ELECTROMYOGRAPHIC FEEDBACK IN THE TREATMENT OF BRUXISM: A PILOT STUDY

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ABSTRACT

ELECTROMYOGRAPHIC FEEDBACK IN THE TREATMENT
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The purpose of this study was to explore potential impact of intensive biofeedback training (EMG) on bruxism. The first question to be dealt with was whether EMG training would, in fact, produce a learned relaxation response in subjects. Assuming that subjects would learn to voluntarily relax their masseter muscles, the next question was whether this voluntary response, if initiated several times during the day and once just before going to sleep at night, would reduce the subject's nocturnal bruxing pattern.

Four subjects, two males and two females, from ages 23 to 55, were selected by a periodontist on the basis of bruxing criteria. Dependent measures included pre- and post-study periodontal observations recorded on a standard dental chart. Pre- and post-study self-report questionnaires were administered, which tested subject awareness of bruxing behavior and subjective reports of pain presumably felt as a result of bruxing. Pre- and post-study masseter muscle tension levels were measured on an Autogenic 1100 EMG machine and data recorded from the illuminated meter of an Autogenic 5100 digital integrator. Bruxism levels throughout the study were measured by the Bruxcore

device (Forgione, 1974), a multicolored, laminated polyvinyl disc covered with microscopic dots and pressed into a tight-fitting plate covering the subject's upper dentition.

The Bruxcore was worn for four consecutive nights, yielding a volumetric index of the subject's bruxing behavior. Baseline EMG levels were measured once per week in a session lasting approximately 20 minutes and consisting of 10 separate 30-second EMG mean readings.

The study followed a time-lagged multiple baseline design to avoid any historical effects on the data as well as to provide a clear representation of the possible effect of biofeedback training on a stabilized baseline measurement of both bruxism and existing muscle tension levels.

After three weeks of baseline data were collected, the subjects underwent intensive biofeedback training. A 30-minute training session was repeated four times per week for three consecutive weeks. During the training sessions, the subjects were instructed in biofeedback procedures and given audio and visual feedback regarding muscle tension. Bruxcore devices were worn concurrently to provide a continuous index of bruxism throughout the study.

After the three-week training program, each subject was asked to return to the office for a one-week follow-up visit consisting of baseline EMG and Bruxcore data collection. This process was repeated one month and six weeks post-treatment, yielding three follow-up measurements. After the sixth week post-treatment, all subjects returned to the periodontist for a post-study observation.

Hypotheses included presumed downward trends in both EMG levels and Bruxcore indexes as well as reductions in bruxing criteria as measured in periodontal observations and subject self-reports. The hypothesized trends in EMG and Bruxcore data were measured by a Testing Against the Trend analysis using Spearman's statistic at the .05 level of confidence. Friedman's Rank Sum was used to determine whether there were any trends across subjects. Subjects' periodontal data were recorded on a dental chart and presented in graphic and descriptive form, as were subject self-report data.

All four subjects demonstrated a learned relaxation response at the .05 level which remained significant throughout the follow-up visits. Only one subject demonstrated a significant downward trend in Bruxcore figures at the .05 level, indicating a reduction in nocturnal bruxism. All four subjects experienced a reduction in bruxing criteria (especially tooth mobility) as witnessed in the post-treatment periodontal observation, and all four subjects reported either less pain or less severe pain as a result of bruxing and/or clenching behavior.

The results of this study offer questionable support for the use of biofeedback training as a main treatment of nocturnal bruxism. However, the data support the existence of a daytime component to the problem. It is this diurnal clenching that appeared to be most amenable to biofeedback-assisted relaxation training, and for which future research efforts should be constructed.

To my parents.

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CHAPTER I

INTRODUCTION

Since the turn of the century, bruxism, the dysfunctional grinding and clenching of teeth, has been of concern to dentists. The effects of bruxism are widely documented and have proven extremely harmful to bruxers' facial musculature and dentition, sometimes leading to development of temporomandibular joint (TMJ) dysfunction and myofacial pain.

Interest has increased in nonfunctional clenching (vertical motion) and grinding (horizontal motion) of teeth because of its likely relationship to oral disease as well as its potential use as a model for studying other psychophysiological disorders (Rugh, 1978).

Bruxism has been found to occur both during a wakeful state and during sleep. It is broadly defined as clenching, tapping, or grinding the teeth: an act not directly related to chewing or swallowing (Nadler, 1960).

Because a variety of terms are used to define bruxism, reports on incidence in the general population tend to be conflicting. Estimates of the prevalence of nocturnal bruxism among adults range from 5.1 percent (Reding et al., 1966) to 88 percent (Olkinuora, 1969),

depending on the method of analysis. No estimates are available of the prevalence of daytime bruxing.

There are conflicting reports concerning differences between sexes in the incidence of bruxism. Walsh (1965) and Molin (1966) reported higher rates of bruxism among females, while Reding (1966), in a study of 2,290 college students, reported no such difference. Solberg (1975) reported that when objective measurements are made, males and females show signs and symptoms of bruxism with the same frequency.

Bruxism occurs in both children and adults, but it is unknown whether adult and childhood bruxism share a common etiology. Abe and Shimakawa (1966), using questionnaire data from 672 adults, reported that 35 percent of childhood bruxers continue to brux as adults. The authors suggest a genetic predisposition, as a result of reports from the same study, that children whose parents grind during sleep are themselves more prone to nocturnal tooth grinding. Support for this hereditary notion can be found from Olkinuora (1969) and Reding (1966), both of whom reported a high incidence of bruxism in subjects whose relatives also grind and clench their teeth. Rugh (1978) states, however, that previous investigations leading to the suggestion of hereditary predisposition have not adequately controlled for environmental and learning factors which may tend to perpetuate the behavior from generation to generation.

The habit of bruxism has many potentially negative effects on dental structures and facial musculature. Abnormal wear of the teeth is most often mentioned as a clinical sign of both acute and chronic

bruxism (Nadler, 1966; Ramfjord & Ash, 1971). Damage to the structures surrounding the teeth (periodontium) has also been cited as a major side effect of chronic bruxism (Glickman, 1972) and has involved recession and inflammation of the gums as well as resorption of the alveolar bone. Hypertrophy of the masticatory muscles, especially the masseter muscle, is most frequently caused by bruxism, and the effects of grinding on the temporomandibular joint have been well documented (Rugh, 1978). Finally, bruxism can give rise to a variety of facial pains, muscle and tooth sensitivity, tooth mobility, and headaches (Vestergaard & Christensen, 1971).

The lack of concise definition as well as an extensive and varied list of symptomatic effects has made it difficult to assess the prevalence of bruxism in the general population accurately. The complexity of bruxism has led researchers to formulate several different theories related to a possible etiology.

Theory

There are three major etiological theories for the origin of bruxism: local/mechanical, psychological, and systemic-hereditary.

Local/Mechanical Theory

Local/mechanical theories propose that minor anatomic defects such as rough cusp ends produce discomfort and tension, which in turn results in bruxism. Missing or elongated teeth, poor restorations, mutilated occlusions, and excessive cuspal inclinations have all been cited as possible causes for grinding behavior (Leof, 1944). More

recent theories have asserted that bruxism results from occlusal abnormalities (Ramfjord & Ash, 1971).

The most common argument for mechanical causes theorizes there is a feedback mechanism between the central nervous system and the proprioceptors of the mandible. If this feedback mechanism is disturbed by an occlusal interference (i.e., poor filling or restoration), an "irritability threshold" may be lowered and grinding may result. When the individual begins to grind his/her teeth, muscular pain is experienced which, in turn, further reduces the irritability threshold and causes even more grinding behavior. A feedback loop evolves in which grinding produces pain that functions to maintain grinding behavior (Ramfjord & Ash, 1971).

Most current dental procedures for treating bruxism are based on this theory. Yet critics have noted that not all bruxers have malocclusions and that not all individuals with malocclusions brux (Olkinuora, 1969).

Psychological Theory

Several different psychological theories have been proposed to account for bruxism. Moulton (1955) and Engel (1951) viewed bruxism and related symptoms from a psychoanalytic perspective and pointed to unconscious sexual and emotional problems as etiological factors. Lefer (1971) suggested that temporomandibular symptoms related to bruxism can be viewed as a conversion reaction usually resulting from emotional conflicts over unconscious needs of dependency or aggressiveness. These conceptual models have not been borne out by experimental verification.

After reviewing eight separate investigations aimed at pinpointing specific personality characteristics of toothgrinders, Olkinuora (1969) reported that too many contradictory results were found to establish a "Bruxism Profile." Personality characteristics were examined by Olkinuora (1969) using the MMPI, Blacky Pictures, Rosenweig Picture Frustration Test, Cornell Medical Index, Maudsley Personality Inventory, the Manifest Anxiety Test, the Guilford-Zimmerman Scale, and the Rorschach Content Test. In a similar review using many of the same tests, Reding (1968) concluded it is impossible to identify a typical "bruxist personality." This conclusion reached by two separate researchers seems consistent with an apparent trend away from attempts to correlate specific personality characteristics with psychophysiological disorders.

In a more recent effort, Olkinuora (1972) differentiated between two types of bruxers, which he termed "strain bruxers" and "nonstrain bruxers." Strain bruxers were found to be more often clenchers and daytime grinders than the nonstrain variety, which was comprised more of hereditarily predisposed nocturnal grinders of a less overtly aggressive nature.

Reding (1968) pointed out the differences between daytime clenchers and nighttime grinders and contended that further study of bruxism should separate the two seemingly unrelated functions.

Olkinuora (1972) also pointed out that bruxism is more common among "anxious" populations. Research exploring anxiety as an etiological factor has been slowed by the general lack of agreed-upon definitions of either anxiety or bruxism. However, there is reasonably

strong evidence to suggest that emotional states such as anxiety can elicit muscular tension, which may in turn play a central role in grinding and clenching behavior (Yemm, 1968, 1971).

Systemic and Hereditary Theories

Early researchers believed that organic brain damage, hyperthyroidism, allergy, endocrine disorders, and hereditary factors might be related to bruxism (Marie & Pietkiewicz, 1907). More recent all-night electroencephalographic studies point to the experience of autonomic discharge on the part of the bruxers. Teeth grinding, it is thought, could be triggered by external or internal physical or emotional sleep-disturbing stimuli (Satoh & Harada, 1973; Reding & Zimmerman, 1968).

Throughout the literature there is no clear-cut theory of the etiology of bruxism. One consistent element, however, is the fact that bruxism requires massive amounts of tension from the muscles of mastication, primarily the masseter muscle. Yemm (1976) cites muscle hyperactivity as a possible origin of bruxism in combination with the presence of externally stressful environments. Upon completion of his study, he concluded there may exist a "central origin" whereby certain people who are predisposed to respond to stress will react by producing a sustained jaw muscle hyperactivity which, in the long run, produces tissue damage, pain, and subsequent clenching and/or grinding to avoid that pain.

This study is built on the common element of muscle tension. If a bruxer can be taught to voluntarily relax the muscles of

mastication, it may then be possible to positively affect the frequency of the habit and the deleterious effects of its chronicity.

Current Treatment Modalities

Mikami (1977) posits four main objectives in the treatment of bruxism: (a) reduce psychic tension, (b) treat the signs and symptoms, (c) minimize occlusal irritations, and (d) break neuromuscular habit patterns. While three of the four objectives are noninvasive in nature, Mikami's emphasis, as a dentist, is to reduce occlusal deficiencies by occlusal adjustment. This procedure is supported in the literature by Ramfjord (1961), Dawson (1974), and Melkas (1971), among others.

Ayer and Gale (1969) developed a procedure called massed negative practice during which the bruxing subject was told to forcibly bite down for a period of 60 seconds several times before going to bed in the evening in an attempt to fatigue the masseter muscles to the point of extinguishing the nocturnal habit. This procedure was later refined by Ayer and Levin (1973) and achieved a reported 75 percent reduction in bruxing behavior.

Heller and Forgione (1975) developed a procedure for measuring frequency and intensity of bruxing behavior. They used a device (called a Bruxcore) to replicate the findings of Ayer and Levin with regard to the efficacy of massed negative practice, but found no significant results in contrast to the 75 percent success rate already mentioned. This large discrepancy in findings was attributed to the fact that previous research in bruxism relied upon self-report in

terms of symptom reduction, whereas implementation of the Bruxcore tended to objectify the data.

After conceptualizing the cause of bruxism to be straight masseter hypertension, Goldberg (1973) developed a procedure for relaxing the muscles of mastication by employing Jacobson's progressive relaxation and hypnotically induced deep relaxation. However, Goldberg's techniques are recorded only in case studies and do not lend themselves readily to scientific evaluation.

Connistraci (1976) reported a procedure employing biofeedback training supplemented with progressive relaxation, autogenic training, and yogic meditational mantrum techniques. Results of this procedure were reported as being very good. All the subjects who completed the training reported symptom relief and positive feelings about what they had learned. Again, with the emphasis on self-report and questionnaires, any scientific appraisal of this multidisciplined procedure is left for future consideration.

Under the classification of dental treatments, the following techniques can be found.

Occlusal adjustment treatment, as reported by Ramfjord (1961), was found to eliminate bruxism in all 34 patients studied. However, 100 percent reduction of bruxism may be difficult to achieve in all cases. Ramfjord and Ash (1971) later reported that, because of the high degree of muscle tonus in bruxers, proper occlusal adjustment may be difficult to achieve.

Another widely recommended technique is the application of occlusal appliances (i.e., occlusal splints and bite plates). The

presumed advantages of these devices are that they are easy and relatively inexpensive to fabricate, and that they will prevent further destruction or wear to teeth and supportive structures (Ramfjord & Ash, 1971).

Recently, several nondental treatments of bruxism have been explored.

In the last 10 years, biofeedback has been employed as a treatment modality. Mixed results have been reported. Solberg and Rugh (1972) asked 15 subjects to wear an EMG biofeedback unit from two to seven days. The authors reported that two-thirds of their subjects improved significantly during the training period. Improvements were measured solely on the basis of self-report.

More support for this technique was presented by Budyznski and Stoyva (1973). They demonstrated (with eight male subjects) that biofeedback training enabled all subjects to reach low EMG levels in masseter muscles within a 20-minute period. No materials were collected to suggest that any of these subjects were bruxers, and no follow-up was reported to substantiate the learned EMG reductions over time.

An electromyographic biofeedback technique employed by Kardachi and Clarke (1977) seems to be the most recent attempt at controlling bruxism through voluntary reduction of masseter tension. In this study, subjects wore the monitors at night and were alerted, vis-à-vis an auditory alarm, whenever the masticatory muscles produced electrical activity beyond an established criterion. The subject then relaxed the masseter muscle below threshold and returned to sleep.

Results of this study demonstrated that all but one of nine subjects significantly reduced the amount of muscle activity (presumably caused by bruxism) as evidenced by receiving progressively fewer alarms per night.

Once again, no direct measurement of the incidence of bruxism was reported. It should also be noted that the subjects reported negative sleep-loss effects caused by continual waking during deep stages of sleep and return to a light-sleep stage. The authors suggested the need for a trained response that might be employed during earlier phases of sleep when the subject might better exercise voluntary control of muscle tension and avoid the deleterious long-term effects of sleep loss.

Conclusion

Bruxism has been and continues to be a difficult habit to measure and treat. Incidence reports of 5 to 88 percent vary according to populations, and such reports are limited to dental patients and college students. Etiological theories include psychological, systemic, and structural problems, and any possible combinations of the three. Problems and possible symptoms arising from the chronic presence of the behavior include: unusual wear in dentition, alveolar bone absorption, decreased ability to open one's mouth, headache, myofacial pain, the TMJ problems.

Most treatment of bruxism has been performed by dentists and consists primarily of the use of oral appliances (splints and bite guards) and occlusal adjustments (grinding teeth down to reduce

premature contact and to assure ample "freeway space"). Behavioral treatments of bruxism include massed practice (to fatigue the muscles of mastication) and the application of general relaxation techniques. All these procedures have been based on clinical observations by dentists and one or another form of patient self-report, leaving in doubt any objective evaluation of potential treatment benefits.

Need

Throughout years of investigation, bruxism has received multi-disciplinary attention from physicians, dentists, physiologists, and psychologists. A review of the literature suggests a complex etiology with possible roots in all four disciplines. The problem is widespread throughout the general population, and much has been written and said about the deleterious effects of its chronicity to bruxers' oral structures and psychological well-being.

Perhaps the greatest problems with research to date have been (a) the lack of objectivity in defining and quantifying the behavior and (b) the absence of accurate measurements of treatment effects.

Recent advances in the development of biofeedback (physiological self-monitoring) have made it possible to teach subjects to voluntarily relax major muscle groups including the muscles of mastication. It may be possible to utilize this relaxation training in the treatment of bruxism. Several recent projects have demonstrated subjects' ability to relax tense masseter muscles, but there are no follow-up data to support the notion that such learning is permanent. There also are no objective data to demonstrate that such a learned

"relaxation response" has a positive impact on bruxing behavior other than the subject's self-report to that effect.

Purpose

The purpose of this study is to explore the efficacy of intensive biofeedback training (EMG) in teaching subjects a specific and measurable "relaxation response" on the masseter muscle and to measure the effects of subsequent application of this "learned" response to the incidence of bruxing behavior.

Follow-up data will be collected on the long-term nature of a learned response and its relationship to bruxing behavior.

The methodological purpose of this study is to respond to problems of objectively measuring bruxism in a nocturnal setting, by using the "Bruxcore" (Heller & Forgione, 1973), a device that promises to yield a more objective scoring of bruxing behavior than has been previously available.

Any refinements in techniques and procedures of biofeedback training and/or measurement will also be reported.

Hypotheses

The following are the research hypotheses, which are stated again in Chapter III and for which results are reported in Chapter IV.

H_1: There will be a decreasing trend in EMG levels from baseline to treatment and follow-up.

H_2: There will be a decreasing trend in bruxcore figures from baseline to treatment and follow-up.

H_3: EMG levels will be positively correlated to bruxcore figures across baseline treatment and follow-up phases.

H_4: The post-treatment clinical observation will report less bruxing criteria than the pre-treatment observation.

H_5: Subjects will report less bruxing criteria present after the study than before the study.

In order to test the difference between pre- and post-treatment EMG levels, baseline EMG data were collected and compared to EMG training levels and post-treatment following EMG scores.

To measure the effect of the EMG training on bruxism, a pre-, during-, and post-treatment baseline level of grinding was obtained by using a Bruxcore device (Heller & Forgione, 1973). Comparisons of the grinding levels pre-, during-, and post-EMG treatment served as the basis for investigating the possible impact of the treatment on the subjects' level of bruxism.

Another test of the possible benefits of EMG training in the treatment of bruxism consisted of a pre- and post-treatment clinical observation by Kenneth Wisser, D.M.D. (licensed periodontist). These observations included measures of tooth mobility, mouth-opening ability, hypertrophy of the masseter muscle, and descriptions of wear facets before and after the treatment process.

Finally, a self-report questionnaire was administered pre- and post-treatment to determine the subjects' impressions with regard to any possible differences in frequency and severity of bruxing behavior and myofacial pain as a result of the treatment process.

Overview

The literature pertinent to the theory and research of bruxism is presented in Chapter II, with a focus on the areas of definition

of the behavior, epidemiology, etiology, current treatment modalities, and measurement problems. Chapter III describes the subject sample, instrumentation (EMG and Bruxcore measurement), training procedures, data-collection procedure, the hypotheses, the analyses, and experimental design. The analysis of data for each hypothesis will be presented in Chapter IV, along with correlational and anecdotal data regarding each subject. Chapter V includes a summary of this investigation, a discussion of the findings and limitations as well as implications for further practice and/or research of the treatment of bruxism with biofeedback.

CHAPTER II

REVIEW OF LITERATURE

This study focuses on the effects of voluntary relaxation of the masseter muscles on the behavior of bruxism. To detail the theory and research upon which this study is based, literature on the definition of the behavior, epidemiology, effects and symptoms, etiology, and current treatment modalities of bruxism will be reviewed.

Definition

The most widely accepted definition of bruxism is that of Nadler (1966): "A nonfunctional voluntary or involuntary mandibular movement, which may occur during the day or night, manifested by the occasional or habitual grinding, clenching or clicking of the teeth."

This phenomenon has appeared in the literature by other names, including bruxomania (Marie & Pietkiewicz, 1907), occlusal habit neurosis (Tischler, 1928), occlusal neurosis (Forhman, 1931), and finally, in the same year, bruxism (Frohman, 1931). Recent German literature employs the term parafunction (Bundgaard-Jörgensen, 1950); however, parafunction has come to denote more generalized oral habits, including finger sucking, nail biting, and even the playing of wind instruments.

The habit of bruxism has been more clearly defined by Ramfjord and Ash (1971) as nonfunctional gnashing and grinding in eccentric excursions or "eccentric bruxism" and clenching as "centric bruxism." They further state that grinding of teeth is more common during the night, while clenching occurs more often during the day. This observation was substantaited by Reding et al. (1966) when in their own studies they noticed marked psychological and neurophysiological differences between nocturnal and diurnal. They chose to use the term bruxism to denote only nocturnal grinding.

Olkinuora (1969) posited that bruxism is a universal phenomenon which only becomes pathological as a result of the intensity and persistence of the habit. He goes on to suggest that bruxing may be conceptualized as a continuum, one end of which represents the nonfunctional gnashing or grinding of teeth occurring in any human being during the day or night, with the most noxious and harmful forms being at the other extreme.

Epidemiology

Since the concept of bruxism has been defined using so many different terms, and considering the unconscious nature of the problem as well as the subjective observation, direct measures of incidence and prevalence are obscure at best (Olkinuora, 1969).

Most early descriptions of the syndrome came from dentists' populations. Boyens (1940) and Leof (1944) reported the symptom to be present in 78 and 81 percent of their cases, respectively; yet total numbers were not mentioned in either. Bundgaard-Jörgensen (1950)

treated 496 patients and reported that 88 percent bruxed. Only 42 patients did not display some form of periodontal and/or temporomandibular joint (TMJ) problems. This seems consistent with Peterson and Dunkin (1956), who found an incidence of approximately 80 percent, but is grossly at odds with Posselt's (1960) report of only 20 percent incidence. This discrepancy may have been due to the lack of a clear definition of bruxism in the early 1950s since it seems that reports appearing from 1960 to the present cite figures closer to the 5-10 percent range.

Reding et al. (1966) provided perhaps the most comprehensive epidemiological study to date when they surveyed 2,290 undergraduate and graduate students at the University of Chicago. They found 117 current bruxers (5.1 percent) in that group.

Walsh (1965) and Molin and Levi (1966) suggested that the frequency of bruxism is greater among women than men. Reding's (1966) study supported that difference, although not with statistical significance.

Several researchers have used incidence reports to demonstrate a potential hereditary link in the bruxism habit. Abe and Shimawakwa (1966) found that 11.6 percent of 336 three-year-olds in their study bruxed, as did 18.3 percent of the 672 parents of those children. They demonstrated (at $p < .05$) that the children of bruxing parents tended to brux more often than those of nonbruxing parents.

Reding et al. (1966) supported the hereditary notion with his findings of a statistically significant association between current

bruxism on the part of the subject and either current or historical bruxism in blood relatives.

Effects and Symptoms of Bruxism

According to Melkas (1971), the bruxing patient may or may not be consciously aware of a bruxing habit, but intense chronic grinding may cause destruction of the clinical crown of the lower anterior teeth. The periodontal ligament may become enlarged and the patient may complain of facial or TMJ pain which can radiate as far as the lower neck.

Chronic bruxism is an important factor in periodontal disease and dysfunction. It can cause lesions in the hard tissues of the teeth, in their supporting structures, and in the temporomandibular joint. Other signs of bruxism include jaw muscle fatigue upon waking, tenderness of the masseter muscle upon palpation, increased tooth mobility, and audible clicking sounds emanating from the jaw area (Connistraci, 1976).

Nadler (1966) cites the following effects: attrition and increased tooth mobility, alveolar bone reactions (including bone absorption), and clicking sounds. Ramfjord (1961) suggests the following as effects of chronic grinding: the appearance of wear facets indicating nonmasticatory patterns of occlusal wear; increased muscle tonus and involuntary resistance to manipulation of the mandible; hypertrophy of the masseter muscles; increased mobility of teeth; clicking sounds; tired jaw muscles; pulpal sensitivity to cold; and a tendency to bite the cheeks, lips, or gums.

Bruxism has also been credited with being a possible cause of many chronic headache symptoms (Berlin & Dressner, 1960; Vestergaard Christensen, 1971).

Rugh and Solberg (1976) report that habitual bruxism can cause TMJ symptoms resulting in a myofacial pain dysfunction syndrome which may be evidenced by the subject's reports of tenderness in the masseter muscle and limited ability to open the mouth.

Xhonga (1973) demonstrated a relationship between dental erosion and bruxism. He compared 15 known bruxers with 15 controls matched for age and sex. By comparing the upper and lower dentitions, with aid of an electron microscope, he demonstrated that shiny wear-facets on the teeth of the bruxers were the result of tiny enamel crystals which had been ground off during bruxing. These enamel crystals, in a pulverized form, serve as a polishing or abrasive medium in bruxism. The pulverized enamel particles are capable of creating damage to the enamel and to the exposed dentine when heavy grinding forces are applied. Less pronounced grooves or scratches were spotted on the nonbruxers. Xhonga (1973) went on to point out that the amount of damage caused by the particles was proportional to the force applied by the muscles of mastication. The role of muscle tension in the origin and evolution of bruxism was quickly supported by several researchers, who pointed out that the presence of excess masseter tension could cause or exacerbate bruxing behavior.

Yemm (1975) concluded from his research of masticatory dysfunction patients that masseter pain was caused by voluntary or involuntary hyperactivity of the masseter muscle after grinding behavior.

Vestergaard Christensen (1971) demonstrated that voluntary tooth grinding can initiate tenderness in the muscles following the period of hyperactivity. A rise in tissue fluid pressure was found within the masseter muscle on the side on which tooth grinding was occurring. This was interpreted as a sign of increased extracellular fluid pressure due to the development of a state of edema caused, presumably, by the grinding behavior.

Bruxism has been directly related to tooth wear, tooth mobility, myofacial pain, temporomandibular joint problems, and a myriad of diseases of the masticatory muscles and gingival areas.

Etiology

While a single etiology for bruxism has not been established, there are several recurring theories throughout the literature, including: organic abnormalities or deficiencies (i.e., malocclusions); psychological causes, including excessive tension, stress reactions, and repressed emotions (usually anger); and finally a systemic theory viewing bruxism as either a result of some other malady (parasites, infections, or disease) or as a neurophysiological reaction to autonomic nervous system arousal.

Nadler (1957) classified the known etiological factors into the following four categories: (a) local dental components in which the grinding habit may be attributed to certain intraoral factors, including malformed restorations, malocclusions, and unpolished surfaces of porcelain dentures which are thought to provide a "trigger" to start the dysfunctional process of mastication; (b) psychological-emotional

elements associated with neurotic symptoms purportedly having developed from excessive stress-producing factors, such as noise, congestion, and rush of city life; (c) systemic factors resulting from diseases including epilepsy, tetanus, and the presence of intestinal parasites; and (d) combination of excessive nervous tension and a poor dental restoration or any other dental factor acting to inhibit the "free-way space" (space between the teeth when the jaw is in a closed position).

Local/Dental Factors

In a review of local dental factors, Melkas (1971) cited the following list:

1. Discrepancies between centric relation and occlusion
2. Tipped or otherwise malposed teeth
3. High restorations
4. Chronic inflammation of the periodontal membrane
5. Differences in or uneven eruption of teeth
6. Presence of unusually steep cusps
7. Tight occlusion
8. Overcarving of restorations
9. Rough or chipped enamel or restoration

Ramfjord (1961) looked at the issue of discrepancies in occlusions when he studied severe bruxers before and after occlusal adjustment. He successfully eliminated bruxing by adjusting occlusions. He said, "discrepancies in occlusion are accompanied by asynchronous contraction or sustained strain in the temporal and masseter muscles

during swallowing." He went on to suggest that the uncomfortable and traumatic position caused by the malocclusion during closure causes the patient to compensate by "fighting back" (bruxing).

Melkas (1971) suggested that teeth that are tipped or otherwise malposed may initiate bruxing since height is increased in some areas, and disrupted normal closure results in a slide or shift of the mandible. He stated the same could hold true of high restorations.

Some patients with chronic periodontitis, according to Malkas (1971), experience a low-grade uncomfortable feeling in the area involved. Patients may describe the sensation only as persistent gnawing or itching. In order to alleviate discomfort they clench the teeth heavily, and have reported sustaining such behavior for long periods. If teeth erupt unevenly, a high contacting surface may manifest itself and incline the patient to eliminate the "prematurity" by bruxing it off. Steep cusps become sharp subsequent to long wear and may irritate the patient who compensates by trying to flatten them (bruxism). Tight occlusion is said to confine the patient. Not having the freedom to roam laterally without running into interferences, they attempt to free up the occlusion by bruxing.

Several authors (Rugh, 1978; Melkas, 1971; Olkinuora, 1969) suggest that poor restorations and the practice of overcarving restorations provide areas of contact which presumably trigger masticatory tension aimed at grinding the surfaces flat.

While local/dental problems make up only one suggested portion of potential etiological factors involved in bruxism, most current treatments for bruxism are in the form of some occlusal repair or

dental procedure. Throughout the list of possible local causes, muscle tension was repeatedly mentioned as playing a prime role in the initiation or maintenance of bruxing behavior.

Experimental evidence to support the role of occlusal deficiencies in the etiology of bruxism has been reported by Jankelsom (1955), who produced bruxism in 10 human subjects by placing acrylic cement on the occlusal surfaces of the teeth. Ramfjord (1961) produced bruxism in 10 Rhesus monkeys by placing high occlusal amalgam fillings in the lower first molars. The bruxism stopped when the monkeys had completely ground down the filling through vigorous grinding.

Psychological/Emotional

A number of authors have suggested that bruxism is a manifestation of psychological dysfunction. The majority of these views have come from the psychoanalytic tradition, which has hypothesized that bruxism is an unconscious expression of repressed oral aggression (Walsh, 1965; Pond, 1968). According to this viewpoint, oral-receptive and oral-aggressive individuals are most likely to brux in an attempt to gratify oral pleasures denied in childhood (Frohman, 1931).

Several authors have suggested that two basic personality types may be found among bruxers (Lefer, 1971; Moulton, 1955). One type can be characterized as ingratiating and passive dependent, whose frustrations presumably lead them to reactive anger and hostility. For these individuals, clenching the jaw is thought to be an attempt to aid repression or suppression of anger. The second type was described as successful, self-supporting, perfectionistic, obsessive and

domineering, for whom grinding functions as an unconscious expression of anxiety avoided during normal daytime activity.

There have been several attempts to validate clinical observations of psychological disturbances among bruxists.

Thaller (1960) compared 25 nonbruxing patients with 25 bruxists using the Cornell Medical Index and found a positive correlation between anxiety and bruxism. Thaller, Rosen, and Saltzman (1967) replicated the same study using 42 bruxers and 44 controls. They found that not only was there a high incidence of anxiety in the bruxers but also that they tended to be "intropunitive."

Vernallis (1955) compared a group of 40 bruxist students with a control group matched for age and sex, intelligence, and educational status. Both groups were administered the Rorschach, the Taylor Manifest Anxiety Scale, the Hypomania Scale of the Minnesota Multiphasic Inventory, the Guilford-Zimmerman Temperament Survey, and the Blacky Picture Test. Bruxism was found to be positively correlated with anxiety, hostility, and hyperactivity.

Molin and Levi (1966), using 103 bruxists and the Maudsley Personality Inventory, found that bruxists had more symptoms of depression and anxiety and significantly more frequent occurrences of muscle tension. The tension described was not limited to the masticatory system but occurred in other areas of the body as well.

In another study of 104 girls and 88 boys 12 years of age, Linquist (1972) found that bruxistic children (as reported by parents witnessing nocturnal grinding) displayed significantly more stress symptoms and nervous disorders than did nonbruxists.

Perhaps the most sophisticated study of the psychological factors in bruxism has been reported by Olkinuora (1972a, 1972b, 1972c) in a series of three related studies. Olkinuora posited there are two distinct types of bruxers. People who grind rather than clench at night instead of during the day and have a genetic predisposition to do so are called "Nonstrain Bruxers." Bruxers who clench during the day or night in response to stress and whose scores on psychological tests are markedly higher on scales measuring emotional disturbance and aggression are termed "Strain Bruxers."

To obtain these data, Olkinuora used the same 69 subjects for all three of his studies and measured performance on the following dependent measures: psychiatric interview, the California Multiphasic Inventory, the Hand Test, the Digit Symbol subtest of the Weschler Adult Intelligence Scale, the Arrow Dot Test, the Buss-Durkee Hostility-Guilt Inventory, the Rosenzweig Picture Frustration Study, and the Alanen Rating Scale. Patients were determined to be "Strain Bruxists" if they reported a connection between beginning of bruxism, severity of bruxism, or pains and/or tiredness in the masticatory muscles and emotional disturbance. "Nonstrain Bruxists" were those for whom the connection did not apply.

In the first two studies, Olkinuora (1972a, 1972b) sought to differentiate between Strain and Nonstrain bruxists. In the third study, Olkinuora (1972c) compared the entire group of 69 bruxing subjects with 42 controls matched for age and sex. From this comparison he concluded that (a) strain bruxists are more emotionally disturbed, have more muscle symptoms, and tend to exhibit more hostility and

aggression than the nonstrain variety; (b) bruxists in general tend to be more emotionally imbalanced, have more headaches and muscle pains, are more meticulous, and achieve more success in school-related matters than do the controls; and (c) nonstrain bruxists tend to be less aggressive, are from a higher social class, and have more relatives who brux than do controls.

The psychological data tend to suggest, although not with unanimity, that a significant number of bruxists are under stress, either from environmental situations or intrapersonal psychological sources. Olkinuora (1972) and Reding (1968) suggest strongly there are at least two distinct types of bruxers. The groups can be differentiated by when they exhibit the habit (day or night) and their degree of manifested aggressiveness and hostility. These findings remain hypotheses rather than established facts due to the lack of validity and reliability found in the dependent measures. Once again, however, it should be noted that most authors agree that stress and excess muscle tension are prime etiological factors in bruxing behavior, combined with differences in character and personality dynamics.

Systemic/Neurophysiological

Early investigators of bruxism felt the disorder was caused by various systemic dysfunctions. Marie and Pretkiewicz (1907), for example, believed that "bruxomania was the result of cortical lesions and occasionally by disturbances of the medulla and pons." They reported bruxing behavior in patients suffering such disorders as paralysis, spasmodic hemiplegia of infancy, chorea, nephritis, and meningitis.

Nadler (1957, 1960) noted that hyperacid urine, hyperthyroidism, endocrine disorders, and hereditary factors may be related to bruxism.

Reding and Zimmerman (1968) pointed out that, although bruxism has been reported in patients with systemic diseases, one cannot infer from these data that bruxism is the result of systemic or neurological dysfunctions.

Since bruxism often appears in several generations of a family unit, many authors have put forth a genetic-hereditary basis for the disorder.

In 1966, Abe and Shimakiawa found that children of bruxistic parents tended to brux more often than children whose parents were not bruxers. Reding and Zimmerman (1966) cited a statistically significant prevalence of nocturnal bruxism among blood relatives of nocturnal bruxists. Perhaps the most recent addition to these data has been from Olkinuora (1972), who suggested that his "nonstrain" bruxing group tended to have more bruxers as blood relatives than the general population and that the "strain bruxers" (daytime clenchers) did not.

It remains difficult to determine the exact origin or method of transmission of bruxism. However, several researchers have suggested a neurophysiological basis which points to autonomic arousal as a possible trigger for bruxing behavior.

Kawamura, Tsukamoto, and Miyoshi (1961) sought to demonstrate a physiological basis for bruxism. They induced grinding behavior in rabbits by electrically stimulating the anteromedial aspect of the cortex, presumed to be the cortical jaw motor area. They found the

grinding to exhibit a regular rhythm that was slower than that of normal mastication.

In a follow-up study using dogs, Kawamura (1961) demonstrated that stimulating limbic system structures may also induce grinding. As a result of this study, they described a feedback mechanism in the brain stem as controlling the function of masticatory muscles. They were further able to isolate particular points on the brain stem that correspond to the contraction of each trigeminal motor nucleus, thus causing the grinding motion found in bruxistic behavior.

Scharer (1971) stimulated the hypothalmic area of the brain and reported a bruxing-like motion in the jaw muscles of rabbits. Scharer found that the rhythmic, forceful contractions of long duration, induced by stimulating the lateral hypothalmic area, appeared only when the cortex was damaged. If the cortex was intact, brief chewing motions were produced. He suggested that the cortex (responsible for higher-order consciousness) has an inhibitory action on jaw muscle activity during normal functioning. However, if this inhibitory function is altered or removed, bruxism can be elicited from stimulation of the same site where chewing-like movements had been induced.

Further support for the notion that bruxism is related to cortical and autonomic mechanisms comes from Yavelow, Forster, and Winninger (1973) in their discussions of mandibular retraining.

> Bruxing and clenching stimulate many motor units, and the resulting force will depend upon the intensity of the pressure, the length of time the pressure is applied, and the mandibular position. The greater the number of motor units stimulated, the greater will be the total strength or pressure exerted upon the teeth. Muscular reaction is inhibited or increased

> by the cortical and/or spinal reticular systems. Muscular reaction is tempered by the limbic system with its mechanism for pleasure and displeasure.

Yavelow (1973) went on to suggest that a patient's emotional difficulties, his/her inability to cope with a stressful situation, can be expressed in anxiety, fear, dependency, or other maladaptive manifestations. These emotions are expressed physically through body tension which may be manifested in abnormal muscular movements or pressures that may precipitate bruxing.

In support of the idea that stress may cause abnormal muscle tension which may in turn evolve into a bruxing pattern, Yemm (1968) demonstrated that, in times of stress, muscle tension increases. Using electromyographic techniques to monitor activity of mandibular elevator muscles in normal human subjects while performing difficult tasks, it was shown that the muscles were more active during the task than when the subject was relaxed.

The task consisted of a series of lights and buttons. The lights were illuminated and the subject was asked to depress the corresponding button over a period of two minutes with intervening rests. Mistakes were marked with a feedback tone alerting the subject to his error. The occurrence of a mistake was associated with an increase in electrical activity of the masseter muscle at the time of the mistake.

Yemm (1969) went on to demonstrate that when normal subjects were asked to repeat the task for a 30-minute period with number of mistakes held constant, the muscular tension response diminished. A group of 10 bruxers did not show the progressive diminution of muscular

response, with most producing as great a response at the end of the session as at the beginning.

The study demonstrated that masticatory dysfunction patients may be less able to adapt to environmental stress. Many of the subjects volunteered that their teeth were clenched at times during the session but none of them reported actively trying to relax their jaw muscles during the task assigned. As a result of this study, Yemm suggests that the clinical picture of masticatory dysfunction (bruxism) could arise as a consequence of muscle hyperactivity in the jaw muscles.

In support of this muscle tension theory, Perry et al. (1960) artificially produced stressful situations for dental students who were being monitored electromyographically (masseter placement). It was clearly demonstrated that increased muscle tonus resulted when more stress was exerted such as contact from the Dean of the Dental School regarding an upcoming exam.

Most bruxism occurs when the subject is asleep. Takahama (1961) noticed in an all-night electroencephalographic and electromyographic study of bruxism that rythmic activity of a kind that does not occur during mastication in the waking state can be found in the masseter muscle during sleep. In the same study, Takahama pointed out that the voluntary discharges were ascertainable in masticatory and wrist muscles before bruxism. The author also noted the incidence of brain waves indicating increased cortical activity preceding the aforementioned changes in pulse and respiration, leading him to surmise a connection with arousal of the autonomic nervous system.

From all-night electroencephalographic studies, Reding et al. (1968) indicated that bruxism could occur in any sleep stage except stage one (Descending Stage), which acts to dispel earlier hypotheses suggesting the occurrence of bruxism exclusively during R.E.M. (Rapid Eye Movement), yet adds to the idea that autonomic arousal found in other sleep disturbances (i.e., somnambulism and enuresis) may also play an integral part in the etiology of bruxism.

The use of electromyographic recording of masseter contraction in times of severe stress seems to indicate a direct relationship between stress and muscle tension. Previous studies have cited the connection between increased muscle tension and increased bruxing behaviors (Perry, 1960; Yemm, 1975). Therefore, it seems reasonable to suggest that, while no clear etiology exists for bruxing behavior, any treatment of the problem must certainly include an attempt to control excess tension in the muscles of mastication. The relationship between excess muscle tension and stress has led several researchers to believe that learning voluntary control of masticatory muscles will provide the necessary inroad to effective treatment and subsequent reduction of bruxing behavior. Several of the treatment modalities testing the muscle tension concept will be discussed in the following section.

Treatment

Since there exists no clear etiology of bruxism upon which to base research of treatment modalities, it has followed that recent and current treatment strategies have adhered to the three major

categories listed in the review of etiological literature (local, psychological, systemic). Treatment can be classified into the following general categories:

1. Dental (including occlusal adjustment, splinting, and oral appliances),
2. Psychological/behavioral, and
3. Physiological monitoring and/or training to reduce muscular tension.

Dental

Treatments fitting this category are an outgrowth of the local/mechanical etiological theories of bruxism. The goal of dental treatment is the elimination of trigger factors leading to bruxism and/or an attempt to prevent further damage to the teeth and supporting tissues of known bruxists.

Perhaps the most popular dental treatment is the occlusal adjustment (i.e., grinding the teeth in such a way as to guarantee no premature contact or violations of the patient's freeway space).

Ramfjord and Ash (1971) noted that occlusal adjustment eliminated bruxism in all 34 patients studied. However, correct occlusal adjustment may be difficult to achieve. In a subsequent project they found that the high degree of muscle tonus found in bruxism patients may make their achievement of proper occlusion difficult. It was stated in the same study that bruxism patients tend to be especially sensitive to changes in occlusion, making the identification and elimination of trigger factors difficult.

A second highly used dental technique involves occlusal applicances, typically constructed from hard acrylic. These devices are to be worn

at night by bruxers in an attempt to interrupt the feedback mechanism maintaining bruxism, thereby curing the problem. The clear advantage of using the occlusal appliance (i.e., splint, bite guard, bite plane) is the ease with which they are fabricated and the tendency toward prevention of further wear to teeth and supporting structures (Sugarman & Sugarman, 1970).

Measurement of the effectiveness of dental treatments of bruxism has been limited to clinical observation and patient self-report.

Psychological/Behavioral

Due to the large number of researchers and practitioners who have conceptualized bruxism as a response to emotional dysfunction, psychotherapy or psychoanalysis has often been recommended for dealing with the hypothesized underlying emotional problem (Thaller, 1965; Shapiro, 1965; Walsh, 1965). No data, however, have been forthcoming regarding the efficacy of such treatments or their lasting effects.

Perhaps a more promising conceptual framework with which to treat bruxism is that of learning theory.

Heller and Strang (1973) developed a classical conditioning model for the treatment of bruxism. After determining a baseline rate of grinding, a treatment was instituted in which a sound blast was administered for three seconds through an earphone whenever the rate of grinding exceeded three grinds per five-second interval. This procedure was followed for seven treatment sessions during which the rate of grinding dropped from 1.75 grinds per minute to .65 grinds per minute. To account for a possible placebo effect, the treatment was

withdrawn for six sessions, after which it was noted that the grinding rate nearly doubled to 1.2 grinds per minute. The reintroduction of the device for 22 sessions decreased the grinding rate to a more stable .45 grinds per minute, at which point treatment was terminated.

Strang and Heller's treatment must be evaluated more closely since only high-intensity grinds produced the audible aversive stimulus. It is entirely possible the subject continued to brux at a lower level of intensity (but perhaps with more frequency), thereby successfully avoiding the threshold above which the noxious stimulus was administered. At the end of their study, Heller and Strang (1973) suggested that an alternative procedure might involve use of the masseter muscle to operate the conditioning device. This would allow the experimenter to more accurately mesure the incidence and intensity of bruxing behavior. A threshold could be established below which no bruxing behavior could possibly occur.

Ayer and Gale (1969), drawing on Hullian concepts of conditioned and reactive inhibition, developed a process of massed negative practice. In massed negative practice, repeated evocation of the response (bruxing) elicits muscular pain and discomfort. Cessation of grinding is reinforced due to the decrease of muscle fatigue and resulting pain. Results of this technique were reported in a single case study of a 26-year-old white male college student whose wife reported a cessation of his bruxing after the tenth night. His practice included tensing for one minute followed by one minute of relaxation repeated for a block of six trials with the entire cycle repeated five times per day for two weeks.

The one-minute tension period tended to cause pain. Ayer and Levin (1973) attempted to replicate the findings of the case report with a larger sample, using shorter clenching and relaxing times. Fourteen female subjects ranging in age from 14 to 56 were instructed to clench and relax for five-second intervals in a block of six trials. They were to repeat each block six times per day for two weeks. The data showed that 11 subjects eliminated the bruxing behavior within nine to ten days, a statistically significant result. No follow-up data were reported.

A more recent study of the massed practice technique by Heller and Forgione (1975) attempted to avoid the methodological problems of using no control group and no clearly objective measurement of the bruxing behavior itself. They employed a bite plate developed by Forgione (1974) as an objective measure of bruxism and utilized a no-treatment control group while measuring the efficacy of the abbreviated massed practice technique (Ayer & Levin, 1973). No difference was found between the treatment group and the control group, a finding the authors attributed to the great variance in frequency and intensity of bruxism from night to night, and to the increased objectivity of their dependent measure.

Physiological Monitoring and Biofeedback

This particular line of research assumes that subjects given biofeedback training will better discriminate between high and low levels of muscle tension. It also assumes that subjects under high

levels of tension will be able to initiate responses (e.g., relaxation) that inhibit tension (Glaros & Rao, 1977).

There have been several studies using electromyographic feedback in the treatment of bruxism.

Solberg and Rugh (1972) asked 15 subjects to wear an EMG biofeedback device for two to seven days. The authors reported that 10 subjects significantly reduced their bruxing behavior.

Subjects reported that using the biofeedback unit increased their awareness of muscle tension and that they eventually learned to reduce the excess masseter tension and ultimately reduce their involuntary bruxing response. The results of this study were based upon self-report of both EMG levels of muscle tension and bruxism episodes.

From this study, Solberg and Rugh (1973) went on to develop a nocturnal device which can be used to wake a subject vis-à-vis an alarm which sounds whenever the threshold EMG level has been exceeded.

In the same year, Budyznski and Stoyva (1973) worked with eight male subjects and demonstrated that low levels of EMG activity in the masseter muscles could be produced in subjects within a 20-minute period. The thrust of this study was more to demonstrate the educative potential of biofeedback than to test the effects of a learned response on the behavior of bruxism. No bruxing criteria were mentioned and no results of reduced bruxing episodes were reported. It was important in that the study demonstrated the ability of subjects to use the feedback to learn a "relaxation response."

Another research effort involving the use of biofeedback in the study of bruxism was that of Kardachi and Clarke (1977). Using a

portable tape recorder to measure bruxism audibly and a compact EMG unit to be worn throughout the night (developed by Solberg & Rugh, 1973), the authors worked with nine subjects, six males and three females ranging from 19 to 38 years of age. Each subject wore the portable EMG unit for a period of seven nights. When they began to brux, sending the EMG level of masseter potential beyond the threshold activating the alarm, they were gently awakened until they reduced their muscle tension below the alarm level. After seven nights, all subjects reported lower levels of bruxism although at levels not considered statistically significant. Equipment failure and a presumed unwillingness to fully cooperate were cited as possible methodological explanations for the results.

The most recent scientific inquiry into the use of biofeedback in the treatment of bruxism was done in 1978 by Kardachi, Bailey, and Ash. The authors compared occlusal adjustment and its effects on subjects known to brux with nighttime biofeedback-assisted relaxation of the masseter muscle. After carefully reviewing the problems of previous studies, the researchers selected 20 subjects and developed two experimental groups (an occlusal adjustment group of three males and one female, and an experimental biofeedback group of two males and two females). The remaining 16 subjects were placed in a false occlusal adjustment control group, a biofeedback control group receiving random alarms without regard to actual EMG levels. These were nonbruxers from whom only baseline EMG levels were taken in an attempt to find differences in grinding behavior between bruxers and nonbruxers. A portable EMG unit was used, which sounded an alarm when the subject

produced any muscular tension in the masseter muscle beyond 100 microvolts (a condition presumed to reflect bruxing behavior). The entire study lasted three consecutive weeks with the first week being baseline EMG data, the second application of either occlusal adjustment in the form discussed by Ramfjord and Ash (1971) or biofeedback training which consisted of the use of the alarm waking the subject whenever the behavior occurred. The third week of the study consisted of follow-up EMG recording. The follow-up procedure was repeated three months later on the experimental groups only to measure the long-term effects of occlusal adjustments and biofeedback training on EMG levels.

The results of Kardachi et al.'s pilot study reflected a great variance in the effects of occlusal adjustments on nocturnal EMG activity. Of the four subjects in this experimental group, the reductions of mean EMG activity were 17.3 percent and 22.7 percent. Two of the subjects actually increased EMG levels by 5.3 percent and 26.1 percent, which was explained by the authors as understandable behavior of "searching" for comfortable positions after occlusal adjustments. One of the subjects experiencing a decrease in EMG activity returned to his pre-treatment baseline and the other maintained a 15 percent reduction after the three-month post-treatment period.

Biofeedback resulted in an average EMG reduction in all four subjects of approximately 70 percent (70.5, 61.2, 80.5, and 72.8). Three of the subjects in this experimental group returned to original baseline levels immediately upon cessation of training, and one maintained a 50 percent reduction as demonstrated by the three-month follow-up. Control groups for both conditions reported no significant

significant change in EMG levels, and the nonbruxist group was consistently much lower in EMG levels than any of the other groups before, during, or after treatment.

The authors concluded that a long-term study of a large population was needed to make any definitive conclusions. But they confessed that such a large sample would be hard to attain because of the inconvenience of the all-night monitoring device necessary for collection of the EMG data.

They also suggested that in order to produce sustained low EMG levels with biofeedback, a training program of several months would be needed. They suggested such training should take place with wakeful subjects who understand biofeedback and wish to control their masseter tension levels.

Perhaps the most significant finding from this study was that EMG levels dropped more consistently and severely during biofeedback training than during or immediately after occlusal adjustments. Since EMG levels were only recorded when the subject was presumed to be bruxing, one can assume that, at least during the time of receiving feedback, physiological monitoring and relaxation of the muscles of mastication have a greater impact on bruxism than occlusal adjustments.

Several problems are evident within the construction of this study, including the lack of a definitive and objective measurement of bruxing behavior (other behavior such as swallowing hard might produce the 100 microvolt threshold used), the short length of time taken to gather baseline data (one week), a potentially insufficient training period (one week), use of a negative reinforcement model (removal of a

sleep-disturbing stimulus subsequent to the desired relaxation response), and nocturnal biofeedback training which does not permit the subject to "learn" the desired response. The authors recognized this problem when they suggested daytime training as an alternative.

The authors suggested that the development of a training procedure to be conducted with motivated subjects in a wakeful state might be the next appropriate step in refining biofeedback training for the reduction of bruxism.

This study seeks to demonstrate the usefulness of such an extensive training procedure while utilizing recently designed, more objective measurements of the bruxing behavior itself--that being the Bruxcore developed by Forgione in 1973.

Conclusion

Bruxism has received scientific attention since the turn of the century. The broad definition of bruxism includes such terms as gnashing, grinding, clenching, and tapping, during the day or night. The most widely accepted definition includes all those terms and emphasizes the nonfunctional nature of the habit. Such variety in defining the problem has led to difficulties in accurately assessing the incidence of bruxism in the general population. Early estimates (from 1940 through the late 1960s) based upon dental patient populations averaged 80 percent of the population, but this figure was shown to be inflated (perhaps due to sampling bias) by Reding et al. (1966) in a study of nearly 2,300 students in which only slightly over 5 percent were found to brux. Subsequent epidemiological studies have

more accurately estimated the incidence to be at approximately 15-20 percent.

The effects of bruxing behavior can be demonstrated most clearly in the dental problems caused by the habitual grinding. Wear facets, tooth mobility, periodontal disease, masseter muscle hypertrophy, myofacial pain, and temporomandibular joint problems exemplify the many potentially deleterious effects of bruxing behavior.

Bruxism is a complex multidisciplinary problem with no single clearly defined etiology. Current theories can be categorized under three headings: local/mechanical, psychological, and systemic/tension induced.

Local/mechanical theories suggest that irregularities in bite (i.e., malocclusions) may trigger the masticatory process at inappropriate times. Malformed restorations or premature violations of the subject's freeway space are also cited as possible mechanical causes for nonfunctional grinding behavior.

Psychological theories suggest repressed emotions (primarily anger) as being the basis for certain predisposed personality types (i.e., hostile, anxious, and intropunitive) to exhibit the oral aggressive or incorporative behavior of biting and grinding. It is felt that this release of subconscious anger is most often accomplished during REM sleep, when the subject is likely to live out the suggested anger reactions through dreams. Scientific inquiry into these hypotheses has been almost nonexistent to date.

The systemic/neurophysiological theories sought to explain the origin of bruxism through the seemingly hereditary nature of the habit

as well as suggesting that it was the by-product of other physiological maladies (i.e., paralysis, parasites, and other diseases). These explanations have fallen short of favor in the scientific community, however, since it has been shown that not all children of bruxing parents brux and not everyone with a supposedly predisposing physical malady tends to brux as part of a total symptomology.

The neurophysiological theories of etiology suggest a "central origin" in which the emotional centers in the brain act to mediate normal expression of subconscious material by inhibiting the body's natural conscious inhibition of slow grinding motion witnessed in bruxing episodes. Excess stress and muscular tension coupled with a rigid perfectionistic personality dynamic are most often suggested to work in tandem, producing a neurophysiological climate for bruxing behavior.

Muscle tension in the masticatory region (masseter) was mentioned in all of the etiological discussions, and serves as the fundamental concept upon which this study will be based. Perhaps by teaching a subject to voluntarily relax the masseter muscle to a profoundly low level, subsequent bruxing behavior can be avoided or at least decreased in frequency and severity.

The treatment of bruxism has taken on three major forms roughly corresponding to the three etiological constructs: dental intervention (i.e., bite splints, oral appliances, and occlusal adjustments), psychological therapies, and physiological training (most recently assisted by the use of physiological monitoring devices or biofeedback).

Although proponents of each treatment method have claimed significant results in reducing the incidence of bruxism, the lack of adequate and objective measurements of grinding behavior and relaxation levels has clouded the success rates of previous studies.

The availability of a more accurate and objective measuring device called a Bruxcore (Forgione, 1973) and the development of a more intensive biofeedback-assisted relaxation training program make it possible to investigate the potential of training subjects to elicit a "learned" relaxation response believed to be sufficient to inhibit bruxing behavior.

CHAPTER III

RESEARCH DESIGN AND METHODOLOGY

This chapter is divided into six sections which cover the following areas: sample, instrumentation, methodology, experimental design, hypotheses, and analysis.

Sample

The sample for this study consisted of four adult subjects chosen by Kenneth Wisser, D.M.D. Three of the subjects were volunteers who had heard of the purpose of the study from their dentist or in a workshop where bruxism had been discussed. These volunteers were then referred to Dr. Wisser for evaluation. The fourth subject was a dental assistant in Dr. Wisser's office who had complained of facial pain and whom Dr. Wisser had evaluated as being a bruxer.

Dr. Wisser's criteria for selecting these adults for the study included wear-faceting, tooth mobility, palpation of the masseter muscle, and a verbal account of the subject's history of grinding behavior.

All four referrals were found to exhibit sufficient signs of bruxing behavior to qualify for this study.

Subject A: Mr. W is a 55-year-old secondary school teacher. He was diagnosed as a bruxer by his dentist and had been fitted with a mouthguard for nighttime wear. Mr. W had previously ground through

one of these guards. He complained of daytime clenching behavior, which resulted in frequent headaches at the end of his school day. Mr. W. reported being aware of clenching his teeth very often during the day.

Subject B: Ms. B is a 23-year-old dental assistant and graduate student at Michigan State University. She had undergone nine years of orthodontic work throughout her childhood and reported experiencing myofacial pain most often manifested in headaches upon waking in the morning. Ms. B. also reported that the facial pain experienced upon waking usually continued through the day, especially when she was under a great deal of stress.

Subject C: Mr. J. is a 29-year-old custom cabinet maker and general contractor. He complained of sore masseter muscles, especially upon waking in the morning, and facial pain which increased in severity throughout the day. Mr. J. also reported finding his jaws locked in tension throughout his working day.

Subject D: Mrs. W. is a 47-year-old housewife and undergraduate student in education at Michigan State University. Mrs. W.'s dentist fitted her for a mouthguard to protect her from damaging her teeth while grinding and/or clenching. She reported experiencing a chronic low-grade headache which often became severe toward the end of the day or on mornings when it was apparent (from masseter fatigue) she had been grinding at night.

All four subjects were diagnosed by Dr. Wisser as being chronic bruxers. They were referred for a pre-experiment interview, during which they were informed about the details of the study. The subjects

were told that they would not receive feedback about their level of bruxing behavior until after the study was completed.

Time commitments, home practice procedures, appointment schedules, and potential benefits were explained in detail to avoid any possible misunderstanding later in the study. At this time the subject consent form was signed and witnessed (Appendix A).

The subjects were then referred again to Dr. Wisser for a pre-study periodontal observation. At this time, verbal consent was obtained to insure that the subjects clearly understood their commitment to the project.

The sample for this study consisted of four adults, two male and two female, aged 23 to 50.

All four volunteers were selected on the basis of the presence of bruxing criteria as observed by Kenneth Wisser, D.M.D., and all were informed about the requirements and potential benefits of their commitment to the study.

Instrumentation

Periodontal Observation

After the subjects were selected and consent forms signed, Dr. Wisser completed a clinical observation of the subjects. Measures of wear facets were taken by visual observations and listed on a dental chart. (See Appendix B.) Masseter hypertrophy was measured subjectively by palpating the masseter muscle, and results were recorded on the dental chart. Tooth mobility was also measured by visual and tactile observation and recorded on the dental chart.

Scoring for tooth mobility was done in the following manner:

0 = No clinical mobility (within normal range)

+ = slight mobility noted (slightly more than physiologic range but less than 1 mm buccal-lingual)[1]

+1 = Clinical mobility approximately 1 mm buccal-lingual

+2 = Clinical mobility approximately 2 mm buccal-lingual but with no mobility in apical direction

+3 = Clinical mobility greater than 2 mm buccal-lingual in addition to mobility in an apical direction

Mouth-opening ability was observed using a Boley gauge to measure interincisal opening between incisal tips of the maxillary and mandibular first incisors and recorded in millimeters on the dental chart.

All the clinical observations were completed before baseline EMG levels were taken or treatment was begun. The clinical measurements were all recorded on a standard dental chart.

This same procedure and observation instrument was used upon completion of the study (six weeks after the final training session), and together with the pre-study observation it served as a pre-post clinical observation.

Self-Report

A pre- and post-treatment self-report questionnaire, filled out by the subjects, was designed to be an easily administered, subjective measure of bruxing behavior. It also served as a report of pain presumably experienced from bruxism. Also included in this instrument

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[1] Buccal = next to or toward the cheek;
Lingual = next to or toward the tongue;
Buccal-lingual = from cheek toward the tongue or from front to back.

was a report of how often the subject noticed bruxing behavior and how often the behavior was noted by a friend and/or spouse. The presence, location, and relative severity of headache and/or facial pain was also documented in relation to the time of day and its duration. Finally, the subjects were questioned about potential pain experienced when chewing or opening their mouths widely.

The self-report questionnaire was administered on the first day of the EMG baseline procedure and again on the last EMG and Bruxcore follow-up visit. A copy of this instrument is included as Appendix C.

Bruxcore

A major problem in bruxism research has been the lack of a practical and objective method of measuring bruxing behavior. Recently a simple, inexpensive, objective method for monitoring and quantifying bruxim was developed by Forgione (1974).

The bruxism-monitoring device, Bruxcore, consists of four differently colored plastic sheets (gray, white, orange, and black) laminated to a total thickness of 0.02 inch with microdots printed in edible ink on the uppermost surface. The dots are 1/180 inch in diameter, fitting 120 dots per inch in a pattern of 14,400 dots per inch. The plastic disc is four inches in diameter and is molded into maxillary plates in a Dent-Sply Vacu-Press. The plates are then trimmed to fit the upper dentition of the subject and worn by the subjects during sleep for four consecutive nights (in this case, Monday, Tuesday, Wednesday, and Thursday). After the four nights of wear, the Bruxcore is examined under a dissection microscope and scored.

In the present study the Bruxcore monitor was scored using the procedures described by Forgione (1974). A Gaertner single-lens microscope was used to detect the number of dots ground away. The following guidelines were employed:

> One point was scored for each dot when at least 1/2 of the dot area was ground away, exposing gray print.
>
> Two points were scored for each dot when at least 3/4 of the dot area exposed the second (white) layer of plastic.
>
> Three points were scored for each dot when at least 3/4 of the dot area exposed the third (orange) layer of plastic.
>
> Four points were scored for each dot when at least 3/4 of the dot area exposed the fourth (black) layer of plastic.
>
> Five points were scored for each dot when at least 3/4 of the dot area was ground through the plastic.

In this manner, a volumetric index of the amount of the plate worn away was obtained for each contact point and a total score for all teeth rendered a bruxism indication for the entire dentition.

In an attempt to validate the use of this device for the study of bruxism, Forgione (1974) selected four white males from 19 to 35 years of age. They were asked to grind a fitted Bruxcore with maximum force for 15-second periods, resting for five minutes between each period to enable scoring, until 80 measures, i.e., 20 minutes of grinding time, were culminated. Measurement settings were of approximately two hours' duration. The procedure was repeated exactly with a second mouthpiece.

The cumulated scores, 80 measures for each subject, were plotted against time. Test and retest correlations of total grinding times per 15-second periods were high enough to demonstrate the reliability of the instrument:

$S_1 = r = 1.00$

$S_2 = r = .99$

$S_3 = r = .98$

$S_4 = r = 1.00$

The high correlations on retest for each subject permitted the random selection of four regression lines, one from each subject. They were:

Run 1 for Subject 1	Slope 1.73	r = .99
Run 2 for Subject 2	1.17	r = .99
Run 1 for Subject 3	.91	r = .99
Run 2 for Subject 4	.58	r = .96

Each regression line was significantly correlated with time beyond the .001 level. Calculated intercepts ranged from 23.64 to -93.11 points on the 2,000-point scale, indicating slight curvilinearity. The standard error of estimate values, i.e., the error score value in estimating Y, were small, ranging from 43.9 (S-1) to 54.3 (S-4). The small size of these estimates indicates that measures, on average, taken approximately three minutes apart, significantly differ from each other.

In descending order, all slopes differed significantly at the .001 level, indicating the individual difference in grinding for each subject over time. This finding indicates that the instrument is suited to the detection of fine changes within a single subject over time, which may in turn allow evaluation of a treatment intervention.

In anticipation of the question of whether or not inserting a plastic mouthpiece would elicit bruxism, a pilot experiment was performed (Forgione, 1974).

Twelve subjects were asked to wear a mouthpiece for four nights in succession. Six subjects were clinically diagnosed as bruxers and six were diagnosed as nonbruxers.

Although the group means differed with regard to scores of four nights' wear, a t-test could not be performed due to lack of homogeneity of variances. However, a Mann Whitney test was performed, revealing that the two groups of scores differed significantly.

If the mouthpiece does elicit bruxism, it is minimal on this recording instrument.

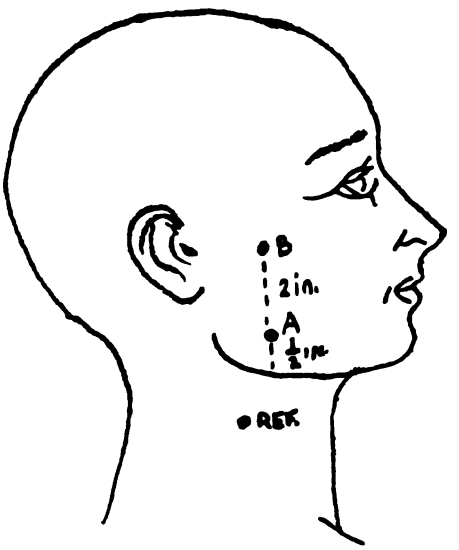
In conclusion, the Bruxcore was found to be a reliable and sensitive detector of grinding behavior over time, and did not appear to elicit sufficient bruxing to argue against use. It was also shown to be more suited to in-depth studies of single subjects over time because of the variance among bruxers relative to amount of bruxing behavior and the potential compounding of that difference in group designs.

Electromyograph

Muscle tension was measured and recorded with the aid of an Autogen 1100 model EMG. The EMG machine functions by monitoring and displaying ongoing electrical activity generated by muscle action (contraction and relaxation). The more electrical activity present in a muscle, the more tense it is; the lower the number of electrical impulses, the more relaxed it is. In feedback electromyography, EMG

activity is first amplified, then rectified, integrated, and rendered in the form of auditory patterns and/or visual displays.

The Autogen 1100 detects muscle activity through three platinum electrodes attached to the surface of the skin directly above the muscle to be measured. In this case, the masseter muscle was being studied and the electrodes were placed as shown in Figure 1.



A & B = active electrodes
Ref. = reference electrode

Figure 1.--Electrode placement.

The subjects were asked to turn their heads slightly to the left, and the reference electrode (R) was placed on the Sterno-Mastical Muscle. The first active electrode (A) was placed at the point of maximum contraction when the teeth were clenched and relaxed. The other active electrode (B) was placed two inches above the first

electrode. All placements were made on the subject's right side since all complained of more pain emanating from that side.

The Autogen 1100 uses integral average measurement EMG amplitude, and the standard bandpass of 100-200 H_2. Other instruments may use a different bandpass or different filtering mechanisms. A microvolt conversion chart is included in Appendix D to permit standardization of EMG readings.

Digital Integrator

EMG readings from the Autogen 1100 were averaged and recorded on a digital integrator (Autogen 5100). The digital integrator (also known as a time period integrator) is a data-processing instrument which performs on-line acquisition and data reduction of certain types of informational inputs. The data analysis is performed over a preselected time interval (in this case, 30 seconds) and the resultant numerical data are read out on a digital display at the conclusion of the interval (in this case, 30 seconds). This provides a means of quantifying both analog and digital information in a form which can be recorded and stored for subsequent reference or additional analysis. The Autogen 5100 was produced by Autogenic Systems, Incorporated, of Berkeley, California, and typically is accurate to ±0.5 percent of the reading with a maximum error of ±0.1 percent. (See Appendix E.)

Methodology

Periodontal Observation

After the subjects were selected and the informed consents obtained, the first step was completion of a pre-treatment clinical

observation by Kenneth Wisser, D.M.D. During this office visit, the subject's teeth were examined visually for wear facets, abnormal markings, gouges, or sharp surfaces which indicate unusual grinding patterns. The location of these markings was documented on a standard dental chart. Tooth mobility was measured tactilly and a subjective record made. Masseter muscle hypertrophy was gauged by tactile palpation of the masseter and a normal, abnormal differentiation was employed. The subject's mouth-opening ability was measured in milimeters. Upon completion of the clinical observation, a plaster impression (working model) was taken of the subject's upper dentition. This model was used by Dr. Wisser initially to confirm visual observations and subsequently as a plate over which to press the Bruxcore plates. At the conclusion of this procedure, the subject was judged once again to be appropriate for the study, and verbal consent was obtained before the next phase commenced.

At this point the subjects were given codes (letters) to insure anonymity, and their working models were taken to the Foundation for Behavior Research in Augusta, Michigan, for production of the Bruxcore devices.

Ten Bruxcore guards were produced (as described previously) for each subject. A record card (see Appendix F) was also produced, indicating date and day of the week the Bruxcore was to be worn, as well as the time it was placed into the mouth and the time it was taken out. These data were acquired to insure a continuity of experience between subjects and to provide a method of reminding the subject to wear the Bruxcore. Heavy-duty, 5" x 8" plastic freezer bags were

used to hold one Bruxcore and one Bruxcore data card. Each subject had 10 such packets corresponding to the nine weeks of data collection and one extra for emergencies. Files for each subject contained self-report and EMG data sheets as well as minimal biographical information and anecdotal notes from each training session.

Bruxcore EMG Baseline Collection

The subjects then reported for their first EMG baseline meeting. Appointments were made on a Friday preceding the Monday they were to begin wearing the Bruxcore device. At the beginning of the first session, the subject self-report questionnaire was completed, and the entire schedule of appointments and expectations for wearing the Bruxcores and keeping records was explained. The subject was then asked to sit in a cushioned chair facing away from the EMG unit and digital integrator to preclude visual feedback of EMG levels. The EMG electrodes were placed in the manner described, and the experimenter took his/her place seated behind the subject (out of sight) and in front of the equipment. The equipment was turned on and verified to be in good working order. At this point, the experimenter gave the following instructions to the subject: "Please sit quietly and relax for the next 15 minutes."

The experimenter then put on a set of headphones, which enabled him/her to hear a warning tone five seconds before the integrator began to compute the average EMG level and again 30 seconds later when the average score was illuminated on a digital readout. The subject's right masseter EMG level was averaged for 30 seconds, after which the

integrator produced the digital score for 30 seconds, then sounded a five-second warning and repeated the process. This procedure was replicated for 10 trials, yielding 10 mean EMG scores for the session. Scores were recorded in the appropriate place on the master EMG data chart and kept in the subject's personal file. (See Appendix G.) After completion of the 10 baseline trials, the electrodes were removed and the subjects were given their first packet containing a Bruxcore and data card. Using a pair of cutical scissors with rounded tips, the Bruxcore was trimmed to fit by cutting the excess plastic near the gingival margin and from the roof of the mouth until the subject reported it to be comfortable. An explanation of wearing schedules (i.e., Monday, Tuesday, Wednesday, and Thursday) was given, data-keeping procedures were explained, and each subject was instructed in the care and maintenance of the Bruxcore (cleaning procedures and emergency trimming, if necessary). The next appointment was scheduled, and the session was terminated.

The same basic procedure was followed for the two subsequent baseline-gathering sessions with the exception that the self-report and record-keeping explanations were not necessary after the initial session.

EMG Training Procedures

After collecting three consecutive Fridays of baseline EMG readings, treatment was begun. The subjects were scheduled for 30-minute appointments starting on Monday of the fourth week and running Monday, Tuesday, Thursday, and Friday for three consecutive weeks.

The frequency of the training sessions was meant to insure the highest potential for learning in the shortest amount of time.

On the first Monday of training, the subject was placed in a cushioned chair, this time directly facing the biofeedback equipment. Again the experimenter was seated directly behind the subject with a clear view of the integrator (for documentation purposes).

At this point the EMG electrodes were positioned in the manner described, and the subject was given a brief explanation of biofeedback in general and masseter training specifically.

Biofeedback was explained as a physiological self-monitoring procedure which acts as a mirror to the body. Electric impulses sent from the brain to the muscles via the central nervous system cause a biochemical reaction in the muscle (muscle contraction). The stronger the impulse, the stronger the resulting contraction. This system is known as muscle potential. The electromyograph picks up the electrical impulse within the muscle and transduces it into an auditory tone and a visual deflection on a meter. The audible tone increases in volume and pitch as the muscle potential increases, and decreases in volume and pitch as muscle potential decreases. The needle on the EMG meter deflects to the right indicating more microvoltage present during muscle contraction and to the left pointing to lower microvoltage values when the subject produces less muscle contraction.

It was explained to the subject that the EMG does nothing to them directly; it simply measures the amount of muscle tension and feeds it back to them so they can use the "new" information to become consciously aware of the amount of tension present in the masseter muscle

at any given time. By receiving feedback frequently and actively attempting to lower the sound emanating from the speaker, it was hoped the subjects would learn to control masseter tension to a fine degree. It was further hoped they would attempt to duplicate the subjective feelings of relaxation present during training several times throughout the day, and to a profound degree before going to bed at night.

After the explanation, questions regarding the basics of the biofeedback procedure were answered. It was then demonstrated that swallowing, coughing, gross muscle movements, blinking hard, or talking would cause the EMG to register large amounts of microvoltage. The subjects were told that any such behavior was allowed, but that when trying to reduce the readings they should refrain from these behaviors as much as possible.

The digital integrator was explained as a simple calculator which averaged the EMG reading over a 30-second period and displayed the read-out digitally to the hundredth of a microvolt. They were told this figure would be illuminated on a meter for them and the experimenter to see. They were welcome to use this information as feedback of a more cumulative nature as well as the second-by-second audio and visual representation of the EMG machine.

Once all questions were answered to the satisfaction of the subject, training was begun. The subject began to hear the audible reflection of masseter muscle tension and to see the EMG meter demonstrating the tension in terms of microvoltage of muscle potential. The experimenter documented the readings from the digital integrator for further reference and comparison. The integrator sounded a bleep,

warning the experimenter (and subject) that in five seconds it would start averaging the EMG level for 30 seconds. In five seconds the illuminated display on the integrator would go black, informing both experimenter and subject that the averaging had begun. Thirty seconds later the second bleep sounded, and the new EMG average appeared on the display meter. The experimenter documented this reading, and the subject was encouraged to use it as feedback about the subsequent 30-second interval. This process was repeated 10 times until 10 EMG averages were obtained. At this point the subject was allowed to relax for approximately five more minutes to complete the feedback practice. The equipment was then shut off and the electrodes were removed. During this procedure, subjects were questioned about how they felt during the session, and an attempt was made to have them articulate the experience of establishing control over muscle tension. During this time, anecdotal information was collected regarding the subject's experiences during the past day, especially with reference to his/her practicing a "relaxation response."

After the subject had been debriefed, the session was terminated. On Fridays the subject was fitted with a new Bruxcore device during the post-training period of the session. Subjects wore the Bruxcore device during the training weeks as well as pre- and post-training to try to establish whether learning to control muscle tension would affect the amount of bruxing, and if so, how much training at what level of success was necessary to produce such a change.

The same basic procedure, without the initial biofeedback explanation, was repeated every Monday, Tuesday, Wednesday, Thursday, and

Friday for the next three weeks, yielding 10 mean EMG readings per session or 40 per week. (Illustration can be found in the design section of this chapter.)

EMG and Bruxcore Follow-up

On the final Friday of the training phase, the subjects were given a new Bruxcore for the next week and asked to return to the office on the following Friday for the first of three post-training follow-up procedures. They were to continue to practice their learned response approximately five times per day and for an extended period at night before going to sleep.

Upon returning to the office one week after their last training session, the used Bruxcore was collected and anecdotal notes were taken regarding the subject's experiences over the previous week, especially as they related to using the relaxation throughout the day in various situations (i.e., in traffic, at work, under stress). The subject was then seated in the cushioned chair facing away from the equipment in the same fashion as in the pre-training arrangement. Once again, the subject was asked to sit quietly and relax for 15 minutes, and the experimenter collected 10 30-second average readings with 30-second rests between. When this procedure was completed, subjects were fitted for their next Bruxcore and asked to wear it beginning on the Monday of the third week from that day. The same procedure was followed for the next session, which occurred one month post-training, and the final session three weeks later or six weeks post-training.

Collection of Post-Training Dependent Measures

On the last follow-up session (six weeks post-training), the subject was asked to complete the self-report questionnaire. An appointment was then made in the subsequent week with Dr. Wisser, at which time they would receive a post-training periodontal observation.

At the end of the final follow-up EMG visit, all Bruxcore devices were sent to Mr. David Keenan and his associates at the Foundation for Behavioral Research in Augusta, Michigan, for scoring.

During the post-training periodontal observation, Dr. Wisser measured wear-faceting, palpated the masseter muscles, measured tooth mobility, and asked subjects for a verbal account of the frequency and severity of their bruxing behavior over the preceding 15 weeks. The data were recorded in the same fashion as in the pre-training observation, documented on subjects' dental charts, and compared as a subjective indication of treatment effects.

Experimental Design

This study employed a modification of the multiple baseline design across subjects as described by Hersen and Barlow (1976). It is also known as a "time-lagged control" design due to the staggered nature of the baseline, treatment, and follow-up collection of data. (See Figure 2.)

In this particular design, a treatment is applied to a matched group of subjects, who are presumably exposed to identical experimental conditions. A baseline of the behavior in question (in this case, bruxism and masseter muscle tension) was obtained for three weeks,

followed by three weeks of biofeedback training (treatment) and three follow-up visits. The design may also be thought of as four separate ABA designs within subjects. Time was lagged to control for environmental effects on target behaviors.

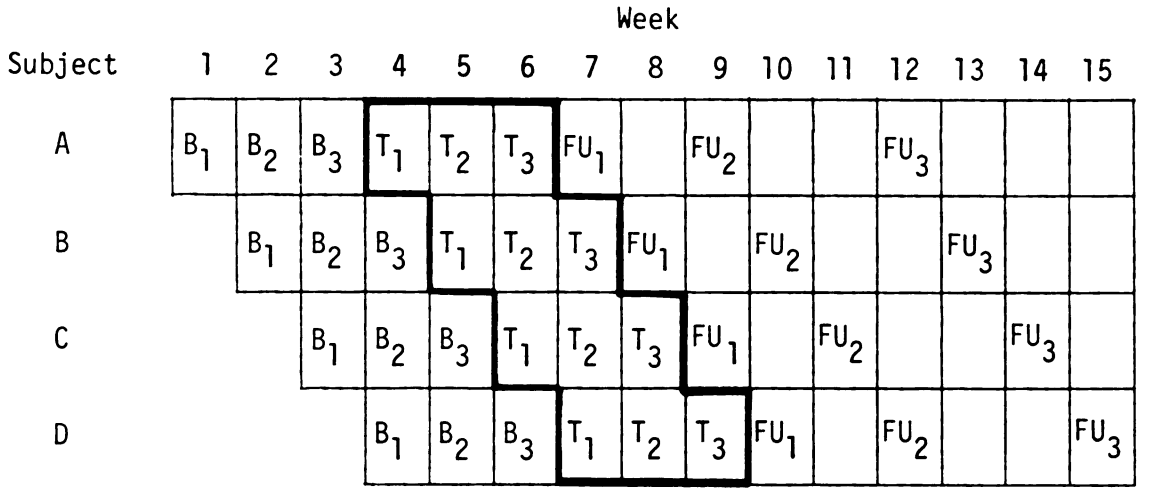


Figure 2.--Time-lagged multiple baseline design.

During the three-week pre-treatment period, baseline data were collected on the subject's level of weekly bruxing behavior (Bruxcore) and 10 weekly samples of existing masseter tension as measured by the EMG machine.

The treatment phase consisted of four weekly training sessions, during which another 10 EMG averages were recorded and weekly Bruxcores gathered.

The follow-up phase consisted of the same data gathering as in the baseline phase. Data were collected and recorded for each subject in the manner shown in Figure 3.

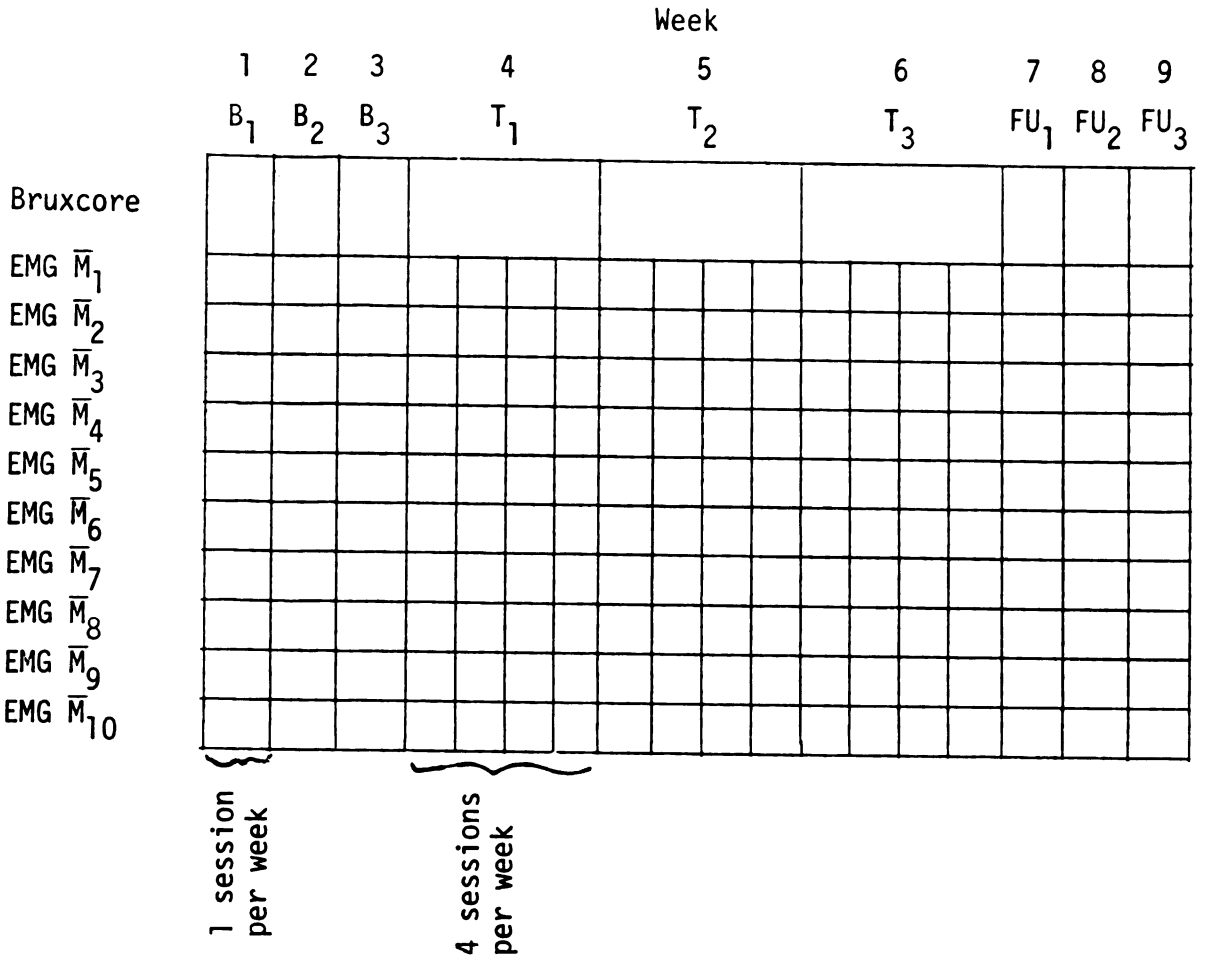


Figure 3.--Master data chart. (Each square under baseline B and Follow-up FU represents one week of data, while each square under treatment T represents one day of data.)

Hypotheses and Analysis

Five hypotheses were generated to empirically test the possible effect of biofeedback training and masseter muscle relaxation of bruxism. All of the hypotheses will be presented in null and alternative form, with the statistical analysis used for each.

The first hypothesis was designed to test the possible effect of biofeedback training on EMG levels of masseter muscle activity.

Null Hypothesis: There will be no trend in EMG levels from baseline to treatment and follow-up.

Alternative Hypothesis: There will be a decreasing trend in EMG levels from baseline to treatment and follow-up.

To test the hypothesis the Test Against the Trend Analysis was used (Lehmann, 1975). The test against the trend uses a Spearman's statistic: $D = (T_i - i)^2$ where T_i is the rank of the i^{th} statistic. Probability of significance was set at the .05 level.

A mean EMG reading was taken from the 10 data points obtained in each visit. The test against the trend was run to see if there was a trend of all resulting nine data points (baseline 1, 2, 3; treatment 1, 2, 3; follow-up 1, 2, 3). Then the test was run over the first six points only (dropping the follow-up). This was done to see if there may have been an initial reduction in EMG levels which could have deteriorated in the follow-up. An additional test was run to see if there was a trend of EMG levels across all four subjects using the Friedman Rank Test (Lehmann, 1975) with probability of significance set at the .05 level.

The second hypothesis was designed to test for the possible effect of biofeedback training on the level of nocturnal teeth grinding as measured by scores obtained from the Bruxcore monitoring device.

Null Hypothesis: There will be no trend in Bruxcore figures from baseline to treatment and follow-up.

Alternative Hypothesis: There will be a decreasing trend in Bruxcore figures from baseline to treatment and follow-up.

Each Bruxcore figure represented the total level of grinding behavior over a four-consecutive-nights period. The Testing Against the Trend analysis was used to see if there was a trend over the entire study period (baseline, treatment, follow-up). Then the test was run over the baseline and treatment only for the purpose of exposing any possible treatment effects which may have deteriorated during the follow-up.

An additional test was run to see if there was a pattern of Bruxcore figures across all four subjects, using the Friedman Rank Test. Probability of significance was set at the .05 level.

The third hypothesis was designed to test for a possible relationship between EMG levels and grinding behavior as measured by the Bruxcore.

Null Hypothesis: There will be no relationship between EMG levels and Bruxcore figures across baseline, treatment, and follow-up phases.

Alternative Hypothesis: EMG levels will be related to Bruxcore figures across baseline, treatment, and follow-up phases.

The test used was the Friedman Rank Sum Test (Lehmann, 1975). A probability of significance level of .05 was used.

The fourth hypothesis was designed to measure the possible effects of biofeedback training on bruxing criteria as recorded in a pre- and post-study periodontal observation.

Null Hypothesis: There will be no difference in pre- and post-study periodontal reports of bruxing criteria.

Alternative Hypothesis: There will be less bruxing criteria recorded in the post-study periodontal observation than in the pre-study observation.

Comparisons of pre- and post-study periodontal observations will be made using tooth mobility, masseter muscle palpation, mouth-opening measurements, and patients' reports of pain or discomfort. The data will be presented in graphic form for tooth mobility and in descriptive written form for the remaining factors.

The fifth hypothesis was designed to measure the possible effects of biofeedback training on bruxing criteria as reported by the subjects on a self-report questionnaire.

Null Hypothesis: There will be no difference in subjects' reports of bruxing criteria before and after the study.

Alternative Hypothesis: Subjects will report less bruxing criteria present after the study than before the study.

Self-reported bruxing criteria include awareness of bruxing over past week, validation of behavior by a friend or spouse, headache and facial pain experienced presumably as a result of bruxing, and pain experienced when opening the mouth wide or chewing.

Data will be presented in graphic form on the factors of awareness of the habit and pain experienced from it. The other factors

will be discussed in the text along with anecdotal notes obtained by the experimenter throughout the study.

CHAPTER IV

ANALYSIS OF DATA

Statistical analysis of data for each hypothesis will be presented along with graphic representations of the results for each subject. Anecdotal notes are presented in the text in the section under Subject Self-Report.

Hypothesis 1

H_0: There will be no trend in EMG levels from baseline to treatment and follow-up.

H_1: There will be a decreasing trend in EMG levels from baseline to treatment and follow-up.

Each score in the baseline and follow-up phases represents the mean of 10 separate EMG readings per session. The treatment scores represent the mean of 10 separate EMG readings per day for four days per week. Lehmann's Testing Against the Trend Analysis was used, and Spearman's statistic was calculated to test the hypothesis at the .05 level of significance.

The EMG readings in Table 1 will be broken down by subject and presented graphically for each. Results of the hypothesis testing will also be presented and explained for each subject.

Table 1.--Mean weekly EMG readings across subjects.

	Baseline			Treatment			Follow-up		
	1	2	3	1	2	3	1	2	3
Subject A	1.543	1.319	1.533	.723	.539	.595	.672	.704	.747
Subject B	11.268	17.068	14.561	1.617	1.200	.838	.719	.880	1.094
Subject C	1.121	1.819	1.472	.498	.445	.456	.461	.525	.474
Subject D	1.858	2.213	1.932	.967	.813	.811	.744	.784	.698

Note: Figures are in microvolts of masseter muscle potential.

Subject A

The data in Graph 4 seem to indicate that Subject A reduced his baseline EMG reading by an average of 57.75 percent after the three weeks of treatment. It must be noted, however, that the follow-up EMG levels reflect only a 51.67 percent decrease from baseline levels, indicating a possible deterioration of the learned response after seven weeks post-treatment. This observation is confirmed by the results of the hypothesis test.

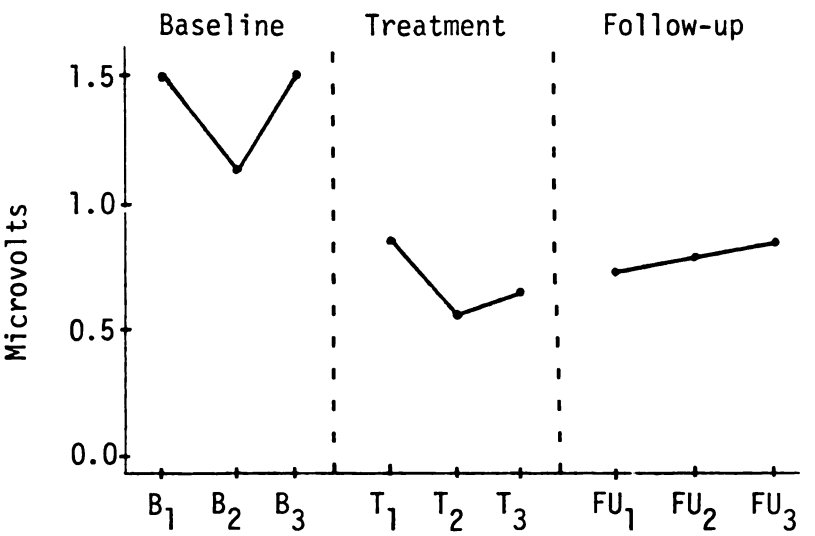


Figure 4.--Subject A EMG levels pre-, during, and post-treatment.

Table 2 indicates that Subject A experienced a decreasing trend in EMG levels from the three baseline sessions through the three treatment weeks. However, when the test was repeated with the three follow-up sessions included, the trend was not significant at the .05 level.

Table 2.--Subject A EMG levels without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Mean reading	1.543	1.319	1.533	.723	.539	.595
Rank	6	4	5	3	1	2
Reading number	6	5	4	3	2	1

$D = 4$ $\quad P\wedge[D \leq 4] = .0167$ Significant at .05 level

Reject H_0--there is a decreasing trend.

Table 3 shows that the decreasing trend in Subject A's EMG levels from baseline to treatment does not maintain stability across the follow-up sessions at the .05 level.

Table 3.--Subject A EMG levels with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Mean reading	1.543	1.319	1.533	.723	.539	.595	.672	.704	.747
Rank	9	7	8	5	1	2	3	4	6
Reading no.	9	8	7	6	5	4	3	2	1

$D = 52$ $\quad P\wedge[D \leq 52] = .0603$ Not significant at .05 level

Do not reject the null hypothesis--there is no significant trend.

Subject B

The data in Figure 5 indicate that Subject B reduced her baseline EMG levels by an average of 91 percent after the treatment phase. Although the follow-up EMG levels appear to be slowly rising from FU_1 to FU_2, the average percentage of decrease in the follow-up phase from baseline levels was 94 percent. There seems to be a stable decreasing trend in EMG levels from baseline through treatment and follow-up. This observation is confirmed by the results of the hypothesis test.

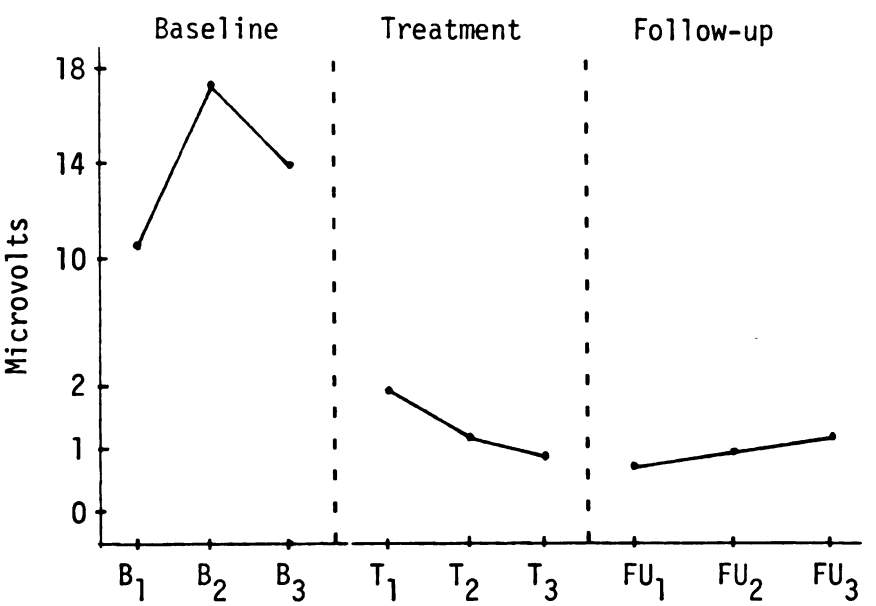


Figure 5.--Subject B EMG levels pre-, during, and post-treatment.

Table 4 indicates that Subject B experienced a decreasing trend in EMG levels from the three baseline sessions through the three weeks of treatment. When the test was repeated with the three follow-up sessions included, the trend remained significant at the .05 level.

Table 4.--Subject B EMG levels without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Mean reading	11.268	17.068	14.561	1.617	1.2	.838
Rank	4	6	5	3	2	1
Reading no.	6	5	4	3	2	1

$D = 6$ $P\wedge[D \leq 6] = .0292$ Significant at .05 level

Reject H_o--there is a decreasing trend.

Table 5 shows that the decreasing trend in Subject B's EMG levels from baseline through treatment and follow-up remained significant at the .05 level.

Table 5.--Subject B EMG levels with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Mean reading	11.268	17.068	14.561	1.617	1.2	.838	.719	.88	1.094
Rank	7	9	8	6	5	2	1	3	4
Reading number	9	8	7	6	5	4	3	2	1

$D = 24$ $P\wedge[D \leq 24] = .0069$ Significant at .05 level

Reject H_o--there is a decreasing trend.

Subject C

The data in Figure 6 indicate that Subject C reduced his baseline EMG levels by an average of 68 percent after the treatment phase. Although the follow-up EMG levels appear to be rising from FU_1 to FU_2, the average percentage of decrease in the follow-up phase from baseline levels was 67 percent. There seems to be a stable decreasing trend in EMG levels from baseline through treatment and follow-up. The hypothesis test in this case verified the initial reduction from baseline to treatment, but when the follow-up data were included, no decreasing trend could be established at the .05 level.

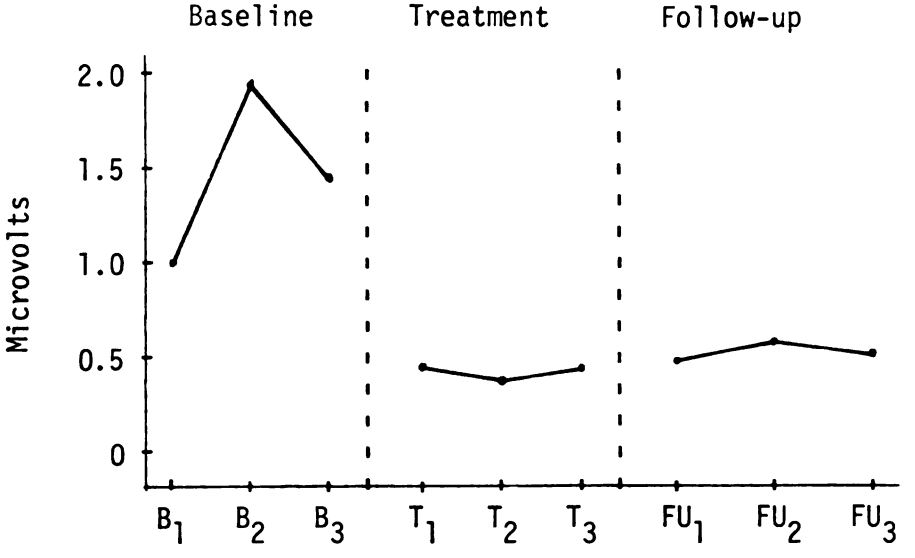


Figure 6.--Subject C EMG levels pre-, during, and post-treatment.

Table 6 indicates that Subject C experienced a decreasing trend in EMG levels from the three baseline sessions through the three weeks of treatment. However, when the test was repeated with the three

follow-up sessions included, the trend was not significant at the .05 level.

Table 6.--Subject C EMG levels without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Mean reading	1.121	1.819	1.472	.498	.445	.456
Rank	4	6	5	3	1	2
Reading number	6	5	4	3	2	1

$D = 8$ $P\wedge[D \leq 8] = .0023$ Significant at .05 level

Reject H_o--there is a decreasing trend.

Table 7 shows that the decreasing trend in Subject C's EMG levels from baseline to treatment does not maintain stability across the follow-up sessions at the .05 level.

Table 7.--Subject C EMG levels with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Mean reading	1.121	1.819	1.472	.498	.445	.456	.461	.524	.474
Rank	7	9	8	5	1	2	3	6	4
Reading no.	9	8	7	6	5	4	3	2	1

$D = 52$ $P\wedge[D \leq 52] = .0603$ Not significant at .05 level

Do not reject H_o--there is no significant trend.

Subject D

The data in Figure 7 indicate that Subject D reduced her baseline EMG levels by an average of 56 percent after the treatment phase. The average percentage of decrease from the baseline to the follow-up phase was even higher at 63 percent. There seems to be a decreasing trend in EMG levels from baseline through treatment and follow-up. This observation was confirmed by the hypothesis test.

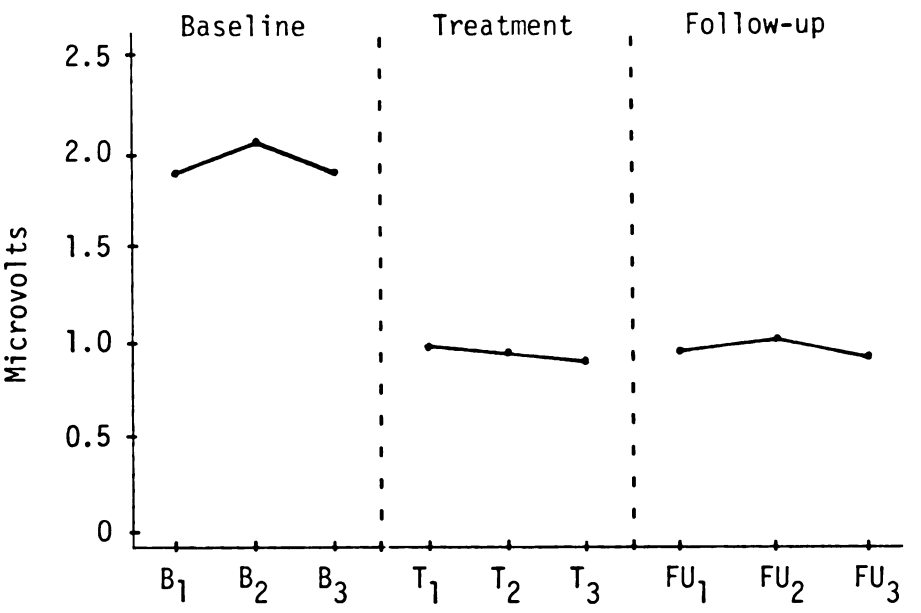


Figure 7.--Subject D EMG levels pre-, during, and post-treatment.

Table 8 indicates that Subject D experienced a decreasing trend in EMG levels from the three baseline sessions through the three weeks of treatment. When the test was repeated with the three follow-up sessions included, the trend remained significant at the .05 level.

Table 8.--Subject D EMG levels without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Mean reading	1.858	2.213	1.932	.967	.813	.811
Rank	4	6	5	3	2	1
Reading number	6	5	4	3	2	1

D = 6 $P\wedge[D \leq 6] = .0292$ Significant at .05 level

Reject H_o--there is a decreasing trend.

Table 9 shows that the decreasing trend in Subject D's EMG levels from baseline through treatment and follow-up remained significant at the .05 level.

Table 9.--Subject D EMG levels with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Mean reading	1.858	2.213	1.932	.967	.813	.811	.744	.784	.698
Rank	7	9	8	6	5	4	3	2	1
Reading number	9	8	7	6	5	4	3	2	1

D = 8 $P\wedge[D \leq 8] = .0004$ Significant at .05 level

Reject H_o--there is a decreasing trend.

Additional Test

Using the Friedman Rank Sum Test, an additional test was run on the data to see if there was a trend across subjects. Table 10 shows that there was a decreasing trend in EMG levels from baseline through treatment and follow-up at the .05 level. All four subjects experienced the same phenomena with regard to a learned relaxation response in their right masseter muscle.

Table 10.--Friedman Rank Sum Test for EMG trends across subjects.

	Subject A		Subject B		Subject C		Subject D		Rank Sum
	$\overline{M}$ Reading	Rank	$\overline{M}$ Reading	Rank	$\overline{M}$ Reading	Rank	$\overline{M}$ Reading	Rank	
B_1	1.543	9	11.268	7	1.121	7	1.858	7	20
B_2	1.319	7	17.068	9	1.819	9	2.213	9	34
B_3	1.533	8	14.561	8	1.472	8	1.932	8	32
T_1	.723	5	1.617	6	.498	5	.967	6	22
T_2	.539	1	1.200	5	.445	1	.813	5	12
T_3	.595	2	.838	2	.456	2	.811	4	10
FU_1	.672	3	.719	1	.461	3	.744	2	9
FU_2	.704	4	.880	3	.524	6	.784	3	16
FU_3	.747	6	1.094	4	.474	4	.698	1	15

$Q = 25.66$ $P\Delta[Q \leq 25.66] < .005$ Significant at .05 level

Reject H_0--there is a decreasing trend in mean EMG readings across subjects.

Hypothesis 2

H_0: There will be no trend in Bruxcore figures from baseline to treatment and follow-up.

H_1: There will be a decreasing trend in Bruxcore figures from baseline to treatment and follow-up.

Each Bruxcore score represents four consecutive nights of grinding behavior. A Testing Against the Trend Analysis was used and Spearman's statistic was calculated to test the hypothesis at the .05 level of significance. The weekly Bruxcore data in Table 11 will be broken down by subject and presented graphically for each. Results of the hypothesis testing will also be presented and explained for each subject.

Table 11.--Weekly Bruxcore data across subjects.

	Baseline			Treatment			Follow-up		
	1	2	3	4	5	6	7	8	9
Subject A	22	27	17	62	49	117	64	21	2
Subject B	638	947	919	916	642	818	720	670	866
Subject C	892	868	629	143	258	179	294	295	290
Subject D	26	167	307	557	22	748	304	313	97

Subject A

The data in Figure 8 show that Subject A actually increased his nocturnal bruxing behavior during the treatment phase. The subsequent reduction of this increase in the follow-up phase seems to indicate the absence of a clear trend in pre-, during, and post-treatment Bruxcore figures. This observation is confirmed by the hypothesis test.

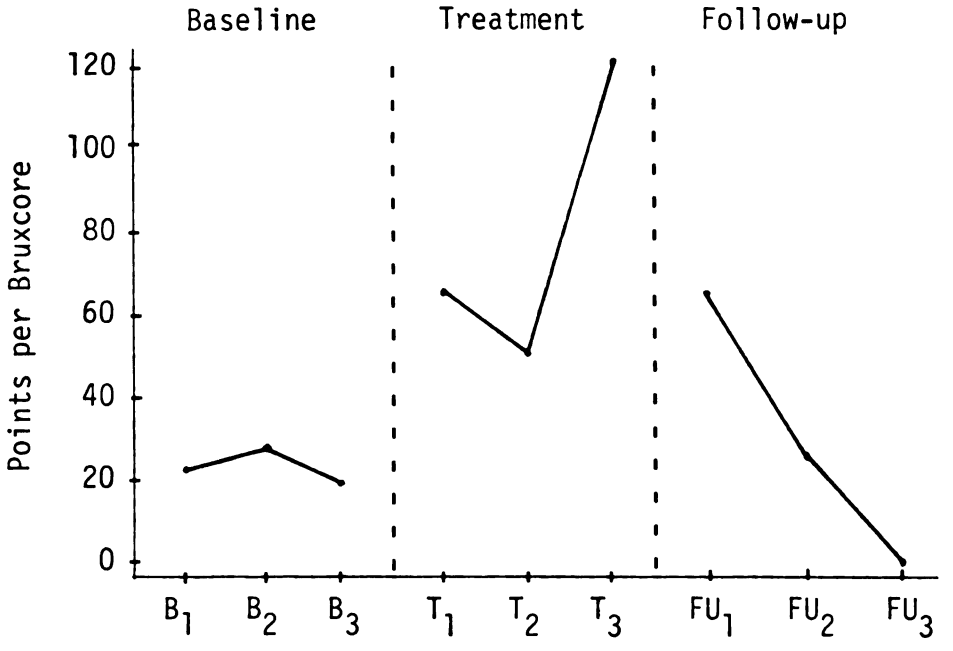


Figure 8.--Subject A weekly Bruxcore figures pre-, during, and post-treatment.

Table 12 shows that there was no significant trend in nocturnal grinding patterns for Subject A from the three baseline weeks through the three weeks of treatment.

Table 12.--Subject A Bruxcore figures without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Bruxcore	22	27	17	62	49	117
Rank	2	3	1	5	4	6
Reading number	6	5	4	3	2	1

$D = 60$ $P\wedge[D \leq 60] > .5$ Not significant at .05 level

Do not reject null hypothesis.

Table 13 shows that when the test was repeated with the three follow-up weeks included, there remained no significant trend in bruxing levels.

Table 13.--Subject A Bruxcore figures with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Bruxcore	22	27	17	62	49	117	64	21	2
Rank	4	5	2	7	6	9	8	3	1
Reading number	9	8	7	6	5	4	3	2	1

$D = 112$ $P\wedge[D \leq 112] = .44$ Not significant at .05 level

Do not reject null hypothesis.

Subject B

The data in Figure 9 indicate that there is no clear trend in the amount of nocturnal bruxing from baseline levels through treatment and follow-up for Subject B. This observation was confirmed by the hypothesis test.

Table 14 shows that there was no significant trend in nocturnal grinding patterns for Subject B from the three baseline weeks through the three weeks of treatment.

Table 15 shows that when the test was repeated including the three weeks of follow-up data, there remained no significant trend in nocturnal bruxing levels.

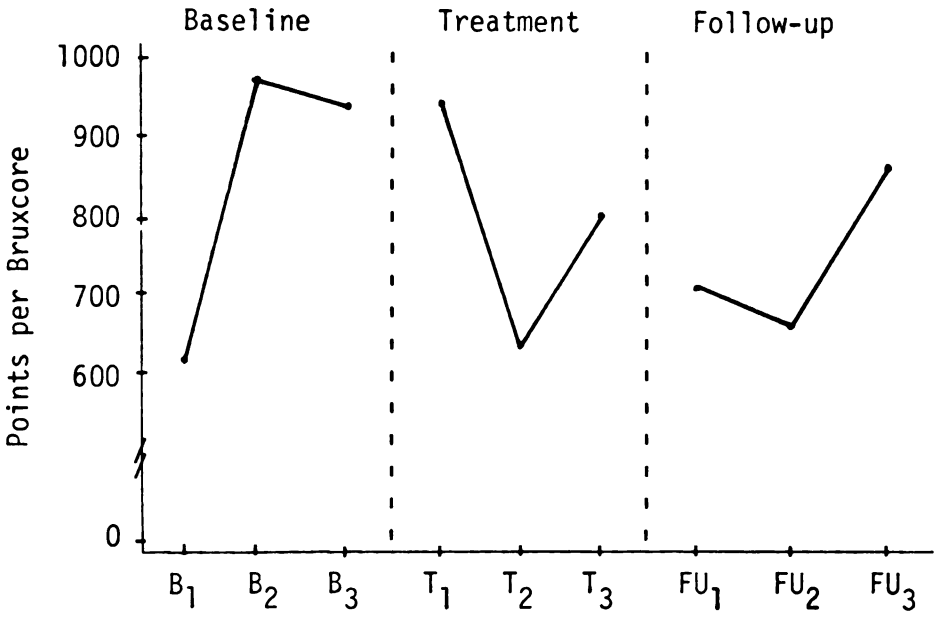


Figure 9.--Subject B weekly Bruxcore figures pre-, during, and post-treatment.

Table 14.--Subject B Bruxcore figures without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Bruxcore	638	947	919	916	642	818
Rank	1	6	5	4	2	3
Reading number	6	5	4	3	2	1

$D = 32$ $P\Delta[D \leq 32] = .4597$ Not significant at .05 level

Do not reject null hypothesis

Table 15.--Subject B Bruxcore figures with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Bruxcore	638	947	919	916	642	818	720	670	866
Rank	1	9	8	7	2	5	4	3	6
Reading number	9	8	7	6	5	4	3	2	1

$D = 104$ $P\wedge[D \leq 104] = .3718$ Not significant at .05 level

Do not reject null hypothesis.

Subject C

The data in Figure 10 show that Subject C experienced a 76 percent decrease in average Bruxcore figures from the baseline phase through the three weeks of treatment. The follow-up data indicate a 63 percent reduction in bruxing behavior from the baseline through the follow-up phase. Subject C seems to have experienced a decreasing trend in nocturnal bruxing behavior from the first week of treatment through termination of the study. This observation was confirmed by the hypothesis test.

Table 16 shows that Subject C experienced a significantly decreasing trend in Bruxcore figures from the baseline phase through the treatment phase.

Table 17 shows that when the test was repeated with the follow-up Bruxcore data included, the decreasing trend maintained significance at the .05 level. Subject C experienced a decreasing trend in nocturnal bruxing from baseline through treatment and follow-up.

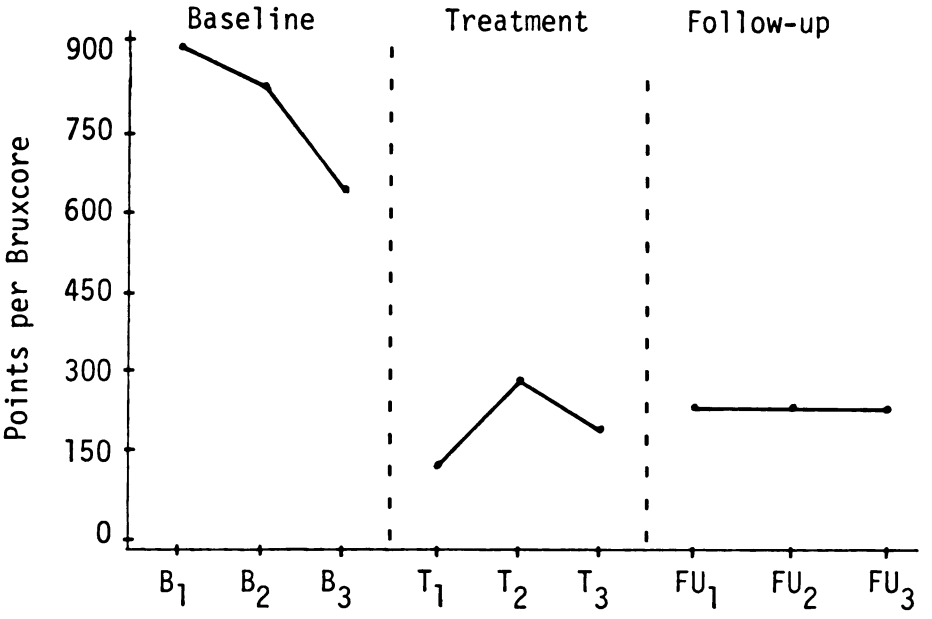


Figure 10.--Subject C weekly Bruxcore figures pre-, during, and post-treatment.

Table 16.--Subject C Bruxcore figures without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Bruxcore	892	868	629	143	258	179
Rank	6	5	4	1	3	2
Reading number	6	5	4	3	2	1

D = 6 $\quad P\wedge[D \leq 6] = .029$ Significant at .05 level

Reject null hypothesis.

Table 17.--Subject C Bruxcore figures with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Bruxcore	892	868	629	143	258	179	294	295	290
Rank	9	8	7	1	3	2	5	6	4
Reading number	9	8	7	6	5	4	3	2	1

$D = 46$ $P\Delta[D \leq 46] = .0429$ Significant at .05 level

Reject null hypothesis--there is a decreasing trend in scores.

Subject D

The data in Figure 11 seem to reflect no clear trend in the pattern of nocturnal bruxing across all phases of the study for Subject D. This observation was confirmed by the hypothesis test.

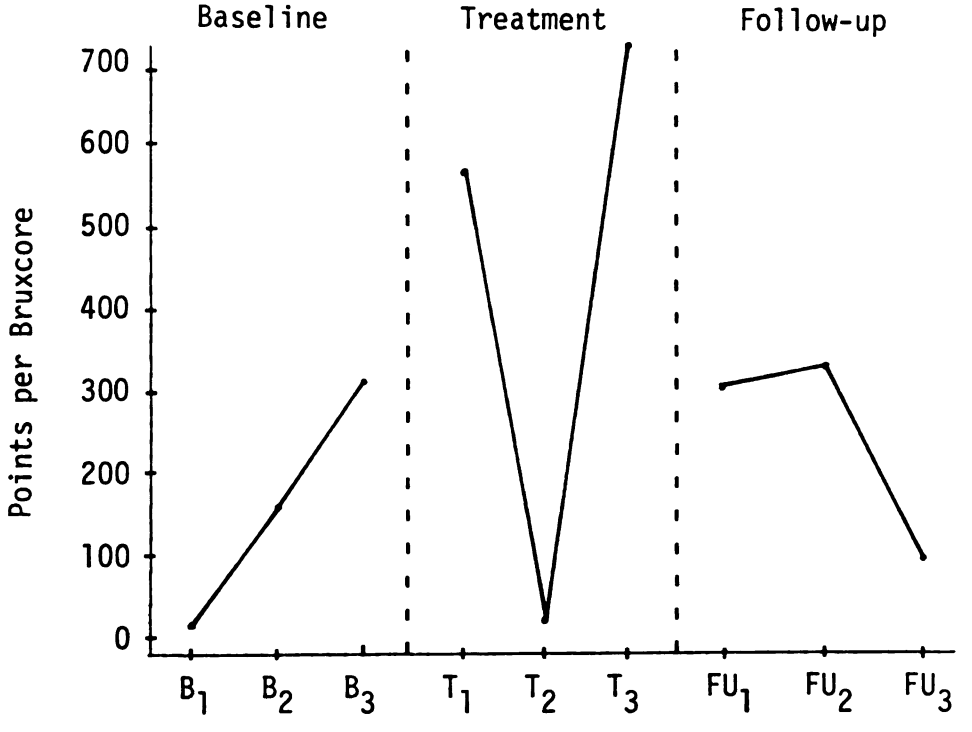


Figure 11.--Subject D weekly Bruxcore figures pre-, during, and post-treatment.

Table 18 shows that Subject D experienced no significant trend in nocturnal bruxing levels from the three baseline weeks through the three-week treatment period.

Table 18.--Subject D Bruxcore figures without follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3
Bruxcore	26	167	307	557	22	748
Rank	2	3	4	5	1	6
Reading number	6	5	4	3	2	1

D = 50 $P\wedge[D \leq 50] < .5$ Not significant at .05 level

Do not reject null hypothesis.

Table 19 indicates that when the test was repeated including the follow-up data, Subject D again experienced no clear trend in nocturnal bruxing behavior.

Table 19.--Subject D Bruxcore figures with follow-up.

	B_1	B_2	B_3	T_1	T_2	T_3	FU_1	FU_2	FU_3
Bruxcore	26	167	307	557	22	748	304	313	97
Rank	2	4	6	8	1	9	5	7	3
Reading number	9	8	7	6	5	4	3	2	1

D = 144 $P\wedge[D \leq 144] < .5$ Not significant at .05 level

Do not reject null hypothesis.

Additional Test

Using the Friedman Rank Sum Test, an additional test was run on the data to see if there was a trend across subjects. Table 20 shows no significant trend in Bruxcore figures across subjects. It cannot be said that all subjects experienced the same phenomena with regard to nocturnal bruxing habits across the phases of this study.

Table 20.--Friedman Rank Sum Test for Bruxcore trends across subjects.

	Subject A		Subject B		Subject C		Subject D		Rank Sum
	Bruxcore	Rank	Bruxcore	Rank	Bruxcore	Rank	Bruxcore	Rank	
B_1	22	4	638	1	892	9	26	2	16
B_2	27	5	947	9	868	8	167	4	26
B_3	17	2	919	8	629	7	307	6	23
T_1	62	7	916	7	143	1	557	8	23
T_2	49	6	642	2	258	3	22	1	12
T_3	117	9	818	5	179	2	748	9	25
FU_1	64	8	720	4	294	5	304	5	22
FU_2	21	3	670	3	295	6	313	7	19
FU_3	2	1	866	6	290	4	97	3	14

Q = 6.6 Not significant at .05 level

Hypothesis 3

H_0: There will be no relationship between EMG levels and Bruxcore figures across baseline, treatment, and follow-up phases.

H_1: EMG levels will be positively correlated to Bruxcore figures across baseline, treatment, and follow-up phases.

The Friedman Rank Sum Test was used

$$\text{test statistic:} \quad Q = \frac{12}{NS(S+1)} \sum_{i=1}^{S} R_i^2 - 3N(S+1)$$

where in this case N = number of subjects

S = number of readings per subject

R_i = sum of ranks for i^{th} reading

Subject A

Table 21 shows that there is no clear relationship between EMG levels and Bruxcore figures for Subject A.

Table 21.--Comparison of EMG levels and Bruxcore figures for Subject A.

	Bruxcore	Ranking	EMG	Ranking	Rank Sum
B_1	22	4	1.543	9	13
B_2	27	5	1.319	7	12
B_3	17	2	1.533	8	10
T_1	62	7	.723	5	12
T_2	49	6	.539	1	7
T_3	117	9	.595	2	11
FU_1	64	8	.672	3	11
FU_2	21	3	.704	4	7
FU_3	2	1	.747	6	7

Q = 3.066 Not significant at .05 level

Do not reject null hypothesis.

Subject B

Table 22 shows that there is no clear relationship between EMG levels and Bruxcore figures for Subject B.

Table 22.--Comparison of EMG levels and Bruxcore figures for Subject B.

	Bruxcore	Ranking	EMG	Ranking	Rank Sum
B_1	638	1	11.268	7	8
B_2	947	9	17.068	9	18
B_3	919	8	14.561	8	16
T_1	916	7	1.617	6	13
T_2	642	2	1.200	5	7
T_3	818	5	.838	2	7
FU_1	720	4	.719	1	4
FU_2	670	3	.880	3	6
FU_3	866	6	1.094	4	10

Q = 10.866 Not significant at .05 level

Do not reject the null hypothesis.

Subject C

Table 23 shows that there is no clear relationship between EMG levels and Bruxcore figures for Subject C. It should be noted, however, that the inclusion of follow-up data caused the positive relationship occurring between the baseline and treatment phases to become insignificant at the .05 level across baseline treatment and follow-up.

Table 23.--Comparison of EMG levels and Bruxcore figures for Subject C.

	Bruxcore	Ranking	EMG	Ranking	Rank Sum
B_1	892	9	1.121	7	16
B_2	868	8	1.819	9	17
B_3	629	7	1.472	8	15
T_1	143	1	.498	5	6
T_2	258	3	.445	1	4
T_3	179	2	.456	2	4
FU_1	294	5	.461	3	8
FU_2	295	6	.525	6	12
FU_3	290	4	.474	4	8

Q = 14 Not significant at .05 level

Do not reject the null hypothesis.

Subject D

Table 24 shows that there is no clear relationship between EMG levels and bruxism figures for Subject D.

A summary of the data yielded from Hypothesis 3 indicates that in no case was the trend in EMG levels related to the trend in Bruxcore levels across baseline treatment and follow-up.

Table 24.--Comparison of EMG levels and Bruxcore figures for Subject D.

	Bruxcore	Ranking	EMG	Ranking	Rank Sum
B_1	26	2	1.858	7	9
B_2	167	4	2.213	9	13
B_3	307	6	1.932	8	14
T_1	557	8	.967	6	14
T_2	22	1	.813	5	6
T_3	748	9	.811	4	13
FU_1	304	9	.811	4	13
FU_2	313	7	.784	3	10
FU_3	97	3	.698	1	4

Q = 7.467 Not significant at .05 level

Do not reject null hypothesis.

Hypothesis 4

H_o: There will be difference in pre- and post-study periodontal reports of bruxing criteria.

H_1: There will be less bruxing criteria recorded during the post-study periodontal observation than in the pre-study observation.

A profile of tooth mobility will be graphically presented for each subject, followed by a comparative listing (pre- and post-study) of (1) wear facets, (2) masseter hypertrophy, (3) pain upon palpation, and (4) maximum mouth opening in millimeters.

Scoring for tooth mobility was done in the following manner:

0 = no clinical mobility

+ = slight mobility noted (slightly more than physiologic range but less than 1 mm buccal-lingual)

+1 = clinical mobility approximately 1 mm buccal-lingual

+2 = clinical mobility approximately 2 mm buccal-lingual

+3 = clinical mobility greater than 2 mm buccal-lingual

Subject A

Figure 12 (a and b) shows slightly less tooth mobility in the upper and lower dentition of Subject A after the study. Figure 12 (b) indicates that teeth numbers 28 and 29 were significantly more stable after the study.

Dr. Wisser observed the following comparisons of bruxing criteria in Subject A before and after the study:

1. Wear facets were observed; wear facets remained unchanged.

2. No masseter muscle hypertrophy was noted before or after the study.

3. Subject A reported no discomfort upon palpation of the masseter muscle before or after the study.

4. Maximum mouth opening before the study was 40 mm, and 41 mm after the study.

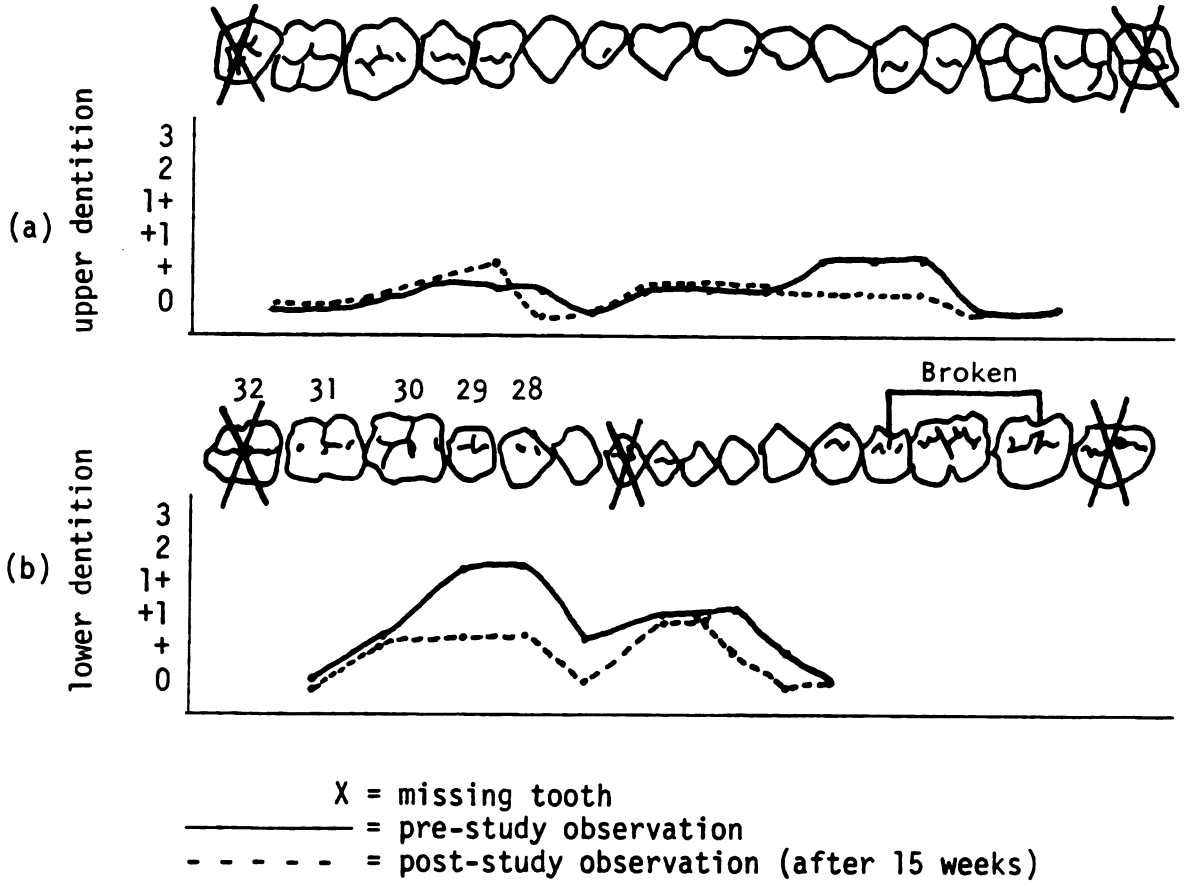


Figure 12.--Tooth mobility pre- and post-study for Subject A.

Subject B

Figure 13 (a and b) shows that there was slightly less tooth mobility in both the upper and lower dentitions of Subject B after the study.

Dr. Wisser observed the following comparisons of bruxing criteria in Subject B before and after the study:

1. Wear facets were noticed before the study and remained unchanged after the study.

2. Slight hypertrophy of the right masseter muscle was noticed before and after the study.

3. Some discomfort was noticed upon palpation of the right masseter muscle before and after the study.

4. Subject B was able to open her mouth to 34 mm before the study and to 38 mm after it.

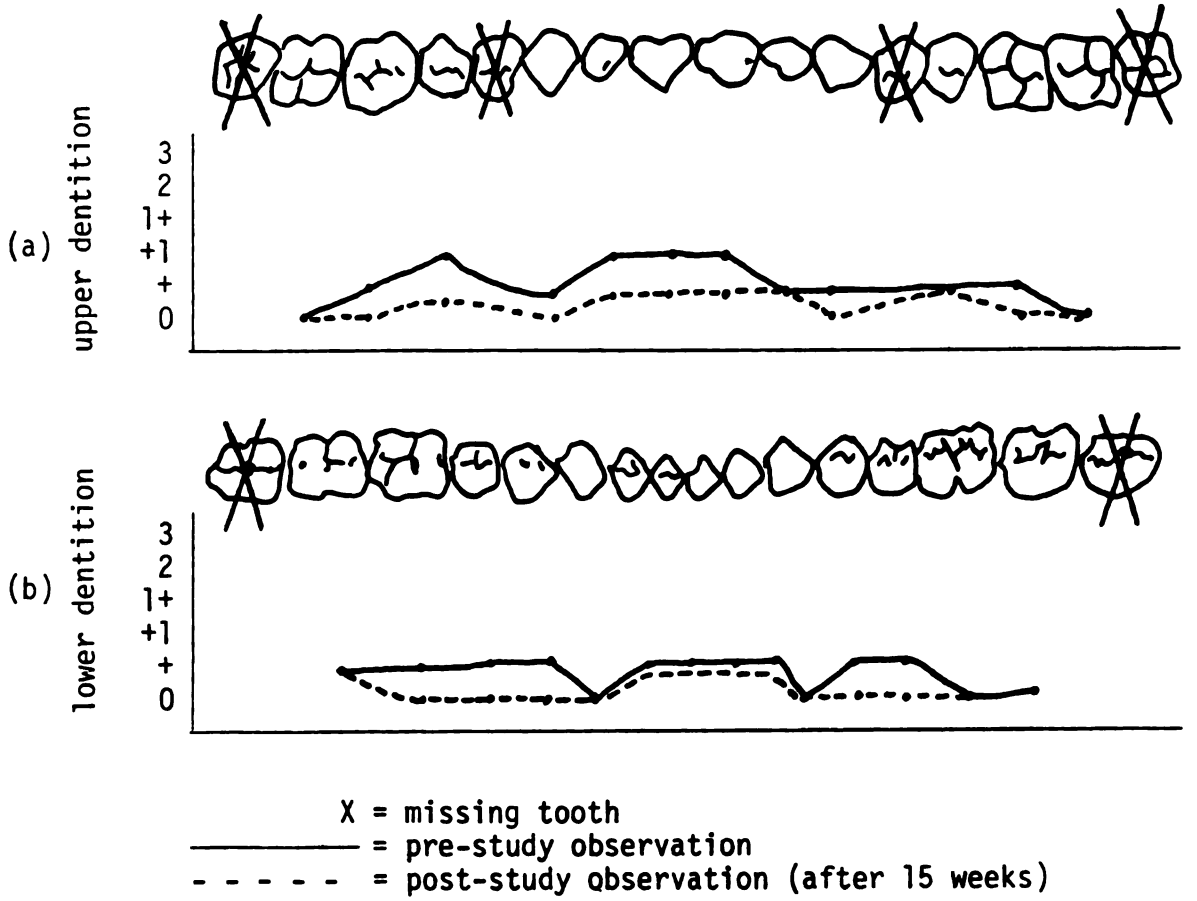


Figure 13.--Tooth mobility pre- and post-study for Subject B.

Subject C

Figure 14 (a) shows slightly less tooth mobility after the study in the upper dentition of Subject C. Figure 14 (b) shows almost no change in the tooth mobility of Subject C's lower dentition.

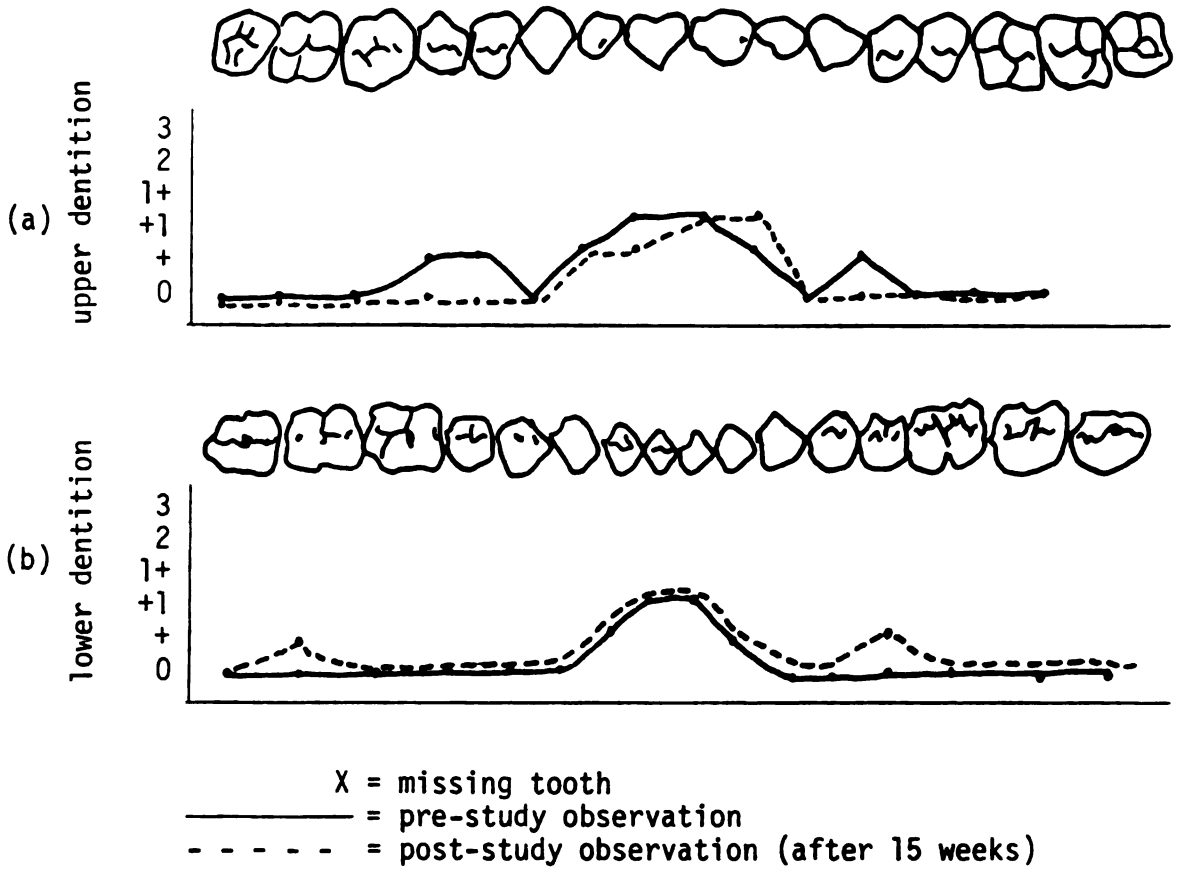


Figure 14.--Tooth mobility pre- and post-study for Subject C.

Dr. Wisser observed the following comparisons of bruxing criteria in Subject C before and after the study:

1. Wear facets were noticed before the study and remained unchanged after it.

2. A slight amount of masseter muscle hypertrophy was noticed before and after the study.

3. Discomfort upon palpation of the masseter muscle was noted before the study, but was absent after the study.

4. Subject C was able to open his mouth to 59 mm pre-study and to 61 mm post-study.

The periodontist reported that Subject C seemed to have reduced his clenching behavior (daytime habit) enough to allow masseter muscles to relax, mouth-opening ability to increase, and facial pain to subside entirely.

Subject D

Figure 15 (a and b) shows slightly less tooth mobility in both the upper and lower dentitions of Subject D after the study. Figure 15 (a) shows that teeth numbers 7 and 8 were significantly more stable after the study.

Dr. Wisser observed the following comparisons of bruxing criteria in Subject D before and after the study.

1. Wear facets were noticed before the study and remained unchanged after the study.

2. Masseter muscle hypertrophy was noticed before and after the study.

3. No discomfort was reported upon palpation of the masseter muscles before or after the study.

4. Maximum mouth-opening ability was measured at 42 mm before and after the study.

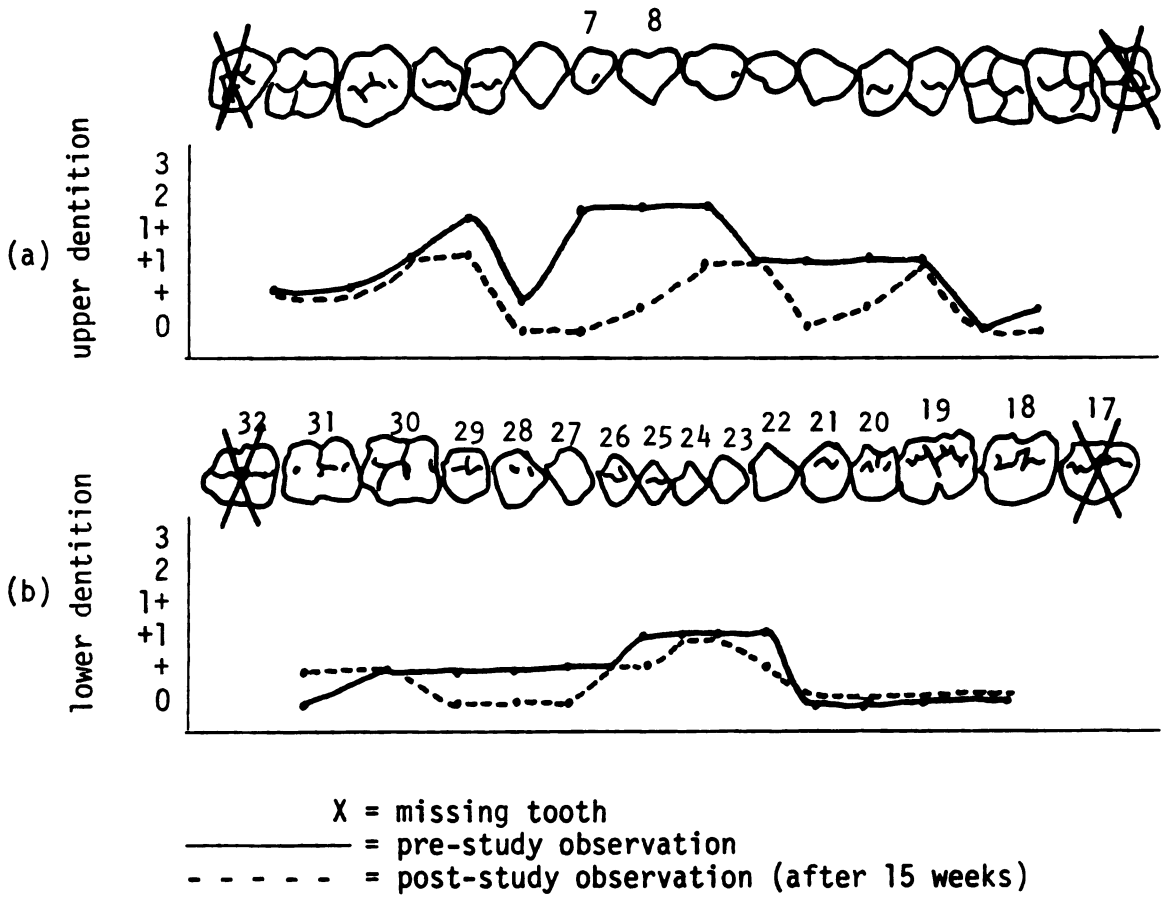


Figure 15.--Tooth mobility pre- and post-study for Subject D.

Ms. W. reported to the periodontist in the pre-study clinical observation that she felt she was a nighttime grinder. She reported waking in the morning with pain in her temples and jaws. After the study, she reported that she realized that she clenched during the day. She stated she had learned to "catch herself" doing it, and by relaxing her facial muscles she could stop clenching. She reported experiencing less pain throughout the day and in the morning.

In summary, according to the periodontal observations there seems to have been a significant reduction in tooth mobility in Subjects A and D. The other subjects demonstrated some stabilizing but did not meet the criterion of a difference in pre- and post-scores of two or more points.

Subject B was able to open her mouth significantly more after the study.

All subjects reported a keener awareness of daytime bruxing. All subjects reported enjoying the relaxation training. Only Subject B reported having trouble generalizing the learned response to her daily routine.

Hypothesis 5

H_0: There will be no difference in subjects' reports of bruxing criteria before and after the study.

H_1: Subjects will report less bruxing criteria present after the study than before the study.

Data regarding this hypothesis were collected from a self-report questionnaire described in Chapter III and included in Appendix C. During a pre- and post-study administration, subjects were asked to recall the number of times throughout the previous week they noticed bruxing behavior, and how often the grinding was noticed by a friend or spouse. They also were asked to rate headache and facial pain presumably experienced from the bruxing on a 1 to 10 scale with 1 representing no pain and 10 representing what they considered to be unbearable pain. Along with severity, subjects were asked to estimate

the duration of the pain and to report whether they experienced pain upon opening their mouths.

A graphic representation of the subjects' answers to these questions (pre- and post-study) will be presented, after which a description of characteristics of the pain experienced and anecdotal notes from conversations with the experimenter will be included.

Subject A

In answer to the question of whether pain was experienced when opening the mouth wide, Subject A reported no pain pre- or post-study.

The pain experienced by Subject A was felt in the jaw muscles both pre- and post-study, and was noticed most often in the late afternoon both pre- and post-study.

Figure 16 shows that Subject A reported having become more aware of his clenching behavior during his working day. He stated on several occasions he noticed himself (three to four times per day) biting down in response to some stressful event, such as having to administer discipline in the classroom. Mr. W expressed that this increased awareness allowed him to control the clenching behavior more than he had previously been able to do. He pointed to the decrease in duration of pain experienced from one hour to several minutes as an outcome of what he considered a heightened awareness and increased ability to control jaw muscle tension.

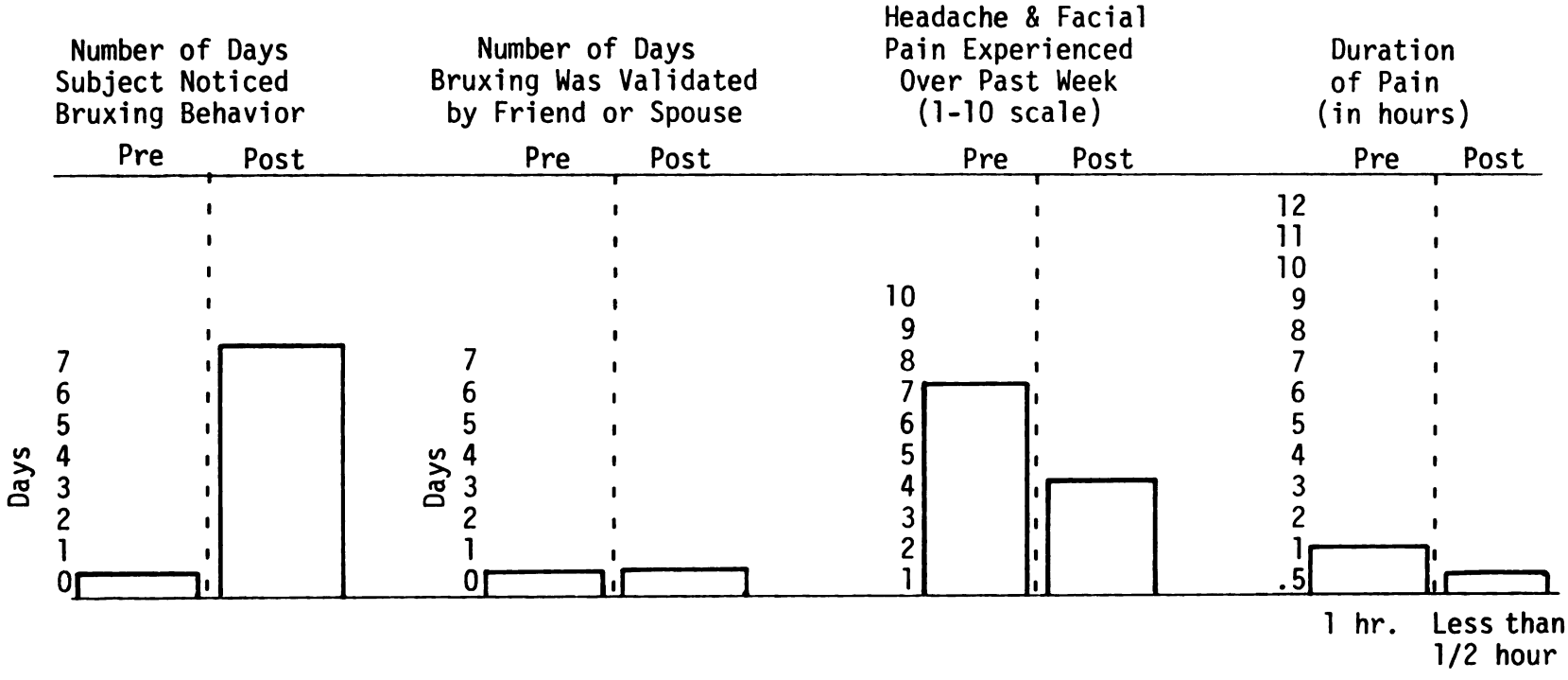


Figure 16.--Subject A pre- and post-study self-report.

Subject B

In answer to the question of whether pain was experienced when opening the mouth wide, Subject B reported yes both pre- and post-study.

The pain experienced by Subject B was felt in the right ear and neck area before the study and the right ear, neck, and forehead post-study.

Ms. B. reported noticing the pain most often in the early morning and late at night pre-study, and early morning, late night, and one to three times during her working day post-study.

Figure 17 shows that after the study, Subject B reported that she had become more aware of her grinding. She now was noticing her muscle tension during the day and reported being able to reduce the duration of the pain by relaxing her facial muscles. She also reported experiencing the same intensity of the pain and that after the study she felt it in her forehead as well as jaw and neck areas. She stated that during exam periods, babysitting duties, and sometimes when work became more hectic than usual, she experienced more pain in the early morning. Ms. B. also reported that relaxing was difficult because it seemed to require great concentration, and such effort was not easy to maintain in the face of the many environmental constraints she felt subjected to daily. In short, she admitted that although she was able to relax in the office and during the "experimental" situation, she felt that generalizing the effect of muscle relaxation to her daily routine was problematic.

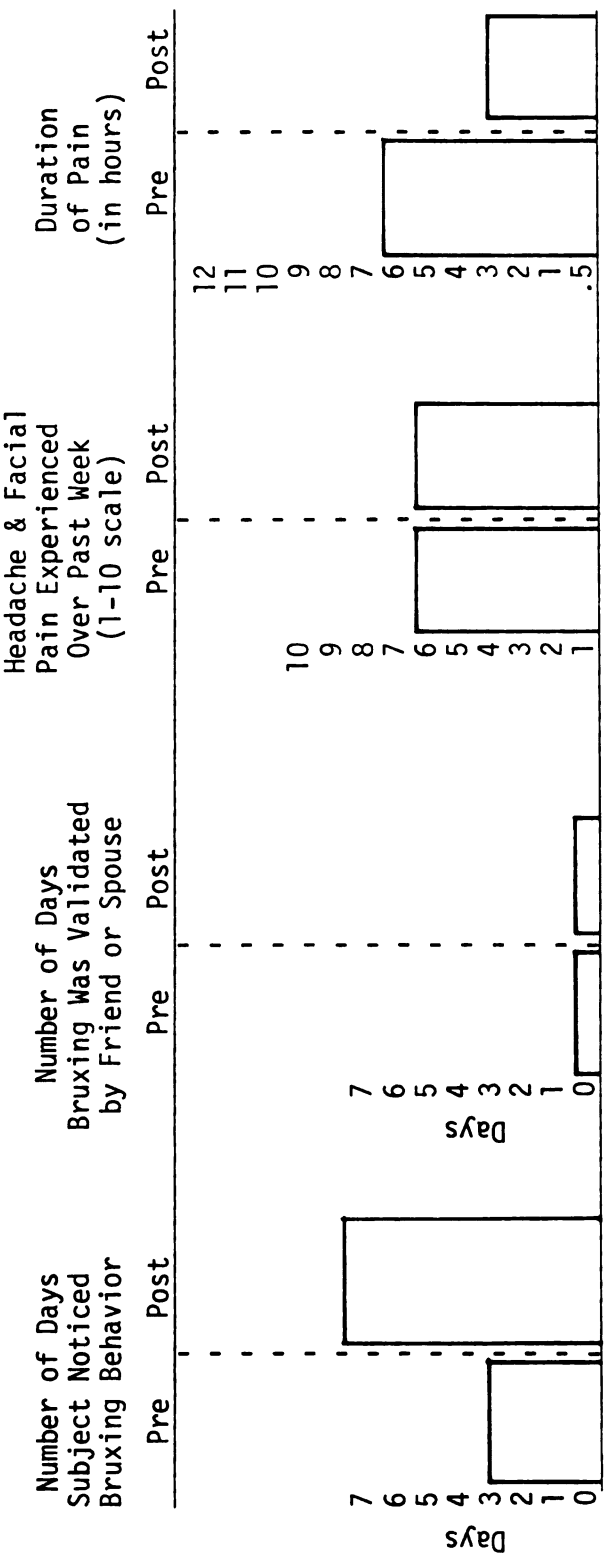


Figure 17.--Subject B pre- and post-study self-report.

Subject C

In answer to the question of whether pain was experienced when opening the mouth wide, Subject C reported yes pre-study and no post-study.

The pain experienced by Subject C was felt in the jaw muscles pre-study, and no pain was reported post-study.

Figure 18 shows that Subject C reported noticing the pain at all times throughout the day, with particular emphasis on late afternoon. Again, he reported noticing no pain after the study at any time, day or night.

Mr. J. had reported clenching his teeth while on the job. He is a carpenter and was particularly aware of clenching while hammering, sawing, and performing any physical labor indigenous to his trade. Nearly half-way through the treatment phase of the study, Mr. J. reported being more aware of his clenching habit and that he was more able to reduce the intensity of the clenching by inducing masseter muscle relaxation.

During the last week of the treatment phase, Mr. J. reported attaining a "second level" of relaxation. The first, he said, was as though he had "stripped off" several layers of tension. The second was more profound and "restful." He reported that while the first level was attainable within minutes of initiation, the second, more profound level required great concentration. He stated that he would strive to attain the "second level" just before bed in hopes of reducing his bruxing habit.

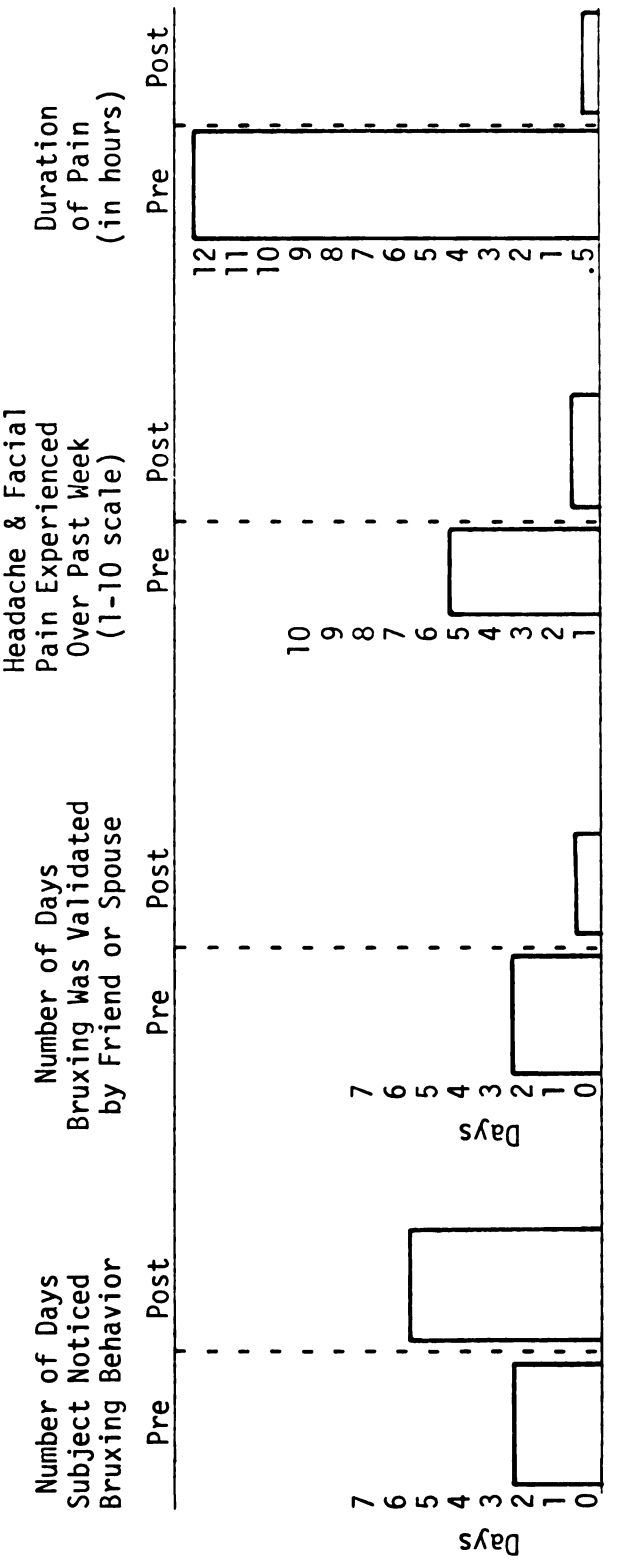


Figure 18.--Subject C pre- and post-study self-report.

Subject D

In answer to the question of whether pain was experienced when opening the mouth wide, Subject D reported yes pre-study and no post-study.

The pain experienced by Subject D was felt in the temples before the study, and no pain was reported after the study.

Ms. W. reported noticing the pain after 2:00 p.m. on days before the study. No pain was reported at any time after the study.

Figure 19 shows that Ms. W. reported experiencing more pain and clenching behavior prior to exams and other school-related stressful events (i.e., reports and projects). She also reported that her response to the question regarding the number of times she noticed bruxing behavior over the previous week was exaggerated because, by coincidence, that happened to be her final exam week. She also reported that after the treatment she never noticed herself grinding or clenching because she "caught" herself beginning to tense before the clenching behavior became chronic.

During the third training session in the second week of the treatment phase, Ms. W. reported she had been headache free for the entire previous day. When asked to explain the significance of this observation, she stated she had experienced some form of headache and facial pain every day for several years, and that she felt that letting her jaws and temples relax had caused her to abort this long-term low-grade ache.

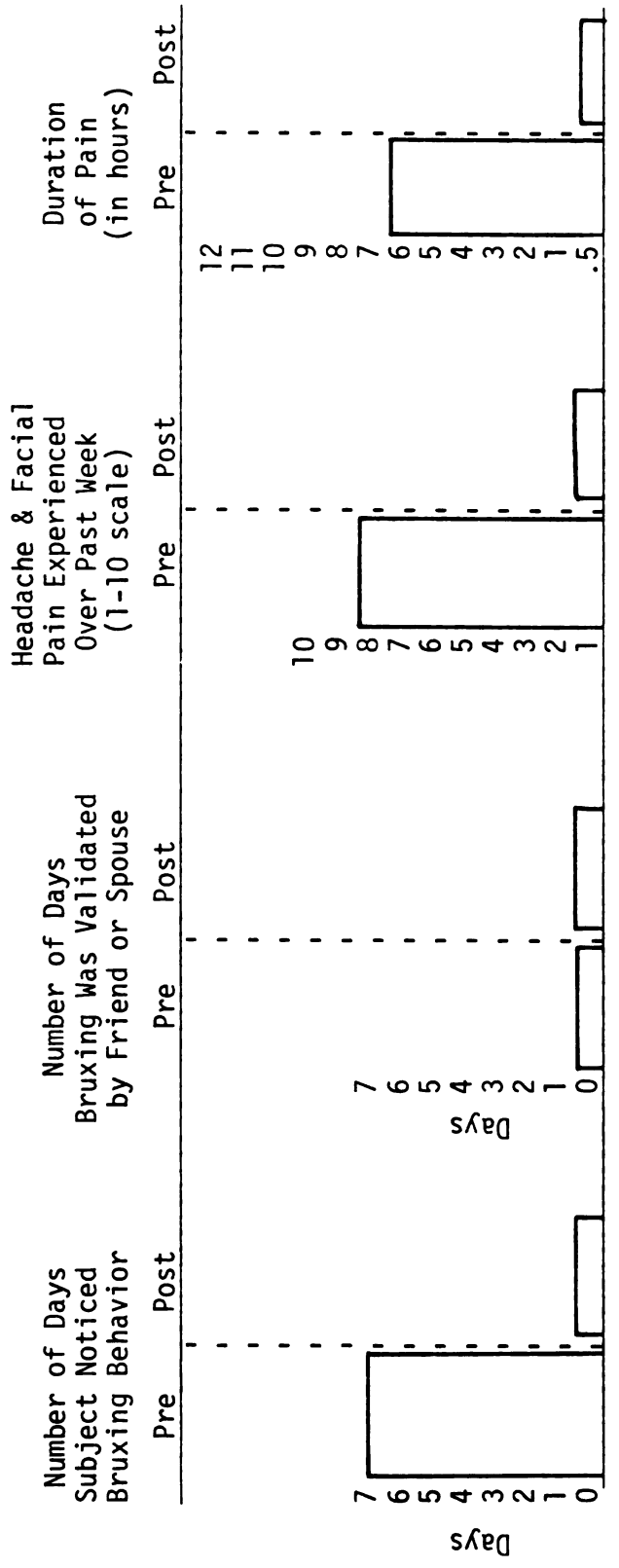


Figure 19.--Subject D pre- and post-study self-report.

In relation to the hypothesis concerning pre- and post-study self-report of awareness of bruxing and pain presumably felt in response to bruxing, it seems that subjects became more aware of the behavior rather than reporting it less often. There also seemed to be a lack of validation of the habit from friends and/or spouses. Subjects tended to report experiencing less headache and facial pain after the study, and those who continued to experience some pain reported a decrease in its duration after the study. Two of the three subjects reporting pre-study pain upon mouth opening reported no such pain after the study. Every subject reported clenching behavior during the day in addition to bruxing at night. Throughout the study, anecdotal notes indicated that the subjects became more aware of this daytime component and reported being more able to affect it with relaxation than the more insidious nocturnal grinding of which they reported being less aware.

CHAPTER V

SUMMARY, CONCLUSIONS, AND DISCUSSION

In this chapter, the study is summarized, conclusions and limitations based on data analysis and methodology are explored in a discussion section, and implications for future research and therapy are presented.

Summary

The primary purpose of this study was to explore potential impact of intensive biofeedback training (EMG) on bruxism. The first question to be dealt with was whether EMG training would, in fact, produce a learned relaxation response in the subjects. Assuming that subjects would learn to voluntarily relax their masseter muscles, the next question was whether this voluntary response, if initiated several times during the day and once just before going to sleep at night, would reduce the subjects' nighttime bruxing pattern.

A review of the literature was conducted in the areas of a historical perspective of bruxism, epidemiology, etiology, and current treatment modalities.

Bruxism has been widely defined as the grinding and/or clenching of teeth during the day or night. It has been reported to exist as a habit in as much as 80 percent of the population, although most conservative estimates approximate a 20 percent figure. It seems to be

equally present in males and females and can be found in nearly any age group and occupational category.

The effects of bruxism range from being a mildly annoying habit causing minimal tooth damage and occasional muscle discomfort, to a severe problem involving the slow deterioration of tooth material, damage to underlying bone structures, and severe myofacial pain or chronic tension headaches.

The cause of bruxism has yet to be clearly established. Current theory suggests any one or a combination of the following possibilities. Local or mechanical problems such as improper bite planes, malocclusions, high restorations, or new bridgework make up one category of potential cause. It is felt that any alteration of "freeway space" (that space between the teeth when they are lightly clenched) will cause the person to grind in an attempt to alleviate the obstruction.

Another etiological factor often considered is that of psychological reactions. There has been much work to determine a personality profile of the bruxer, but much of this work remains conflicting and speculative in nature. One of the most stable findings from this premise, however, is that most bruxers tend to exhibit high levels of anxiety and muscle tension. It seems that, during periods of intense stress, muscle tension is initiated at high levels and often manifested in the muscles of mastication. The grinding and clenching then is assumed to be a reaction to stress on the part of the subject, and an attempt to actively cope with it or defend against it.

A final classification of possible cause is physiological or systemic in nature. Studies have been done in which the bruxing subject could not produce the behavior in a wakeful state. Only during deep sleep can the behavior be observed in some chronic bruxers. At these times the grinding motion is slower and more intense than in the wakeful clenching type. Although each of these possible etiologies seems logical and accurate to some extent, there remains no definite origin for bruxing behavior nor any clear understanding of its evolution into a potentially deleterious syndrome.

Treatments for bruxism have been nearly as prolific as etiological theories. Dental treatments include occlusal adjustments, ranging from oral surgery to simply grinding the teeth down to a point that they cannot possibly touch prematurely. A less severe strategy is that of manufacturing a bite splint or mouthguard to be worn by the patient at night. This device is thought to retrain tense muscles to a point where they do not violently contract causing bruxism, while at the same time protecting patients' teeth from further damage caused by grinding.

Other treatments include massed negative practice, a behavioral technique involving voluntary contraction of the masseter muscle to the point of fatigue, presumably causing the muscle to rest during the night rather than spasm in a bruxing fashion. Relaxation procedures have been employed, including progressive relaxation and variations of meditative techniques.

All these treatment modalities, from the irrevocable surgical and dental intervention to the less invasive instructive procedures,

have relied upon patient self-report for outcome measures. Both the level of relaxation attained and the subsequent abatement of the bruxing behavior have been measured by clinical observation and patient report sometimes validated by a friend or spouse who may have witnessed the behavior or its possible reduction.

It is the ambiguous and often conflicting nature of these self-reported results which has provided the impetus for this study in several ways. First, it was an attempt to provide a more objective measurement of muscle tension and bruxing levels. Second, it was an attempt to measure more clearly the impact of biofeedback training on masseter muscle tension levels, and finally to more accurately assess the possible impact of a learned relaxation response on bruxism.

Four subjects, two males and two females, from ages 23 to 55 were selected by a periodontist on the basis of bruxing criteria. Dependent measures included pre- and post-study periodontal observations recorded on a standard dental chart. Pre- and post-study self-report questionnaires were administered which tested subject awareness of bruxing behavior and subjective reports of pain presumably felt as a result of bruxing. Pre- and post-study masseter muscle tension levels were measured on an Autogenic 1100 EMG machine and data recorded from the illuminated meter of the Autogenic 5100 digital integrator. Bruxism levels throughout the study were measured by the Bruxcore device (Forgione, 1974), a multicolored, laminated polyvinyl disc covered with microscopic dots and pressed into a tight-fitting plate covering the subject's upper dentition. The Bruxcore was worn for four consecutive nights, yielding a volumetric index of the subjects'

bruxing behavior. EMG levels were measured once per week in a session lasting approximately 20 minutes and consisting of 10 separate 30-second EMG mean readings.

After three weeks of baseline data were collected, the subjects underwent intensive biofeedback training. A 30-minute training session was repeated four times per week for three consecutive weeks. During the training sessions the subjects were instructed in biofeedback procedures and given audio and visual feedback regarding muscle tension. Bruxcore devices were worn during this time to provide a continuous index of bruxism throughout the study.

After the three-week training program, each subject was asked to return to the office for a one-week follow-up visit consisting of baseline EMG and Bruxcore data collection. This process was repeated one month and six weeks post-treatment, yielding three follow-up measurements. After the sixth week post-treatment, all subjects returned to the periodontist for a post-study observation.

The study followed a time-lagged multiple baseline design to avoid any historical effects on the data as well as to provide a clear representation of the possible effect of biofeedback training on a stabilized baseline measurement of both bruxism and existing muscle tension levels.

Hypotheses included presumed downward trends in both EMG levels and Bruxcore indexes as well as reductions in bruxing criteria as measured in periodontal observations and subject self-reports.

The hypothesized trends in EMG and Bruxcore data were measured by a Testing Against the Trend analysis using Spearman's statistic at

the .05 level of confidence. Friedman's Rank Sum was used to determine whether there were any trends across subjects. Subjects' periodontal data were recorded on a dental chart and presented in graphic and descriptive form, as were subject self-report data.

Discussion

This section will discuss conclusions drawn from the results of the statistical analysis and limitations of the study.

Hypothesis 1: EMG Levels

When the subjects' EMG levels were tested for a possible decreasing trend from baseline through the treatment phase, all four subjects experienced such a trend at the .05 level of significance.

It may be observed then that all subjects experienced a general reduction of EMG levels during the biofeedback training phase of the study. When the EMG levels for the three follow-up sessions were added to the analysis, however, only Subjects B and D maintained the decreasing trend to the .05 level. The EMG levels of Subjects A and C were significant at the .06 level which, while seeming rather stable, could have occurred by chance. It would seem that all subjects learned a relaxation response but that perhaps Subjects A and C began to lose the ability albeit only slightly over the follow-up phase. The Friedman Rank Sum Test performed across subjects indicated that all subjects experienced a similar decreasing trend at approximately the same rate throughout the study. In this case it would appear that the biofeedback training had the desired effect of teaching each subject to voluntarily reduce masseter muscle tension significantly below baseline

levels. While these data seem to reflect a promising relationship between biofeedback training and voluntary control of muscle tension, it must be remembered that there are no normative data in the literature relative to masseter muscle EMG readings. Therefore, the levels recorded for each subject reflect only what was going on for each individual and in no way indicated how that individual compared to a "normal reading" or a known threshold of relaxation. The absence of such normative standards prohibits any conclusions regarding minimum levels of EMG activity needed to insure the presence of a learned relaxation response. In this study, the presence of such a response was assumed on the basis of the difference in pre- and post-training EMG levels. Another factor which may have obscured the results somewhat is that of artifact in the EMG readings. Eye blinking and swallowing along with random facial movements were not controlled. Subjects seemed to learn not to do these behaviors while the digital integrator was averaging their EMG activity, but once again it is presumed that since learning was the same for all subjects, fluctuations in the amount of artifact present in EMG levels were randomly distributed. With due consideration of these limitations it seems reasonable to observe that after receiving biofeedback training all four subjects could voluntarily relax their masseter muscles. The next step was to measure bruxing levels throughout the study and test for a possible treatment effect.

Hypothesis 2: Bruxing Levels

When the subjects' Bruxcore levels were tested for a possible decreasing trend from baseline through the treatment phase, only

Subject C experienced such a trend at the .05 level of significance. When the test was run including data from the follow-up weeks of Bruxcore wear, Subject C remained the only person to experience a significantly decreasing trend. The additional test to see whether there was any trend across all four subjects was not significant at the .05 level. There was no clear trend; hence the subjects cannot be said to have experienced the same phenomenon with regard to nocturnal bruxing patterns in any of the phases of this study. Subject C, however, did experience a significant decreasing trend and can be said to have reduced his nocturnal bruxing below baseline levels during and after the biofeedback training.

Several limitations regarding the Bruxcore device must be considered when reflecting upon the possible conclusions drawn from the data presented above. The experimenter had no way of insuring that the subjects would actually wear the device when requested to do so. In this study, subjects were asked to fill out a card (Appendix F) indicating the time of day at which the device was inserted and removed. All subjects returned every card completely and appropriately filled; that is, each subject reported wearing the Bruxcore when asked to do so. On this point the experimenter can only assume that the subjects were being truthful.

Another potential error factor is that of the subjective nature of scoring the Bruxcore devices. The scoring procedure was accomplished by subjective observation through a microscope. In an attempt to account for some of the potential error in this scoring method, one Bruxcore from each phase for each subject or a total of 12 Bruxcores

were rescored by a second technician. These scores were compared with those done by the original scorer, yielding the following reliability coefficients:

Subject A r = .96

Subject B r = .98

Subject C r = .97

Subject D r = .97

Whereas these figures seem to reflect a high degree of scorer reliability, the data are still subject to some error.

Another limitation is the variability of bruxing scores within subjects. Forgione (1974), Kardachi and Clarke (1977), Reding (1968), and Rugh (1978) have all suggested that bruxing behavior varies greatly from person to person and within a given person from time to time. This variability has made any comparative study of the habit a difficult task, especially when using a large group of subjects. The results of this study seem consistent with the literature in this regard. Perhaps a three-week baseline was not sufficient to clearly establish a pattern from which to measure possible treatment effects.

Perhaps the greatest limitation regarding the Bruxcore device and its use in measuring bruxing behavior is the fact that the subjects only wore them at night. Thus only a record of nocturnal bruxing was obtained. Throughout the study it became obvious that each subject exhibited some form of bruxing behavior during the day as well. However, this daytime component was not objectively measured and must remain speculative, as do all the data pointing to a reduction of this part of the overall problem.

Hypothesis 3: Correlation of EMG Levels to Bruxism Levels

The null hypothesis stating there would be no relationship between EMG levels and Bruxcore figures across baseline, treatment, and follow-up could not be rejected at the .05 level for any subject. While it seems that all subjects experienced a similar decreasing trend in EMG levels after the biofeedback training, the Bruxcore levels were so variable within and across subjects that no clear correlation could be established. Perhaps with more baseline weeks some of the variability could be reduced to permit the testing for such a positive correlation in future studies.

Hypothesis 4: Periodontal Observation

As a result of the pre- and post-treatment periodontal observations, it was demonstrated that tooth mobility was reduced somewhat in all subjects. In Subjects A and D, two of the teeth measured, rated two increments more stable in the post-treatment observation. The periodontist identified these changes as being the most significant indications of a reduction in bruxing behavior. However, it must be noted that neither Subject A nor D experienced a significant reduction in nocturnal bruxing behavior as measured by the Bruxcore device. It is possible that both Subject A and D experienced a reduction in the amount of clenching and biting behavior exhibited during the daytime. It is also possible that the reduction of this diurnal parafunction could have allowed for their teeth to become significantly more stable over the 15-week duration of the study.

Consistent with these findings were the measurements of maximum mouth opening. Subjects A, B, and C increased their maximum opening potential and Subject D remained the same. Although none of these increases are significant by themselves, taken in combination with tooth mobility changes they seem to indicate the absence of chronic masseter muscle tension presumably responsible for limitations of maximal opening potential.

Pain upon palpation of the masseter muscle was noticed in Subjects B and C before the treatment and only in Subject B after the study. Subject C had reported catching himself clenching during the performance of his daily work as a carpenter. After the biofeedback training he reported consciously relaxing his jaws whenever he became aware of the clenching, and he reported to the periodontist that he had noticed the absence of facial pain and tension headaches. These had been routine occurrences before the study. The data once again suggest a clear differentiation between diurnal and nocturnal bruxism, with the daytime problem appearing more amenable to muscle relaxation training than the unconscious habit.

In general, the periodontal observations recorded what appear to be significant reductions in bruxing criteria in Subjects A, C, and D, with fewer such indications for Subject B.

There were several limitations with regard to the pre- and post-study periodontal observations. One of the generally agreed-upon criteria for the diagnosis of bruxism is the size of the periodontal ligament. If the ligament is enlarged, it is a sign of inflammation often caused by bruxism. Absence of such enlargement is a sign the

bruxing behavior is not severe or is nonexistent. X-rays are required for examination of the periodontal ligament. X-rays are costly and carry some level of risk, especially when administered too often. Not including the examination of this structure limits the accuracy of the diagnosis of bruxism and limits the potential for post-study comparison since inflammation might be more amenable to treatment than some of the other outcome measures used.

Wear facets were used as a pathonomonic indication of the presence of bruxing behavior. Unfortunately, wear facets are rather permanent in nature and don't change enough to be noticed within the space of 15 weeks. No method of measuring wear facets has been developed fully enough to use in this fashion. Once again, the limitation is that this is one less possible outcome measure for the efficacy of the treatment.

The periodontal observations were recorded on a standard dental chart. Questions were asked of the subjects, and their answers were written down in the margin of that document. A more organized forced-choice type of form would have yielded much more comparable data. Also, the fact that only one periodontist was used limits the generalizations that can be made from the data since there is almost certainly some variance among how other practitioners observe and record the signs of bruxism.

Hypothesis 5: Subject Self-Report

The hypothesis regarding subjects' self-reported bruxing criteria was also generally demonstrative of a reduction from pre- to post-study, with several noteworthy exceptions.

In response to the question of how often over the past week the subject had noticed bruxing behavior, only Subject D responded that she had noticed significantly less grinding. Subjects A, B, and C all reported noticing significantly more episodes over the final week than were observed in the week prior to the beginning of the study. It remains unclear as to whether this increase in the data reflects a rise in actual bruxing behavior or might better be explained as a heightened awareness of the habit where such an awareness previously had been absent. When questioned verbally about their response to question one, Subjects A, B, and C reported that they had become more aware of the daytime clenching rather than having actually bruxed more often. This lack of clarity with regard to the meaning of subject responses to the questionnaire reflects one of the greatest limitations of the present study.

The self-report was simply too short and administered too infrequently. The questions were worded to inquire about the subjects' bruxing behavior over the previous week, and were primarily open-ended in nature. While limiting the breadth of responses to one week, it also allowed subjects to give answers that were difficult to interpret and compare.

At least one question on the self-report questionnaire seems to have been inappropriate. Only Subject C reported any validation of the bruxing behavior by a friend or spouse (and in a direction suggesting there was less bruxing after the treatment). This question has been used extensively in the self-report section of previous research. It seems with only one subject of four responding to it,

that perhaps the continued reliance upon such validation should be vigorously questioned. It also seems reasonable that the continued use and refinement of the Bruxcore device could serve as a more viable alternative.

All subjects reported a reduction in the duration and severity of headache and facial pain after the study. This was very subjective but seems to indicate again the presence of a diurnal element which seems to be very amenable to biofeedback training. Perhaps this conscious aspect of bruxism is simply the subject's awareness of the pain and tension created by the nocturnal habit, but even if that is true, the application of voluntary relaxation seems to provide relief from symptomatology.

In summary, Subject A's EMG levels went down while his bruxing level remained the same. His periodontal observation reflected a downward trend in tooth mobility, with two teeth significantly more stable after the treatment. His self-report reflected a reduction in severity and duration of headaches and myofacial pain.

Subject B's EMG readings decreased significantly while her Bruxcore figures did not change. Her periodontal observation reflected roughly the same tooth mobility pattern pre- and post-study, and her self-reports were very similar before and after the study.

Subject C's EMG readings reflected a decreasing trend, as did his Bruxcore figures. His periodontal observation demonstrated a reduction in tooth mobility patterns, reduced pain upon palpation in the masseter muscle, and increased mouth-opening ability. Subject C's self-report reflected decreased validation of the behavior and decreased severity

and duration of facial pain. In fact, he reported a total absence of headache or facial pain after the treatment.

Subject D's EMG levels were found to be decreasing, but her Bruxcore figures reflected no such trend. Her periodontal observations indicated a significant reduction in tooth mobility patterns while the balance of clinical criteria remained the same. Subject D's self-report indicated a decreased number of times over the previous week that the behavior was noticed, and decreased severity and duration of headache and facial pain. In fact, she reported during the middle of the second week of treatment that she had become headache-free for the first time in several years. The absence of pain was maintained throughout the remainder of the study.

Limitations

Perhaps the greatest general limitation to this study is the small number of subjects. Although this was intended to be a pilot study, generalizations from the sample of four subjects are to be considered tenuous at best. One of the reasons for such a small subject sample was the financial cost of producing and scoring the Bruxcore device. The cost of this procedure was approximately $4.50 per Bruxcore. It was this factor, along with the 80 half-hour sessions of baseline, treatment, and follow-up data collection, that placed constraints on the scope of this study.

Another general limitation was that the experimenter could never be sure that the subjects actually practiced the learned relaxation response either during the day or just before going to bed as they

were requested to do. Every time the subjects came to the office they were questioned about their practice habits. They were encouraged to develop reminders to relax, word formulas to call their attention to the state of their masseter muscles. However, with a study of this type, there remains no way of forcing subjects to practice; hence the data are subject to yet another important source of potential variance.

In the analysis of the data an ordinal-type statistic was used rather than an integral statistic. This limited the usefulness of the results since fine discriminations between subjects and within subjects from baseline through follow-up were lost. Increasing the sample size and using a more traditional group experimental design might make the use of an integral statistic more feasible. It would seem that such a statistic would be preferable when dealing with a phenomenon like bruxism, which varies in severity between subjects and within each subject from observation to observation.

A final limitation to the conclusions drawn from available data is that only biofeedback was used as a treatment modality. Forgione (1974) and Rugh (1978) have reported findings with regard to the use of biofeedback in conjunction with such practices as progressive relaxation and massed negative practice. The current study was intended to isolate the potential treatment effects of biofeedback without adjunctive treatment modalities. Perhaps some combination of counseling or psychotherapy and biofeedback training could provide the subjects with a more integrated concept of their bruxing habit, and hence more effective voluntary control over it.

Implications

The main purpose of this study was to act as a pilot for future research. To that end it has provided some interesting implications. First, it seems that people who brux at night may also clench during the day. All four subjects reported some awareness of a daytime habit, as well as reduction of pain experienced in the daytime after learning the relaxation. Subject A, in fact, evidenced little nocturnal bruxing throughout the study but experienced increased tooth stability, reduction in severity and frequency of headache and facial pain, and reported catching himself clenching while tending to his classroom duties. Subject A volunteered to wear the Bruxcore during the day to monitor his clenching, but since it was beyond the scope of this study he did not do so. It seems this may well be the area of most promise for future research. This may also lead to the use of biofeedback training in the study and treatment of TMJ problems and myofacial pain dysfunction syndrome, since there is thought to be a strong daytime clenching component inherent in each.

A daytime treatment (i.e., learning to voluntarily relax the muscles of mastication) may have limited use in treating a nighttime problem since, if the problem has roots in the subconscious, one might expect a consciously learned response to be ineffective in its treatment.

Future research in treating the daytime component of bruxism should include refinements of self-reports and expansions of the periodontal observations. More baseline data points with regard to

the Bruxcore may act to reduce some of the normally high variances among subjects and allow for greater generalization of results.

Subjects B and D, both females and both students, experienced extremely low Bruxcore readings during the same week of treatment. Anecdotal notes reveal this was the week following mid-term examinations. This may point to a stress factor in bruxism which, while not being at all clear to the experimenter, most certainly has implications for future phenomenological research in the area.

In terms of the implications of this study for current and future treatment modalities, it seems that biofeedback training does have an impact on subject learning, and that this learning was basically the same for all subjects. It also was demonstrated to be a learned response with some longevity, a point often disputed in current biofeedback-related research.

There can be little doubt that when only one of four subjects significantly reduces his bruxing habit as a result of biofeedback training alone, there is no call for abandonment of current dental practices in favor of such a treatment. However, there are strong indications that biofeedback works to alleviate many of the symptoms caused by bruxing and clenching as well as providing a tool for the subject to use in the active reduction of the daytime component of the problem. This alone seems enough to warrant the continued use of biofeedback-assisted relaxation training in a supportive or adjunctive role with current dental treatments.

APPENDICES

APPENDIX A

SUBJECT CONSENT FORM FOR BRUXISM STUDY

APPENDIX A

I understand that participation in this study will involve two visits to Dr. Kenneth Wisser, D.M.D., for the purpose of clinical observation and the construction of one impression of my upper dentition at no charge to me. I will then be asked to wear a Bruxcore night guard for four consecutive nights (Monday, Tuesday, Wednesday, and Thursday) for up to 12 weeks and to visit Mr. Cornellier's office on Friday of every week to deliver and pick up these bite plates. At that time electromyographic (EMG) electrodes will be placed on my cheek for monitoring purposes only. From three to five weeks into this process I will be asked to come to Mr. Cornellier's office for a 30-minute biofeedback training session every Monday, Tuesday, Thursday, and Friday for three consecutive weeks at no cost to me. I further understand that my confidentiality is guaranteed through this process and that at any time I may remove myself from further study.

I have freely given my consent to participate in this study being conducted by Vincent Cornellier under the supervision of Dr. William C. Hinds. I have not been offered nor do I expect to receive any remuneration to my participation in this research.

_____
Signature of participant

_____
Date

_____
Signature of witness

_____
Date

APPENDIX B

SAMPLE DENTAL CHART

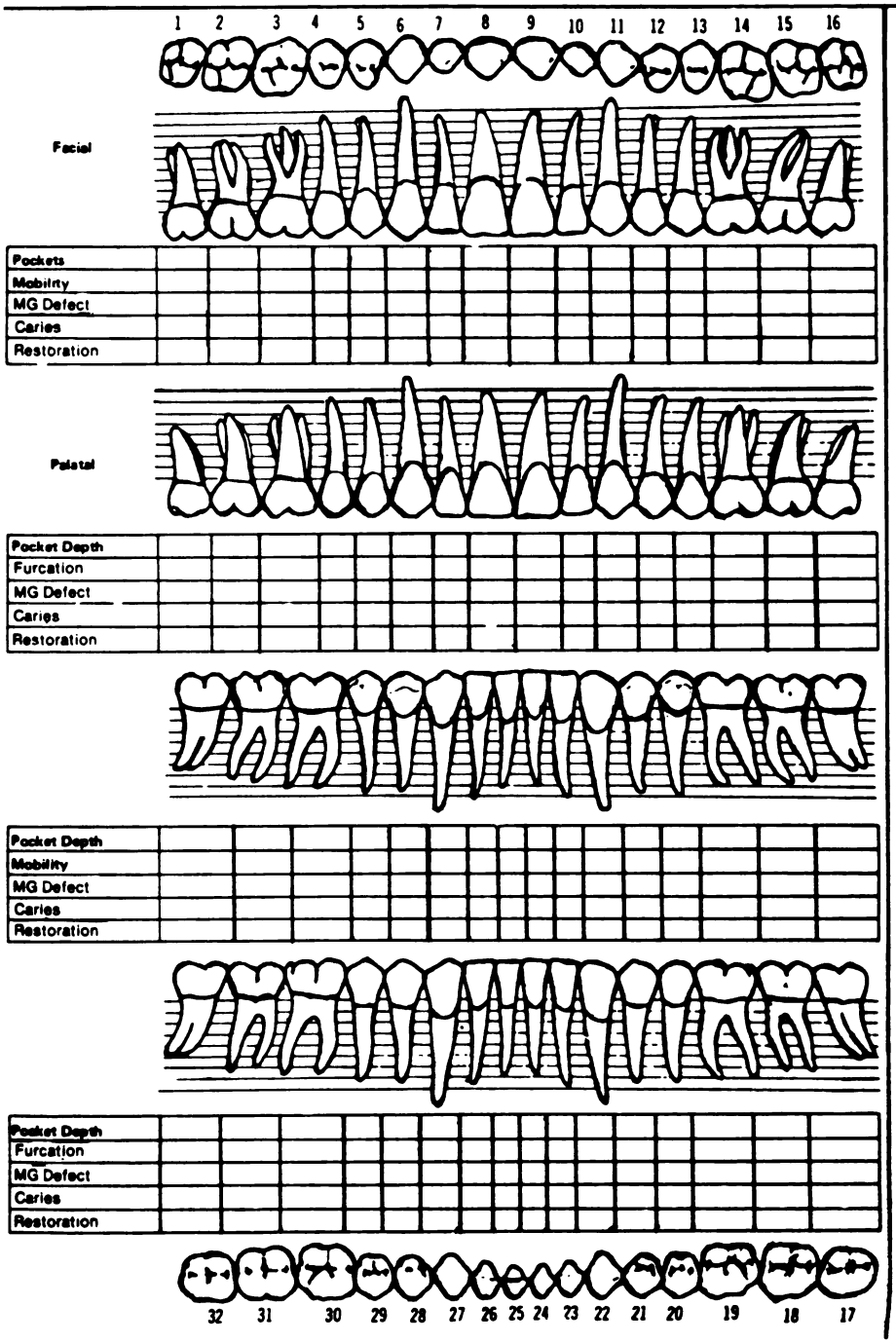
APPENDIX B

NAME

HOME ADDRESS: Street City Zip Phone

BUSINESS ADDRESS: Street City Zip Phone

REFERRED BY:



CASE NOTES:

APPENDIX C

SELF-REPORT QUESTIONNAIRE

APPENDIX C

Name _____ Date _____

1. How often over the last week have you noticed bruxing behavior?

2. How often over the last week have you been informed by a spouse or friend of your bruxing behavior?

3. Have you had facial pain or headaches over the past week? _____
If so,
   a. Where was the pain located?
   b. What time of day or night was the pain noticed?
   c. Please rate the intensity of the pain on a scale (with 1 being no pain and 10 being what you consider unbearable).
      _____
   d. How long, in hours, did the pain last?

4. Do you experience pain when chewing food or opening your mouth wide?

APPENDIX D

EMG MICROVOLT CONVERSION CHART

APPENDIX D

This table is based on research being done at ASI with computerized power frequency spectrum analysis of the EMG recorded by surface electrodes.

Method of EMG Amplitude Measurement and Calibration	Bandpass 100-200Hz.	100-500Hz.	100-1000Hz.	20-500Hz.	20-1000Hz.
Integral Averaged	1	1.56	1.67	2.66	2.73
Peak to Peak	3.14	4.90	5.25	8.36	8.59

To compare readings taken from the Autogen 1100 (100-200Hz. bandpass) with readings taken from another EMG instrument, select from the above chart the method of EMG amplitude measurement and bandpass which most closely match the one used on the other EMG instrument. Multiply the reading from the Autogen 1100 by the value listed on the table for the desired bandpass and method of amplitude measurement.

There are two limiting factors on the accuracy of this table: filter attenuation of out-of-bandpass EMG activity and artifacts, and the system noise level. For example, the BIFS Model B-1, used in some published research, has a bandwidth of 95-1000Hz. which closely matches that of the 100-1000Hz. in the table. This unit is calibrated for peak-to-peak readings, giving a factor of 5.25. However, the amplified input noise of the model B-1 would limit its usefulness at approximately 3uV peak-to-peak and below. This error is difficult to assess due to the fact that complete instrument specifications are usually not available, but an EMG instrument cannot accurately measure EMG signals equal to or less than its amplifier input noise level.

APPENDIX E

ASI INTEGRATOR (5100)

APPENDIX E

Specifications:

1. Functions: Time Integration, Percent Time Computation, Event Counter

2. Computer Time Intervals: 1 second, 15 seconds, 30 seconds, 1 minute, 2 minutes, 5 minutes, 10 minutes, 20 minutes, 40 minutes

3. Rest Time Intervals: 0.1 second, 5 seconds, 15 seconds, 30 seconds, 1 minute, 2 minutes, 5 minutes

4. Accuracy: ∫2, ∫3, ∫4: ±0.1% F.S. plus ±0.2% reading
   ∫1 (signal conditioning inputs): maximum: ±1% F.S.
   typical: ±0.5% F.S.
   %1,%2,%3: ±0.1% F.S.
   Count: ±1 count

5. Inputs: ∫1A: ÷10 absolute value, ±40 VDC, Zin = 1 Megohm
   ∫1B: x1 absolute value, ±4 VDC, Zin = 100K ohms
   ∫1C: ÷2 absolute value, ±8 VDC, Zin = 200K ohms
   ∫1D: AC x10 absolute value, ±0.4 VDC, Zin = 10K ohms, 1uf
   ∫1B-C: x1 differential, ±4VDC, Zin = 100K ohms each side
   ∫2, ∫3, ∫4: x1, ±4VDC, Zin > 100 Megohms
   %1, %2, %3: +3 VDC, Zin > 100 Megohms
   Count: +3 VDC, Zin > 100 Megohms

6. Outputs: Scotchflex 3461-0000 (or equivalent) 20 conductor double-readout P.C. edge connector. For pin connections, see Section IV-E.

7. Physical: Power: two 9-volt batteries (NEDA 1603)
   Size: 4-1/4" high x 17-3/4" side x 8" deep
   Weight: 14 lbs.

APPENDIX F

BRUXCORE LOG

APPENDIX F

DATE	DAY	TIME IN	TIME OUT

REFERENCES

REFERENCES

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