A MULTIFACETED TREATMENT FOR MYOFASCIAL–PAIN DYSFUNCTION:
A COMPARISON OF TREATMENT COMPONENTS

DISSERTATION

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By

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This study compared the clinical effectiveness of cognitively oriented stress-coping training with and without biofeedback training to biofeedback training only in the treatment of myofascial pain dysfunction (MPDS). These groups were also compared to a fourth treatment consisting of pseudo-biofeedback plus stress-coping training. Subjects were 32 adults suffering from MPDS who had failed to previously profit from other treatments. Subjects averaged 33.5 years of age and 58.7 months of myofascial pain.

Treatment consisted of 10 individual sessions over a five-week period. Stress-coping training was designed to teach subjects to monitor their cognitive responses to stress-eliciting situations and to learn cognitive coping skills. Biofeedback training was designed to provide relaxation skills that would enable subjects to reduce masseter muscle tension (EMG). Subjects receiving pseudo-biofeedback training did not receive veridical feedback training.

Prior to and following the conclusion of treatment, resting levels of masseter EMG were obtained from each subject and the psychosomatic checklist and Beck Depression
Inventory were completed. Subjects also provided nine weeks of self-monitored recordings of hourly myofascial pain. A clinical evaluation of each subject's MPDS signs and symptoms was conducted two-weeks prior to and two-weeks following the conclusion of treatment. This evaluation provided an objective assessment of each subject's MPDS symptoms and was utilized to rate the outcome of treatment.

Results indicated that all four groups led to significant reductions in selective symptoms of MPDS. Though numerically large differences in outcome existed between the biofeedback only and other treatment groups, these differences rarely achieved statistical significance. Consequently, there was little evidence to unequivocally demonstrate that any one treatment was more efficacious than another. Only subjects receiving veridical biofeedback training showed significant reductions in masseter EMG activity. Reductions in masseter EMG activity were not correlated with self-reported symptom improvement.

Overall, the findings provide strong support for the use of stress-coping treatment strategies in the treatment of MPDS. The findings also suggest that biofeedback/relaxation training is not an indispensable or even necessary component of treatment since stress-coping training that did not rely on any feedback training was as effective as those that did.
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A MULTIFACETED TREATMENT FOR MYOFASCIAL-PAIN DYSFUNCTION:
A COMPARISON OF TREATMENT COMPONENTS

Myofascial pain-dysfunction syndrome (MPDS) is a chronic, troubling, and not uncommon syndrome (Wepman, 1980). MPDS is defined as a functional disorder of the masticatory system and is presently believed to be the equivalent of a muscle contraction headache in the muscles of mastication (Scott & Gregg, 1980). Incidence of MPDS has been estimated to occur in about 20% of the population (Carlsson & Gale, 1977), although in this 20%, the disorder is often not severe enough to seek clinical treatment (Agerberg & Carlsson, 1972). The average age of patients in treatment is approximately 30 years, although almost all studies report wide ranges of ages of their patients in treatment from late teens to the sixth decade (Kaye, Moran, & Fritz, 1979; Heloe & Heloe, 1975). The majority of patients seeking treatment for MPDS are female. In a recent review Kay et al. (1979) reported that 80.2% of MPDS patients were female.

There has been much confusion regarding the myofascial pain-dysfunction syndrome as it has generally been defined or identified in terms of a set of symptoms rather than through its etiology or by specifying the exact diagnosis (Rugh & Solberg, 1976). Historically, the symptoms have covered a broad spectrum, with impaired hearing, sensations of burning in
the tongue and throat, stuffiness, earache, dryness of the mouth, dizziness, tinnitus (Costen, 1934), mandible hypomobility (Schultz, 1937), pain in the temporomandibular joint and associated muscles, joint noises, and altered muscle function (Schwartz, 1959) have all been reported. Over a period of time, the disorder has been more narrowly defined by reducing the number of symptoms included in the definition. Symptoms have been included or excluded usually on the basis of certain etiological assumptions. Schwartz (1958), for example, excluded many of Costen's (1934) original symptoms on the basis of his conviction that the disorder was of nonorganic origin and basically a functional problem of the masticatory musculature. Although Schwartz (1958) found no single cause for the disorder, he did emphasize that "reacting to stress seems to be more important than any organic problem (e.g., malocclusion) that the patient may have" (p. 210). Schwartz's work had a profound influence on the thinking of many individuals interested in temporomandibular joint problems and focused research on the role of stress, muscle tension, and emotional states and their relationship to the symptoms of the temporomandibular joint syndrome.

Laskin (1969) introduced the term myofascial pain-dysfunction syndrome to identify a subgroup of temporomandibular joint patients which he felt comprised the bulk of patients having pain and dysfunction. Laskin (1979) proposed a primarily
psychological theory of the etiology of MPDS which emphasized the role of stress, muscle tension and spasm in the development of the disorder.

**MPDS: Signs and Symptoms**

Presently, there is some agreement about the definitional signs and symptoms of MPDS (Laskin, 1969; Bell, 1969; DeBoever, 1973; Kay et al., 1979; Scott & Gregg, 1980). The most common finding and complaint which brings patients for treatment is pain, often of a unilateral origin (Laskin, 1969; Kay et al., 1979). Though interpretation of pain is very subjective and varies with the individual patient (DeBoever, 1973) it is most often described as a dull ache felt in the ear or preauricular area, that may radiate to the angle of the mandible, the temporal area, or the lateral cervical region (Laskin, 1969; Kaye et al., 1979). The pain can be relatively constant, but more often is reported as worse on arising in the morning, or else as relatively mild in the morning but gradually worsening as the day progresses. It frequently also is exacerbated at mealtime (Laskin, 1969; Scott & Gregg, 1980).

The next most common finding is muscle tenderness (Laskin, 1969; Kaye et al., 1979). Although this symptom is not usually reported by the patient, it is easily determined via digital palpation of the masticatory muscles by the examiner (Kaye et al., 1979). The most frequent areas of tenderness are the lateral pterygoid muscles and the region distal and superior to the maxillary tuberosity (Kaye et al., 1979; Laskin, 1969).
It is presumed that a muscle tender to palpation is under abnormal tension and is in some degree of spasm (Laskin, 1969).

The third most common symptom is a clicking or popping noise in the temporomandibular joint. Patients having this symptom alone, however, are not included in the category of myofascial pain-dysfunction syndrome (Laskin, 1969; Rugh & Solberg, 1976).

Limitation of jaw function is the fourth cardinal symptom of MPDS (Laskin, 1969; Kaye et al., 1979). This symptom may be characterized either by an inability to open as wide as usual or by deviation of the mandible on opening.

Besides having one or more of the four cardinal symptoms of pain, tenderness, joint sounds, and limitation of opening, the patient considered to have the uncomplicated myofascial pain-dysfunction syndrome would also have the following negative characteristics: (a) absence of radiographic evidence of deterioration of the temporomandibular joint, and (b) lack of tenderness in the temporomandibular joint when palpated via the external auditory meatus (Bell, 1969; Laskin, 1969). The significance of these negative characteristics in establishing the final diagnosis lies in their indication that the primary site of the problem is in the masticatory musculature of the joint.

Although the symptoms discussed above are generally recognized as indicating or defining MPDS, there is considerable variability in the set of symptoms reported by any single
patient (Kaye et al., 1979). For example, a significant percentage of the patient population has been reported to suffer from frequent headaches (Dawson, 1974; Kaye et al., 1979). Since MPDS, similar to headaches, is attributed to increased muscle tension, the finding of a high incidence of headaches in this population is not surprising. It has been argued that many chronic "headaches" may well be referred pain from the muscles of mastication and as such symptomatic of MPDS (Butler, Folke, & Brandt, 1975; Alderman, 1976).

Head and neck swelling is another symptom which has been noted in a significant percentage of the MPDS patient population (Kaye et al., 1979). Head and neck swelling may be caused by, among other things, hypertrophic muscle groups (Mahan, 1976).

Occlusal habits such as the clenching and grinding of the teeth (i.e., bruxism) are also commonly noted in MPDS patients (Laskin, 1969; Kay et al., 1979). Such habits place the head and neck musculature in a state of hypertension (Gelb & Tarte, 1975) and have been hypothesized as the hidden factor that initiates the myofascial pain-dysfunction syndrome (Laskin, 1969; Mahan, 1976).

In summary, myofascial pain-dysfunction syndrome, implying muscle and fascial pain, is a more recent and accurate term. Such a differential diagnosis indicates that the patient's disorder is a functional problem of the masticatory musculature and specifically eliminates patients with pain due to
joint anomalies or disease (e.g., osteoarthritis). This distinction forms the essential basis for understanding the etiology of the myofascial pain-dysfunction syndrome.

**Etiology of MPDS**

A number of different theories have been proposed to explain the etiology of the collection of signs and symptoms of MPDS. Discrepancies in occlusion, or maxillo-mandibular relationships, or both, formed the basis for most of the early theories of the etiology of the temporomandibular joint syndrome (Laskin, 1969). While originally it was suggested that mandibular overclosure was the prime initiating factor (Costen, 1934; Harris, 1938), more refined theories related to occlusal disharmony have subsequently been developed (Dawson, 1974; Perry, 1969; Ramfjord & Ash, 1971). The occlusal disharmony theory of MPDS is still widely ascribed to by dentists today (Scott & Gregg, 1980). This theory holds that occlusal discrepancies require one or both condyles of the mandible to be held forward from their ideal hinge position. Because the lateral pterygoid muscle is responsible for the continual bracing of the mandible in a forward position, the muscle eventually becomes spastic and painful. This pain causes muscle splinting. The pain, muscle spasm, and splinting of associated muscles produces the limitation and deviation of the mandible which is characteristic of MPDS.

Recently, a primarily psychological theory of the etiology of myofascial pain-dysfunction syndrome has been proposed by
Laskin and his co-workers (1969). This theory arose in part from several observations: (a) the vast majority of the population has occlusal problems that could theoretically cause the syndrome, and (b) these occlusal problems are evenly distributed between men and women. Yet, only a small percentage of the population who has malocclusion actually has these symptoms (Scott & Gregg, 1980), and it is generally believed women are more prone to these problems than men (Kaye et al., 1979). In addition, (c) it occurs in people without teeth, and (d) it occurs in people with ideal occlusion. Advocates of the psychological theory believe that psychological stress per se (without occlusal disharmonies) can cause the muscles of mastication to be hyperactive and hence go into spasm.

Specifically, Laskin (1969) views masticatory muscle spasms as the primary factor responsible for the signs and symptoms of MPDS. Figure 1 depicts Laskin's (1969) proposed scheme for explaining the pathogenesis of the myofascial pain-dysfunction syndrome. As can be seen, muscle spasms can be initiated in one of three ways: muscular overextension, muscular overcontraction or muscular fatigue. Thus the theory admits that adverse mechanical factors, may, indeed, cause some instances of MPDS. However, the theory is primarily psychological in nature because Laskin (1969) hypothesizes the most common cause of muscle spasm to be muscle fatigue produced by involuntary tension-relieving
oral habits such as clenching or the grinding of the teeth (i.e., bruxism). The psychological concept of "response specificity" to stress (Cameron, 1974; Rugh & Solberg, 1976) appears applicable here. That is, individuals have been found to have relatively consistent, unique, physiological response patterns to a variety of stressful situations. For the MPDS patient, the grinding and clenching of teeth in response to stressful life events may serve as both a tension-relieving mechanism as well as a precursor of the syndrome.

TENSION → ORAL HABIT ← "Dental Irritation"

MUSCULAR FATIGUE

Muscular Overextension → MYOSPASM → Muscular Overcontraction

MYOFASCIAL-PAIN-DYSFUNCTION SYNDROME

Contracture → Occlusal Dysharmony → Degen. Arthritis

Altered Chewing Pattern

Figure 1. Etiology of the myofascial pain-dysfunction syndrome. Although there are three means of entry into the syndrome, the arrows indicate the most common pathway (Laskin, 1969).
The development of spasm in one or more of the masticatory muscles, regardless of whether it is caused by fatigue, overcontraction, or overextension, not only leads to pain and limitation, but also may produce a slight change in jaw position so that the teeth do not occlude properly (Laskin, 1969; Bell, 1969). If this abnormal jaw relationship persists, the teeth may then gradually shift to accommodate to the new position. Laskin (1969) reasons that this is why some patients suddenly exhibit occlusal disharmonies when the spasm is relieved and the unbalanced musculature returns the jaw to its original relationship.

In addition to producing possible alterations in occlusion, persistent myospasm also can cause two other organic changes (Laskin, 1969; Bell, 1969), namely: degenerative arthritis and contracture. Thus, MPDS, although originating as a functional problem in most instances, ultimately may lead to organic changes. The condition then becomes self-perpetuating because all of these organic changes result in an altered chewing pattern with attendant reinforcement of the original spasm and pain. Furthermore, the possibility that MPDS may ultimately result in organic changes poses a dilemma for the clinician since it may be difficult to determine clinically whether the patient belongs in the category of the myofascial pain-dysfunction syndrome or if the pathologic condition is primary (Bell, 1969; Laskin, 1969).
Laskin's (1969) theory, which is currently widely accepted (Scott & Gregg, 1980), offers several advantages over previous theories. On the one hand, not only does it adequately explain the origin of various signs and symptoms, it also shows how occlusal disharmonies can arise in a functioning dentation as a result rather than as a cause of the problem. Furthermore, the theory also explains how diverse etiologic factors can produce like symptoms. Similarly, it offers a logical explanation of how different forms of therapy may be successful in what symptomatically appears to be the same condition.

Both the older occlusal disharmony theory and the more recent psychological theory share the assumption that pain is due to muscle hyperactivity. The hyperactivity is believed to produce pain by the following course of events (Scott & Gregg, 1980). The muscles consume oxygen faster than normally functioning muscles. This causes a buildup of lactic acid, potassium and histamines in the intermuscular spaces and this buildup irritates the afferent nerve fibers in the muscle blood vessel walls. This is perceived by the individual as a dull, radiating, deep pain (Harris, 1974).

In addition, other changes in the central nervous system may occur after patients have been experiencing pain for a prolonged period of time. Patients who are chronically in pain are almost invariably depressed (Sternbach, 1974). The depressive states have been linked with lower levels of
at least two monoamine neurotransmitters, e.g., norepinephrine and serotonin. Lower levels of serotonin have been associated with increased pain sensitivity and/or reactivity in humans (Messing & Lytle, 1977) as well as alterations of normal sleep patterns (Thomas, Tiber, & Schireson, 1973). In brief, chronic MPDS patients may exhibit a number of central nervous system changes which may make any successful intervention more difficult.

**Evidence for Psychological Factors**

Since Laskin's (1969) theory of the etiology of MPDS has served as the basis for the increased usage of psychological treatment strategies (e.g., biofeedback, relaxation), the evidence for this theory will be examined more closely. Research into the psychological factors of MPDS have utilized a variety of etiological models in an attempt to specify the exact nature of the psychological factors involved. For example, psychoanalysts (Moulton, 1955; Lefer, 1966; 1971) have viewed MPDS as a "conversion reaction manifested in the oral cavity" usually resulting from emotional conflicts over unconscious needs for dependency, aggressiveness, or depression. However, as Rugh and Solberg (1976) argued, such theories are abstract, unverifiable and add little to the development of a reliable testing method to determine the etiology of MPDS.

Considerable effort has also been directed at attempts to identify specific personality traits or characteristics
which are correlated with MPDS patients. A wide variety of personality characteristics have been reported by the various investigators (Lupton, 1969; Solberg, 1972; Gross & Vacchiano, 1973; DeBoever, 1973; Shipman, 1973). MPDS patients have been found by different investigators to be: dependent, narcissistic, obsessive, sadistic, rigid, domineering, autocratic, perfectionistic, hypernormal, responsible, overgenerous, aggressive, imaginative, neurotic, emotionally unstable, hypochondriacal, and depressed. Although many of the personality characteristics reported are not incompatible or mutually exclusive, there is little evidence of consistency in the results (Rugh & Solberg, 1976). Considering the diversity of results reported, it is difficult to maintain that patients with MPDS represent a homogenous group with respect to personality characteristics. A similar conclusion has been reached regarding other psychosomatic disorders such as low back pain and tension headaches (Buck & Hobbs, 1959; Lipowski, 1968; Bandler, 1958). Recently the whole concept of individuals possessing fixed personality characteristics which determine behavioral patterns has been questioned (Mischel, 1968; 1973).

Similarly, research on MPDS patients and their perceptual style (Lupton, 1966) and specific attitudes (Graham, Graham, & Kabler, 1960) has generally been supportive of psychological factors in MPDS, yet has lacked consistency and often suffer from possible alternative explanations (Rugh & Solberg, 1976). In conclusion, the concepts utilized in the above approaches
(i.e., psychoanalytic, personality trait, etc.) have not been developed in such a manner to lend themselves to experimental evaluation and employment in a verifiable etiological theory of MPDS.

Germane to the psychological theory of MPDS (Laskin, 1969) is a body of research on the role and relationships of emotional states (e.g., anxiety, anger), muscle tension, and pain. Recall that Laskin (1969) proposes that in the majority of MPDS cases the etiology is due to stress which provokes muscle tension and/or oral habits such as teeth clenching or grinding. If prolonged, such habits result in muscular fatigue which in turn produces spasm in one or more of the masticatory muscles resulting in pain, limitation of movement and muscle tenderness. The stress, muscular tension (i.e., hyperactivity), and pain theory of MPDS involves three major assumptions (Rugh & Solberg, 1976) which may be examined independently: (a) that emotional states such as anxiety elicit muscle tension, (b) that persistent muscular tension (i.e., hyperactivity) results in pain syndrome, and (c) that this sequence of causal relations leads to symptoms reported by MPDS patients.

Regarding the first assumption, there exists relatively strong experimental evidence that emotional states such as anxiety, fear, and frustration can elicit increased muscular activity (Malmo, 1957; Goldstein, Grinker, Heath, & Shipman, 1964). Furthermore, individuals have been found to have
relatively consistent, unique, physiological response patterns (i.e., response specificity) to a variety of stressful situations (Grinker, 1966). For example, a "muscle responder" will respond repeatedly with tension in the same set of muscles to a wide range of emotional stimuli. Individuals vary with respect to the degree to which they exhibit response specificity, however, the effect has been demonstrated under a wide variety of stimuli (Goldstein et al., 1964) and with several types of subjects (Shipman, Heath, & Oken, 1970). Response specificity does not exclude the possibility of several bodily responses to stress. It merely specifies that the same set of bodily responses will be repeated regardless of the particular stress. Gold, Lipton, Marbach, and Gurion (1975) have found that the MPDS patient was apt to have more than one specific psychophysiological disorder resulting from stress.

Additionally, investigators have found that emotional factors such as anxiety, frustration, and fear frequently elicit increased masseter and temporalis muscular activity (Yemm, 1968, 1969, 1971, 1972; Thomas et al., 1973). Thus investigations leave little doubt that emotional states can elicit generalized muscle tension.

Concerning the second assumption, it appears equally clear that muscular hyperactivity can cause pain. Yemm (1976) concluded in his extensive review of the neurophysiological studies of MPDS that Christensen (1971) has presented "the main experimental support for the clinical contention that
MPDS is caused by muscle hyperactivity" (p. 33). In the study, Christensen (1971) asked 10 healthy subjects to unilaterally grind their teeth for 50 minutes. Following the bruxing, nine of the 10 subjects reported pain similar to myofascial pain. The muscular pain reached a maximum about two hours after the bruxism session and lasted from one to seven days. Similar results were also reported in a subsequent study (Christensen, 1975).

Several problems with Christensen's (1971, 1975) studies limit the conclusions that can be drawn. For one, Christensen (1971, 1975) created pain in most of the muscles of mastication, however, most investigators (e.g., Franks, 1965) have indicated that the muscle tenderness in MPDS is most specific to the lateral pterygoid. Scott and Lundeen (1980) attempted to extend Christensen's model of MPDS by creating an experimental model of MPDS involving the lateral pterygoid. In the study, half of the 30 healthy volunteer subjects were asked to vigorously thrust or protrude their jaw for five minutes. The other half of the subjects were asked to clench lightly on a tongue blade (control group). The experimental group showed a significantly higher pain level than the control group, rating the intensity of their pain between moderate and severe (approximately 5 on a 0-10 scale). Subjects reported that the pain was located in front of the ear, and had a similar, deep, dull quality of a muscle pain. Thus this study also demonstrated that muscle hyperactivity causes pain.
While the above studies indicate that emotional states may elicit muscular tension and that prolonged muscular tension can cause pain, the question remains as to whether this sequence is responsible for the pain reported by MPDS patients. Several lines of evidence summarized below suggest that this third assumption is true.

Several studies (Grieder, 1973; Lupton, 1966; Kaye et al., 1979) have indicated that MPDS patients are frequently found to be bruxists. Grieder (1973), for example, reported that 97% of the MPDS patients examined showed evidence of bruxism. Other evidence indicates that patients with MPDS have greater masseter and temporalis activity during stress than normals (Yemm, 1969, 1971, 1972; Thomas et al., 1973). For example, Yemm (1969) utilized an experimental task which involved subjects pressing buttons in complex ways to correspond to serially illuminated lights. Yemm found that initially normal subjects showed high masseter activity levels, but these high initial levels diminished as the subjects became accustomed to the task (performance did not improve on the brief task). In contrast to adaptation shown by healthy controls, the patients with MPDS showed no adaptation and hence had high levels of muscle activity during the entire task.

Thomas et al., (1973) also compared MPDS patients to healthy controls. They tested their subjects on the following "frustration" inducing task. Subjects were asked to complete a puzzle form board as a supposed measure of intelligence.
The experimenter deliberately interrupted the subject and made derogatory comments about each subject's poor performance. Under these extreme conditions, the MPDS patients showed statistically higher masseter muscle activity, and maintained it for a longer period of time, than the controls. The results of the above studies provide evidence that muscle hyperactivity is a common response in MPDS patients and can be elicited by emotional stimuli accompanying physical effort.

The relationship of emotional stress and muscular tension has also been observed outside the laboratory setting (Solberg & Rugh, 1972; Rugh & Solberg, 1974; Rugh & Solberg, 1975). In a series of studies, MPDS patients were provided with portable electromyographic (EMG) devices which signaled the patient when masseter muscular activity exceeded a pre-set threshold. In one study (Rugh & Solberg, 1974), patients wore the device during their normal daily activity from four to seven days. Rugh and Solberg (1974) reported that patients were surprised to find how frequently they clenched their teeth without being aware of their behavior. Each patient was able to report stressful stimuli which consistently elicited the clenching. Furthermore, several patients reported that they discovered they clenched their teeth while ruminating upon past or upcoming stressful events such as divorce proceedings or school exams.

In an attempt to demonstrate the relationship between stress and bruxism in the natural environment, Rugh and Solberg
(1975) utilized portable EMG recording devices which store masseter muscle activity above 20 microvolts (uV) for the duration that the devices are worn. A group of nocturnal bruxists wore these devices during sleep for periods of up to three months and unilateral masseter activity was recorded. The recordings indicated that bruxism behavior may vary greatly from night to night and is correlated with the previous day's stress level. That is, stressful daytime situations were found to be correlated with high levels of grinding on the following night.

A final study to be reported is a rather unique attempt to demonstrate via biochemical measurement that MPDS patients are under greater emotional stress than a nonpatient group (Evaskus & Laskin, 1972). Urinary concentrations of catecholamines and 16-hydroxy steroids were compared in MPDS patients and a control group. Evaskus and Laskin (1972) reported that MPDS patients had significantly high urinary concentrations of both substances. The authors concluded that this indicated that MPDS patients are under greater emotional stress than normal individuals. However, while it is true that emotional stress increases catecholamine concentration, it is also true that muscular effort increases catecholamine concentration (Frankenhaeuser, 1971). Thus, it may be that the differences in catecholamine concentrations were due to hyperactive masticatory musculature of the MPDS patients. Yet, whether a result of emotional stress or muscle hyperactivity, Evaskus
and Laskin's (1972) data provide strong evidence for an emotional muscular tension theory.

In summary, the above evidence indicates that emotional factors may play a significant role in the etiology of MPDS. MPDS patients react to emotional states such as anxiety, frustration, fear, and anger with increased activity in the masticatory muscles. Prolonged tension in these muscles can produce pain symptoms which are commonly found in MPDS patients.

Treatment of MPDS

One of the most important aspects of the successful management of any disorder is the understanding of its etiology so that a rational treatment plan can be formulated. Traditionally, dentists have focused on organic factors in treating MPDS and a variety of treatment methods have been employed: the occlusion has been adjusted or the mandible repositioned mechanically; biteguards have been constructed for the patient to wear at night; patients have undergone physiotherapy consisting of moist heat and muscle exercise; the muscles have been injected with local anesthetics or sprayed with ethyl chloride; or muscle relaxants and tranquilizers have been prescribed. Various authors (e.g., Greene & Laskin, 1974; Dawson, 1974) have reported high degrees of success utilizing these treatment strategies, however, their success appears related to the period of the disorder when treatment is initiated. Carlsson and Gale (1977) reported
that it is not uncommon to be able to have a 50% cure rate when MPDS patients are treated in the initial stages (i.e., first 3 months) of the disorder. Carlsson, Gale, and Ohman (1975) claimed that the effective relief of pain early in the history of the myofascial pain disorder is most likely the result of spontaneous remission. Laskin and his co-workers (Goodman, Green, & Laskin, 1976; Laskin & Greene, 1972) have provided evidence that improvement in the early stages of MPDS may be due to nonspecific treatment effects. For example, dentists commonly perform occlusal equilibration on MPDS patients in an attempt to eliminate altered proprioceptive feedback due to faulty occlusion and hence reduce muscle hyperactivity. Goodman et al. (1976) have shown that "mock" equilibration involving an elaborate rationale, the construction of dental casts on the first dental visit, and grinding on the non-occluding surfaces of the teeth by a slow-speed handpiece on two subsequent visits, produced total or nearly total remission of the pain symptoms in 16 of 25 (64%) of the patients. Placebo effects are potent factors in any treatment of pain (Beecher, 1959; Evans, 1974) and providing the patient with a mental awareness of the problem along with the assurance and understanding of a doctor-patient relationship can do much to initially reduce and sometimes eliminate the symptoms of MPDS. Therefore, the effectiveness of a treatment method for MPDS should not be evaluated in patients whose pain is of recent origin but
rather in patients who have been refractory to conservative treatments (Carlsson & Gale, 1977).

The traditional methods of treating MPDS such as occlusal adjustment and biteguards have often proven to be ineffective or prompt relapse has occurred. Laskin (1969) has suggested that the primary reason for these treatment failures is that clinicians have failed to recognize that the initiating factors for the syndrome are emotional rather than physical. Consequently, following treatment, the patient continues to clench and grind his teeth in response to psychological stress. Only after continued failures to respond to treatment would a patient finally be referred for psychological treatment (Lupton, 1966; Atterbury, 1980). Historically, the mode of psychotherapy attempted with these patients was based on analytically derived models (Moulton, 1968). As stress has come to be accepted as a major etiological component of MPDS, attention has focused on the use of EMG biofeedback-assisted relaxation training as a treatment (Carlsson et al., 1975; Carlsson & Gale, 1977; Berry & Wilmont, 1977; Gessel, 1975; Dohrmann & Laskin, 1978).

An early application of biofeedback to temporomandibular joint function was conducted by Budzynski and Stoyva (1973) who showed that normal healthy volunteers could readily relax their masseter muscles significantly below the levels obtained by untrained controls. Budzynski and Stoyva compared two types of electromyographic feedback (auditory versus digital).
to two control groups (either an irrelevant feedback of constant low frequency tone or a no feedback condition).

To ensure high motivation to relax among all groups, participants were told that they would be paid according to their performance, relative to other participants in the group.

Subjects in both the feedback conditions significantly reduced the electromyographic levels below the two control groups. The standard deviations of electromyograph recordings in the two feedback conditions were markedly smaller than the two control conditions. The finding suggests that among the normal volunteer subjects, the feedback treatments when compared to simple instructions to relax, were uniformly helpful in producing more profound relaxation with less variable results.

Since this initial analogue study, there has been increasing interest in applying biofeedback to the treatment of MPDS. Carlsson et al., (1975) described the successful treatment of a 21 year old female patient who had previously undergone numerous traditional treatments (e.g., equilibrium of the dentation, biteguard, injection of local anesthetics) without relief from pain. The researchers reported that the strategy of biofeedback training was to initially make the patient aware of the level of tension in the masseter muscle and then to teach her to reduce it. Following 18 biofeedback sessions, the patient was pain free and maintained this through a 6-month follow-up assessment.
In an extension of this case report, Carlsson and Gale (1977) reported on the treatment of 11 patients suffering from chronic (occurring for an average of nearly seven years) myofascial pain. Conventional occlusal and other dental treatments had previously failed for all these patients. During an average of eight treatment sessions over an interval of about two months, the patients were provided with visual feedback of surface EMG levels recorded from their masseter muscles. Additionally, they were provided with relaxation instructions and given practice in reducing muscle tension levels with and without the feedback.

The results indicated that most of the patients were able to reduce masseter EMG levels substantially over the course of the training interval. Clinical evaluation found five of the 11 patients to be free of symptoms, while three were rated to be significantly improved, one was rated as slightly better, and two showed no change in reported symptomatology. Follow-up evaluations were conducted over a 4 to 15 month period, with a general maintenance of the classification breakdown reported at the end of the treatment interval.

Aside from the obvious problems in interpreting results from such a weak experimental design, one of the researcher's findings directly challenges the conclusion that the treatment effects noted were due to the biofeedback procedure per se. Carlsson and Gale (1977) found no correlation between success in establishing muscular relaxation during the
treatment session and symptom relief. In fact, the two 
"treatment failures" turned out to be the best relaxers. The 
finding suggests that something other than the presumably 
biofeedback-mediated muscle relaxation was responsible for 
the changes in symptoms noted for nine of the 11 patients. 
Furthermore, the finding highlights the need for researchers 
to develop adequately controlled experimental designs that 
may isolate and evaluate the role of individual treatment 
components and nonspecific factors.

Berry and Wilmont (1977) reported on the treatment of 
35 outpatients suffering from chronic myofascial pain-
dysfunction syndrome. Each patient received, on the average, 
three sessions of audiovisual feedback of masseter EMG 
activity. For all subjects, regardless of the site of the 
pain and dysfunction, feedback reflected the EMG activity of 
only the right masseter muscle. In addition to the biofeed-
back training, 25 of the patients were initially provided 
with plastic occlusal covers for the lower teeth. Berry and 
Wilmont (1977) viewed this treatment as only a temporary, 
though often effective, means of reducing the bruxism and 
clenching that triggers myofascial pain.

The sole evaluation of the treatment effectiveness was 
self-reported changes in symptoms. Relief was reported by 24 
(69%) of the patients in three to five months though two ini-
tially successful cases relapsed, and two patients did not 
remain in treatment. Although no evaluation of the relationship
between the bioelectrical and clinical responses was attempted and no attempt was made to assess maintenance of the clinical gains, the authors contend that these results compare quite favorably to those generally found with purely occlusal treatment. The basis for this conclusion was a survey conducted previously by the authors of 100 cases of MPDS treated with occlusal adjustment. The following record of symptomatic relief was reported:

fourteen percent were relieved in less than 2 months;  
25% were relieved in less than 2-3 months; 21 % were relieved in 4-6 months; 24 % were relieved in 7-12 months (half of these had residual minor symptoms); 16% were relieved in more than 12 months (although this group included the 'chronic relapsers') (Berry & Wilmont, 1977, p. 259).

Thus, biofeedback therapy, when evaluated by an anecdotal case design, appears to be a more cost-efficient treatment technique for MPDS than traditional occlusal therapy. However, several issues cloud the interpretation of the results. For one, the direct analysis of biofeedback training per se is not possible since the treatment was confounded by the use of biteguards. Secondly, the authors attributed relief from pain that occurred months after training to biofeedback even though it is well documented that MPDS patients may experience periods of remission (Carlsson et al., 1973).
Dohrmann and Laskin (1978) recently reported the first controlled evaluation of biofeedback therapy for myofascial pain-dysfunction syndrome. They randomly assigned 24 outpatients to one of two groups: group 1 (16 patients) received auditory feedback of surface masseter EMG activity with gradual shaping to reduce EMG levels; group 2 (8 patients) sat quietly for each session as their masseter EMG levels were monitored. Patients in group 2 were not told about biofeedback but rather that "the jaw muscles involved would be exposed to a low-grade electrical current that would block the muscles' activity and thereby cause relaxation" (p. 657). Two 30-minute sessions each week were held for patients in both groups and all patients were provided with instructions for twice-daily 20-minute home relaxation sessions.

Group 1 patients showed a significant decrease in mean EMG levels pre- to posttreatment (6.30 uV to 3.52 uV); group 2 patients showed no significant decreases in EMG levels across the sessions. Self-reported "pain values" dropped by more than 80% over the course of treatment for group 1 patients, with a 50% reduction in pain values noted for group 2 patients. Group 1 patients showed a significant increase in jaw opening without discomfort (28.5 mm to 36 mm, pre-to-post) while group 2 patients did not significantly improve on this measure. Similar results were found for changes in masticatory muscle tenderness and joint sounds during jaw movement. Both self- and rater-examinations of overall treatment success revealed
the following distribution for group 1: 10 patients (62.5%) were judged completely successful, six patients (37.5%) were rated partially successful, and no treatment failures were noted. Group 2 subjects self-rated more favorably (3 complete, 5 partial successes) than did the examiner (2 complete, 2 partial successes, and 4 treatment failures). At the end of treatment, five of the group 2 patients required additional treatment (e.g., Valium, bite plates, psychological counseling). Although only one of the group 1 patients required additional treatment immediately following biofeedback therapy, three more patients in that group needed additional treatment by the 12-month follow-up interview. Thus, over the course of the entire treatment and follow-up period, 12 (75%) of the biofeedback patients, and only 2 (25%) of the control patients, needed no additional treatment for their MPDS.

While the design and execution of this experiment are perhaps the best in this area of biofeedback application, several issues again cloud the interpretation of the results. One is the obvious problem of differential rational credibility (Kazdin & Wilcoxon, 1976). It is likely that some group 2 patients implicitly questioned the validity of their electrical stimulation treatment and differences in patient belief in treatment efficacy may confound meaningful comparisons between groups. Second, the design of the study does not permit a direct analysis of the contribution of biofeedback therapy per se since the treatment was confounded with home
relaxation instructions. Finally, a more crucial issue in the evaluation of outcome concerns the relationship between the bioelectric response that is being modified and the clinical relief of symptoms. Dohrmann and Laskin (1978) reported a large difference in the baseline EMG levels between the two groups (6.31 ± 3.28 uV for group 1; 11.7 ± 8.27 uV for group 2). Although this difference was not statistically significant (due to the large variability in group 2), group 2 patients still averaged nearly twice as great an initial EMG level as group 1 patients. While group 2 did not show a statistically significant decrease in EMG levels over the treatment interval (again because of the great variability in scores), these patients showed a proportionately greater decrease in EMG level than group 1 patients. Consequently, the study is unable to demonstrate whether therapeutic benefits are due to changes in the patients' perceptions, or whether the changes are due to alterations in muscular activity.

In summary, the use of biofeedback therapy for the treatment of MPDS may only be called promising at this time. The majority of the studies are in the pre-experimental stage of research design and what little controlled research has been done (Dohrmann & Laskin, 1978; Stenn, Mothersil, & Brooke, 1979) does not thoroughly evaluate many important issues of nonspecific influence and component contribution. Close scrutiny of the research also reveals several methodological
inadequancies that should be addressed by future researchers.

One inadequacy in much of the research is the failure to include multiple dependent measures, i.e., measures of all of the myofascial pain signs and symptoms: pain, jaw limitation, joint sounds, and muscle tenderness upon palpation (Scott & Gregg, 1980). Most of the studies have only evaluated the patients for self-reported pain occurring in the natural environment. Green and Laskin (1973) reported that some patients admitted reduced pain or even the complete remission of pain and regarded themselves as "significantly" improved even though they still had marked limitation of the motion of the mandible and obvious joint sounds. Thus, it does not appear that the remission of one symptom (usually pain), is highly correlated with the remission of other symptoms. More recent research (e.g., Dohrmann & Laskin, 1978) have found a corelational relationship between the remission of pain and the remission of other signs and symptoms of MPDS. Future research should continue to investigate this finding further.

Another methodological inadequacy in the research studies is the failure to assess the patients' perceptions of the treatment credibility and expectancies for change. Recent publications have seriously questioned the adequacy of comparative treatment designs which do not account for variations in patient expectancy and credibility of treatment
(Kazdin & Wilcoxon, 1976). To this author's knowledge, no published study on the treatment of MPDS has even attempted to assess the effects of varying cognitions among subjects in different treatment conditions.

Another criticism of the current research is that it suffers from both "patient and treatment uniformity myths" (Kiesler, 1966, p. 129). That is, little research effort has been directed to identify which type of MPDS patient is a good candidate for biofeedback therapy. For example, Gessel (1975) observed that MPDS patients who were older, more depressed, have had the pains for a relatively longer period, and who may be reinforced for the pain are the least successful with biofeedback/relaxation procedures. Other researchers (e.g., Carlsson and Gale, 1977) have failed to support this contention. The problem, however, is that these evaluations are of a questionable validity since they relied on unsystematic clinical judgments rather than quantitative measurement. Future researchers should continue to address this issue by employing a wide-ranging predictor battery (Blanchard, 1981) that would include psychological, physiological, and structural data on MPDS patients undergoing treatment.

The most crucial problem posed by the current research, however, is the failure to establish a relationship between pain, pain reduction, and muscle tension (EMG). Close inspection of studies conducted by Dohrmann and Laskin (1978) and
Carlsson and Gale (1977) suggests that their patients' improvement was not associated with lowered day-to-day masseter muscle tension. If there is no or low correlation between the bioelectric response that is being modified and the clinical relief of symptoms, the basis for biofeedback treatment may be questioned.

Germane to the above issue is a recent review of the application of biofeedback in the regulation of pain (Turk, Meichenbaum, & Berman, 1979). The authors contended that biofeedback training, which places its greatest emphasis on maladaptive physiological functioning is a too restrictive view of pain. That is, in pain syndromes, consideration must also be given to the patient's coping patterns and life style (Genest & Turk, 1979). Teaching voluntary control of physiological functioning may not be sufficient, since patients not only must control their physiology, but they must be capable of dealing effectively with their environment (Shapiro & Schwartz, 1972).

Biofeedback training for the control of MPDS directly addresses only the maladaptive physiological responses to stressful situations and ignores those psychological factors that initiate and contribute to such responses. Yet, similar to muscle contraction headaches (Bakal, 1975; Dalessio, 1972), a three-stage process is believed to produce MPDS: (a) the individual responds to psychological stress, which (b) may produce prolonged tension/contraction in the muscle of
mastication, and which (c) may subsequently lead to the production of myofascial pain and dysfunction. Thus, Turk et al., (1979) argue that to successfully treat pain syndromes, a therapeutic regimen that addresses the first stage (response to psychological stress) such as self-control strategies (Goldfried & Trier, 1974; Meichenbaum, 1977) or stress-coping training (Holroyd, Andrasik, & Westbrook, 1977) should be combined with treatments that focus on the second stage (maladaptive physiological responding). The failure of biofeedback procedures to specifically address individuals' maladaptive appraisals and behaviors in the natural environment may account for (a) the equivocal data, (b) for the relatively high number of patients who drop out of biofeedback treatment prematurely, as well as (c) the individual differences in the ability to benefit from feedback training (Lazarus, 1977; Turk & Genest, 1979).

A related issue that has yet to be systematically evaluated in the treatment of MPDS is whether interventions aimed at modifying patients' maladaptive physiological responding (i.e., biofeedback/relaxation) are indispensible or even a necessary component of treatment. That is, are therapeutic interventions that focus solely on altering individuals maladaptive cognitive responses that are assumed to mediate the occurrence of prolonged muscle tension in and of themselves an efficient and effective treatment of MPDS. Blanchard (1981) has labeled such approaches to the treatment
of psychophysiological disorders as "preventive" since the overall goal is to
prevent the excessive physiologic arousal to sources of conflict/stress either through reducing or eliminating the conflicts or through very early detection of arousal and prevention of its occurrence (p. 247)
Pioneering work with a "preventive" form of treatment to psychophysiological disorders has been provided by Holroyd and his co-workers (Holroyd & Andrasik, 1978; Holroyd et al., 1977), with a cognitive approach to tension headaches. In one study, Holroyd et al., (1977) used a cognitive control treatment designed to train patients to cope more adaptively with environmental and individual stylistic responses that instigated muscular tension. Specifically, patients were trained to deliberately interrupt the sequence of maladaptive thoughts preceding their emotional responses at the earliest possible point and to engage in cognitive control techniques incompatible with further stress and tension (e.g., cognitive reappraisal, attention deployment, fantasy).

This cognitive control regimen was employed with 10 tension headache patients who were compared to patients who received either biofeedback or no specific treatment. Training consisted of eight biweekly sessions with a 15-week follow-up. At the termination of treatment and at follow-up, only the cognitive control group demonstrated substantial improvement on frequency, duration, and intensity of headaches.
Interestingly, only the biofeedback group demonstrated significant reductions in EMG activity.

Only one study (Stenn et al., 1979) on the treatment of MPDS has attempted to address the above issues. In treating MPDS patients, Stenn et al. (1979) utilized a multifaceted treatment regimen consisting of biofeedback training and cognitive behavior therapy. This regimen was employed with 11 patients who had experienced MPDS for at least one year's duration and had failed to respond to previous traditional treatments. Patients received eight 1-hour weekly sessions. During the first half of each session, all patients were hooked up to EMG masseter electrodes and provided relaxation instructions, but only half of them received feedback. During the second half of each session, each patient received individualized cognitive behavior therapy such as assertiveness training (Smith, 1975), rational-emotive therapy (Ellis & Harper, 1968), and stress inoculation training (Meichenbaum & Cameron, 1974), to help them deal with specific stress-inducing situations. The extent to which each of the above modules was used varied from patient to patient depending upon their specific concerns. In summary, all patients received relaxation training (with the concomitant recording of EMG) and cognitive behavior therapy. The only differential treatment was that six of the subjects received EMG biofeedback while five did not.
Results at the termination of treatment and at a 3-month follow-up showed that subjects in both treatment conditions reported a significant reduction in pain and other symptoms than prior to therapy. However, subjects who were provided the auditory feedback about their masseter muscle tension showed significantly greater reductions in their pain levels and number of symptoms than subjects for whom the feedback was deleted.

Thus, Stenn et al. (1979) provide preliminary evidence that a multifaceted treatment consisting of biofeedback and cognitive behavior therapy is an effective treatment for MPDS. Furthermore, the multifaceted treatment regimen was superior to cognitive behavior therapy without biofeedback. However, several issues warrant the use of caution in interpreting the results. One is that the comparative efficacy of the two treatment regimens was based on a very limited number of patients. Second, the direct analysis of the contribution of biofeedback therapy per se is not possible since the treatment was confounded with relaxation training. Furthermore, it is unclear what biofeedback contributed since results indicated that both groups were equally skilled at reducing masseter EMG levels. Not only does the finding again directly raise the question of the assumed causal relationship between muscle tension and MPDS, but it also suggests the activity of nonspecific treatment effects due to biofeedback training. Stenn et al.'s (1979) failure
to utilize adequate control procedures constitutes, perhaps, the most serious methodological flaw in their study. In explaining their results, Stenn et al. proposed that the superior outcome on the part of patients who received feedback may have been due to the development of an expectation that they had greater control over their muscle tension even though in the actual relaxation sessions both groups of patients had decreased the level to a similar degree. The authors' failure, however, to assess variations in their patient's perceptions of treatment credibility and expectancies for therapeutic change leave their proposition purely speculative.

Summary and Purposes

The use of biofeedback assisted relaxation and other stress management training procedures in the treatment of myofascial pain and dysfunction is only in its infancy at this time. Although results appear promising many important issues of nonspecific influence and component contribution have not been evaluated. A more crucial question revolves around the proposed causal relationship between pain, pain reduction, and muscle tension. Several studies (Stenn et al., 1979; Dohrmann & Laskin, 1978; Carlsson & Gale, 1977) suggest that their patients' improvement was not associated with lowered day to day masseter muscle tension. Future researchers should address themselves to this preliminary finding.
A related issue is whether biofeedback training is a too restrictive approach to the treatment of myofascial pain. That is, should not the primary focus of treatment be on the patient's coping pattern and life style with biofeedback being utilized as an adjunctive technique (Turk & Genest, 1979). Preliminary evidence (Stenn et al., 1979) indicates that such a multifaceted treatment regimen is effective in ameliorating the symptoms of MPDS.

Finally, an additional issue that has yet to be systematically investigated in the treatment of MPDS is whether biofeedback/relaxation training is an indispensible or even necessary component of treatment. That is, is a "preventive" treatment approach (Blanchard, 1981) which aims to (a) teach patients to recognize sources of conflict, or stress, and their concomitant physiologic arousal, and then (b) to learn more adaptive means of coping with them, in and of itself an effective treatment of MPDS. Certainly such a coping skills intervention is cost-effective and more readily available than biofeedback therapy.

The present study was designed to investigate the above issues. A multifaceted treatment program consisting of biofeedback training and cognitively oriented stress-coping training was compared to biofeedback training alone and stress-coping training alone. A fourth treatment group consisting of a biofeedback "placebo" treatment (pseudo-biofeedback) plus stress-coping training was utilized in
order to serve as a more adequate comparison to the multi-
faceted treatment condition. Specifically, the pseudo-
biofeedback plus stress-coping treatment group was designed
to control for the nonspecific factors in biofeedback training
as well as the possible effects due to greater treatment
contact time.

The present study was also designed to minimize the
methodological inadequacies inherent in previous studies on
the efficacy of biofeedback and/or stress-coping approaches
to the treatment of MPDS. Specifically, the methodological
improvements were: (a) a larger sample size; (b) the provi-
sion of a rationale for treatment pretested for equivalence
on ratings of credibility/expectancy; (c) continued assess-
ment of credibility/expectancy during treatment; (d) a
multiple outcome battery utilizing a variety of viewpoints;
(e) a design which allows a more direct analysis of the
contribution of biofeedback training since the pseudo-biofeed-
back condition was not confounded by home relaxation training;
and (f) the use of a variety of objective pretreatment measures
in an attempt to more thoroughly identify variables predictive
of response to treatment.

Method

Subjects

Subjects were obtained by referral from the dental,
periodontal, and oral surgery clinics of the Medical Univer-
sity of South Carolina (M.U.S.C.) Dental School. Subjects
were referred because they had pain symptoms considered to be of temporomandibular joint origin. Potential subjects were contacted by the principal investigator and informed that a myofascial pain management program was being offered at the M.U.S.C. Dental School. They were told why their pain syndrome was viewed as being amenable to the treatment program and that the program was being conducted for research purposes and thus free of cost. Additionally, they were provided a brief description regarding the parameters of involvement in the program (e.g., frequency of individual sessions, maintenance of a pain diary, etc.) if they both desired and qualified to participate. If the individual expressed either a desire to participate or to discuss the program more fully, an interview appointment was scheduled.

Interested participants met individually with one of the co-investigators at the M.U.S.C. Dental Clinic to have the details of the myofascial pain management program explained to him/her and have any questions answered. If the individual expressed a desire to participate, he/she was asked to:

(a) read and sign an informed consent form (Appendices A, B, and C) appropriate to his/her predetermined group assignment;
(b) respond to a demographic questionnaire; (c) undergo an interview and clinical examination of his/her pain syndrome.

Inclusion criteria for participation were: (a) a differential diagnosis of myofascial pain-dysfunction syndrome of at least six months duration which, by the subject's report, had
failed to respond to previous traditional treatments (e.g., physiotherapy, occlusal equilibration, biteguards, tranquilizers). The diagnosis was determined by an interview and clinical examination. Additionally, a radiographic exam was required if the subject's medical history revealed any evidence of arthritis or rheumatism (see signs and symptoms evaluation below): (b) no evidence of physical trauma to the head or neck in the subject's history; (c) age between 18 and 60; (d) apparent absence of severe psychopathology (i.e., psychosis, organic brain damage) as determined from historical documentation; (e) at least the equivalent of a ninth grade education in order to assure reading and comprehension skills sufficient to permit participation.

Between April and September, 1981, 47 individuals were interviewed and evaluated for participation in the study. Thirty-eight subjects met the criteria for participation and 37 were willing and able to participate. Five subjects (two in the biofeedback only group, and one each from the three other treatment conditions) did not complete the study. Two subjects reportedly began new employment which precluded their continued involvement in the study, while two subjects simply stated they no longer had the time to participate. A final subject did not return for his third treatment session and various efforts to contact him were unsuccessful.

Subjects were assigned to one of four treatment conditions: (a) biofeedback plus stress-coping training; (b)
pseudo-biofeedback plus stress-coping training; (c) biofeedback only; (d) stress-coping training only. Subjects were assigned to treatment conditions according to their sex and their order of presentation at the screening interview and evaluation. As a means of assuring proportional representation of each sex in each of the treatment conditions, separate randomized block running schedules were used for male and females. With regard to subjects who did not complete the study, the next same-sex subject who met the criteria for participation served as a replacement for each of these subjects. A total of 32 subjects completed the study, eight per treatment condition. There were seven females and one male in each treatment condition. All of the subjects were Caucasian and ranged in age from 19 to 59 years (M = 33.5 years). Average years of education was 13.5 (S.D. = 2.0) and myofascial pain difficulties ranged in onset from 6 months to 15 years prior to the beginning of this study (M = 58.7 months).

Instruments

**Demographic data questionnaire.** Sex, age, marital status, race, occupation, number of years of education, and referral source was obtained from the subjects during the screening interview and evaluation (Appendix D).

**Signs and symptoms evaluation.** During the initial contact, each subject underwent an evaluation of the signs and symptoms of their pain syndrome in order to obtain a
differential diagnosis of MPDS. The evaluation was conducted at the M.U.S.C. Dental School and consisted of an interview and clinical exam conducted by two dentists who served as co-investigators in the present study. The interview (Appendix E) obtained information regarding the history and characteristics of the subject's pain syndrome (i.e., nature and location of the pain, length of illness, previous treatments, oral habits, current medication). Any history of bruxism and clenching, as well as chronic headaches and discomfort related to bite, was also determined. Additionally, any history of physical trauma to the head or neck was determined.

The clinical examination was also conducted by the dentists. Half of the subjects (n = 16) were evaluated independently by both dentists who were blind to the other's assessment. This format allowed for an assessment of the reliability between raters of the presence, absence, and measurement of the signs and symptoms of the subject's pain syndrome. An outline of the clinical examination can be found in Appendix F. Germane to the present study was a record of the presence or absence of the following signs and symptoms found in MPDS (Laskin, 1969; Scott & Gregg, 1980):

(a) muscle tenderness as determined by the subjective report of the subject when the masticatory muscles were palpated bilaterally. Additionally, any other areas of reported muscle tenderness such as the condylar leads (Snow, 1977)
were recorded; (b) limitation of jaw opening as measured in two ways. The first was a measure in millimeters between the right maxillary and mandibular central incisors at the maximum opening the subject could accomplish, regardless of discomfort. The second was a similar measure of the maximum opening the subject could accomplish without any discomfort. In addition, any deviation of the mandible in opening and closing was recorded; (c) the presence or absence of joint sounds on opening or closing. Additional analysis of the subject’s oral cavity (e.g., occlusal analysis, wear facets, periodontium exam, etc.) was conducted in an attempt to evaluate individual differences in response to treatment. Finally, patients with a positive history of arthritis or rheumatism underwent a series of tomograms in order to rule out structural damage to the temporomandibular joint.

Approximately two weeks after the final treatment sessions, subjects were once again assessed at the M.U.S.C. Dental School where the dentists re-evaluated each subject’s signs and symptoms (utilizing the same examination as for the pre-test). This posttreatment evaluation was also utilized by the dentists to judge the outcome of the treatment. In addition, at the conclusion of this posttreatment evaluation, subjects were asked to rate the outcome of treatment. Outcome was judged on the following 7-point scale:

1. Significantly worse—large increases in the frequency, duration, and intensity of pain; exacerbations in muscle tenderness, etc.
2. Clearly worse—moderate increases in the frequency, duration, and intensity of pain; exacerbations in muscle tenderness.

3. Slightly worse—small but noticeable increase in the subject's MPDS signs and symptoms.

4. No change.

5. Slight improvement—small but noticeable decrease in myofascial pain activity.

6. Significant improvement—a large decrease in myofascial pain activity as well as other signs and symptoms, but not totally symptom-free.

7. Symptom-free—no myofascial pain activity; no muscle tenderness.

**Masseter EMG.** Masseter EMG was assessed during a 10 minute resting period at the first treatment session and following each treatment session for participants receiving biofeedback or pseudo-biofeedback training. Subjects in the stress-coping only treatment condition were monitored for a 10-minute resting period prior to their initial treatment session and for a 10-minute resting period following the tenth and final treatment session. This allowed for an assessment of the impact of each of the treatment procedures on the muscles of mastication responses that have been hypothesized to be crucial to the genesis and maintenance of myofascial pain (Laskin, 1969; Scott & Gregg, 1980). Subjects were seated in a comfortable reclining chair located
in an Industrial Acoustics Corporation electrically shielded, sound attenuated chamber at the VA Medical Center's psychophysiological laboratory. Masseter EMG activity was monitored from masseter surface electrode placements (Budzynski & Stoyva, 1973) directed to a Grass Model 7P3 preamplifier and 7D polygraph. A Med Associates Inc. modular system was used to quantify the data. The raw EMG output from the preamplifier was fed into a Med Associates EMG coupler (bandpass filter 8-1000 Hz) which fed into an analogue to digital converter. Average masseter EMG was printed out in microvolts every ten seconds. Feedback was produced through a Med Associates' voltage-controlled oscillator. Utilizing the integrated output of the Grass 7P3 preamp, a Med Associates Threshold Comparator set the criterion levels for feedback purposes.

**Self-monitoring pain diary (SMPD).** This self-report data sheet was adapted from the work of Budzynski, Stoyva, and Adler (1970), and Stenn et al., (1979) for use in this study (Appendix G). Subjects were trained to record, for each hour of their waking day: (a) the degree of myofascial pain, according to a 7-point scale anchored by adjectives and behavioral descriptions; (b) medication consumption; (c) mood ratings, according to nine defined categories; and (d) environmental setting. Additionally, as part of an ongoing homework assignment for subjects receiving stress-coping training, they were asked to describe their thoughts,
activities, etc., that seem to accompany changes in the degree of myofascial pain and/or mood ratings.

Subjects were required to maintain the SMPD for at least two weeks prior to initiating treatment continuing through a two week post-treatment assessment. From the SMPD, four dependent measures (Holroyd et al., 1977) were computed for each week: (a) myofascial pain activity—a composite measure consisting of the product of myofascial pain intensity times duration; (b) total duration—the total number of hours of myofascial pain reported; (c) elevated duration—the total number of hours of myofascial pain reported with clinically significant intensity, recorded only for myofascial pain achieving an intensity rating greater than 3; (d) mean intensity—defined as myofascial pain activity divided by myofascial pain total duration.

Psychosomatic checklist. Subject's rating of the frequency of occurrence of 18 common psychosomatic complaints was obtained on the psychosomatic checklist (Cox, Freundlich, & Meyer, 1975) before treatment and at the end of the final treatment session.

Beck Depression Inventory (BDI). The BDI (Beck, Ward, Mendelson, Mock & Erbaugh, 1961) is a widely used 21 item self-report questionnaire designed to measure behavioral manifestations of depression. It was utilized in the study to provide a measure that might be helpful in predicting individual differences in response to treatment. BDI
assessments were obtained before treatment and at the end of the final treatment session.

Self-Control Schedule (SCS). The SCS (Rosenbaum, 1980) is a 36-item self-report inventory designed to assess individual tendencies to apply self-control methods to the solution of behavioral problems. The inventory was utilized to provide a measure that might be helpful in predicting individual differences in response to treatment. It was suspected that individuals high in self-control tendencies might be more responsive to self-control procedures such as those employed in the present study. SCS assessments were obtained before treatment.

Credibility and outcome expectancy measure. In order to assess participants' perceptions of the credibility of the treatment they received and their expectancy for therapeutic change, they were required to respond to a 7-point scale evaluating the following (Appendix H): (a) the likelihood of their recommending the treatment to a friend suffering from myofascial pain; (b) how important they thought it that the treatment be made available to other myofascial sufferers; and (c) how much therapeutic change they expected to receive from participating in the treatment program. Credibility ratings were obtained at the first session and at the end of the final treatment session. Expectancy ratings were obtained at the conclusion of the first and sixth session.
Procedure

Subjects accepted for treatment following the interview and clinical evaluation of their pain syndrome were provided the SMPD on which to record daily myofascial pain activity. Subjects were instructed to maintain such recordings for a two week period prior to their scheduled first treatment session. Subjects were assigned to one of four treatment conditions: (a) biofeedback plus stress-coping training; (b) pseudo-biofeedback plus stress-coping training; (c) biofeedback only; (d) stress-coping training only. Subjects receiving stress-coping training were assigned to one of three therapists representing two therapists conditions (i.e., principal investigator versus other therapist).

Treatment

Treatment procedures were administered during 10 bi-weekly individual sessions conducted at the psychophysiological laboratory facility of the Charleston Veteran's Administration Medical Center. During the first session all subjects initially met individually with the biofeedback therapist who administered pretreatment measures. The objective of the first treatment session was to familiarize subjects to the rationale, equipment, and procedures of their 5-week treatment program. Additionally, at the conclusion of the first treatment session, subjects were required to rate the credibility of their treatment program and their expectations for success. Specific treatment procedures were as follows:
Biofeedback training. Two groups, biofeedback plus stress-coping training and biofeedback only, received biofeedback training. The treatment was adapted from the method described by Dohrmann and Laskin (1978). During the first session, after subjects had completed pretreatment measures, they were: (a) introduced to the theory of EMG feedback training and how such training would help in bringing about a remission of their symptoms; (b) acquainted with the equipment and how it would be used to teach them to control the level of tension in their muscles of mastication. Subjects were seated comfortably in the biofeedback chamber. The skin over the sights of electrode placement was prepared with alcohol rub and then a conductive electrode paste was applied. Two surface electrodes were placed over the main body of the masseter muscle on the side of maximal pain, with a reference electrode placed behind the ear (Budzynski & Stoyva, 1973). Placement was standardized by asking the subject to clench his/her jaw and by positioning the electrodes over the bulging area of the masseter muscle.

During the initial session, after the electrodes were correctly attached, subjects were provided auditory feedback of existing muscle tension. They were informed that the tone of the auditory signal was directly proportional to the amount of muscle tension. The subjects were asked to clench and relax their jaws to show them that a high tone did, in fact, indicate increased muscle activity and that they did
have control over the tone. The auditory feedback was then turned off and subjects were asked to simply close their eyes, sit still, and relax while they became accustomed to the equipment. The therapist then left the room to calibrate the polygraph and record a 10-minute baseline. The mean EMG recording during this baseline period served as the measure of pretreatment muscle tension. At the conclusion of this period, each subject was given instructions to follow at home. They were told to avoid possible clenching or grinding of their teeth, especially during periods of stress. Additionally, subjects were provided taped instructions of cue controlled relaxation techniques (Bernstein & Borkovec, 1973). Subjects were instructed to practice relaxation at home for at least twenty minutes, twice a day. The above instructions were repeated after each session. Following the fourth session, subjects were additionally instructed to employ their relaxation skills at the initial signs of myofascial pain.

Subjects assigned biofeedback training received nine 30-minute training sessions. During the initial five minutes of each session, after the electrodes had been attached, subjects were asked to sit still and rest comfortably while the therapist recorded their level of masseter EMG activity. This information was utilized to adjust the criterion level for training. Biofeedback training then commenced and subjects were instructed to utilize their relaxation skills to
keep the frequency of the feedback tone as low as possible or to turn it off completely. Each session consisted of three 4-minute periods of training separated by 1-minute segments of rest in which the subject could swallow, change positions, etc. An important aspect of training involved a shaping procedure that was accomplished by varying the gain of the feedback loop. As subjects learned to relax the masseter muscle and decrease or turn-off the tone, the gain of the feedback loop was gradually increased. Each subject was then obliged to further decrease the level of EMG activity to keep the auditory feedback off or at a low frequency, thus making the task progressively more difficult. The initial criterion level for feedback was the mean EMG level obtained during the pretreatment session. Following this, the criterion level utilized was the level used at the completion of the previous training session. Criterion levels were adjusted at the beginning of a session or during the 1-minute rest periods if the subject, during the previous training period, was able to maintain feedback at the no-tone level approximately 80% of the time. Budzynski, Stoyva, and Adler (1970) reported that this strategy does not produce frustration while allowing for learning to occur.

Following the final 4-minute training session, subjects were informed that they would receive no further biofeedback, but that they should continue relaxing and focus on the feelings of warmth and heaviness as all tension leaves the body.
During this time, average EMG recordings from thirty 10-second trials (i.e., 5 minutes) was recorded. Mean EMG level from these trails served as the measure of EMG activity during that session.

**Stress-Coping training.** Three groups (biofeedback plus stress-coping training, pseudo-biofeedback plus stress-coping training, and stress-coping training only) received stress-coping training. This treatment focused on altering maladaptive cognitive and behavioral responses that were assumed to mediate the occurrence of an individual's emotional reactions. Specific procedures were adapted from cognitively oriented therapy procedures (e.g., Goldfriend, 1979; Meichenbaum, 1974; Turk, 1977) and are outlined in Appendix I. The procedures were designed to maximize the occurrence of causal reattribution and the development of self-monitoring and cognitive coping skills. However, as Meichenbaum (1974) recommended, training was sufficiently flexible so that it could be adapted to individual needs and changing environmental demands. Consequently, the focus of particular therapeutic procedures varied somewhat for each individual depending upon the nature of the stressful situations in question. Similar to previous studies on cognitively oriented stress-coping training (e.g., Holroyd et al., 1977; Meichenbaum, Turk, & Burnstein, 1975) the present treatment program adhered to a three-stage strategy.
1. The initial strategy was to provide subjects an explanation of their problem which encouraged them to attribute it to relatively specific cognitive and behavioral deficiencies rather than to external stimuli or complex inner dispositions. The treatment rationale (Appendix J) emphasized that MPDS results, ultimately, from stress and that stress reactions are directly influenced by labels, expectations, and self-statements. Concrete examples were utilized to illustrate the variety of events that can be perceived as stressful by different individuals and the way that thoughts and self-statements induce such stress. The goals of treatment were specified.

After the treatment rationale was presented, didactic examples were utilized to further illustrate the various ways specific beliefs, thoughts, or behaviors might exacerbate stress. Once subjects recognized that self-statements mediate emotional arousal, the role that irrational or unrealistic expectations and beliefs play in predisposing an individual to experience stress was illustrated and discussed.

In summary, the initial therapeutic task was to convincingly present an alternative framework in which the subject could view his/her problem, one that emphasized those cognitive and behavioral variables that are potentially under his/her control.

2. The second stage of treatment involved instruction in the monitoring of stress responses so that the subject
learned to identify specific eliciting stimuli and on-going cognitive and behavioral responses to stress. The therapeutic task was to assist the subject in identifying patterns of covert and overt events that regularly precede, accompany, and follow stressful interactions. In the present treatment program, several methods were utilized to achieve the goal: (a) Homework assignments—utilizing the self-monitoring pain diary, subjects were trained to monitor daily changes in the degree of myofascial pain, mood, environmental settings, and to describe their thoughts and activities that seem to correlate with these changes. The homework assignments were reviewed during each session so that the relationship between cognitive and emotional responses could be identified. (b) From a review of homework assignments and other discussions, a list of stressful situations was constructed. Beginning with relatively easy situations, the subject and therapist focused on identifying: (1) the cues that triggered tension and anxiety; (2) how the subject responded when anxious (withdrawal, attack, etc.); (3) the subject's thoughts prior to becoming aware of tension, while tense, and subsequently; and (4) the way in which these cognitions appeared to contribute to the subject's tension and myofascial pain.

It was expected that instruction and practice in self-monitoring would serve to create evidence which supported a cognitive interpretation of emotional distress and thus allow subjects to conclude that what they need to learn is how to stop thinking irrationally in certain situations.
3. The third and final stage of treatment involved teaching subjects alternate ways of coping with stressful situations via didactic instruction, modeling and graduated practice. In general, subjects were encouraged to employ signs of impending distress as a signal to engage in cognitive coping skills strategies designed to alter the stressful interaction or to manage the subject's emotional responses. Several cognitive coping skills strategies were primarily taught in the present training program. The strategies provided were designed to enable the subject to employ each of three main types of intrapsychic coping responses that have been indentified by Lazarus and his co-workers (Lazarus, Averill, & Opton, 1974): cognitive reappraisal, attention deployment, and fantasy. Specifically, subjects were instructed in the use of cognitive restructuring as a coping skill (e.g., "What am I thinking to induce my distress?" "What are the facts?"); self-instructional training (Meichenbaum, 1974) so as to learn to emit coping self-instructions when dealing with a stress-eliciting event ("Calm down, you can handle this, concentrate on what needs to be done."); and imagery ("Imagine myself for a moment care-free, at the beach.").

**Pseudo-biofeedback training.** One group, pseudo-biofeedback plus stress-coping training, received pseudo-biofeedback training. This treatment was designed to control for the non-specific effects of biofeedback training. Furthermore, since
subjects who received pseudo-biofeedback training also
received stress-coping training, this group was equated with
the biofeedback plus stress-coping training group in terms
of treatment contact time and thus served as a more adequate
comparision group.

During the first session, subjects receiving pseudo-
biofeedback training were not informed about EMG biofeedback.
Rather they were provided the following instructions adapted
from the work of Dohrmann and Laskin (1978):

The application of electric currents to the body as a
means of therapy has a history that dates back to ancient
times. Recent progress has been rapid and numerous, new
applications of electric currents are being utilized in
medicine today. One of the most promising areas in
which electric currents are being utilized is as muscle
relaxant. During this 30-minute portion of treatment,
your painful jaw muscles will be exposed to a low-grade
electrical current that will block the muscles' activity
and thereby cause relaxation. However, be assured that
the current is of such a low intensity that it is completely
harmless and you will not feel anything.

Subjects were then seated comfortably in the biofeedback
chamber in order to get acquainted with the relaxation techni-
que and be assured that the technique was not painful. In
order to maximize the functional similarites between the
pseudo-biofeedback and biofeedback conditions, subjects in
both groups were prepped and hooked up to the electrodes in the same manner. However, pseudo-biofeedback subjects were not provided with auditory feedback, but simply asked to sit still and relax quietly for 15 minutes while they were supposedly receiving electrical stimulation via the electrodes. During the last 10 minutes of the initial session, a baseline recording of EMG activity was obtained. Pseudo-biofeedback subjects were also given home instructions to avoid possible clenching or grinding of their teeth, especially during periods of stress. However, pseudo-biofeedback subjects were not provided taped instructions of relaxation techniques, instead, they were asked to lie down in a quite place at home and relax for twenty minutes, twice a day. The above instructions were repeated after each session.

Subjects in pseudo-biofeedback training also received nine 30-minute training sessions. During each session, they merely sat quietly and were relaxed by the supposed receiving of electrical stimulation. During the last five minutes, subjects were informed that the electrical current had been turned off, but that they were to sit quietly and focus on the relaxed feelings of warmth and heaviness throughout their body. During this time, average EMG readings from thirty 10-second trials (i.e., 5 minutes) were recorded. Mean EMG level from these trials served as a measure of EMG activity during that session.
The multifaceted treatment groups (biofeedback plus stress-coping and pseudo-biofeedback plus stress-coping) differed from stress-coping only and biofeedback only treatment groups in several ways. The focus of treatment in the former was on altering both the cognitive and physiological responses to stressful situations rather than just one response system as in the biofeedback only and stress-coping only groups. Consequently, the treatment rationale offered subjects in the multifaceted treatment groups emphasized that myofasical pain resulted from both psychological stress and sustained masseter muscle tension. Treatment sessions also differed in length of time with the multifaceted treatments sessions lasting approximately 75 minutes compared to the 30- to 40- minute sessions accorded biofeedback only and stress-coping only subjects. During the initial 0.5 hour of each session, subjects receiving multifaceted treatment met individually with the biofeedback therapist for biofeedback or pseudo-biofeedback training as described above. At the conclusion of each training session, subjects were directed to an adjoining therapy room where they met individually with their therapist for cognitively oriented stress-coping training.

Therapists. The principal investigator and two fellow clinical psychology interns at the Medical University of South Carolina served as therapists for the stress-coping training treatment. Because of time constraints and previous commitments, therapists were not able to treat equal number
of subjects. Consequently, half of the subjects in each of the stress-coping treatment conditions \((n = 12)\) were seen by the principal investigator, while the other half were divided \((n = 6)\) among the two other therapists. All therapists had received extensive training in cognitive behavioral techniques and were experienced in utilizing such procedures in individual therapy. Additionally, therapists were provided manuals describing the therapeutic procedures and instructions concerning the procedures to be followed in each session. Furthermore, treatment sessions were discussed regularly by the therapists to encourage comparability of treatments and conformity to the treatment manuals.

The biofeedback training was administered by the biofeedback therapist employed at the psychophysiological laboratory at the VAMC. This individual has extensive background in biofeedback therapy and presently is completing his doctorate in psychology with an emphasis on psychophysiological techniques.

**Results**

Analyses were performed to (a) test for differences across conditions in biographic/demographic variables, level of depression, treatment credibility and expectancies for change, as well as possible therapist differences in outcome. (b) Assess outcome, and (c) identify variables predictive of response to treatment.
Treatment group composition. Table 1 summarizes the biographic/demographic characteristics of the four treatment groups. One way analyses of variance revealed no significant difference between the groups for the variables of age, $F(3,28) = .82$, $p > .05$, number of years of education, $F(3,28) = .65$, $p > .05$, number of months experiencing myofascial pain problems, $F(3,28) = 48., p > .05$, or number of previous treatments, $F(3,28) = 1.36$, $p > .05$. The chi-square statistic determined that these groups were also not different regarding marital status, $\chi^2 (9) = 7.97$, $p > .05$.

Table 1
Summary of Biographic and Demographic Characteristics of Treatment Groups

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BFSC</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>35.8</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>6.4</td>
</tr>
<tr>
<td>Number of Years Education</td>
<td></td>
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<tr>
<td>Mean</td>
<td>12.9</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>1.3</td>
</tr>
<tr>
<td>Number of Months of Symptoms</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>63.9</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>54.3</td>
</tr>
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</table>
Table 1--Continued

<table>
<thead>
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<th>Characteristics</th>
<th>Group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>BFSC</td>
</tr>
<tr>
<td><strong>Number of Previous Treatments</strong></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>3.1</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>1.06</td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
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<tr>
<td>Married</td>
<td>87.5%</td>
</tr>
<tr>
<td>Single</td>
<td>0%</td>
</tr>
<tr>
<td>Divorced</td>
<td>12.5%</td>
</tr>
<tr>
<td>Widowed</td>
<td>0%</td>
</tr>
</tbody>
</table>

*Note. BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback and stress-coping; BF = Biofeedback only; SC = Stress-coping only.*

**Treatment credibility and expectancies for change.** Group means for the treatment credibility and subjects' expectancies for therapeutic change scores at the various measurement periods are presented in Table 2. One way analysis of variance computed on the mean treatment credibility scores revealed no significant difference between the groups at Session 1, \( F(3,28) = .54 \), or at the conclusion of treatment, \( F(3,28) = .30 \), both \( p's > .60 \). Analyses of Session 1 and Session 6 subjects' expectations for change scores also revealed no significant differences between groups, \( F(3,28) = .20 \) and \( .90 \) respectively, both \( p's > .40 \). Thus, treatment groups did not differ with regard to their perception of the credibility.
of their treatment nor their expectancies for therapeutic change. Additionally, inspection of Table 2 reveals that all the treatment procedures were rated as highly credible at both evaluations across groups.

Table 2

Means of Treatment Credibility and Subjects' Expectancies for Therapeutic Change Scores

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>BFSC</th>
<th>PSC</th>
<th>BF</th>
<th>SC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment Credibility&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Session 1</td>
<td></td>
<td>13.1</td>
<td>11.8</td>
<td>12.9</td>
<td>13.1</td>
</tr>
<tr>
<td>Posttreatment</td>
<td></td>
<td>13</td>
<td>13.8</td>
<td>13.4</td>
<td>13.5</td>
</tr>
<tr>
<td>Expectancy for Change</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Session 1</td>
<td></td>
<td>5.1</td>
<td>5.3</td>
<td>5.4</td>
<td>5.5</td>
</tr>
<tr>
<td>Session 6</td>
<td></td>
<td>5.8</td>
<td>5.3</td>
<td>6.0</td>
<td>5.8</td>
</tr>
</tbody>
</table>

<sup>a</sup>Sums of two 7-point scales.

Note. BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback and stress-coping; BF = Biofeedback only; SC = Stress-coping only.

Level of depression. Gessel (1975) observed that MPDS patients who were more depressed in combination with other characteristics are more likely not to benefit from biofeedback/relaxation treatment procedures. Consequently, it was important to determine whether treatment groups differed from one another with regard to depression level prior to
intervention. Group means for the BDI scores obtained prior to treatment are presented in Table 4. A one-way analysis of variance revealed no significant differences between the groups, \( F(3,28) = 1.02, p > .35 \), indicating that groups were not different prior to intervention.

**Therapist effects.** Because of the small sample size, data for all three stress-coping training conditions seen by the same therapist were pooled in the analysis to determine if subjects responded differently to therapists. As mentioned, therapists were not able to treat equal number of subjects, consequently, subjects were assigned to one of two therapist conditions, principal investigator versus "other therapist." To determine whether subjects in the "other therapist" condition responded differently to the two therapists, one way analyses of covariance were performed on the four myofascial pain measures and psychosomatic checklist at posttreatment (with pretreatment scores as covariate). None of these analyses achieved significance (all \( p \)'s > .05). Since these two therapists produced similar treatment outcome, the data was pooled in the analysis to determine if subjects responded differently to the two therapist conditions (i.e., principal investigator versus "other therapist"). Analyses of covariance revealed no significant therapist effects for myofascial pain activity, \( F(1,21) = .50 \), total duration, \( F(1,21) = .001 \), elevated duration, \( F(1,21) = 2.96 \), mean intensity, \( F(1,21) = 1.1 \), or psychosomatic
symptoms, $F(1, 21) = .15$, all $p$'s > .05. Since the two therapist conditions produced similar treatment outcomes, in subsequent analyses therapist condition was collapsed.

**Outcome**

Examination of pretreatment scores presented in Tables 3 and 4 reveals occasional differences among groups on the dependent measures. Although separate analyses of variance revealed that none of these differences were significant (all $p$'s > .05), analysis of covariance (with pretreatment scores as the covariate) was employed to provide the most sensitive comparative assessment of treatment differences. In those cases where significant differences were found, the Newman-Keuls test (applied to the adjusted means) was used to determine where the differences were. To determine whether each treatment condition resulted in significant pretreatment to posttreatment differences on the variety of dependent measures, $t$-tests for correlated means were assessed separately for each group. The mean difference scores, $t$ and $p$ values obtained from the $t$-test analyses are presented in Tables 7 and 8 (see Appendices K and L).

**Myofascial pain recordings.** The four myofascial pain measures (i.e., myofascial pain activity, total duration, elevated duration, mean intensity) were computed for each week of the study. Prior to analyses, pain recordings obtained for each of the 2-week periods during pretreatment and posttreatment were computed into mean weekly averages.
These are presented in Table 3. Inspection of Table 3 indicates that all treatment conditions showed a marked reduction in myofascial pain recordings with the biofeedback only group showing the more modest improvement.

### Table 3

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>BFSC</th>
<th>PSC</th>
<th>BF</th>
<th>SC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Myofascial Pain Activity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td></td>
<td>198.3</td>
<td>173.3</td>
<td>174.4</td>
<td>177.6</td>
</tr>
<tr>
<td>Posttreatment(^a)</td>
<td></td>
<td>40.9</td>
<td>61.6</td>
<td>107.5</td>
<td>65.2</td>
</tr>
<tr>
<td><strong>Total Duration</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td></td>
<td>85.3</td>
<td>85.3</td>
<td>88</td>
<td>78.1</td>
</tr>
<tr>
<td>Posttreatment(^a)</td>
<td></td>
<td>33.1</td>
<td>43.1</td>
<td>60.3</td>
<td>47.3</td>
</tr>
<tr>
<td><strong>Elevated Duration</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td></td>
<td>9.9</td>
<td>7.4</td>
<td>9.8</td>
<td>7.5</td>
</tr>
<tr>
<td>Posttreatment(^a)</td>
<td></td>
<td>0</td>
<td>.88</td>
<td>2.52</td>
<td>1.6</td>
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<tr>
<td><strong>Mean Intensity</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td></td>
<td>2.4</td>
<td>1.9</td>
<td>2.0</td>
<td>2.2</td>
</tr>
<tr>
<td>Posttreatment(^a)</td>
<td></td>
<td>.82</td>
<td>1.21</td>
<td>1.66</td>
<td>1.04</td>
</tr>
</tbody>
</table>

\(^a\)Adjusted means.

Note: BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback and stress-coping; BF = Biofeedback only; SC = Stress-coping only.
Analysis of covariance conducted on the posttreatment myofascial pain activity scores while suggestive of a trend \( (p = .09) \), failed to reveal significant treatment differences, \( F(3,27) = 2.38 \). The \( t \)-tests \( (df = 7, \text{one-tailed}) \) for correlated means presented in Table 7 (see Appendix K) revealed that all treatment groups showed a significant reduction in myofascial pain activity at posttreatment \( (\text{all } p \text{'s } < .0025) \).

Analysis of covariance conducted on the posttreatment total duration component measure of myofascial pain activity also failed to reveal significant treatment differences, \( F(3,27) = .734, p > .05 \). The \( t \)-tests \( (df = 7, \text{one-tailed}) \) for correlated means once again revealed that all treatment groups showed a significant reduction in total pain duration at posttreatment \( (\text{all } p \text{'s } < .04) \).

Analysis of covariance conducted on the posttreatment elevated duration component measure of myofascial pain activity approached but did not reach significant treatment differences, \( F(3,27) = 2.62, p = .07 \). The \( t \)-tests \( (df = 7, \text{one-tailed}) \) for correlated means again revealed that all treatment groups showed a significant reduction in elevated duration at posttreatment \( (\text{all } p \text{'s } < .023) \).

Analysis of covariance conducted on the posttreatment mean intensity measure of myofascial pain activity did reveal significant treatment differences, \( F(3,27) = 3.06, p < .05 \). Newman-Keuls test conducted on the adjusted posttreatment means further revealed that the biofeedback plus stress-coping
group differed significantly from the biofeedback only group, but none of the other treatment groups differed significantly from one another. The t-tests (df = 7, one tailed) for correlated means revealed that all treatment groups showed a significant reduction in mean myofascial pain intensity at posttreatment (all p's < .03).

In summary, all treatment groups showed significant reductions on the four component measures of myofascial pain at posttreatment evaluation. While comparison of treatment effects approached significance on the myofascial pain activity and elevated duration measures, it was only on the mean intensity measure where significant treatment differences were obtained with the biofeedback plus stress-coping group reporting lower mean intensity than the biofeedback only group.

**Objective MPDS measures.** From the signs and symptoms evaluation conducted by the dentists prior to treatment and two weeks following the conclusion of treatment, four dependent measures were computed: (a) number of muscle areas tender to palpitation; (b) maximal jaw opening without discomfort; (c) maximal jaw opening regardless of discomfort; and (d) presence or absence of joint sounds on opening or closing the jaw. The pretreatment and adjusted posttreatment means for the first three measures are presented in Table 4.

During the pretreatment evaluation, half of the subjects were evaluated independently by each of the dentists who were blind to the other's assessment. Pearson product-moment
correlation coefficients between dentists' ratings were computed for each of the dependent measures. The inter-rater reliability was .94 for the number of muscle areas tender to palpitation, .87 for maximal jaw opening without discomfort, .77 for maximal jaw opening regardless of discomfort, and 1.0 for the presence or absence of joint sounds.

Table 4

Pretreatment and Posttreatment Means for the Non-myofascial Pain Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>BFSC</th>
<th>PSC</th>
<th>BP</th>
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<tr>
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<td>Posttreatment(^a)</td>
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<td>3.3</td>
<td>5.5</td>
<td>4.3</td>
</tr>
</tbody>
</table>

\(^a\)Adjusted means.

\(^b\)Measured in millimeters.

\(^c\)Expressed as microvolts.

\(^d\)Larger scores represent a lower incidence of psychosomatic symptoms.

Note. BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback and stress-coping; BF = Biofeedback only; SC = Stress-coping only.

Analysis of covariance conducted on the mean number of muscle areas tender to palpitation at posttreatment failed to reveal significant treatment differences, \(F(3,27) = .42, p > .05\). The \(t\)-tests (\(df = 7\), two-tailed) for correlated means conducted on the objective measures of MPDS are presented in Table 8 (see Appendix L). The results revealed that all treatment groups reported significantly fewer number of muscle areas tender to palpitation following treatment (all \(p\)'s < .05).

Analyses of covariance conducted on the maximal jaw opening without discomfort, and regardless of discomfort, at posttreatment assessment also failed to reveal any
significant treatment differences, \( F(3,27) = .76 \) and \( .42 \) respectively, both \( p > .05 \). Furthermore, \( t \)-tests (df =7, two-tailed) for correlated means revealed that none of the treatment groups showed significant pre- to post differences on these two measures (all \( p > .05 \)).

The chi-square statistic was used to analyze the presence or absence of joint sounds, since these data did not conform to the covariance or variance model. The chi-square statistic indicated that, prior to treatment, the groups did not differ with regard to the percentage of subjects with joint sounds as a symptom, \( \chi^2(3) = .58, p > .05 \). Separate chi-square statistics conducted for each group revealed that none of the groups showed a significant change in the percentage of subjects with this symptom as a result of treatment. The \( \chi^2(1) \) values ranged from 0.0 to 1.07, with all \( p > .05 \). Additionally, a chi-square analysis conducted on the total number of subjects reporting joint sounds as a symptom at pretreatment versus posttreatment regardless of treatment group, also failed to reveal any significant difference \( \chi^2(1) = 1.07, p > .05 \).

Electromyographic activity. Examination of Table 4 reveals that mean resting levels of masseter electromyographic activity were very similar for the four treatment groups prior to treatment, \( F(3,28) = .70, p > .05 \). Analysis of covariance conducted on the masseter EMG scores obtained at the conclusion of the final treatment session revealed significant
treatment differences, $F (3,27) = 7.45, p < .001$. Newman-Keuls's test conducted on the adjusted posttreatment means revealed that the biofeedback plus stress-coping group differed significantly from the pseudo-biofeedback plus stress-coping and stress-coping only groups but not from the biofeedback only group, which did not differ significantly from the pseudo-biofeedback plus stress-coping and stress-coping only groups. The $t$-tests ($df = 7$, two-tailed) for correlated means (see Table 8 in Appendix L) comparing Session 1 and posttreatment masseter EMG levels revealed that only the biofeedback plus stress-coping and biofeedback only groups showed a significant reduction in masseter EMG, $t = -2.64$ and $-8.65$, respectively, both $p$'s $< .05$. Thus, biofeedback training was effective in teaching clients to effectively reduce their level of masseter muscle activity. However, reductions in masseter electromyographic activity were not found to be significantly related to myofascial pain activity improvement, $r (32) = .12, p > .05$.

Additional measures. Pretreatment and adjusted posttreatment means for the psychosomatic checklist and Beck Depression Inventory are presented in Table 4. Analysis of covariance conducted on the posttreatment psychosomatic checklist scores failed to reveal significant treatment differences, $F (3,27) = .58, p > .05$. The $t$-tests ($df = 7$, two-tailed) for correlated means presented in Table 8 (see Appendix L) revealed that all treatment groups reported
significantly fewer psychosomatic symptoms following treatment (all p's < .05).

Analysis of covariance conducted on the posttreatment BDI scores also failed to reveal significant treatment differences F (3,27) = .30, p > .05. The t-tests (df = 7, one tailed) for correlated means (see Appendix L) revealed that all the treatment groups except the biofeedback only group reported a significant reduction in depressive symptoms following treatment (p's < .05).

Patient and examiners' rating of outcome. The patients' and dentists' opinion about the immediate success of treatment are presented in Table 5. Inspection of Table 5 reveals that, in general, patients were more optimistic in their rating of outcome than the dentists. Yet, there does appear to be agreement between the dentists and subjects in the biofeedback plus stress-coping group. From the patients' ratings of outcome, it can be seen that the majority of patients, regardless of treatment condition, rated themselves as significantly improved or symptom free at the conclusion of treatment. In contrast, the dentists rated 75% of the patients in the biofeedback plus stress-coping group as significantly improved or symptom free, while only 50% of the subjects in each of the other treatment conditions received such a favorable outcome rating. No subject in the biofeedback only group was rated by the dentists as being symptom free.
Table 5

Patients and Dentists' Ratings of Treatment

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Ratings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Symptom Free</td>
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<tr>
<td>Biofeedback and Stress-Coping</td>
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</tr>
<tr>
<td>Patients' Rating</td>
<td>4 (50%)</td>
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<tr>
<td>Dentists' Rating</td>
<td>3 (37.5%)</td>
</tr>
<tr>
<td>Psuedo-Biofeedback and Stress-Coping</td>
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</tr>
<tr>
<td>Patients' Rating</td>
<td>3 (37.5%)</td>
</tr>
<tr>
<td>Dentists' Rating</td>
<td>1 (12.5%)</td>
</tr>
<tr>
<td>Biofeedback Only</td>
<td></td>
</tr>
<tr>
<td>Patients' Rating</td>
<td>1 (12.5%)</td>
</tr>
<tr>
<td>Dentists' Rating</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Stress-Coping Only</td>
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</tr>
<tr>
<td>Patients' Rating</td>
<td>3 (37.5%)</td>
</tr>
<tr>
<td>Dentists' Rating</td>
<td>1 (12.5%)</td>
</tr>
</tbody>
</table>

Comparisons among outcome measures. Table 6 presents a Pearson-product moment correlation matrix comparing each pair of dependent outcome measures at posttreatment. Inspection of
Table 6 reveals that all the myofascial pain recording component measures, the Beck Depression Inventory, and psychosomatic checklist were significantly correlated with one another. Masseter electromyographic activity obtained at the conclusion of treatment was positively correlated with elevated duration and mean intensity measures of myofascial pain. Number of muscle areas tender to palpitation was significantly correlated with the elevated duration of myofascial pain. Finally, maximal jaw opening without discomfort and maximal jaw opening regardless of discomfort were significantly correlated with one another but not with any other outcome measures.

**Prediction of Response to Treatment**

In order to determine variables related to MPDS symptom improvement all pretreatment scores, treatment credibility and expectancies for therapeutic change ratings, and various biographic/demographic variables were correlated with MPDS symptom improvement scores. Posttreatment improvement scores were computed by the formula \(((\text{pretreatment} - \text{posttreatment})/\text{pretreatment}) \times 100\%)\). Eight significant correlations were found (\(p = .01\) to guard against spurious findings). Pretreatment elevated duration was inversely related to improvement in myofascial pain activity, \(r = -.40, p = .01\), and total duration, \(r = -.42, p = .007\). Yet, pretreatment elevated duration and mean intensity were positively associated with improvement in elevated duration, \(r\)'s = .55 and .50, respectively, both \(p\)'s < .002. Pretreatment myofascial pain activity was inversely related with improvement in total duration,
Table 6

Pearson-product Correlation Matrix of Outcome Measures at Posttreatment

<table>
<thead>
<tr>
<th></th>
<th>BDI</th>
<th>PCL</th>
<th>MPA</th>
<th>TD</th>
<th>ED</th>
<th>MI</th>
<th>EMG</th>
<th>Tender</th>
<th>Max</th>
<th>Max/Pain</th>
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</thead>
<tbody>
<tr>
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<td>-</td>
<td>.61**</td>
<td>.60**</td>
<td>.32*</td>
<td>.45*</td>
<td>.25</td>
<td>.25</td>
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<td>-.21</td>
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<tr>
<td>PCL</td>
<td></td>
<td>1.0</td>
<td>-</td>
<td>-.61**</td>
<td>-.59**</td>
<td>-.30*</td>
<td>-.36*</td>
<td>-.02</td>
<td>-.24</td>
<td>.09</td>
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<td></td>
<td>.96***</td>
<td>.53**</td>
<td>.66**</td>
<td>.21</td>
<td>.16</td>
<td>-.09</td>
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<tr>
<td>TD</td>
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<td>-</td>
<td>.40**</td>
<td>.57**</td>
<td>.19</td>
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<td>-.12</td>
</tr>
<tr>
<td>ED</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.0</td>
<td>-</td>
<td>.55**</td>
<td>.30*</td>
<td>.36*</td>
<td>-.06</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.0</td>
<td>-</td>
<td>.32*</td>
<td>.24</td>
<td>-.19</td>
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<tr>
<td>EMG</td>
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<td>1.0</td>
<td></td>
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<td>.14</td>
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<tr>
<td>Tender</td>
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<td></td>
<td>1.0</td>
<td>-</td>
<td>.08</td>
</tr>
<tr>
<td>Max</td>
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<td></td>
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<td></td>
<td></td>
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</table>

Note. BDI = Beck Depression Inventory; PCL = Psychosomatic Checklist; MPA = Myofascial Pain Activity; TD = Total Duration; ED = Elevated Duration; MI = Mean Intensity; EMG = Electromyographic activity; Tender = Number of muscle area tender to palpitation; Max = Maximal Jaw Opening regardless of discomfort; Max/pain = Maximal jaw opening without discomfort.

Note. *p < .05; **p < .01; ***p < .001.
$r = -.41, p = .009$. Pretreatment maximal jaw opening without discomfort was inversely related with improvement in maximal jaw opening without discomfort, $r = -.57, p = .001$. The frequency of psychosomatic symptoms was inversely related with improvement in number of areas tender to palpitation, $r = .46, p = .004$ (higher scores on psychosomatic checklist represent lower incidence of psychosomatic symptoms). Finally, number of months experiencing myofascial pain problems was positively associated with improvement in the number of muscle areas tender to palpitation, $r = .41, p = .01$. The Self-Control Schedule, Beck Depression Inventory, and other various biographic/demographic characteristics were not found to be related to MPDS symptom improvement.

**Discussion**

The belief that psychological stress is the major etiological component of MPDS has served as the basis for the increased usage of psychological treatment strategies. Previous research has primarily focused on the use of EMG biofeedback-assisted relaxation training and the results, though preliminary, appear promising. Little research has been directed toward determining whether other psychological stress-management techniques in conjunction with biofeedback or alone may be effective in the treatment of MPDS. This possibility is the primary focus of the present study. A standard outcome methodology comparing biofeedback plus stress-coping training to stress-coping training only and biofeedback
only treatment conditions was utilized. A fourth treatment group consisting of a pseudo-biofeedback condition plus stress-coping training was included to control for the non-specific effects of biofeedback as well as the greater therapy contact time inherent in a multifaceted treatment regimen.

The results of the present study indicate that all four groups led to significant reductions in selective symptoms of MPDS. The data indicate that at the conclusion of treatment all groups showed significant reductions on the four component measures of self-reported pain (myofascial pain activity, total duration, elevated duration, mean intensity), number of muscle areas tender to palpitation, and frequency of psychosomatic symptoms. Overall, there is little evidence to unequivocally demonstrate that any one treatment is more efficacious than another. Only on the mean intensity component measure of myofascial pain were significant treatment differences obtained. A posteriori paired comparisons using the Newman-Keuls procedure indicated that the biofeedback plus stress-coping group reported significantly greater reductions in mean intensity than the biofeedback only group, but none of the other treatment groups differed significantly from one another.

Collectively, the results of this study provide further support for the use of psychological stress-coping treatment strategies in the management and treatment of MPDS since all
treatment groups led to significant reductions in symptomatology. The results are especially encouraging since the patients used in the study had suffered from MPDS of long-standing duration which had failed to respond to previous traditional treatments.

Of particular interest in the present study is the finding that the stress-coping only treatment condition is as effective as the other treatment conditions in significantly reducing MPDS symptomatology. This treatment strategy is certainly cost-effective since it requires less therapy contact time and does not require expensive biofeedback apparatus. The treatment is designed to teach patients to identify cognitive and behavioral responses to stress and to employ cognitive coping skills. Unlike Stenn et al.'s (1977) study, the treatment is not confounded by relaxation training. Thus, to this author's knowledge, this is the first controlled study to indicate that a "preventive" treatment approach aimed toward providing general skills for coping with psychological stress is effective, in and of itself, in the treatment of MPDS. The finding also suggests that biofeedback and/or relaxation training is not an indispensible or even necessary component in the treatment of MPDS. Results from the present study support the pioneering work by Holroyd and his co-workers (Holroyd & Andrasik, 1978; Holroyd et al., 1977) who successfully utilized a cognitively oriented stress-coping approach to tension headaches. Additionally, results
from the present study add to a growing body of evidence (Beck, 1976; Goldfried, 1979; Turk & Genest, 1979) supporting the effectiveness of therapeutic procedures designed to alter patients' cognitions in the treatment of anxiety and stress-related disorders.

Besides pain and muscle tenderness, limitation of jaw opening and joint sounds have also been defined as cardinal symptoms of MPDS (Scott & Gregg, 1980). Yet, none of the treatment conditions in the present study had a significant effect on these symptoms (i.e., maximal jaw opening, maximal jaw opening without discomfort, joint sounds). The results of the present study, however, are consistent with previous research on the treatment of MPDS (e.g., Dohrmann & Laskin, 1978; Greene & Laskin, 1972) which has also failed to obtain significant treatment effects on these symptoms of MPDS. Inspection of Table 6 reveals that limitation of jaw opening outcome measures are not correlated with pain and muscle tenderness measures. One possible interpretation of the finding is that pain and muscle tenderness are the most common complaints of individuals experiencing MPDS and thus, as in the present study, reported by all patients. On the other hand, limitation of jaw opening and joint sounds are not reported by all subjects and may only be present in severe cases of MPDS or in patients who display particular structural characteristics (e.g., malocclusion). For example, only 69% of the subjects in the present study had joint
sounds. If limitation of jaw opening was not a symptom for all the subjects then the great variability in the obtained measurements which occurred in this study should be expected. Such a great variability in the scores, however, only serves to reduce the chances of obtaining significant treatment effects. In any case, limitation of jaw opening and presence of joint sounds do not appear to be effective measurements for assessing the outcome of treatment of MPDS.

In summary, the results of this study provide strong support for the use of stress-coping treatment strategies in the treatment of MPDS. Previous investigations have primarily focused on the use of biofeedback therapy and have found it to be effective in the management of MPDS symptoms. Results from the present study provide support for the use of cognitively oriented stress-coping techniques whether in conjunction with biofeedback therapy or alone in the treatment of MPDS. Results also suggest that biofeedback/relaxation training is not an indispensible or even necessary component of treatment since stress-coping training that did not rely on any feedback training was as effective as those that did. The finding is of importance since such a coping skills intervention is a more readily available, cost-effective treatment than biofeedback training.

Overall, the results from this study appear to have implications for a growing body of research on the behavioral treatment of stress-related pain syndromes such as MPDS.
muscle contraction and migraine headaches. Turk et al., (1979) argue that to successfully treat pain syndromes a therapeutic regimen that addresses an individual's response to psychological stress needs to be combined with treatments that focus on the individual's maladaptive physiological responding. In the present study, the biofeedback plus stress-coping training treatment condition represents such a combined therapeutic regimen. The results from the present study indicate that such a multifaceted treatment regimen is efficacious in the treatment of MPDS. However, as mentioned, there is little evidence to indicate that the biofeedback plus stress-coping treatment is more efficacious than the other treatment groups. Thus, results from the present study do not support Stenn et al.'s (1979) finding that a combined treatment of cognitive-behavior therapy with biofeedback is superior to cognitive-behavior therapy alone. Results from the present study are consistent with results from other studies that have compared the combination of cognitive therapy and relaxation training procedures with cognitive procedures alone. These studies have found combined treatments to be less or equally effective to cognitive interventions (Holroyd, 1976; Holroyd & Andrasik, 1978; Osarchuk, 1977) but they have not been shown to be more effective than cognitive interventions alone. A number of investigators have explained these findings by assuming that patients are unable to master both of these techniques in the brief
treatment time typically allowed in these studies (Goldfried, 1977; Meichenbaum, Gilmore, & Fedoravicious, 1971). Yet, in the present study participants in the treatment conditions which received veridical biofeedback training did show significant reductions in masseter EMG level following treatment. The finding suggests that these individuals were quite effective in mastering the relaxation training procedure.

Masseter electromyographic activity was not significantly associated with MPDS symptom prior to treatment, and reductions in masseter electromyographic activity were not correlated with myofascial pain activity improvement following treatment. This weak association between resting levels of masseter electromyographic activity and myofascial pain activity has been found in a number of recent studies on the treatment of MPDS (Dohrmann & Laskin, 1978; Carlsson & Gale, 1977; Stenn et al., 1979). Thus, the exact relationship between masseter muscle tension and MPDS remains unclear. The present findings, however, add to a growing body of evidence (e.g., Andrasik & Holroyd, 1980) that suggests that the learned reduction of EMG activity may play only a minor role in outcomes obtained with biofeedback. Yet, it does seem likely that muscle contraction responses to specific stressful situations might contribute to MPDS even though these responses might not be elicited in the relaxed laboratory assessment situation. Therefore, research on MPDS would probably benefit from the development of methods for
assessing such responses to laboratory as well as to real-life stressors.

The finding that biofeedback plus stress-coping, pseudo-biofeedback plus stress-coping, stress-coping only and biofeedback only treatments yielded similar outcomes that were not statistically significant from one another suggests that elements common to these interventions may have accounted for the results. For example, the provision of a causal explanation for distressing symptoms may have served to increase subjects' belief in their ability to cope with their symptoms (Frank, 1974; Murray & Jacobson, 1978). Although in the present study explanations of the therapeutic process were slightly different in each of the treatment groups, they all emphasized that subjects could master their symptoms. Thus, subjects were informed that cognitive distortions or masseter muscle contraction responses eliciting myofascial pain could be combatted by engaging in specific coping responses or by understanding their historical antecedents, which were no longer present and therefore need not influence current responses. Such explanations likely increase individuals' belief in their ability to cope with previously debilitating symptoms and might be expected to lead to a greater initiation and persistence of coping behavior (Bandura, 1977).

Similarly, several researchers (e.g., Holroyd & Andrasik, 1978; Meichenbaum, 1976; Turk et al., 1979) suggests that cognitive therapeutic approaches as well as EMG biofeedback
relaxation training may bring relief from pain syndromes by producing alterations in person-environment interactions which improve an individual's ability to cope with stress. Consequently, interventions that directly train flexible coping skills and focus on person-environment interactions, such as the stress-coping training regimen in the present study, might be expected to be more effective than a procedure that indirectly trains a single coping skill (i.e., reduced masseter EMG). Clearly, further research will be required to assess the extent to which patients' coping efforts are related to alleviation of myofascial pain. As with most research efforts that focus on the outcome of treatment, the present study is not able to discern these variables since the active components within each procedure is not known.

Appropriate statistical and research methodology dictates that treatment differences exceed chance probability to be considered statistically significant. In the present study, numerically large differences exist between the biofeedback only group and the other treatment groups with regard to the experience of pain at the conclusion of treatment. As mentioned, except on the mean intensity pain measure, these differences are not statistically significant. Yet, the author cannot help but be impressed by the obtained differences. In treatment outcome research a distinction is often made between
statistical and clinical significance. For example, pain changes that attain statistical significance may have only slight effects on the patient's functioning and thus be of questionable clinical significance. Clinically significant symptom changes imply that the changes are of functional utility for patient.

In the present study, the distinction between clinical versus statistical significance may be entertained from two perspectives: (a) Are the statistically significant pain reductions reported by all four groups of clinical significance? (b) Are the numerically large, but statistically non-significant differences between treatment groups of clinical significance? To examine these issues, individual improvement scores were tabulated for all subjects for the myofascial pain activity measure at posttreatment. Improvement scores were computed as previously defined. These data are summarized in Table 9. Positive scores indicate improvement and are assigned by intervals from 0% to 100% improvement. As can be seen in Table 9, the majority of subjects in each of the four groups show improvement scores of 50% or better. Such reductions in pain activity are expected to have impact upon the subject's functioning and thus the obtained statistically significant pain reductions appear clinically relevant.
Table 9

Distribution of Individual Subject Improvement Scores for Myofascial Pain Activity at Posttreatment

<table>
<thead>
<tr>
<th>Degree of Improvement</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BFSC</td>
</tr>
<tr>
<td>90-100</td>
<td>4</td>
</tr>
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<td>80-89</td>
<td>2</td>
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<tr>
<td>70-79</td>
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<td>60-69</td>
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<td>50-59</td>
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<tr>
<td>10-19</td>
<td>0</td>
</tr>
<tr>
<td>0-9</td>
<td>0</td>
</tr>
</tbody>
</table>

Note. BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback and stress-coping; BF = Biofeedback only; SC = Stress-coping only.

Further inspection of Table 9, however, also reveals that subjects in the treatment conditions which provided stress-coping training either alone or in conjunction with biofeedback or pseudo-biofeedback are, in comparison to subjects in the biofeedback only group, more likely to obtain improvement scores approaching a myofascial pain free condition. For example, 75% of the subjects in the biofeedback plus
stress-coping group, 50% of the subjects in the stress-coping only group, and 38% of the subjects in the pseudo-biofeedback plus stress-coping group show improvement rates of 80% or better. In comparison, none of the subjects in the biofeedback only group show such improvement in their pain symptomology. In general, subjects in the biofeedback only group show less promising improvement in myofascial pain activity, with improvement scores ranging from 0% to 79%. Additionally, differences between treatment groups are suggested by the patients' ratings of the outcome of treatment. Fifty percent of the patients in the biofeedback plus stress-coping group and 37.5% of the patients in each of the stress-coping only and pseudo-biofeedback plus stress-coping groups rated themselves as symptom free at the conclusion of treatment. In comparison, only one patient (12.5%) in the biofeedback only group rated herself as symptom free.

Thus, while there is a lack of statistically significant treatment differences, individual improvement score analyses and patients' ratings of the outcome of treatment suggest treatment differences that may be clinically relevant. The pattern of findings suggests that the biofeedback only treatment may be less efficacious than the other treatment conditions in reducing and ameliorating the pain symptoms of MPDS. Considering the limited number of subjects used in each group, it may be best to view the present research as a pilot study since exceedingly large differences between groups
would have been required in order to attain an acceptable degree of statistical significance. It would be highly desirable to replicate this study using a larger sample size. If the magnitude of the differences obtained in the present study could be replicated in larger samples, then potentially important clinical results may also attain statistical significance.

Overall, this study presents several distinct improvements over previous research, yet several inadequacies exist which limit the findings. Foremost, is the lack of follow-up data. The present study only reports pre- to posttreatment effects occurring over an approximate nine-week interval. Ephemeral effects are of little interest to clinicians whose interests is in stable change. Reasonable stability of change can be judged only after a minimum follow-up of six months. The author appreciates this methodological mandate and currently a nine-month follow-up of patients is being conducted. While not presented in this study, follow-up data will be analyzed and reported before submission for professional publication.

A lack of effective control procedures is another inadequacy in this study. All of the treatment conditions provided participants with active strategies designed to enable them to reduce stress in their lives. Because of ethical considerations and time constraints, the study did not utilize a waiting-list or attention placebo control group. Two sources
of rival hypotheses that can be offered to explain the efficacy of the treatment strategies utilized in the present study are regression-to-the-mean and placebo effects. Miller (1974) argues that pain patients are much more likely to seek treatment when they are feeling worse. Since physiological systems tend to fluctuate between periods of exacerbation and those of amelioration, it is possible to obtain a sample of volunteers whose pain will show reduction due to spontaneous fluctuation, regressing toward the mean level. The point also underscores the necessity of incorporating extended follow-up periods.

Placebo effects are potent factors in any treatment of pain and are especially potent in an impressive treatment such as biofeedback, with its complex mechanical equipment. Yet, it must be considered that this study only included patients who had suffered from MPDS of long-standing duration which had been refractory to traditional treatments. It could be argued that such treatment-resistant patients are, in all probability, also "placebo-resistant" and that treatment effects are unlikely to only represent regression to the mean. On the other hand, it is possible that the rationale and credibility of the present treatments affected patients' expectancy and consequent response to treatment in a more favorable way than previous traditional treatments. Unfortunately, there are no data to support or refute this. In any case, future research should utilize attention-placebo and/or waiting-list control groups.
An attempt was made in the present study to improve upon the evaluation of the outcome of treatments for MPDS. In this regard, multiple methods, including objective and subjective measures, were used to assess various aspects of the MPDS experience. The dentists' examination of the signs and symptoms of MPDS served as a reliable, objective measure of outcome. The self-monitoring pain diary (SMPD) represented a more intense effort to evaluate patients for self-reported pain in the natural environment. Patients were required to report the intensity of their pain for every waking hour during the length of the study. Compliance was excellent and allowed for the evaluation of the pain experience along several dimensions (i.e., total pain activity, duration, elevated duration, mean intensity). Other aspects of the MPDS experience such as depression and the frequency of psychosomatic symptoms were also assessed via self-report inventories. The significant treatments effects obtained on these measures suggests that treatment effects generalized to other stressors.

Another manifestation of pain behavior is analgesic medication usage. In the present study, no attempt was made to control the analgesic regimen of the patients in order to isolate treatment effectiveness. At attempt was made via use of the SMPD to have patients record their daily medication consumption. However, compliance was so variable across subjects that a statistical treatment of this data was not
attempted. Consequently, the impact of the present treatments upon pain symptoms may be confounded by concurrent medication usage. Future research should attempt to avoid this possible confound by either utilizing patients who are willing to discontinue pain medications during the course of the study or by more vigorously obtaining data on this factor and submitting it for appropriate statistical treatment.

Finally, an attempt was made in this study to identify variables which predict specific treatment outcome. The results are somewhat disappointing since few meaningful relationships were found. The results of these analyses, however, do not support Gessel's (1975) observation that MPDS patients who are older, more depressed, and have had their pains for a relatively longer period are least successful with psychological treatment strategies. Indeed, in the present study, patients who had myofascial pain problems for a longer period of time were more likely to show greater improvements in the number of muscle areas tender to palpitation.

Considering the variety of physical, psychological, and medical factors that may play an etiological role in MPDS, rather than seeking to determine whether a particular treatment approach is effective, future research should direct its efforts toward identifying subject characteristics that predict specific outcome, given particular treatments. It is recommended that wide ranging predictor batteries which include demographic, psychological, physiological and
medical/structural data be utilized in future studies on the treatment of MPDS. Furthermore, if future studies are able to utilize larger sample sizes, as recommended, this will allow for the use of statistical techniques such as multiple discriminant analysis which may be helpful in identifying variables predictive of outcome.

In conclusion, the results of this study provide strong support for the use of cognitively oriented stress-coping treatment strategies in the treatment of MPDS. It is hoped that this particular investigation will serve as a stimulus for further refinements and improvements in research on the treatment of MPDS.
Appendix A

INFORMED CONSENT AGREEMENT (H.R. #1,428)

I, ____________________________, do hereby consent to participate in an experimental research project conducted by Mr. Lewis R. Waid, aimed at helping me to cope with the symptoms and problems associated with myofascial-pain dysfunction syndrome. Mr. Lewis R. Waid has explained orally to me and I have read and fully understand the attached description of this investigational treatment program. Additionally, I realize that this research project does not involve either medical or dental treatment.

I fully understand the following:

I understand that my participation in the investigational treatment program is contingent upon my undergoing a clinical examination at the MUSC Dental School Clinic to determine if the history and signs and symptoms of my pain syndrome are appropriate for the experimental treatment procedures. That if I do qualify for the research project that I will be asked to complete several forms, attend ten (10) individual treatment sessions (2 per week), practice certain procedures taught to me, and retain some daily records. Furthermore, I understand that each treatment session will be divided into two halves: The first half of each session will take place in a psychophysiological laboratory where tension in my masseter muscles will be monitored and measured and I will be provided specific skills that should enable me to relax and thus reduce the level of muscle tension. The second half of each session will be utilized to identify specific stress producing situations in my life and to learn new and perhaps more effective ways to handle and thus cope with such situations. I also understand that at the completion of treatment I will once again undergo a clinical examination at the MUSC Dental School Clinic to reassess the signs and symptoms of my pain syndrome. Furthermore, I understand that while this treatment program may have therapeutic effects, no guarantee of this is made.

The possibility of discomfort and risk that might arise in the procedure: Some people may experience some temporary discomfort/pain when undergoing the clinical examination and/or practicing some of the techniques.

Benefits: This program may help to ameliorate or modify some of the pain or discomfort which I presently experience.
Additionally, the program may assist me to cope more adaptively with stress and/or muscle tension in my daily life.

The myofascial pain patients involved in this study have all received previous alternative conservative treatments such as soft diet, physiotherapy, occlusal splint, muscle relaxants, etc. The results of these previous efforts have not been completely successful.

Mr. Lewis R. Waid has agreed to answer any inquiries that I may have concerning the procedures and has informed me that I might also contact the Medical University of South Carolina Institutional Review Board for Human Research (803-792-4148) directly. This Board administers the agreement with the United States Department of Health and Human Services covering protection of human subjects.

I understand that in the event of physical injury resulting from the research procedures to the participant, reasonable medical treatment is available free through the Medical University of South Carolina (or Veterans Administration Medical Center, if applicable). Financial compensation is not available for medical treatment elsewhere, loss of work, or other expenses.

I understand that if I am a patient at the Veterans Administration Medical Center that my participation in this study will be kept confidential.

I also understand that I am free to withdraw my consent and discontinue participation at any time. Discontinuation will in no way jeopardize my ability to receive treatment now or in the future at this Institution.

PERSON OBTAINING CONSENT

SIGNATURE OF PARTICIPANT

WITNESS

WITNESS

DATE
Appendix B

INFORMED CONSENT AGREEMENT (H.R. #1,428)

I, ____________________________, do hereby consent to participate in an experimental research project conducted by Mr. Lewis R. Waid, aimed at helping me to cope with the symptoms and problems associated with myofascial-pain dysfunction syndrome. Mr. Lewis R. Waid has explained orally to me and I have read and fully understand the attached description of this investigational treatment program. Additionally, I realize that this research project does not involve either medical or dental treatment.

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relaxants, etc. The results of these previous efforts have not been completely successful.

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I understand that in the event of physical injury resulting from the research procedures to the participant, reasonable medical treatment is available free through the Medical University of South Carolina (or Veterans Administration Medical Center, if applicable). Financial compensation is not available for medical treatment elsewhere, loss of work, or other expenses.

I understand that if I am a patient at the Veterans Administration Medical Center that my participation in this study will be kept confidential.

I also understand that I am free to withdraw my consent and discontinue participation at any time. Discontinuation will in no way jeopardize my ability to receive treatment now or in the future at this Institution.

______________________________    ________________________________
PERSON OBTAINING CONSENT        SIGNATURE OF PARTICIPANT

______________________________    ________________________________
WITNESS                        WITNESS

______________________________
DATE
Appendix C

INFORMED CONSENT AGREEMENT (H.R. #1,428)

I, ____________________________, do hereby consent to participate in an experimental research project conducted by Mr. Lewis R. Waid, aimed at helping me to cope with the symptoms and problems associated with myofascial-pain dysfunction syndrome. Mr. Lewis R. Waid has explained orally to me and I have read and fully understand the attached description of this investigational treatment program. Additionally, I realize that this research project does not involve either medical or dental treatment.

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I understand that my participation in the investigational treatment program is contingent upon my undergoing a clinical examination at the MUSC Dental School Clinic to determine if the history and sign and symptoms of my pain syndrome are appropriate for the experimental treatment procedures. That if I do qualify for the research project that I will be asked to complete several forms, attend ten (10) individual treatment sessions (2 per week), practice certain procedures taught to me, and retain some daily records. Furthermore, I understand that each treatment session will take place in a psychophysiological laboratory where tension in my masseter muscles will be monitored and I will be provided specific skills that should enable me to relax and thus reduce the level of muscle tension. I also understand that at the completion of treatment I will once again undergo a clinical examination at the MUSC Dental School Clinic to reassess the signs and symptoms of my pain syndrome. Furthermore, I understand that while this treatment program may have therapeutic effects, no guarantee of this is made.

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Benefits: This program may help to ameliorate or modify some of the pain or discomfort which I presently experience. Additionally, the program may assist me to cope more adaptively with stress and/or muscle tension in my daily life.
The myofascial pain patients involved in this study have all received previous alternative conservative treatments such as soft diet, physiotherapy, occlusal splint, muscle relaxants, etc. The results of these previous efforts have not been completely successful.

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I understand that if I am a patient at the Veterans Administration Medical Center that my participation in this study will be kept confidential.

I also understand that I am free to withdraw my consent and discontinue participation at any time. Discontinuation will in no way jeopardize my ability to receive treatment now or in the future at this Institution.

__________________________________________
PERSON OBTAINING CONSENT

__________________________________________
SIGNATURE OF PARTICIPANT

__________________________________________
WITNESS

__________________________________________
WITNESS

______________________________
DATE
Appendix D

DEMOGRAPHIC DATA SHEET

Name ________________________________  Sex: (1) Male ____

Address ______________________________  Female ____

___________________________  Phone # _____________

Age _____  Martial Status: (1) Married ____  (2) Single ____

(3) Divorced or Separated ____  (4) Widow ____

Race:  White ____  Black ____  Other: ____________________

Number of years of education _______________________________

Occupation:  ____  (1) Higher executives, major professionals

____  (2) Business managers, lesser professionals

____  (3) Administrative personnel, minor professional

____  (4) Clerical and sales worker, technicians

____  (5) Skilled manual employees

____  (6) Machine operators and semi-skilled employees

____  (7) Unskilled employees

____  (8) Unemployed

Specific Type of job ______________________________

Referred from:  Dental School ________________

VAMC __________________

Private __________________

Other ____________________
Appendix E

MPDS INTERVIEW

Name __________________________ Date __________

Subject's description of pain (nature and location) __________________________

________________________________________________________________________

________________________________________________________________________

For how long? __________________________

Pain in right, left, or both sides of face? __________________________

Which side is pain greatest? __________________________

Does pain come and go, or constant? __________________________

Is it worse in morning or night? __________________________

Does subject grind teeth at night? __________________________

Does subject "clench" teeth in daytime? __________________________

Has subject had previous treatments for this condition (describe)? __________________________

________________________________________________________________________

Current medication? __________________________

________________________________________________________________________

Any history of physical trauma to head or neck? __________________________
Appendix F

Clinical Examination

Name_________________________ Date________________

Sex _____ Race _____ Age _____ Date of Birth _________

1. Chief Complaint____________________

2. TMJ
   a. Tenderness to palpation (+ or -)
      1. Pre auricular
      2. Intra auricular
   b. Noises (click, pop, crepitus)
   c. Opening to first pain:
      maximal:
   d. Deviation on opening and closing (draw and label)
      Right ___________ Left ___________
      Back ___________ mm
      Forward ___________
   e. Movements producing pain____________________
   f. Any restriction or locking jaw movements________________________

3. Muscle Tenderness (Mark area with X and circle most severe)


4. Bruxism, clenching or parafunctional habits YES NO
   When most noticeable _________________________
5. _____ Is there or ____ was there TMJ pain  YES : NO
   When is/was it most noticeable or severe__________

6. _____ Is there or ____ was there any muscle spasm  YES  NO
   When is/was it most noticeable or severe__________

7. Has there been any discomfort related to your bite  YES  NO
   When ________________________________

8. _____ Is there or ____ was there any chronic headache  YES  NO
   When is/was it most noticeable or severe__________

9. Any arthritis or rheumatism elsewhere in the body  YES  NO
   Where____________________________________

10. Any systemic disorders  YES  NO
    If so, list________________________________

11. Previous treatment________________________________

12. Types of occlusion: Cuspid protected _____ Group
    Function _____  Other _____

13. Angles classification: Class I ____  Class II ____
    Class III ____

14. Occlusal analysis:
   a. Overlap of anteriors: Horizontal___mm
      Vertical_______mm
   b. Altered vertical dimension ______________________
   c. First tooth contact in centric relation __________
   d. Amount and direction of discrepancy to max contact:
      __________mm
Appendix F—Continued

e. Posterior contacts:
(1) Protrusive
(2) Working
(3) Balancing

15. Teeth (erosion, facets, sensitivity)
Appendix G

SELF-MONITORING PAIN DIARY

DIRECTIONS (make entries for each hour)

Column 1: Degree of Myofascial Pain (list one)

0 = No soreness, pain or ache.
1 = Some slight stiffness or soreness, tender, but no pain or ache.
2 = Slight, dull ache or pain; able to ignore and in no way limits activities.
3 = Moderate, dull ache or pain; not able to ignore; disconcerting; but does not limit jaw movement.
4 = Piercing pain or ache exacerbated by use of jaw; impossible to ignore.
5 = Severe fascial pain radiating to other areas of the head; activities quite limited.
6 = Intense fascial pain radiating to other areas; incapacitating; may feel swollen; jaw movements nearly impossible.

Column 2: Medication (list as many as necessary)

A = ______, dosage____ B = ______, dosage____
C = ______, dosage____ D = ______, dosage____
E = ______, dosage____ F = ______, dosage____

Column 3: Mood (list all those that apply)

z = depressed, sad, "blue"  y = happy, joyful
x = angry, resentful, annoyed  "up"
v = fearful, afraid  w = pleased, contented
s = tense, anxious  t = romantic, sexy
q = rested, active, alert  r = tired, sleepy

Column 4: Place (list as many as apply)

1 = home  2 = work/school  3 = neighbor/friend's
4 = outside  5 = restaurant/store home
6 = car, bus, place, etc  7 = recreational area
8 = ______
# SELF-MONITORING PAIN DIARY

Week ____________________  Date ____________________

Name ____________________

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<tr>
<th>Col.#</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Activities and thoughts correlated with mood change</th>
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Appendix H

Expectancy/Credibility Assessment Device

(1) If you had a friend who suffers from myofascial pain like yourself, what is the likelihood that you would recommend that he undergo the present treatment?

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<th>1</th>
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<tbody>
<tr>
<td>Definitely would not recommend</td>
<td>Definitely would recommend</td>
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</table>

(2) After hearing the rationale behind this treatment program, how important do you believe it is that this treatment program be made available to other myofascial pain sufferers?

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<th>5</th>
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<td>Not Important</td>
<td>Very Important</td>
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</table>

(3) How much therapeutic change do you expect to receive from participating in this treatment program?

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<th>7</th>
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<tbody>
<tr>
<td>No change whatsoever</td>
<td>Slight improvement</td>
<td>Significant improvement</td>
<td>Total remission of symptoms</td>
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Appendix I

Manual of Stress-Coping Procedures

The cognitive coping strategies that will be utilized in the present study are adapted from Goldfried's (1979) systematic rational restructuring techniques, Meichenbaum's (1977) self-instructional training, and Turk's (1977) imagery-coping strategies. The specific therapeutic guidelines of these strategies are described below.

Rational restructuring. The goal of cognitive restructuring as a coping skills intervention is to provide clients themselves with the ability to adopt a more reasonable perspective on potentially upsetting events. The following four steps are involved in this procedure: (a) presentation of rationale; (b) overview of irrational assumptions; (c) analysis of subjects' unrealistic self-statements which mediate their maladaptive emotions; (d) helping subjects to modify their unrealistic self-statements.

1. Presentation of rationale. The therapists's goal is to help subjects recognize the general assumption that many maladaptive feelings are the result of what we tell ourselves about situations, rather than the situations themselves. Subjects will be provided a treatment rationale that emphasizes that MRS results from stress and that stress reactions are directly influenced by our labels, expectations, and self statements. In discussion with the subject, concrete examples
will be provided that illustrate that differential emotional reactions can be attributed to differing self-statements.

In describing and illustrating the basic rationale, the therapist should point out that in many situations one may not literally "tell oneself" things which lead to emotional upset. However, because of the overlearned nature of our association with certain situations, our self-statements may have reached the point where the labeling process is more or less automatic. The goal here is to have subjects accept the premise that, even though they may not deliberately tell themselves certain things prior to or during emotionally upsetting situations, they nonetheless react disproportionately, as if they view the situation in a given way.

Through this initial step (Session 1), the therapist will describe the significance of self-statements in a fairly general way without relating it to the subject's particular case. It is important to have the subject agree with what has been described thus far before carrying the procedure any further.

2. Overview of irrational assumptions. The goal of this step is to have subjects acknowledge the irrational or unrealistic nature of a series of beliefs that individuals frequently hold. Various irrational self-statements that typically appear in clinical cases will be presented (e.g., the expectation that it is essential to receive approval and
love from others in order to have any feelings of self-worth, the notion that perfection is required in all accomplishments in order to see oneself as anything but a failure). The therapist's task is to play the devil's advocate, that is, instead of trying verbally to convince subjects that these thoughts are irrational, an attempt will be made to have the individual arrive at this viewpoint by offering his own arguments regarding the unreasonableness of each of these beliefs. Social psychology literature suggests that such an approach is more likely to facilitate attitude change (Brehm & Cohen, 1962). Again, through this step everything continues to be discussed on an objective basis, by minimizing personal involvement it makes it easier for the client to generate believeable counterarguments.

3. Analysis of the subject's unrealistic self-statements which mediate his/her maladaptive emotions. The therapeutic task, at this point, is to identify and review the specific situations in which the individual's own problems arise and then, in Socratic-like fashion, have him/her come to the conclusions that his/her maladaptive feelings are mediated by irrational self-statements and unrealistic expectations.

The irrationality of a subject's self-statements will be analyzed at two levels: (a) the likelihood that the subject is correctly interpreting the situation, and (b) the ultimate implication of the way in which the subject has labeled the event. As an example, consider the case of young male
who reacts with disproportionate emotional upset when unsuccessful in obtaining a date with a particular female. His interpretation of the event is that "she does not like me." At the first level of analysis, one could examine the extent to which this belief is rational from a probabilistic point of view. That is, were there any other reasons which may have determined her refusal. The second-level analysis can be approached as follows: "Let's assume for a moment that she really does not like you. Why should this make you so upset?" The goal of such a series of questions is to lead the subject to recognize that his emotional overreaction is being mediated by one or more held irrational beliefs (e.g., everybody must love me).

Some subjects may find it difficult to identify their feelings and thoughts. In such cases, it may be useful to employ imagery techniques. Subjects will be asked to close their eyes and picture as vividly as they can the situation they find troublesome. Once they do this, they will be asked to also picture how they felt at that time and to determine what they are thinking. In essence, they will be instructed to "think aloud" so the therapist can prompt and otherwise assist them in identifying cognitions that contribute to their emotional reactions.

4. Helping subjects to modify their unrealistic self-statements. The goal, during this step, is to have the individual take what he/she knows in "theory" and put it into
actual practice. The subject is instructed to utilize his/her emotional reaction as a signal or cue for him/her to stop, think, and ferret out what he/she is telling himself/herself about the situation which may be responsible for this upset. In the process of breaking up what heretofore may have been an automatic reaction, the subject will become more aware of his inappropriate self-statement, and then may proceed to reevaluate this belief more rationally. The irrational self-statement is then replaced with more of a realistic appraisal of the situation and note is taken of the resulting decrement in stress.

To provide subjects with the opportunity to reevaluate their stress producing self-statements rationally, behavioral rehearsal via overt role playing or in imagination will be employed. Subjects will be instructed to "think aloud" so that the therapist can prompt and assist them in evaluating and modifying unrealistic assumptions. In addition to the practice received during treatment sessions, subjects will be assigned homework, utilizing the SMPD, to identify stress-provoking situations and how they utilized cognitive restructuring in vivo.

Self-Instructional training. This cognitive coping strategy, adapted from the work of Meichenbaum and his co-workers (1977), will be utilized to augment training in cognitive restructuring. The procedures are designed to help subjects in the generation of sets of coping self-statements
which assess the reality of the situation, control negative thoughts, help to "psych up" the subject to confront a stressful situation, and be self-reinforcing. In brief, the purpose of self-instructional training is to have participants develop a problem-solving, task-oriented set when confronting a stressful situation.

Rationale. Self-instructional training will sequentially follow training in cognitive restructuring. The following rationale will be provided: "Research has suggested that one of the important points in becoming more skilled in handling stress is to have available a number of alternative strategies that can be used and to be able to shift among them when the situation warrants. So far you have been learning how to modify the unrealistic expectations and self-statements which mediate stressful reactions, yet we all from time to time face stressful situations that appear so overwhelming that it is difficult to know where to start in order to manage them. We have found that when a stressful situation is divided into components or stages people are better able to keep the situation manageable, that is, keep things from getting out of control. Thus, by breaking the stressful situation into its elements one has an opportunity to deal with each of these stages one at a time. We have identified four stages in any stressful situation: (a) a preparatory stage; (b) a confrontation stage; (c) critical moments; and (d) self-evaluation."
Training. Subjects will identify stressful experiences or problem situations and be instructed to view the stress reaction as a series of four stages, rather than as one massive panic attack. Through discussions with therapist, modeling, and behavioral rehearsal, subjects will learn to emit positive coping self-statements during each phase in order to "make it through the stressor." While the statements listed for each phase below are geared toward anger provocation (Novaco, 1977) similar kinds of statements can be used for other kinds of stressful situations.

Phase 1 - Preparing for the Stressor
This could be rough, but I know how to deal with it.
I can work out a plan to handle this.
Easy does it.
Remember, stick to the issues and don't take it personally.
Don't worry; worrying won't help anything.

Phase 2 - Confronting and Handling the Stressor
You can meet the challenge.
As long as I keep my cool, I'm in control.
You don't need to prove yourself.
Don't make more out of this than you have to.
There is no point in getting mad.

Phase 3 - Coping with Feelings at Critical Moments
My anger is a signal of what I need to do.
Time for problem solving.
Time to take a deep breath.
He probably wants me to get angry, but I'm going to deal with it constructively.

Phase 4 - Reinforcing self-statements

Good, I handled that pretty well.
I could have gotten more upset that it's worth.
Shake it off, everything is going fine.
That's going a good job!

Guided-Imagery Practice. Imaginal scenes have been used extensively by cognitive-behavioral therapists for the control of many avoidant behaviors and, as in the present treatment, to make clients aware of their maladaptive thoughts, feelings and beliefs. The purpose of using imagery as a coping skill is to divert attention away from the stressful experience. Since imaginative techniques have proved quite efficacious for some individuals, it is quite possible that some practice might enhance the effects attributed to imagery coping strategies. The guided-imagery practice utilized in the present study is adapted from the work of Turk (1977).

Rationale. Guided-imagery practice will sequentially follow training in self-instruction. The following rationale will be provided: A final strategy that you may wish to utilize when dealing with stressful situations involves mental imagery. By focusing your attention on some vivid, pleasant imagery you will be able to divert yourself from attending to unpleasant feelings, thoughts and sensations. No doubt you have utilized mental imagery before in your life, for example,
you have probably had some form of mental image as you lay on the beach, daydreamed in a boring class, while waiting for a bus, etc. When we reminisce about these occasions we can see pictures in our mind's eye of the situation, the people present and who was doing what. Sometimes these pictures are so vivid, that it is almost as if we were reliving those situations. Can you recall having this type of experience?

(discussion) What I would like to do next is to have you try to imagine several scenes so you can get a feeling for what I mean?"

**Training.** Subjects will then be asked to make themselves comfortable and relaxed with their eyes closed. Subjects will then be guided through two mental fantasies. Following practice, subjects will be queried as to their involvement in the imagery technique. Further discussion will emphasize that this is a form of "imaginative inattention" that will allow them to ignore unpleasant situations, thoughts or feelings by focusing on scenes incompatible with the disturbing thoughts or sensations. The key to the technique is to make the image as vivid and as real as possible. The more vivid and the more you feel as if you are really there the more absorbing it becomes. Subjects will be informed that they should have all the details of the fantasy worked out before proceeding. Additionally, they should include all the characteristics of a scene to make it as real as possible, including sounds, smell, touch as well as vision. Subjects will then be asked to
develop a pleasant scene that would attract their attention in such a way so as to block out other sensations. They will be asked to practice utilizing this imagery technique at least four times during the coming week.
Appendix J

Treatment Rationale

The following treatment rationale was presented to subjects during the start of the initial session of stress-coping training. The content was appropriately revised for subjects who received stress-coping training only. That is, for stress-coping training only subjects no mention was made of a multifaceted treatment approach.

As we have indicated to you, your pain syndrome has shown that the most common cause of MPDS is muscle spasms produced by chronic oral habits such as the clenching or grinding of the teeth. These oral habits appear to serve as involuntary tension relieving mechanisms and thus it can be said that MPDS results, ultimately, from stress and tension. To put it another way, a three stage process is generally believed to produce myofascial pain and dysfunction: (1) The individual's response to psychological stress, which (2) may result in the prolonged use of tension relieving oral habits leading to muscle fatigue and spasm, which (3) subsequently leads to the production of myofascial pain and dysfunction.

Consequently, the present treatment program is a two-pronged attack. During the first half of each session, we hope to help you relieve the muscle fatigue and spasms by relaxing the masseter muscles. Joe Giacinto has already
talked specifically to you about how that goal will be accomplished. During this portion of treatment, I wish to work with you on the first stage of the process, that is, how you deal with stress. Specifically, my goal is to train you in specific skills which should enable you to be more effective in identifying and coping with stressful life events.

The first step in achieving this goal is for you to understand the role that thinking plays in our maladaptive feelings like anxiety, tension and depression. People often think that many of the maladaptive emotions they experience are the result of the particular situation they find themselves. However, on closer inspection it can be seen that most maladaptive feelings are the result of what we tell ourselves about situations, rather than the situations themselves. For example, let's take a situation in which a man is aiming a gun at you on a city street. Certainly, you would become very anxious and uptight. However, there is nothing intrinsically anxiety provoking about the size and shape of the gun being aimed at you. Rather, your bad feelings are the result of your expectations or thoughts that the gun might seriously hurt you. On the other hand, if another individual who had never seen or heard of a gun before found himself in the same situation, his reaction would probably be minimal. The cause of emotional reactions is not the event itself but one's interpretation of the event. Perhaps another example will help in allowing you to understand the role one's
thinking or self-statements play in maladaptive emotions. Let's say there are two different individuals who are getting ready to attend the same party. One individual is feeling very calm about the whole thing, and in fact, is looking forward to the evening ahead. His thoughts about the situation may run as follows: "It should be a fun and interesting party tonight. There will probably be a number of people there that I don't know and that will give me the chance to make some new friends. Also, there will be some people there I haven't seen in a long time, it will be great to renew some friendships." The second individual, on the other hand, is very nervous and uptight. His thoughts are, "I don't know how well I will do tonight. I hate parties where I don't know anyone. I'm not sure if I will be able to say or do the right things. I don't want to look foolish, especially since there will a lot of people there." Thus, you can see that differential emotional reactions displayed by these two individuals is attributed to their differing self-statements.

Now, you might be thinking that you really do not talk to yourself this way. Let me assure you that often, in many situations, one may not literally "tell oneself" things which lead to emotional reactions. However, this lack of awareness is probably due to the fact that many of our associations with certain situations are so overlearned that our self-statements may have reached a point where the labeling process
Appendix J—Continued

occurs more or less automatically, rather than being carefully thought out.

Thus, the goal of this portion of treatment is for you to learn how to better cope with stress by exerting more control over your thinking processes. The first step in this process will involve you identifying stressful situations. Next, we will work together in identifying the thoughts, self-statements and expectations you have prior to becoming stressed, while stressed, and subsequently. This will be achieved by having you monitor your thinking daily in an attempt to identify the activities, thoughts, and self-statements that seem to accompany changes both in your mood and pain syndrome. Remember, exacerbations in pain probably indicate that you are under significant stress.

Finally, after we have identified the types of beliefs, expectations, and self-statements that you make which often result in the experience of unwanted emotional reactions, we will train you in how to combat this. That is, you will receive training in how to employ signs of impending distress as a signal to engage in cognitive strategies incompatible with the further occurrence of stress responses. Specifically, you will receive training in how to reappraise your attitudes toward the distress-eliciting event (What am I thinking to induce my distress? What are the facts?), to emit coping self-instructions (Calm down, concentration on the present – there is no point in catastrophizing) and how to
use imagery in order to divert your attention away from the stress provoking thoughts. Are there any questions?
Table 7

Summary of Mean Difference Scores, \( t \) and \( p \) Values for the Four Myofascial Pain Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>BFSC</th>
<th>PSC</th>
<th>BF</th>
<th>SC</th>
</tr>
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<tbody>
<tr>
<td>Myofascial Pain Activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Difference(^a)</td>
<td></td>
<td>-148</td>
<td>-116</td>
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<tr>
<td>( t )(^b)</td>
<td></td>
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<td>.0006</td>
<td>.0006</td>
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<td>.0005</td>
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<tr>
<td>Total Duration</td>
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<td></td>
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<tr>
<td>Mean Difference(^a)</td>
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<td>( t )(^b)</td>
<td></td>
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<tr>
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<td>.0055</td>
<td>.004</td>
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<td>.035</td>
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<tr>
<td>Elevated Duration</td>
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<td>Mean Intensity</td>
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<tr>
<td>Mean Difference(^a)</td>
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<tr>
<td>( t )(^b)</td>
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<td>-4.26</td>
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<tr>
<td>( p )(^c)</td>
<td></td>
<td>.0015</td>
<td>.002</td>
<td>.025</td>
<td>.0005</td>
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\(^a\)Pre- to Posttreatment  
\(^b\)Degree of freedom equals 7.  
\(^c\)One-tailed.
Appendix L

Table 8
Summary of Mean Difference Scores, t and p Values for the Non-Myofascial Pain Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>BFSC</th>
<th>PSC</th>
<th>BF</th>
<th>SC</th>
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<tbody>
<tr>
<td><strong>Number of Tender Areas</strong></td>
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<td></td>
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<tr>
<td>Mean Difference&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>-3.13</td>
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</tr>
<tr>
<td>t&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>-3.15</td>
<td>-4.21</td>
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</tr>
<tr>
<td>p&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td>.016</td>
<td>.004</td>
<td>.02</td>
<td>.042</td>
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<tr>
<td><strong>Jaw Opening Without Discomfort</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td>1.15</td>
<td>4.21</td>
<td>3.65</td>
<td>-1.91</td>
</tr>
<tr>
<td>t&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>1.19</td>
<td>1.91</td>
<td>1.16</td>
<td>-1.02</td>
</tr>
<tr>
<td>p&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td>.27</td>
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<td><strong>Maximal Jaw Opening</strong></td>
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<tr>
<td>Mean Difference&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>t&lt;sup&gt;b&lt;/sup&gt;</td>
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<td>1.18</td>
<td>.84</td>
<td>.96</td>
<td>-.01</td>
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<tr>
<td>p&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td>.28</td>
<td>.43</td>
<td>.37</td>
<td>.99</td>
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<tr>
<td><strong>Masseter EMG Activity</strong></td>
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<tr>
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<td>-.746</td>
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<td>-2.64</td>
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<td>.034</td>
<td>.577</td>
<td>.0001</td>
<td>.42</td>
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<tr>
<td><strong>Psychosomatic Symptoms</strong></td>
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<tr>
<td>Mean Difference&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>2.88</td>
<td>4.25</td>
<td>3.75</td>
<td>3.0</td>
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<tr>
<td>t&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>2.76</td>
<td>2.93</td>
<td>2.35</td>
<td>2.45</td>
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<tr>
<td>p&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td>.028</td>
<td>.022</td>
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Appendix L--Continued

Table 8--Continued

<table>
<thead>
<tr>
<th>Measure</th>
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<th>PSC</th>
<th>BF</th>
<th>SC</th>
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<tr>
<td>Beck Depression Inventory</td>
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<td>Mean Difference$^a$</td>
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<td>$t^b$</td>
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<td>$p^c$</td>
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<td>.034</td>
<td>.008</td>
<td>.189</td>
<td>.019</td>
</tr>
</tbody>
</table>

$^a$Pre- to Posttreatment

$^b$Degrees of freedom equals 7.

$^c$Two-tailed

Note. BFSC = Biofeedback and stress-coping; PSC = Pseudo-biofeedback plus stress-coping; BF = Biofeedback only; SC = Stress-coping only.
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