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by

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The undersigned certify that they have read and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled Psychological Correlates of Myofascial Pain Dysfunction submitted by Frank W. McGrath in partial fulfilment of the requirements for the degree of Ph.D. in Counselling.

Supervisor

Date January 16, 1985

Dedication

To My Dad

The late James Raymond McGrath

son of Francis Clarence McGrath, M.D.

who always wanted his son

to become a doctor.

Abstract

The present investigation was designed to explore the possibility that the severity of Myofascial Pain Dysfunction (MPD) might be determined by the level of environmental stress combined with the style of coping with that stress. In this study a Life Events Checklist was defined as a measure of environmental stress. The Ways of Coping Checklist, which measures seven coping styles, provided the remainder of the psychological predictor variables. Clinical measures of symptom severity, organicity and malocclussion were defined as nonpsychological predictor variables. Subjective and objective measurements of the extent and severity of symptoms were the criterion variables at intake. At follow-up a third criterion variable of subjective improvement was obtained.

Life events were remarkably uncorrelated with symptom severity and only the coping response of "minimizing threat" significantly contributed to subjective MPD. "Not Seeking social support" significantly contributed to the dental assessment of MPD. The dental assessment of "organicity" was the strongest predictor of both subjective and clinical MPD.

Since the literature on MPD was found to be equivocal on the diagnostic criteria for the syndrome, this problem area was examined through a factor analysis of eleven symptoms which were rated by the patients at intake. Distinct factors were identified which seemed to measure MPD PAIN and temporomandibular joint DYSFUNCTION respectively. The PAIN factor showed significant correlations with various coping strategies and the DYSFUNCTION factor was related to evidence for organicity in the joint and malocclussion of the teeth.

Implications of these findings were reviewed and suggestions for further research on MPD were proposed.

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Finally, I would like to acknowledge my special friend Debra, who has provided a valuable perspective and an invaluable relationship in which to survive the whole experience.

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I. INTRODUCTION

The past two decades have seen an increasing interest in masticatory muscle pain and dysfunction. This may be due, in part, to its reported epidemic proliferation among industrialized nations (Hanson & Nilner, 1975). The similarity of masticatory muscle pain to pain of dental origin, its inconsistent response to treatment, and the divergent views on diagnosis and etiology have provoked the interest of dental, medical, and psychological researchers. As investigations have progressed it has become clear that, in addition to dental considerations, complex physiological and psychological phenomena are involved.

This chapter contains eight major sections. Myofascial Pain Dysfunction (MPD) is defined in the first section. This is followed by a description of the anatomical features of the disorder. In the next two sections a review of the epidemiological studies of MPD preceeds a discussion of response to treatment. The last four sections identify the problem to be considered in the present research, the purpose of the study, the research design and general hypotheses to be tested.

A. Definition of Myofascial Pain Dysfunction

In order to explore the incident rates, etiology and treatment of any disorder it is imperative that agreement be made as to the diagnostic criteria. Masticatory muscle pain has been variously called Costen's Syndrome, Facial Pain Syndrome, Temporomandibular Joint (TMJ) Dysfunction and Myofascial Pain Dysfunction. The diagnostic criteria over the years have included impaired hearing, sensations of burning on the tongue, stiffness, earache, tinnitus, dryness of the mouth, dizziness (Costen, 1934), specific occlussal characteristics (Thompson, 1964), mandible hypermobility (Schultz, 1937), pain in the temperomandibular joint, pain in the masticatory muscles, joint noises and altered muscle funtioning (Schwartz, 1959). The permutations and combinations of symptoms which have been used in research has resulted in unreplicable findings and great disagreement as to the etiology of facial pain disorders (Laskin, 1969).

Rugh and Solberg (1976) in their review of the literature, defined TMJ dysfunction as exhibiting the following characteristics: degenerative organic change in the joint, tenderness in the joint area, sounds during condylar movements, limitations of mandibular movement and pain and tenderness of the muscles of mastication. Following the work of

Laskin (1969) they described a subcategory of TMJ patients that exhibited the above symptoms except for those related to degeneration of the joint itself. This subcategory was found to make up approximately 95% of the TMJ patients and was termed Myofascial Pain Dysfunction. Generally it seems that TMJ dysfunction clearly requires direct medical intervention to rehabilitate the joint. Since MPD symptoms are most likely to have psychological features in their etiology and treatment they have become the focus of the present study.

For the purposes of the present research MPD will be defined according to Laskin's (1969) criteria as exhibiting the following constellation of symptoms:

1. MPD patients *must* have pain and tenderness of the muscles of mastication and limitations of mandibular movement.
2. MPD patients *may* have sounds during condylar movement (ie popping, clicking and crepitus of the joint).

In reviewing the literature it is apparent that TMJ dysfunction and MPD have been used synonymously in nearly all studies predating Laskin's (1969) review. Weinberg (1979) reports that even in recent years researchers tend to confound the two disorders. Weinberg (1980) and others (Moss, Garrett & Chiodo, 1982) indicate that MPD may *develop* into TMJ dysfunction and that a differential diagnosis may, at times, be difficult. Thus, in order to incorporate all of the relevent literature into the present review it has been necessary to report on both MPD and TMJ studies. Those TMJ studies which clearly present a diagnosis of "degenerative joint disorder" *per se* will be excluded from the review. Since the great majority of TMJ patients are thought to have MPD (Laskin, 1969) the exclusion of degenerative joint disorder studies will probably not result in misleading conclusions.

B. Anatomical Considerations

The masticatory muscles consist of three pairs of closing and one pair of opening muscles which normally function in a balanced synchrony. Forward, lateral, raising and lowering movements require the coordination of these muscles around a double lever arrangement of mandible (lower jaw) and maxillae (upper jaw). The condyles are posterior protuberances of the mandible and rest in sockets or fossae of the temporal bone to form

the fulcrum of a third class lever. This is known as the temporomandibular joint. Thus when the jaw is closed the force is distributed first on the condyle and then along the teeth.

The major jaw closing muscles are the masseter, temporal and internal pterygoid. The masseter is near the surface of the skin in front of and below the ear and attaches to the body of the mandible and maxillae . The temporal muscle extends from the posterior region of the mandible and spreads upwards over the entire temporal region of the cranium. The internal pterygoid is on the inside of the mandible beneath the masseter muscle. The jaw opening muscle is the lateral pterygoid. It extends from the condyle area of the mandible forward to a point near the upper cheek bone. In order to open the jaw the lateral pterygoid pulls the condyle forwards. Thus the condyle is both stationary when acting as a fulcrum, and movable when under the influence of the lateral pterygoid. At times one condyle may be stationary and the other moving (eg. when grinding on molars).

C. Epidemiological Studies of Myofascial Pain Dysfunction

The nature and extent of MPD have been the subject of numerous clinical and epidemiological studies. Investigations of patients referred to dentists for MPD have found that 70 to 90% are female and that most patients are between the ages of 20 and 40 (Schwartz & Cobin, 1957; Franks, 1964; Carraro, Caffesse & Albano, 1969). Studies of patient populations, while providing interesting and practical information, tell us little about the extent of the disorder in the general population. Elucidation of this question has been gained through various epidemiologic studies.

Many of the epidemiologic studies prior to 1971 have been confounded by unsystematic examination methods and sampling bias (see Helkimo, 1976 for a review). Recent studies have been more consistent in regards to diagnosis with only some improvement in sampling errors. For example, Posselt (1971) examined 269 young dental nurses and found that 21% had symptoms of a magnitude requiring treatment. Hansson and Nilner (1975) examined 1,069 persons employed at a Swedish shipbuilding yard and found 79% had some sign of masticatory dysfunction and 25 to 30% were judged as needing treatment. Finally Solberg, Woo and Houston (1979) found that 26% of 739 first year UCLA students reported subjective signs of dysfunction.

Studies employing broader sampling methods but using a less reliable questionnaire procedure have been conducted by Agerberg and Carlsson (1972, 1973) in Sweden. These researchers found that of 1,215 responders (a 91% response rate) in a single town, 50% reported some symptom of dysfunction of the masticatory system. Frequent facial pain, however, was reported by only 12% of the responders. Both age and sex distribution in this study were found to be balanced.

Helkimo (1974, 1976) attempted to systematize examination methods by developing two indices of masticatory dysfunction. These scales give numerical values for "anamnestic" (subjective) symptoms as well as for "clinical" symptoms. Using this scale he examined a northern population in Finland and found that 26% needed treatment. He also found that as symptom severity increased there was a higher incidence of headache, neck, shoulder and general joint and muscle pain. He noted that there was no sex or age imbalance in symptomatology. Greene and Marbach (1982) reviewed seven Scandinavian research studies which employed the Helkimo Dysfunction Index and found that symptoms were present in 12 to 80% of the population with from 13 to 60% requiring treatment. These researchers concluded that the nature of the symptoms and the various expectancies of subjects and researchers accounted for the variability in the studies.

The few studies of children reported have been even less rigorous in design than those of the adult population. Geering-Gaerney and Rakosi (1971) examined 281 school children ages 8 to 14 and found symptoms in 41% of the students. None of these students were found to have "other symptoms as found in adults". Presumably very few of these youngsters required treatment. Lindquist (1974) in a study of bruxism in 12 year old twins found muscle tenderness in 27% of his subjects. Finally, Belfer and Kaban (1982) reported that 10% of their patients who were treated for facial pain were children between the ages of 10 and 16. These studies indicate that MPD symptoms are distributed in children similar to those in adults with the possible exception that severity is less pronounced in children.

In summary the literature indicates that the incidence of MPD can range from 12% to 80% depending on the sampling procedure, diagnostic criterion and various psychological factors that may be active during the reporting and collecting of data. In addition approximately 25% of the "normal" population has been found to be in need of

treatment. This does not, however, mean that one out of four people seek treatment for MPD. It is known that the incidence of people seeking treatment is much less than the number of persons who have "treatable symptoms". Marbach and Lipton (1978) for example report that only 4% of people in need of treatment for facial pain actually seek care.

These findings are similar to reports on low back pain. Greene and Marbach (1982) found that in both MPD and low back pain the distinguishing feature between a clinical and normal population was that the latter did not consider themselves sick. Thus psychological considerations of expectancy, illness behaviour and personality were seen as important variables in both syndromes.

D. Nature of the Problem of Response to Treatment

Notwithstanding the difficulties in interpreting the epidemiological data, there does appear to be an increasing number of MPD patients seeking treatment. A review of the treatment literature reveals that there are at least twenty possible procedures that a patient with MPD could be subjected to. These include conservative dental treatment such as reassurance and advice, masticatory muscle exercises, spraying the skin with refrigerant liquids (Cohen, 1978), physiotherapy (Weinberg, 1980) and splint therapy (Carraro & Caffesse, 1978). Radical dental procedures include occlussal rehabilitation (Kruger & Dale, 1982), injection of sclerosing agents or cortisone into the joint (Hankey, 1956), injection of a local anesthetic into painful muscles (Cohen, 1978), partial or complete immobilization of the mandible (Roydhouse, 1958), condylectomy (Rowe, 1960), and condylotomy (Ward 1961). There is also a group of treatments which focus on muscle tension *per se*. These include pharmacological treatments (Cohen, 1978), and biofeedback (Olson, 1977). Finally, there is a series of treatments which focus on underlying psychological problems or abilities to cope with stress. Psychoanalysis (Moulton, 1966), pharmacological treatment of affective disorders (Lipton, 1969), behaviour management (Rugh & Solberg, 1976), cognitive behaviour modification (Stenn, 1979), relaxation training (Scott, 1980) and stress management (Haber, Moss and Kuczmienczyk, 1983) all focus on the presumed underlying deficits of MPD patients. Considering this range of treatment an observation made by Berry (1963) may still hold today: "For many patients the only course is to try

different methods in succession in an empirical fashion with the hope that, by exclusion, the final diagnosis may appear and present a rational form of treatment" (p.1123)

Each of the treatment strategies cited have demonstrated some effectiveness with MPD and there seems to be little evidence that one or other general style is more effective on the total patient population. The fact that many clinicians choose treatment styles on the basis that other patients have improved following similar procedures results in a circular and erroneous logic (Laskin & Greene, 1972; Shipman, Greene & Laskin, 1974; Cohen, 1978). This is particularly the case when one considers the placebo factor operating with these patients. For example Goodman, Greene and Laskin (1976) found that 64% of MPD patients respond to a mock equilibration procedure. Although there is clearly a population of MPD patients who will probably respond to *any* treatment there is also, just as clearly, a population of patients who require specialized treatment strategies.

Only a few studies in MPD have focused on psychological factors which may have implications for the severity of symptoms or predict response to these various treatment strategies. Small (1974), Schwartz, Greene and Laskin (1979) and Gessel (1975) found that certain personality factors may be predictive of treatment outcome. Also Speculand, Goss, Spence, and Pilowsky (1981) determined that patient stress levels and other factors affecting the patient/dentist relationship may be useful in developing alternative modes of treatment. This data as well as related research on the etiology of MPD may be productively combined in the development of a psychological diagnostic procedure for use with new MPD patients. Based on this diagnostic strategy it may then be possible to suggest adjunctive psychological treatment strategies.

E. Statement of the Problem

Although there is evidence that psychological factors contribute to the etiology and response to treatment of MPD there is little concensus on the nature or extent of this contribution. Consequently there is no acceptable means for assessing MPD patients at intake to identify the psychological problems that may be pertinent to treatment. This absence of concensus also impairs the development and utilization of effective psychological treatments.

F. Purpose of the Study

The present research is designed to explore the relationships between MPD symptomatology, level of environmental stress and coping strategies. In addition, the interrelationships between subjective symptoms, objective symptoms, duration of symptoms, organicity, and malocclussion are examined. The analysis of these relationships provides a basis on which to make specific hypotheses for future investigations.

G. Design of the Study

Eighty new MPD patients completed a questionnaire which assessed level of environmental stress, coping strategies and MPD symptoms. Dentists also completed an objective diagnostic assessment instrument. Two months following initiation of dental treatment the patient completed a follow-up assessment of treatment outcome. Treatment outcome was defined as the patient's subjective rating of overall improvement .

A discriminant function analysis was undertaken to: a) predict patient and dentist ratings of MPD symptoms and b) predict subjective treatment outcome from the level of stress, coping strategy and severity of presenting symptoms.

A factor analysis of all the measures was undertaken to determine their interrelationships.

II. LITERATURE REVIEW

The literature on the etiology and treatment of MPD can generally be divided into three areas. There are researchers who consider malocclussion as the primary determinant of MPD and see splint therapy and occlussal equilibration as necessary to restore the bite and balance the TMJ condyles. A second group of researchers see environmental stress as the primary cause and muscle hyperactivity as a specific maladaptive stress response - similar to a tension headache. A third group of researchers incorporates both occlussal and stress factors in the etiology and suggest that the two acting in combination result in the constellation of symptoms known as MPD. These three theoretical perspectives are examined in this chapter at length. Additional sections explore the research on psychological correlates of MPD and introduce the concept of interactionism.

A. Occlussal Factors

The most common theoretical position on the etiology of MPD considers that condylar displacement in the glenoid fossae as a result of maloclussion of the teeth is the primary causal factor. Granger (1955) noted that the neuromuscular pattern of mandibular movement instinctively attempts to guide the mandible into a position of maximal tooth contact. The neuromuscular system is designed to operate with a centric relationship between the condyle, disc and glenoid fossae. Harmonious functioning occurs when centric occlussion (i.e., the position of maximal tooth contact) and centric relationship of the TMJs correspond. When one or the other of these relationships becomes eccentric there arises opposing muscular forces between jaw-closing-muscles and lateral pterygoid muscles. In this model malocclussion results in wear in the joint, further strain on the teeth or supporting structures, and spasm of the muscles.

Berry (1963) used the concept of "limit of tolerance" in the masticatory system to explain why some people develop certain symptoms while others with similar morphology develop different symptoms or are symptom free. He suggests that the teeth, bones, joints, muscles and nerves of the masticatory system normally adapt to each other to provide painless and satisfactory functioning in spite of considerable deficiencies that may be present in any part of the system. Each individual, however, has their own biologically determined limit of tolerance beyond which adaptation without dysfunction cannot occur.

Berry points out that the component of the system which breaks down may not necessarily be that under the greatest strain but is the one which cannot adapt furthur to strain on the total system. He points out that dental treatments function to alter the pattern of masticatory movements and thus impact the total system, allowing the healing process to occur wherever necessary.

Shore (1968) expanded on this analysis by demonstrating, through radiographic procedures, that the normal mandibular closing arc is interfered with at the first point of contact of maloccluding teeth. The teeth may be displaced, but at the same time a new occlussal relationship is formed and a modified mandibular arc develops. This results in a displacement of the condyles, muscle spasm and TMJ degeneration. Treatment then is seen to require occlussal therapy so that the normal mandibular arc may be regained. Splint therapy and the more permanent occlussal equilibration procedure (whereby teeth are ground towards a centric occlussal relationship) are the most common dental procedures. Weinberg (1972, 1975, 1977) in a series of studies refined radiographic procedures for examining condylar displacement so that centric relationships between the condyle and occlussion could be determined. He found that bilateral assymmetric TMJ spaces, and posterior or superior condylar displacement were very often associated with muscle spasm.

The fact that occlussal therapy produces improvement in up to 95% (Thompson 1971, Rothwell 1973) of MPD patients is one of the strongest arguements for the occlussal theory of MPD. Further support comes from Ramfjord (1961) who examined occlussal relationships and electromyographic recordings of masticatory muscles in 32 patients with "TMJ joint and muscle pain". All patients were reported as symptom free following complete occlussal adjustment. In addition EMG recordings indicated that balanced muscular activity was restored. Although this study is impressive it is not clear if Ramfjord was reporting on only his successfully treated patients and possibly biasing the effectiveness of the treatment.

Zarb and Thompson (1970) reported that over 90% of their patients with "temporomandibular joint dysfunction" responded to splint therapy and remained symptom free after a 2 1/2 year follow-up. These researchers noted that those patients who responded most rapidly to splint therapy were those with conspicuous dental

disharmonies. Posselt (1971) also found that 74% of his patients who were treated by occlussal equilibration were symptom free at a two year follow-up. Thus the effectiveness of occlussal therapies has been demonstrated in these, and other studies, to be between 75 and 90%.

In spite of its demonstrated efficacy many researchers dispute the occlusal theory of MPD etiology. Goodman, Greene and Laskin (1976) criticize the "circular logic" behind the assumption that malocclussions cause MPD. The fact that the rationale appears sound, that most MPD patients have minor or major dental disharmonies and that patients improve following occlussal therapy is thought not to be "empirical" but rather "appealing". Several researchers (Thomson, 1959; Franks, 1964 and Posselt, 1971) have found that the same types of occlussal disharmonies are distributed equally among populations of patients with MPD and randomly selected normal individuals. In addition the fact that four out of five patients with MPD are women is not adequately explained by the occlussal theories.

To make their point Goodman et al (1976) designed an elaborate experiment in which 25 MPD patients received mock equilibration of their occlussion. Their procedure included the dentists expressed optimism, an elaborate assessment and actual grinding on non-occluding surfaces. Even though all patients had occlussal abnormalities and no occlussal therapy was undertaken, 16 of these patients reported "total or nearly total remission". In other studies Green and Laskin (1971, 1972, 1974) have reported that up to 94% of MPD patients will respond to non-occlussal or even placebo therapies.

In defense of the occlussal theory Weinberg (1979a, 1979b) emphasized the need to differentiate MPD from TMJ dysfunction patients as has been suggested by Laskin (1969). Both Weinberg and Laskin report that TMJ dysfunction patients are most likely to have structural anomalies and require occlussal equilibration. Refined radiological procedures followed by occlussal correction based on centric condylar relationships were found to be necessary for effective treatment of a high percentage of TMJ dysfunction patients. Weinberg defines the studies of patients who respond to placebo therapies as restricted to MPD patients.

In his strongest statement in support of occlussal therapy, Weinberg (1979a, 1979b) cites his own research which has found that 96% of TMJ dysfunction patients have malocclusion. He also, however, notes that the incidence of malocclussion in the

normal population is 77%. This data raises the critical question referred to earlier. If 77% of "normals" have malocclussion and several other researchers have found that TMJ patients have similar occlussal relationships as do non-patients (Thomson, 1959; Posselt 1971; Solberg, Flint & Brantner, 1972) then why do some individuals with malocclussion develop TMJ dysfunction while others do not?

There seems to be clear evidence that other factors are involved in MPD beyond occlussal disharmonies. Within this structural model the concept of "limit of tolerance" as presented by Berry(1963) provides some theoretical speculation. Most of this theory, however, is not subject to empirical validation. Indeed there is some circularity in the logic that MPD sufferers have less tolerance for stress in the masticatory system. Although it is clear that malocclussion and condylar displacement are important in the development and treatment of MPD, additional explanations of predisposing or precipitating factors are necessary if our understanding of the syndrome is to be complete.

B. Masticatory Muscle Hyperactivity

Schwartz (1955) was one of the first researchers to suggest that MPD pain may derive from hyperactive masticatory muscles. His clinical examinations of TMJ patients found muscle tenderness to be an important concomitant symptom and suggested that dentists provide therapy to improve muscle power, elasticity and co-ordination. Sicher (1955) examined neurological and structural relationships in the masticatory system and described a proprioceptive system which may operate to keep the muscular system in balance. He suggested that spasms of the muscles of mastication could be caused by overclosure, premature contacts or mental tension. Jarabak (1956), Kydd (1959), Ramfjord (1961) and Thompson (1970) performed EMG evaluations of the masticatory muscles of MPD patients and found hyperactivity before occlussal treatment and a reduction in hyperactivity after treatment. Chaco (1973) also examined four patients with Costen's syndrome and hyperactive masticatory muscles who were treated with valium. In this study, valium resulted in symptom remission and a reduction to normal EMG levels. Thus clinical EMG investigations have consistently supported the involvement of hyperactive musculature in MPD.

Studies of the "silent period" in the masticatory muscles following the "jaw jerk reflex" have also supported the thesis that muscular dysfunction causes MPD. Normal muscular functioning is characterized by a brief period of inactivity following involuntary stretching of a tense muscle fiber. In normal masseteric functioning a tap to the chin when the teeth are clenched is followed by a silent period ranging from 20-30 milliseconds (msec). MPD patients, however, have been found to have silent periods averaging 80 msec (Bessette, Bishop & Mohl, 1971), 57 msec (Bessette, Mohl & DiCosimo, 1974) and 55 msec (Bailey, McCall & Ash, 1977). These studies have determined that a silent period greater than 35 msec is indicative of MPD and that increased duration correlates with symptom severity.

The significance of these findings suggests that there is a loss of the inhibitory capacity of the nerves innervating the masseter muscle in MPD patients. In normal subjects there is an involuntary inhibition of jaw closing muscles following tooth contact. This inhibition may originate in the periodontal mechanoreceptors or in nerves of the muscle fiber itself (Bessette et al, 1971). With MPD clenching of the teeth is associated with more inhibition than is found in normal functioning. Thus the dysfunction of the masticatory muscles is seen to involve a breakdown in the normal process of inhibition following tooth contact.

Laboratory studies of muscular involvement in the development of MPD have been conducted in which attempts were made to experimentally produce similar symptoms. Christensen (1975) asked healthy adults to grind or clench their teeth for 20 to 30 minutes. Twenty of the 21 subjects reported MPD like pain. Scott and Lundeen (1980) completed a similar study in which muscle hyperactivity was induced by forward thrusting of the mandible to extend the lateral pterygoid muscle. Results again indicated that MPD like symptoms were produced following prolonged masticatory muscle activity. These studies provide *analogue* evidence that muscular hyperactivity may be involved in the etiology of MPD. The similarity of symptoms to those of MPD, as well as their relatively long duration suggest that such activities as clenching or bruxing could produce the clinical symptoms. Other researchers have noted the high incidence of bruxism in MPD patients (Toller, 1976). Ramfjord (1961) published the first comprehensive electromyographic study of bruxism in MPD patients in which he found immediate remission of bruxism

following the use of occlussal splints. Newton (1969) subsequently proposed that occlussal splints reduce muscle hyperactivity through passively stretching the jaw closing muscles.

The possibility that premature tooth contacts might directly initiate bruxism has been proposed by several researchers. Ramfjord (1961), and Newton (1969) have presented neurophysiological theories for the development of such muscular dysfunction. These theories involve reflex pathways, periodontal mechanoreceptors and possible mediation via the central nervous system. Yemm (1976), however, reports that there is no experimental evidence for these reflex pathways and Moss, Garrett and Chiodo (1982) found that the evidence for occlussal generation of bruxism is at best equivocal. Thus there is some theoretical support for locally initiated hyperactivity but there is little experimental evidence for operation of the reflex pathways which are proposed.

In summary it is apparent that muscle hyperactivity is frequently found in MPD patients. The hyperactivity appears to involve excessive clenching, grinding or bruxism and a lowered ability to inhibit activity after tooth contact. The possibility that muscle hyperactivity is directly caused by malocclussion has been suggested but there is little evidence of how this might occur. With the lack of such experimental evidence for local causes the possibility of centrally mediated hyperactivity has received considerable attention.

C. Environmental Stressors

Early studies found that increased masseter and temporal EMG activity were associated with such stressors as hostility on the part of an interviewer (Kydd, 1959). Perry, Lammie, Main and Teuscher (1960) examined dental students while they were being questioned about upcoming final examinations and found significantly increased masseteric activity. Yemm (1969a) found elevated masseter activity in MPD patients during a task involving high response uncertainty. Yemm (1969b) also examined the *recovery* of masseter muscles in MPD patients. He found that MPD subjects consistently failed to demonstrate reduction of EMG activity in their masticatory muscles within the two minute interval between exposures to stressful tasks. Non MPD subjects readily demonstrated a reduction in EMG activity between exposures. This research indicates that MPD patients

not only respond to stressors through tensing their masticatory muscles but also may not be able to recover resting muscle levels as rapidly as non-MPD subjects. The implication is that MPD subjects maintain elevated muscular activity long after stressful events have passed.

The question of whether MPD patients demonstrate a generalized or specific muscle overresponsiveness has been considered by Mercuri, Olson and Laskin, (1979). They found that EMG levels in the masticatory muscles of MPD subjects were more responsive to experimental stressors than were those of controls. In addition these researchers found that control subjects had a higher EMG response in the gastrocnemius muscles in the same stressful situations. These studies suggest the possibility that MPD results from a specific localization of the stress response in masticatory muscles.

Further evidence of a CNS mediated stress response in MPD patients is reported by Berry (1969) who found that MPD patients suffered from a high incidence of stress related disorders such as migraines and low back pain. The incidence of stress related disorders was 10 times that of the normal population. Evaskis and Laskin (1972) also investigated the stress levels of MPD patients by measuring urinary catecholamine and 17 hydroxysteroid levels. These chemicals have been determined to be excreted by persons under stress. The 17 hydroxysteroid level of MPD patients was 33% higher and the catecholoamine level was 118% higher than that of controls. Based on previous research which suggested that particular kinds of stress effect the excretion of these compounds Evaskis and Laskin (1972) concluded that MPD patients were commonly under stressors which involved uncertainty and ambiguity.

More direct studies of the enivironmental stress facing MPD patients has been reported by Stein, Hart, Loft and Davis (1982) and Moody, Kemper, Okeson, Calhoun and Parker (1982). These researchers used the Schedule of Recent Events (SRE) developed by Holmes and Rahe (1967). The SRE is a checklist of stressful experiences that has been found to correlate with stress related illnesses. In the Stein *et al* (1982) study MPD patients scored an average of 161.8 on the SRE compared to 93.4 for the control subjects. In the Moody *et al* (1982) study MPD patients scored an average of 216. These two studies are perhaps the most convincing in demonstrating that enviromental stressors play a significant role in the development of MPD.

In summary MPD patients appear to have experienced a high incidence of environmental stress prior to the onset of their symptoms. They also have relatively more stress related disorders such as migraine and low back pain. Finally, physiological measures have determined a specific localization of the stress response in the masticatory muscles of some MPD patients.

D. Psychophysiologic Theories

Over the years many researchers have considered the possibility that both environmental and physiological factors must be present for MPD to develop. Schwartz (1955, 1959) together with Moulton (1955) proposed that *predisposing, contributing* and *precipitating* factors could account for the development of MPD. These structural factors such as the manddible/maxillae relationship may *predispose* certain individuals to develop TMJ symptoms. Occlussal abnormalities could be seen as *contributing* to this predisposition. *Precipitating* factors might include a sudden or continuous stretch of the jaw muscles, some dental procedures or "subconcious oral activity" such as clenching or grinding of the teeth. The primacy of occlussal abnormalities was retained in this model but a schema of TMJ development which included psychological and muscular factors was now being proposed.

Laskin (1969) presented a similar psychophysiologic model which clearly considered masticatory muscle spasm as the primary factor responsible for the MPD symptoms. Laskin describes muscular overextension, overcontraction or fatigue as initiating spasm. Muscle fatigue, as the result of chronic oral habits such as bruxing or clenching were thought to account for most of the MPD disorders. These chronic oral habits were described as "tension relieving mechanisms" that are largely independent of occlussal factors. Prolonged muscle spasm was seen to produce a slight change in jaw position *to which the teeth accommodate*. Thus, according to Laskin's model, occlussal disharmonies *result* from imbalanced muscular functioning. Laskin also suggested that continued jaw function with the condyles in an abnormal position leads to muscle spasm and degenerative arthritis. Thus the MPD syndrome, "although originating as a functional problem in most instances, ultimately can lead to organic disease" (Laskin, 1969, p.50).

Rugh and Solberg (1976) have formulated a more balanced psychophysiologic theory. These researchers suggest that the symptoms reported by any one patient develop from a unique constellation of structural, environmental, and psychological factors. According to Rugh and Solberg the possibility that instability of the "gnathic structure" is a result of inherited, developmental or dental factors (including occlussal interferences) cannot be dismissed. Similarly stress *per se* cannot be seen as unicausal since individuals vary in their perception of stress. Finally, the response to stress and reporting of symptoms is also a function of the individual's psychological makeup and tolerance for pain.

In Rugh and Solberg's model the structural, personality, and situational considerations are seen as important in determining the patients presentation of, as well as response to treatment. This multicausal model emphasizes the complexity of the MPD phenomenon. It suggests that numerous factors determine not only the etiology of MPD but also the patients response to the various therapeutic procedures. These authors are emphatic in their contention that a psychological as well as a dental assessment should be a routine part of the MPD intake procedure. They predict that those patients who report the strongest psychological maladjustment or who have other related stress disorders are likely to have the most unfavourable prognosis for conventional dental therapy.

E. Psychological Correlates

The impetus for exploring psychological correlates of MPD in depth has come from case reports of significant psychopathology in TMJ patients (Engel, 1957) as well as the response of many patients to psychotherapy and counselling (Moulton, 1955; Lupton, 1969). These findings as well as those which have indicated a relatively significant response of patients to placebo splints (Sutcher 1969) and placebo drugs (Greene & Laskin, 1971) strongly support the contention that psychological variables are important in the etiology of, and reponse to treatment of MPD.

The possibility that MPD may be an expression of intrapsychic phenomena was first expounded by psychoanalytic theorists. Moulton (1955) suggested that TMJ dysfunction is an hysterical conversion reaction resulting from unconcious emotional conflicts. Lefer (1965) provided an elaborate psychoanalytic explanation of TMJ pain in

which TMJ symptoms were utilized to gain security in the form of a transference relationship with the dentist. According to Lefer the patient was seen as having poor ego boundaries and unmet dependency needs. Clenching of the teeth was explained as the result of an excessive effort to control anger in a dependency relationship. Other researchers have relied on clinical impressions to suggest that TMJ patients are rigid, perfectionistic, domineering and obsessive-compulsive (Lesse, 1956) or tense, apprehensive and overreacting to pain (Kydd, 1959). The presence of stable psychopathology is not consistently supported ,however, as other assessments have found no such evidence (Gessel, 1973).

Related to the conception that psychological factors can predict MPD is the hypothesis that emotions such as anxiety, hostility, apprehension and anger are important mediators. The presence of anxiety in MPD has received consistent clinical and empirical support (McCall, Szmyd and Ritter, 1964; Solberg, Flint and Brantner, 1972; Molin, Edmon and Schalling, 1973). Unfortunately, the concept of anxiety has not been rigorously defined beyond "the emotional reaction accompanying the perception of threat or uncertainty" (Lazarus and Averill, 1972) and must be inferred through the observation of behavioral or physiological changes or through verbal reports of individuals. Thus it is unclear whether the consideration of anxiety as a phenomenon which results from stressful experiences and causes physiological reactions is more useful than considering stress as directly causing a specific physiological response such as muscular tension. In addition, as Rugh and Solberg (1976) point out, the incorporation of "anxiety" into the etiology of MPD may be misleading since it has not yet been demonstrated that anxiety is the cause of rather then the result of MPD. It seems less confusing, then, at this point, to consider psychological correlates of MPD independently from an hypothetical emotional mediating factor.

Recently researchers have attempted to describe an MPD personality. Using the MMPI Lupton (1966) found narcissistic, responsible, autocratic and overgenerous traits in TMJ patients. Shipman, Greene and Laskin (1974) also examined MMPI profiles of MPD patients and found high scores on conversion hysteria, hypochondriasis, depression and psychopathic deviate scales. Gross and Vacchiano (1973) using the 16PF found high scores for insecurty and hostility among his patients and Molin, Edman and Schalling

(1973) using the Eysenck Personality Inventory described MPD patients as neurotic. Concurrently other researchers (Solberg, Flint and Brantner, 1971; Schwartz, Greene and Laskin, 1979) have found MMPI profiles to be within normal limits with elevated scores on only the neuroticism and depression scales. The inconsistency in the attempts to describe an MPD personality has led many researchers to suggest that there may be personality subgroups of MPD patients (Solberg, Flint and Brantner, 1972; Lupton and Johnson, 1973; Rugh and Solberg, 1979).

An examination of the research on psychological correlates of MPD suggests that there may be three MPD personality types. Lascelles (1966), Fine (1971), Rothwell (1972), Cohen (1978) and others have found a high incidence of neuroticism. The extremely neurotic individuals may also have depressive symptomatology (Belfer, 1982) or signs of psychosis (Kaban and Belfer, 1981). A second category of MPD patients could be classified as hypernormal or stoical. Lupton (1966, 1969) and Lupton and Johnson (1968, 1973) have conducted extensive research on the stoical, narcissistic MPD patient. These patients tend to deny weaknesses in coping with stress and are prone to psychosomatic symptoms. A third group of MPD patients have been described as normals who are under extreme environmental stress or are simply beyond their capacity to cope with stress (Fine, 1971; Gross, 1973; Heloe and Heiberg, 1980).

In summary, there is little evidence to indicate that MPD is correlated with one specific personality trait. MPD patients do not present as a well defined group and it appears that a variety of personality types are susceptible to the disorder. A hypothetical subcategorization of MPD patients is possible and has been presented based on the present authors interpretation of the literature. These personality correlates of MPD will be of limited value, however, unless they can be utilized to make specific clinical predictions. Rugh and Solberg (1976) have pointed out that efforts to diagnose subgroups of MPD patients should focus on providing clinically useful information which will aid in treatment planning.

F. Interactionism and Coping

The difficulties in accounting for psychological factors in the prediction of MPD or other stress related illnesses may be inherent in the measurement of personality and emotional responsiveness. Traditionally these phenomena were viewed from the perspective of trait theory or situationism. Thus, depending on one's orientation, psychological functioning was determined either by long standing intrapsychic variables or by the environment. Unfortunately efforts to predict future behaviour on the basis of one or the other have not been rewarding (see Endler & Magnusson, 1976). For this reason the prediction of adaptation has shifted to an examination of how persons and situations interact to determine outcome.

The interactional perspective attempts to take into account psychological factors which mediate the impact of the environment on the organism. Factors such as perception and appraisal become important, if not crucial, predictors in this model. Although one might continue to argue that perception and appraisal are a reflection of particular personality traits (eg. Haan, 1977) the fact remains that the "personality" literature has not been very fruitful in predicting adaptive outcomes - particularly in the MPD research. Singer (1984) points out that even if we could determine maladaptive personality profiles then, "by definition" there will be no therapuetic interventions forthcoming from the research. "Those who have the wrong or inappropriate personality are just out of luck" (Singer, 1984, 2306).

Another way of describing the means by which people perceive, appraise and react to stress has been termed "symbolic interactionist" or the "situational-attributional-informational" perspective (Singer, 1984). In this model adaptation is seen as a learned pattern of cognitive and affective reactions which are triggered by the immediate environment. These reactions may be called strategies, defenses, or responses. They may also be called styles when they generalize to other situations. In this model there is an assumption that interventions can be developed to learn new reactions to the environment.

Although not purely interactionistic in nature it may be possible to begin to assess adaptation through examining coping strategies and environmental stressors. This will provide a static picture of what is, by definition, a dynamic process. Metaphorically this is

similar to trying to predict the end of a motion picture from several still frames. Although highly tenuous some themes may be identified and if enough "still frames" are put together one approaches a very close representation of the dynamic relationships as well as the outcome.

The possibility that particular coping strategies or styles are associated with specific patterns of physiological responses and hence to specific illness outcomes is receiving increasing support (Lipowski, 1977, Lazarus and Folkman, 1982). For example Linden and Feurstein (1981) have described a tendency for hypertensives to be disposed toward threat as opposed to challenge appraisals and to react to this threat with anger. In regards to treatment outcome Hackett and Cassen (1973) determined that myocardial infarction patients do better if they deny having had a heart attack.

An important contribution to our understanding of MPD may be obtained by analyzing coping styles among MPD patients along with their current level of environmental stress. The recent research of Lazarus and Folkman (1982, 1983) allow us to develop hypotheses about how these styles may influence adaptation (i.e., MPD symptomotology). An analysis of the previously reviewed psychological correlates provides support for some specific predictions. MPD patients have been most consistently described as neurotic or stoical. The term "neurotic" has been difficult to define in terms of coping styles. It would appear that neuroses is a description of resultant symptomatology rather than of a process of reacting to the environment (see previous discussion on anxiety). The term "stoical", however, has already been described by Lupton (1969) in terms of coping behaviors. Stoics are more likely to have "responsible, generous, and managerial responses to stress." They tend to deny problems and consequently to avoid talking to others about problems. Overall they have an investment in appearing strong and self-sufficient.

It may be possible to identify stoicism on the basis of the assessment instruments provided by researchers on coping. Three coping responses in particular, appear to be equivalent to the research findings that MPD patients are stoical. These are, minimizing threat, self blame and avoiding social support. All of these would be expected to be positively correlated with MPD symptomatology.

G. Conclusions on the MPD literature

It appears that several variables may be helpful in predicting symptom severity and the success of conservative dental treatment for MPD patients. Those patients who are reported to have different levels of malocclussion or organicity of the joint would be expected to respond differently. Perhaps the duration of symptoms is the critical determinant of response. The evidence for elevated stress levels in MPD patients would also suggest that treatment which does not focus on stress would be less effective for these patients. Personality studies have given indirect evidence that certain "types" of individuals may not respond to dental treatment. Individuals who are "stoical" may require adjunctive therapeutic efforts in addition to those provided by the dentist. An innovative style for operationally defining these psychological factors has been provided by researchers in coping. This style makes it possible to operationally define "stoic" in terms of the coping responses minimizing threat, self blame and avoiding social support.

We are thus left with the research questions addressed in Chapter I. Can severity of MPD symptomatology be predicted by measures of life events and coping styles? And, is it possible to predict non responders to conventional MPD therapy on the basis of severity of dysfunction, level of environmental stress and psychometrically determined coping styles? Finally, how do these predictors compare to nonpsychological predictors such as malocclussion, organicity or duration of symptoms? If we can answer these questions it may be possible to identify those MPD patients who should be directed to adjunctive psychological therapies.

III. DESIGN AND PROCEDURE

Field studies on stress and illness have been of two general forms - retrospective and prospective. The former assess life events as they are recalled by already symptomatic individuals. The latter requires a current assessment of life events in healthy individuals followed by an examination of the presence of symptoms later in life. Each of these approaches have their own shortcomings in regards to either implementation or interpretation of results.

The retrospective study presents serious problems of interpretation since it relies on *a posterior* data. In stress research identified patients (i.e., already ill) are normally asked to recall stressful events and these recollections are compared with those of non patients. Correlations of .30 to .40 have been found between life events and the presence of illness. Interpretation of this correlation is difficult because of the phenomenon known as "systematic retrospective falsification". This is the confounding of criterion and predictor variables through the previous knowledge of one influencing the assessment of the other. For example the presence of the illness in the "patient" population may affect the recollection of stressful events. This direct contamination of the predictor variable is particularly problematic when both variables are measured concurrently.

The prospective design precludes some of the problems inherent in retrospective research. By predicting future illness on the basis of current reports of life events systematic falsification is less probable. Although there is reliance on self reports there is less chance that the subject will connect the two measures. When reporting life events he/she has no knowledge of his/her future illness. Later, when illness is recorded, particularly if there are clinical measures available, it is doubtful that knowledge of life events is directly contaminating this data.

The major criticism of prospective studies is not one of contamination but of practicality. If the incidence of an illness is say 10% it would require *a priori* assessments of life events in 1000 subjects to obtain 100 "hits". Although providing invaluable information a study of this scope is beyond the means of many researchers.

The first part of the present study will be retrospective in nature. It will involve the assessment of life events, coping styles and MPD symptomotology via self-report at time of intake. Thus the population under study will be self selected as having facial pain. This

procedure eliminates the necessity of examining up to 400 normal subjects to obtain the 80 required to make valid predictive statements. The problem of retrospective falsification remains, however, and these predictor variables must be reevaluated in a prospective study. The second part of the present research will thus be prospective in nature. This will be an attempt to predict response to treatment on the basis of all of the variables obtained from the intake procedure. Response to treatment will be determined at a two-month follow-up through the readministration of parts of the original self-report questionnaire. Subjects will be asked to evaluate their level of symptoms at follow-up and these will be compared to symptom severity at time of intake.

A. Instrumentation

The instruments required for the proposed investigation include measures of MPD symptomatology, response to treatment, life events and coping styles. Appendix II shows the final questionnaire with all assessment measures included.

Measurement of MPD symptomatology. The diagnosis of MPD has been notoriously inconsistent. Helkimo (1976) attempted to standardize the assessment of symptom severity through a multidimensional rating scale. The Helkimo Dysfunction Index is composed of two parts. The clinical index includes ratings of five symptoms each measured on a three point scale. Impaired range of movement, impaired joint function,(TMJ sounds, deviation, luxation or locking of the jaw), tenderness to palpation of masticatory muscles, tenderness to palpation of temporomandibular joint and pain on a movement of the mandible are all assessed by the dentist. Each has been operationally defined and the procedures are well-known to dentists working in the area. From this scale an overall index of clinical symptom severity can be obtained which ranges from five for no symptoms to 15 for severe dysfunction.

The Helkimo subjective index is a rating scale filled out by the patient on fatigue of the jaw muscles, difficulty in opening the mouth wide, locking or luxation and muscle pain.On the basis of the literature review it was decided to add subjective symptoms of joint clicking, scraping, pain of the shoulder muscles, back pain and headaches which have often been associated with MPD. In addition it was decided to expand the Likert rating scale from three to five in order to provide more variability in the data. This revised

subjective index provides a range of severity from eight to 40.

To date there have been no reports of the reliability or validity of the Helkimo instrument. The Helkimo Dysfunction Index does present considerable face validity and appears quite straight forward to implement. For this reason, and since there is no comparable alternative, this instrument will provide the criterion variables of clinical and subjective symptom severity.

Response to Treatment: At follow-up each patient was asked to rate their symptoms on the above subjective rating system via telephone interview (Appendix III). They were then asked to rate their overall improvement on an eight point scale from "very much worse" to "completely symptom free".

Life Events: Holmes and Rahe (1967) were the first to systematically assess environmental stress in terms of life events. The Social Readjustment Rating Scale (SRRS) consisted of 43 life events derived from case histories of 5000 patients. Years of research have confirmed the original finding that "the greater the impact of life change the greater the probability that the life change would be associated with disease onset" (Holmes and Masuda, 1974). Notwithstanding these "associations" several criticisms of this instrument have been brought forth and have resulted in improvements in subsequent measures.

A major criticism of the SRRS has been that it samples only a small number of possible events (Dohrenwrend, 1974), and that many of the events sampled are "illness related" (Hudgens, 1974). In addition to measuring a relatively small number of highly stressful events the scale enquires about major personal injury, pregnancy and health of the family which are all directly related to illness. Also the sampling of events from a patient population may have resulted in a list that is less meaningful for the general population (Dohrenwend & Dohrenwend, 1974). Finally the SRRS has been criticized for over sampling negative stressful experiences when recent literature has pointed to the similarity of the adaptation process following positive stressful experiences such as a promotion or marriage (Rahe, 1974).

Dohrenwend, Krasnoff, Askenasy and Dohrenwend (1978) have developed a life events scale which attempts to remedy the above difficulties. The Psychiatric Epidemiology Research Interview Life Events Scale (PERI) seems to offer considerable

improvement on the SRRS.

To improve the sampling procedure Dohrenwend *et al* drew their respondents from an urban population (New York City) through a stratified random sampling procedure. Possible items were generated by questioning the respondents about "the last major event in your life." Through this method as well as a review of relevant literature a 102 item checklist of possible stressful experiences was developed. Although these events included some illness-related items most of these were eliminated. Finally an improved balance of positive and negative experiences was obtained.

Previous researchers had developed ratings of the impact of stressful events through a sample of convenience. Professionals, students or patients were asked to rate the items as to the amount of disruption associated with the event. Dohrenwend *et al* (1982) improved this procedure by obtaining a stratified random sample of 92 raters. Each rater was asked to provide a number indicating "the amount of change or adjustment involved in each event when compared to marriage". Marriage was given a rating of 500. The arithmetic mean of the ratings for each event became the "weighted" score for that event. The range of these weighted scores was 163 to 1036. The sum of weighted scores was then defined as a measure of total life events. To simplify the analysis in the present study the ratings were divided by 10 to give a range of weighted scores from 16 to 104. These weighted scores will be retained in the present study in spite of the criticisms of Rahe(1974) and others that weighted and unweighted life events are highly correlated.

Using the PERI it will be predicted that weighted life events are positively correlated with symptom severity in MPD patients.

Coping Measures: Although the measurement of coping is still in its infancy several promising research instruments have been developed. Two of these are based on theoretical assumptions whereas a third measure has been empirically derived. Gleser and Ihilevich (1969) constructed the Defense Mechanism Inventory to tap the relative intensity of five groups of defenses in the psychoanalytic sense. Haan (1977) formulated a more general taxonomy of 10 ego processes which are grouped as either coping, defense mechanism or fragmentation. Both of these instruments have demonstrated an acceptable level of reliability and internal validity but have run into problems of external validity or

general interpretation.

Lazarus and Folkman (1982) have developed a questionnaire which samples a broad range of possible coping responses and which, when factor analyzed, yields several meaningful categories. The subject is asked to identify and elaborate on "the situation that has been the most stressful to you in the last month". After this is done they simply check all of the 66 possible coping responses "applied to them". The factor analysis of the 66 item checklist reveals seven general categories of coping responses. These factors are problem solving, wishful thinking, mixed coping, growth, minimizing threat, seeking social support and blaming self. The present researcher suggests that this instrument will provide a measure of stoicism. The factors "minimizing threat" and "self blame" are consistent with the literature descriptions of stoicism. Also the factor "seeking social support" would be expected to have a negative relationship to stoicism.

Thus it may be predicted that symptom severity in MPD patients will correlate positively with "minimizing threat" and "self blame" and negatively with "seeking social support".

Nonpsychological variables. Three additional independent measures were included in the present study for comparisons to the psychological predictions. As there is considerable support in the literature for the MPD symptomotology to be related to the extent of malocclussion and/or evidence of organic deterioration in the joint these measures were included. Also the duration of symptoms would be expected to be related to symptom severity. In consultation with two dentists Likert scales for clinically distinguishable levels of malocclussion and organicity were developed. Duration of symptoms was determined by the patient ratings of how long they have been experiencing facial pain. Six month intervals were provided for this rating.

Additional data was collected at intake for later analyses of variations in the experience of pain and the operation of secondary gains. Questions on age of patient and marital status were also included for descriptive purposes. This additional data is not included in the present analysis and will not be considered further.

B. The Sample

The subjects for the study were 85 patients who presented at dentists offices in Alberta with MPD as defined by Laskin (1969).

The dentists were instructed to include all patients who presented with pain and tenderness of the muscles of mastication as well as limitations of mandibular movement (Laskin, 1969). Patients with advanced organic deterioration of the TMJ were excluded from the study. Approximately 40 percent of the subjects were obtained from a single practice. In this practice all patients presenting with facial pain were required to complete the questionnaire as part of the intake procedure. The remainder of the subjects came from 11 offices throughout Alberta where it was more difficult to control selection bias on the part of the dentist. Generally, it is expected that resistant patients in these practices were not required to complete the questionnaire.

C. Procedure

Patients filled out the intake questionnaire (Appendix II) in the first or second appointment and before treatment was administered. The questionnaire included sections on life events, coping styles, subjective MPD symptoms, clinical MPD symptoms and demographic information. Some of the questionnaires contained additional sections on Locus of Control, Depression, and Anxiety which had been included in the original research design. These sections were abandoned halfway through the study because of the difficulty in obtaining data outside of the single practice described above. The long questionnaire required 60-90 minutes to complete. The short questionnaire required 30-45 minutes to complete and significantly improved the response.

The dentist completed the brief clinical-diagnostic section at the end of the initial visit.

Each subject was contacted by phone after a two month interval to obtain the follow-up data. At that time they were asked to indicate their present severity of MPD symptoms as well as rate their overall symptoms on the 8 point Likert scale "very much worse" to "completely fine". Approximately 70% of the original subjects were contacted for follow up.

D. Statistical Analysis

The indepentent variables of subjective and clinical symptomatology were determined by summing patient and dentist ratings respectively. They were then correlated with each other to determine if they could be collapsed into one criterion variable. The independent variable of life events was determined by totalling the weighted scores of the events checked off by each subject and dividing by 10. The independent variables of coping styles were obtained by totalling the responses on each of the seven categories. Malocclussion, organicity, and duration of symptoms were obtained directly from the Likert scales. Pearson r correlations were calculated between the dependent and independent variables. Finally, a stepwise discriminant function of the twelve predictor variables onto the two criterion variables was undertaken. At this time the specific research questions on these correlations were examined. Question 1 (see next section) was explored by examining the discriminant function for significant contribution of the life events and coping scores to variance accounted for in predicting symptom severity.

The above analysis was repeated for the dependent measure of response to treatment. Included in the discriminant function analysis for predicting response to treatment were the subjective and clinical ratings. This permitted the comparison of the nonpsychological independent measures which could be obtained at intake with the psychological measures as determinants of treatment outcome. Question 3 was examined through a discriminant function for the significance of the contribution of the life events and coping scores to variance accounted for in predicting treatment outcome.

E. Research Questions

1. Will the psychometric measures of life events and coping responses improve the prediction of MPD symptom severity at intake over the physical measures of maloculussion, duration of symptoms and evidence of organicity in the temporomandibular joint?
2. Will the psychometric measures of life events and coping responses improve the prediction of subjective improvement of MPD symptoms at a two month follow-up over the physical measures of maloculussion, duration of symptoms and evidence of

organicity in the temporomandibular joint.

3. Is it possible to identify theoretically defendable factors for the constellation of symptoms reported by MPD patients which have unique relationships with coping strategies and stress?

IV. RESULTS

In this chapter the data is organized, analyzed and conclusions are drawn. The chapter begins with a demographic and descriptive information. The research questions are then explored through correlation, multiple regression and factor analysis of the data.

A. Demographic and Descriptive Data

Table I summarizes the demographic and descriptive information collected on each subject at intake. This table indicates that 85% of the subjects were female, 74% were under 36 years of age, 65% were married and 46% reported symptom duration of more than two years. The figures for sex ratio, age, and duration of symptoms are commensurate with previous reports. Data on marital status have not previously been reported.

Table II presents the distribution of subjective symptom severity across subjects. Approximately 80% of the subjects reported joint clicking, fatigue of the jaw muscles, difficulty in opening the mouth wide, pain in the shoulder muscles, low back pain and headaches. Fifty percent reported locking of the jaw, light headedness, indigestion and constipation. Only 30% reported scraping of the joint and less than 10% reported fainting. It would appear that the subject population can indeed be described as suffering from MPD according to Laskin's (1969) criteria. Only one subject reported a complete unawareness of symptoms even though the dentist had identified impaired mobility of the mandible.

The subjects in this study were also asked to briefly describe "the event or situation that has been most stressful to you during the last month." These anecdotal records may serve to concretize some of the discussions to follow. Many of the "situations" were described in a cursory manner such as "marital problems" or "arguement with a friend." Others, however, were more descriptive and provide insight into the stress / coping contexts of the subjects.

Three representative descriptions follow:

> Subject #05: After a straining year, my husband and I had another fight. I had been severely depressed and frustrated lately and could not take anymore. I wanted a separation and he would never agree. I knew I was emotionally sick

Table I

Demographic and Descriptive Data at Intake
Expressed in Percentages (N=85)

Sex	Male	15%	Female	85%
Age	16-25 years	20%	26-35 years	49%
	26-35 years	20%	more than 46 years	6%
Martial Status				
	Single	35%		
	Married / Common Law	65%		
Duration of Symptoms				
	less than 6 months	29%	6-11 months	12%
	12-24 months	14%	more than 24 months	46%

and my body was warning me of problems. This last fight was enough to make me almost have a nervous breakdown. I took the kids and spent two weeks in the womens shelter, where I filed for divorce. He threatened the worse, as he always does, but I can't take anymore and don't care if he carries out his threats - even his threat to commit suicide.

Subject #47: My 87 year old mother is in a nursing home. As I am the only child I feel quite responsible for her care. This has always been with me. This past month I had back, neck, arm and leg pain almost the whole month.

Subject #72: I have had three different supervisors at work in the last eight months each of whom has different priorities and different ways of doing things. Frustrating!

The term "frustrating" seems to summarize the situations of these and many other subjects. A common theme appears to be involvement in situations which are perceived to be beyond the subjects control or in which the subjects own needs are not being heard or met.

Given this general impression of the subject populations it is now appropriate to turn to a statistical exploration of the responses of the subjects.

B. Data Analysis

An initial decision in the analysis of the data was whether the clinical dysfunction index and the subjective dysfunction index were measuring a single phenomenon which could be defined as myofascial pain dysfunction. Should subjective dysfunction and clinical dysfunction be highly correlated it could be assumed that any relationships found with one would also apply to the other. A low correlation would require two separate analyses as well as an explanation of any differences found.

A priori a correlation of 0.80 or better was determined to be the criteria for accepting the hypothesis that a common phenomenon was measured. This cut off was based on the correlation commonly used in studies of interrater reliability. Table III shows that the pearson correlation between these two dependent measures was 0.17. The

Table II

Subjective Symptom Severity at Intake

Expressed in Percentages (N=85)

	None	Mild	Moderate	Severe	Extreme
clicking	17%	30%	31%	15%	7%
scaping	70%	19%	10%	2%	0%
stiffness / fatigue	15%	13%	30%	2%	0%
opening	22%	19%	22%	19%	18%
pain / shoulder	25%	25%	26%	15%	9%
headaches	24%	17%	24%	17%	18%
low back	31%	18%	31%	16%	4%
light head	46%	37%	13%	2%	2%
fainting	94%	6%	0%	0%	0%
indigestion	58%	21%	6%	15%	0%
constipation	64%	17%	11%	8%	0%

Table III

Pearson Correlation Coefficient between the dentist ratings

of Clinical Dysfunction and the patient ratings of Subjective Symptom Severity

Pearson r	Significance
0.17	.065

variance shared by these measures is thus less than 3% and the hypothesis of one common variable was rejected. The two measures must be assumed to be independent and separate analyses undertaken for the purpose of further hypothesis testing.

C. Research Questions

Research Question 1: Will the psychometric measures of life events and coping responses improve the prediction of MPD symptom severity at intake over the physical measures of maloculussion, duration of symptoms and evidence of organicity in the temporomandibular joint?

Table IV presents the correlation coefficients between subjective dysfunction and all of the independent variables. The subjective report of the coping response of "minimizing threat" was significantly correlated with subjective dysfunction. as were "problem solving", "evidence of organicity" and "severity of malocclussion".

Table V presents the correlation coefficients between clinical dysfunction and all the independent variables. Only "evidence of organicity" and "severity of malocclussion" were significantly correlated with clinical dysfunction.

Table VI summarizes the discriminant function equation for predicting subjective dysfunction. For the purposes of this analysis mild dysfunction was arbitarily defined as a score of 21 or less whereas moderate to severe dysfunction was defined as a score of 22 or greater. This also allowed for approximately equal cell sizes of 39 for mild dysfunction and 46 for moderate to severe dysfunction. Following the stepwise method with a significance level for inclusion in the equation set at 0.05 it was found that only three variables could be entered into the equation. In order of decreasing additional variance accounted for these were a) organicity as determined by the dentist, b) duration of symptoms and c) the use of the coping response "minimizing threat" as reported by the patient. The actual equation is reported at the bottom of Table VI.

Similarly Table VII summarizes the discriminant function equation for predicting clinical dysfunction. Mild clinical dysfunction was defined as a score of 10 or less whereas moderate to severe dysfunction was defined as a score of 11 or more.

Even though clinical and subjective dysfunction are poorly correlated the "evidence of organicity" as determined by the dentist is the most significant discriminator of severity

Table IV

Correlation Coefficients at Intake

Independent Variables vs Subjective Dysfunction

Independent Variable	Subjective Dysfunction	Significance
Total Life Events	-.02	.44
COPING RESPONSES		
problem solving (C_1)	.19	.04*
wishful thinking (C_2)	.04	.37
mixed coping (C_3)	.09	.22
growth (C_4)	.03	.39
minimizing threat (C_5)	.19	.04*
seeking social support (C_6)	.05	.32
blaming self (C_7)	-.10	.18
Duration of symptoms	.17	.06
Evidence of organicity	.56	.02*
Severity of malocclussion	.22	.03*

* Exceed 0.05 level of significance.

Table V

Correlation Coefficients at Intake

Independent Variables vs Clinical Dysfunction

Independent Variable	Clinical Dysfunction	Significance
Total Life Events	.025	.42
COPING RESPONSES		
problem solving (C_1)	.08	.25
wishful thinking (C_2)	.11	.17
mixed coping (C_3)	.10	.19
growth (C_4)	.15	.10
minimizing threat (C_5)	.03	.41
seeking social support (C_6)	.16	.09
blaming self (C_7)	.16	.09
Duration of symptoms	.07	.27
Evidence of organicity	.56	.000*
Severity of malocclussion	.54	.000*

* Exceed 0.05 level of significance

Table VI

Discriminant Function at Intake

Predicting Subjective Dysfunction at Intake (STMJ)

From the Independent Variables

Independent Variable	B	Significance
Evidence of organicity (ORG)	.11	.01
Duration of Symptoms (DUR)	.06	.04
COPING RESPONSE		
minimizing threat (C_3)	.06	.05
Constant	.74	

Equation STMJ = .74 + .11 (ORG) + .06 (DUR) + .06 (C_3)

Multiple R = .39

R Square = .15

Variables included in this analysis: duration of symptoms, organicity, malocclussion, total life events, seven coping responses.

Table VII

Discriminant Function at Intake Predicting
Clinical Dysfunction at Intake (CTMJ)
from the Independent Variables

Independent Variable	B	Significance
Evidence of organicity (ORG)	.16	.00
Severity of malocclussion (MAL)	.14	.02
COPING RESPONSE seeking social support (C_6)	-.10	.02
Constant	.94	

Equation: .94 + .16(ORG) + .14(MAL) - .11(C_6)

Multiple R = .64

R Square = .41

Variables included in this analysis: duration of symptoms, organicity, malocclussion, total life events, seven coping styles, clinical dysfunction at intake and total subjective symptoms at intake.

on both dependent measures. In order of decreasing additional variance accounted for the following variables were entered into the equation for predicting clinical dysfunction: a) evidence of organicity as determined by the dentist, b) severity of maloclussion as determined by the dentist and c) the use of the coping response of "seeking social support". The latter variable was negatively correlated with clinical dysfunction. Thus in predicting both subjective dysfunction and clinical dysfunction the psychological variables contributed to the prediction of symptom severity. This contribution, however, was neither consistent nor all inclusive. The equation for predicting clinical dysfunction is reported at the bottom of Table VII.

Research Question 2: Will the psychometric measures of life events and coping responses improve the prediction of subjective improvement of MPD symptoms at a two month follow-up over the physical measures of maloculussion, duration of symptoms and evidence of organicity in the temporomandibular joint?

Table VIII presents the correlation coefficients between subjective improvement and all of the possible independent measures including clinical dysfunction and subjective dysfunction at intake. None of the dependent measures were significantly correlated with subjective improvement.

Table IX summarizes the discriminant function equation for predicting subjective improvement. For the purposes of this analysis mild improvement was defined as a score of 5 or less whereas moderate to better improvement was defined as a score of 7 or more. Following the stepwise method and with a significance level for inclusion set at .05 it was found that only two variables could be entered into the equation. In order of decreasing significance these variables were a) malocclussion at intake and b) clinical dysfunction at intake - both being variables determined in the dental assessment. The subjective reports of life events and coping responses did not enter into the equation at a significant level. The equation for predicting subjective improvement is reported at the bottom of Table IX.

Research Question 3: Is it possible to identify theoretically defendable factors for the constellation of symptoms reported by MPD patients which have unique relationships with coping strategies and stress?

Table VIII

Correlation Coefficients at Follow-Up

Independent vs Dependent Variable

Independent Variable	Subjective Improvement	Significance
Total Life Events (LESUM)	-.12	.23
COPING RESPONSES		
problem solving (C_1)	-.07	.33
wishful thinking (C_2)	.13	.20
mixed (C_3)	-.06	.34
growth (C_4)	-.13	.21
minimize threat (C_5)	.04	.39
seek social support (C_6)	-.11	.24
self blame (C_7)	-.05	.39
Duration (DUR)	-.16	.16
Organicity (ORG)	-.03	.44
Malocclussion (MAL)	-.30	.03*
Clinical Dysfunction at intake (CTMJ)	.10	.28
Subjective Dysfunction (STMJ) at Intake	.09	.30

* Exceed 0.05 level of significance

Table IX

Discriminant Function at Follow-Up Predicting Subjective Improvement (DSUBJ) from the Independent Variables - Stepwise Method.

Independent Variable	B	Significance
Malocclussion (MAL)	-.31	.007
Clinical Dysfunction at Intake (CTMJ)	.07	.046
Constant	1.63	

...tion: DSUBJ = 1.63 - .31(MAL) + .07(CTMJ)

Multiple R = .43

R Square = .19

Variables included in analysis: duration of symptoms, organicity, malocclussion, total life events, seven coping responses, clinical dysfunction at intake and total subjective symptoms at intake.

This research question was examined through a factor analysis of the eleven subjective dysfunction scales plus two "placebo" scales which were included in the questionnaire. The placebo symptoms were "constipation" and "indigestion" and thus had some face validity as stress measures. There is no support in the literature, however, for these symptoms to be related to MPD. These two variables would not be expected to correlate highly with any of the MPD symptoms. High ratings of these symptoms in MPD patients might indicate that patients are reporting general "somatic dysfunction" rather than TMJ related dysfunction.

The factor analysis is summarized in Table X. Both non-rotated and rotated varimax matrices were generated. The varimax rotation with three factors provided the most theoretically parsimonious matrix. Three distinct factors with eigen values greater than 1.0 appeared in this rotation. Factor one was composed of five scales each of which had weights greater than 0.50. Factor two was composed of six different scales each having weights greater than 0.55. Factor three was composed of the two placebo scales and the clinical index with weights exceeding 0.66.

The first factor accounts for 46% of the total variance in the subjective symptoms. The scales in this factor could be described as PAIN symptoms related to either muscle or vascular tension. Thus jaw pain, low back pain, headaches, light headedness and fainting comprise factor one. The relationship between these symptoms seems to confirm reports that MPD patients also have shoulder pain, back pain and headaches as concomitant symptoms. One might predict that treatment which effected MPD would effect these other symptoms.

The PAIN factor could be theoretically "explained" as a function of muscle tension. The argument may have to be stretched to account for light headedness and fainting although muscular tension which effects blood flow is not an uncommon phenomenon. The literature on ischemic pain supports the notion that severe vascular constriction can result from prolonged muscular tension.

The second factor accounts for 24% of the variance of the symptoms. These symptoms seem to be related to the physical evidence of MPD. Clicking, scraping and locking of the TMJ, and difficulty in opening the TMJ are all related to joint function. A fifth symptom in this factor was fatigue of the jaw muscles which could be perceived as a

Table X

Factor Analysis of Subjective Symptoms
Placebo Symptoms and the Clinical Index

Independent Variable	Factor 1	Factor 2	Factor 3
clicking of TMJ		0.60	
scraping of TMJ		0.54	
tiredness of jaw muscle		0.66	
difficulty in opening wide		0.75	
locking of TMJ		0.70	
pain in shoulder muscles	0.59		
headaches	0.77		
low backpain	0.56		
light headedness	0.70		
fainting	0.59		
pain in jaw muscles	0.68		
indigestion			0.78
constipation			0.66
CLINICAL INDEX			0.70

result of, or response to, joint dysfunction (i.e., the result of the muscles trying to coordinate the jaw to minimize clicking, scraping etc.) Patients appear to perceive jaw fatigue differently from jaw pain. Even though the fatigue symptom falls in this factor, the factor will be interpreted as a measure of DYSFUNCTION in the joint.

The third factor composed of the clinical index plus constipation and indigestion will not be of further theoretical interest but will be briefly considered before proceeding to a discussion of the first two factors. Of note is the fact that the clinical index as determined by the dental assessment, was more closely related to this third factor than to either the PAIN or DYSFUNCTION factor.

What this third factor is measuring could be speculated on. One might interpret indigestion, constipation and dentist rated clinical symptomatology to be "physical" ratings. On the other hand they may be seen to be accessing "somatic" well being. These hypotheses appear very tenuous to the present researcher and to pursue them further would be more conjecture than substantive.

Table XI presents the correlation coefficients for the PAIN factor and the independent variable at intake. Total life events was not significantly correlated with PAIN at intake. Six of the seven areas of coping, however, were correlated with PAIN at the .05 level. Four of these, problem solving, wishful thinking, mixed coping and seeking social support are correlated with the PAIN factor at the .01 level. The nature of the relationship of these coping responses to PAIN does not, however, follow the expected directions. Generally all of the coping responses increase as PAIN severity increases. Only one of these correlations, "minimizing threat" is consistent with the predictions from the MPD literature.

Table XII summarizes the discriminant function equation for predicting PAIN at intake from the independent variables. For the purpose of this analysis mild pain was defined as a score of less than 12 and moderate to severe pain was defined as a score of greater than 13. Following the stepwise method with a significance level for inclusion in the equation set at 0.05 it was found that three coping responses and duration of symptoms could be entered into the equation. In order of decreasing additional variance accounted for the independent variables were a) mixed coping, b) duration of symptoms, c) self blame coping and d) growth coping. Total variance accounted for was 0.35.

Table XI

Correlation Coefficients at Intake

Independent Variables vs the PAIN Factor

Independent Variable	PAIN factor	Significance
Total Life Events	.14	.11
COPING RESPONSES		
problem solving (C_1)	.19	.04*
wishful thinking (C_2)	.28	.007*
mixed coping (C_3)	.42	.000*
growth (C_4)	.18	.05
minimizing threat (C_5)	.20	.04
seeking social support (C_6)	.28	.007*
blaming self (C_7)	-.06	.29
Duration of symptoms	.29	.005*
Evidence of organicity	.01	.45
Severity of malocclussion	-.06	.29

* Exceed 0.05 level of significance

Table XII

Discriminant Function at Intake

Predicting the PAIN Factor at Intake from

the Independent Variables

Independent Variable	B	Significance
Coping Response: Mixed (C_3)	.67	.000
Duration of symptoms (DUR)	.08	.003
Coping Response: Self blame (C_7)	-.14	.003
Coping Response: growth (C_4)	.05	.05
Constant	1.0	.000

Equation: PAIN = .67 + .10(C_3) + .08(DUR) - .14(C_7)

Multiple R = .59

R Square = .35

Variables included in analysis: duration of symptoms, organicity, malocclussion, total life events, seven coping responses, clinical dysfunction at intake and total subjective symptoms at intake.

Table XIII presents the correlation coefficients for the DYSFUNCTION factor and the independent variables at intake. Total life events was negatively, although not significantly, correlated with DYSFUNCTION. None of the coping responses correlated with DYSFUNCTION. Only evidence of organicity and severity of malocclussion significantly correlated and these were highly significant.

Table XIV summarizes the discriminant function for predicting DYSFUNCTION at intake from the independent variables. For the purpose of this analysis mild DYSFUNCTION was arbitarily defined as a score of less than 12 and moderate to severe DYSFUNCTION was defined as a score of 12 or more. Following the stepwise method with a significance level for inclusion in the equation set at 0.05 it was found that only severity of malocclussion could be entered into the equation. Total variance accounted for was only 0.10.

D. Summary of Results

The research questions can all be answered in the affirmative. The analysis was complicated by the fact that the two measures of dysfunction were very poorly correlated with each other. Clinical dysfunction as measured by the dentist was found to be poorly correlated, statistically, to the patient's self report of dysfunction.

Life events were remarkably uncorrelated with severity of symptoms whether measured by the dentist or rated by the patient. Similarly the stress coping responses of "self blame" and "seeking social support" were not significantly correlated with symptom severity. Only the coping response of "minimizing threat" was significantly correlated with subjective dysfunction as predicted.

When considering the utility of the non-psychological measures in predicting symptom severity it was determined that the dental assessment of organicity was the strongest predictor of both subjective and clinical dysfunction. The coping responses of "minimizing threat" and "seeking social support" also significantly contributed to the prediction of symptom severity but in an inconsistent manner. The former was useful in predicting subjective dysfunction whereas the latter was useful in predicting clinical dysfunction.

Table XIII

Correlation Coefficients at Intake

Independent Variables vs the DYSFUNCTION Factor

Independent Variable	Dysfunction Factor	Significance
Total Life Events	-.14	.10
COPING RESPONSES		
problem solving (C_1)	.20	.04*
wishful thinking (C_2)	.03	.40
mixed coping (C_3)	.13	.12
growth (C_4)	.16	.07
minimizing threat (C_5)	.14	.10
seeking social support (C_6)	.01	.45
blaming self (C_7)	-.02	.41
Duration of symptoms	.03	.39
Evidence of organicity	.27	.006*
Severity of malocclussion	.31	.002*

* Exceed 0.05 level of significance.

Table XIV

Discriminant Function at Intake

Predicting the DYSFUNCTION Factor at Intake

from the Independent Variables

Independent Variable	B	Significance
Evidence of molocclussion (MAL)	.16	.004
Constant	1.07	.000

Equation: DYSFUNCTION = 1.07 + .16(MAL)

Multiple R = .31

R Square = .10

Variables included in analysis: duration of symptoms, organicity, malocclussion, total life events, seven coping responses, clinical dysfunction at intake and total subjective symptoms at intake.

A factor analysis of the "subjective dysfunction" subscales yields two distinct factors of direct clinical interest. The first factor, accounting for nearly half of the total variance in dysfunction seems to be a measure of pain. The Pain factor is significantly correlated with all of the seven coping styles in a manner of inconsistent with the present researcher's hypotheses. Together with duration of symptoms, these coping styles predict 35 percent of the variance in PAIN.

The second factor accounts for one half of the remaining variance and seems to measure joint dysfunction. This factor has little relationship with coping or stress level as measured in this study. The dentists' assessments of evidence for organicity in the joint and malocclussion of the teeth were significantly correlated with DYSFUNCTION. Together they accounted for only 10 percent of the variance in DYSFUNCTION.

V. DISCUSSION

This chapter addresses three general areas of discussion. First the difficulties encountered in running the study are reviewed with a focus on how these difficulties created sampling bias. Second the findings of the present study are discussed and the implications of these findings are cited. Finally recommendations for improving the present research design are made and hypotheses for future exploration are generated.

A. Sampling Difficulties

The incidence of MPD in the general population has been found to be around 25% (Helkimo, 1974, 1976). If we assume that 10% of those with symptoms seek treatment (a generous estimate - see Marbach and Lupton, 1978) we are sampling less than three percent of the general population. More importantly this sampling bias is determined by severity of symptoms. Thus patients in this study are self selected according to one of the dependent measures - subjective symptoms. This reduces the variability of the dependent measure and limits attempts to account for this variance.

When the researcher began this study he contacted approximately 30 dentists in the province of Alberta who treated MPD. Of these 8 agreed to particpate in the study. A few months later a province wide advertisement of the project solicited four additional dentists. Overall less than two percent of the provinces' dentists participated in the study.

Within their offices there was clearly some bias in the selection of patients who completed the questionnaire. From the dentists' descriptions, patients who did not fill out the questionnaire were those who said they did not have time. It might be hypothesized that those with high stress levels were self-selected from the study. This bias would probably effect the variability of the independent measures - particularly the life events scores. A notable exception to this trend was found in one office where patients were required to complete the questionnaire as part of the intake procedure. This one office accounted for approximately 40% of the sample. So that, in spite of their protestations, some "stressed out" subjects were included in the sample. It is possible, however, that required participation did not remediate the problem since 15% of the returned questionnaires were not scorable. A few patients filled out the first page and left the remainder blank, others left out important sections. The resistance of these patients, then

may have been transferred from the dentist to the questionnaire.

Approximately another 30% of the patients included in the intake sample were unavailable for the follow-up interview. This occurred for various reasons, the most common being that they could not be contacted at the telephone numbers given. Others had not returned to the dentist for treatment or had lost their splint. Only patients who had complied with the splint therapy treatment were included in the follow-up assessment.

The possibility that patients who dropped out of the study were somehow different from the remainder of the subjects was examined through a multivariate analysis of variance (MANOVA) between these two groups. Table XV summarizes the MANOVA for the non-psychological variables, age of patient, duration of symptoms, evidence of organicity, severity of malocclussion, subjective symptom severity and clinical dysfunction. The overall F was significant allowing the univariate test of each variable. Both age of patient and clinical dysfunction were significantly different between the two groups. An examination of means indicates that patients who dropped out of the study were younger and had less clinical symptomatology compared with those who remained in the study.

An interpretation of the finding on age differences could be that the younger patients were more mobile than the older patients and thus were more difficult to locate at follow-up. The fact that they had less clinical symptomatology is more difficult to account for. We can make the logical interpretation that those with less severe symptoms are less motivated to pariticipate in treatment. It is then possible to equate, at least in the patients mind, research with therapy and generalize lack of motivation to participation in the present study. Another interpretation is that these patients were "cured" and not interested in follow-up. The important conclusion from this analysis is that the variance in one of the dependent measures may have been reduced through attrition.

A second analysis of these two groups on the psychological measures found no significant differences (Table XVI). Even though life event scores were 40% higher for the attrition group this did not prove statistically significant in the MANOVA.

Although some of the preceeding difficulties were anticipated, the extent of the bias in dentist participation, and patient compliance were not predicted. The overall impact of these sampling difficulties probably contributed to decreased variability and

Table XV

Multivariate Analysis of Variance Summary Table

Follow-up Group (G_1) versus Attrition Group (G_2) at Intake

On the Non-Psychological Variables

Hotellings multivariate test of significance

s=1, m=2, N=37 1/2 (G_1=60, G_2=25)

Value	F	df	Significance of F
0.26	3.31	(6,77)	.008

Univariate F-tests with (1,82) degrees of freedom

Variable	F	Significance
Age of patient	5.63	.02
Duration of Symptoms	0.19	.66
Evidence of Organicity	2.41	.13
Severity of Malocclussin	0.47	.50
Clinical Dysfunctin	8.84	.004

Table XVI

Multivariate Analysis of Variance Summary Table
Follow-up (G_1) versus Attrition Group (G_2) at Intake
on the Psychological Variables

Hotellings multivariate test of significance
s=1, m=3, N=37 (G_1=60, G_2=25)

Value	F	df	Significance of F
0.12	1.27	8,76	0.31

Variables included in this analysis: life events, seven coping respones.

consequently variance in both the dependent and independent measures. The fact that these biases were, in most cases, systematic leads the researcher to be cautious in his interpretation of the results from the study. The decrease in variability may also account for the absence of support for many of the hypothesized relationships.

B. Clinical versus Subjective Symptoms

The lack of a significant correlation between dentist and patient assessment of MPD is perhaps the most unexpected finding of the study. The shared variance of 3% suggests that how the dentist diagnoses MPD is much different from the patient's decision to seek treatment. Alternatively the scales which were developed to assess both subjective and clinical symptoms may simply be unreliable measures of MPD.

The possibility that the subjective scales were not measuring a common underlying variable was also explored. These findings indicate that, in fact, MPD as rated by the patient is composed of two distinct factors. These factors are the actual physical dysfunction of the joint (factor DYSFUNCTION) and the pain which emanates from the joint and spreads to the head and down the spine (factor PAIN).

Notably the clinical index was found to be positively correlated with DYSFUNCTION ($r=0.36$) but negatively correlated with PAIN ($r=-0.23$). Even higher correlations with the clinical index were found with the PLACEBO factor (indigestion and constipation) which were expected to have no relationship with MPD. On the basis of this post hoc analysis one is left questioning the statistical utility of the clinical index. As noted earlier, part of this may be explained by the fact that the attrition group had significantly lower clinical symptoms. Also the poor correlations with any of the subjective scales may be due, in part, to the number of dentists who determined the ratings on this index. Markedly different criteria for assessing mobility, joint function and pain may be a result of different training, orientation or clinical experience.

The unreliability and questionable validity of this measure should not be seen as reflective of dental assessments but specifically a property of the clinical index as used in this study. The particular assessment style of each dentist probably includes much more than that sampled by this questionnaire. Also, statistical weakness does not necessarily preclude the clinical utility of the index in planning a treatment approach.

C. Life Events

Life events was remarkably unrelated to any measure of MPD. A positive correlation between MPD and life events is consistently supported in the literature and one would expect that severity of symptoms would be related to "severity" of environmental stress. Again the simple explanation of these negative findings is in the limitations of the assessment instrument.

The differences between the previous studies and the present research may also account for the absence of collaborative results. The 102 item life events scale incorporated into the present design is much more comprehensive than the 43 item Social Readjustment Rating Scale. Although improvements in item selection supported the use of the former scale there has been little research evidence of its validity or reliability.

The fact that the present scale did not focus exclusively on serious, negative stressors may have allowed many patients to deny or distort environmental stress. For example, it is much easier to deny a "serious family arguement" than "spouse died". As described in Chapter II one of the predicted coping styles of MPD patients is to deny or minimize stress. If this coping style is used it can also be seen as inconsistent with the admission of stressful experiences.

The sampling bias described previously in this chapter probably also accounts for the non-relationship between life events and MPD. It is conceivable that self selection at both ends of the continuum of life events affects the present sample. Only those with serious dysfunction and possibly high levels of stress sought treatment. Also, at the other extreme, only those whose level of stress did not preclude the time or energy to fill out the questionnaire were included in the sample. The remaining subjects may have been "highly stressed" compared to the norm of the general population but the "within" group variance may have been minimal. This hypothesis is supported by the fact that the attrition group had a 40 per cent higher Life Events score than the remainder of the sample.

For the present, however, the hypothesis of a correlation between life events and severity of MPD must be rejected.

D. Coping Responses

Two of the coping responses were found to have utility in predicting symptom severity at intake. "Minimizing threat" improved the discrimination between mild and moderate-to-severe subjective symptoms. The more the subject "minimized threat" the greater the symptoms at intake. The total variance accounted for, however, with this variable plus duration of symptoms and evidence of organicity was only 0.15.

Similarly, the coping response of "seeking social support" improved the discrimination between mild and moderate-to-severe clinical symptoms. The less the subject sought social support the greater the symptoms. Total variance accounted for with this variable plus malocclussion and organicity was 0.41.

It should be noted that there were some marginal relationships with other coping responses and symptom severity. "Problem solving" correlated with subjective dysfunction ($r=0.19$, $p=.04$) but did not enter into the discriminant function. Also "self blame" correlated with clinical dysfunction ($r=.16$, $p=.09$) and did not enter into the discriminant function.

From the correlations described above we get a tentative picture of a possible "coping" profile of the MPD patient. MPD patients may tend to rely on themselves in stressful situations. They blame themselves for their difficulties and do not seek support from others to solve their problems. They could be described as showing an internal locus of control (Rotter, 1966). They tend to minimize problems - perhaps seeing them as solvable - or simply showing a pervasive optimism. The fact that they have knowledge of several problem solving styles could also be seen as consistent with this "supernormal" profile. These self-reliant people must see themselves as having the answers to problems. A critical question would be how effective the problem solving of MPD patients is? It is possible that the "broad shoulder" on these people may lead to taking on more stress and eventually overloading their ability to adapt?

An extension of this argument may account for the absence of consistent psychological information on MPD patients. These patients may be very reluctant to admit weakness to an interviewer or on a questionnaire for that matter. Or it may be that MPD patients are generally "intact" individuals who, because of their coping styles, take on more than they can handle. Several telephone interviews come to mind where subjects reported

they had only a few minutes between several responsibilities they were incurring simultaneously.

E. PAIN versus DYSFUNCTION

The factor analytic determination of two types of MPD symptoms provides another picture of the coping responses associated with symptom severity. Subjects appear to be aware of two general but separate symptom patterns. They report DYSFUNCTION in the joint as one category of symptoms and PAIN in the jaw, neck, back and head as another category. The fact that the dependent variable "subjective symptoms" was composed of two independent symptom patterns may account for the absence of significant relationships with the independent measures.

An investigation of the relationship between PAIN and the independent measures found that six of the coping responses were significantly correlated with PAIN but in an inconsistent way. Three of these entered into a discriminant function along with duration of symptoms to account for 35% of the variance in the PAIN factor. The general pattern of these relationships suggests that the PAIN factor is related to a general increase in the reporting of all coping responses. Two hypotheses generated from this interpretation are that MPD patients either see themselves as "having all the answers" or their experience with pain has resulted in their need to access all of their coping resources.

The DYSFUNCTION factor showed no relationship to the coping strategies or life events. Both evidence for organicity and severity of malocclussion correlated highly with DYSFUNCTION but only "malocclussion" accounted for significant variance in the discriminant function analysis. The conclusion by many researchers that malocclussion causes MPD is not surprising considering its relationship to DYSFUNCTION. Researchers who attend to the physical symptoms of the TMJ would probably find the relationships described above.

Support for this interpretation comes from an examination of the clinical index in the factor analysis of symptoms reported in Chapter IV. The clinical index had a low negative correlation with the PAIN factor and a higher positive correlation with the DYSFUNCTION factor. This is somewhat surprising when one considers the fact that three of the five scales in the clinical index direct the dentist to assess pain. We must assume

that the dentist attends to the physical side of MPD more than the pain experienced by the patient. The fact that the training of dentists and traditional focus of dentistry has been on repairing physical dysfunction may account for these findings. The present study suggests that many researchers also attend to the dysfunction in the TMJ to the exclusion of the experience of pain and subsequently conclude that malocclussion is the primary determinant of etiology and treatment.

If researchers were to attend to only the pain associated ith MPD it is probable that only psychological determinants would be found. Clearly research must focus on both aspects of the disorder. It would appear that the present findings are consistent with the psychophysiological theory proposed by Rugh and Solberg (1976).

F. Suggestions for Further Research

Further research into the psychological correlates of MPD should address the difficulties cited above. The sampling problem may be somewhat corrected by shortening the questionnaire. The life events questionnaire provided little useful information predicting symptom severity or response to treatment and the focus probably should shift to clarifying the coping responses used by MPD subjects. Another method of decreasing the sampling bias would be to locate dentists who could require patients to complete the questionnaire. In the present study one office was able to get an 85% participation rate. Even this level could be improved by personal interviews of patients.

Examination of the reliability and validity of the present coping questionnaire would be a first step in clarifying the nature of coping deficits, if any. This might be achieved by observations of subjects who report various coping responses. Presentations of stressful situations or the examination of their response to hypothetical stressful events could provide some validity data. Having spouses fill out questionnaires on each other may be a useful measure of reliability. If both husband and wife agree on a coping strategy it is more likely to be actually used.

The question of the effectiveness of coping strategies was alluded to earlier. It may be more valuable to know the effectiveness of a strategy than whether it is reported to be used. Many researchers believe that all types of coping are effective in different situations.

Richard Lazarus (personal communication) reports that current research suggests that flexibility in coping strategies is the most adaptive style. This hypothesis could be investigated through expanding the questionnaire to include opportunities to report the process of coping rather than specific responses. One might measure the effectiveness of a style or the flexibility in using strategies by giving MPD patients real stressful situations to respond to.

Finally the determination of distinct PAIN and DYSFUNCTION symptomatology needs to be explored further. A priori predictions of these factors may confirm their existence. Also of interest is the finding that coping responses are related to PAIN and malocclussion is related DYSFUNCTION. It is possible that an assessment of the relative balance of these two symptoms could be a valuable diagnostic approach. MPD patients who present with relatively severe PAIN may have more psychological problems and could benefit from retraining in coping strategies. Those who present with more DYSFUNCTION might require only occlussal treatment. This could be explored by offering retraining in coping styles to groups of MPD patients and predicting that those who presented with more severe pain would benefit most.

Alternatively these patients seem to be increasing all of their coping responses as PAIN severity increases. It may be fruitful to compare coping responses in MPD patients with other pain patients to determine similarities and differences. The differences may be in reaction to the pain or as predeterminants of the particular pain response.

Overall the general approach in this study of using multivariate research design should be maintained. If any conclusion can be made from the present study it is that MPD is multidetermined and even the determinants probably effect different aspects of MPD symptomatology.

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APPENDIX I

RESEARCH STUDY ON FACIAL PAIN QUESTIONNAIRE

Researchers from the University of Alberta are conducting a study of pain. Frank McGrath of the Department of Educational Psychology and Dr. Norman Thomas of the Department of Dentistry are interested in your responses to the enclosed questionnaire. The 180 items ask about various areas of your life and will provide us with valuable information for helping people like yourself. The questionnaire should take only 30 minutes to complete. Please complete it before leaving the office today.

In about two months we will be contacting you again. At that time, we would like to obtain additional information.

Your co-operation in truthfully answering these questions will help us develop better treatment procedures for pain sufferers. Your specific responses will be kept completely confidential and we will destroy the forms after adding up your scores. We will need your name, address and telephone number so that you can be contacted for the second questionnaire.

If you require further information please contact:

Frank W. McGrath
6-145h Education North
University of Alberta
T6G 2E1

Phone: Work: 432-3226 / Res.: 435-6686

Name: _____

Address: _____

Telephone: _____

PLACE A CHECK (X) AT THE APPROPRIATE ANSWER

1. Age:

   16-25 __a 26-35 __b 36-45 __c 46-55 __d 55+ __e

2. Martial Status:

   Single __a

   Common-law __b

   Married (first marriage) __c

   Divorced __d

   Remarried __e

3. How many months have you had the pain?

   less than six __a 6-11 __b 12-17 __c 18-24 __d more than 24 __e

4. On a scale from a (none) to e (extreme) rate the present severity of the following symptoms?

a) Pain of the jaw muscles	_a	_b	_c	_d	_e
b) Clicking in front of ear	_a	_b	_c	_d	_e
c) Scraping sound in front of ear	_a	_b	_c	_d	_e
d) Stiffness or fatigue of jaw muscles	_a	_b	_c	_d	_e
e) Difficulty in opening the mouth wide	_a	_b	_c	_d	_e
f) Locking of the jaw	_a	_b	_c	_d	_e
g) Pain in shoulder muscles	_a	_b	_c	_d	_e
h) Headaches	_a	_b	_c	_d	_e
i) Low back pain	_a	_b	_c	_d	_e
j) Light headedness	_a	_b	_c	_d	_e
k) Fainting	_a	_b	_c	_d	_e
l) Indigestion / stomach cramps	_a	_b	_c	_d	_e
m) Constipation / diarrhea	_a	_b	_c	_d	_e

LIFE EVENTS SCALE

Directions: Indicate which of the following events have occurred to you in the past twelve months. Check (x) all items which apply to you.

		Yes	
	School		
1.	Started school or a training program after not going	___	34
2.	Changed school or training programs	___	26
3.	Graduated from school or training program	___	32
4.	Had problems in school or in training program	___	27
5.	Failed school training program	___	30
6.	Did not graduate from school or training program	___	30
	Work		
7.	Started work for the first time	___	37
8.	Returned to work after not working for a long time	___	35
9.	Changed jobs for a better one	___	47
10.	Changed jobs for a worse one	___	36
11.	Changed jobs for one that was not better and no worse than the last one	___	25
12.	Had trouble with a boss	___	32
13.	Demoted at work	___	38
14.	Found out that was not going to be promoted at work	___	35
15.	Conditions at work got worse, other than demotion or trouble with the boss	___	32

		Yes	
16.	Promoted	___	37
17.	Had significant success at work	___	35
18.	Conditions at work improved, not counting promotion or other personal successes	___	32
19.	Laid off	___	33
20.	Fired	___	41
21.	Started a business or profession	___	47
22.	Expanded business or professional practice	___	48
23.	Took on a greatly increased work load	___	41
24.	Suffered a business loss or failure	___	51
25.	Sharply reduced work load	___	25
26.	Retired	___	46
27.	Stopped working, not retirement, for an extended period	___	46
	Love and Marriage		
28.	Became engaged	___	41
29.	Engagement was broken	___	31
30.	Married	___	50
31.	Started a love affair	___	38
32.	Relations with spouse changed for the worse, without separation or divorce	___	53

		Yes	
33.	Married couple separated	___	52
34.	Divorce	___	63
35.	Relations with spouse changed for the better	___	52
36.	Married couple got together again after separation	___	56
37.	Marital infedelity	___	56
38.	Trouble with in-laws	___	31
39.	Spouse died	___	82
	Having Children		
40.	Became pregnant	___	42
41.	Birth of first child	___	58
42.	Birth of a second or later child	___	45
43.	Abortion	___	37
44.	Miscarriage or stillbirth	___	46
45.	Found out that cannot have children	___	52
46.	Child died	___	104
47.	Adopted a child	___	46
48.	Started menopause	___	34
	Family		
49.	New person moved into household	___	30

		Yes	
50.	Person moved out of the household	___	33
51.	Someone stayed on in the household after he was expected to leave	___	29
52.	Serious family argument other than with spouse	___	26
53.	A change in the frequency of family get togethers	___	23
54.	Family member other than spouse or child dies	___	46
	Residence		
55.	Moved to a better residence or neighborhood	___	44
56.	Moved to a worse residence or neighborhood	___	46
57.	Moved to a residence or neighborhood no better or no worse than the last one	___	24
58.	Unable to move after expecting to be able to move	___	31
59.	Built home or had one built	___	55
60.	Remodelled a home	___	31
61.	Lost a home through fire, flood, or other disaster	___	41
	Crime and Legal Matters		
62.	Assaulted	___	38
63.	Robbed	___	31

		Yes	
64.	Accident in which there were no injuries	___	25
65.	Involved in a lawsuit	___	41
66.	Accused of something for which a person could be sent to jail	___	49
67.	Lost driver's licence	___	25
68.	Arrested	___	48
69.	Went to jail	___	57
70.	Got involved in a court case	___	30
71.	Convicted of a crime	___	54
72.	Acquitted of a crime	___	47
73.	Released from jail	___	50
74.	Didn't get out of jail when expected	___	47
	Finances		
75.	Took out a mortgage	___	32
76.	Started buying a car, furniture, or other large purchase on installment plan	___	26
77.	Foreclosure of mortgage or loan	___	46
78.	Repossession of car, furniture, or other items bought on installment plan	___	29
79.	Took a cut in wage or salary with a demotion	___	40

		Yes	
80.	Suffered financial loss or loss of property not related to work	___	45
81.	Went on welfare	___	42
82.	Went off welfare	___	35
83.	Got substantial increase in wage or salary without a promotion	___	35
84.	Did not get expected wage or salary increase	___	34
85.	Had financial improvement not related to work	___	52

Social Activities

86.	Increased church or synagogue, club, neighborhood, or other organizational activities	___	27
87.	Took a vacation	___	27
88.	Was not able to take planned vacation	___	22
89.	Took up new hobby, sport, craft or recreational activity	___	28
90.	Dropped hobby, sport, craft, or recreational activity	___	18
91.	Acquired a pet	___	16
92.	Pet died	___	20
93.	Made new friends	___	25

		Yes	
94.	Broke up with a friend	___	33
95.	Close friend died	___	46
	Miscellaneous		
96.	Entered the armed services	___	41
97.	Left the armed services	___	36
98.	Took a trip other than a vacation	___	25
	Health		
99.	Physical health improved	___	56
100.	Physical illness	___	67
101.	Injury	___	56
102.	Unable to get treatment for illness or injury	___	61

COPING QUESTIONNAIRE

The purpose of this questionnaire is to find out the kinds of situations that trouble people in their day-to-day lives, and how people deal with them.

Part I

Directions: Take a few moments and think about the event that has been the most stressful for you during the last month. By *"stressful"* we mean a situation which was difficult or troubling to you, either because it made you feel bad or because it took effort to deal with it. It might have been something to do with your family, with your job, or with your friends.

In the space below, please describe the most stressful event of the past month. Describe what happened and include details such as the place, who was involved, what you did, what made it important to you, and perhaps what led up to the situation. The situation could also be one that is going on right now as well as one that has already happened. Don't worry about making it into an essay -- just put down the things that come to you.

Part II

Directions: Thinking about the situation you have just described, put a check in the *"Yes"* or *"No"* column for each item, depending on whether that item applied to you.

(To help keep the situation in mind): I am talking about the situation in which

_____

_____

_____

		Yes	No
1.	Just concentrated on what you had to do next - the next step	___	___
2.	You went over the problem again and again and again in your mind to try to understand it.	___	___
3.	Turned to work or substitute activity to take your mind off things.	___	___
4.	You felt that time would make a difference, the only thing to do was to wait.	___	___
5.	Bargained or compromised to get something positive from the situation.	___	___
6.	Did something which you thought wouldn't work, but at least you were doing something.	___	___
7.	Got the person responsible to change his or her mind.	___	___
8.	Talked to someone to find out more about the situation.	___	___
9.	Blamed yourself.	___	___
10.	Concentrated on something good that could come out of the whole thing.	___	___
11.	Criticized or lectured yourself.	___	___
12.	Tried not to burn your bridges behind you, but leave things open somewhat.	___	___
13.	Hoped a miracle would happen.	___	___
14.	Went along with fate; sometimes you just have bad luck.	___	___
15.	Went on as if nothing had happened.	___	___
16.	Felt bad that you couldn't avoid the problem.	___	___

		Yes	No
17.	Kept your feelings to yourself.	___	___
18.	Looked for the "silver lining," so to speak; tried to look on the bright side of things.	___	___
19.	Slept more than usual.	___	___
20.	Got mad at the people or things that caused the problem.	___	___
21.	Accepted sympathy and understanding from someone.	___	___
22.	Told yourself things that helped you to feel better.	___	___
23.	You were inspired to do something creative.	___	___
24.	Tried to forget the whole thing.	___	___
25.	Got professional help and did what they recommended.	___	___
26.	Changed or grew as a person in a good way.	___	___
27.	Waited to see what would happen.	___	___
28.	Did something totally new that you never would have done if this hadn't happened.	___	___
29.	Tried to make up to someone for the bad thing that happened.	___	___
30.	Made a plan of action and followed it.	___	___
31.	Accepted the next best thing to what you wanted.	___	___
32.	Let your feelings out somehow.	___	___
33.	Realized you brought the problem on yourself.	___	___
34.	You came out of the experience better than when you went in.	___	___
35.	Talked to someone who could do something concrete about the problem.	___	___
36.	Got away from it for a while; tried to rest or take a vacation.	___	___
37.	Tried to make yourself feel better by eating, drinking, smoking, taking medication, etc.	___	___
38.	Took a big chance or did something very risky.	___	___

		Yes	No
39.	Found new faith or some important truth about life.	___	___
40.	Tried not to act too hastily or follow your first hunch.	___	___
41.	Joked about it.	___	___
42.	Maintained your pride and kept a stiff upper lip.	___	___
43.	Rediscovered what is important in life.	___	___
44.	Changed something so things would turn out all right.	___	___
45.	Avoided being with people in general.	___	___
46.	Didn't let it get to you; refused to think too much about it.	___	___
47.	Asked someone you respected for advice and followed it.	___	___
48.	Kept others from knowing how bad things were.	___	___
49.	Made light of the situation; refused to get too serious about it.	___	___
50.	Talked to someone about how you were feeling.	___	___
51.	Stood your ground and fought for what you wanted.	___	___
52.	Took it out on other people.	___	___
53.	Drew on your past experiences; you were in a similar situation before.	___	___
54.	Just took things one step at a time.	___	___
55.	You knew what had to be done, so you doubled your efforts and tried harder to make things work.	___	___
56.	Refused to believe that it had happened.	___	___
57.	Made a promise to yourself that things would be different next time.	___	___
58.	Came up with a couple of different solutions to the problem.	___	___
59.	Accepted it, since nothing could be done.	___	___
60.	Wished you were a stronger person -- more optimistic and forceful.	___	___
61.	Accepted your strong feelings, but didn't let them interfere with other things too much.	___	___
62.	Wished that you could change what had happened.	___	___

		Yes	No
63.	Wished that you could change the way you felt.	___	___
64.	Changed something about yourself so that you could deal with the situation better.	___	___
65.	Daydreamed or imagined a better time or place than the one you were in.	___	___
66.	Had fantasies or wishes about how things might turn out.	___	___
67.	Thought about fantastic or unreal things (like the perfect revenge or finding a million dollars) that made you feel better.	___	___
68.	Wished that the situation would go away or somehow be over with.	___	___
69.	Did something different from any of the above.	___	___

_____

_____

_____

In general, is this situation one

a. that you could change or do something about?	___	___
b. that must be accepted or gotten used to?	___	___
c. that you needed to know more about before you could act?	___	___
d. in which you had to hold yourself back from doing what you wanted to do?	___	___

If you checked "Yes" more than once, underline the statement which best describes the situation.

FOR DENTISTS USE ONLY

Please circle 0, 1, 3, or 5 for each symptom

A. Symptom: Impaired range of movement / mobility

Criteria:

Normal range of movement	1
Slightly impaired mobility	2
Severely impaired mobility	3

B. Symptom: Impaired TM-joint function

Criteria:

Smooth movement without TM-joint sounds and deviation on opening or closing movements 2mm	1
TM-joint sounds in one or both joints and / or deviation >2mm on opening or closing movements	2
Locking and / or luxation of the TM-joint	3

C. Symptom: Muscle pain

Criteria:

No tenderness to palpation in masticatory muscles	1
Tenderness to palpation in 1-3 palpation sites	2
Tenderness to palpation in 4 or more palpation sites	3

D. Symptom: Temporomandibular joint pain

Crtieria:

No tenderness to palpation	1
Tenderness to palpation laterally	2
Tenderness to palpation posteriorly	3

E. Symptom: Pain on movement of the mandible

Criteria:

No pain on movement	1
Pain on 1 movement	2
Pain on 2 or more movements	3

F. Symptom: Radiographic evidence of organic deterioration of TMJ (if radiographs have been done) -- check here ___ if not applicable

Criteria:

No organic deterioration	1
Slight organic deterioration	2
Mild organic deterioration	3
Severe organic deterioration	4

G. Symptom: Malocclusion

Criteria:

No malocclusion	1
Mild malocclusion	2
Moderately severe malocclusion	3
Severe malocclusion	4

H. Treatment Plan - check one or prioritize the three most important treatments you plan to use. (please specify drugs if used.)

Splint therapy ___
Removable prosthetics ___

Fixed prosthetics ___
Orthodontics ___
Occlusal Equilibration ___
Physiotherapy ___
Chiropraxy ___
Biofeedback ___
Relaxation ___

Counselling ___
Pharmacology
a) _____
b) _____
Kinesiography ___
Myomonitoring ___
Vitamins ___
Nutrition
Surgery ___
Other - please specify _____

I. Sum A + B + C + D + E = _____.

APPENDIX II
TELEPHONE FOLLOW-UP QUESTIONNAIRE

Name: _____

Date: _____

Telephone: _____

Dentist: _____

1. Compared to when you filled out the questionnaire the symptoms are

   a. very much worse ___
   b. quite a bit worse ___
   c. a little worse ___
   d. the same ___
   e. a little better ___
   f. quite a bit better ___
   g. very much better ___
   h. completely better ___

2. Have you had any other treatment for TMJ?

   _____

3. On a scale from 1 to 5 what is the present severity of the following symptoms?

a) Pain of the jaw muscles	_a	_b	_c	_d	_e
b) Clicking in front of ear	_a	_b	_c	_d	_e
c) Scraping sound in front of ear	_a	_b	_c	_d	_e
d) Staffiness or fatigue of jaw-muscles	_a	_b	_c	_d	_e
e) Difficulty in opening the mouth wide	_a	_b	_c	_d	_e
f) Locking of the jaw	_a	_b	_c	_d	_e
g) Pain in shoulder muscles	_a	_b	_c	_d	_e
h) Headaches	_a	_b	_c	_d	_e
i) Low back pain	_a	_b	_c	_d	_e
j) Light headedness	_a	_b	_c	_d	_e
k) Fainting	_a	_b	_c	_d	_e
l) Indigestion/ stomach problems	_a	_b	_c	_d	_e
m) Constipation/diarrhea	_a	_b	_c	_d	_e