TREATMENT OF ACUTE ARTHROGENOUS PAIN IN THE HUMAN TEMPOROMANDIBULAR JOINT WITH AN ORAL ORTHOPAEDIC APPLIANCE

by

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ABSTRACT

The biomechanical events that accompany functional loading of the human mandible are poorly understood. Computer simulations has emerged as an indirect way to demonstrate the principles of jaw mechanics. The modelling of condylar load distributions for various clenching tasks has lead to the observations that deviations in form and osteoarthritic changes as most commmonly found in the central and lateral regions of the articulation reflect habitual compressive loading of the temporomandibular joints.

Speculation has existed that compressive stresses as measured during simulated unilateral tooth clenching, offer a functional correlate for regional differences in articular pathology. It has been suggested due to the indirect measuring of the effects of these loads, that well-known progressive deterioration of the discs and articular surfaces are largely brought about by persistent non-working side compression of the temporomandibular joint structures. This in the short term is believed to lead to arthralgia of sufficent magnitude that patients often seek treatment by dental clinicians.

In the first study, an existing 3D FEM model of the human mandible, modified to include an opposing oral orthopaedic device, was utilized to test for the effect of two clenching tasks on the compressive stresses measured at the level of the

condylar heads when an orthopaedic dental appliance with unilateral occlusal contacts was placed between the teeth. It was found that the level of compressive stress in the contralateral side is twice that of the working side joint.

A clinical study was then performed in order to test the hypothesis that in acute articular pathology, the use of an orthopaedic appliance designed to reduce the load to the painful joint can positively influence the resolution of arthrogenous pain in the short term. Specifically, it was proposed that this could be achieved with an appliance designed with unilateral occlusal support.

A group of patients diagnosed with unilateral articular pathology were randomized into two treatment groups, one was treated with a conventional flat appliance, the other with unilateral occlusal contacts removed from the side contralateral to the painful joint. In both cases, a VAS was used to assess pain in both joints with and without the mechanical stimulus provided by biting on a force transducer placed between the teeth. Patients were followed for 3 weeks after initial testing. It was found that painful symptoms improved in the range, as measured by the VAS, and in the degree of pain for subjects treated with the unilaterally supported appliance. It was also discovered that the range and magnitude of the bite force increased concomittantly with the reduction in painful symptoms.

Collectively the studies suggest that muscle activity is sensitive to differences in occlusal support offered by an intra-oral appliance, and that such a device can be used to modify articular loading and/or to control muscle use in such a way as to speed up the resolution of painful intrarticular symptoms in TMJ patients.

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INTRODUCTION

The human masticatory apparatus can be lookedi upon as a biomechanical system of complex interrelated components made up of muscles, bones, ligaments, nerves and teeth, all of which play a role in the proper function and health of the being. The mandible is analogous to a limb that crosses the midline with over 27 muscles influencing its action with tendons and ligaments of inmeasurable numbers and complexity affecting its functions. Muscles are recruited as required to move the bony components against gravity with the aid of other passive forces and function to provide forces, directed in such a manner as to maintain the articulation of the temporomandibular joint in an optimum position to withstand loads, and to provide mobility.

The craniomandibular articulation with its component parts is vital in determining general posture and movement. One can easily speculate, that with changes in morphology, excessive muscle tensions, parafunctional movements, persistent loading of connective or bony tissues and/or with trauma, the whole articulation can progressively destabilize or break down, leading to the malfunction of this most crucial apparatus.

Temporomandibular disorders (TMD) is a broad term that encompasses a variety of clinical conditions that involve the stomatognathic system.

Although TMD has been viewed as one syndrome, current thought increasingly supports the view that what is now termed as TMD or by some CMD (Craniomandibular Disorders) is a variety of disorders often of multifactorial etiology, and presenting with overlapping signs and symptoms. Unfortunately, many studies in the past have not differentiated between the broad terms for classification of these disorders and what is now believed to be a subgroup of these, temporomandibular joint disorders. The signs and symptoms of these disorders manifest themselves in the masticatory musculature, the temporomandibular joints, teeth and periodontal structures, and cervical musculature. They are a major cause of non-dental pain in the orofacial region and have been seen to develop post trauma to the head, post-motor vehicle accidents, and appear commonly following excessive levels of oral parafunctional activities, often exacerbated by stress and cognitive influences.

Most commonly, these disorders are treated by general dental practitioners, by means of intra-oral appliances, which are designed to alter tooth contacts, joint forces, and masticatory muscle use. The appliance therapy is often supplemented by analgesics, anti-inflammatories, habit modification education, physical therapy and a variety of biofeedback and behavioral altering techniques.

Oral orthopaedic appliances, commonly referred to as splints, have been used with a good measure of success.

The reduction of painful symptoms with appliance therapy has been reported commonly to reach levels of 70% of clinical success.

Standard appliances are usually worn during the day and/or night, and are designed to balance forces between the jaws. Research in the past has not managed to explain the biological reason for the reported improvement in symptoms. It is not known whether their beneficial effects occur because these devices actively reduce force on the affected joint, change its movement patterns, or because tooth contact is altered and thus muscle activity is influenced, or simply because the appliance changes the habits and modifies the behaviour in the affected individual.

Loading that exceeds the physiological limits of the temporomandibular joints and its associated structures may present itself initially as arthrogenous pain and pain to the muscles of mastication, the pre-auricular area, and angle of the lower jaw. Pain is often aggravated by chewing and jaw movement. Complaints of restricted jaw opening are commom, as well as distressing joint sounds described as clicking or popping.

Tissues of the temporomandibular joint react to functional demands by alterations in the soft and hard tissues. The role of articular mechanics in the progression of change in the joint has been implied but not proven.

Loading demands beyond the physiological limits of the tissues of the joint often lead to remodelling. This constitutes the adaptive processes that the form can undergo under function.

As the biomechanics of the temporomandibular joints and the concommittant musculature are complex and not fully understood, and vary with the anatomy and physiology of each individual, it is difficult to predict the effects that a standard design appliance has on any one person. Knowledge of the actual change which may be responsible for the beneficial results would clearly improve treatment planning and predictability of success. The increased level of prediction for each treatment approach could in turn, speed up the resolution of the disorder.

Research in jaw mechanics suggests that the forces in the joints are normally compressive, but can be altered by tooth contact patterns, and the way the jaw muscles are recruited and activated. Recent studies that have looked at the influence of mechanics on the temporomandibular joint and the possible effects on its function, suggest a probable role of joint biomechanics on symptomatology. Thus, it seems theoretically possible to increase or decrease temporomandibular joint forces by altering the appropriate combination of tooth contacts, and by encouraging particular patterns of masticatory muscle use.

An increased level of success both in terms of improved predictability as to the treatment approach , and improvement in the speed of recovery from symptoms, as well as a decrease in the magnitude of the pain, by articular decompression would be a desirable effect from an oral orthopaedic appliance.

Thus any novel oral appliance, designed specifically to reduce loading in an acutely-painful temporomandibular joint, could resolve arthrogenous pain more effectively than conventionally-designed ones reported previously in the literature, and would be a useful clinical approach for managing specific clinical temporomandibular disorders.

CLASSIFICATION, ETIOLOGY AND MANAGEMENT OF TEMPOROMANDIBULAR JOINT DISORDERS

CLASSIFICATION OF TEMPOROMANDIBULAR DISORDERS

Temporomandibular disorders are a major cause of non dental pain in the orofacial region. Their most frequent symptom is pain, usually localized around the muscles of mastication, the preauricular area, and the temporomandibular joint. The pain is commonly aggravated by excursive movements of the jaw and mastication. There is also often what has been described as clicking, popping, grating and crepitus coming from inside the joint complex. Patients usually complain of earaches, headaches, jaw aches, toothaches and facial pain. Often a history of trauma is elicited, and of parafunctional activities such as bruxing or clenching, and of stress.

Definition of the disorders has undergone substantial changes in the last few years. The term TMD itself has come to be regarded as too broad, because individuals with TMD present with quite distinct variations in signs and symptoms, and probably have various mechanisms underlying their disease.

The term "TMJ" has become regarded in the popular press as the "disease" of the 70's, 80's and 90's, due to the increased attention this complex

articulation has attracted from the profession. Due to the ever increasing costs to society and to the need to bring health care costs under control, and with the proliferation of diagnostic and therapeutic devices, there have been major efforts made to improve the diagnostic and management guidelines existing for this multifactorial disorder. A historical review can be found in the Guidelines for Classification, Assessment and Management of Temporomandibular Disorders by McNeill, (McNeill 1993) and in Dworkin (1992).

Laskin (1969), described what is now considered to be a specific subgroup category within TMD called Myofascial Pain Dysfunction Syndrome (MPD). MPD was proposed for use by clinicians for diagnostic and treatment planning purposes and for researchers for the purpose of defining subject groups. For the diagnosis to be made, four positive and two negative signs had to apply.

The positive inclusionary signs were:

- Unilateral pre-auricular pain.
- Muscle pain on palpation.
- Joint noises (clicking, popping).
- Limited mandibular opening.

The negative exclusionary signs were:

- No joint tenderness upon palpation of the external auditory meatus.
- No clinical, radiographic, or biochemical evidence of organic disease.

All patients with TMD, but not categorized as MPD, were considered arthritic patients (McNeil 1993). The operational definitions for eliciting a positive or negative sign were not provided, nor were there clear descriptions on the amount of pressure needed to palpate a muscle or an objective measure of pain considered. It also unfortunately did not differentiate between intra-articular or so called internal derangements of the discs and muscular problems. It did serve nevertheless for many years as the operational definition for temporomandibular disorders.

During the late 70's, based on a conference convened to establish guidelines for the clinical diagnosis of TMD (Laskin et al. 1983), a nomenclature system suggested by Bell (1982) was decided upon. Bell's system also lacked clear operational definitions and reproducibility or validity testing, but it did serve as an expansion on Laskin's. It suggested five subgroups:

- Acute Muscle Disorders
- Disc Interference Disorders
- Inflammatory Disorders
- Chronic Mandibular Hypomobilities
- Growth Disorders

In 1985, Eversole and Machado (1985) proposed another classification system for TMD. This system specified three main categories:

- Myogenic Facial Pain.

- Internal Derangements.
- Degenerative Joint Disease.

This simplification eliminated the subgroupings made by Bell for muscle pain and simplified the disc derangements categories. As this system was proposed for research as well as clinical purposes, specific exclusionary and inclusionary criteria were developed. Unfortunately, again no operational definitions for the examinations were given. It also has proven to be too exclusionary, since for example, a myogenic facial pain could not present with joint sounds. These exclusions make it difficult to categorize patients into the proposed subgroups. Nevertheless, it has been shown to capture the more acceptable grouping for TM dysfunction (i.e., muscle disorders and pain, joint disorders and pain and arthritis).

The problem of multiple diagnoses and how to deal with these when attempting to define a presumably homogenous reasearch population has not really been solved. Although individual clinicians are succesful in diagnosing the simpler forms of TMD, the management of the more complex and more chronic problems often require a multidisciplinary approach. To facilitate this approach Dworkin (1992) suggested a multiaxial diagnostic system that develops concurrent physical, psychological and social conditions along two axes, and the newly proposed Research Diagnostic Criteria (RDC), (Dworkin Ed. 1992) has been an attempt to do just this.

The base for the two axes are a clinical examination and a chronic pain disablilty and psychological-based questionnaire. This classification has gone a long way in providing some operational definitions and criteria to aid in the examination and evaluation of the multifactorial presentation of signs and symptoms for a heterogenous group of patients. The parameters that are suggested for use by researchers have not yet been fully validated, but at least give some guidance in how best to group patients and what tests to use, and the method doesn't ignore the multidimensional aspect of these disorders.

For clinician's use, the clinical diagnostic nomenclature systems detailed by Clark et al. in 1989 and McNeill et al. in 1993 continue to be used, and be of use in the assessment and treatment of individual cases for they are generally consistent with patient symptomatic presentations.

The presence of multiple symptoms thus remains an issue. To some extent, researchers have ignored the underlying sources of the disorders and have focused unduly on the multiplicity of symptoms and signs for this multifactorial disorder. Researchers need to continue addressing the issue of symptom overlap and continue developing a system which allows multiple attributions to distinct subgroups, as well as scoring specific symptoms when multiple signs and symptoms exist. Only in this way can the relative strength of each subgroup be assessed.

The pooling of heterogenous groups has unfortunately caused much confusion. Clinicians have not been concerned with the problems of forming homogenous groups and since they have been mostly involved in the treatment of these disorders, the criteria available in the past has not proven very helpful in the study of the disorder. All too often the broader term of TMD has been equated with, what is now accepted as a subgroup of the classification, TMJ disorders.

For research purposes, the refinement of existing clinical methodology and the addition of more objective methods to quantify signs and symptoms can only benefit any study that deals with repeated measurements over a period of time. Methods need to be tested for reliability and validity and compared to some "Gold Standard", which to date continues to be the examination and history that clinicians are most adept at.

Data on sensitivity, specificity, and predictive values exist, for some examinations and questionnaires, but further work is needed before these can be used both as research and clinical tools in order to facilitate the communication between researchers and clinicians.

In summary, the definition of TMD and its various subgroups has progressed steadily since the concept of MPD was brought forward by Laskin.

Fortunately clinicians are more routinely observed to describe their patients in a more detailed fashion, and rarely is the broad concept of "Temporomandibular Disorder" considered a sufficient diagnosis. "TMJ" continues to simply be the anatomical name for the articulation, not an unknown disease entity, and temporomandibular joint disorders is increasingly studied as a disease entity in itself.

ETIOLOGY OF TEMPOROMANDIBULAR JOINT DISORDERS

-Early Theories of Etiology-

Since the beginning of this century a variety of etiologic factors have been suggested as the cause of pain and dysfunction in the temporomandibular articulation system. One of the earliest proposed and most persistent theories, was that temporomandibular joint disorders are caused by abnormal structure both of the teeth and jaws. Ever since the pioneering work by Edward Angle (1900), who today is credited with describing the essential features of a "normal" occlusion, dentists have regarded abnormal structure as a major etiologic factor in temporo-mandibular disorders. Costen (1934) described a more specific structural joint disorder, when he postulated that ear and temporomandibular joint pain were signs and symptoms caused by the compression of vascular and neural elements, specifically the chorda tympani nerve and tympanic plate, due to displacement of the mandibular condyles. Most structural abnormality theories of the first half of the century, centered on

occlusal abnormalities due to lack of teeth, malpositions of teeth and the so called abnormal occlusions. The structural occlusal model only became succesfully challenged in the 50's by Sicher, in the late 1960's by the multifactorial model of temporo-mandibular dysfunction of Laskin (1969) and by a new structural or morphological concept, that of the disc displacement model (See Clark (1991) for an historical perspective).

Theories by the middle to late 1970's became more multifactorial in approach and allowed for joint and muscle abnormalities. These could be induced by trauma, parafunctional habits, hypermobility of tissues and of course malocclusions of the jaws.

-Current Theories-

It is interesting to note, that to this day, the occlusal abnormality theory has survived, even though evidence exists to the contrary (Helm and Petersen 1989). While clearly, others feel that the scientific evidence available does not warrant the rejection of the hypothesis that occlusal factors are part of a causal complex of craniomandibular disorders. (Kirveskari and Alanen 1993). It nevertheless has been established that many patients with naturally occuring, so called occlusal abnormalities, do not present with signs or symptoms of temporomandibular joint disorders, while others with ideal occlusions exhibit disease.

Unfortunately, the evidence for the prevalence of other etiologic factors such as trauma, joint laxity, parafunctional habits, stress related behaviours and other anatomic susceptible abnormalities needs to be more convincingly presented.

Temporomandibular joint disorders are diverse and often multifactorial, and most probably a universal etiology does not exist. According to McNeill (1983), there are certain factors that increase risk and are therefore predisposing. Others may cause the onset of signs and symptoms and can be referred to as initiating factors. Still others that enhance the progression of TMJ disorders and are called perpetuating. Any of these factors under different circumstances may serve any of these roles.

Many factors are thought to affect the dynamic balance or equilibrium between the different components of the masticatory system. Loss of structural integrity, altered function, or biomechanical overloading in the system can compromise adaptability and increase the likelihood of dysfunction by disrupting this equilibrium. (Parker 1990, McNeill 1993). The exact prevalence of the various etiologic factors has therefore not been firmly established. (Clark 1991). For the purposes of this review however, etiologic agents or factors in TMD are divided into 4 major groups: Trauma; Anatomic or Biologic; Systemic Pathophysiological and Psychosocial Factors.

Trauma.

The best estimates indicate that long lasting effects from an injury or trauma account for 10 to 30% of TMD patients. A history of trauma can be elucidated from a majority of adults suffering from TMD signs or symptoms, compared to a much smaller number in non-patients (Pullinger et al. 1985). Katzberg (1985), reported a history of trauma in 26% of paediatric patients. Commonly, blows to the mandible during sports injuries and abuse during artistic performance were reported by Howard (1990). Iatrogenic trauma from dental procedures, as well oral intubation associated with the administration of general anaesthesia has also been implicated. (Harkins and Marteney 1985, Taylor and Way 1968). Evidence also exists for the presence of temporomandibular joint symptoms after motor vehicle accidents (Kronn 1993 and Braun et al. 1992) and for signs of altered function of the temporomandibular joint after cervical extension-flexion injuries, the so-called Whiplash injuries, even though the mechanism whereby these injuries develop remains controversial (Weinberg and Lapointe 1987, Howard et al. 1991).

Another form of trauma associated with joint disorders has been hypothesized to originate from adverse loading of the system due to parafunctional habits. Tooth clenching and grinding, lip biting, and abnormal posturing of the jaw all are common in asymptomatic individuals but have been suggested as initiating and/or perpetuating factors joint disorders (see Travell and Simons 1983,Faulkner 1990,

Rugh and Harlan 1988, and Scharer 1974). Parafunctional habits have been most frequently assessed by self-reporting, questionnaires and tooth wear. These have been critized for being too subjective and relying primarily on memory and verbal reports. Limitations of such measures were recently reviewed by Marbach et al. (1990).

Anatomic or Biologic Factors

These comprise the developmental and genetic factors, encompassing skeletal malformations, past alterations to the dentition or what historically have been viewed as malocclusions.

Historically as was mentioned above, the profession has viewed malocclusions as a primary etiologic factor for temporomandibular joint disorders. Interferences in occlusions (both non-working and working) plus centric discrepancies, have commonly been associated with these disorders. However, reviews of the literature fail to support tooth position discrepancies as a common etiologic factor (See Marbach 1990, De Laat et al. 1986, Seligman 1991 among others).

Changes in the structure of the component parts of the articulation have been associated with the loss of occlusal support and lack of molar teeth. Studies of skeletal remains, (Granados 1979, Whittaker 1989) and of patients with osteoarthrosis,

(Akerman et al. 1988, Tegelberg 1987, Kopp 1977 to name a few), have correlated loss of molar support with bony changes. The highest correlation with osteoarthrosis seems to be increasing age however, not the lack of teeth (Whittaker et al. 1990).

Studies of living, apparently normal populations, have not shown an association between tooth loss and tempormandibular joint disorders (Helkimo 1974, Kirveskari and Alanen 1985, Wilding and Owen 1987, Pullinger at al. 1990, among others). The loss of moderate changes in vertical dimension (5mm) does not seem to be enough cause for TMD symptoms (Rivera-Morales and Mohl 1990). In a study comparing a control group to well-defined diagnostic groups rather than to specific symptoms, Selligman and Pullinger (1989) found selective occlusal variables somewhat associated with some TMJ disorders.

In a further review of the role of intercuspal relationships in TMD, the same authors in 1991 concluded that in skeletal anterior open bites, reduced overbite and increased overjet were all associated with osteoarthrosis but they lacked the specific parameter to define a patient population. Therefore, they concluded that there was no evidence that overbite and overjet play a role in the pathophysiology of nonarthrotic disorders. They also found a relationship between unilateral retruded contacts and the lack of a centric slide with the presence of disc displacements. Also there was no relationship between crossbites and the presence of disease.

This review should be consulted for a more detailed look at the skeletal and occlusal relationships to TMD. Nevertheless, the association remains between anatomical factors such as an altered condylar positions with concomitant capsular alterations, but not as the cause of the pathophysiology (Selligman and Pullinger 1991).

Systemic Pathophysiological Factors

These can include degenerative, endocrine, neoplastic, vascular and metabolic disorders, which can act simultaneously at a central or local level (Byrd and Stein 1990, Hagberg et al. 1990). Degenerative muscle changes can result from ankylosis and lack of use of the articulation (El-Labben et al. 1990).

It has also been suggested for all joints that the lack of intra-articular lubrication and alterations in synovial fluid viscosity may initiate disc derangements and clicking (Toller 1961). The content and make up of synovial fluid has been implicated in the modulation of pain in inflammation of the TMJ (Israel 1989, Quinn and Bazan 1990).

Systemic joint laxity has been suggested as a contributing factor to TMJ disorders. It has been reported more prevalent in females and a correlation has been made with the number of lax joints and the appearance of a craniomandibular disorder (Westling 1989, 1990, 1992, Buckingham et al 1991, Bates 1984). Other studies however have found no relationship (Chun et al.1990, Blasberg et al. 1991).

If true, this together with hormonal cyclical changes could help explain some of the increased incidence of TMJ disorders in females (Olson et al. 1988), but further work is needed to confirm it.

Other factors include the arthritides. Of these, the most common is osteoarthrosis (Kopp 1977). The distinction between an adaptive response by intra-articular tissues to increased joint loads and the pathologic one is somewhat blurred. (A discussion of loading and its efects can be found elsewhere). Studies have evaluated enzymatic and other metabolic by-products in degeneration of the TMJ (Kopp 1983, Israel 1989). On the other hand, there is strong evidence that rheumatoid arthritis affects the temporomandibular articulation (Akerman et al. 1988) and that it can be modulated by injections of glucocorticosteroids (Wenneberg 1991), suggesting that inhibition of the degenerative process may be achieved by a reduction in the inflammatory process.

Histologic changes have been observed on condylar cartilage as a response to functional loads (Hansson and Oberg 1977, Hansson and Nordstrom 1977 among others) as has the remodelling of the articular tissues (Baldioceda et al. 1990, Luder l993). The presence of changes in the structure of the temporomandibular joint with age is as expected with all aging tissues. Nevertheless, the presence or absence of intra-articular changes, be it disc derangement and/or bony remodelling cannot be ruled out as normal anatomic variation in aging individuals, nor can it be

said that it is found solely as a result of the systemic pathophysiological factors reviewed above.

Psychosocial Factors

These factors include individual, social and societal variables that impact on the behaviour and the ability of the patient to function and adapt to change. There has been some evidence that patients who suffer from temporomandibular disorders experience more anxiety than controls and that many of the symptoms expreseed may be somatizations of emotional disturbances (Gerschman et al. 1987, McCreary et al. 1991). These patients often have a history of other stress-related disorders (Gold et al. 1975).

The role of stress and mediating cognitive variables, ie: the feeling of contolling one's own life situation, may be important in the etiology and progression of temporomandibular disorders (Stockstill and Callahan 1991).

While the relationship of psychosocial disorders to TMD is still not clear, clinical reports suggest that the psychosocial conflicts and distress of pre-existing psychiatric conditions may be a contributing factor to the etiology of TMD, and may exacerbate and/or affect the adaptive capabilities of the individuals to the temporomandibular disorder (Lipowski 1988).

Psychosocial factors may predispose certain individuals to TMD and may also perpetuate the disorder once chronicity is established. Careful consideration of psychosocial factors therefore seems important to the diagnostic evaluation of TMD patients (McNeill 1993).

MANAGEMENT OF TEMPOROMANDIBULAR DISORDERS

Increasingly the management of patients with TMD attempts to deal with these patients in a similar manner to those of other orthopaedic or rheumatologic disorders. Its primary goals are to reduce pain, decrease adverse loading, and restore normal function.

To achieve these goals the clinician tries to develop a multidisciplinary approach. Often there is a need for the rehabilitation program to incorporate treatments for both the physiological as well as the psychological component of the disorder, and to decrease or remove all contributing factors that can be ascertained from a history and examination.

-Reversible Therapies-

It is important in planning treatment to remember that TMD can be transient and self-limiting. The progression of a mild form of the disorder to a more

serious condition has not been shown to be the natural course of TMD (Greene and Laskin 1983, Fricton 1991). The body has powerful healing and remodelling capabilities that cannot be ignored (Nickerson and Boering 1989). This conservative approach to therapy has resulted repeatedly in positive treatment outcomes (Fricton 1991, Carlsson 1985, Randolph et al. 1990, Okeson et al. 1986 to name a few).

The conservative approach to the management of TMD depends primarily on the education of the patient with regard to the various treatment approaches, as well as addressing etiological, contributing and/or perpetuating factors that may be playing a role. This approach towards "self care" depends largely on motivation and cooperation by the patient. The prevention of further injury to the temporomandibular system, as in other musculoskeletal injuries, depends on rest and allowing healing to proceed without further insult (Randolph et al. 1990, Hodges 1990). Self-care instructions emphasize habit modification, avoidance of heavy chewing (soft diet), awareness of parafunctional activities of the tongue, lips and jaw, and mild forms of physiotherapy and exercise (Danzig and VanDyke 1983, Curl 1993).

Changing persistent maladaptive habits also play an important part in cognitive behavioral intervention. This sometimes requires a structured programme of behaviour modification that may involve lifestyle changes and often needs to be individualized (Rugh 1988).

Biofeedback is an example of structured therapy designed to alter behaviour that has been used alone and in conjunction with other therapies, to alter bruxing behaviours and muscle tonic activity (Solberg and Rugh 1972, Rugh and Johnson 1981, Pierce and Gale 1988).

Psychotherapy can also be part of a multidisciplinary approach to TMD therapy. This is especially the case in chronic pain patients, where symptoms may serve as a somatic metaphor that both expresses and resolves pre-existing or concurrent psychological conflicts (Levinson 1990). When TMD is part of the patient's coping mechanism, treatment efforts with cognitive-behavioral management or pharmacologic therapy will not be sufficient to resolve the conflict. The aid of a skilled psychotherapist is an integral part of management.

Pharmacotherapy has been shown to be an important adjunct to aid in patient comfort and rehabilitation. Nevertheless, its use needs to be controlled for misuse and abuse since relying on narcotics or analgesics for TMD patients may lead to tolerance and dependency (Ready 1979). The most effective agents for the management of TMD in acute conditions include analgesics, non-steroidal inflammatories, corticosteroids and muscle relaxants. In addition, anti-depressants are used in some chronic pain patients (Gregg and Rugh 1988, McNeill 1991).

Physical therapies are most commonly used as adjuncts to other treatments (Clark 1990). These involve posture training (Curl 1993, Hackney 1993), exercise and relaxation (Carlson et al. 1991), mobilization and manipulation (VanDyke 1990, Minagi et al. 1991). Agents often used in conjunction to the above are thermotherapy, utilizing surface heat or by means of cryotherapy or coolant therapy (Nelson 1988, Burgess et al. 1988, Travell 1952, Travell and Simons 1983); Electrotherapy in the form of electrogalvanic stimulation (EGS) (Murphy 1983), or transcutaneous electrical stimulation (TENS) (Binder 1981, Gold et al.1983, Wessberg et al. 1981, Moystad 1990); Acupuncture (Johansson et al. 1991, Raustia 1985); Laser therapy (Hanson 1989, Bezuur 1988); and Ultrasound (Esposito 1984). For detailed reviews of these modalities the reader should consult Okeson 1993 and McNeill 1993. Orthopaedic appliance therapy is primarily regarded as a reversible modality. It is reviewed in detail later.

-Irreversible Therapies-

Occlusal therapy and its role in establishing a cause and effect relationship to TMD has been a subject of much debate. Although dental treatment per se may be necessary for patients with TMD, many believe that it is infrequently necessary for the purpose of directly treating TMD (McNeill 1991).

As reported above there is little evidence, that normal and natural occlusal variation is a direct cause of TMD (Seligman and Pullinger 1991). Controlled studies have failed to show an association between occlusal interferences and TMD.

Occlusal adjustment has been reported to be effective in reducing symptoms of TMD (Magnusson 1983), but it has not been shown to be more effective than reversible modalities. (Wenneberg 1988). Limited occlusal adjustment may be indicated where certain interferences apparently provoke acute symptoms, especially as a result of dental restorative interventions (Scharer 1966). Occlusal adjustment is not viewed as a preventive modality for TMD (Goodman 1976).

It has been suggested that restorative therapy, since it is an irreversible treatment option, should never be regarded as an initial form of therapy (McNeill 1993), and the benefits of occlusal restorative therapy has been questioned (Plasmans et al. 1988). However, once symptoms are under control, its role could be considered. It would seem intuitively obvious that a stable occlusion and an optimum platform for the even distribution of forces and loading stresses to the teeth and joints, might be a desirable biomechanical state for the continuation of health for the teeth and articulation (Hannam 1984, Hylander 1985, Faulkner et al. 1987).

Orthodontic therapy, and in the extreme, orthognathic surgery, can be viewed as improving occlusal stability and thus providing more optimum surfaces to

distribute the stress. Their use as a treatment modality has to be viewed in the same manner as any occlusal therapy. When used after repositioning appliance therapy, orthodontic therapy has not been proven to be any more succesful than appliance therapy alone (Bradley 1989). In fact, orthodontic therapy in itself has been viewed as possibly destabilizing the stomatognathic system (Greene 1982). In contrast with this view, some long term studies have shown the risk factor of orthodontics itself to be negligible (Kremenak et al. 1992).

Surgery is considered an appropriate an effective form of management for specific temporomandibular disorders. The American Association of Oral and Maxillofacial Surgeons has set criteria in order to maximize the potential for succesful outcomes in surgical therapies of the TMJ. These have been reviewed by McNeill (1993). Arthroscopy, a commonly used mode for lavage and visualization of the joint may in the future be superceded by arthrocentesis as a conservative surgical therapy (Nitzan et al. 1991).

Arthrotomy, and in general open surgical techiques are today only used for advanced disease states, such as ankylosis, neoplasias, persistent painful disc derangements and severe osteoarthrosis. Discoplasty and discectomy when performed have been considered to be succesful (McCarty and Farrar 1979) for internal derangements, but the measure of success has not been made clear.

Alloplastic prostheses presently seem to be contraindicated due to tissue rejection problems and material breakdown, and condylectomies are presently performed infrequently. (For a review see McNeill 1993).

ORTHOPAEDIC APPLIANCE THERAPY

The occlusal appliance, commonly referred to as a splint, is a removable device most often made out of acrylic, that fits over the occlusal surfaces of the teeth in either the maxillary or mandibular arch creating a prosthetic surface that allows for varying tooth contacts with the opposing arch.

These appliances, in a variety of designs, have been commonly accepted as part of the primary form of therapy for temporomandibular disorders. Since the etiology and interrelationships of these disorders are often ill-defined and complex, the initial therapy is commonly reversible and non-invasive. There is a broad set of clinical experiences by enumerable practitioners dating back decades to justify the use of these types of appliances.

A critical review by Clark (1984) found their effectiveness to vary depending on the masticatory disorder being treated. In general there appears to be a 70 percent rate of "clinical success" with their use.

The precise effect and role of these appliances has yet to be defined. Several theories have been brought forward to explain how the interocclusal appliances actually work.

-The Occlusal Disengagement Theory-

This is based on the premise that the appliance provides the patient with an ideal occlusal scheme that eliminates any occlusal disharmonies, abnormal muscle activity and stabilizes the TMJ's (Ramfjord and Ash 1971, Posselt 1968). The design of the appliance is such that it is most often adjusted into a centric relation position, when the posterior teeth are in contact with the appliance, with posterior separation and anterior contact only on protrusive and canine rise during lateral disclusive paths of mandibular movement. It is often referred to as the full arch stabilization splint.

The occusal disengagement theory is based primarily on malocclusions as the primary etiology for TMD. Most studies use the splint in combination with other therapies, usually occlusal adjustments and/or prosthetic rehabilitation and counselling (Franks 1965, Zarb 1975, Agerberg and Carlsson 1974, Magnusson and Carlsson 1980, Suvineen and Reade 1989) . Few studies have utilized the stabilization splint as the sole mode of therapy (Goharian and Neff 1980, Greene and Laskin 1972, Carraro and Caffesse 1978). However, its "clinical success" cannot be considered as proof that the occlusal disengagement theory is correct.

The resolution of subjective symptomatology, as it is commoly reported, may be the result of force redistribution, occlusal stabilization, relaxation of muscle activity or simply habit modification or by the appliance working as a placebo.

The stabilization type splint has been used to treat both temporomandibular intra-articular pain and muscle dysfunctions. The differentiation between these diagnostic categories has often been blurred. The success rates have varied in part, due to poor control as to the diagnostic criteria for temporomandibular joint disorders. Even so in studies that have looked at the short term effectiveness to such a therapy, intra-articular pain seem to respond better than TMJ sounds or mandibular movement limitations (Tsuga et al. 1989, Caffesse 1978).

-Lack of Vertical Dimension Theory-

This theory is based on the concept that an interocclusal splint can restore the previously lost vertical dimension, thus restoring muscles and the articulation to a more "advantageous" position. The design of the appliance is often similar to the stabilization splint, but more care is placed in increasing the thickness and height of the appliance, thus supposedly reestablishing the original vertical. This treatment has been commonly suggested for patients with loss of posterior occlusal support and muscle hyperactivity.

Since the measurement of vertical dimension is often difficult to make quantitatively, the application of this approach is guarded (Rugh 1981). The adaptation of humans to changes in muscle length has been shown to be quite good (Goldspink 1976, Hellsing 1984). Nevertheless, some claims have been made for the use of varying splint heights to treat certain muscle symptoms (Manns et al. 1983). It is also pertinent to add that any interocclusal device increases the vertical dimension : thus a change in symptomatology would be expected for this reason alone, if this theory were the only factor involved in affecting temporomandibular symptomatology. The effect in any case may prove to be only temporary (Jaffe 1991).

-Mandibular Realignment Theory-

It has been theorized that by changing the relationship of the two jaws with an interocclusal appliance, various musculoskeletal symptoms improve as the muscles achieve a "neuromuscular balance". The assumption lies in the malrelatioship that is said to exist between the jaws causing this imbalance. Jaw manipulating techniques can be utilized to achieve this new jaw position (Celenza et al. 1978). Another approach to mandibular realignment is a muscle determined position determined by trancutaneous low frequency electrical stimulation of the elevator musculature (Jankelson 1979). No studies of treatment of TMJ pain have been done using this approach, and there are no specific biological descriptions of what is precisely meant by "neuromuscular imbalance".

-Temporomandibular Repositioning Theory-

Similarities exist between this approach and the one mentioned previously. Both assume that the mandibular position is incorrect. This approach attempts to alter the actual position of the condyle in the fossa (Weinberg 1979, 1980). This method is contemplated when a change in the position of the condyles is presumed to be necessary in order to attempt the correction of an intracapsular displacement of the disc. If succesful it is expected that the altered disc-condyle relationship will alleviate the clicking joint. The basic premise of this theory is that the disc repositioning actually occurs, which after a period of healing or readaptation allows the mandible to return to a non-treatment position. Alterations of disc-condyle positions have a guarded prognosis due to the possible remodelling changes that can occur in the joints (McNamara 1979, Mongini 1980), and the possible need for permanent alterations to the tooth to tooth relationships that need to follow in order to stabilize the jaws long term.

-Cognitive Awareness and/or Placebo theory-

The term placebo has been applied to many types of substances used in a variety of different ways. The most common definition refers to a material that does not contain any active medicine and is pharmacologically inert, a 'pure placebo'. An orthopaedic appliance can be categorized as such a device.

An 'impure placebo', refers to any substance or device used as placebo that is not believed to be totally inert. It is also important to note that it has been known that you can demonstrate objective physiological changes in patients given placebos.

The cognitive awareness theory is based on the concept that an interocclusal appliance of any design will alter behaviour. This change in behaviour, whatever this may be, is enough to alter abnormal muscle activity, parafuntional jaw movement or jaw loading forces. Greene and Laskin (1972) as well as Rubinoff et al. (1987) found that a non-occluding splint proved just as succesful as an occluding one, in ameliorating symptoms, suggesting that cognitive awareness may play a significant role in the effectiveness of intra-oral appliances.

The modification or altering of one's behaviour is a general theory that has been shown to apply to most succesful therapeutic interventions (Rugh and Solberg 1976).

ORTHOPAEDIC APPLIANCE (SPLINT) TYPES

-The Stabilization Splint-

This is the most commonly used intra-oral device by dental practitioners for the treatment of temporomandibular joint disorders. It is adjusted for use in

condylar positions ranging from centric occlusion, to habitual and hinge positions. As we have seen above, it is used in the treatment of masticatory dysfunction, temporomandibular joint pain, clicking joints, bruxism, and in the treatment of disorders of incoordination and limitation of jaw movement. It has been used most succesfully for the treatment of muscle related disorders (Okeson 1993). In addition, myogenous pain disorders seem to respond best to part time use (often night-time wear), while intracapsular disorders are better managed by continous use (Wilkinson et al . 1992).

-The Anterior Repositioning Splint-

This intraoral device incourages the mandible to be positioned in a more anterior tooth relationship. Its presumed goal is a "better" condyle-disc relationship so that the coordination of jaw movement can proceed without pain and joint noises. However, the elimination of joint sounds has not been shown to position the condyle-disc complex necessarily in the intermediate zone. Arthrography (Tallents et al. 1986), and CT scanning (Manco and Messing 1986), have shown that even with the absence of sounds, the position of the complex is not as thought pior to the repositioning. Lundh and Westesson (1989) followed a group of patients treated with a repositioning appliance long term, and found that pain and clicking were mostly eliminated by the use of this type of appliance initially, but the clicking returned in 74% of cases. Okeson (1988) retro-spectively looked at 40 patients treated with a repositioning splint for eight weeks and then phased out with a step-back procedure. 80% of patients

presented free of pain and clicking at eight weeks. Thirty months later 66% had a return of joint sounds, with 23% reporting joint pain. The author concluded that if resolution of pain is the primary objective, repositioning has a good long term prognosis, but a repositioning splint is of limited value in the permanent elimination of joint sounds. Similar conclusions were reported by Clark et al. (1988).

-Bite Planes- (Anterior).

This appliance is worn over the maxillary teeth and provides contact only for the mandibular anterior teeth. It leaves the posterior teeth out of contact, in order to eliminate their influence on the function of the masticatory system. Has also been called a re-programming appliance usually used for brief periods in anticipation of occlusal adjustment. It has been recommended for use in the treatment of muscle disorders by Ramfjord and Ash (1983), Posselt (1968) and others that have believed the primary etiology of the disorders to be malocclusion based. The main drawback to an anterior bite plate is the uncontrolled supra-eruption of the posterior teeth.

- Bite Planes - (Posterior).

This appliance type is usually fabricated for the mandibular posterior teeth. It has been commonly referred to as the Gelb Splint (Gelb 1977). It has been advocated for use in cases of severe loss of vertical dimension and jaw repositioning.

Once again, long term use is not advocated due to the potential for supra-eruption of teeth and/or intrusion of the occluding teeth.

-Pivot Appliances-

This appliance covers one arch and usually provides a single posterior contact on each side. It was originally designed with the idea that it would lessen intra-articular pressure and unload the temporomandibular joints in order to help treat noisy joints (Posselt 1968). It has been shown to actually produce the opposite effect by Ito et al. (1986). Bilateral pivots tended to intrude the condyles in the fossa in a superior and anterior direction, and did not cause distraction of the joints.

-Soft Appliances-

This splint is fabricated from resilient materials adapted to either the maxillary or mandibular teeth. It's most common application is as a protective device for the prevention of traumatic injuries of the teeth or jaws during sports. They have been advocated for use by clenchers and bruxers with the belief that the softness allowed for the more even distribution of loads to the joints (Posselt 1968). In fact, Okeson (1987) showed that their use increased nocturnal masseteric activity.

The anecdotal reporting of the reduction in symptoms in some patients using soft appliances, could be explained by the cognitive awareness theory.

In summary, it is accepted that a patient's resolution of symptoms is due to many of the factors outlined above. That a majority of TMD patients benefit by a reduction in symptomatology, is reason enough to consider this form of reversible therapy for these patients. Complications, and iatrogenic sequelae of appliance therapy can be prevented with proper use and design. Appliances probably should not be designed to allow tooth movement unless desired. They should also be hygienic and easily tolerable in order to promote compliance.

PAIN

Pain is the most common symptom of disease or injury that compels patients to seek medical or dental attention. The treatment of pain is associated with enormous costs to society. As for its effects on our health and lifestyle, chronic pathologic pain serves no clear biological benefit to humans, yet imposes emotional, physical, and social stresses to the sufferer and society. In contrast, acute symptomatic pain serves a distinct biological function, since it warns the subject that something is amiss. Pain as an experience is complex. It includes sensations evoked

by noxious stimuli and the reactions to such stimuli. Pain sensation refers to the ability to discriminate the quality, location, intensity and duration of the stimulus. Human reactions to these sensations vary from individual to individual. Attentional, cognitive, motivational and emotional variables modify the behaviour elicited by the noxious stimulus. This multidimensionality of pain has evolved in great part from the landmark work of Melzack and Wall (1965). The reaction to pain may depend on the meaning of the situation in which pain occurs. Cultural backgrounds influence individual reactions and responses. Level of stress, anxiety, past experience and memory affect the response to the noxious stimulation.

ARTICULAR PAIN MECHANISMS

In humans the control mechanism for musculoskeletal pain in general, has been studied mostly on the processing of superficial nociceptive information from such tissues as skin. Small-diameter, slow conducting primary afferent fibers are usually associated with the responses to noxious stimuli. Articular tissues are not only innervated by nociceptor fibers that transmit pain, but also by larger diameter fibers, (group II and III) primary afferents. The latter are associated with low threshold receptors (e.g. Ruffini-like, Pacicinian-like, Golgi tendon organs). These low-threshold receptors respond to non-noxious mechanical stimuli or movements and are considered to play a role in perceptual and reflex responses related to articular movements. While most work has been directed at limb joints, some is available on

the TMJ. There is reasonable evidence that articular receptors do play a role in the detection of joint movements, but controversy exists about the extent of their contribution to joint position sense (Burke et al. 1988, Clark et al. 1989, Dubner et al. 1978).

Recently the view has been substantiated that group III and IV primary afferents, and perhaps Group II, are also involved in responses to noxious stimulation of articular tissues. These afferents terminate in the peripheral tissues as free nerve endings, and they can be activated by mechanical or chemical means, or even non-noxious low threshold distortions of the articular tissues. These studies have also documented the progressive increase in activity as the intensity of the stimulus increases (Mense 1986, Schaible and Schmidt 1988).

Enhancement of articular afferent activity has also been demonstrated in experimentally induced arthritis, and this activity has been shown to be counteracted by analgesics. These responses to peripheral mechanisms may contribute to our ability to code the intensity of articular pain, the hyperalgesia and allodynia that can occur in a traumatized or inflammed joint, and to the spontaneous pain and to symptoms and signs related to movement that are commonly observed clinically (Guibaud 1988). There has been some evidence that sensitization of afferents supplying limb and trunk muscles may occur, when the activation threshold is lowered by chemicals such as bradykinin or prostaglandins (Mense 1986).

Sympathetic afferents supplying articular tissues, may also be playing a modulatory role. This has been suggested in inflammatory conditions and in pain associated with articular tissues (Basbaum and Levine 1991).

INNERVATION OF THE TEMPOROMANDIBULAR JOINT

The temporomandibular joint differs from most other joints in the body, in that its condyle not only rotates but also translates. It is technically considered a ginglymoarthrodial joint (Bell 1990). It is a compound synovial joint formed by the mandibular condyle fitting into the squamous portion of the temporal bone. A pliable water-filled intra-articular disc composed of dense fibrous connective tissue separates the two bony surfaces and divides the joint into two synovial compartments. The disc emerges at its periphery with a capsular sheath that surrounds the joint, that gets its support from peripheral ligamentous attachments particularly at its medial and lateral borders. Three of the masticatory muscles have projections on to the disc. The pars profunda of the masseter muscle, the superior head of the lateral pterygoid and the temporalis muscle. The articular disc starts in fetal development, as a structure with vascular elements but its central portion becomes compressed and loses its vascularization as development progresses embryologically.

-Peripheral Innervation-

The mandible in the adult is a single bone that crosses the midline of the body, having arisen laterally and fused in the midline, and has at both its ends these two articulating surfaces. Thus, afferent inputs from the TMJ's to the central nervous system probably are associated with integrative processes that differ from other joints. Sensory and motor branches of the Fifth Cranial nerve supply the muscles that move this joint as well the joint itself. The innervation was first detailed by Thilander in 1961. The central portion of the disc was found to be avascular and not innervated. This portion is believed to receive the load and compressive forces of mastication and movement as it cushions this joint in function.

Studies carried out on human and animal tissues have produced contradictory results as to the innervation of the disc and capsule. However, most of these were carried out by metallic impregnation that can produce inconsistent results (Thilander 1961, Bernick 1962, Wink 1992). Recently, immunohistochemical techniques that detect neurospecific structural or neuroactive peptides has allowed for more specific markers of neural elements . Thus, some evidence has surfaced lately that, not only is there innervation of the capsular and perivascular fibers, but fibers were indentified in areas of the joint disc previously considered avascular . The TMJ does contain free nerve endings, but specialized receptors have not been found in abundance. The afferents supplying these receptors are nearly all less than 10μm in

diameter and are carried primarily in the branches of the auriculotemporal nerve. The richest innervation has been found in the posterolateral aspect of the capsule of the joint (Morani et al. 1994). Conclusive evidence as to the organization of the innervation in the articular disc and articulating surfaces has been scant. In autopsy specimens nerve fibers were reported penetrating the disc from the pericapsular tissues, and structures resembling Pacinian corpuscles and Golgi tendon organs have been identified (Wink et al. 1992).

Few studies have concentrated on the properties of nociceptor afferents supplying the TMJ. Group III and IV afferents do supply the tissues of the joint, as well as larger, faster conducting afferents. Recordings have been made, from these apparently low threshold non-nociceptive afferents in responses to jaw movement and changes in position. (Klineberg 1971, Kawamura and Abe 1974, Lund and Matthews 1981). However, the data has not been conclusive. Nociceptive input clearly can be expected to exist from the temporomandibular joint as it does from limbs, but its evidence has proven elusive.

In the last few years it has become clear that primary afferent nerve fibers responding exclusively to noxious stimuli are found innervating the face and extremities. Dissatisfaction with specificity theory that argued that pain was a primary or specific sensation with its own specialized receptors and pathways plus its inability to explain some of the characteristics of clinical pain, led to central summation

theories that proposed that stimulus intensity and summation were the critical determinants of pain (Sessle and Wu 1991).

Some studies have also shown that neuropeptides with a probable trophic function are released in synovial fluids (Larsson et al. 1989, Mapp et al. 1990). These neuropeptides can modulate the production of immunoglobulins, stimulate T lymphocytes, and induce production of PGE-2 and collagenases in synovial cells. Many of the neuropeptides are believed to be involved in vasoregulatory and inflammatory mechanisms. Thus, it is conceivable that some nerves observed in the TMJ capsule may be responsible for the release of some of these peptides. The identification of prostaglandins and leukotrienes in the synovial fluid of painful and dysfunctional TMJ's has been presented by Quinn and Bazan (1990).

The output of the transmission neurons is also known to be influenced by descending control mechanisms from the brain that relate to cognitive, motivational, and affective processes.

-Central Afferent Projections-

The central projection sites within the trigeminal brainstem sensory nuclear complex from deep craniofacial tissues have not been fully explained.

Those of large diameter jaw muscle afferents whose cell bodies are located in the trigeminal mesencephalic nucleus have been well documented (Dubner et al. 1978, Capra 1987). Bulk labelling of these afferents has resulted in labelling of the rostral components of the trigeminal brainstem as well as in its subnucleus caudalis. The projection of small diameter afferents from the TMJ has not been examined. Nonetheless, we could speculate that deeper structures, like an articulating joint, may also project to each subdivision of the trigeminal brainstem complex.

-Central Neuronal Mechanisms-

Neurones in the dorsal horn of the spinal cord are responsive to non-noxious mechanical stimulation of limb joints, but can also be activated by noxious articular stimuli, from bilateral joints. It is also becoming clear that both nociceptive specific (NS) and wide dynamic range neurons (WDR) in the spinal dorsal horn receive nociceptive inputs from articular tissues and that, like spinal dorsal horn neurones activated by noxious stimulation of muscle, they receive convergent inputs from afferents supplying superficial tissues (Menetrey et al. 1980, Schaible et al. 1987a). The convergence of superficial and deep inputs on the same neurone is considered an integral mechanism underlying the hyperalgesia, poor localization, and referral that characterize many conditions manifesting pain from joints and other deep structures.

With respect to TMJ afferents inputs, it has been documented in the rat and cat subnucleus caudalis (Sessle 1987), that a substantial population of caudalis neurones receive TMJ nociceptive inputs. Upon a variety of stimuli, TMJ afferents are preferrentially excited by cutaneous nociceptive (WDR and NS) neurones. These neurones are predominantly located in laminae I-II and V-VI of the subnucleus caudalis. In view of findings by Sessle and others that cutaneous or intraoral nociceptive neurones also exist in more rostral components of the trigeminal braistem complex, some of which receive jaw muscle afferent inputs, it is likely that rostral neurones also contribute to brainstem mechanisms underlying deep craniofacial pain.

It suggests that the caudalis neurones responsive to the TMJ and craniofacial muscles afferent inputs, represent critical neural elements underlying the transmission of acute orofacial pain, and that these deep nociceptive inputs show considerable convergence in transmitting deep nociceptive information. The convergence of mechanosensitive fields is consistent with the view of pain referral mechanisms. These probably contribute to the apparent spread of pain to adjacent tissues from the site of injury or inflammation which is often reported in craniomandibular injuries (Sessle and Wu 1991).

-Thalamocortical Projections-

The role of the thalamus and cerebral cortex in nociceptive mechanisms pertaining to pain perception is poorly understood. Only limited information is known about the thalamocortical processing of deep nociceptive information. Some activity can be evoked in the cat ventromedial and basal thalamus from the stimulation of forelimb and hindlimb afferents. Noxious mechanical or algesic stimulation of joints or muscles, as well as cutaneous noxious stimuli, activates single neurones in the ventrobasal thalamus and its immediately sorrounding regions and in the SI somatosensory cortex (Guibaud 1991).

A major input to the posterior thalamus originates from the dorsal column-medial lemniscal system and the rostral components of the trigeminal brainstem complex that transmit primarily non-nociceptive information from the spinal and orofacial regions. Another important input is the spinothalamic tract, which originates in the spinal dorasal horn: its trigeminal analogue is the thalamic projection from subnucleus caudalis. Axons originating in caudalis represent a major portion of the input of the trigeminal to the posterior thalamus. (For further review of the neurobiology of facial and dental pain Sessle 1987 should be cosulted).

SUMMARY

Pain by its nature is a subjective symptom. Its conscious perception is generally evoked by noxious stimuli and described as a multidimensional process involving both a subjective evaluation of the sensory aspects of the stimulus (intensity, duration, location) as well as the emotional or affective component to that stimulus.

In the temporomandibular joint and its associated structures, pain is not always due to joint pathology or traumatic injury. Since the TMJ capsule has been shown to be supplied with free nerve endings, it is not surprising that pain may emanate from this joint. The most common symptoms of TMJ pathology are limitation of movement and tenderness on palpation. Pain in the joint has been known to be referred to other areas of the head and neck.

The detailed mechanisms underlying acute and especially chronic pain have yet to be clarified. This is partly a reflection of the multidimensional nature of pain, since we now do understand it as a multifactorial experience encompasssing sensory-discriminative, affective, cognitive and motivational dimensions. The mechanisms involved in our understanding of pain are receptor specificity, central convergence and summation, inhibition, and descending control.

BIOMECHANICS OF THE TEMPOROMANDIBULAR JOINT ARTICULATION

ARTICULAR LOADING

For over a century, researchers analyzing the mechanics of the mammalian mandible, have looked at the forces developed in varying planes, and how they relate to mandibular function with the thought that the mandible, acts as lever during mastication with the mandibular condyle acting as a fulcrum (Hylander 1975) .

For the human temporomandibular joint (TMJ), several investigators have commented upon the possibility of differential mechanical loading within the joint, with the stress bearing portion being located along the articular surfaces of the eminence of the temporal bone and the head of the mandibular condyle (Mofett 1964, Oberg 1971). It has become evident that forces reactive to functional demands are in part directed through the articulation of the jaw and these are unevenly dissipated through the tissues (Hylander 1979). The evidence for differential loading appeared originally from studies by Moffet et al. (1964), who demonstrated how the lateral aspect of the joint remodels differently than the medial aspect with increased function. These patterns have been seen as highly indicative of possible differences in mechanical loading of the articular components of the temporomandibular joint.

Degenerative studies have also suggested differential loading within the joint. Oberg et al. (1971) noted that the majority of disc perforations were found on the lateral aspect of the joint. Since arthrotic changes are often related to local mechanical factors, these data seem to support the view that the central and lateral aspect of the TMJ experiences more load and stress, and therefore more remodelling change than the medial aspect.

Knowledge of the distinct microheterogenous regional specialization of proteoglycans, the distribution of complex collagens and of the fibrillar orientation in the extracellular matrix of the disc, has elucidated much about the possible reactions to forces acting on this tissue and has helped in the formulation of a model for its mechanical function (Mills et al. 1994).

There are various reasons for the preponderance of stresses to be located on the lateral or posterolateral aspect of the TMJ. One is related to the distortions that the mandible goes through during the power stroke in chewing. The mandibular corpus of primates has been shown by Thilander (1979) to evert at its lower border and invert at the coronoid process, causing the lateral aspect of the condyle to get pressed against the articular eminence during differential occlusal loading.

These observations have been replicated in modelling studies, where measurements of the rotational deformations with simulated tooth clenching tasks in a computerized simulation were observed, and the rotational distortion was shown to originate at the rami, due to the action of the elevator muscles, and manifested in the dental arch and at the condyles (Korioth and Hannam 1994).

Mohl (1988), advanced another reason for the increased loading of the lateral aspect of the TMJ. He suggested that the lateral pole of the condyle, due to its angulation and position with relation to the transverse axis of rotation, traverses differently at its medial and lateral poles during the power stroke in mastication. Another, perhaps more important explanation for differential loading, is related to the mediolateral translation of the condyle relative to its position in the fossa during unilateral mastication. In these instances the ipsilateral condyle exhibits a lateral shift, commonly referred to as Bennett's shift, during the opening phase of mastication. This in turn, is followed by a medial shift of the same condyle from this lateral position during the closing phase. The medial shift is therefore correlated with the occurence of the power stroke and with it, the increase in condylar reaction force. While, at the ipsilateral condyle its position is shifted to a more lateral one, relative to the eminence. This presumably causes the lateral and central part of the condyle and its disc, to be pressed against the more lateral portion of the fossa, while the contact is reduced between the medial portion of the condyle and its eminence.

This would suggest, that in the early phase of the power stroke in mastication, the lateral aspect of the ipsilateral joint may experience more stress than its medial aspect. Once centric occlusion is reached, the stress bearing surface of the joint no longer oppose each other in a steep medial-to-lateral gradient of increasing reaction force. Instead, the TMJ reaction force will be more evenly distributed along the articulating surfaces.

-Condylar Forces-

In vivo animal experiments have measured condylar forces during jaw function. Force transducing devices were attached on bony surfaces in the subcondylar region (Hylander 1979), or directly on top of the condyles (Brehnan et al. 1981 and Boyd et al. 1990). Metallic prostheses have been implanted in the ramus near the joint (Hohl and Tueck 1982), and hydrostatic synovial fluid pressure has been analyzed within the superior aspect of the TMJ space (Roth et al. 1984 and Ward et al 1990).

The forces recorded by Hylander in primates were correlated with movement of the mandible, to distinguish between compressive and tensile strains. During mastication, the condyle was found to be loaded compressively. Forces were generally greater on the balancing side temporomandibular joint, than on the working

side. Changing bite positions altered the level of force. The condyle was also shown to be loaded during opening jaw movements.

The vast majority of studies have suggested the TMJ to be load bearing and higher forces to be transmitted through the balancing side articulation during unilateral mastication (Brehnan 1981, Thilander 1979, Hohl 1982, Roth 1984, Ito 1986, Boyd 1990, Ward 1990, Korioth 1994, Ferrario 1994). Some differences in the results are believed to be due to difficulties in detecting the chewing or biting side (especially from animal experiments), a crucial point in the correct interpretation of the data, since the ratio of bicondylar force distribution is dependent on the bite point location (Boyd 1990), others to the ratio and resultant of masticatory muscle forces (Ito 1986, Ferrario 1994).

Condylar forces have been estimated to be between 250-350 Newtons during intercuspal clenching (Koolstra et al. 1988 and Nelson 1986). The greater forces are being transmitted bilaterally through the TMJ's during intercuspal and incisal clenches, and through the balancing side articulation during unilateral isometric molar biting as well as left group function clenching tasks. However, the inclusion of a molar contact on the balancing side of a clench decreases the total amount of forces acting on the balancing side condylar region (Korioth and Hannam 1994).

The site and number of occlusal contacts, and the direction of application, influence the activity of the masticatory musculature during clenching (Williamson and Lundquist 1983, Shupe et al. 1984, MacDonald and Hannam 1984, Belser and Hannam 1985, Manns et al 1987). The degree of muscle activity has been shown to be sensitive to differences in occlusal support throughout the arch (Wood and Tobias 1984). The differential removal of contacts on a bite plane, significantly altered the general distribution of muscle activity. When one balancing contact was left intact, the general activity remained much the same. It was apparent that the mandibular condyle of the balancing side may have been dispersing the load that was previously transmitted by the balancing tooth contact.

The Finite Element Model (FEM) of Korioth and Hannam (1994) predicted this relationship as well and confirmed the importance of a balancing side molar contact in "stress-breaking or splinting" the balancing side TMJ during simulated clenching activities.

Further substantiation can be attributed to the clinical findings of Minagi et al. (1990), where the authors attribute the lack of balancing tooth contacts to the prevalence of joint sounds. They hypothesized that the presence of balancing contacts was protective to the the temporomandibular joint in certain occlusions. This has been corroborated by condylar movement observations made by Hagiwara et al. (1994).

Condylar forces during maximum clenching in the intercuspal position have been estimated to be around 250-350 Newtons and 250 Newtons for incisal clenching (Koolstra 1988, Nelson 1986). Forces are assymetrical during unilateral clenching with values being reported during first molar clenching perpendicular to the occlusal plane, at 25/15% of total muscle force (Faulkner 1987). The reason for this assymetry and the functional significance of morphological variation in the muscles of mastication has been reviewed by Weijs (1989). Miller (1991) should also be consulted for a review of the function and form of masticatory musculature.

Evidence presented above increasingly supports the assumption, since direct measurements in the human have not been possible, that during normal functioning there is symmetrical loading of the dentition and the joints. The magnitude of these loads changes with varying muscle recruitments for each particular task. The bite forces and the loads these muscles produce are usually directed at angles that allow a "stable" resistance by the anatomy. That is, during tooth clenching, condylar forces are developed which are more or less perpendicular to the temporal eminence when the jaw finishes the power stroke with teeth together, and roughly parallel to the occlusal plane or slightly more anterior to it, during rest when the condylar head is in the fossa.

-Bite Forces-

Bite forces in vivo have been shown to vary considerably between populations. So called "primitive" peoples eat less refined diets and thus rely more heavily on the use of their dentitions for the trituration of their foods. They have been shown to have stronger maximal bite force values than so called "developed" societies (Carlsson 1974).

Maximal bite forces vary in vivo considerably. Typically though, they range from 300-450 Newtons in the molar region, and about 1/3 of that strength in the incisor region. Helkimo (1977) showed gender differences in the mean values for bite force in a Scandinavian population, with women having about 2/3 the strength of men. Values for bite force are seen to decrease with increasing age partially due, it is believed, on the age-dependent deterioration of the dentition seen in those populations.

In general, bite force does not seem to be closely related to general muscle force and skeletal dimensions (Linderholm and Wennstrom 1970). However, bite force is reported to be associated with a long mandibular corpus and a small gonion angle (Ringquist 1973).

The exertion of a maximal bite force is affected when the mandible is placed in eccentric positions. Bite forces measured with the mandible in extreme lateral, retrusive or protrusive positions reduce the overall level of the bite force (Molin 1972, Leff 1966). Mechanical advantage is an important contributing factor to the force developed by a muscle. Subjects with a so called "long face" have a relatively large mandibular plane angle and a shorter ramus and develop lower molar biting forces as compared to normal subjects (Throckmorton et al.1980 and Proffit et al. 1983).

Another factor is tooth anatomy. It could also be that molars by having a larger occlusal table have a larger supportive area closer to the action of the muscles (Carlsson 1974). The vertical separation between the jaws is also important. Manns et al. (1979) have reported that an intermaxillary distance of 15 to 20mm of jaw opening, is the optimal masseter muscle length and thus at this opening the optimal bite force can be generated. Hellsing and Hagberg (1990) reported differences in maximal bite force with changes in head angulation and hyoid bone to mandible relationships. They found that the force increased with an altered head position.

Peripheral feedback either from the periodontal ligaments, intradental, or joint and/or muscle receptors affects the control of bite forces. Lund and Lamarre (1973) reported that anaethesia around the teeth reduced the force of maximal jaw closing contractions by 40%. In contrast, Hellsing (1980) showed an increase in the maximal bite force in both anaethetized and non anaesthetized subjects.

He also concluded that elevator muscle vibration did not influence maximum bite force. Lately, Teenier and Throckmorton (1991) showed there was no change in the voluntary maximal bite force levels or the levels of integrated EMG levels in anaesthetized subjects.

It has also been observed that in patients with reduced periodontal ligament support, due to periodontal disease or at its extreme due to loss of teeth and therefore wearing dental prostheses, that the level of bite force is decreased (Laurell 1985, Williams et al. 1985, Lundquist et al. 1986). This is in contrast to patients with implanted prostheses where the level of bite force has been seen to return to a pre-edentulous level or even surpass it (Haraldson et al. 1979).

Patients with disturbances of the craniomandibular system such as pain from muscles or joints are reported to have lower maximal bite force values than healthy subjects (Helkimo et al. 1975, Molin 1972). In the patient group studied, as the pain subsided the bite forces increased. They also observed no significant differences between the affected and non affected sides.

In patients with a history of clenching and grinding habits it was found by Helkimo and Ingervall (1978) that these patients differed from the norm because they achieved lower levels of bite force than the controls.

In a measure of endurance with a 50-Newton bite force in patients suffering from temporomandibular joint disorders, it was observed by Stegenga et al. (1992) that TMJ pain was the main reason for the cessation of the biting effort, in contrast to control subjects who ceased biting due to muscle fatigue.

-Masticatory Forces-

Several techniques have been developed over the years to measure the human masticatory force. The instruments used for this purpose are referred to as gnathodynamometers. The earliest of these date back to the late 19th century (See Klaffenbach 1936). The level of biting force needed to crush different types of nuts was measured by these biting instruments. These were made of spring steel arms, which were held between the teeth by the subjects with varying degrees of comfort.

Rugh and Solberg (1972) in their review, looked at the historical use of bite blocks to measure forces and credit G.V. Black in 1895 for recognizing that bite forces, were dependent on the degree of separation between the jaws, and for noting that the force was not being exerted equally across the occlusal surface of the teeth.

Major advances were made by the late 1940's, as variable inductance and wire strain gauges became available. Howell and Manly in 1948 developed a variable inductance type of gauge, with interchangeable bite plates, in order to attempt

to deal with the interocclusal separation at the time of measurement, which was shown to affect the level of attainable force, at varying degrees of jaw separation. Biting forces were applied to a spring steel plate that moved a silver foil near an inductance coil, as the inductance changed a meter type circuit changed the variation in inductance to a DC current, which then indicated the level of biting force.

Later, small strain gauges were mounted within a single tooth. Anderson (1956), Scott and Ash (1970). Strain gauges have been placed under artificial teeth, Brudevold (1951), Yurkstas and Curby (1953), Atkinson and Sheperd (1967) and others.

Strain gauges have been placed inside acrylic bite blocks in order to measure force distribution and the effect of varying the position of the bite point on muscle forces (Kikuchi et al. 1994).

Most commonly the indirect measurement of bite force has been performed using bite forks (Haraldson et al.1979, Helkimo and Ingervall 1978, Rugh and Solberg 1972) to mention a few. The masticatory force is highest when the food is initially crushed between the teeth and the values are highest for hard foods such as nuts and carrots (Carlsson 1974). Levels of force can reach as high as 400 Newtons during normal mastication (Hagberg 1987).

ARTICULAR STABILITY

There is little literature regarding the stability of this joint and its supporting structures and constraints. It therefore remains unclear to what extent the configuration of the articular fossa and the condylar head and disc, under loaded and non-loaded conditions, contributes to mobility and/or stability of the articulation. It may be hypothesized that the less the joint elements are congruent, the less stable the system would be (Osborn 1985).

Mandibular movements are controlled by muscles, their innervation, and by biomechanical constraints such as the hard and soft tissue anatomy of the joint and the dentition. Osborn (1985) suggests that during jaw movement, and especially during mastication, the function of the disc is to not only spread the forces exerted on the articulation, but also to limit the movement of the condylar head into the retrodiscal tissues and the temporal bone. Thereby, allowing the articulating surfaces as well as the disc, sufficient freedom to rotate and slide in the fossa and down the eminence. He believes that in reality the disc destabilizes the condyle by keeping the incongruent bony surface slippery by its low coefficient of friction, and it is the annulus, to which both the

anterior and posterior thickened bands contribute, which maintains stability and which can do so without muscle action.

Williams and Warwick (1980) in their review of joint function state that joints are maximally stressed at the limits of their movement and it is in this position that the surfaces become congruent and 'closely packed', and in this way able to resist further movement.

Osborn sees the temporomandibular joint as an exception. It is not braced and closely packed when maximally loaded but it is held "balanced" by its supporting ligaments and musculature as the stresses are placed on it in order to achieve stability (Osborn 1989).

Hesse and Hansson (1988) have suggested that close-packing only occurs in joints when the supporting ligaments have been stretched to their limit and are taut. This is not the case when teeth intercuspate and transmit loading forces during mastication or hyperfunction in humans.

The mechanics of the other soft tissues found intra-articularly would lead us to question the idea of the articulation as ever close-packed. Scapino (1991) has expressed, that upon articular loading we find retrodiscal compression and venous collapse, and during opening, reduced retrodiscal pressures cause the expansion of loose connective tissue fibers and venous spaces, with blood being drawn into the articular region. This venous cushion would impart a viscoelastic damping to the

loading stresses placed on the articulation and provide for some give as loads were placed on it thus destabilizing any close packing tendencies.

Others have viewed stability, and the role the disc plays in it, as the ability of the condylar head and soft tissues to remain statically positioned in the fossa as compressive forces pass through the articulation. Okeson (1993) has lately viewed the purpose of the disc, as an interpositional medium of dense fibrous connective tissue that protects, and stabilizes the condyle in the fossa.

At first these apparently different views seem to contradict each other, but actually with further understanding the views expressed above are not that dissimilar. There is common support in the literature for the role elevator muscles and the supporting ligaments play, and these are widely understood to play a major role as the stabilizers of the temporomandibular joint articulation. Optimum joint position being achieved only when the articular discs are properly interposed between the condyles and the articular fossae. An unstable joint then would include one, in which the disc and intra-articular tissues fail to provide the articulating surfaces with the necessary support required, for loaded condylar motion.

The role of the musculature in generating compressive forces on the articulation has been discussed above. It is not difficult then, to presume that these muscle forces generated by the elevator musculature contribute to the apposition of

the condylar head, in the fossa, with interpositional fibrous damping being provided by the disc and the retrodiscal connective tissues.

During jaw opening the role of the musculature is less clear. We have discussed above that some view the role of the ligaments as restraining elements during jaw opening. Osborn (1989) has expressed that the temporomandibular ligament restrains the condyle against the eminence during opening and mastication, and that the sphenomandibular ligament, even though may exhibit some slack with the teeth in contact, restrains the size of the gape during maximum opening.

The musculature of the jaw is expected to have passive muscle tension as the muscles are lengthened during opening, with each muscle exerting increases in tension depending on their attachemnt position relative to the jaw movement. Some muscles can be expected to be lenghthened more than others, and also have different trajectories of movement during different functions (Goto et al. 1994). The varying tensions generated by these muscles have not been directly measurable.

Lately, computer assisted models have been used to study the dynamic changes in jaw muscle tension and the effect of jaw movements on articular compression. The forces generated are directed approximately perpendicular to the rear slope of the articular eminence, and seem to peak at maximum jaw opening, with a total compressive thrust of 40-50 Newtons. This seems to indicate that in a normal

joint during voluntary jaw movements, the system has a capability to produce sufficient muscle tension to mainatain appositional articular loading during jaw opening and may play a significant role, in stabilizing the temporomandibular joint. (Langenbach and Hannam, in press)

SUMMARY

In order to understand the craniomandibular system, increasing efforts have been made to understand muscle contraction, jaw motion, masticatory and joint forces, as part of the theoretical framework used to diagnose and manage disorders of the craniomandibular system.

Biomechanical models have combined the anatomy of the system, with their primary function of developing tension, to discover the direction of forces developed by the muscles of mastication, relating these to loading patterns and changes observed to occur in the system under function.

FEM models have confirmed the prediction that the mandibular condyles are load bearing, with greater force magnitudes being transmitted bilaterally during intercuspal and incisal clenching, as well as through the balancing side articulation during unilateral clenching. In symmetrical biting, contact positions that lie anterior to

the canine region cause compressive condylar reaction forces, while more posterior molar biting locations create higher tooth forces.

During asymmetrical unilateral occlusal tasks, higher condylar reaction forces on the non-working or balancing side will occur. On the working side, the condylar reaction forces will tend to decrease and may even cause the working condyle to distract or decompress for posterior tooth contacts.

Bite force measurement has provided a variable that can be related to factors such as the state of the occlusion, the type of dental treatment undertaken, condition and function of the articulation and its related musculature. The intraoral measurement of these forces has proven most reliable with the use of strain gauges attached to a bite fork.

Individuals of varying anatomy and morphology can produce similar biting forces. Joint stability increases for more distant bite locations, and the differential activity of working and balancing jaw muscles helps to ensure the moderation of intra-articular forces while accounting for higher bite forces.

In applying biomechanical principles to loading forces on the bony jaw elements, studies have tried to show a correlation between muscle forces and the counteracting role played by the teeth, the state of the occlusion, the role of the disc

and connective tissue components, that play a part in the proper fuctioning of this articulation and perhaps try to point to why and how the system does reach its physiological limit, and break down.

STATEMENT OF THE PROBLEM

The etiology of human temporomandibular joint disorders is due to a number of factors. Of these the role of adverse articular mechanics in the progression of change in the joint has been implied but not proven. It has been observed that loading beyond the physiological limits of the soft and hard tissues of the joint may lead to acute inflammation in the short term and remodelling, in the long term. It is possible that loading which exceeds the physiological limits of the structures, under clenching, grinding, abusive habits or primary trauma leads to disordered conditions that often present themselves initially as smatognathic pain. Evidence exists for a significant innervation in pericapsular connective tissues. It has been shown that the trigeminal subnucleus receives nociceptive information from components in the TMJ, and one could infer that afferent input from pain fibers may also be important with respect to referred pain from the joint. Afferent input in humans is cognitively perceived, and can be expressed verbally as pain.

Although, it has proven difficult to measure directly loads within the human temporomandibular joint, model simulations support the suggestion that higher loads occur in the area of the joint during clenching tasks and that the contralateral or balancing side of a clench measures even higher loading patterns. In patients with missing posterior tooth support, the load in the joints is higher than with contacts present. Pain may ensue that can often be moderated by providing prosthetic appliances which reestablish these posterior contacts.

Interocclusal orthopaedic appliances or splints have been used routinely for the treatment of temporomandibular joint disorders because of their ease of use, and when properly managed, because of their efficacy. The value of such orthopaedic interocclusal appliances have been evaluated in a number of studies in the past, and thus various theories have been proposed with regard to the method of action of different appliances, but at present no conclusive testing of these theories has been presented.

In the present thesis, it is hypothesized that in acute articular pathology, the use of an orthopaedic appliance designed to reduce the load to the painful joint can positively influence the resolution of arthrogenous pain in the short term. Specifically, it is proposed that this could be achieved with an appliance designed with unilateral occlusal support.

METHODS AND MATERIALS

In order to test the hypothesis two separate studies were carried out, one on a computer to verify the biomechanical effect of the test device, and the other a clinical study to test the hypothesis on patients suffering from an articular disorder.

BIOMECHANICAL MODEL

The first study made use of an existing computer model of the mandible. The model was based on a technique known as finite element modelling (FEM). A freshly dissected human mandible was imaged with the aid of computerized tomography (CT) scans at 2mm intervals. The outer and inner cortical outlines were retraced, digitized and assembled into a three dimensional finite element mesh with available software (I-DEAS 6, SDRC, Milford, OH). The simulations were carried out on a unix based Hewlett Packard 9000 series, 380 mini-computer work station. This model was developed by Korioth (1993) and the full description of the process can be found elsewhere. (Some details of this model are described below for clarification).

Finite element modeling is an advanced numerical technique developed for engineering structural analysis. It subdivides the complex geometry of a complicated structure like the human jaw into a mesh of smaller elements. These elements are then interconnected at specified points (nodes) on the element

boundaries with defined degrees of freedom. The use of mathematical functions allows for the generation of a series of equilibrium equations (one per nodal degree of freedom) that, when summed across the entire geometry and solved simultaneously, define the structure in question.

The reconstructed mandible was divided into multiple interconnected elements. The structure of the temporomandibular joints was modelled as a two layered "cap", in which the first layer consisted of the combined thicknesses of the articular fibrocartilages, and the second consisted of the temporal cortical bone. This permitted analysis of the stress distribution on the condyles and allowed for some theoretical buffering effect of the articular disc against the temporal bone.

In collaboration with Korioth, the FE model was used to simulate two clenching tasks on an acrylic appliance that was modelled with similar characteristics to an orthopaedic maxillary occlusal splint. This appliance was assigned the material properties of heat cured acrylic and it optionally opposed all mandibular cusps. The model was loaded with multiple force vectors and rigidly restrained from movement at the maxillary teeth and the endosteal surface of the temporal bone. Groups of parallel vectors simulated nine pairs of masticatory muscles (superficial and deep masseter; anterior, middle and posterior temporalis; medial pterygoid; superior and inferior lateral pterygoid; and anterior digastric), assumed to be directly attached to the bony surface. Their directions were derived algebraically as unit vectors from single

vectors of muscular attachment available in the literature. The magnitude of the total muscle force exerted by each muscle during isometric contraction was given by the product of a Weighting Factor (representing maximum possible tension) and a Scaling Factor (representing muscle activation levels. The orthogonal vector force components were subsequently proportioned between the nodes comprising a specific area of muscle attachment. (see Korioth and Hannam 1994).

The two occlusal tasks modelled clenching in the intercuspal position (ICP) with clenching with and without contralateral tooth contact against the acrylic appliance. The contact of the cusps tips opposing the acrylic splint were either evenly distributed across the arch (Bilateral Contacts), or removed on one side from the incisor region to the last distal contact. (Unilateral Contacts). The ensuing compressive stresses (negative sign), also termed Minimum Principal Stresses, were then analyzed for each task and for each joint and expressed in MegaPascals.

(See page following for colour photocopy of Model)

FE model of a human jaw with overlaying intraoral orthopaedic acrylic splint. Model was made in collaboration with Korioth (Korioth, 1994).

CLINICAL STUDY

Two methods of assessment were used in the clinical part of the study, and are reviewed here.

THE MEASUREMENT OF ORAL BITE FORCES

Bite force measurements were made in order to standardize the level of masticatory force exerted and to replicate the mechanical stimuli directed to the temporomandibular joints for each subject at each assessment session.

Instruments that measure human forces are known as Gnathodynamometers. The earliest efforts at estimating the force required to crush certain food stuffs dates back to the 19th century. Black (1895) and other early pioneers in the years that followed, developed gnathodynamometers using spring steel arms which the subjects bit upon. As many others have since, he encountered a variety of problems. (See review by Rugh and Solberg 1972). The thickness of the metal, its ease of use, the recognition that biting forces depended on the separation between the teeth, the calibration and the method used to record the level of bite force contributed to a variety of designs.

For the purposes of this study a thin bite plate of sufficient strength and plastic properties to withstand high clenching forces was required. The instrument described below is modelled after a design by Rugh and Solberg (1972) which consisted of a cantilever bridge and strain gauge arrangement with a bite plate thickness of 1.5mm before the placement of balsa wood bite pads which were added for stabilization of the cuspal contacts. Construction was simplified by using a set of ASH articulating forceps, modified to form a dual cantilever bridge arrangement. Small strain gauges were mounted on both sides of one of the forceps handles. The twin gauges provided for temperature compensation and doubled the gauge factor. A positioning bar was placed at the edge of the bite pad in order to keep the orientation of the bite force as perpendicular to the occlusal plane as possible, and in order to reproduce the location of the occlusal force on the dental arch. Several materials were tried to cushion and stabilize the occlusal contact points. Cotton wood sticks were cut to fit the occluding surfaces, and were cemented into place and soaked in water before use in order to soften the wood. The force transducer was attached to an Ammeter in order to measure the bite force in Newtons. It was calibrated with an Instron Tensile Tester, and found to be linear. (Calibration chart in Appendix 1).

EXPERIMENTAL MEASUREMENT OF PAIN

The level of symptoms elicited by the clenching effort at each assessment session, for each subject, was recorded in order to obtain an objective measure of the level of pain that the clenching effort exerted on the temporomandibular joints.

In many studies, only the presence or absence of pain has been reported (Clark 1984). When pain has been assessed, the use of rating scales or visual reports are most frequently used. The most commonly used measure in studies of experimental and clinical pain in humans has been the subjective verbal report.The human subject is unique, in that he or she can provide introspective verbal reports of pain. Both verbal and non-verbal scales have been developed to evaluate different aspects of pain perception. The evolution of these approaches traces back to the more traditional ordinal scales, which asked patients to rate their pain as mild, moderate or severe, to the present approach of using numerical scales that incorporate the principles of cross modality matching (Stevens 1975). In such experiments, subjects are asked to match the level of a modality, such as line length, elapsed time, handgrip force, or in this case occlusal force, with various levels of stimuli from another modality (sound, light, weight, etc.). This provides for a bias-free ratio of data that allows for parametric analyses of the responses to the stimuli.

A seminal verbal test that became a standard for the multi-dimensional mesurement of pain was the McGill pain Questionnaire. This classification scheme was described by Melzack (1975) and others since, to distinguish among different pain syndromes and to be sensitive to some therapeutic manipulations. Other verbal scales, such as the Verbal Descriptor Checklist, allows for a quantitative measurement of the sensory and affective dimensions of pain by using words that share a commonality across different pain syndromes. Numerical values, previously assigned to the descriptors by a comparable population using cross modality matching techniques, result in a scale with ratio characteristics. Thus, allowing a parametric, multidimensional assessment of the subjects painful experience. (Gracely, McGrath and Dubner 1978), (Heft, Gracely and Dubner 1984), (Duncan, Bushnell and Lavigne 1989).

One of the most widely used non-verbal measurement techniques is the visual analogue scale (VAS). This measure is a form of cross modality matching. It consists of a horizontal line, of predetermined length, with each end representing an extreme state for the criterion under consideration, defined by appropriate opposing words or phrases. The subject is asked to mark his or her status somewhere between these two extremes, by placing a mark on a 10-cm line. It has been found preferable not to mark intermediate reference points on such a scale as this influences the responses and their uniform distribution and cluster around the reference points are lost. This technique is valid and reliable as a measuring tool for both clinical and

experimental pain (Price et.al. 1983). A review of the assessment of pain in the temporomandibular joint can be found in Stegenga et al. (1993). The VAS format used in this study is shown on Appendix 2.

METHODOLOGY OF CLINICAL STUDY

The clinical study involved the recruitment of two groups of subjects who were either symptomless (Normals) or diagnosed with primarily a unilateral joint disorder. All were females between the ages of 25 and 45. This selection was due to the predominance of TMJ disorders in women. They were recruited either by responding to a posting requesting volunteers for this research study (Normals) or by presenting with temporomandibular joint symptoms at one of two clinics; A University based clinic that specilalizes in treating patients with craniomandibular symptoms, and the private dental clinic of the author. (This constituted the Treatment Group).

The clinical examination and history taking procedure was common to all subjects. It consisted of the history questionnaire and examinations forms as discussed by Widmer from La Resche (1992), following the guidelines set by Dworkin et al. (1992), for a set of standardized research diagnostic criteria for temporomandibular disorders or RDC/TMD. The relevant criteria are summarized in Appendix 3.

These criteria are intended primarily for research purposes, allowing standardized methods for gathering relevant data and making possible comparison of findings among different investigators. The diagnostic system is nonhierarchical and allows for the possibility of multiple diagnoses for a given subject. For the purposes of this study the most important clinical condition for inclusion in the treatment group was the presence of primarily unilateral arthralgia (an Axis I Group III diagnosis). Any sign of disc displacement or Group II diagnosis did not rule the subject ineligible, if in addition to the displacement and or disc incongruency, arthralgia was present in one joint. The history of the symptoms at the time of examination varied from three days to three weeks and had to be of acute origin, that is, of sudden onset and persistent. As will be discussed later, subjects suffering from Axis I, group I, (muscle disorders) primarily were excluded from the study, as were patients suffering from bilateral craniomandibular symptomatology.

Normal subjects, (comprised of 6 subjects) who were the "normal controls", showed an absence of symptoms upon history taking and examination. These subjects were asked to clench maximally on the gnathodynamometer's bite pads and to maintain this clench for 2 to 3 seconds. Immediately upon performing the task, the measurement of the bite force was recorded for both sides of the arch, as was the measure of any sensation elicited, (or its absence) for each joint. The articular sensation was measured with the aid of the visual analogue scale, which required the

subjects to assess their sensation by marking a position on a straight line with predetermined end-points at 10mm of length.

Patients affected with disorders (12 subjects) were randomly assigned into Groups I and II (Treatment Groups) in order to test the efficacy of two treatment appliances. They followed the the same protocol as above with regards to clenching on the force transducer and notating their discomfort on a VAS scale. The treatment approach varied by the type of occlusal appliance used. As these were symptomatic patients, with a history of an acute onset unilateral arthralgia, Group I subjects received a conventional maxillary oral orthopaedic appliance commonly used in the treatment of temporomandibular joint disorders. This appliance was a flat-plane, occlusally balanced appliance (Intra-oral Splint) which was worn 24 hours a day except at meals. At 3 sessions following the initial placement of the appliance (approximately one week apart) these patients were asked to return for follow up assessment, at which time the pain and force measurement protocol was repeated and the splints adjusted as necessary.

Group II subjects, (comprised of 6 subjects) underwent an identical protocol to the other treatment group. They were provided with a modified orthopaedic appliance after ascertaining which side (temporomandibular joint) was the one presenting with painful symptoms. A unilateral occlusal support on this appliance was maintained or augmented on the side of the arthralgia and removed from the

contralateral side. This was done with the use of cold-cure acrylic and acrylic burs as used conventionally when adjusting occlusal appliances. The altered occlusal scheme was expected to reduce the load and possible compression that the painful joint was hypothesized to be under during the treatment period. This altered occlusal balance was maintained during the 3-4 week treatment period. At 3 sessions following the initial placement of the appliance, these patients were also re-assessed for their symptoms. The level of arthralgia was measured after each clenching effort on the gnathodynamometer (force transducer), as was the level of bite force for each clenching effort. At the completion of the treatment period the appliances were permanently modified to the more conventional, balanced bilateral contact design, providing equal support to both joints. This is necessary to prevent any unwarranted tooth movement. In this group if the pain worsened during treatment, the subject's treatment protocol was modified immediately to the conventionally designed appliance and the subject's treatment reassessed.

All subjects in Group I and II were treated for the time necessary for the resolution of all symptoms, or until such time that they decided to discontinue any further treatments. Their treatments and pain control were handled in the most ethical and professional manner possible. All subjects were asked to abstain from analgesics and anti-inflammatory medications and alternative treatments during the study period. If this was not possible, they were removed from the study and treated for their symptoms in the conventional manner. If it was deemed necessary to place any

subject on additional treatment, this was undertaken following the study period. Patients were asked to keep a record of appliance use during the treatment period.

Figures presented in the results illustrate the level of pain immediately after performing the clenching task on the force transducer plotted against the pre-treatment session and the 3 post-treatment sessions.

RESULTS

MODEL PREDICTIONS

The compressive stresses at the site of the condylar heads during the simulated intercuspal clench, as analyzed by the IDEAS software for this study, are presented in the Table below.

	BILATERAL CONTACTS		UNILATERAL CONTACTS	
	Right Condyle	Left Condyle	Right Condyle	Left Condyle
Total	-151	-140	-230	-115
Max.	-8.1	-7.1	-12.2	-6.5
Avg.	-3.7	-3.4	-5.6	-2.8

Measurements are in MPa (1 Mpa = 1x10E+06 Nm^2). Negative sign indicates compressive stress. Data in Table reports the compressive stresses as measured by the FE model for the two clenching tasks.

The data clearly supported the view that the location of occlusal contacts affects the magnitude of compressive stress on the supporting condyle. There was a dramatic (two-fold) increase in the compressive stresses experienced by the condylar head on the side contralateral to the contacting side. In other words, when the right occlusal contacts were removed from contacting the acrylic splint, the condyle experienced a marked increase in compressive stress. It was also noted that the working side (Left) condyle experienced LESS compression when the occlusal pattern was modified from bilateral to unilateral support. It is presumed that the addition of the acrylic material interposed between the two arches absorbed some of the stresses at the tooth contact locations. This also decreased the condylar load levels when compared with clenching activities without the interposed acrylic as presented by Korioth and Hannam (1994).

CLINICAL STUDY

The level of pain at the start of the examination of the normal group, as measured by the VAS scale, was zero.

The "normal" control group (6 subjects) did not present with any notation on the VAS scale above the level of 1 for right and left sided tasks. These values were typical of asymptomatic subjects. Repeated attempts at eliciting pain intensity levels above 1 for either joints were unsuccesful, related to the clenching tasks

(the mechanical stimulus) on the force transducer. The absence of symptoms helped to confirm grouping these subjects in the asymptomatic control group.

The levels of bite force varied between 100 Newtons and 200 Newtons, well within the normal ranges for these individuals as reported by other workers. Repeated tries at the clenching task (during the same sitting), were of similar levels for left and right sided clenching on the gnathodynamometer.

All the patients receiving treatment, either with the conventional balanced appliance or the unilaterally contacting appliance, benefited to some extent from wearing the orthopaedic device over the 3 to 4 week treatment period. The difference in pain levels (as notated on the VAS), infers that those provided with the unilaterally contacting appliance showed the most profound change in their symptoms over this treatment period.

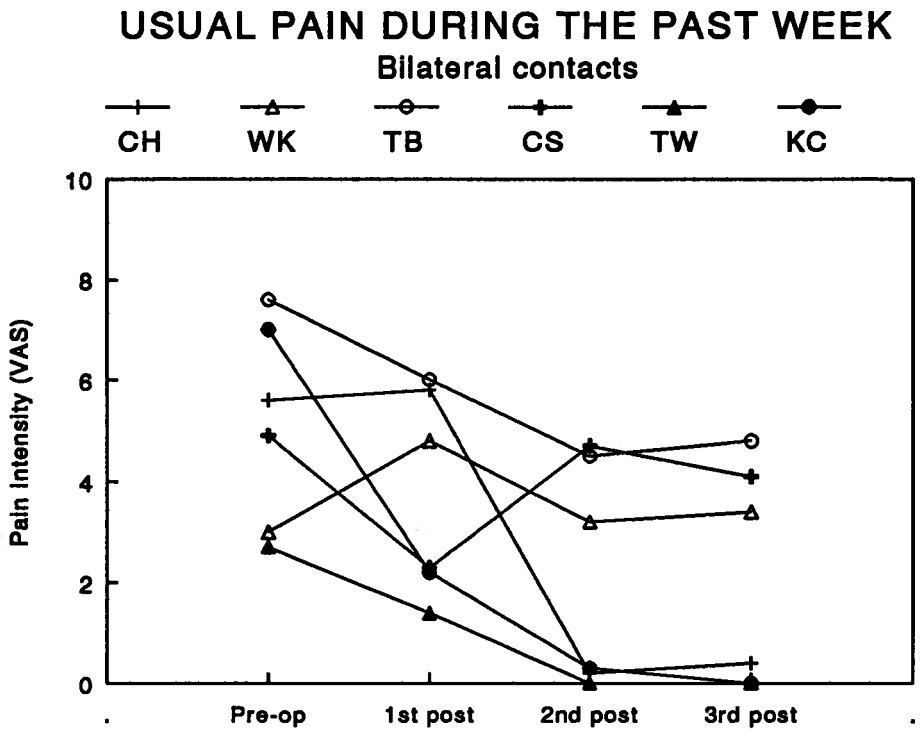
The data are presented as scatter plots in Figures 1 through 5, which represented the pain intensity reported on the VAS scale as plotted against the PRE-OPERATIVE and 3 POST-OPERATIVE sessions. They highlight the variance in the symptomatology of both treatment groups from the PRE-OP SESSION, that is the level of perceived pain in and around the temporomandibular joint before the delivery of any appliance, through the 3 POST-OP SESSIONS at which each re-assessment of symptoms was performed.

Figure 1 depicts the level of pain in both treatment groups in response to the first question on the VAS scale, viz. "Rate the intensity of the usual pain during the last week." Both groups showed an overall decrease in symptoms over the treatment period. What is most striking here is the change in variance from the pre-op to the 3rd session post-op, in the new appliance group. The range in symptoms at the start of the treatment was similar for both groups; From 2.5 to 7.8 in Group I and 2.2 to 8.6 in Group II. This range, decreased most markedly in Group II, so much so, that for all 6 subjects the total range in the VAS at the end of the treatment period was 1.5 to 3.0. This was in marked comparison to Group I, where the range stayed the same, though decreased in terms of pain intensity.

Figure 2 depicts the change in symptoms perceived at the time of questioning. This rating of the pain was in answer to the question: 'Rate the intensity of the pain at present.'

The VAS range was virtually unchanged when the 1st POST-OP assessment was compared with the level at the PRE-OP session in the figures above. But here again, the change in the range of the level of pain for all subjects notably changed especially for Group II, the group treated with the "new" appliance, by the 3rd POST-OP visit. The pain level in most cases dropped to less than half that perceived at the start of the treatment for this Group. This change was not as striking for the conventionally treated group.

a)



b)

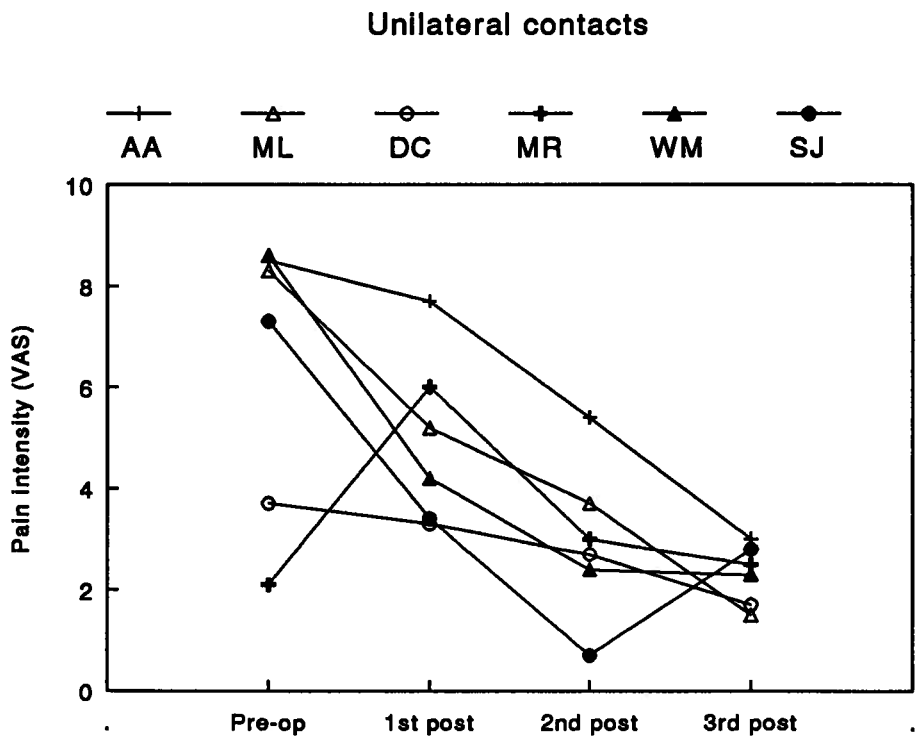
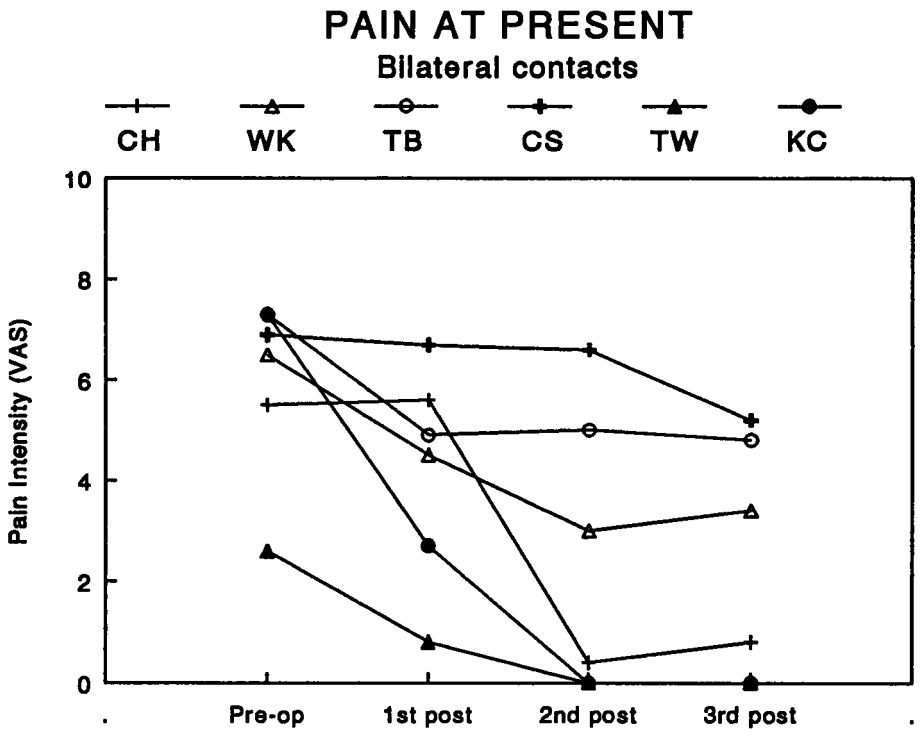


Figure 1 depicts the level of pain (in VAS) during the past week for both treatment groups (**a:** bilateral and **b:** unilateral contacts), plotted against the 4 assessment sessions.

a)



b)

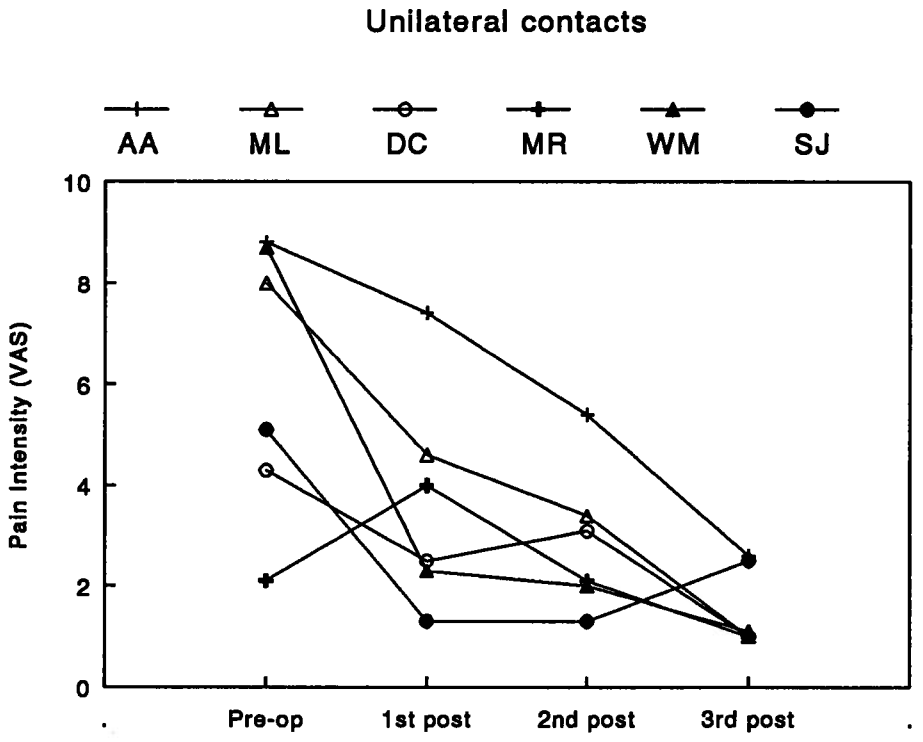
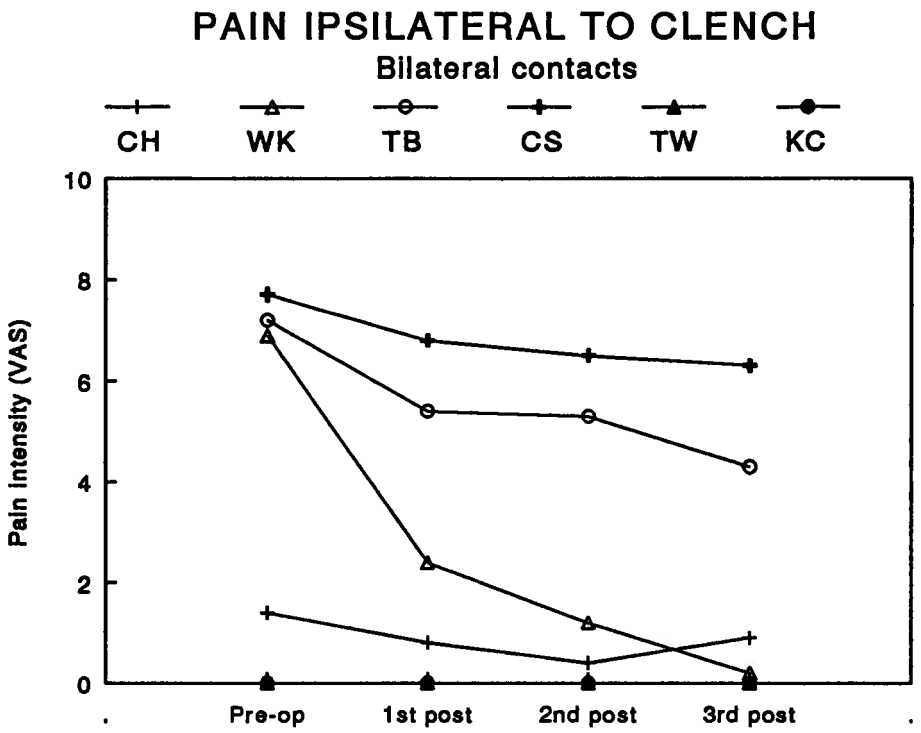


Figure 2 depicts the pain at the time of examination (in VAS) for both treatment groups (**a:** bilateral and **b:** unilateral contacts), plotted against the 4 assessment sessions.

Figure 3 depicts the level of pain perceived on the painful joint when the subject clenched on the side ipsilateral to the painful joint. For the group treated conventionally, the level of pain was not markedly changed when the load and stress on the joint was directed ipsilaterally. However, the effect on the unconventionally treated group was most noticeable. The change in the level of pain elicited by the clenching effort was similar to that recorded when these subjects were required to rate the level of pain "at present", without it being triggered by the clenching task.

The most striking change in the level of pain perceived, was found when the subjects were required to clench on the side opposite to the painful joint (Figure 4). This task was designed to elicit the greatest level of load to the painful joint. Consistent with the hypothesis that a clenching effort on the side contralateral to a joint causes the greatest load on that joint. The level of pain for the six subjects in Group II was highest for this task. All subjects were able to elicit pain levels in the painful joint higher than their 'resting' level of pain. The range however was low here, levels measuring from 5.2 to 8.6 at the PRE-OP SESSION and from 0.5 to 3.2 at the end of the treatment period. The change in the elicited level of pain was more dramatic than the one depicted in Figure 2b. This might be expected, since the level of pain was enhanced by the clenching task.

a)



b)

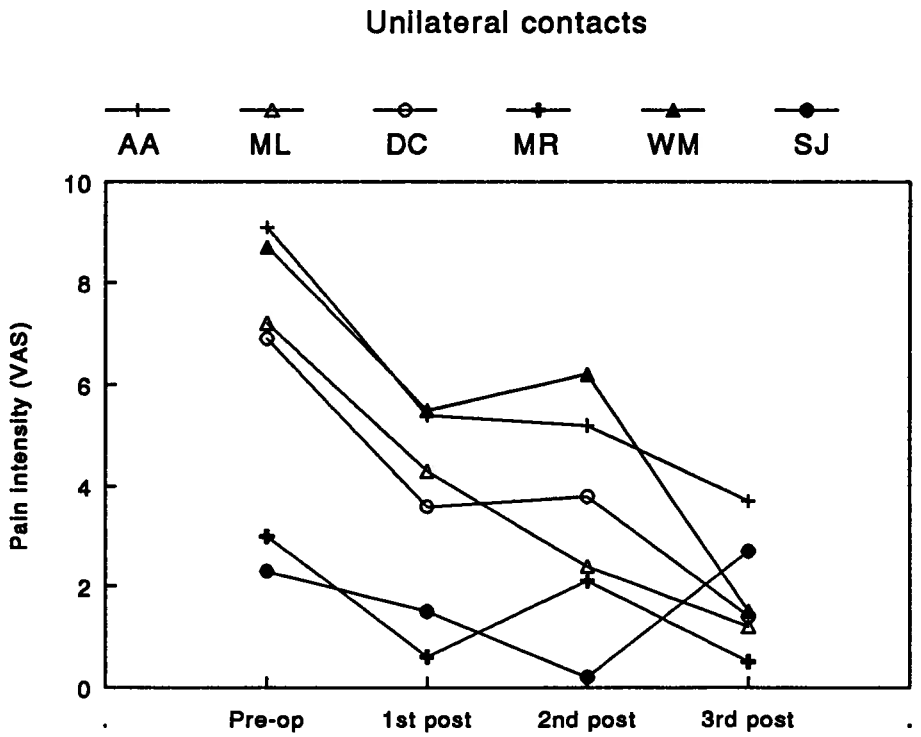
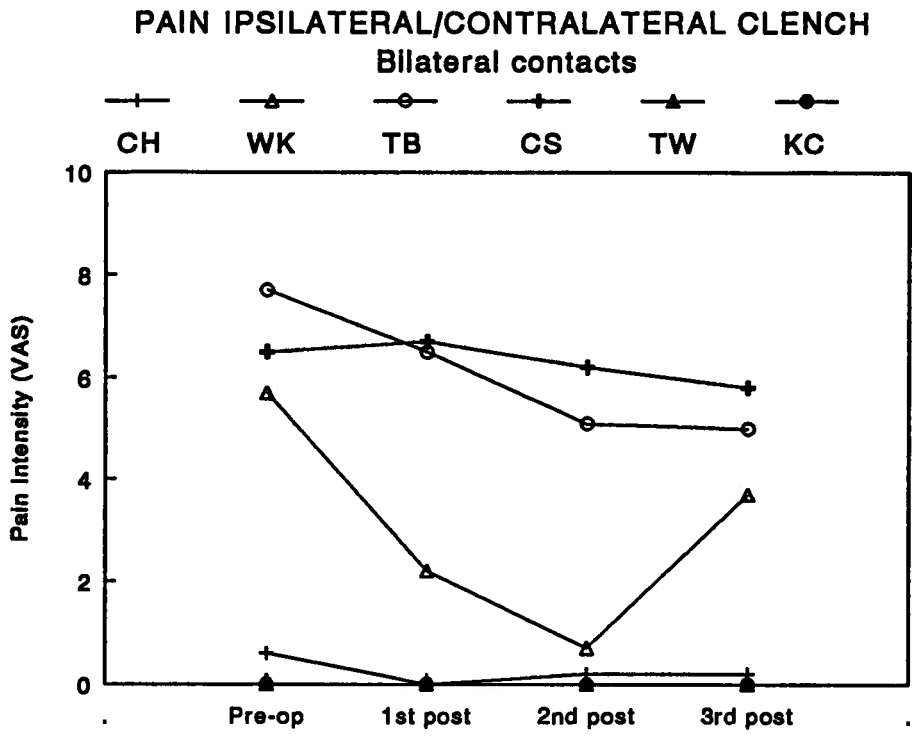


Figure 3 depicts the level of pain (in VAS) elicited by the clench on the ipsilateral side to the painful joint (**a:** bilateral and **b:** unilateral contacts), plotted against the 4 assessment sessions.

a)



b)

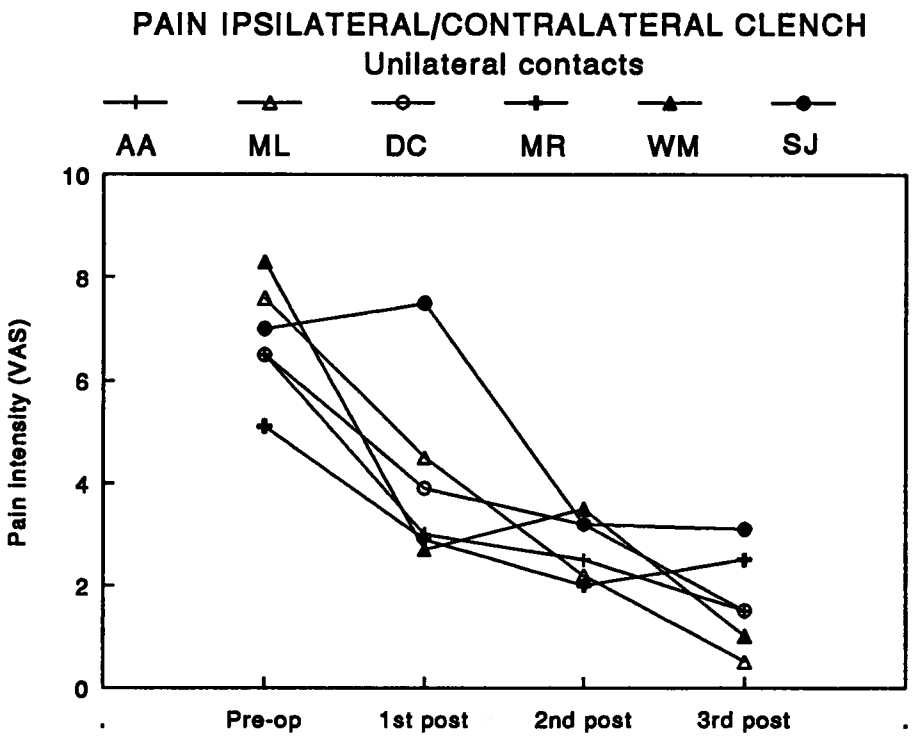
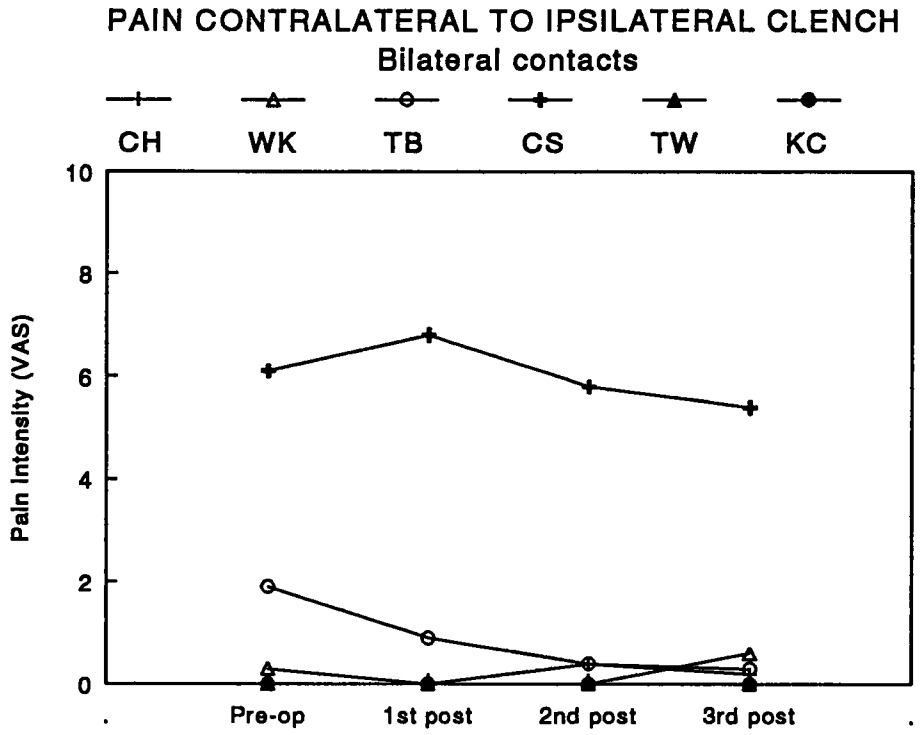


Figure 4 depicts the level of pain (in VAS) elicited by the clench on the contralateral side to the painful joint (**a:** bilateral and **b:** unilateral contacts), plotted against the 4 assessment sessions.

Finally, it was observed that when the clenching effort was directed ipsilateral to the painful joint but the pain level measured at the opposite joint, the level of pain to that joint also decreased noticeably for some Group II subjects (Figure 5). Suggesting that not all symptomatology was strictly unilateral. In addition, a clenching effort on the side of the painful joint apparently produced an excerbation of the normal loading and stress that the joint undergoes in normal function. Subjects in Group I did not exhibit such a marked change in symptoms for either the ipsilateral clench or the contralateral clench.

The force levels as measured by the gnathodynamometer (force transducer) were collated for all patients treated with appliances and as shown on Figure 6. The most salient feature was the general level of increase in the clenching effort that occured as the treatment progressed. This was true for both Groups I and II. As with the pain levels, the amount of clenching effort increased most dramatically when the measure was recorded while the subjects clenched on the side opposite the painful joint.

a)



b)

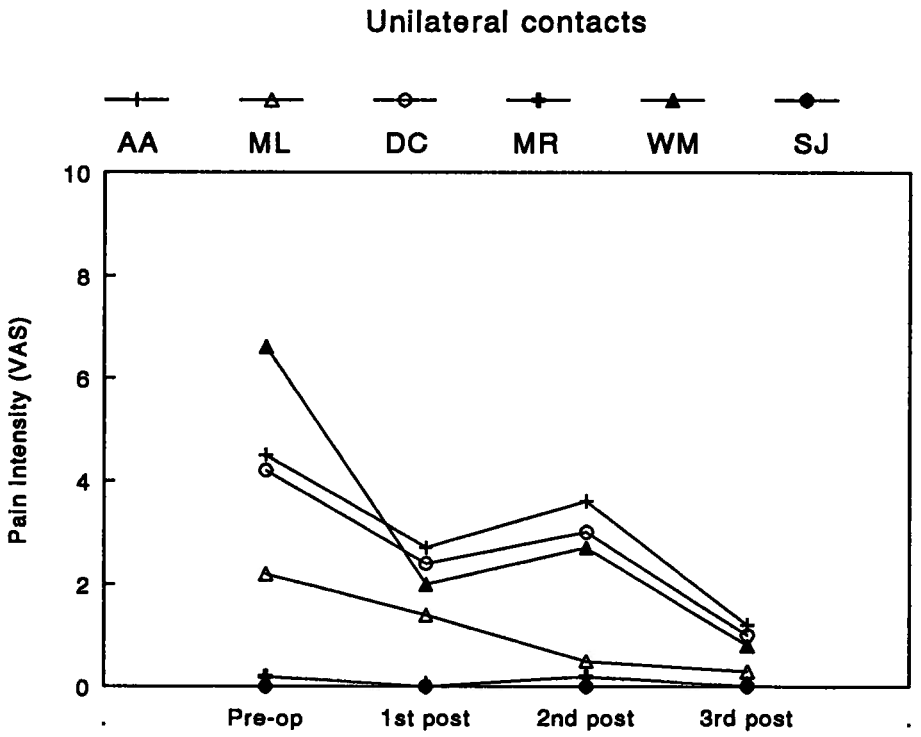


Figure 5 depicts the level of pain at the contralateral joint (in VAS) elicited by the clench on the ipsilateral side to the painful joint (**a:** bilateral and **b:** unilateral contacts), plotted against the 4 assessment sessions.

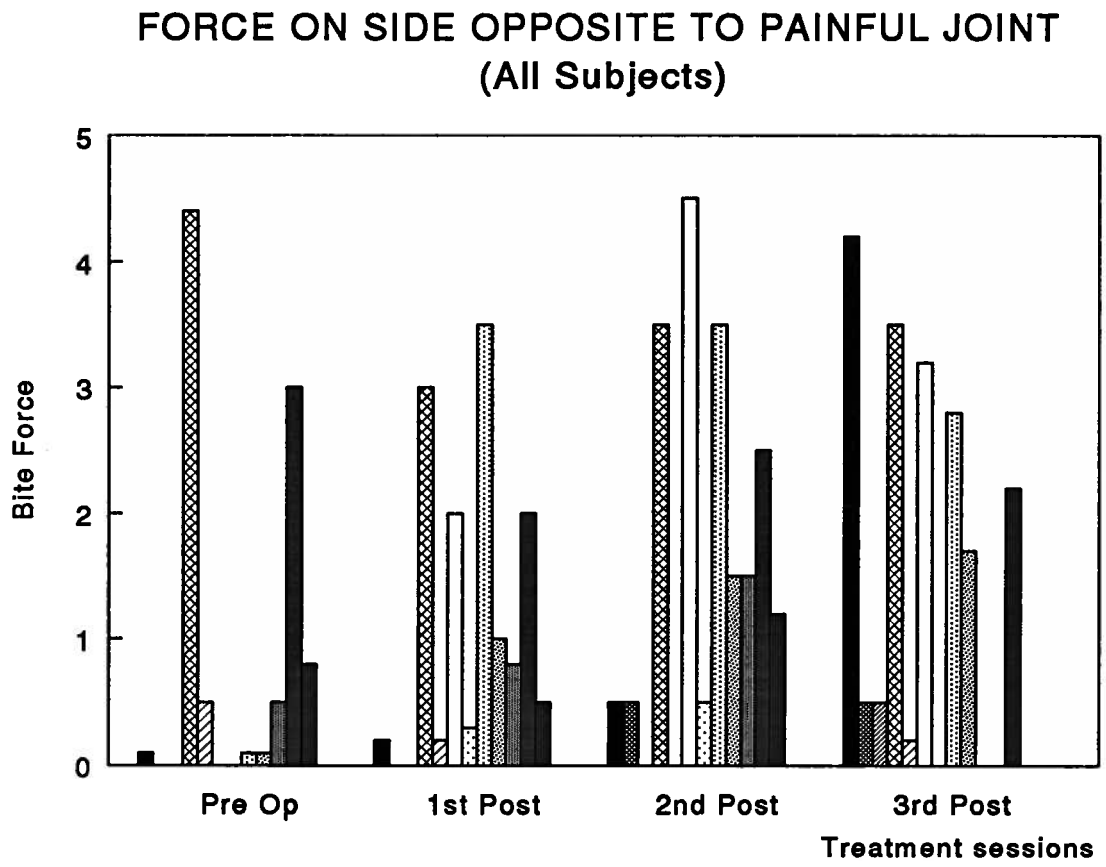


Figure 6 depicts the level of bite force as measured by the gnathodynamometer for all subjects during the study period. One unit of bite force equals about 20 N.

DISCUSSION

Mandibular condylar forces during jaw function have been measured in vivo in experimental animals. Transducers have been placed on bony surfaces in the subcondylar region (Hylander 1979) or directly on top of the condyles (Brehnan et al 1981 and Boyd et al. 1990). Metallic prostheses have been implanted in the ramus of the baboon near the TMJ (Hohl and Tucek 1982), and hydrostatic synovial fluid pressure has been measured within the superior compartment of the joint (Ward et al. 1990). Computerized models of the mandible (Korioth and Hannam 1994, Ferrario and Sforza 1994) support the view of the temporomandibular joint as a loaded joint.

During unilateral mastication higher loads are transmitted through the balancing side articulation than through the working side articulation, even though the latter authors suggest that with a change in the ratio of the muscles this imbalance can be altered, perhaps unconciously, as a compensation for the altered joint conditions. One must also be cautious when comparing models, due to differences in the calculation of the condylar reaction forces. Ferrario and Sfoza only accounted for the EMG potentials recorded over the masseter and temporalis when calculating reaction forces, in contrast to the EMG data used in the Korioth model, which made use of muscle data as presented by MacDonald (1982) for 9 pairs of muscles. This difference alone may have affected their conclusions. Nevertheless, condylar load distribution may be altered by cognitive influences.

Miller (1991) has presented data regarding the average muscle activation levels associated with various occlusal devices to predict changing loading patterns.

The results obtained in the present modelling study confirmed the dependency of condylar load distribution on the type of clenching task. The simulations support the notion that higher loads and stresses are observed on the condyles contralateral to the clenching effort. The patterns of stress distribution are also task dependent as was found by Korioth and Hannam (1994) for bilaterally symmetrical tasks, with the force distribution patterns differing between sides. This could be related to the asymmetry of the muscle forces loading the mandible, as the recruitment of the different muscles changes with the level of occlusal support. Only the superior aspects of the condylar surfaces were taken into account when analyzing the data, but the complete model came into play for the calculation of these compressive stresses including its inherent bending and rotational deformations.

These results attest to the usefulness of the FE technique for modelling forces on 3-D surfaces, and for developing a biomechanical justification for the hypothesis. At present there are no other methods for obtaining such a confirmation in the human temporomandibular joint.

The site and number of occlusal contacts affects the levels of muscle activity. Wood and Tobias (1984) showed that with the removal of six contacts on one

side of the arch of an occlusal splint, muscle activity decreased by 21%, with the authors suggesting that the balancing condyle absorbed the load previously taken by the balancing side contacts. One would expect the articular loads to decrease as the muscular activity decreased, due to the lack of contacts on the balancing side. This notion is supported by the model's predictions of higher loads in the contralateral or balancing side of a clench, and is not in conflict with a change in this imbalance being possible with a changing muscular balance or by changing cognitive influences.

The differences in the compressive stresses presented in the modelling study for both clenching tasks, attest to the possible biomechanical effect that an appliance designed to alter the location of occlusal contacts and with it, the direction and level of the muscular effort must have when placed intra-orally between the mandible and the skull. It is interesting to speculate on the possible effect the appliances designed to alter the biomechanical environment may have on the resolution of temoporomandibular joint disorders. It must be remembered that for this study the muscle forces were equal for both tasks simulated by the model. The structure, muscle architecture, and muscle recruitment were kept the same, but there is as wide variation in the architectural anatomy of patients who seek dental treatment. These anatomical and physiological differences may sum with different patterns of muscle use, to create load and stress characteristics that differ markedly for each task and possibly for each individual.

The recruitment of subjects for the clinical study, proved to be a challenge due to the need to include only subjects who suffered primarily from unilateral joint arthralgia. Efforts were made at the outset to keep both treatment Groups similar with respect to histories and diagnoses in order to test the two orthopaedic appliance designs, but in retrospect Group I, the conventional treatment group, ended up with a slighly different profile from Group II. Both groups did have a preponderance for unilateral joint symptoms, but as was discovered later, the degree of the myalgias contributing to the overall symptomatology of 2 subjects in Group I could have affected the comparison. If one looks at Figures 3a and 3b one can observe that for subjects TW and KC the level of articular pain was nearly non-existent during the performance of the clenching tasks. By POST-OP SESSION 2, as is evident in Figure 2a, it was found that these two subjects had recovered most remarkably from their symptoms. It might be postulated that these subjects did not have a preponderance of arthralgia, but were suffering from both arthrogenous and myogenous symptoms, which in turn improved drastically with the reduction in activity that was possibly influenced by the conventional flat plane orthopaedic device.

A more optimum design for the random assignment of the patients to both treatment groups, would in retrospect have included the blinding of the examiner, since bias may have crept in, in the assignment of suitable patients to each treatment group. In addition, the presence of symptoms bilaterally could also have been due to tenderness in the lateral pterygoid muscle. At present there is no reliable method that

can be used to differentiate pain emanating from the lateral pterygoid muscle which lies in close apposition to the joint and intracapsular pain. Anaesthetizing differentially the capsule of the joint and the muscle could prove beneficial in the future, in order to mutually exclude the influence of each structure on the symptomatology.

The improvement of symptoms in the conventional treatment group could also be explained by any of the theories presented in the review of the literature above, for the use of these appliances, most significantly the cognitive and behavioral modification aspect of treating these disorders.

In a recent study by Linde and Isacsson (1990) an attempt was made to differentiate by clinically apparent signs, patients with disc displacement versus patients with myogenic craniomandibular disorders. They pointed out that signs of TMJ tenderness appear in 1% to 13% of the general population, while they appear in 36% to 55% in patients with nonspecified CMD. In their own study, they found that a tender TMJ was present in 33% of the patients they examined with primarily myogenic CMD. This difficulty in recruiting a pure sample of TMJ sufferers is not new. It may be, as they suggest, that some myogenic CMD patients have crepitation and some restriction in condylar translation and arthralgia because of some sub-clinical joint pathosis. Another possibility, may be our present inability using the CMD/RDC criteria for diagnosis and evaluation, to separate clearly enough these two sub-groups of CMD patients.

It is known from the work of Sessle (1987), that caudalis neurones responsive to the temporomandibular joint and craniofacial muscle afferent inputs, represent critical neuronal elements underlying the transmission of acute orofacial pain, and that these deep nociceptive inputs show considerable convergence in transmitting nociceptive information. The convergence of mechanosensitive fields is consistent with the view of pain referral mechanisms. Thus the possibility of "mistaking" arthralgia for myalgia is quite plausible and relevant to this study.

Expressing the results presented above in terms of a range instead of a mean or a specific stastistical test was a decision mitigated on the problems encountered in the size of the sample. With the present results it is expected that we will be able to more closely plan for a statististically valid sample in the future, since the range of our symptoms will allows us to plan for the optimum sample size. In terms of the variation in symptoms between visits, this may have been due to the somewhat irregular intervals of time between visits that occured due to scheduling difficulties, and this could have clouded the data. Given the constraint of possible iatrogenically caused tooth movement possible with a unilaterally supported oral orthopaedic appliance, a two test period of pre-determined length maybe all that is needed to show significant changes in pain levels which would in turn facilitate the statistical analysis and the regular scheduling of assessment sessions.

Flat occlusal appliances such as those conventionally used by most dental clinicians can produce exactly the opposite effect that is desired. While it is well established that in most cases the use of a flat-plane occlusal appliance decreases muscle activity, the design of itself cannot guarantee that a patient will gradually decrease muscle effort, in some cases, muscle activity during clenching on full-arch bilaterally supported appliances can actually increase, compared to clenching on natural teeth (Miller 1991). It is quite possible and perhaps a common occurrence in Group I subjects, that the effect of the appliance worn 20 plus hours a day, is to produce higher levels of muscle activity, with increasing loads to both temporomandibular joints than is hypothetically desired. In contrast, the lack of occlusal contacts on the side contralateral to the painful joint, as in Group II subjects, may reduce muscular activity as suggested by Wood and Tobias (1984). If so, the theoretical suggestion that the compressive stresses on the painful joint would be reduced (or perhaps even turned into traction of the joint) is understandable. This introduces a new hypothesis for muscle activation, based on the number and location of occlusal contacts.

It is sobering to consider the effects that may occurr when patients in the alternative treatment group clench and function while wearing the "new appliance". From the biomechanical perspective, standard appliances can be predicted to have different effects on muscle an articular symptoms. An appliance could have a profound effect on pain in one subject, yet a similar design in another with identical symptoms

could have a far less profound effect because it simply does not affect the local tissue mechanics. Customization of the so-called "new appliance" to reduce the apparent load and compressive stress to the painful joint may be justified, at least provisionally, according to our results, and could contribute to a reduction in inflammation brought about by intracapsular changes. This could be achieved by asymmetric ramps on the appliances, or anterior bite blocks of varying thicknesses, or by behavioral modification brought about by functionally altering the use of these appliances in combination with the above. Clearly, efforts to modify muscle use will require more than just altered occlusal conditions, since these cannot be maintained for extended periods of time due to the propensity of teeth to move. Nevertheless, the combination of such a biomechanical approach together with altered behavioral activity could prove useful in a staged treatment protocol, especially over the short term.

The intracapsular causes of pain have been reviewed earlier. Bradykinins and excessive particulate debris produce the release of prostaglandins and other mediators. The subintimal tissue of the synovial membrane is highly innervated, and early inflammatory changes may lead to a loss of lubricating ability of the hyaluronic acid and chondroitin sulfate present inside the joint, causing increased friction (Moses 1991). Lavage of the TMJ space has proven succesful in the short term (Quinn 1989). Inflammation and pain probably continues until there is a reduction in joint overloading. It is interesting to speculate on the possible findings that assays on Group III patients would show at the start of treatment and at the conclusion of a specific treatment

period. It is perhaps this combination of the change in the biomechanical milieu together with a change in the local mediating factors that may lead to more effective long term success in treating patients with TMJ arthralgia.

Another important factor in the measurement of efficacy of a treatment approach is the amount and level of compliance. Patient compliance while taking medicines can be assessed directly, but the degree of use of an appliance, and the manner of its use, are very important in the assessment of efficacy. In this study, patient compliance was monitored by the manner in which the appliance was handled at each re-assessment visit and also by the thoroughness, frequency and quality of the notes found in the patient diaries. Attendance at each assessment session also served as a measure of compliance. However, the difference between groups could not be measured with respect to the level of compliance.

Erskine et al. (1990) have suggested that when pain is experienced as acute, as in these two subject groups, it is considered relatively novel, and the accuracy of recall for its intensity is greater than that when pain is either chronic or when the patient has episodic experiences of pain. It is evident from Figures 1a and 1b that the memory of the pain was unaffected, from pain perceived to exist over the previous week.

The studies of Helkimo et al. (1975) and Molin (1972), suggest that patients with disturbances of the craniomandibular system, such as joint and muscle

pain, have lower maximal bite force capabilities than healthy subjects. This was again borne out in the present study. What appears most evident is that as pain decreases, the level of the bite force increases. What was not shown was a difference between the sides for the level of the bite force. Molin also failed to show a difference in the level of bite force between the affected side and the non-affected side.

An incidental finding during this study was the re-appearance of contacts on the occlusal appliances in the side from which they were removed. This was noticed often during the re-assessment sessions for Group II subjects. a simple explanation for this would be the possible change in oral habits that may have occured during the study period. In addition, some evidence exists for condylar repositioning alterations that may be occuring with altered appliance designs (Hagiwara 1994), plus the possible phenomenon described by Scapino's (1991), where during articular loading there is compression of the retrodiscal tissues with venous collapse followed by blood being drawn in once the retrodiscal pressures are decreased. This "decompression" of the painful joint was most interesting since it appeared to coincide with a reduction in symptomatology.

The term placebo has been applied to many types of substances used in a variety of different ways. The most common definition refers to a material that does not contain any active medicine and is pharmacologically inert. This has also been called a "pure placebo". An "impure placebo" refers to any substance or device

used as placebo that is not believed to be totally inert. Orthopaedic appliances can result, as with any treatment, in effects deemed to be placebo-like in nature. Green and Laskin (1972) have suggested that 40% of patients suffering from TM disorders respond favorably to placebos. A positive placebo may result simply from a positive doctor-patient relationship. Reassurance that the appliance provided for the treatment will be effective may be the most significant factor influencing the placebo effect. In this study attempts were made to remove this effect by providing all treatment subjects with an appliance for which the difference in treatment approach was not made obvious to them. It is conceivable nevertheless, that the clinician providing the treatment inadvertently treated both treatment groups differently. Although this was believed not to be the case, one way to avoid the problem in the future could be the blinding of the clinician as to the treatment approach or by having several trained operators.

CONCLUSIONS

1) The FEM model of the human mandible supported the hypothesis that the side contralateral to the working side of a static clench exhibits the higher compressive stress at the condylar head.

2) Patients diagnosed to be suffering from unilateral temporomandibular joint arthralgia and randomized into two treatment groups received a unilaterally supported oral orthopaedic appliance and were found to have a smaller range of painful symptoms and seemed to show a more dramatic improvement in symptoms than the conventional appliance treatment group, as measured by a VAS.

3) Bite force measurements increased in magnitude as the level of the pain decreased for both patient groups.

4) Future studies are indicated on a larger sample of patients to confirm the efficacy of short term unilateral appliance use in a clinical environment.

CLOSING COMMENTS AND DIRECTIONS FOR FUTURE RESEARCH

The comparison of benefit to risk considerations for two or more treatment modalities is not trivial and a decision not easily made. This is partly because most factors that affect benefits or risks are either not major considerations in specific situations or are so similar between alternatives therapies that they do not greatly impact the benefit to risk concept or decisions based on this concept. It is not often the case that a single factor is the sole basis for choosing a treatment approach for a particular patient. It is also true that not all patients need to have the same factors influencing the decision for the use of a specific approach.

The term benefit includes numerous concepts beyond that of a treatment efficacy. Efficacy relates to how well a treatment achieves its objectives, in a comparative way (eg., treatment A improves symptoms better than treatment B) or in a non comparative way (Treatment A improves patients' disease measures). Benefits also include quality of life considerations at the individual patient level or at the collective patient level . For a society, benefits should more often be considered in terms of the improved public health and improved utilization of resources, money and time. There are different types of risks in comparing two treatment approaches. A patient's failing to improve could be viewed as a type of risk, since the alternative treatment or no treatment at all might have provided the patient with a greater likelihood of improvement.

In the use of appliances there is often relative risks that should be considered. The term risk usually refers to the probability that a given experience yields a deleterious reaction. Under carefully controlled conditions the use of a unilateral appliance need not involve any risk. Even a patient's failing to improve on by its treatment could be viewed as a type of risk, since the alternative treatment might have provided the patient with a greater likelihood of achieving a therapeutic effect. This was not the case in this study.

Present treatment modalities for TMJ arthralgias may not be truly beneficial in shortening the long term resolution of the disease or disorder, but need to be considered a benefit in the short term reduction of the symptoms associated with the disorder. The benefit of considering the biomechanical approach to the treatment of this painful condition may be the enhanced capability of this treatment approach to reduce intracapsular inflammation and ensuing pain. The overall long term effect on the resolution of the symptomatology as compared to a conventional appliance may not be any greater but the quality of life in the short term may be positively affected.

Future considerations are the continuation of this study with perhaps greater emphasis being paid to the recruitment of patients with reproducibly similar symptomatologies. The diagnostic criteria available unfortunately still relies on a clinical diagnosis as the gold standard in assessing patients with CMD.

There is an increasing need to develop more objective tests, such as synovial fluid assays, or muscle biopsies, that may enable clinicians to rule out or rule in, the presence of synovitis mediating factors or muscle disorders in treating TMJ disorders.

An additional aspect that needs to be considered is the level of muscle activity that may be occuring during the use of these appliances. It would prove most interesting in the future to be able to monitor the changes in muscle activity during the treatment period. This would allow more precise correlation of the level of muscscular effort during function or parafunction with the observed symptoms at re-assessment sessions.

In addition, further studies could involve the monitoring of articular changes via the use of recording dental and condylar positions. We know little about condylar motion during function and even less during the treatment of temporomandibular joint disorders with oral orthopaedic appliances.

All future approaches should include the further development of hypotheses regarding the contribution of local mechanics to disordered function. These hypotheses could be tested with intra-oral appliances tailored to anatomical variations in individuals, and could be designed to achieve specific physical and behavioral effects in these patients.

BIBLIOGRAPHY

Agerberg G and Carlsson GE (1974). Late results of treatment of functional disorders of the masticatory system. J Oral Rehab 1:309-316.

Akerman S, Kopp S, Nilner M, Petersson A and Rohlin M (1988). Relationship between clinical and radiologic findings of the temporomandibular joint in rheumatoid arthritis. Oral Surg Oral Med Oral Pathol 66:639-643.

Anderson DJ (1956). Measurement of stress in mastication. J Dent Res 35:664-673.

Angle EG (1900). Treatment of malocclusion of the teeth and fractures of the maxillae: Angle's System 6th ed. Philadelphia SS. White Dental Manufacturing Co.

Atkinson HF and Sheperd RW (1967). Masticatory movements and the resulting force. Arch Oral Biol 12:195-202.

Baldioceda F, Pullinger AG and Bibb CA (1990). Relationship of condylar bone profiles and dental factors to articular soft-tissue thickness. J Craniomandib Disord Facial Oral Pain 4:71-79.

Basbaum AI and Levine JD (1991). The contribution of the nervous system to inflammation and inflammatory disease. Can J Physiol Pharmacol 69:647-651.

Bates RE, et al. (1984). The relationship between internal derangements of the temporomandibular joint and systemic joint laxity. J Am Dent Assoc 109:446.

Bell WE (1982). Clinical management of temporomandibular disorders. Chicago: Year Book Medical Publications.

Bell WE (1990). Temporomandibular Disorders. Classification, Diagnosis, Management. 3rd. ed. Chicago, Year Book Medical Publishers pp 166-176.

Belser UC and Hannam AG (1985). The influence of altered working side occlusal guidance on masticatory muscles and related jaw movement. J Prosthet Dent 53:406-413.

Bernick S (1962). The vascular and nerve supply to the temporomandibular joint of the rat. Oral Surg 15:488-498.

Bezuur NJ, Habets LLMH and Hansson TL (1988). The effect of therapeutic laser treatment in patients with craniomandibular disorders. J Craniomandib Disord Facial Oral Pain 2:83.

Binder SA (1981). Applications of low and high voltage electro-therapeutic currents. In Wolf WL (ed) Electrotherapy. NY Churchill Livingstone pp 1-25.

Black GV (1895). The force exerted in the closure of the jaws. Dental Cosmos 6:469-484.

Blasberg B, Hunter T and Phillips S (1991). Peripheral joint hypermobility in individuals with and without temporomandibular joint disorders (abs 98). J Dent Res 70 (Special Issue) pp 278.

Block LS (1947). Diagnosis and treatment of disturbances of the temporomandibular joint especially in relation to vertical dimension. JADA 34(2):253-260.

Boyd RL, Gibbs C, Mahan PE, Richmond AR and Laskin JL (1990). Temporomandibular joint forces measured at the condyle of Macaca Arctoides. Am J Orthod Dentofac Orthop 97:472-479.

Bradley GR (1989). The effect of splint therapy and orthodontic extrusion on TMJ symptoms. Thesis St Louis University, Missouri.

Braun BL, Di Giovanna A, Schiffman E, Bonnema J and Fricton J (1992). A cross-sectional study of temporomandibular joint dysfunction in post cervical trauma patients. J Craniomandib Disord Facial Oral Pain 6:24-31.

Brehnan K, Boyd RL, Laskin J, Gibbs C and Mahan P (1981). Direct measurement of loads at the temporomandibular joint in Macaca Arctoides. J Dent Res 60:1820-1824.

Brudevold F (1951). A basic study of the chewing forces of a denture wearer. J Am Dent Assoc 43:45-51.

Buckingham RB, Bruan T, Harinstein DA, Oral K, et al. (1991). Temporomandibular joint dysfunction syndrome: A close association with systemic joint laxity (the hypermobile joint syndrome). Oral Surg Oral Med Oral Patol 72:514-519.

Burgess JA, Sommers EE, Truelove EL and Dworkin SF (1988). Short-term effect of two therapeutic methods on myofascial pain and dysfunction of the masticatory system. J Prosthet Dent 60:606.

Burke D, Gaudevia SC, and Macefield G (1988). Responses to passive movement of receptors in joint, skin and muscle of the human hand. J Physiol (London) 402:347-361.

Byrd KE and Stein ST (1990). Effects of lesions to the trigeminal motor nucleus on temporomandibular disc morphology. J Oral Rehabil 17:529-540.

Capra NF (1987). Localization and central projections of primary afferent nerves that innervate the temporomandibular joint in cats. Somatosens Res 4:201-213.

Carlson CR, Okeson JP, Falace DA, Nitz AJ and Anderson D (1991). Stretch-based relaxation and the reduction of EMG activity among masticatory muscle pain patients. J Craniomandib Disord Facial Oral Pain 5:205-212.

Carlsson GE (1974). Bite force and chewing efficiency. In Kawamura Y (ed). Frontiers in Oral Physiology Vol 1 Basel, Karger, pp 265-292.

Carlsson GE (1985). Long-term effects of treatment of craniomandibular disorders. J Craniomand Pract 3:337-342.

Carraro JJ and Caffesse RG (1978). Effect of occlusal splints on TMJ symptomatology. J Prosthet Dent 40:563-566.

Celenza FU and Nasedkin JN (1978). Occlusion: the state of the art. Chicago, Quintessence.

Chun DS and Koskinen-Moffet M (1990). Distress, jaw habits, and connective tissue laxity as predisposing factors to TMJ sounds in adolescents. J Craniomandib Disord Facial Oral Pain 4:165.

Clark GT, Adachi NY and Dornan MR (1990). Physical medicine procedures affect temporomandibular disorders. A review. J Am Dent Assoc 121:151-161.

Clark GT (1984). A critical evaluation of orthopaedic appliance therapy: design, theory, and overall effectiveness. J Am Dent Assoc 108:359-364.

Clark GT and Adler RC (1985). A critical evaluation of occlusal therapy: Occlusal adjustment procedures. J Am Dent Assoc 110:743-750.

Clark GT, Lanham F and Flack VF (1988). Treatment outcome results for consecutive TMJ clinics patients. J Craniomandib Disord 2:87.

Clark GT, Beemsterboer PL and Rugh JD (1981). Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. J Oral Rehabil 8:279-286.

Clark GT, Seligman DA, Solberg WK and Pullinger AG (1989a). Guidelines for the examination and diagnosis of temporomandibular disorders. J Craniomand Disorders 3:7-14.

Clark GT (1991). Etiologic theory and prevention of temporomandibular disorders. Adv Dent Res 5:60-66.

Clark FJ, Grigg P and Chapin JW (1989). The contribution of articular receptors to proprioception with the fingers in humans. J Neurophysiol 61:186-193.

Costen JB (1934). A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol Rhinol Laryngol 43:1-15.

Curl DD (1993). Chiropractic management of capsulitis and synovitis of the temporomandibular joint. J Orofac Pain 7:283-293.

Danzig W and VanDyke AR (1983). Physical therapy as an adjunct to temporomandibular joint therapy. J Prosthet Dent 49:96-99.

De Laat A, Van Steenberghe D and Lesaffre E (1986). Occlusal relationships and TMJ dysfunction. Part II. Correlation between occlusal and articular parameters and symptoms of TMJ dysfunction by means of stepwise logistic regression. J Prosthet Dent 55:116-121.

Dubner R, Sessle BJ and Storey AT (1978). The neural basis of oral and facial function. Plenum Press, New York.

Duncan GH, Bushnell MC and Lavigne GJ (1989). Comparison of verbal and visual analogue scales for measuring the intensity and unpleasantness of experimental pain. Pain 37:295-303.

Dworkin SF (1992). Research Diagnostic Criteria for temporomandibular joint disorders. Final project report. Dworkin (ed). NIDR Epidemiology and Oral Disease Prevention Program.

Dworkin SF (1992). Research Diagnostic Criteria for Temporomandibular Disorders: Review, Classification, Examinations and Specifications, Critique. J Craniomand Disorders 6:301-355.

El-labben NG, Harris M, Hopper C and Barber P (1990). Degenerative changes seen in masseter and temporalis muscles in limited mouth opening and TMJ ankylosis. Oral Surg Oral Med Oral Pathol 19:423-425.

Erskine A, Morley S and Pearce S (1990). Memory of pain : a review. Pain 41:255-265.

Esposito CJ, Veal SJ and Farman AG (1984). Alleviation of myofascial pain with ultrasound therapy. J Prosthet Dent 51:106.

Eversole LR and Machado L (1985). Temporomandibular joint internal derangements and associated neuromuscular disorders. J Am Dent Assoc 110:69-79.

Farrar WB (1972). Differentiation of temporomandibular joint dysfunction to simplify treatment. J Prosthet Dent 28:629-636.

Faulkner KDB (1990). Bruxism: A review of the literature. Part I and II. Aust Dent J 35:266-276 and 355-361.

Faulkner MG, Hatcher DC and Hay A (1987). A three dimensional investigation of temporomandibular joint loading. J Biomechanics 10:997-1002

Franks AS (1965). Conservative treatment of temporomandibular joint dysfunction: a comparative study. Dent Pract 15:205-210.

Fricton JR (1991). Recent advances in temporomandibular disorders and orofacial pain. J Am Dent Assoc 122:25-32.

Fricton JR (1991). Clinical care for myofascial pain. Dent Clin North Am 35:1-28.

Gausch K and Kulmer S (1977). The role of retrodisclusion in the treatment of the TMJ patient. J Oral Rehab 4(1):29-32.

Gelb H (1977). Current management of head, neck and TMJ pain and dysfunction. Philadelphia, WB Saunders Co.

Gerber A (1971). Kiefer gelenk und zahnokklusion. Dtsch Zahnarztl Z 26:119.

Gerschman JA, Wright JL, Hall WD, Reade PC, Burrows GD and Holwill BJ (1987). Comparisons of psychological and social factors in patients with chronic orofacial pain and dental phobic disorders. Aust Dent J 32(5):331-335.

Goharian RK and Neff PA (1980). Effect of occlusal retainers on temporomandibular joint and facial pain. J Prosthet Dent 44:206.

Gold S, Lipton J, Marbach J and Gurion B (1975). Sites of psychophysiological complaints in MPD patients: II Areas remote from the orofacial region.(Abs 165) J Dent Res 54 (Special Issue).

Gold N et al. (1983). TENS therapy for treatment of MPD syndrome. (Abs 676). J Dent Res 62 (Special Issue):244.

Goldspink G (1976). The adaptation of muscle to a new functional length. In Anderson DJ and Mathews B (eds). Mastication. Bristol. John Wright and Sons Ltd.

Goodfriend DJ (1933). Symptomatology and treatment of abnormalities of the mandibular articulation. Dent Cosmos 75:844-852.

Goodman P, Greene CS and Laskin DM (1976). Response of patients with myofascial pain dysfunction to mock equilibration. J Am Dent Assoc 92:755-758.

Goto TK, Langenbach G, Korioth T, Hagiwara M, Tonndorf M and Hannam AG (1994). Functional movements of Human Jaw Muscle Attachments. (Abs) J Dent Res 73263 (Special Issue).

Gracely RH, Mc Grath P and Dubner R (1978). Ratio scales of sensory and affective verbal pain descriptors. Pain 5: pp 5-29.

Greene CS (1982). Orthodontics and the temporomandibular joint. Angle Orthod 52:166-172.

Greene CS and Laskin, DM (1972). Splint therapy for the myofascial pain dysfunction (MPD) syndrome: A comparative study. J Am Dent Assoc 84: 624-628.

Greene CS and Laskin DM (1974). Long term evaluation of conservative treatment for myofascial pain dysfunction syndrome. J Am Dent Assoc 89(6):1365-1368.

Greene CS and Laskin DM (1983). Long term evaluation of treatment for myofascial pain dysfunction syndrome. J Am Dent Assoc 107:235-238.

Gregg JM and Rugh JD (1988). Pharmacological therapy. In Mohl ND, Zarb GA, Carlsson GE, Rugh JD (eds). A textbook on occlusion. Chicago, Quintessence Publ Co. pp 351-375.

Guibaud G (1988). Peripheral and central electrophysiological mechanisms of joint and muscle pain.Edited by R.Dubner, GF Gebhart, and MR Bond. In Proceedings of the Vth World Congress on Pain. Elsevier, Amsterdam pp 201-215.

Guilbaud G (1991). Central neurophysiological processing of joint pain on the basis of studies in normal animals and in models of experimental arthritis. Can J Physiol Pharmacol 69:637-646.

Hackney PT, Bade D and Clawson A (1993). Relationship between forward head posture and diagnosed internal derangement of the temporomandibular joint. J Orofac Pain 7:386-390.

Hagberg C, Hellsing G and Hagberg M (1990). Perception of cutaneous electrical stimulation in patients with craniomandibular disorders. J Craniomandib Disord Facial Oral Pain 4:120-125.

Hagberg C (1987). Assessments of bite force: A review. J Craniomandib Disord Facial Oral Pain 1:162-169.

Hanamura H, Houston F, Hylander H, Carlsson GE, Haraldson T and Nyman S (1987). Periodontal status and bruxism: A comparative study of patients with periodontal disease and occlusal parafunctions. J Periodontol 58:173-176.

Hannam AG (1984). Optimum occlusal relationships are essential for craniomandibular harmony. Position Paper. In Klineberg I and Sessle B (eds). OroFacial Pain and Neuromuscular Dysfunction, Mechanisms and Clinical Correlates Advances in Biosciences. Oxford, Pergammon Press. pp 167-175.

Hansson P et al. (1986). Influence of naloxone on relief of acute oro-facial pain by transcutaneous electrical nerve stimulation (TENS) or vibration. Pain 24:323.

Hansson T and Nordstom B (1977). Thickness of the soft tissue layers and articular disk in temporomandibular joints with deviations in form. Acta Odontol Scand 35:281-288.

Hansson T and Oberg T (1977). Arthrosis and deviation in form in the temporomandibular joint. A macroscopic study on a human autopsy material. Acta Odontol Scand 35:167-174.

Haraldson T, Carlsson GE and Ingervall B (1979). Functional state, bite force and postural muscle activity in patients with osseointegrated oral implant bridges. Acta Odontol Scand 37:195-206.

Harkins SJ and Marteney JL (1985). Extensive trauma: A significant precipitating factor in temporomandibular joint dysfunction. J Prosthet Dent 54:271-272.

Heft MW, Gracely RH and Dubner R (1984). Nitrous oxide analgesia: a psychophysical evaluation using verbal descriptor scaling. J.Dent Res 63:129-132.

Helkimo E, Carlsson GE and Helkimo M (1977). Bite force and state of dentition. Acta Odontol Scand 35:297-303.

Helkimo M (1974). Studies on function and dysfunction of the masticatory system. III. Analysis of anamnestic and clinical recordings of dysfunction with the aid of indices. Swed Dent J 67:165-182.

Helkimo E and Ingervall B (1978). Bite force and functional state of the masticatory system in young men. Swed Dent J 2:167-175.

Helkimo E, Carlsson GE and Carmeli Y (1975). Bite force in patients with functional disturbaces of the masticatory system. J Oral Rehabil 2:397-406.

Hellsing D (1984). Functional adaptation to changes in vertical dimension. J Prosthet Dent 52:867.

Hellsing G (1980). On the regulation of bite force in man. J Oral Rehabil 7:403-411.

Hellsing G and Hagberg C (1990). Changes in maximum bite force related to extension of the head. Eur. J Orth 12:148-153.

Helm S and Petersen PE (1989). Mandibular dysfunction in adulthood in relation to morphologic malocclusion at adolescence. Acta Odontol Scand 47:307-314.

Hesse JR and Hansson TL (1988). Factors influencing joint mobility in general and in particular with respect of the craniomandibular articulation: a literature review. J Craniomand Disorders: Facial Oral Pain 2:19-28.

Hodges JM (1990). Managing temporomandibular joint syndrome. Laryngoscope 100:60-66.

Hohl TH and Tueck WH (1982). Measurement of condylar loading forces by instrumented prosthesis in the baboon. J Maxillofac Surg 10:1-7.

Howard JA (1990). Temporomandibular joint disorders, facial pain and dental problems of performing artists. In Sataloaff R, Brandfonbrener A, Lederman R, (eds.) Textbook of performing Arts Medicine, New York, Raven Press Ltd. pp 111-169.

Howard RP, Benedict JV, Raddin Jr. JH and Smith HL (1991). Assessing neck extension-flexion as a basis for temporomandibular joint dysfunction. J Oral Maxillofac Surg 49: 1210-1213.

Howell AH and Manly RS (1948). An electronic strain gauge for measuring oral forces. J Dent Res 27:705.

Huskisson EC (1974). Measurement of pain. The Lancet 2:1127-1131.

Hylander WL (1975). The human mandible: lever or link? Am J Phys Anthropol 43:227.

Hylander WL (1979). An experimental analysis of temporomandibular joint reaction force in macaques. Am J Phys Anthropol 51:433-456.

Hylander WL (1985). Mandibular function and temporomandibular joint loading. Craniofacial Growth Series # 16. Ann Arbor. Univ. of Michigan.

Israel HA (1989). Synovial fluid analysis. Oral Maxillofac Surg Clin North Am 1:85-92.

Ito T, Gibbs CH, Marguelles-Bonnet R, Lupkiewicz SM, Young HM, Lundeen HC and Mahan PE (1986). Loading on the temporomandibular joints with five occlusal conditions. J Prosthet Dent 56:478-484.

Jaffe A, Tal M and Ehrlich J (1991). Effect of occlusal bite-raising splint on electromyographic, motor unit histochemistry and myoneuronal dimension in rats. J Oral Rehabil 18:343

Jankelson B (1979). Neuromuscular aspects of occlusion. Dent Clin North Am 23:157-168.

Lieb MM (1977). Oral orthopaedic. In Gelb H, (ed). Clinical management of head, neck and TMJ pain and dysfunction. Philadelphia, WB. Saunders.

Johansson A, Wenneberg B, Wagersten C and Haraldson T (1991). Acupuncture in treatment of facial muscular pain. Acta Odontol Scand 49:153-158.

Katzberg R, Tallents R, Hayakawa K, Miller T, Goske M and Wood B (1985). Internal derangements of the temporomandibular joint: findings in the pediatric age group. Radiology 154:125-127.

Kawamura Y and Abe K (1974). Role of sensory information from temporomandibular joint. Bull Tokyo Med. Dent Univ Suppl 21:78-82 .

Kikuchi M, Korioth TWP and Hannam AG (1994). Bite force distribution during tooth clenching in man. (Abs) J Dent Res 73:446.

Kirveskari P and Alanen P (1985). Association between tooth loss and TMJ dysfunction. J Oral Rehabil 12:189-184.

Klaffenback AO (1936). Gnathodynamics. Journal of the American Dent Assoc 23:371-382.

Klineberg IJ (1971). Structure and function of temporomandibular joint innervation. Ann R Coll Surg Engl 49:649-661.

Koolstra JH, van Eijden TMGJ, Weijs WA and Naeije MA (1988). A three dimensional mathematical model of the human masticatory system predicting maximum possible bite forces. J Biomech 2:563-576.

Kopp S (1977). Clinical findings in temporomandibular joint osteoarthrosis. Scand J Dent Rest 85:434-443.

Kopp S, Wenneberg B and Clemensson E (1983). Clinical, microscopical and biochemical investigation of synovial fluid from temporomandibular joints. Scand J Dent Res 91:33.

Korioth TWP (1993). Phd.Thesis. University of British Columbia.

Korioth TWP and Hannam AG (1994). Deformation of the human mandible during simulated tooth clenching. J Dent Res 73(1):56-66.

Korioth WP and Hannam AG (1994). Three dimensional finite element modelling of human mandibular biomechanics. Part II: Dental and Condylar forces during clenching (In press).

Kremenak CR, Kinser DD, Melcher TJ, Wright GR and Harrison SD, Ziaja RR and Harman HA (1992). Orthodontics as a risk factor for temporomandibular disorders II. Am J Orthod Dentofac Orthop 101:21-27.

Kronn E (1993). The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. J Oral Facial Pain 2:209-213.

Langenbach GEJ, Ng WT and Hannam AG (1994). Dynamic modelling of the human masticatory system. Archs Oral Biol (In press).

Larsson J, Ekblom A, Heriksson K and Theodorsson E (1989). Immunoreactive tachikinins, calcitonin gene realted peptide and neuropeptide in human synovial fluid from inflammed knee joints. Neurosci Lett 100:326-330.

Laskin DM (1969). Etiology of the myofascial pain dysfunction syndrome. J Am Dent Assoc 79:148-153.

Laskin DM and Block L (1986). Diagnosis and treatment of myofascial pain dysfunction syndrome. J Prosthet Dent 56:76-84.

Laskin DM, Greenfield W, Gale E, Rugh J, Neff P, Alling C and Ayer WA (1983). The president's conference on the examination, diagnosis and management of temporomandibular disorders. Chicago American Dental Association.

Laurell L (1985). Occlusal forces and chewing ability in dentitions with cross-arch bridges. (Thesis). Swed Dent J (suppl 26).(From Hagberg 1987).

Leff A (1966). Gnathodynamics of four mandibular positions. J Prosthet Dent 16:844-847.

Lerman MD (1974). The hydrostatic appliance: a new approach to treatment of the TMJ pain-dysfunction syndrome. J Am Dent Assoc 89(6):1343-1350.

Levinson NA (1990). Psychologic facets of esthetic dental health care: a developmental perspective. J Prosthet Dent 64:486-491.

Linde C and Isacsson G (1990). Clinical signs in Patients with Disk Displacement Versus Patients with Myogenic Craniomandibular Disorders. J Craniomand Disorders: Facial & Oral Pain 4:197-204.

Lindeholm H and Wennstrom A (1970). Isometric bite force and its relation to general muscle force and body build. Acta Odontol Scand 28:679-689.

Lipowski ZJ (1988). Somatization: The concept and its clinical application. Am J Psychiatry 145:1358-1368.

Luder HU (1993). Articular degeneration and remodelling in human temporomandibular joints with normal and abnormal disc position. J Oral Fac Pain 4:391-403.

Lund JP and Lamarre Y (1973). The importance of positive feedback from periodontal pressoreceptors during voluntary isometric contraction of jaw closing muscles in man. Journal Biologie Buccale 1:345.

Lundh H and Westesson P-L (1989). Long term follow-up after occlusal treatment to correct abnormal temporomandibular joint disc position. Oral Surg Oral Med Oral Pathol 67:2.

Lund JP and Matthews B (1981). Response of temporomandibular joint afferents recorded in the gasserian ganglion of the rabbit to passive movements of the mandible. In Oral-facial sensory and motor functions. Ed. by Y Kawamura and R Dubner. Quintessence Tokyo. pp.153-160.

Lundquist LW, Carlsson GE and Hedegard B (1986). Changes in bite force and chewing efficiency after denture treatment in edentulous patients with denture adaptation difficulties. J Oral Rehabil 3:21-29.

MacDonald JWC (1982). Occlusal contacts and jaw muscle activity in parafunctional clenching. MSc. Thesis. University of British Columbia.

MacDonald JWC and Hannam AG (1984). Relationship between occlusal contacts and jaw closing muscle activity during tooth clenching: Part I. J Pros Dent 52:718-729.

MacDonald JWC and Hannam AG (1984). Relationship between occlusal contacts and jaw closing muscle activity during tooth clenching: Part II. J Pros Dent 52:862-867.

Magnusson T and Carlsson GE (1983). Occlusal adjustment in patients with residual or recurrent signs of mandibular dysfunction. J Prosthet Dent 49:706-710.

Magnusson T and Carlsson GE (1980). Treatment of patients with functional disturbances in the masticatory system. A survey of 80 consecutive patients. Swed Dent J 4:145-153.

Manco LG and Messing SG (1986). Splint therapy evaluation with direct sagittal computed tomography. Oral Surg 61:5.

Manns A, Miralles R and Guerrero F (1981). Changes in electrical activity of the postural muscles of the mandible upon varying the vertical dimension. J Prosthet Dent 45: 438-445.

Manns A, Valdivia J,Mirralles R and Pena MC (1988). The effect of different occlusal splints on the electromyographic activity of elevator muscles. J Gnathol 7:61-73.

Manns A, Miralles R, Santander H and Valdivia J (1983). Influence of the vertical dimension in the treatment of myofascial pain-dysfunction syndrome. J Prosthet Dent 50:700-709.

Manns A, Chan C and Miralles R (1987). Influence of group function and canine guidance on electromyographic activity of elevator muscles. J Prosthet Dent 57:494-501.

Manns A, Miralles R and Palazzi C (1979). EMG, bite force and elongation of the massester muscle under isometric voluntary contraction and variations of vertical dimension. J Prosthet Dent 42:674-682.

Manns A, Miralles R, Valdivia J and Bull R (1989). Influence of variation in anteroposterior occlusal contacts on electromyographic activity. J Prosth Dent 61:617-623.

Mapp PI, Kidd BL, Terry JM, Revell PA, Ibrahim NBN and Blake DR (1990). Substance P-Calcitonin gene realted peptide and C-Flanking peptide of neuropeptide Y-immunoreactive fibers are present in normal synovium but depleted in patients with rheumatoid arthritis. Neurosci 37:143-153.

Marbach JJ, Raphael KG, Dohrenwend BP and Lennon MC (1990). The validity of tooth grinding measures: Etiology of pain dysfunction syndrome revisited. J Am Dent Assoc 120:327-333.

McCarty WL and Farrar WB (1979). Surgery for internal derangements of the temporomandibular joint. J Prosthet Dent 42:191-196.

McCreary CP, Clark GT, Merril RL, Flack V and Oakley ME (1991). Psychological distress and diagnostic subgroups of temporomandibular disorder patients. Pain 44:29-34.

McNamara JA and Carlson DS (1979). Quantitative analysis of temporomandibular joint adaptations to protrusive function. Am J Orthod 76:593-611.

McNeill C (1983). Craniomandibular (TMJ) disorders-state of the art. Part II: Accepted diagnosis and treatment and modalities J Prosthet Dent 49:393-397.

McNeill C (1991). Temporomandibular disorders: Guidelines for the diagnosis and management. J Calif Dent Assoc 19:15-26.

Melzack R (1975). The McGill pain questionnaire: major properties and scoring methods. Pain pp 277-299.

Melzack R and Casey KL (1968). Sensory, motivational and central control determinants of pain: a new conceptual model. In Kenshalo DR (ed), The Skin Senses. Thomas, Springfield, IL. pp 423-443.

Menetrey D, Chaouch A and Besson JM (1980). Location and properties of dorasal horn neurones at origin of spinoreticular tract in lumbar enlargement of the rat. J. Neurophysiol 44:862-877.

Mense S (1986). Slowly conducting afferent fibers from deep tissues: neurobiological properties and central nervous actions. Prog Sens Physiol 6:140-219.

Miller AJ (1991). Craniomandibular Muscles: Their role in function and form. CRC Press Inc. Boca Raton, Florida.

Mills DK, Fiandaca DJ and Scapino RP (1994). Morphologic, microscopic, and immunohistochemical investigations into the function of the primate TMJ disc. J Orofac Pain 8:136-154.

Minagi S, Nozaki S, Sato T and Tsuru H (1991). A manipulation technique for treatment of anterior disk displacement without reduction. J Prosthet Dent 65:686-691.

Minagi S, Watanabe H, Sato T and Tsuru H (1990). The relationship between balancing-side occlusal contact patterns and temporomandibular joint sounds in humans: Proposition of the concept of balancing-side protection. J Craniomandib Dis Fac Oral Pain 4:251-256.

Moffett BC, Johnson LC, McCabe JB and Askew HC (1964). Articular remodelling in the adult human temporomandibular joint. Am J Anat 115:119-142.

Mohl ND (1988). The temporomandibular joint. In Mohl, Zarb, Carlsson and Rugh. (eds). A textbookof Occlusion. Chicago, London, Berlin, Sao Paulo, Tokyo and Hong Kong. Quintessence Publishing Co. Inc.

Molin C (1972). Vertical isometric muscle forces of the mandible. A comparative study of subjects with and without manifest mandibular pain dysfunction syndrome. Acta Odontol Scand 30:485-499.

Mongini F (1980). Condylar remodelling after occlusal therapy. J Prosthet Dent 43:568-577.

Morani V, Previgliano V, Schierano GM and Ramieri G (1994). Innervation of the human temporomandibular joint capsule and disc as revealed by immunohistochemistry for neurospecific markers. J Orofac Pain 1: 36-41.

Moses JJ and Topper DC (1991). A functional approach to the treatment of temporomandibular joint internal derangement. J Craniomand Dis Fac Oral Pain 5:19-27.

Moystad A, Krogstad BS and Larheim TA (1990). Transcutaneous nerve stimulation in a group of patients with rheumatic disease involving the temporomandibular joint. J Prosthet Dent 64:596-600.

Murphy GJ (1983). Electrical physical therapy in treating TMJ patients. J Craniomandibular Pract 1:68.

Nelson GJ (1986). Three dimensional computer modelling of human biomechanics. MSc. Thesis. The University of British Columbia.

Nelson SJ and Ash MM (1988). An evaluation of a moist heat pad for the treatment of TMJ/Muscle pain dysfunction. J Craniomandibular Pract 6:355.

Nickerson JW and Boering G (1989). Natural course of osteoarthrosis as it related to internal derangement of the temporomandibular joint. Oral Maxillofac Surg clin North Am 1:27-45.

Nitzan DW, Dolwick MF and Heft MW (1990). Arthroscopic lavage and lysis of the temporomandibular joint. A change in perspective J Oral Maxillofac Surg 48:798-801.

Oberg T, Carlsson GE and Fajers CM (1971). The human temporomandibular joint. A morphologic study on a human autopsy material. Acta Odontol Scand 29:349-384.

Okeson JP (1993). Management of Temporomandibular Disorders and Occlusion. 3rd. Edn. Mosby Year Book Co. St. Louis, Mo.

Okeson JP, Kemper JT and Moody PM (1982). A study of the use of occlusion splints in the treatment of acute and chronic patients with craniomandibular disorders. J Prosthet Dent 48:708-712.

Okeson JP (1988). Long term treatment of disc -interference disorders of the TMJ with anterior repositioning occlusal splints. J Prosthet Dent 60:611.

Okeson JP and Hayes DK (1986). Long term results of treatment for temporomandibular disorders: An evaluation by patients. J Am Dent Assoc 112:473-478.

Okeson JP (1987). The effects of hard and soft occlusal splints on nocturnal bruxism. J Am Dent Assoc 114:788.

Olson GB, Peters CJ and Franger AL (1988). The incidence and severity of premenstrual syndrome among female craniomandibular pain patients. J Craniomandib Pract 6:330.

Osborn JW (1985). The disc of the human temporomandibular joint: design, function and failure. J Oral Rehab 12:279-293.

Osborn JW (1989). The temporomandibular ligament and the articular eminence as constraints during jaw opening. J Oral Rehab 16:323-333.

Parker MW (1990). A dynamic model of etiology in temporomandibular disorders. J Am Dent Assoc 120:283-289.

Pavone B (1985). Bruxism and its effects on natural teeth. J Prosthet Dent 53:692-696.

Pierce CJ and Gale EN (1988). A comparison of different treatments for nocturnal bruxism. J Dent Res 67:597-601.

Plasmans PJJM, Kuipers L, Vollenbrock HR and Vrijhoef MMA (1988). The occlusal status of molars. J Prosthet Dent 60:500-503.

Posselt U (1968). Physiology of occlusion and rehabilitation, 2ed. Philadelphia, F.A. Davis Co.

Posselt U and Wolff IB (1963). Treatment of bruxism by bite guards and bite plates. J Can Dent Assoc 29:773-778.

Clark GT (1984). A critical evaluation of orthopaedic interocclusal appliance therapy: Design, theory, and overall effectiveness. J Am Dent Assoc 108:359-364.

Price DD, McGrath PA, Rafii A and Buckingham B (1983). The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. Pain 17:45-56.

Proffit WR, Fields HW and Nixon WL (1983). Occlusal forces in normal and long-face adults. J Dent Res 62:566.

Pullinger AG and Seligman DA (1991). Trauma history in diagnostic groups of temporomandibular disorders. Oral Surg Oral Med Oral Pathol 71:529-534.

Pullinger Ag, Baldioceda F and Bibb CA (1990). Relationship of TMJ articular soft tissue to underlying bone in young adult condyles. J Dent Res 69:1512-1518.

Pullinger AG, Montiero AA and Lui S (1985). Etiological factors associated with temporomandibular disorders. J Dent Res 64 (Spec Iss):269, Abs# 848.

Quinn J (1989). Pathogenesis of TMJ chondromalacia and arthralgia. Oral Maxillofac Surg Clin North Am 7:57.

Quinn JH and Bazan NG (1990). Identification of prostaglandin E2 and leukotriene B4 in the synovial fluid of painful, dysfunctional temporomandibular joints. J Oral Maxillofac Surg 48:968-971.

Ramfjord SP and Ash MM (1983). Occlusion. 3rd ed. Philadelphia, WB Saunders Co, pp 481-509.

Ramfjord SP and Ash MM (1971). Occlusion 2ed. Philadelphia, W.B. Saunders Co.

Randolph CS, Greene CS, Moretti R, Forbes D and Perry HT (1990). Conservative management of temporomandibular disorders: A post treatment comparison between patients from a university clinic and from private practice. Am J Orthod Dentofac Orthop 98:77-82.

Raustia AM and Pohjola RT (1986). Acupuncture compared to stomatognathic treatment for TMJ dysfunction III. Effect of treatment on mobility J Prosthet Dent 56:616.

Ready LB and Hare B (1979). Drug problems in chronic pain patients. Anaesthesiol Rev 6:28-31.

Rindquist M (1973). Isometric bite force and its relation to dimensions of the facial skeleton. Acta Odontol Scand 31:35-42.

Rivera-Morales WC and Mohl ND (1991). Relationship of occlusal vertical dimension to health of the masticatory system. J Prosthet Dent 65:547-553.

Roth TE, Goldberg JS and Behrents RG (1984). Synovial fluid pressure determination in the temporomandibular joint. Oral Surg 57:583-588.

Rubinoff MS, Gross A and Mccall WD (1987). Conventional and nonoccluding splint therapy compared for patients with myofascial pain dysfunction syndrome. Gen Dent Nov/Dec.

Rugh JD and Harlan J (1988). Nocturnal bruxism and temporomandibular disorders. Adv Neurol 49:329-341.

Rugh, JD and Solberg, WK (1972). The measurement of human oral forces. Behav Res Meth and Instru Vol 4:125-128.

Rugh JD and Johnson RW (1981). Temporal analysis of nocturnal bruxism during EMG feedback. J Periodontol 52:263-265.

Rugh JD and Solberg WK (1976). Psychological implications in temporomandibular pain and dysfunction. Oral Sci Rev 7:3-30.

Rugh JD and Solberg WK (1975). EMG studies of bruxist behaviour before and during treatment. J Cal Dent Assoc 3:56.

Rugh JD and Robins W (1981). Oral habit disorders. In Ingersoll B (ed). Behavioral aspects in dentistry. New York, Appleton-Century Crofts, Chap.10 pp 179-202.

Rugh JD and Drago CJ (1981). Vertical dimension: a study of clinical rest position and jaw muscle activity. J Prosthet Dent 45:670-675.

Rugh JD (1988). Behavioral therapy. In Mohl ND, Zarb GA, Carlsson GE and Rugh JD (eds). A Textbook in Occlusion, Chicago. Quintessence Publ. Co. pp 329-338.

Scapino RP (1991). The posterior attachment: its structure, function and appearance in TMJ imaging studies. Part I. J Craniomand Disorders: Facial Oral Pain 5:83-95.

Schaible HG and Schmidt RFT (1988). Time course of mechanosensitivity changes in articular afferents during a developing experimental arthriits. J Neurophysiol 60:2180-2195.

Schaible HG, Schmidt RF and Willis WD (1987a). Convergent inputs from articular, cutaneous and muscle receptors onto ascending tract cells in the cat spinal cord. Exp Brain Res 66:479-488.

Scharer P and Stallard E (1966). The effect of occlusal interference on the tooth contact occurrences during mastication. Helv Odont Acta 10:49-56.

Scharer P (1974). In Kawamura Y (ed). Frontiers of Oral Biology. Basel, S Karger. pp 293-322.

Scott IS and Ash MM (1970). Micro miniature intraoral telemetry system. Proceedings of the International Telemetering Conference (From Rugh and Solberg 1972).

Seligman DA and Pullinger AG (1991). The role of functional occlusal relationships in temporomandibular joint disorders: A Review. J Craniomandib Disord Facial Oral Pain 5:265-279.

Seligman DA and Pullinger AG (1991). The role of intercuspal relationships in temporomandibular disorders: A Review: J Craniomandid Disord Facial Oral Pain 5:96-106.

Seligman DA and Pullinger AG (1989). Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord Facial Oral Pain 3:227-236.

Sessle BJ and Wu JW (1991). Mechanisms of pain arising from articular tissues. Can J Physiol Pharmacol 69:617-626.

Sessle BJ (1987). The Neurobiology of Facial and Dental Pain: Present Knowledge, Future Directions. J Dent Res 66(5): 962-981.

Sheikholeslam A, Holmgren K and Riise C (1986). A clinical and electromyographic study of the long term effects of an occlusal splint on the temporal and masseter muscles in patients with functional disorders and nocturnal bruxism. J Oral Rehabil 13:137-145.

Shupe RJ, Mohamed SE, Christensen LV, Finger IM and Weinberg R (1984). Effects of occlusal guidance on jaw muscle activity. J Prosthet Dent 51:811-818.

Solberg WK and Rugh JD (1972). The use of biofeedback devices in the treatment of bruxism. South Calif Dent Assoc J 40:852-853.

Solberg WK, Clark GT and Rugh JD (1975). Nocturnal electromyographic evaluation of bruxism patients undergioing short term splint therapy. J Oral Rehabil 2:215-223.

Spilker B (1991). Guide to Clinical Trials. Raven Press Ltd.

Stegenga B, Broekhuijsen ML, De Bont LG and Van Willigen JD (1992). Bite force endurance in patients with temporomandibular joint osteoarthrosis and internal derangements. J Oral Rehab 19:639-647.

Stegenga B, de Bont LGM and Boering G (1993). Temporomandibular joint pain assessment. J Orofac Pain 7:23-37.

Stevens SS (1975). Psychophysics: Introduction to its perceptual, neural and social prospects. Wiley, New York.

Stockstill JW and Callahan CD (1991). Personality hardiness, anxiety and depression as constructs of interest in the study of temporomandibular disorders. J Craniomandib Disord: Facial Oral Pain 5:129-134.

Suvinen T and Reade P (1989). Prognostic features of value in the management of temporomandibular joint pain-dysfunction syndrome by occlusal splint therapy. J Prosthet Dent 61:355-361.

Tallents CR et al. (1986). Clinical and arthographic evaluation of temporomandibular joint sounds. Oral Surg 62:373.

Taylor RC and Way WL (1968). Temporomandibular joint problems in relation to the administration of general anaesthesia. Oral Surg 26:327-329.

Teenier TJ and Throckmorton GS (1991). Effects of local anesthesia on bite force generation and electromyographic activity. J Oral Maxillofac Surg 49(4):360-365.

Tegelberg A and Kopp S (1997). Clinical findings in the stomatognathic system for individuals with rheumatoid arthritis and osteoarthritis. Acta Odontol Scand 45:65-75.

Thilander B (1961). Innervation of the temporomandibular joint capsule in man. An anatomic investigation and a neurophysiologic study of the perception of mandibular position. Trans R Sch Dent 1.

Throckmorton GS, Finn RA and Bell WH (1980). Biomechanics of differences in lower facial height. Am J Orthod Dentofac Orthoped 77:410.

Toller PA (1961). The synovial apparatus and temporomandibular joint function. Br Dent J 111:355-362.

Travell JG and Simons DG (1983). Myofascial pain and dysfunction. The trigger point manual. Baltimore. The Williams and Wilkins Co. pp63-74.

Travell J (1952). Ethyl chloride spray for painful muscle spasms. Archs Phys Med 33:291.

Tsuga K, Akagawa Y, Sakaguchi R and Tsuru H (1989). A short term evaluation of the effectiveness of stabilization-type splint therapy for specific symptoms of temporomandibular joint dysfunction syndrome. J Prosthet Dent 61:610-613.

VanDyke A and Goldman SM (1990). Manual reduction of displaced disc. Cranio 8:350-352.

Ward DM, Behrents RG, Goldberg JS (1990). Temporomandibular synovial fluid pressure response to altered mandibular positions. Am J Dentofac Orthop 98:22-28.

Weinberg S and Lapointe H (1987). Cervical Extension-Flexion Injury (Whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 45:653-656.

Travell JG and Simons DG (1983). Myofascial Pain and Dysfunction: The trigger point manual. Baltimore, Williams and Wilkins. pp 63-158.

Weinberg LA (1979). Role of condylar position in TMJ dysfunction-pain syndrome. J Prosthet Dent 41(6):636-643.

Weinberg LA (1980). The etiology, diagnosis and treatment of TMJ dysfunction-pain syndrome: Part II. Differential diagnosis. J Prosthet Dent 43:58-70.

Weinberg LA (1980). The etiology, diagnosis and treatment of TMJ dysfunction-pain syndrome: Part III. Treatment J Prosthet Dent 43:186-196.

Wenneberg B, Nystrom T and Carlsson GE (1988). Occlusal equilibration and other stomatognathic treatments in patients with mandibular dysfunction and headache. J Prosthet Dent 59:478-483.

Wessberg GA et al. (1981). Transcutaneous electrical stimulation as an adjunct in the management of myofascial pain dysfunction syndrome. J Prosthet Dent 45:307.

Westling L (1989). Craniomandibular disorders and general joint mobility. Acta Odontol Scand 47:293.

Westling L (1992). Temporomandibular joint dysfunction and systemic joint laxity. Swed Dent J (suppl 81).

Westling L, Carlsson GE and Helkimo M (1990). Background factors in craniomandibular disorders with special reference to general joint hypermobility, parafunction and trauma. J Craniomandib Disord: Facial Oral Pain 4:89-98.

Whittaker DK, Jones JW, Edwards PW and Molleson T (1990). Studies on the temporomandibular joints of an eighteenth century London population (Spitalfields). J Oral Rehabil 14:35-42.

Widmer CG (1992). (ed). Examination and History Data Collection. Part III. J Craniomand Disorders 6:335-345.

Wilding RJC and Owen CP (1987). The prevalence of temporomandibular joint dysfunction in edentulous non-denture wearing individuals. J Oral Rehabil 14:175-182.

Wilkinson T, Hansson TL, McNeill C and Marcel T (1992). A comparison of the success of 24-hour occlusal splint therapy versus nocturnal occlusal splint therapy in reducing craniomandibular disorders. J Craniomandib Disord: Facial Oral Pain 6:64.

Williams PL and Warwick R (1980). Gray's Anatomy, 36th Edition. Churchill Livingstone, Edinburgh.

Williams WN, Levin BDS and LaPointe LL (1985). Bite force discrimination by individuals with complete dentures. J Prosthet Dent 54:146-150.

Williamson EH and Lundquist DO (1983). Anterior Guidance: Its effect on electromyographic activity of the temporal and masseter muscles. J Pros Dent 49:816-823.

Wink CS, St Onge M and Zimny ML (1992). Neural elements in the human temporomandibular articular disc. J Oral Maxillofac Surg 50:334-337.

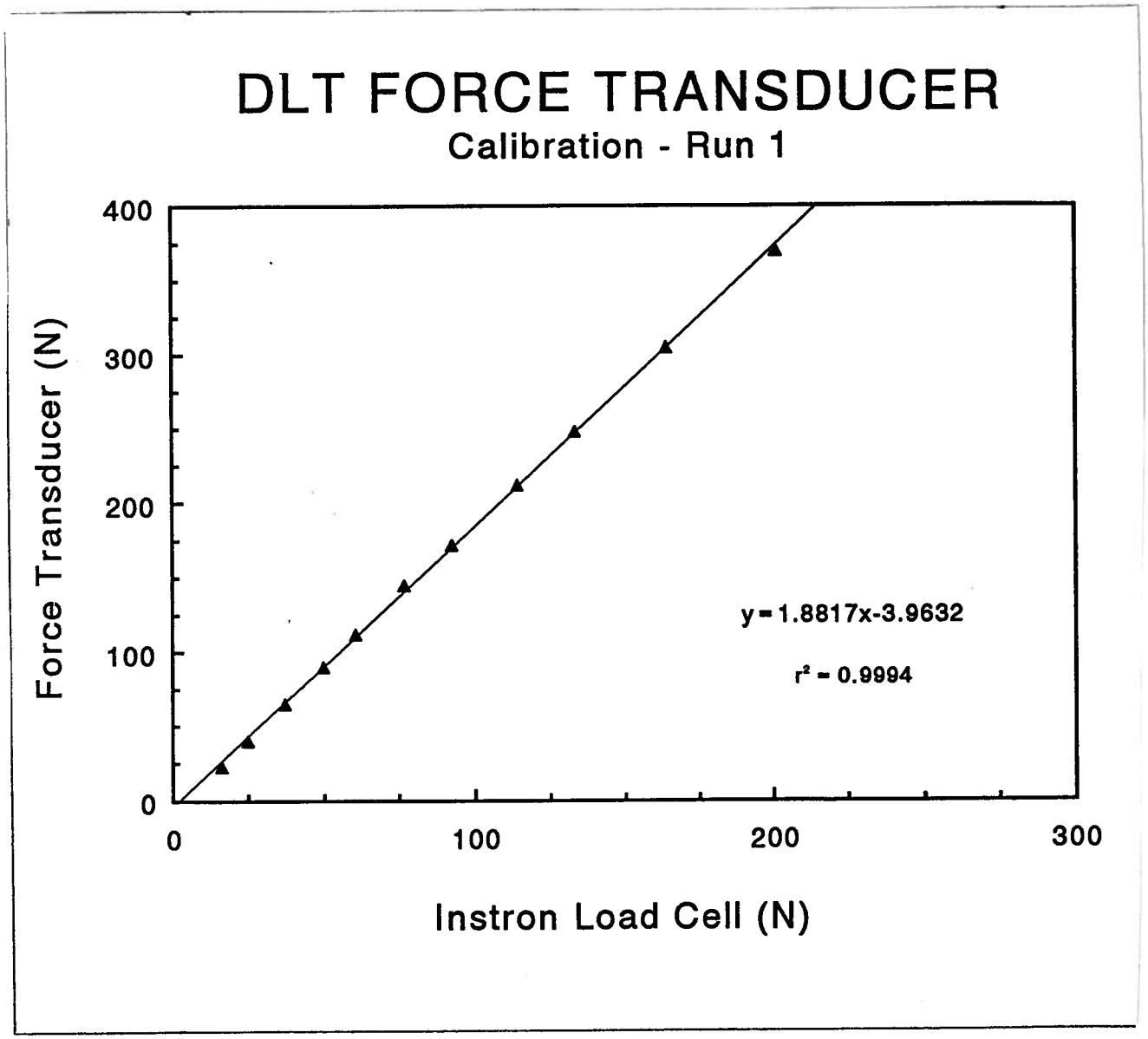
Wood WW and Tobias DL (1984). EMG response to alteration of tooth contacts on occlusal splints during maximal clenching. J Prosthet Dent 51:394-396.

Yurkstas A and Curby WA (1953). Force analysis of prosthetic appliances during function. J Prosthet Dent 3:82-87.

Zarb GA and Thompson GW (1975). The treatment of patients with temporomandibular joint pain dysfunction syndrome. J Canad Dent Assoc 41:410-417.

Appendix 1 Force transducer calibration.

Force measured by the force transducer (in N) plotted against the force applied by an Instron Load Cell. The equation shows the relationship between both axes.



Appendix 2 Questionnaire used to measure pain levels during all four sessions.

VAS QUESTIONNAIRE

Rate the intensity of the USUAL PAIN during the LAST WEEK by placing a (/) somewhere on the line below.

|----------|

No pain — Most intense pain imaginable.

Rate the intensity of the PAIN at present.

|----------|

No pain — Most intense pain imaginable.

Rate the intensity of the PAIN on the RIGHT JOINT while clenching on the RIGHT SIDE.

|----------|

No pain — Most intense pain imaginable.

Rate the intensity of the PAIN on the LEFT JOINT while clenching on the RIGHT SIDE.

|----------|

No pain — Most intense pain imaginable.

Rate the intensity of the PAIN on the RIGHT JOINT while clenching on the LEFT SIDE.

|----------|

No pain — Most intense pain imaginable.

Rate the intensity of the PAIN on the LEFT JOINT while clenching on the LEFT SIDE.

|----------|

No pain — Most intense pain imaginable.

Appendix 3 Research diagnostic criteria for TMD disorders (Axis I)

I. MUSCLE DIAGNOSES

a. Myofascial pain
b. Myofascial pain with limited opening

II. DISC DISPLACEMENTS

a. Disc displacement with reduction
b. Disc displacement without reduction, with limited opening
c. Disc displacement without reduction, without limited opening

III. ARTHRALGIA, ARTHRITIS, ARTHROSIS

a. Arthralgia
b. Osteoarthritis of the TMJ
c. Osteoarthrosis of the TMJ

From: Dworkin (1992)
RDC (Ed) L. LeResche et al.