NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, tests publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30.

THE UNIVERSITY OF ALBERTA

THE ABILITY OF OCCLUSAL SPLINT THERAPY TO INCREASE TEMPOROMANDIBULAR JOINT SPACE

BY

PAUL WILLIAM MAJOR

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

MASTER OF SCIENCE

IN

CLINICAL SCIENCES

FACULTY OF DENTISTRY

EDMONTON, ALBERTA

SPRING 1988

ISBN 0-315-42816-3

THE UNIVERSITY OF ALBERTA

RELEASE FORM

Paul William Major

The Ability of Occlusal Splint Therapy to Increase Temporomandibular Joint Space

Master of Science in Clinical Sciences

1988

Permission is hereby granted to THE UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific purposes only.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

Box 149,
Edmonton, Alberta
T0B 0E0

April 12, 1988

The University of Alberta

Faculty of Graduate Studies and Research

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled: "The Ability of Occlusal Splint Therapy to Increase Temporomandibular Joint Space" submitted by Paul William Major in partial fulfilment of the requirements for the degree of Master of Science in Clinical Sciences (Orthodontics).

Supervisor

External Examiner

Date: April 12th 1988

ABSTRACT

Condylar displacement (increased joint space) using three different forms of occlusal splint therapy for an 8 week period was evaluated. Thirty two subjects with a clinicoradiographic diagnosis of disc displacement without reduction were sequentially divided into three groups. The first group received a splint with total tooth contact and anterior guidance (CO splint), while the second and third group each received a pivotal splint constructed with a 1 mm shim between the condylar and articular portions of the articulator. The third group also had orthodontic traction to mechanically displace the condyles. Changes in condylar position were determined using standardized serial lateral transcranial radiographs and articulator determined condylar displacement recordings. Subjectively ranked pain level at the beginning and end of the study period was recorded for each patient.

Initial placement of all three splint types produced significant condylar displacement. The pivotal splints with orthodontic traction produced significantly larger posterior and inferior condylar displacement over the study period of which the inferior displacement persisted after splint removal. There was a very significant correlation between the reduction in subjective pain level and the amount of inferior condylar displacement.

ACKNOWLEDGEMENTS

Many people have freely given their time and support in the completion of this project. I would like to acknowledge the following people:

Each member of my thesis committee (Dr. K. Glover, Dr. D. Hatcher, Dr. K. Compton, Dr. R. Holland) for their suggestions and supervision in the preparation of this thesis.

A special thanks to Dr. K. Glover for his thoughtful suggestions, encouragement and friendship throughout my graduate training.

To Dr. D. Hatcher for providing the enthusiasm and motivation for me to pursue academic goals.

To Ms. Carol Gervais and Mrs. Brigitte Klemp for so willingly providing their clinic assistance.

To Mrs. Maureen Dmytrash for her many hours of typing.

To Dr. D. Scott for his knowledge and many hours of valuable assistance in completing the statistical component of this project.

To Mr. D. Hone for willingly sharing his time and expertise in constructing the occlusal splints used in this study

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

One of the treatment objectives in orthodontic therapy is to optimize the comfort, efficiency, and longevity of the dentition, supporting structures, and the temporomandibular joints.[1] The diagnosis and management of temporomandibular joint dysfunction are common problems facing the practicing orthodontist. A recent survey has shown that 33% of adolescents presenting for routine orthodontic care already have incipient symptoms of temporomandibular joint dysfunction.[2] Our understanding of the pathogenesis of temporomandibular joint dysfunction has improved substantially in recent years. This information can be utilized to assist in the differential diagnosis of this complex problem. Unfortunately a review of current literature reveals a lack of scientific information to permit comparison of different forms of therapy. Various treatment modalities should be designed to meet specific treatment objectives based on effective differential diagnosis and current principles of pathogenesis. Systematic scientific investigation into the effectiveness of different treatment modalities is needed.

a. STATEMENT of the PROBLEM:

The term degenerative joint disease is used interchangeably with the term osteoarthritis, and is defined as "the pathologic destruction of articular tissue and bone that occurs whenever articular remodeling does

A review of medical literature suggests that degenerative joint disease is the result of an imbalance between the stress applied to the joint and its ability to tolerate and adapt to that stress. Since, under normal conditions the tissues of the joint can adapt to joint loading, then degenerative joint disease should be viewed as a failed relationship between mechanical and biologic factors. It occurs in instances where the physical loading imposed upon the joint is increased because of altered anatomy or function, or in situations where the tissues adaptive ability and resistance to stress is lowered.

The most common internal derangement of the temporomandibular joint involves a stretching of the fibrous attachment of the disc to the capsule, allowing the disc to become displaced from its normal position in relation to the condyle. Disc displacements may produce a tissue response in a continuum from remodeling (adaptation) to degenerative joint disease (failure of adaptation). All the available evidence suggests that the avascular disc has no capacity for remodeling and once displaced without reduction will undergo permanent deformation with little chance of repositioning. Signs of degenerative joint disease such as crepitation, non-adaptive osseous changes and perforation of the posterior attachment are found primarily in patients with non-reducing disc displacements. In the progression of

internal derangement to degenerative joint disease, the morphology as well as the position of the disc is important.

The treatment of degenerative joint disease of the temporomandibular joint is aimed at arresting the degenerative process, reducing discomfort and establishing optimum joint function. Since disc displacement without reduction which precedes degenerative joint disease results in permanent non-reversible soft tissue changes, it is generally accepted normal joint anatomy cannot be restored. Joint loading is intimately involved in degenerative joint disease, and therefore reduction in loading forces is a reasonable objective in patients with non reducing disc displacement where the adaptive ability and resistance to stress is lowered.

Splint therapy to "offload" the temporomandibular joint can be designed to work in two ways. The first splint type is constructed to increase the vertical dimension of occlusion and provide an even contacting, non-interfering occlusion. This will passively reduce joint loading by reducing muscle activity. The second splint type is designed to physically separate the joint components to reduce, if not eliminate, compressive forces. An increase in joint space between the condyle and the articular eminence would potentially allow for reduction of the degenerative process. If the occlusal pattern of the splint is altered so that joint space must

be increased in order to achieve maximum intercuspation, it may be possible to utilize proprioceptive feed back with splint therapy to physically separate the joint space. Alternatively, interarch mechanical traction in combination with appropriate splint design should create an increase in joint space.

b. PURPOSE of the STUDY:

The objective of this study is to evaluate the clinical effectiveness of splint therapy to increase temporomandibular joint space. This will be achieved by analyzing radiographic joint space measurement, changes in condyle position during the course of splint therapy, and articulator determined condyle displacement recordings. Joint space measurements will be correlated with condyle displacement recordings for three different splint techniques.

c. RESEARCH QUESTIONS:

i. Is condyle position significantly altered by initial splint placement?

ii. Is there a significant difference in condyle position during the study period between the three groups of patients treated with different splints?

iii. Do changes in condylar position remain following removal of the splint?

iv. Is there a correlation between radiographically determined and articulator determined values for condylar displacement?

v. Is there a significant difference in joint space during the study period between the groups of patients treated with different splints?

vi. Is there a reduction in pain level associated with the different forms of splint therapy?

vii. Is there a correlation between the subjective pain level changes and the amount of condylar displacement?

d. HYPOTHESIS:

i. H_O! The condyle position is not significantly altered by initial splint placement.

H_A! The condyle position will be significantly altered by initial splint placement.

ii. H_O! There will be no statistical difference in condyle displacement during the study period between the three groups.

H_A! There will be a statistical difference in condyle displacement between the three groups.

iii. H_O! The condyle position following splint therapy is not different than prior to splint therapy.

H_A! The condyle position will be different following splint therapy.

iv. H_O! There is no significant correlation between radiographically determined and articulator determined values for condylar displacement.

H_A! There is a significant correlation between radiographically determined and articulator determined values for condylar displacement.

v. H_O! There is no significant change in joint space during the study period between the three groups.

H_A! There is a significant change in joint space during the study period between the three groups.

vi. H_O! There is no difference in pain level change between the three groups.

H_A! There is a significant difference in pain level change between the three groups.

vii. H_O! There is no significant correlation between the subjective pain level change and the amount of condylar displacement.

H_A! There is a significant difference between the subjective pain level change and the amount of condylar displacement.

II. LITERATURE REVIEW:

a. Normal Anatomy and Function of the Temporomandibular Joint

The temporomandibular joint is classified as a synovial compound joint.[5] and was first described in detail by Rees in 1954. Interposed between the articular surface of the condyle and the articular eminence of the temporal bone is the articular disc. This disc separates the joint into a superior joint component which forms a freely movable sliding joint and an inferior joint compartment which forms a pure rotational joint. The fully developed and normally functioning disc has a characteristic biconcave shape as seen on sagittal section. The thin central portion of the disc is firm and devoid of blood vessels, nerves and synovial tissues, reflecting its adaptation to compressive stress. The disc is thickened anteriorly and posteriorly into what Rees termed the anterior and posterior band. These thickened areas represent those peripheral parts of the disc not subjected to the pressure which in the early life of the individual, flattened the central articular zone.[6] This disc contour does not interfere with free rotary movement between the disc and condyle, yet it effectively resists anterior or posterior displacement of the disc from the condyle during translatory movements.[7]

The nonvascularized articular surfaces of the temporomandibular joint and clavicle are composed of dense

fibrous tissue rather than hyaline cartilage as in other synovial joints.[8] This dense fibrous articular tissue best withstands shearing forces, whereas hyaline cartilage found in other joints best withstands compressive forces.[9] Compression implies an axial loading perpendicular to the joint surface. Condyle movement coincident with loading in combination with incongruity of the opposing joint surfaces dictates that most of the forces are non axial.

The disc attachments merge with the articular capsule around the periphery of the disc dividing the joint into its separate superior and inferior synovial compartments. The posterior attachment, often referred to as the bilaminar zone, is rich in neurovascular tissue and is composed of loose collagenous tissue with scattered elastic fibres. Collagenous fibres of the posterior attachment are not normally concentrated into recognizable superior and inferior band[10] as suggested by Rees.[4] As translation of the condyle takes place, the stretching superior retrodiscal attachment exerts an ever increasing posterior traction force on the disc.[5] It is the only structure capable of applying posterior traction on the disc in a posterior direction. Anteromedially the disc merges into the fibres from the upper head of the lateral pterygoid muscle. Posteriorly the joint capsule attaches just behind the squamo tympanic fissure and merges with the posterior attachment of the disc. The medial wall of

the capsule is fairly loose and weak while lateral wall is loose posteriorly but is strengthened anteriorly by the triangular shaped temporomandibular ligament. This flat sheet of dense collagenous tissue passes downward and backward from the root of the zygoma to the posterior neck of the mandible below the lateral pole of the condyle.

Electromyographic studies have confirmed that the two heads of the lateral pterygoid muscle function independently in the normal patient.[11,12] On opening the lower head contracts while the upper head relaxes ensuring that the posterior thick band is not pulled anteriorly to the condylar articulating surface. During closing as the jaw is clenched, the upper head of the lateral pterygoid muscle contracts pulling forward on the disc, stabilizing it between the articular eminence and the condyle. The position of the disc is determined by the state of tonus of the upper head of the lateral pterygoid muscle, counterbalanced by the posteriorly directed pull of the bilaminar zone and the wedging resistance of the posterior band of the disc.

Using a computer assisted model, Osborn[13] calculated theoretical joint loading produced by various combinations of muscle action. Temporomandibular joints are normally loaded during function but utilizing the posterior band of the temporalis and the inferior head of the lateral pterygoid muscles, the neuromuscular system can create a negative or separating joint force. Jaw closure using

this muscle combination is very inefficient and is unlikely to occur except in unusual circumstances.

When the teeth are not in occlusion, sensory and proprioceptive signals from the muscles and joints dominate mandibular movements. The periodontal receptors come into play as the teeth are stimulated by the contact of food or opposing teeth. The meshing of inclined planes guiding the teeth into maximum intercuspation is the final determinant of jaw position at the end of the chewing stroke or during clenching. This tooth dictated position must be harmonious with that determined by muscle action. Disharmony between these two factors may result in a disruption of normal joint function.[8]

b. Mandibular Dysfunction

As pointed out by Farrar[14] the problem of definition and differentiation of dysfunction is difficult and perplexing. The boundaries separating "ideal", "normal", and "abnormal" cannot be clearly defined. Furthermore, the various forms of dysfunction though not identical are intimately related with overlapping symptoms, etiology, pathogeneses and sequelae. An understanding of each form of dysfunction is essential to establish an accurate diagnosis on which to define treatment objectives and ultimately treatment modalities.

i. Myofacial Pain Dysfunction

The term myofacial pain dysfunction refers to that group of patients who suffer an ill-defined muscle

imbalance causing symptoms of headache, aching and/or tenderness of the various muscles of the head and neck, and peri-temporomandibular joint tenderness.[4]

Clark[15] differentiated pain and dysfunction of the muscles of mastication into three categories: myalgia, trismus or splinting, and mandibular incoordination. Myalgia is considered to mean both the patient's subjective pain in the muscles as well as tenderness to palpation of these muscles. A strong correlation has been demonstrated between jaw muscle hyperactivity and the symptoms of myalgia.[16] While there is broad agreement that muscle fatigue and spasm resulting from hyperactivity are responsible for the pain and tenderness, the question of the cause has remained controversial.

One hypothesis for the origin of MPD problems is based on the assumption that various occlusal disharmonies can disrupt normal neuromuscular function.[17] Clinicians who subscribe to this hypothesis spend a good deal of time observing, recording, and analyzing occlusal relationships when examining patients with MPD. Treatment is then directed at correction of the occlusal disharmony to "eliminate the cause".[18,19] The modalities of occlusal correction could include equilibration, orthodontics, and full mouth prosthetic reconstruction.[20]

One alternative hypothesis is that MPD is caused primarily by emotional stress rather than mechanical factors.[21] Subscription to this psychophysiologic

theory would indicate conservative dental treatment modalities.

Various investigators have tested the relationship between the cervical spine with its associated musculature and the masticatory system. Cervical posture changes can affect the mandibular path of closure,[22] mandibular rest position,[23] and masticatory muscle activity.[22,24] Increased masticatory electromyographic levels have been noted with cervical backward bending, suggesting that the cervical apophyseal joints can neurologically directly alter muscular activity about the jaw.[25] An abnormal postural position frequently adopted is that of the forward-head posture, which Kendell[26] defined as a slumped rounded back with hyperextension of the cervical spine. This anterior head position relative to the normal weight bearing axis of the spine, will lead to increased gravitational forces on the head with resultant strain on the cervical apophyseal joints, ligamentous structures, and muscles. Therefore the forward-head posture may alter the neuromuscular influences of the cervical system on the masticatory system.[22] Physiotherapy is an important treatment modality for the relief of pain and restoration of function in the musculoskeletal system.[27]

There is strong clinical evidence that occlusal splints have a major effect on masticatory myalgia symptoms. Agerberg and Carlsson[28] in a controlled study found a 71% decrease in MPD symptoms with splint therapy.

Carrao and Caffesse[19] evaluated the effect of splint therapy as the only treatment for 27 patients with muscle pain. They reported 57% of the patients as cured, 26% as improved and 15% as worse or no change. Magnusson and Carlsson[29] using a control group found a distinct decrease in muscle tenderness in patients with MPD when treated with splint therapy.

Solberg et al[30] measured nocturnal muscle activity in eight patients with bruxism and found that insertion of a full arch occlusal splint reduced muscle activity. When the occlusal splints were removed, pretreatment EMG levels returned. Clark et al[31] evaluated 25 patients with MPD who were treated with full arch stabilization splints. This study confirmed earlier research and showed that full arch maxillary splints affect the nocturnal muscle activity; however the effects observed were inconsistent. Only 52% of the patients showed the expected reduction in nocturnal EMG levels, while 24% showed no change and 20% had an increase in nocturnal muscle activity. Interestingly the authors reported that the majority of the patients showed subjective symptom improvement. Even some cases with an increase in nocturnal EMG levels reported subjective improvement.

Several factors may account for the differential effectiveness of splint therapy. Splint design, including differences in vertical dimension and occlusal contact pattern, along with factors such as the cause of the

original symptoms, should be considered. Reduction in nocturnal muscle activity may not be a necessary condition for symptom improvement. Alternate explanations for improvement of symptoms with splint therapy may include: redistribution of oral forces, reduction of parafunctional muscle activity during waking hours, and the placebo effect of receiving "TMJ treatment."[16]

Greene and Laskin[32] evaluated the long term status of 135 patients treated for MPD with a variety of conservative modalities. The specific form of therapy did not seem to be the essential factor in determining the long term success or failure in these patients. The authors concluded that psychological factors within the patient and the dentist-patient relationship determine treatment outcome. Psychotherapy may form an important aspect of the treatment of MPD.[33]

Masticatory trismus or splinting implies that the normal pain free range of motion is decreased as a result of protective muscle activity in the jaw closing muscles.[15] Reduced range of motion as a result of trismus must be carefully differentiated from other mechanical causes. Although trismus is a common finding in patients with mandibular dysfunction and occlusal splints are often used to treat patients with this disorder, there are no studies published that have evaluated the range of motion before and after splint therapy.

Mandibular incoordination is interpreted to mean any of the following: decreased speed of mandibular closing movement; lack of coordinated or smooth lateral, protrusive, or opening movements; or a decreased proprioceptive ability as measured by mandibular kinesthetic tests.[15] Because some patients can perform reproducible mandibular movements and others cannot, the hypothesis has been developed that a pantograph can be used to determine the coordination of mandibular movements.[34] Pantographic tracing reproducibility is quantified with a pantographic reproducibility index (PRI). Beard and Clayton[35] evaluated the ability of 15 patients with mandibular dysfunction to make reproducible lateral and protrusive jaw movements. The reproducibility of these movements improved significantly with splint therapy, but removal of the appliance without any follow-up occlusal adjustment resulted in a return to the original state of muscle incoordination. Unfortunately, no comment was made in this study as to the correlation between muscle incoordination and subjective symptoms. In a 1985 literature review on the use of the pantographic reproducibility index, Clayton[36] concluded that the RPI can be used to determine the presence or absence of TMJ dysfunction. It can be used to evaluate the success of treatment modalities such as occlusal splints, occlusal adjustments, and restorative treatment on clinical success in experimental patients.

In 1986, Clark and Lynn[37] found that as a group, patients with mandibular dysfunction differ significantly from control subjects in the accuracy of right and left lateral movements and in the maximum velocity of a retrusive jaw movement. However they found a lack of correlation of questionnaire data and clinical examination data with horizontal plane jaw movements. It was concluded that jaw movement reproducibility tests are positive for mandibular dysfunction in general, but have no specific diagnostic implications.

ii. Internal Derangement

The term internal derangement implies an intracapsular alteration or mechanical fault which interferes with the smooth action of the joint.[38] Internal derangement of the temporomandibular joint has been redefined and classified as an abnormal relationship of the disc to the condyle.[39] Subclassification can be made on the basis of disc displacement with or without reduction. In 1951, Ireland[40] described a progression from clicking, to clicking with intermittent limited opening (closed lock), to permanent limited opening, and finally to crepitus. He proposed that the mechanism for these observations was an anteriorly displaced disc. Convincing evidence of the existence of internal derangements has come from surgical observation and from gross histologic examination of autopsy material.[41,42,43,44]

Condylar path tracings have further supported the

concept of anterior displacement of the disc. Farrar[45] found that in people with clicking joints the path of the condyle deflected inferiorly as the click occurred. This might indicate a dislocation of the disc with respect to the condyle prior to the click. Similarly, Omnell[46] radiographically observed an increase in joint space immediately prior to the click.

More recently the use of temporomandibular joint arthrography has allowed visualization of the disc by injection of contrast material into the joint spaces. Several investigators have reported descriptions of normal and abnormal temporomandibular joint arthrograms.[47,48,49,50] These arthrographic observations have been correlated with clinical histories of clicking and locking, and with surgical observations.[51,52] Joints with reciprocal clicking consistently showed disc displacement with reduction. The posterior band was positioned anterior or antero-inferior to the condylar head in the closed jaw position. On opening the disc regains its normal relationship after the condyle clicks forward under the posterior thick band. This disc reduction may occur early, intermediate, or late as determined by the degree of interincisal opening at that moment. At some point during closing, the disc displaces forward once again into an anteriorly displaced position. The timing of the opening and closing clicks do not necessarily correspond. Closing clicks are often less

detectable and may require loading of the joint to be reproduced. In an autopsy study, Westesson[38] found that 38% of the 26 joints with reducing anterior disc displacement reduced without clicking. In approximately one half of the joints with reciprocal clicking, the disc was only partially anteriorly displaced. A significant proportion of reducing displaced discs also showed deformation. Neither the degree of displacement nor the degree of deformation could be diagnosed by the joint sounds.

A dissection study on autopsy specimens using high speed cinematography has shown that clicking is most frequently caused by the condyle forcibly seating against the disc and temporal component after having rapidly passed over the posterior band of the disc.[53]

In the joint with a non-reducing anteriorly displaced disc, the disc remains in its abnormal position during the opening and closing movements. Clicking sounds were not generally heard, although crepitus could sometimes be detected. Although disc displacement without reduction is usually preceded by disc displacement with reduction, only 50% of the patients could recall previous clicking.[54] Furthermore, only 50% of the patients could recall an acute onset.

Several studies have correlated joint sounds and arthrographic findings with joint morphology on fresh autopsy specimens.[38,55] Disc deformation was rarely

seen in joints with normal disc position. In joints with partial anterior disc displacement, the deformation occurred somewhat more frequently and was consistently located in the displaced region of the disc. Seventy seven percent of the 32 joints with completely anteriorly positioned discs showed disc deformation.[38] There was a range of variation in the extent of disc deformation between joints and also between different mediolateral regions of the same disc. The most frequent disc deformation was enlargement of the posterior band. All but one of the 11 non-reducing anteriorly positioned discs were deformed. It was concluded that disc deformation was preceded by disc malposition.[38]

Since the temporomandibular disc does not have the capacity to undergo cellular remodelling,[59] deteriorative changes in its internal architecture will occur with abnormally directed or excessive forces. Scapino[56] reported on the histopathology associated with disc displacement without reduction. On gross examination disc deformation is accompanied by the loss of distinction between the regions of the disc. Flexure of the disc occurs with jaw opening, with a greater frequency of upward folding. The forward location of the transverse collagen fibers of the posterior band, along with a disruption of the normal fibers of the central portion of the disc by the addition of transverse fibers, were interpreted as deteriorative changes associated with disc

flexure. He also reported fibrosis of the anterior part of the posterior attachment associated with compressive loading. When the disc is in an anteriorly displaced position, the condyle articulates with the posterior attachment of the disc rather than the disc itself. This fibrosis has the maladaptive consequence of reducing the elasticity of the posterior attachment, further reducing the possibility of recapturing the disc. Articulation on areas of the disc other than the central bearing area of the disc may lead to perforation.

Perforations are identified with arthrographic examination by observation of simultaneous filling of both inferior and superior joint spaces.[58] In autopsy specimens examined by Westesson *et al*[38,59] perforations were seen only in joints with anterior disc displacement, and were confined to the posterior attachment in most cases. This differs from an earlier autopsy study by Oberg *et al*[42] which reported perforations to be located in the disc. Graham *et al*[58] found perforations in both the posterior attachment and the disc proper. Moffett[3] suggested that discs that continue to be compressed in one area may become thinned in that area and eventually perforate.

Medial disc displacement is another form of internal derangement of the temporomandibular joint.[60,61] However since sagittal sectioning does not show this displacement well, a study on frontally sectioned

temporomandibular joints would help to increase current understanding of this type of internal derangement.

Positioning of the posterior band anterior or anteromedial to the condyle creates an obstruction to normal movement of the condyle in these directions. Katzberg *et al*[62] studied the translation of the condyle to maximum opening in; normal patients, patients with clicking, and patients with locking. Patients with chronic locking were usually able to translate the condyle to a point posterior or inferior to the height of the eminence. The normal patient went a little further and the patient with clicking could often translate even further to a point anterior to the eminence.

Treatment of internal derangements includes surgical and nonsurgical methods. The nonsurgical treatment of anterior disc displacement with reduction was pioneered by Farrar[63] and involves using a mandibular anterior repositioning splint designed to recapture the disc in its correct anatomic relationship to the condyle. The mandible is moved forward into a non-click position with the least possible opening and forward shift. This therapeutic position is maintained through incorporation of guiding inclines which allow the antagonist teeth to occlude with the appliance. The splint is to be worn 24 hours per day except for cleaning.[64] Over a period of time the splint is adjusted to return the condyles as near as possible to the hinge position without clicking.

The mechanism by which repositioning splint therapy works has not been established. It has been suggested by Polwick and Riggs[65] that if the disc and condyle are kept properly aligned the posterior soft tissue attachment of the disc may "heal", the disc may recontour, and/or osseous remodelling may occur. The prognosis for successful anterior repositioning appliance therapy to recapture the displaced disc diminishes significantly with later opening clicks.[10] Patients with late opening clicks and early closing clicks have a very poor prognosis.[65] Bell et al[64] reported that the short term success of reducing anteriorly displaced discs in the correct position is about 50%. This is comparable to the success rate reported by Clark.[66] Tallents et al[67] found that 15% of patients with painful clicking of the temporomandibular joints do not have a reducing disc, and will therefore not respond to splint therapy directed at reduction of the displacement. In a recent study Manzione et al[68] found that 26 out of 56 of clinically recaptured discs as indicated by a normal range of motion without clicking, still had an anteriorly displaced disc when examined by arthrography. Three of the 26 unsuccessfully treated patients developed anterior displacement without reduction during splint therapy, and one of these was shown to have an associated disc deformation.

Patients with acute anterior displacement without reduction will complain mostly of pain and limitation of

opening.[69] There may or may not be a history of previous clicking. Many will have noticed the onset of pain at the same time clicking disappeared. These patients experience pain on maximum opening (which is restricted) and on forced occlusion. There may be associated neuromuscular discomfort attributable to myalgia. Widening of the joint space on maximum opening in combination with posterior condylar position at maximum intercuspation were described by Farrar[45] as signs of disc displacement without reduction. In normal joints the joint space is larger at the interocclusal position than at maximum opening.

In cases with a short duration of locking there is a possibility of unlocking the joint with manual manipulation.[45] After manual repositioning the disc must be stabilized in the reduced position with an anterior repositioning splint.[70] In contrast the long term or chronic closed should usually not be manually manipulated because of the probability of existing disc deformation.

If the anteriorly displaced disc cannot be reduced and maintained with conservative methods, the clinician has two options. The internal derangement can be accepted as status quo for that patient, or surgical intervention can be undertaken. Potential surgical options include menisectomy, condylotomy, high condylar shave and surgical repositioning of the disc.[69,71]

Westesson[72] has suggested the following criteria for surgical intervention in the management of internal derangements of the temporomandibular joint:

1. All forms of nonsurgical therapy should be attempted first.
2. Arthrograms should document pathologic changes in the joint.
3. The patient should have pain or considerable dysfunction.
4. The pain should emanate from the joint proper.

According to Farrar and McCarty,[14,45,48] and Weinberg[73], considerable insight into the position of the disc can be obtained by studying condylar position as seen on transcranial or tomograph radiographs. According to these authors, a reduction in posterior-superior joint space can be correlated with anterior displacement of the disc. It was further suggested that treatment should be aimed at reestablishment of normal joint space.

There are several well documented studies which point out the futility of such an exercise. Blashke and Blashke[74] measured joint space in 50 subjects with asymptomatic temporomandibular joints using corrected lateral tomographs. Although the condyles were most often centered in the glenoid fossa in the sagittal plane, the standard deviations around the mean joint position were very large. Some condyles would have been classified as posteriorly or anteriorly displaced according to the

principles of Farrar, McCarty and Weinberg. A study by Hansson et al[75] attempted to correlate condyle position with joint symptoms and structural radiological changes. The various "displaced or deviate" radiologic condylar positions with the mandible in centric occlusion did not correlate with any clinical signs or symptoms. Katzberg et al[76] measured the posterior-to-anterior joint space ratio from linear tomographs in 50 patients. Arthrograms were performed to assess for disc displacement. No statistically significant difference in joint space measurements was found between normal joints and joints with disc displacement without reduction. Hatcher et al[77] measured the joint space on bilateral serial tomographs of 56 patients. The joint space varied from the lateral and medial regions in 36% of the joints examined. Diagnostic interpretation of joint space must include a three dimensional analysis to take into account regional variations. Methods that do not consider joint space in three dimensions may not accurately represent joint anatomy.

iii. Degenerative Joint Disease

The term degenerative joint disease is used interchangeably with the term osteoarthrosis,[78] and is defined as "the pathologic destruction of articular tissue and bone that occurs whenever articular remodelling does not maintain equilibrium between form and function."[3]

The shape of the temporomandibular joint structures is dynamic and subject to continual change. The growth process is not only expansive but leads to a change in the shape of the structure through a mechanism of differential bone deposition and resorption.[79] At the end of growth remodelling continues and involves both the soft and hard tissues. Remodelling of the hard tissues is by progressive substitution of primary bone through a system of secondary osteotomes and osteotomic remnants. Bone trabeculae tend to realign in sites in which remodelling is more active. If the rate of bone resorption does not coincide with the rate of deposition it leads to a change in shape. remodelling is said to be progressive when proliferation of tissue occurs and regressive when osteoelastic resorption is evident.[8] Soft tissue remodelling is evidenced by increased activity in the proliferative zone of the articular tissue.[80] Early remodelling changes appear to be largely progressive, with thickening of the articular soft tissue taking place along with osseous change. Such change takes place more on the condylar than the temporal surface, and little or none occurs in the disc.[81] When bone resorption prevails over bone deposition it leads to flattening and concavities.

In contrast to remodelling which involves reshaping without surface defects, degenerative joint disease is characterized by a breakdown in the continuity of the

articular surface.[83] Blackwood[84] described in detail normal remodelling in the temporomandibular joint and he concluded that in physiological remodelling the superficial articular tissue remains largely unchanged, with considerable activity occurring in the underlying tissue and bony zones. By contrast in degenerative joint disease one of the earliest observed changes was loss of the normal arrangement of cells in the soft articular tissue. The number of cells was decreased with the remaining cells becoming clumped together and irregularly distributed. In addition, the fibrillar matrix became disorganized.

Using light and electron microscopy, DeBont[85] described the existence of several different zones within the articular tissue of healthy condyles. These are the articular surface, articular zone, proliferative zone, fibrocartilagenous zone, calcified cartilage zone, and subchondral bone. Normal articular surfaces exhibit a nearly structureless layer of fine fibrils about two microns thick, which seems to correspond to the lamina splendens described in other joints.[86] The articular zone is composed of collagen fibrils arranged in sheets and running parallel to the articular surface. The density of collagen fibers is greater than in the normal hyaline cartilage of other joint surfaces.[87] Between the collagen fibrils lies the mucopolysaccharide ground substance acting as a physical binding agent. Long

spindle shaped fibrocytes have blade like processes of plasma membrane intruding between adjacent fasciculi of collagen bundles. Fibrocytes within the proliferative zone are shorter and spindle shaped. Collagen fibrils within the fibrocartilagenous and calcified cartilagenous zones are organized in randomly oriented bundles.

Changes in the ultrastructure of the articular surface of the condyle in degenerative joint disease have been well documented.[87,88,89,90,91,92] Toller et al[93] found that the surfaces of all pathologic condyles showed loss of lamina splendens, a reduction in collagen fibril size, and evidence of disassociation of both the collagen and its surrounding ground substance. There appears to be an alteration of the ground substance which allows separation of the collagen bundles. This may correlate with the observed loss of mucopolysaccharides seen in studies of degenerative joint disease in cartilage surface joints.[86] Toller et al[93] suggested that the disordered surface layer may affect the joint sliding properties by creating a greater frictional coefficient contributing to further breakdown. Deeper levels showed aggregations of structures, which the authors termed "verifom bodies", and which appear to be collections of abnormal amounts of elastic tissue. Where there were large aggregates of those structures, there was a general disruption of the surrounding matrix and a reduction of normal collagen. It appeared that there was a general

weakening of the fibrous articular structure which allowed a physical breakup and the appearance of clefts in the articular surface. The veriform bodies occurred in association with cytoplasmic abnormalities in some of the fibroblasts, and a correlation between disturbance of cell function under stress and the appearance of veriform bodies was suggested.

Studies by Liem et al[90] and DeBont et al[91,94] also found degeneration and alteration in collagen fibril size in the surface layers and abnormal amounts of elastic fibres, lipid globules and cellular abnormalities in the deeper layers. They concluded that the collagen fibre network disintegration and fatty acid degeneration are osteoarthritic changes in the articular matrix which impair the mechanical properties and play a role in the progression of degenerative joint disease. Liem et al[90] also noted numerous mineral containing vesicles in the region adjoining the subchondral bone. This distribution suggests changes in the local concentration of calcium which could alter the various physico-chemical properties of the tissue such as the water binding capacity, thus reducing the tissue elasticity.

The first signs of degenerative joint disease at the macroscopic level appear in the articular cartilage as fibrillation of the surface. Fissuring, flaking, and abrasion of the cartilage often results in denudation of the bone, and after some time the breakdown may be seen in

a radiograph.[95] In cartilagenous joints osteoarthrosis begins as a focal lesion in the cartilage without clinical symptoms. In later stages clinical signs and symptoms become evident, and in the final stages degenerative radiographic signs appear.[94] This has led to the common acceptance of degenerative joint disease as a noninflammatory breakdown.[96,97,98]

Kopp et al[99] analyzed the synovial fluid from 29 temporomandibular joints that were painful and tender to palpation. Of the 29 joints, 61% had radiographic erosions. Of the joints with erosions, 24% showed an increased concentration of inflammatory cells and plasma proteins in the synovial fluid, indicating increased capillary permeability due to inflammation.

The etiology of degenerative joint disease is not yet fully understood, but both local and systemic factors have been recognized.[78] A review of medical literature suggests that degenerative joint disease represents an imbalance between the stress applied to the joint and its ability to tolerate and adapt to that stress. It can occur in instances where the physical loading imposed upon the joint is increased because of altered anatomy or function, or in situations where the tissues adaptive ability and resistance to stress is lowered.

Strong evidence that the temporomandibular joint is loaded during function has been presented.[100,101,102] The mandible acts as a Class III lever system with the

condyle as its stress bearing fulcrum. Standlee et al[102] utilized a three dimensional photoelastic stress analysis to visualize the intensity and direction of stresses within the condyle generated by various occlusal forces. Functional stresses were found to concentrate in a unidirectional fashion in the neck of the condyle. Adaptation to withstand this concentration of stress is evidenced by heavy cortication and trabecular reinforcement. Functional stresses in the condyle are reduced by its elliptical shape. The translating nature of the condyle requires that it act as a fulcrum in many different positions. This creates an environment of non-preferential stress direction which is confirmed when the condyle was sectioned, revealing uniform trabecular bone and thin cortical bone. The fact that the condyle appears adapted to varied light forces rather than heavy repeated unidirectional forces could be the reason it degenerates rather than reinforcing under heavy cyclical forces.

It seems reasonable to postulate that occlusal forces generated during function are transferred to the articular surfaces of the joints, and that their intensity and pattern depend on the characteristics of the occlusal scheme. remodelling and the consequent change in condyle shape is a biologic adaptation to new functional demands imposed by changing occlusal schemes during life. If the adaptive capacity is exceeded because of the intensity or

duration of the applied forces, a degenerative process will begin. Correlations have been found between dental attrition and degenerative joint disease, and between loss of posterior teeth and degenerative joint disease.[42,103,104] Richards and Brown[105] were able to demonstrate a positive correlation between the degree of degenerative joint changes and the severity of dental attrition in Aboriginal skeletal material. In contrast Eversole et al[106] found no correlation between the degree of dental attrition and the severity of degenerative joint disease when examining skeletal specimens from a contemporary American population. Tooth loss with the associated tipping and overeruption of teeth can contribute to joint dysfunction as a result of occlusal interferences and altered neuromuscular function.[107] The number of remaining teeth or occluding pairs of teeth is correlated with the presence of degenerative joint disease. Biomechanical analysis of the mandibular complex predicts that joint loading is increased as the point of application of the bite force is moved anteriorly. Furthermore, unilateral loss of tooth support will increase loading in the contralateral joint.[108] Prolonged unilateral chewing produces the same overloading.[109]

There is ready agreement that major trauma to synovial joints is likely to lead to degenerative joint disease.[78] Whether degenerative joint disease can be

attributed to the cumulative effect of repetitive minor trauma on normal articular tissue is open to debate. The response of cartilage to one or more deliberate, superficial lacerations has been studied,[110] but this is a poor model of minor everyday injuries. Single or repetitive blunt injuries are closer to the style of trauma most likely encountered in everyday life. Radin et al[111] found that repeated impact loading produced an increased rate of osteogenesis in subchondral bone evidenced radiographically as subchondral sclerosis. He concluded any such change leading to increased stiffness of the bones may increase the susceptibility of the articular cartilage to trauma caused by impact. This supports the concept that microfractures in trabecular bone occur early and are associated with loss or degeneration of those cartilage molecules that resist compressive stress. Temporomandibular joint clicking usually associated with disc displacement with reduction may represent repetitive impact loading,[53] and therefore may lead to subchondral sclerosis.

Degenerative joint disease usually occurs in a joint area formerly subjected to remodelling.[82] The influence of functional changes on temporomandibular joint remodelling is well documented. Animal experiments have shown that a change in mandibular position obtained by intraoral or extraoral devices, induce characteristic remodelling and changes in shape of the articular

surface.[112,113] Anterior condyle placement caused by the application of Class II forces is accompanied by osteogenesis on the posterior superior aspect of the condyle along with increased periosteal deposition on the post glenoid tubercle. Distal displacement of the condyle with Class III forces produced regressive remodelling of the posterior condylar surface and the anterior surface of the post glenoid tubercle.[114]

Radiographic observations using serial tomography showed condylar remodelling with typical shape changes in cases with condylar displacement in maximum intercuspation.[115] According to Mongini, anterior displacement may lead to flattening of the anterior condylar surface, whereas posterior displacement is accompanied by flattening or concavities in the posterior aspect of the condyle.[82,116] Changes in shape may be different between the lateral and medial components of the same joint. Hansson et al[117] concluded that the thickness and arrangement of the articular tissues probably reflects the rate of growth and loading of the condyle and temporal component of the joint. According to Hansson[118] the lateral part of the joint is exposed to the largest functional and parafunctional loads. In an autopsy study, Hansson and Oberg[119] found arthritic lesions were most often localized in the lateral portion of the temporal fossa, and not at all in the most medial portion. Lesions were markedly fewer in the condyle than

in the temporal component. In contrast deviations in form without loss of surface integrity were more common in the condyle. They concluded that these deviations inform which represent remodelling occur more readily in the condyle because of its larger concentration of undifferentiated mesenchyme. It was also suggested that deviations in condyle form are accompanied by an increase in unfavorable biomechanical loading which leads to a degenerative lesion in the temporal component whose capacity to resist loading may be exhausted.

There is another potential explanation for the differences in the frequency of degenerative lesions between the condyle and temporal component. There could be greater frictional loading between the disc and the temporal component than the condyle and disc. As pointed out by Bell[5] the upper joint compartment is subject to translatory movement while the lower undergoes rotational movement.

Oberg et al[42] found arthrotic lesions in 22 of 102 autopsy condyles. Of the 22 condyles with arthrotic lesions, four showed extensive changes while 18 showed only local changes. Thirteen had changes in the lateral portion of the condyle, while five had changes in the central portion and none had changes in the medial portion.

Solberg et al[120] in an autopsy study of 96 joint specimens from young adults, found localized surface

changes were common. These remodelling areas are in juxtaposed functional areas of the articular surfaces. Local modifications of the condylar surface and overall condylar shape appeared to be interrelated adaptive responses to functional stimuli. Irregular condylar forms were well correlated with the shape of the temporal component in the sagittal, horizontal, and frontal planes. Frontally flat and gable shaped condyles articulated against flat and inverted V-shaped temporal components. Each temporomandibular joint component seems to have its own capacity for maturity, adaptive, and degeneration. More than half the 90 temporomandibular joints in the age group between 13 and 38 years manifested different histologic patterns in the medial, central, and lateral components.[123]

To date little research has been done to analyze mandibular symmetry as an etiologic consideration. Underdeveloped condyles, unilateral defects in condylar form and asymmetric lengths of the two mandibular rami can be recognized radiographically.[121,122] Their analysis may provide information on the effect of supporting muscle function and deviants of mandibular movement.

Anatomic studies have shown that degenerative joint disease is found with increasing frequency in older persons,[42,124,125] leading to the suggestion that age is a predisposing factor in the development of degenerative joint disease. DeBont et al[126] found that the fibrous

component of the matrix of aged articular tissue of the temporomandibular joint, consists of a well organized collagen network occasionally interspersed with elastic fibres. The total amount of collagen does not decrease with ageing. However, the proteoglycan content does decrease and its composition is changed. The proportion of keratin sulfate increases and chondroitin sulfate concentration decreases. Finally, the cellularity decreased with ageing resulting in loss of the proliferative zone. Such age-related changes in the articular tissue affects its mechanical properties, which may facilitate pathogenesis of degenerative joint disease.[127] The increased presence of elastic fibres may cause an age-related loss of tensile fatigue stress observed in hyalin cartilage.[126] Ali[128] suggested that the amount of collagen crosslinks and the binding quality of the protoeogylcans is altered.

Lack of effective joint lubrication may exacerbate articular tissue failure by increasing the frictional resistance during loaded joint movements. Recently one of two glycoproteins associated with the lubrication fraction of the synovial fluid has been found to be of particular importance.[78] Whether a failure of synthesis or secretion of this glycoprotein "lubricin" is part of the etiology of degenerative joint disease is not known.

In clinical studies clicking frequently preceded the development of temporomandibular degenerative joint

disease.[129,131] In a study of 127 autopsy joint specimens, the position and configuration of the disc was related to degenerative joint disease.[123] Normal articular surfaces were associated with discs in the normal position. Twenty five percent of the joints with anterior disc displacement but normal disc configuration had degenerative changes. Fifty percent of the cases with disc of even thickness laterally, and 90% of the cases with biconvex discs showed degeneration. These findings support the assumption that internal derangement particularly with disc deformation, may progress to degenerative joint disease. This concept of disease progression from internal derangement to degenerative joint disease has been clinically confirmed with arthrogram studies. Westesson[59] conducted a radiographic study including arthrograms on 128 patients with internal derangement. Structural hard tissue changes were seen in 50% of the patients with anterior disc displacement without reduction but seldom in patients with disc displacement with reduction. Degenerative changes were always found in joints with disc perforation. He concluded that degenerative hard tissue changes occurred predominantly in patients with advanced internal derangement which was interpreted as signs of progression of the disease.

Anderson and Katzberg[132] confirmed Westesson's findings with a tomographic and arthrographic study of 141

patients with temporomandibular joint dysfunction. Nine percent of the patients with a reducing displacement showed signs of degeneration. Thirty nine percent of the patients with non-reducing disc displacement, and 60% of the patients with perforations had degenerative changes. They concluded that internal derangement of the temporomandibular joint likely precedes degenerative joint disease.

The prevalence of degenerative joint disease has been well documented. Oberg et al[42] in an autopsy study of 115 joints evenly distributed for age, found 22% of the adult joints showed evidence of degenerative joint disease. The frequency of degenerative changes increased significantly with age, with the majority occurring after 40 years of age.[130] Blackwood[124] reported degenerative changes in over 40% of his sample of 400 temporomandibular joints removed from cadavers over 40 years old. Rohlin et al[55] reported degenerative joint disease in 34% of their autopsy specimens. A range of 8 to 16% of patients treated at special TMJ dysfunction clinics, were diagnosed as having degenerative joint disease.[129,133,134] Abnormalities of structure and shape of the bony components were found in 50% of the joints of 124 patients examined for mandibular dysfunction. Cortical and subcortical erosions were recorded in 15%, sclerosis in 33%, and deviation in shape such as flattening in 46% of the joints. Females seem

more prone to degenerative joint disease than males, especially in the aged.[42,129]

In spite of their obvious shortcomings, radiographic findings form the basis for diagnosing temporomandibular degenerative joint disease. It is often difficult if not impossible to radiographically differentiate between degenerative changes and adaptive remodelling.[136] The radiographic abnormalities reported as being characteristic of degenerative joint disease are: reduced joint space indicating a loss of articular cartilage, flattening, subchondral sclerosis, osteophytes, and erosions or loss of the cortical lining.[137] Kopp and Rockler[138] concluded that reduced anterior and superior joint space may be due to destruction of articular soft tissue, particularly when in association with crepitation. However, they also pointed out that reduced joint space is probably not an indicator of degenerative joint disease if molar support is present and the radiographs are exposed with the mandible in the intercuspal position. In their sample of 64 patients, no patients with a complete compliment of teeth had evidence of reduced joint space. They also concluded that subchondral sclerosis is most probably an adaptive response to increased functional loading. Flattening of the condyle and articular eminence is frequently associated with bony condensation or subchondral sclerosis. Oberg et al[42] found that flattening m-

occur in the absence of arthrosis. In the absence of other signs of degeneration such as surface erosions or irregularities, flattening is usually representative of remodelling. Condylar flattening and subchondral slcerosis is often present as an adaptive response in joints with reducing disc displacement.

Rasmussen[141] found that erosions were predominantly located in the lateral pole of the condyle. Osteophytes and lipping were found in the anterior part of the condyle and even then only in advanced cases. Osteophytes can be caused by apposition of bone or simulated apposition due to destruction of adjacent bone. This may create an overestimation of the frequency of osteophytes.

There is a clear risk of under-diagnosing degenerative lesions in temporomandibular joint radiographs. Very early changes in the degenerative process may occur in the articular soft tissue long before radiographic signs are visible.[139,140] Degenerative changes in the temporal component must be pronounced to be detected radiographically.[140] The absence of radiographic changes cannot exclude the presence of degenerative lesions. Added to these difficulties are the uncertainties of interpretation and observer variability.[142]

Clinical findings in degenerative joint disease of the temporomandibular joint are not specific. Kopp[143] concluded that the clinical signs and symptoms of patients

with temporomandibular degenerative joint disease do not differ from those of other patients with mandibular dysfunction except for crepitation. Rohlin et al[55] found that 10 out of 12 joints with crepitation had degenerative changes while the remaining two had extensive remodelling. Ogus[88] and Toller[129] reported that the principal clinical findings in osteoarthrosis of the temporomandibular joint are pain on movement or biting, reduced range of motion, joint tenderness to palpation, and crepitus. Rasmussen[89,131,135] reported the clinical findings during the course of degenerative joint disease. During the early painful phases the mobility was restricted with pain in excursive movements and tenderness of the joint capsule and masticatory muscles to palpation. As the pain ceases, mobility improves and crepitation may appear. The best clinical criterion for degenerative joint disease seems to be crepitation in the later stages of the disease.[144,145] In earlier phases, pain and tenderness in the joint and muscles may be a consequence of lesions of the joint proper, without the joint having shown any signs of radiographic changes.

Kopp and Rockler[138] found no correlation between the number of radiographic abnormalities and the degree of pain or dysfunction. Berrett[135] pointed out that there is a population segment with degenerative joint disease but few if any symptoms. Under certain circumstances such a patient with no symptoms may suddenly become

symptomatic. Trauma in general is the most common cause. Such trauma may include automobile accidents, extensive dental procedures and excessive stretching of the mouth during general anesthesia intubation procedures.

Treatment of degenerative joint disease can be divided into several approaches. Prescription of analgesics and rest often combined with light jaw exercises to maintain or increase the range of motion, is a common method of treatment. Degenerative joint disease is generally regarded as noninflammatory and therefore anti-inflammatory drugs are generally indicated.

Treatment with intra-articular injections of steroids is controversial, and there is a general reluctance to use corticosteroids in the treatment of degenerative joint disease.[98] Ppswillo[146] demonstrated that the articular surface in Macacus irus monkeys showed histologic damage after six injections with hydrocortisone. Toller[147] found no radiographically detectable damage to the articular surface in humans with a single intra-articular injection of up to 40 mg of prednisolone trimethylacetate. Intra-articular steroid treatment has most success in patients over 30 years of age. The older the patient the greater the likelihood of clinical improvement.[147,129] Toller recommended it not be used in younger age groups. Long term (5 year) results suggest that such successfully treated patients continued pain free. In cases where there is evidence of articular

erosion before injection, an advancement of the lesion with reduction in size of the condyle can be expected.

In a double blind study of 17 patients with osteoarthritis of the knee, Friedman and Moore[148] found that pain relief with the steroid was temporary with no difference within four weeks. Rasmussen[149] reported that steroid injection results in a higher frequency of irregularity of the condyle as seen in transpharyngeal radiographs. He also reported a short term reduction in symptoms, but the long term results were comparable to those after conventional therapy. Majersjo[98] reported that when intra-articular injections of corticosteroids were used the symptoms disappeared rapidly but recurred after some time.

Kopp et al[150] recommended intra-articular injection following the failure of conservative treatment in people with long term pain and tenderness of the joint. Due to the uncertainty regarding the local side effects of corticosteroid, high molecular weight sodium hyaluronate has been investigated as an alternative. Namiki et al[151] reported that 71% of the 45 osteoarthritic knees injected with hyaluronate had effective relief. They found that there was effective relief only if degenerative changes were mild to moderate. In a blind study using injections of sodium hyaluronate and betamethorsone, both drugs were found to reduce the signs and symptoms in patients who had failed to respond to more conservative

treatment.[150] No statistical significance between drugs in their short term therapeutic effects could be found. However, the long term effect on the temporomandibular joint is not known.

Occlusal splint therapy is also a common treatment modality for degenerative joint disease (DJD). Unfortunately only a few uncontrolled clinical reports discuss occlusal splint therapy procedures for patients with radiographically confirmed degenerative joint disease. Mongini[152] used splints and other forms of occlusal therapy to reposition the mandible into a concentric condylar position. He reported radiographic evidence of remodelling in seven out of eleven patients who had flattened degenerative condylar surfaces and abnormal joint position before treatment. Weinberg[153] discussed similar treatment but offered no specific evidence of remodelling as a result of treatment. Isberg-Holm and Ivarsson[154] reported reconstruction and reshaping of a condyle to normal following splint therapy.

Although research has established the general fact that condylar remodelling does occur in animals,[112,113] the effect of a repositioning splint on the degenerative condyle has not been strongly established. Much research could be done to quantify remodelling changes or a decreased progression of DJD in humans with occlusal splints. Furthermore, the need to remodel condyles simply because they do not appear normal must be questioned.

Given current concepts regarding the etiology of degenerative joint disease, splint therapy should be aimed at reducing loading in the temporomandibular joint. Ito et al[155] investigated joint loading associated with several different splint designs. Splints without posterior tooth support resulted in an increased joint loading during clenching. Likewise splints with unilateral posterior contact create increased loading in the contralateral joint, with distraction in the ipsilateral joint. Therefore bilateral centric stops on posterior teeth appear important for protecting the joints from excessive loading, particularly during parafunctional activities.

Occlusal splint therapy can reduce joint loading indirectly by reducing muscle hyperactivity[31] and provide symptom relief by decreasing coexistent neuromuscular symptoms.[28,29,30] Traction of the vertebral column has been suggested as a means of treatment of acute cases with prolapsed disc where conservative treatment has failed.[156] Vertical traction of the temporomandibular joint, causing an increased distance between the osseous components of the joint may be an effective means of eliminating joint loading. Such an effect can reportedly be obtained using spring mechanisms between the maxilla and mandible,[157] acrylic resin splints to gradually increase vertical dimension of occlusion,[156] and pivoting splints.[158,159] Ito et

al[155] found clenching immediately following placement of splints with bilateral posterior pivots did not distract the condyles. Lous[160] reported using pivoted splints with a chin strap and headgear apparatus to emphasize the tilting effect as a means to treat disc displacements. Rasmussen[149] reported that treatment of degenerative joint disease with pivotal splints resulted in relief from pain, but increased the extent of the radiographically observed degenerative disease.

Several researchers have reported that degenerative joint disease runs a clinical course of one to three years followed by a natural regression of symptoms usually ending with little or no permanent disability.[28,29,129,135,154,161,162] Rasmussen[141] in a five year study of 119 patients found that degenerative joint disease has a radiographically evident destructive and repair phase terminating in healing. The destructive phase lasts I - 1 I/2 years and the repair phase lasts I - 2 1/2 years. In 75% of the 119 cases examined, the entire course took less than 18 months. All cases completed the healing phase by three years. Once the healing phase was completed the joints seemed quite stable. Although Hanson et al[163] and Greene and Markovic[164] reported definite improvement in subjective symptoms, they did not identify dramatic radiographic evidence of healing over a six and three year period.

Rasmussen[149] compared the effect of various

treatment modalities on the subjective symptoms of patients with degenerative joint disease. Treatment included flat plane splints, pivotal splints, steroid injection, and no treatment. No significant difference was found in the duration of presence of symptoms with the different treatment modalities. Yoshimar et al[165] in a five year study found a gradual improvement in range of motion in all patients with degenerative joint disease. In 12 months, 36% were pain free and at the end of five years, 92% were pain free with 8% still reporting significant pain.

Joint crepitus persists in many patients long after subjective symptoms subside.[129] Hansson et al[163] conducted a clinical and radiographic six year follow-up study of patients with crepitus. At the end of six years, 59% had no clinical change in the crepitation.

Surgical intervention in the treatment of degenerative joint disease is used as a last resort when conservative treatment provides little or no relief.[129] Menisectomy is frequently used with favorable short term relief from pain and functional disturbances.[146,166,167,168,169] In a long term follow-up study of 15 patients treated with menisectomy, Eriksson and Westesson[170] found all the patients were pain free. None had subjective experiences of dysfunction. Two thirds of the joints were crepitant, and all the joints radiologically showed structural hard tissue changes. These included the presence of

osteophytes and flattening of the condyle and eminence. They concluded that menisectomy provides long term relief from joint pain and eliminates subjectively perceived dysfunction, despite the clinical and radiographic evidence of degenerative joint disease.

As pointed out by Majessjo and Carlsson[161] many factors influence the result of degenerative joint disease treatment. The general health of the patient as well as psychologic factors may play a large role. Agerberg and Carlsson[28] described patients with degenerative joint disease as "mentally and physically" delicate patients. Many will have coexisting dentofacial and/or local occlusal disturbances.

The aim of treatment of degenerative joint disease, is to shorten its natural course or at least to make it more tolerable.[129] It is hoped that treatment during the active phases of the disease will relieve pain, preserve function, and prevent or minimize deformity. Once the disease process has stabilized, treatment is aimed at minimizing temporomandibular joint loading. These objectives can be achieved best with a multi-professional approach.

c. Temporomandibular Joint Radiography

Radiology of the temporomandibular joint is the only means available to clinically evaluate temporomandibular joint morphology, including the spatial relationship between the condylar and temporal component of the joint.

A radiograph which presents a two dimensional image of a three dimensional object, has definite limitations in temporomandibular joint study. This problem is further compounded by the fact that a temporomandibular joint cannot be radiographed by conventional cephalometric methods without other radio-opaque structures being superimposed on the film. Successive radiographs are required to study changes in condyle position related to any given treatment or manipulation. The radiographs must be reproducible and represent the spatial relationships of the same joint components without distortion. The radiographs must also be of sufficient quality and clarity to allow proper interpretation of joint components. Changes in condyle position can be quantified either by superimposition of successive radiographic tracings, or by an analysis of the joint space between the condylar and temporal components of the joint.

To resolve the problem of superimposition in temporomandibular joint radiography, radiologists utilize an oblique projection similar to that proposed by Schuller[171] for examination of the temporal bone. In the quest for the best representation of joint structures a number of variations of this basic projection technique have been reported. All of these projections are collectively known as oblique lateral transcranial projections, where the central ray is angled superiorly approximately 15 to 25° degrees against the horizontal

plane and posteriorly ranging from 0 to 15 degrees against the frontal plane.

In 1957 Updegrave[172] described a standardized method with the patient's head positioned on an inclined table. The superior and posterior angles were both 15 degrees. Lindbroom[173] used the same angulations on an upright seated individual. Madsen[171] described a method in which the patient was recumbent with the head turned to one side. The projection to the joint region was 25 degrees for the superior angulation and 15 degrees for the posterior angulation.

Weinberg[174] examined the potential error in joint space analysis introduced by alteration of the projection angle. If the x-ray source is 8 inches away from the patient, the x-ray source-to-film distance is approximately 14 times the object-to-film distance. By mathematical analysis it can be calculated that a 15 degree change in the projected angle of the central x-ray beam, will only produce a 2% distortion of a temporomandibular joint image size. If for instance a joint space of 3.81 mm was being measured, a 15 degree change in the x-ray projection would only create a .14 mm error in image size. In his dry skull study it was found that a 15 degree change in projection angle could alter the projected joint space measurement by a maximum of .5 mm. Since clinical variations would be considerably less than 15 degrees, he concluded that joint space

measurements are accurate and reproducible in the clinical setting.

Weinberg[175] proposed the concept that variation between consecutive radiographs is due to alterations in the superimposition of adjacent osseous structures onto the temporomandibular joint. Because the superimposed skeletal structures are closer to the x-ray source then the joint, alterations in x-ray projection angles will change the pattern of superimpositions projected onto the film. Using metallic markers attached to various aspects of internal bony structures, Weinberg identified the superimposed bones. The posterior clinoid process is often projected over the anterior joint space, and the petrous portion of the temporal bone often superimposes on the posterior joint space. Alterations in the amount of superimposition of these structures onto the joint spaces can create the illusion of an altered joint space.

Weinberg[176] luted .010 wire to the temporomandibular joint fossa and the opposing surface of the condyle in an anteroposterior direction in each of four equally spaced positions. By taking radiographs with the wire in different locations within the joint he was able to identify which parts of the joints were being visualized. The central and medial portions of the condyle are projected inferiorly to the condylar image on the film and therefore are not seen on the radiograph. The x-ray beam will almost always pass through the lateral one third of

the condyle in a such a way that the lateral one third of the fossa and condyle are accurately represented on the radiograph. Weinberg also reported that the anteroposterior joint space proportions remain constant between the lateral and medial aspect so that if the condylar position was concentric in the lateral one third it would be concentric in the medial on third, even though the temporomandibular joint space may decrease proportionately. It should be noted however, that this conclusion was based on a single skull, with no presentation of actual data or statistical analysis.

Bergarstadt and Wictorin[177] placed three metallic markers on the lateral part of the fossa and condyle in corresponding positions. Radiographs were taken at two degree intervals as the superior projection angle was moved from 16 to 25 degrees. Significant distortion for the projection angles between 20 and 25 degrees was noted particularly in the vertical measurement. The authors concluded that the superior angle should be as small as possible without excessive superimposition of bony structures, and that caution should be exercised in evaluating positional relationships in the vertical dimension.

Eckerdal and Lundberg[178] also investigated the risk for diagnostic error produced by sequential images of the same temporomandibular joint taken with different projection angles. Transcranial radiographs of a skull

with steel identification markers in different parts of the joint were compared for 17°, 22° and 27° superior angulations. Even a 5° difference in angulation produced a significant displacement of the markers relative to the radiographic outlines of the joint. The authors noted that an evaluation of joint space differences could seriously alter the clinician's diagnosis of posterior displacement versus concentricity, if the projection angulations are not duplicated. In contrast with Gergerstadt and Wictorin, the validity of joint space evaluation was found to be higher in the vertical than horizontal dimension. Reproducibility of a series of transcranial radiographs can be evaluated by the image of the auditory canal since if projectional angles are the same all outlines of the auditory canal should be identical.

Aquilino et al[179] evaluated the accuracy with which relative joint space measurements taken from transcranial radiographs record the true positional relationship between the condyle and glenoid fossa. They compared the radiographic measurements between three pairs of lead markers placed separately on the medial, central, and lateral aspects of the joint, with actual measurements of joint space obtained from an impression of the joint space. Differences in joint space measurements are found to exist between the medial, central, and lateral parts of a single joint. Furthermore, there were considerable

variations between the true joint space and the radiographically measured joint space at all anatomic locations. Anteroposterior joint space ratios derived from transcranial radiographic measurements did not coincide with the joint space ratios derived from direct measurement. The authors concluded that neither actual joint space dimensions, nor the relative anteroposterior position of the condyles in the glenoid fossa can be accurately recorded with transcranial radiographs. Joint space measurements were reproducible on successive radiographs if skull position and radiographic projections were identical.

Weinberg[174] reported that condylar asymmetry does not affect the superior portion of the joint so that condylar position can be determined if radiographic measurements are limited to this region. He suggested that temporomandibular joint radiographs can only record the relative position of joint structures with joint space ratios, and no attempt should be made to obtain true anatomic dimensions based on joint space measurements.

Kundert[180] in a sample of 11 dental students with asymptomatic joints, established the threshold value of condylar displacements detectable with identical projection transcranial radiographs. The subject's head was fixated in a rigid position with the mandible fixated to the maxilla by a splint specially designed to allow the mandible to move directly forward in .2 mm intervals. A

series of five radiographs were taken at the different mandibular positions. Uniformity of radiographs was checked by reproducibility of the auditory meatus and the position of radiopaque markers relative to bony structures. The mean threshold value of perceptibility of condyle position differences on serial radiographs taken with identical projections was found to be .61 ± .16 mm. The author concluded that the smallest difference in condylar position perceptible in clinical situations is .2 mm. The threshold is primarily influenced by the technical quality and interpretability of the radiograph. The posterior joint space was reported to have less radiographic distortion than the anterior joint space.

Laminography, (sometimes referred to as tomography), is a specialized technique used to minimize superimposition of adjacent structures in a diagnostic radiograph. Rosenberg[181] provided a good review of the history and theory of laminography. The x-ray source moves in one direction, while the film moves move simultaneously in the opposite direction. The source and film are rotated in a constant relationship about an axis lying in the plane of the section to be projected. Planes other than the plane of the section to be projected experience relative displacements on the film and are blurred which result in a radiographic image of only the desired plane.

Ricketts[182] first introduced the use of laminography

for the evaluation of temporomandibular joints. In a follow-up study he used laminographs to quantitatively evaluate condyle position relative to the temporal component of the temporomandibular joint.[183] In a sample of 50 patients with no clinical manifestation of joint disorder, the average distance from the anterior surface of the condyle to the articular eminence was 1.5 mm. The average distance from the top of the condyle to the floor of the fossa was 2.5 mm, and the average condyle was located 7.5 mm anterior to the center of the external auditory canal.

A sound basis for understanding lateral tomograms of the temporomandibular joint was presented by Eckerdal[184] in his study of the correlation between the tomographic image and the histologic anatomy of 27 autopsy specimens. To define the diagnostic possibilities and limitations of tomography as applied to the temporomandibular joint, a series of tomograms taken from different regions of the joint was analyzed for both qualitative and quantitative accuracy. Analysis of structural features requires better contrast and sharpness than does evaluation of joint relationships alone. Eckerdal found that the lateral and medial poles of the condyles, which together comprise one third to one half of its mediolateral width, may not be satisfactorily defined in a tomographic series. Lack of clarity is due to the superimposition of adjacent bony structures not completely blurred, as well as geometric-

morphologic factors within the joint. The parts of the joint which are reproduced with sufficient sharpness to allow clear identification of their contours are regarded as belonging to the tomographic layer. The thickness of the tomographic layer was determined to be 3 ± 1 mm when using a polytome with hypocycloidal motion. In a tomogram every partial layer contained in the tomographic layer can to some extent contribute to the production of image contours. Large morphologic differences between adjacent anatomic layers contained within the tomographic layer may result in a composite image in which the object may be distorted or indistinct. The better radiographic quality of the central part of the joint is an expression of the high degree of morphologic symmetry in the central portion when compared to the morphologic incongruity present in the medial and lateral portions of the joint. Eckerdal also noted that there may be distortion in tomography if there is a large angle between the horizontal axis of the joint and the perpendicular of the tomographic plane.

Individual anatomic variations in the shape and angulation of more than 2900 condyles were investigated and documented by Yale.[185] The horizontal angulation between the long axis of the condyle and the frontal plane (transmeatal line) ranged from 0° to 30° in 99% of condyles with an average angulation of approximately 13°. Variation in vertical condylar angulation ranged from -45° +35° with an average of +5°. The distribution

of vertical angulation was broad with 11.2% having a negative angle, 28.8% having a zero angle, and 60% having a positive angle of $+5^{o}$o $+35^{o}$. Preti et al[186] found that the angle between the condylar long axis and the frontal plane varied from -2 to $+33^{o}$ with a mean of 21^{o} . The standard deviation was 4.2^{o} for the right side and 5.3^{o} for the left side. The maximum difference between the right and left side in the same individual was 23^{o}. Taylor[187] found the horizontal condylar long axis varied between 11^{o} and 35^{o} to the frontal plane in 95% of his sample. He also observed that "when an individual's two condyles were compared, differences in angulation were commonly found". Yale[185] also found this asymmetry in condylar long axis angulation in over 50% of the mandibles examined.

Updegrave[188] stated, "when we consider the gross dissimilarities and variations between individuals and frequently between the right and left sides of a single individual, using a standard fixed angle technique is all but unscientific".

Rozencweig and Martin[189] analyzed a series of tomographic films of the same condyle position, exposed with a variation in angulation in increments of 5 degrees. The projected form of the condyle and the joint space thickness are profoundly modified from one film to the other. This effect can be explained by simple geometry. (See Figure 1)

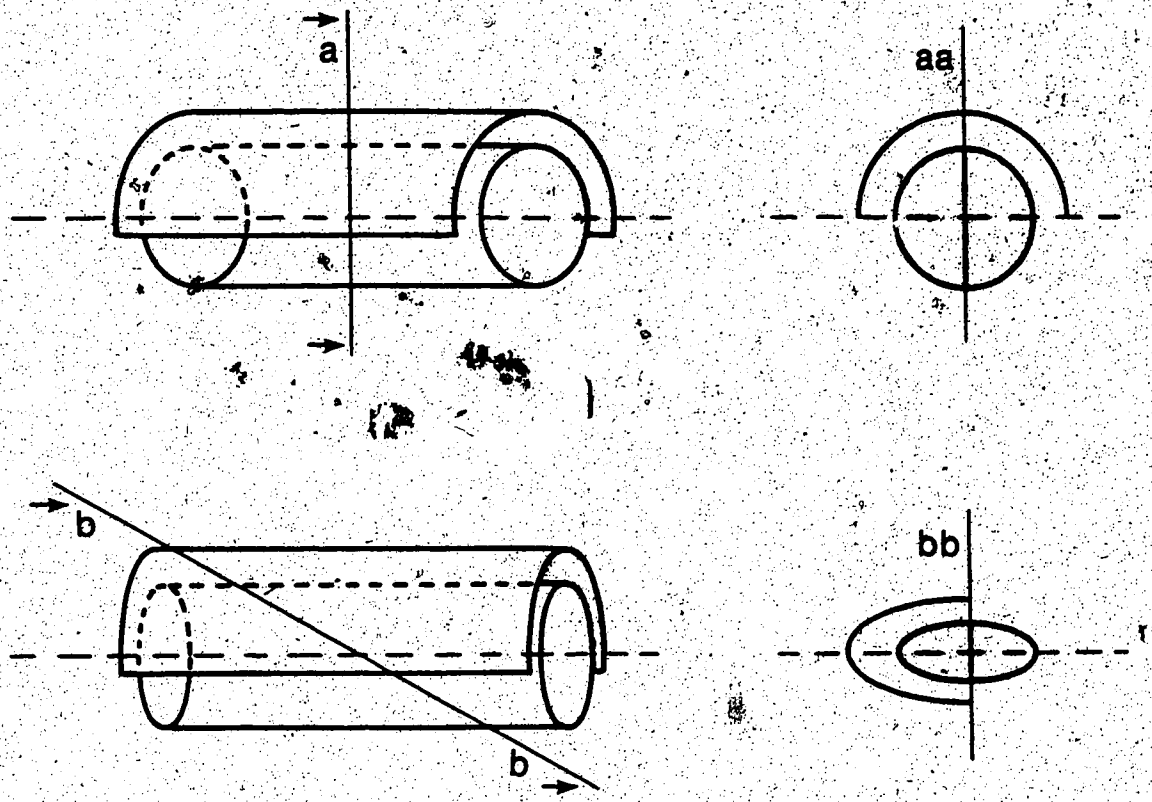


Figure 1. The effect of altered tomographic angulation.

According to Preti et al[190] the accuracy and reproducibility of lateral tomograms of temporomandibular joints depends on the following:

1. Suitable head position so that the condylar long axis is perpendicular to the path of the x-rays.
2. To avoid distortion of condylar position the face should not rest on the table.
3. Correct centering of the temporomandibular joint.
4. Precise programming of layers to include the entire structure.

Rozencweig [189] pointed out that to be sure of the exact position of the condyle on the tomograph, the teeth

must be placed in a stable reproducible occlusal contact.

The submento-vertex projection of the temporomandibular joints is used to measure the horizontal long axis of the condyle, as well as to determine the ideal depth of cut in lateral tomographs. According to Beckwith et al[191] three factors in the submento-vertex projection could produce a significant error:

1. Magnification distortion of the radiograph.
2. Inaccurate determination of the midsagittal plane.
3. The effect of head rotation on depth of cut determination.

It was the contention of the authors that all measurements should be taken from machine and not anatomic landmarks. The midsagittal plane was determined at the center of the headholder, and a straight piece of .030 inch wire was centered in this position over the cassette holder. All radiographs taken with this wire in place show a definite radiopaque line representing the center of the headholder. This line was used as the zero point from which future depth of cut measurements could be made. The anatomically determined midline deviated between +4 mm and -5 mm from the machine midline. In a dry skull study the authors showed that as the condylar anguiation increased, the depth of cut decreased. This was due to the condyle moving closer to the midsagittal plane as the head was rotated to maintain the tomographic plane perpendicular to

the horizontal condylar long axis.

Lysell and Peterson[192] evaluated how different x-ray angulations in submento-vertex projections influence the apparent horizontal angulations of the condylar long axis. When the beam direction was shifted ±14 degrees from a true submento-vertex projection (perpendicular to Frankfurt Horizontal plane), the maximum condylar angulation change was 6 degrees. It was the author's conclusion that since the projection seldom varies as much as ± 14 degrees from a true submento-vertex, the clinician can anticipate good agreement between the measured and true horizontal angulation. They recommend relating condylar angulation to ear plug indicators rather than to skeletal reference points to avoid the variability related to the complex anatomy of the temporal bone as seen in submento-vertex views. Inter-examiner differences are due mostly to difficulty in identifying the medial and lateral outlines of the condyle, combined with difficulty in interpreting the wide range of condylar shapes. Beckwith et al[191] found that the same examiner tracing the same condyle could differ as much as 5 degrees.

Aquilino et al[179] compared the subjective classification of joint space ratios made by knowledgeable observers with actual joint space measurements. They concluded that condyle-fossa relationships cannot be reliably classified by subjective evaluation from temporomandibular joint radiographs.

Omnell and Peterson[193] compared the diagnostic information obtained utilizing a standard technique with an individualized technique for both transcranial radiographs and lateral tomographs. The individualized tomographs were found to show the most structural changes, followed by the individualized transcranials. The standardized transcranial was found to be the least accurate to diagnose condyle-fossa relation and structural changes.

The clinical choice of projection techniques in temporomandibular joint radiography is a matter of interpretation based on the relative advantages and limitation of each projection technique and their relation to the desired diagnostic information. Transcranial radiographs have the benefit of low equipment cost. They have been shown to have good clarity and reproducibility, but unfortunately allow visualization of the structural morphology and spatial relationships only in the lateral part of the joint. Lateral tomographs generally have less distortion and afford views of the joints at different depths. Tomographs have less clarity than transcranial radiographs particularly in the medial and lateral parts of the joints. Serial tomography will allow visualization of the three dimensional nature of the joint morphology and spatial relationships.

Eckerdal and Lundberg[194] compared the diagnostic information available from conventional lateral oblique

transcranial projections and serial lateral tomographs with the histologic morphology and spatial relations. They concluded that the complex pattern of superimpositions seen in transcranial radiographs are poorly understood, the diagnostic information inferred by joint space analysis is exaggerated. The tomographic images agreed well with the true morphology as seen in histologic sections. Spatial relations seen in the medial and central parts of the joints are significantly different from the lateral parts.

Larheim and Tveito[195] compared the reproducibility of temporomandibular joint radiographs using the lateral oblique transcranial projection with the lateral tomographic technique. Thirty five individuals were radiographed with each technique on two separate occasions. The transcranial projections were made with a standard 25 degree superior and 15 degree posterior angulation. The tomograms were taken for the central part of the joint with the plane of sectioning oriented perpendicular to the horizontal long axis of the mandible. Joint space measurements of linear dimensions tended to be more reproducible on conventional radiographs than on tomograms. Although no significant difference was demonstrated between measurements on repeated tomograms, the standard deviation was .74 mm, which is almost double the standard deviation of .38 mm seen in transcranials.

The lateral oblique transcranial projection gives

ambiguous information about the condyle-fossa relationship in a caudo-cranial direction. When the superior joint space is reduced two interpretations are possible; the condyle is either displaced superiorly or laterally. Conversely, a large joint space may be explained as an inferior or medial displacement.[196]

Axial tomography as described by Fairovich and Omnell[196] required placement of the tomographic plane parallel to the orbit-meatal line. According to these authors the main advantage of axial tomography is the information it offers regarding condylar horizontal inclination and shape, and the relative position between the bony parts of the condyle and temporal portion of the joint, both in the anteroposterior, as well as the mediolateral direction. Simultaneous multisectional axial tomography may allow observation of the joint space at different levels. Reproducibility of depth of cut and tomographic plane angulation has not yet been established.

Frontal tomography was proposed by Rozencweig[197] in 1975 to allow visualization of the superior surface of the condyle from the medial to lateral pole with the condyle seated in the fossa. Superior joint space relations can be analyzed with satisfactory clarity in both the medial and lateral aspects of the temporomandibular joint. The plane of focus passes through the condyle perpendicular to the Frankfort horizontal plane. Gushing[198] modified the angulation of the frontal tomographic plane to visualize

the articulating anterosuperior surface of the condyle and posterior slope of the articular eminence. Conventional posteroanterior and submento-vertex cephalometric radiographs were used to determine the location and orientation of the condyle, and a corrected lateral tomography was used to determine the angulation of the articulating surfaces relative to Frankfort horizontal plane. The patient's head is situated so the central x-ray beam is perpendicular to the major axis of the condyle and tangent to the articular surfaces.

d. Summary

The majority of articles reviewed fall into two broad categories of anatomically related and functionally or dysfunctionally related. Renewed interest, most occurring within the past ten years has broadened the knowledge of static and functional anatomy related not just to the temporomandibular joint but to associated contiguous structures as well. Studies involving computer assisted models of skeletal and muscular components in three planes of space have added to our conceptual knowledge, however; the available literature does not address the dimension of time as well as it does the other parameters. Long term studies of treatment efficacy have yet to be reported with most of the current literature reporting case follow-ups of six months.

The psychological aspect of TMJ pain/stress management

is relatively unreported in the dental literature. This component of management is currently receiving an intensified interest in pain and/or stress induced management of discomfort.

Interest in TMJ has resulted in the addition of a number of journals which enable authors to publish their articles with less delay than with the major journals. Whether they survive the current interest level has yet to be seen.

This literature review, although not exhaustive, has been an attempt to provide the reader with a broad understanding of the very complex problem of mandibular dysfunction. An understanding of the current concepts of etiology, pathogenesis, diagnosis and treatment are essential to the practicing dentist. Recent advances have produced a large volume of scientific literature which is difficult to condense and assimilate into a manageable form. Areas specific to the research questions were covered in detail.

III. MATERIALS AND METHODS:

Approval of the study protocol was obtained by the Human Ethics Committee of the Faculty of Dentistry, University of Alberta (Appendix A). Each participant signed the approved consent form (Appendix B).

a. Sample:

The sample used in this study consisted of the first 32 suitable patients presenting to the Temporomandibular Investigation Unit at the University of Alberta and having a clinicoradiographic diagnosis of disc displacement without reduction requiring "offloading" splint therapy. Patients who were pregnant or had a history of temporomandibular joint surgery were not included in the study. All patients accepted into the study had a sufficient number of natural posterior teeth to provide adequate support and retention for the splint appliance. Each patient was assigned a coded model identification number and a coded radiograph identification number.

The subjects were sequentially assigned into three groups so that every third patient was placed into a particular group. The distribution of subjects according to mean age, age range, sex and the right and or left temporomandibular joint diagnosed as having disc displacement without reduction are shown in Table I.

Table 1. Distribution of subjects

	Total No. of Patients	Age (Years) Mean	Age (Years) Range	Male:Female Ratio	Total No. of Joints Affected L	R	L & R
Group 1	10	23.8	13-48	2:8	6	4	0
Group 2	11	28.6	17-42	3:8	10	4	3
Group 3	10	24.8	17-36	0:10	9	4	3
	31						

b. Procedure:

Prior to acceptance into the study each patient underwent a thorough subjective and objective temporomandibular joint evaluation by members of the Temporomandibular Investigation Unit (Appendix B). As part of the questionnaire each patient was asked to subjectively rank their pain level on a scale of zero to ten with zero representing no pain and ten representing extreme pain. This subjective value was recorded again at the end of the study for each patient.

Maxillary and mandibular alginate impressions were taken for each patient and immediately poured with vacuum mixed Vel-Mix dental stone. A SAM(a) bite fork was

---

(a) SAM Prazisionstechnik, Texisstrasse 43, D-8000, Muchen 19, Germany. Distributed in North America by Great Lakes Products, Buffalo, N.Y. 14216.

curved to adapt to the patient's Curve of Spee. Softened green stick compound was placed on the upper surface of the bite fork in three separate locations, tempered in a 120° Fahrenheit water bath and placed on the occlusal surface of the patient's maxillary teeth to index the central incisors and first molars. The modeling compound was cooled and trimmed leaving only a light index to ensure an accurate fit. With the ear rods and nasal reference indicator properly and firmly positioned the bite fork was placed into the mouth in its correct position relative to the indexed teeth and attached firmly to the SAM anatomic facebow. The facebow was removed from the patient and transferred to the SAM 2 system articulator. The [redacted] maxillary model was seated in the compound index and attached to the mounting ring of the articulator with fast setting Snow White #2(b) impression plaster.

A centric occlusion bite registration was taken using Regisil(c) bite registration material. The bite registration was placed over the teeth on the maxillary cast. The mandibular cast was set into the registration and attached to the lower mounting plate with fast set plaster.

(b) Kerr Sybron, Romulus, Michigan 48174

(c) L.D. Caulk Company, Division of Dentsply International Inc., Mildford, Delaware 19963

constant reference points on the joint portion of the temporal bone.

As outlined in the previous analysis, the contours of each glenoid fossa, articular eminence, and condyle were marked directly on the radiographs with a sharp 4H pencil. The reference template was placed over the radiographs so that the point where the 90 degree radial met the arc was located at the reference point marked on the radiograph as the most superior central portion of the fossa. The template was pivoted around the reference point until the best fit of the glenoid fossa was obtained. Two additional reference points were placed at the outside tip of the two 30 degree radials by perforating the radiograph with a sharp probe.

The distance between the condyle surface and the surface of the temporal bone was measured along each 15 degree radial. Superimposition reference points and the bony contours of the glenoid fossa and articular eminence were traced onto matte acetate with a sharp lead pencil from the series A radiograph. The remaining radiographs in the series were individually placed over the tracing to transfer the template reference points onto the radiographs with a sharp probe. Using the template positioned with the reference points, the joint space was measured and recorded for each radial (Figure 7).

All joint space measurements were made to the nearest 0.1 mm using electronic calipers. For the purpose of data

recording and analysis the center radial was assigned the number 0. The 15°, 30°, and 45° radials to the right of the center radial were assigned the numbers 1, 2, 3. The radials to the left of the center radial were assigned the numbers -1, -2, and -3.

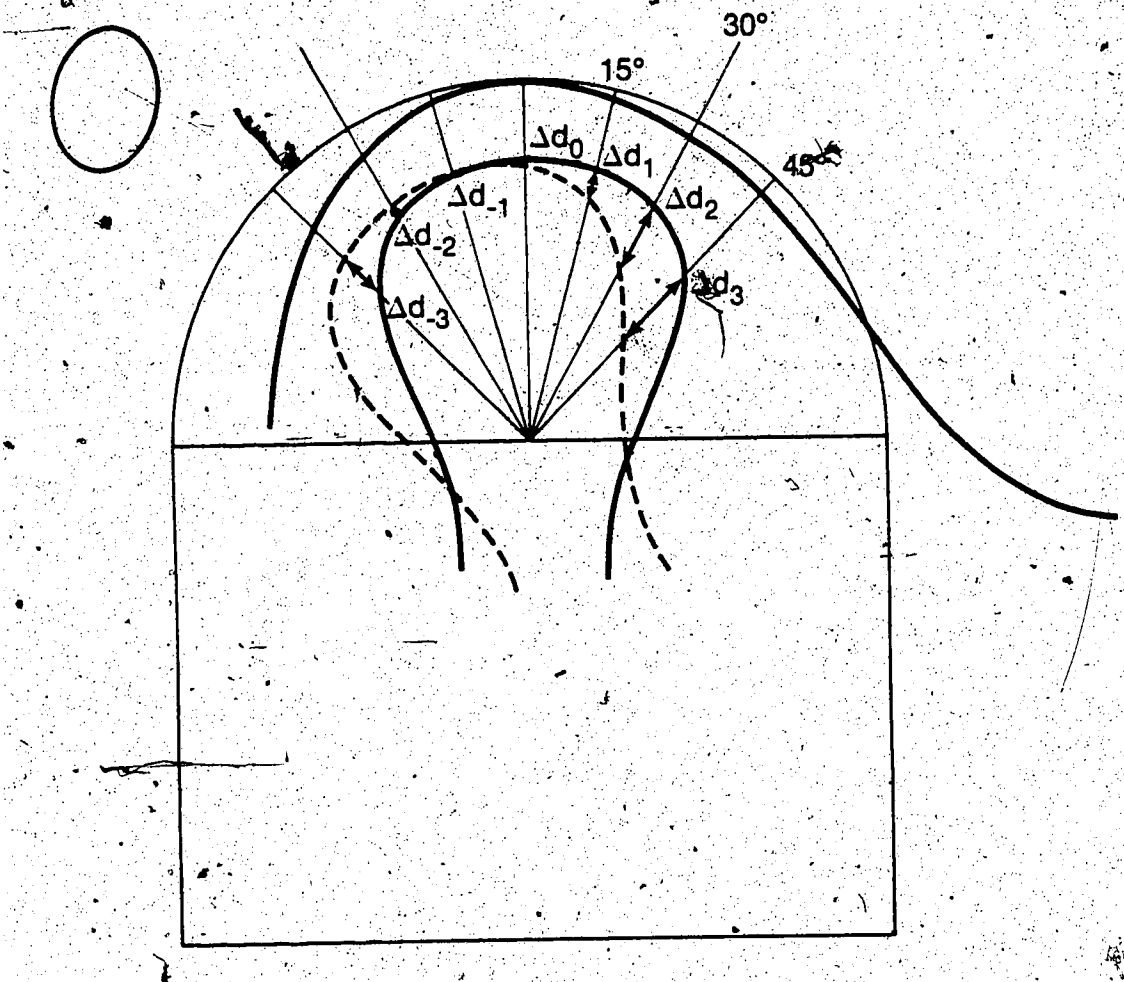


Figure 7. Alteration in joint space with condyle displacement

e. Data Analysis:

Data derived from the radiographic and P.I. measurements were entered and verified onto a data list file for analysis by the SPSS-X package on the University of Alberta's Amhdal main frame computer. The patient model and radiograph identification numbers were decoded and the data was reorganized by experimental group. For the purpose of data analysis the experimental groups were identified by the following "Case Types"; 1 (CO), 2 (pivotal), 3 (pivotal with orthodontics). Data variable labels were defined and listed (Appendix C).

Changes in joint spaces at each template radial (-3, -2, -1, 0, 1, 2, 3) were determined by calculating the difference between the measured joint spaces of the appropriate radiographs. Changes in condyle position between A and A(SI), A(SI) and B(SI), and between A and B were determined for the left and right side on each patient. A positive (+) value was assigned to increased joint space, while a negative (-) value was assigned to reduced joint space.

With the data organized by case type, the mean, standard error of mean, standard deviation and range was calculated for each variable (Appendix D). An analysis of variance was performed for all the mean changes in condyle position. Where the significance of F value was less than .05 it was concluded that there was a significant difference for the variable by case type. Contrast

coefficient matrices utilizing a one-way analysis of variance that assessed both pooled and separate variance estimates were performed for each variable found significant by the initial analysis of variance.

Correlation analysis was performed to compare the recorded M.P.I. values for condylar position change to the radiographically determined (B-A) value for the same coordinate.

Changes in the patient's subjective pain levels during the study period were calculated and recorded. With this data organized by case type the mean, standard error of mean, standard deviation and range were calculated. Contrast coefficient matrices utilizing a one-way analysis of variance that assessed both pooled and separate variance estimates were performed for each variable. Correlation analysis was performed to compare the change in pain level to the amount of condyle displacement produced during the study period (B-A). This correlation was done with the data organized according to the diagnosed status of each joint (non closed lock and non degenerative vs chronic closed lock vs degenerative). The mean, standard error of mean, standard deviation and range of the initial joint space was calculated for each radial. An analysis of variance was performed using the mean joint space of each radial.

Intra examiner reliability for radiographic determination of condyle displacement was established by

IV. RESULTS:

Condylar displacement determined radiographically using X and Y coordinates was calculated and recorded.

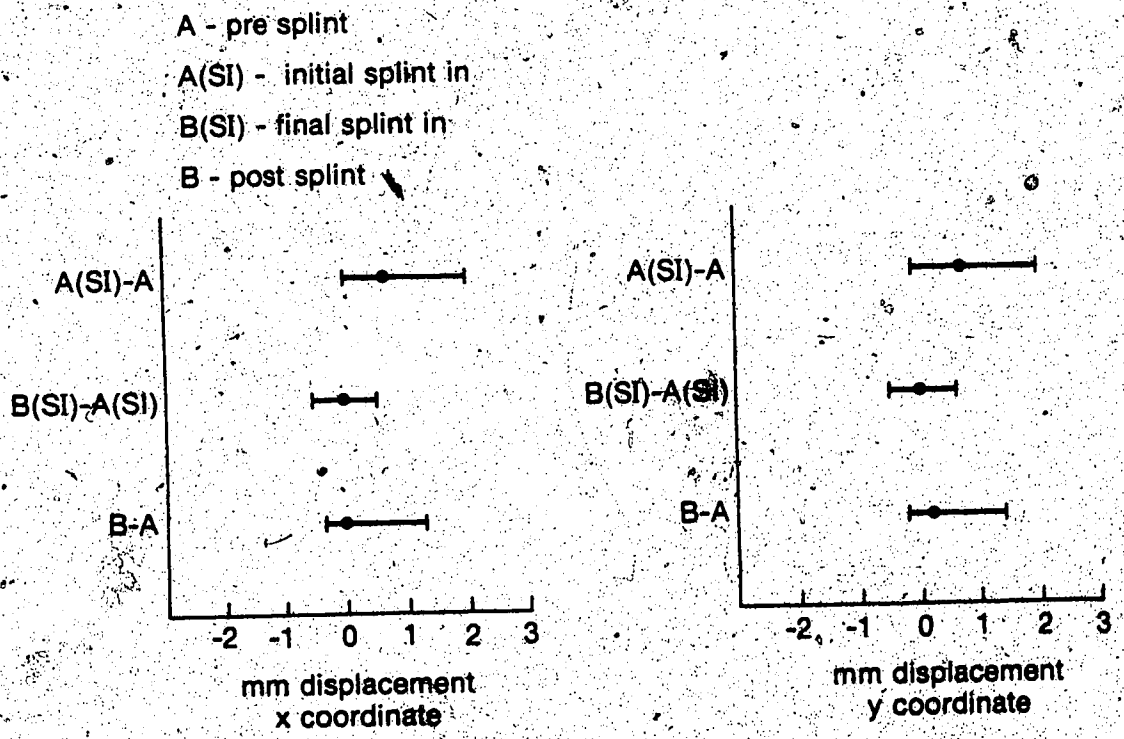


Figure 8. Radiographically determined mean condylar displacement combined left and right for Case Type I.

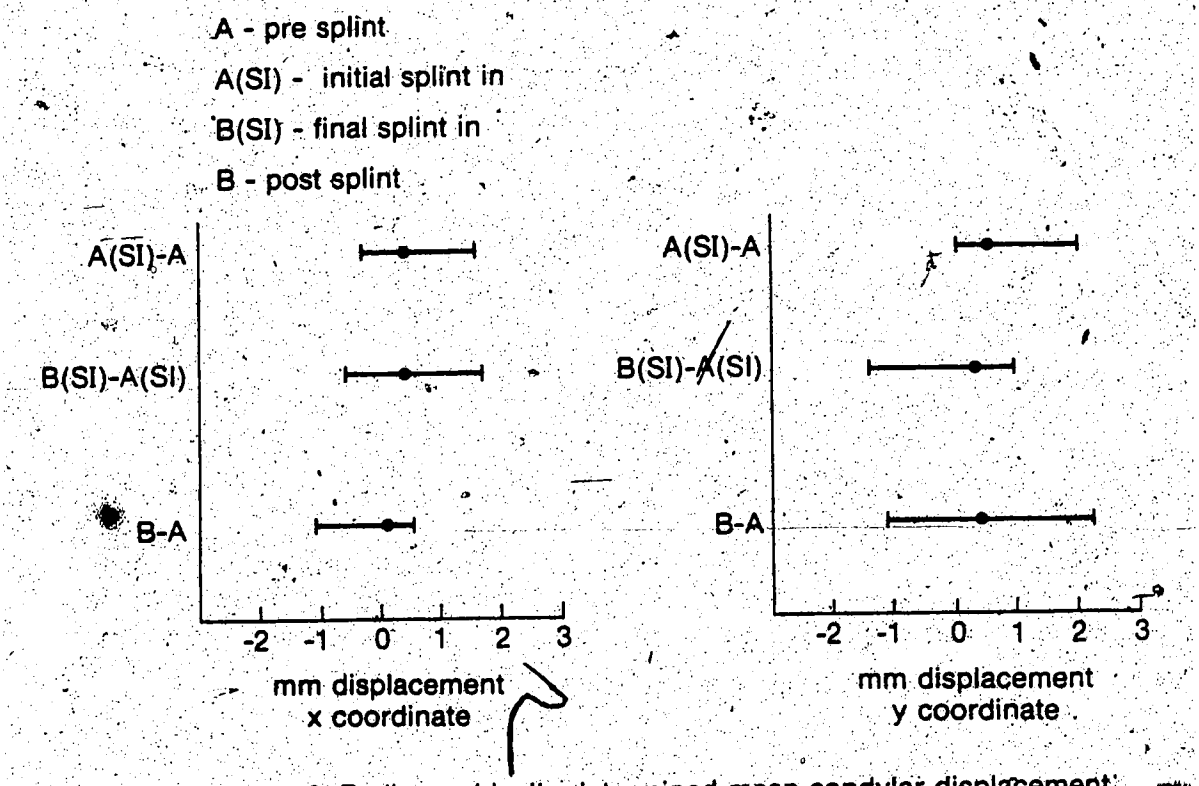


Figure 9. Radiographically determined mean condylar displacement combined left and right for Case Type II.

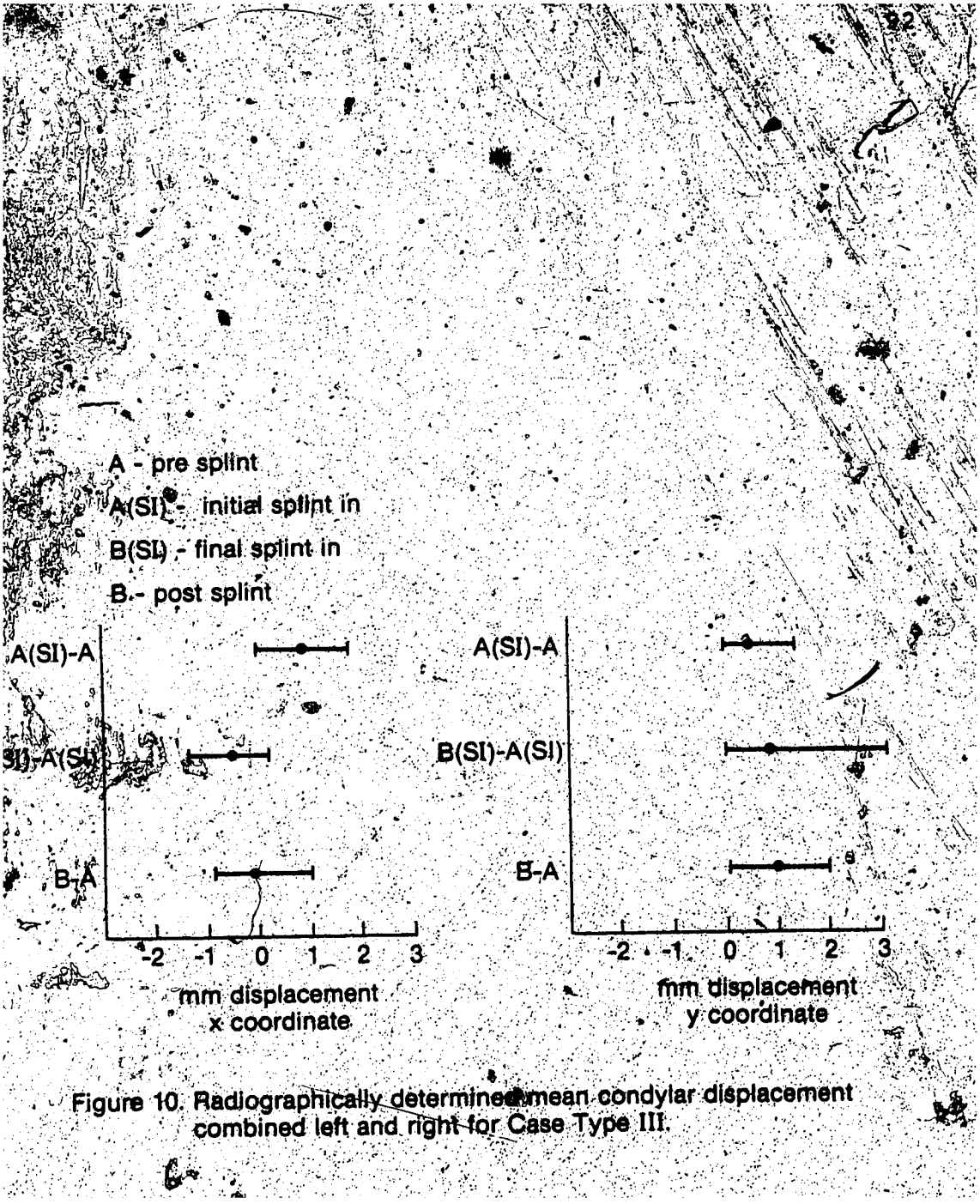


Figure 10. Radiographically determined mean condylar displacement combined left and right for Case Type III.

Correlation coefficients calculated to compare the similarity between radiographically determined vs M.P.I. determined changes in condyle position were all significant at the $p \leq .000$ level for both the left and right joints using an X and Y coordinate system.

Table 2. Comparison of radiographic and articular determined values of condylar displacement

	Left		Right	
	X	Y	X	Y
Correlation Coefficient	0.877	0.757	0.82	0.925
Probability	0.000	0.000	0.000	0.000

One-way analysis of variance was performed for each viable using 2 degrees of freedom for between groups sum of squares and 28 degrees of freedom for within sum of squares. The results of the one-way ANOVA yielded a F probability value that had a value less than or equal to .05 to reject the Null hypothesis. Only variables with significant values were subjected to contrast comparisons by case type to determine which contrasts were most likely to contribute to the overall F probability. The results of variable contrasts by Case Type for X and Y coordinate

condylar displacement are reported in Tables 3 and 4. Because of the very high level of correlation only radiographic values are reported.

Table 3. Significant values for one-way contrasts (X coordinate)

	Left B(SI)-A(SI)	Right B(SI)-A(SI)
Contrast 1 C type 1 vs. 2		
Contrast 2 C type 1 vs. 3	**	*
Contrast 3 C type 2 vs. 3	***	*

A - pre splint
ASI - initial with splint
BSI - final with splint
B - post splint

*p≤0.05
**p≤0.01
***p≤0.001

Table 4. Significant values for one-way contrasts (Y coordinate)

	Left		Right	
	B(SI)-A(SI)	B-A	B(SI)-A(SI)	B-A
Contrast 1 C type 1 vs. 2				
Contrast 2 C type 1 vs. 3	**	***	***	***
Contrast 3 C type 2 vs. 3	***	**	***	***

A - pre splint
ASI - initial with splint
BSI - final with splint
B - post splint

*p≤0.05
**p≤0.01
***p≤0.001

Changes in condyle position were not significantly different between groups of patients treated with a Case Type I splint and patients treated with the Case Type II splints for any of the measured variables. The Case Type III splint with orthodontic traction did not produce a significantly different condyle displacement with initial placement. However, there is a significantly larger inferior and posterior condylar displacement over the 8

week period the splint was worn. The inferior displacement persisted even with removal of the splint.

With the data reorganized into three groups according to the diagnosed joint condition (normal, chronic closed lock and degenerative) the mean, standard error of mean, standard deviation and range for the joint spaces at each radial was calculated and recorded. An analysis ($P < .05$) of variance did not identify any significant difference in mean joint spaces between the three groups.

Differences in joint space measurements were calculated from the recorded measurements. An analysis of variance by case type was applied to each variable. All variables with a significance of F value less than .05 were subjected to contrast comparison by case type to determine which controls were most likely to contribute to the overall F probability (Tables 5 and 6)

Table 5. Significant values of left joint space change for one-way constrasts

	Case Type 1 vs 2	Case Type 1 vs 3	Case Type 2 vs 3
A(SI)-A -45° radian		**	**
B(SI)-A(SI)-A -45° radian		***	***
B(SI)-A(SI)-A -30° radian		***	***
B(SI)-A(SI)-A -15° radian		***	***
B(SI)-A(SI)-A 0° radian		*	***
B-A -30° radian			**
B-A -15° radian		*	**

A - pre splint
ASI - initial with splint
BSI - final with splint
B - post splint

*$p \leq 0.05$
**$p \leq 0.01$
***$p \leq 0.001$

Table 6. Significant values of right joint space change for one-way constrasts

	Case Type 1 vs 2	Case Type 1 vs 3	Case Type 2 vs 3
B(SI)-A(SI)-A -30° radian		**	**
B(SI)-A(SI)-A -15° radian		***	***
B(SI)-A(SI)-A 0° radian		**	**
B-A -15° radian		***	**
B-A 0° radian		**	*

A - pre splint
ASI - initial with splint
BSI - final with splint
B - post splint

*p≤0.05
**p≤0.01
***p≤0.001

Changes in pain levels during the study period were calculated and recorded (Table 7)

Table 7. Change in subjective pain level on a scale of 0 to 10

	X	S. Error	S.D.	Range
Case Type I	-1.0	0.5	1.5	5.0
Case Type II	-3.0	0.7	2.4	8.0
Case Type III	-4.6	.5	1.5	5.0

Contrast coefficient matrices utilizing a one-way analysis of variance was performed and recorded (Table 8)

Table 8. One-way contrast of pain level change by case type.

	Valve	S. Error	T Value	D.F.	T. Prob.
Contrast 1 Case Type 1 vs 2	-2.0	0.79	-2.5	28.0	0.018
Contrast 2 Case Type 1 vs 3	-3.6	0.79	-4.5	28.0	0.000
Contrast 3 Case Type 2 vs 3	-1.6	0.81	-2.0	28.0	0.059

Change in pain level was significantly different between the groups of patients treated with a "C.O." splint and patients treated with the "offload" splint ($P < .05$). The offload splint with orthodontic traction did reduce pain levels significantly more than the CO splint ($P < .001$) but not significantly more than the "offload" splint.

Correlation coefficients to compare change in pain level with the amount of condyle displacement was calculated and recorded (Table 9).

Table 9. Comparison of pain level change with the amount of condylar displacement.

	L		R		Combined	
	X	Y	X	Y	X	Y
Correlation Coefficient	0.116	-0.575	0.076	-0.431	0.110	-0.665
Probability	NS	XXX	NS	XXX	NS	XXX

*p≤0.05
**p≤0.01
***p≤0.001

DISCUSSION

Initial placement of all three splint designs resulted in a significant anterior and inferior condylar displacement with no statistically significant difference in the magnitude of displacement between groups. Each splint design produced a similar increase in vertical dimension of occlusion as the mandible rotated clockwise around a transverse hinge axis. Brewka[200] reported that the true hinge axis is located posterior and inferior to the center of each condyle. Rotation about this axis produces a predictable forward downward movement of the articular surface of the condyle along the slope of the articular eminence. If the location of this hinge axis is known the amount of this translation could be calculated with simple geometry (Figure 11).

In this paradigm if the distance (L) between the first molar and the hinge axis is 60 mm and the distance (l) from the hinge axis to the articulating surface of the condyle is 12 mm; and the mandible is rotated open a distance of 3 mm at the first molar, then the condyle would translate anteriorly and inferiorly 0.6 mm. The mean condylar displacement for the left and right joints of the three groups together was 0.73 mm anterior and 0.59 mm inferior. Condylar position did not change

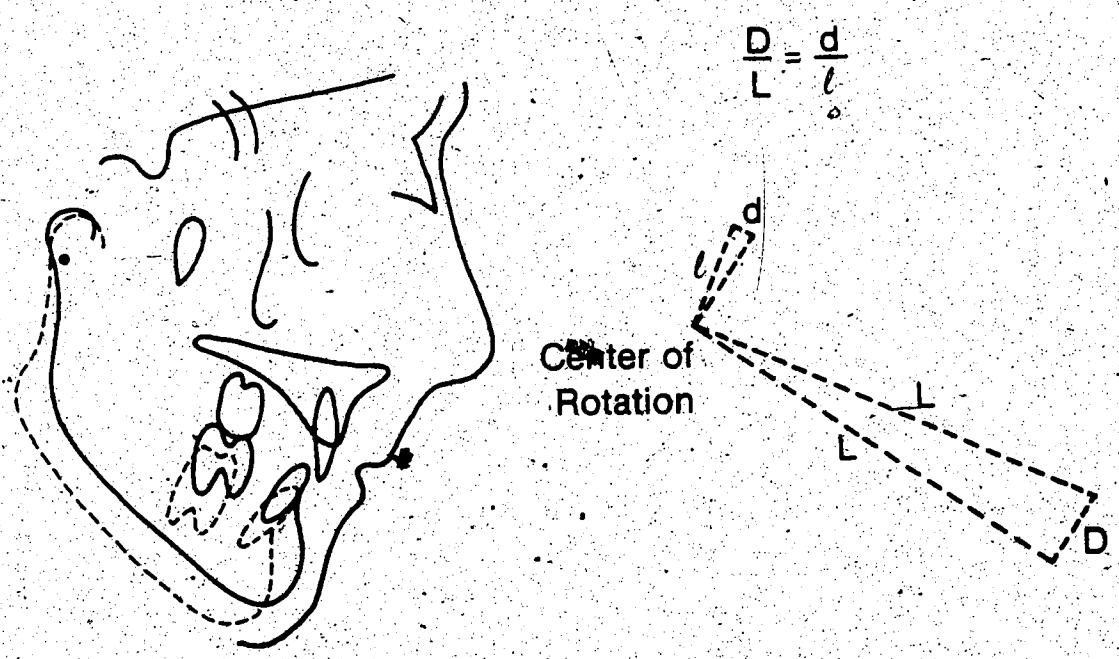


Figure 11. Displacement of the condylar articular surface in hinge axis movement.

significantly with the CO splint during the study period.

The mandible was stabilized at the position of initial insertion by indexing the opposing teeth evenly on the splint. Although pivotal splint therapy did not produce a significant condylar displacement when the group as a whole was analyzed, there were significant changes in condylar position for some individuals as evidenced by the variability. Pivotal splint therapy with orthodontic traction did produce significant inferior and to a lesser extent posterior condyle displacement. The mean inferior displacement was very close to the 1 mm thickness of the shims placed between the articulator components during splint construction. Pure rotation of the mandible around the pivot point introduced by the splint would result in an inferior and slightly posterior condyle displacement (Figure 12).

For rotation to occur around the pivot point a resultant force must be applied anterior to the pivot. In group 3 this force was applied in the form of orthodontic elastics while in group 2, rotation of the mandible is more difficult to explain. All of the elevator muscles attach to the mandible posterior to the pivot point. Using a mathematical model, Osborn[13], found that simulated contraction of the inferior lateral pterygoid and posterior temporalis muscles simultaneously produce a force couple. These non colinear opposing force vectors

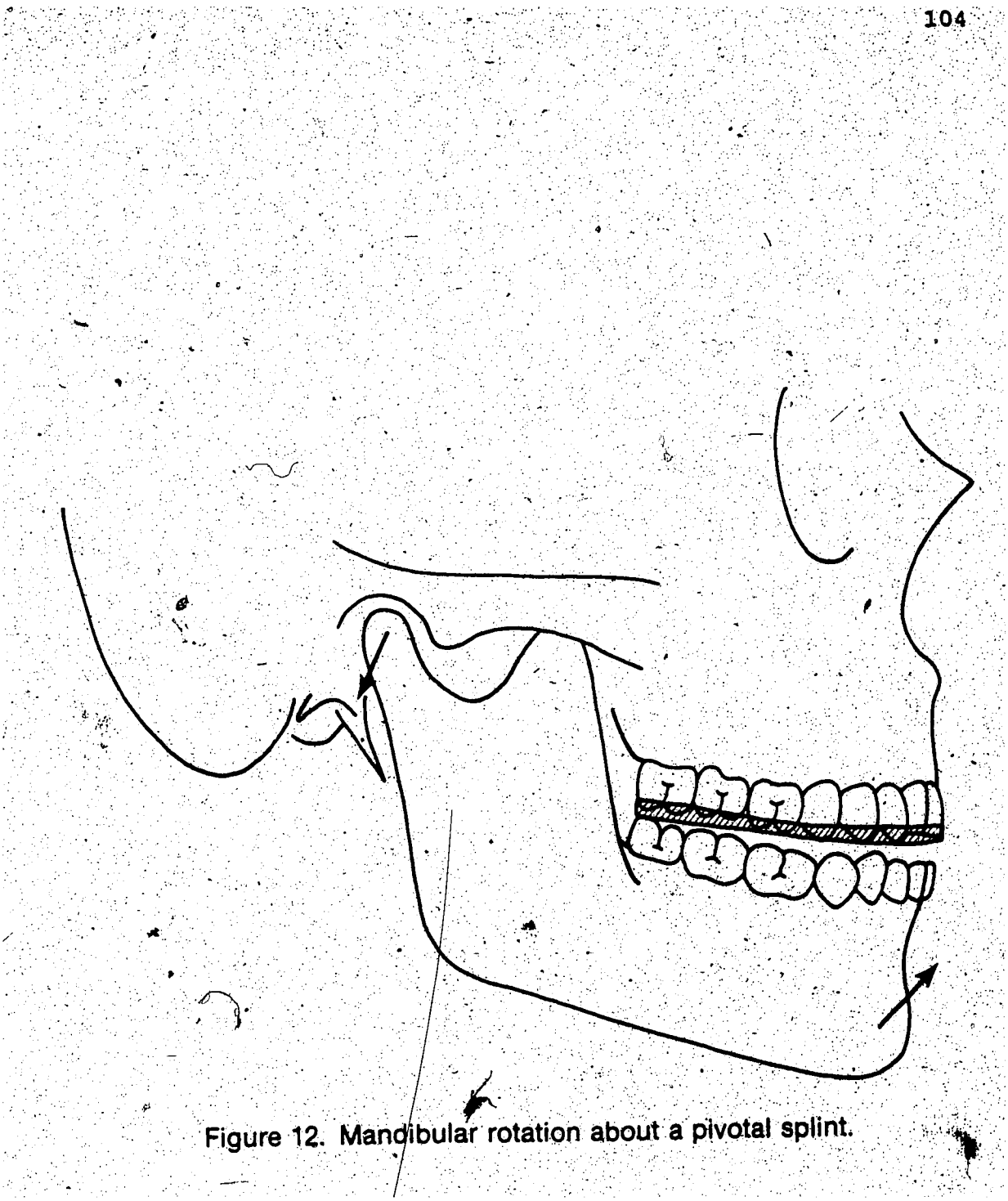


Figure 12. Mandibular rotation about a pivotal splint.

produce mandibular rotation about a point located within the region bordered by the forces. The position of axis of rotation would depend on the magnitude and direction of the resultant forces. (Figure 13)

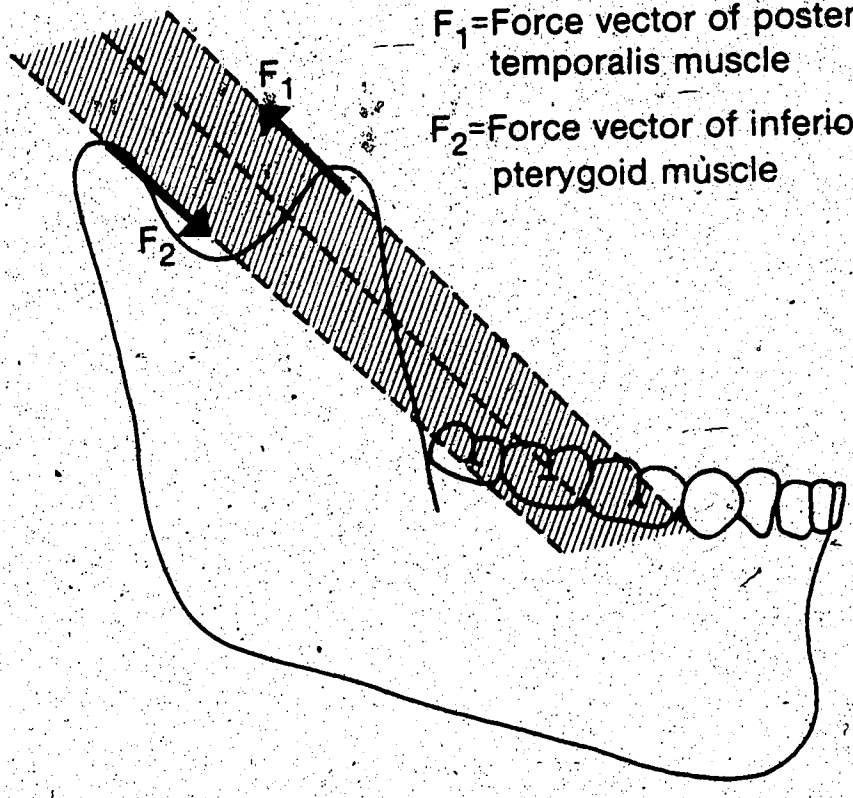


Figure 13. Theoretical model of the force couple produced by the posterior temporalis and inferior lateral pterygoid muscles.

Counterclockwise rotation of the mandible would require the relaxation of the other elevator muscles at the same time as contraction of the inferior lateral pterygoid and posterior temporalis muscles. The anteroposterior position of the splint pivot in relation to overall mandibular morphology is important. All pivotal splints were constructed with the pivot point as posterior as possible. Tradowski et al[201,202] measured mandibular rotation associated with pivot points placed at different anteroposterior locations along the occlusal plane. In patients with an Angles Class I occlusal relationship, a pivot placed behind the mesial one third of the mandibular first molar produced a counterclockwise mandibular rotation. Conversely, a pivot placed ahead of the first molar produced a clockwise rotation. Missing posterior teeth, particularly second and third molars, would reduce the potential for inferior condylar displacement with pivotal splint therapy.

The wide range of inferior condyle displacement noted with the pivotal splints (Group 2) could have several other possible explanations. Many patients with disc displacement without reduction also have myofacial pain dysfunction with myalgia and/or trismus. Spasm in the major elevator muscles such as the masseter and medial pterygoid muscles could inhibit mandibular rotation about the pivot point. Perhaps physical therapy including such treatment modalities as Transcutaneous Electrical Neural

Stimulation (TENS) or ultrasound may promote muscle relaxation and improve the prognosis of achieving inferior condylar displacement. Cephalometric analysis of craniofacial morphology to determine potential muscle force vectors and how they relate to the pivot point, may also provide some prognostic information. Other factors such as the laxity of capsular ligaments, intracapsular adhesions and preexisting condylar displacement may also be important.

Inferior condylar repositioning did not relapse during post therapy centric occlusion recordings. The maintenance of condylar repositioning indicates that condylar displacement is most likely associated with neuromuscular reprogramming. Proprioceptive feedback from the periodontium has been shown to be a significant influence in neuromuscular programming.[203] Although not recorded in the present study it is possible that prolonged removal of the splint could result in a return to the presplint condylar position as the muscles reprogram to achieve maximum intercuspation. Long term maintenance of the condyle in the displaced position using splint therapy may induce remodelling of the articular tissue thereby maintaining this condylar position. The duration of splint wear that would be required to induce this amount of remodelling is unknown. Mongini[152] and Isberg-Holm *et al*[154] reported radiographic evidence of reconstruction and reshaping of degenerative condyles with

long term occlusal splint therapy.

The net change in subjective pain level was calculated. A limitation of this ordinal based subjective assessment is that the patient must start with moderate to severe pain to show a significant change. If the initial pain were only mild the degree of change would be difficult to quantify. Individual perception of pain is only loosely linked to the instigating circumstance and is subject to psychological, social and cultural factors. Despite some obvious limitations, Wolff[124] concluded that magnitude estimation using subjective rating is an useful technique for human pain measurement.

The group of patients treated with a CO splint did not experience a statistically significant mean reduction in pain, while patients treated with pivotal splints experienced significant reduction in subjective pain level ($P < 0.05$) with high variability. The group of patients treated with combined pivotal splint therapy and orthodontic traction had the largest mean reduction in pain level. Pain level decreased significantly more than with the CO splint ($P < 0.000$) but not significantly more than the pivotal splint without orthodontic traction ($P < 0.05$). Although the present study showed that pivotal splint therapy with or without orthodontic traction is significantly more effective at reducing pain levels than a CO splint, the response of an individual cannot be predicted. Some patients did not improve in their

reported pain level. What can be concluded is that the best splint therapy approach to reduce pain levels is a pivotal splint with orthodontic traction.

The etiology of pain may be difficult to localize and in the case of mandibular dysfunction may involve a combination of muscles, joints and soft tissues. Patients with disc displacement without reduction are functioning with the innervated bilaminar zone between the articular surface of the condyle and articular eminence. Degenerative changes of the articular surfaces whether inflammatory or not, could directly result in pain. Myalgia is also a source of pain in many patients with these forms of internal derangement. While there is general agreement that muscle fatigue and spasm resulting from muscle hyperactivity are responsible for the pain and tenderness, the question of the cause remains undefined. Masticatory muscle myalgia may be a secondary protective mechanism to reduce joint movement. Other factors may include occlusal disharmony, emotional stress and cervical dysfunction.

The highly significant correlation ($p = .001$) between inferior condylar displacement and subjectively reported reduced pain levels in patients diagnosed as having disc displacement without reduction or degenerative joint disease, supports the concept that treatment should be aimed at aggressively increasing joint space. It is hypothesized that inferior condylar displacement will

result in elimination of joint loading. This theoretically will reduce pressure on the innervated bilaminar zone and degenerative articular surfaces. Inferior condylar displacement may also be linked to reduced contraction of the elevator muscles, thereby reducing pain resulting from myospasm of these muscles.

The eight week study period was inadequate to be able to draw conclusions about the long term effect on clinical pain. As described by Kopp[125] patients with mandibular dysfunction experience a wide fluctuation of symptoms even without treatment. A follow up to this study should be undertaken to quantify the long term stability of pain reduction. It is hypothesized that the patients will continue to experience improved comfort if properly managed.

Reduced joint loading associated with the measured inferior condylar displacement may not occur during function such as mastication. All that has been measured was that the condyles were displaced at the end of the study with the splint in place and with the mandible closed passively into centric occlusion without the splint.

Beyond the conclusion that inferior condylar displacement of the condyles and therefore reduced joint loading can be accomplished with appropriate splint therapy, little can be concluded regarding the tissue response. If it is accepted that degenerative joint

disease is the result of an imbalance between the forces applied to the joint and its ability to adapt to that stress, a reduction in joint loading by inferior condylar displacement should optimize tissue response.

Potential biologic liabilities associated with the different forms of splint therapy remain unknown. An eight week study period was chosen to minimize the risk of undesired tooth movement. Prolonged use of vertical orthodontic traction would likely result in supereruption of the involved teeth. Prolonged use of a pivotal splint without traction may also result in dental extrusion if mandibular repositioning to achieve total tooth contact did not occur. It is also possible that long term use of the pivotal splint, even once mandibular repositioning has occurred, could result in the intrusion of posterior teeth until the original condylar position is reestablished.

Counterclockwise mandibular rotation around the splint pivot point produced a posterior open bite similar to the amount of inferior condylar displacement. If the change in mandibular position were maintained for a sufficient period of time to become irreversible, the now permanent change in occlusion would need to be addressed. This could be accomplished by selective splint adjustment to allow progressive supereruption of posterior teeth, occlusal adjustment, orthodontic therapy or prosthodontics.

Transcranial projections were chosen over lateral

tomographic techniques to identify changes in bony joint relationships because of their superior reproducibility, clarity and reduced equipment cost. Transcranial projections only represent spatial relations of the lateral one third of the joint which may significantly differ from the medial and central parts. For this reason diagnostic information inferred by joint space analysis has been confined to changes in joint relations. Direct measurement of joint spaces to determine changes in condylar position represent a composite of condylar rotation and bodily displacement. This is evidenced by the significant change in joint space associated with increased vertical dimension.

The highly significant correlation between M.P.I. values and radiographically recorded value for bodily condylar displacement supports the validity of both techniques. Although M.P.I. values are consistent with radiographic values they are less versatile. M.P.I. values can only be determined where there has been no change in occlusal morphology. Condylar displacement recorded in X and Y coordinates is easier to interpret than changes in joint space.

Sample size was a limitation with this study, particularly in the clinical measurement of pain. Wolff[124] suggests that in clinical studies there should be 30 patients per independent variable. Therefore the ideal sample for this study would be 90 patients, which

was not technically feasible with patient availability and time constraints.

VI. CONCLUSIONS:

1. There is a very significant correlation between radiographically determined and articulator (M.P.I.) determined values for condylar displacement in the X and Y coordinates.
2. Condylar position was altered significantly by initial placement of all three types of splints.
3. Changes in condylar position were not significantly different between groups of patients treated with a C.O. splint and patients treated with a pivotal splint.
4. The pivotal splint with orthodontic traction did produce a significantly larger inferior and posterior condylar displacement persisted after removal of the splint.
5. Changes in joint space were not significantly different between groups of patients treated with C.O. splints and patients treated with pivotal splints.
6. Pivotal splint therapy with orthodontic traction did produce reduced posterior joint space and increased superior joint space during the study period which was maintained following splint removal.
7. There was a significant reduction in subjective pain levels using the pivotal splint compared to the C.O.

splint.

8. There was an apparent but not statistically significant reduction in subjective pain levels using the pivotal splint with orthodontic traction compared to the pivotal splint.
9. There was a statistically significant correlation between the reduction in subjective pain level and the amount of inferior condylar displacement.
10. There was not a statistically significant correlation between the reduction in subjective pain level and the amount of horizontal condylar displacement.

Clinical Cautions:

This thesis presents insufficient data to permit an adequate comparison of different forms of splint therapy and to establish a priority for their use. The basic principle of using conservative reversible forms of therapy, whenever possible, should be followed. Centric occlusion splints designed to provide total tooth contact without intentionally repositioning the mandible are considered to be a reversible form of therapy.[205] All teeth not stabilized by the splint or by opposing occlusal contact may be subject to drifting. Twenty four hour splint wear for an extended period of time should be discouraged.

The long term effects of permanently altered condylar position is unknown, and will remain subject to question

until more research is done. Jaw positioning occlusal splints may produce irreversible effects. Although the short term clinical results reported in this study are promising it must be stressed that the long term occlusal, neuromuscular and intracapsular effects are unknown.

Suggestions for further study:

1. Long term followup to determine if changes in condyle position will remain stable.
2. Long term followup to monitor pain levels.
3. Long term followup to monitor and assess remodelling within the joints.
4. Develop alternative means of assisting pivotal splints in mandibular repositioning.
5. EMG studies to assess neuromuscular response to mandibular repositioning.

BIBLIOGRAPHY

1. Roth, R.: Functional occlusion for the orthodontist. J.C.O. 15: No. 1,2,3,4, as reprinted AES Comp., 390, 1981-1982.

2. Williamson, E.H.: Temporomandibular dysfunction in pretreatment adolescent patients. Am.J.Orthod. 72: 429, 1977.

3. Moffett, B.C.: Definitions of temporomandibular joint derangements. In Moffett, B.C. and Westesson, P., (editors): Diagnosis of internal derangements of the temporomandibular joint. Proceeding of a Continuing Dental Education Symposium, Seattle, Feb. 17-18, 1984.

4. Rees, L.A.: The structure and function of the temporomandibular joint. Br.Dent.J. 96: 96, 1954.

5. Bell, W.E.: Understanding temporomandibular biomechanics. J.Craniomand.Pract. 1:27, 1983.

6. Moffett, B.C.: Histologic aspects of emporomandibular joint derangements. In Moffett, B.C., and Westesson, P. (editors). Diagnosis of internal derangements of the temporomandibular joint. Proceedings of a Continuing Dental Education Symposium, Seattle, Feb. 17-18, 1984.

7. Osborn, J.W.: The disc of the human temporomandibular joint: design, function and failure. J.Oral Rehab. 12:279, 1985.

8. Bell, W.E.: Temporomandibular disorders: Classification, diagnosis, management. Year Book Medical Pub. Inc., Chicago, 1982.

9. Moss, M.L.: The functional matrix concept and its relationship to temporomandibular joint dysfunction and treatment. Dent.Clin.N.Amer. 27:445, 1983.

10. Maloney, M.B.: Internal derangements of the temporomandibular joint. I Clinical and radiological diagnosis. Aust.Dent.J. 30:253, 1985.

11. Juniper, R.P.: Temporomandibular joint dysfunction: A theory based on electromyographic studies of the lateral pterygoid muscle. Brit.J.Oral Surg. 22:1, 1984.

12. Juniper, R.P.: The superior pterygoid muscle? Brit. J.Oral Surg. 19:121, 1981.

13. Osborn, J.W.: Predicted pattern of muscle activity during clenching derived from a computer assisted model: symmetric vertical bite forces. J. Biomechanic 18:599, 1985.

14. Farrar, B.: Differentiation of temporomandibular joint dysfunction to simplify treatment. J.Prosthet.Dent. 28:629, 1972.

15. Clark, G.T.: A critical evaluation of orthopedic interocclusal appliance therapy: Effectiveness for specific symptoms. J.A.D.A. 108:364, 1984.

16. Clark, G.T., Beemsterboer, P.L., and Rugh, J.D.: Nocturnal masseter activity and the symptoms of masticatory dysfunction. J.Oral Rehab. 2:279, 1981.

17. Graham, M.M., Buxbaum, J., and Stalling, L.M.: A study of occlusal relationships and the incidence of myofacial pain. J.Prosthet.Dent. 47:549, 1982.

18. Christensen, L.V.: Facial pains and the jaw muscles: A review. J.Oral Rehab. 8:193, 1981.

19. Carraro, J.J., and Caffesse, R.J.: Effect of occlusal splints on TMJ symptomology. J.Prosthet.Dent. 40:563, 1978.

20. Mongini, F.: Condylar remodelling after occlusal therapy. J.Prosthet.Dent. 43:568, 1980.

21. Lupton, D.E., and Johnson, D.L.: Myofacial pain dysfunction syndrome, attitudes, and other personality characteristics related to tolerance for pain. J.Prosthet.Dent. 29:323, 1973.

22. Goldstein, D.F., Kraus, S.L., William, W.B., and Glasheen-Wray, M.: Influence of cervical posture on mandibular movement. J.Prosthet.Dent. 52:421, 1984.

23. Cohen, S.: A cephalometric study of rest positions in edentulous persons: Influence of variations in head position. J.Prosthet.Dent. 7:467, 1957.

24. McLean, L.F., Brenman, H.S., Friedman, M.G.: Effects of changing body position on dental occlusion. J.Dent.Res. 52:1041, 1973.

25. Bratzlavsky, M., and VanderEcken, H.: Postural reflexes in cranial muscles in man. Acta Neurol.Belg. 77:5, 1977.

26. Kendell, H.O., Kendell, F.P., and Boynton, D.A.: Posture and pain. Huntington, N.Y. 1952, Robert E. Krieger Publishing Co. Inc., p 154.

27. Selby, A.: Physiotherapy in the management of temporomandibular disorders. Aust.Dent.J. 30:273, 1985.

28. Agerberg, G., and Carlsson, G.E.: Late results of treatment of functional disorders of the masticatory system. J.Oral Rehab. 1:309, 1974.

29. Magnusson, T., and Carlsson, G.E.: Treatment of patients with functional disturbances of the masticatory system. A survey of 80 consecutive patients. Swed.Dent.J. 2:145, 1980.

30. Solberg, W.K., Clark, G.T., and Rugh, J.D.: Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint treatment. J.Oral Rehab. 2:215, 1975.

31. Clark, G.T., Beemsterboer, P.O., Solberg, W.K., and Rugh, J.D.: Nocturnal electromyographic evaluation of myofacial pain dysfunction in patients undergoing occlusal splint therapy. J.A.D.A. 99:602, 1979.

32. Greene, C.S., and Laskin, D.M.: Long-term evaluation of conservative treatment for myofacial pain dysfunction syndrome. J.A.D.A. 89:1365, 1974.

33. Pomp, A.M.: Psychotherapy for the myofacial pain dysfunction syndrome: A study of factors coinciding with symptom remission. J.A.D.A. 89:629, 1974.

34. Shields, J.M., Clayton, J.A., and Sindledecker, L.D.: Using pantographic tracings to detect TMJ and muscle dysfunctions. J.Prosthet.Dent. 39:80, 1978.

35. Beard, C.C., and Clayton, J.A.: Effects of occlusal splint therapy on TMJ dysfunction. J. Prosthet. Dent. 44:324, 1980.

36. Clayton, J.A.: A pantographic reproducibility index for use in diagnosing temporomandibular joint dysfunction: A report on research. J.Prosthet. Dent 4:827, 1985.

37. Clark, G.T., and Lynn, P.: Horizontal plane jaw movements in controls and clinic patients with temporomandibular dysfunction. J.Prosthet.Dent. 55:730, 1986.

38. Westesson, P., Bronstein, S.L., and Liedberg, J.: Internal derangement of the temporomandibular joint: Morphologic description with correlation to joint function. Oral Surg.Oral Med.Oral Path. 59:323, 1985.

39. Dolwick, M.F., Katzberg, R.W., Helms, C.A.: Internal derangement of the temporomandibular joint: Fact or fiction? J.Prosthet.Dent. 49:415, 1983.

40. Ireland, V.E.: The problem of the "clicking jaw". Proc.R.Soc.Med. 44:191, 1951.

41. Blackwood, H.J.J.: Pathology of the temporomandibular joint. J.A.D.A. 79:118, 1969.

42. Oberg, T., Carlsson, G.E., and Fajers, C.M.: The temporomandibular joint: A morphologic study on human autopsy material. 29:349, 1971.

43. Hansson, T.L., Solberg, W.K., and Pen, M.D.: Temporomandibular joint changes in young adults. J.Dent.Res. 58:267, 1979.

44. Lipton, J.S., Dolwick, M.F., and Warner, M.R.: Sagittal anatomy of human cadaver TMJ. J.Oral Maxillofac.Surg.

45. Farrar, W.B.: Characteristics of the condylar path in internal derangements of the TMJ. J.Prosthet. Dent. 39:319, 1978.

46. Omnell, K., Randow, K., and Oberg, T.: Clicking and temporary "locking" in the temporomandibular joint. Dentomaxillofac Radiol. 5:33, 1976.

47. Wilkes, C.: Arthrography of the temporomandibular joint in patients with TMJ dysfunction syndrome. Minn.Med. 61:645, 1978.

48. Farrar, W.B., and McCarty, W.L.: Inferior joint space arthrography and characteristics of condylar paths in internal derangements of the TMJ. 41:548, 1979.

49. Dolwick, M.F., Katzberg, R.W., Helms, C.A., and Bales, D.J.: Arthrotomographic evaluation of the temporomandibular joint. J.Oral Surg. 39:793, 1979.

50. Blaske, D., Solberg, W.K., and Sanders, B.: Arthrography of the temporomandibular joint: Review of current status. J.A.D.A. 100:388, 1980.

51. Eriksson, L., and Westesson, P.L.: Clinical and radiological study of patients with anterior disc displacement of the temporomandibular joint. Swed.Dent.J. 7:55, 1983.

52. Eriksson, L., Westesson, P.L., and Rohlin, M.: Temporomandibular joint sounds in patients with disc displacement. Int.J.Oral Surg. 14:428, 1985.

53. Isberg-Holm, A.M., and Westesson, P.O.: Movement of the disc and condyle in temporomandibular joints with and without clicking. A high speed cinematographic and disection study on autopsy specimens. Acta.Odontol.Scand. 40:165, 1982.

54. Schwartz, H.G. and Kendrick, R.W.: Internal derangements of the temporomandibular joint: Description of clinical syndromes. Oral Surg. 58:24, 1984.

55. Rohlin, M., Westesson, P.L., and Ericksson, L.: The correlation of temporomandibular joint sounds with morphology in fifty-five autopsy specimens. J.Oral Maxillof.Surg. 43:194, 1985.

56. Scapino, R.P.: Histopathology associated with malposition of the human temporomandibular joint disc. Oral Surg. 55:382, 1983.

57. Moffett, B.: Histologic aspects of temporomandibular joint derangements, in Moffett, B.C. (ed): Diagnosis of Internal Derangements of the Temporomandibular Joint, Vol. 1 Seattle, Univ. of Wash., 1984, p 47.

58. Graham, G.S., Ferraro, N.F., and Simms, D.A.: Perforations of the temporomandibular joint meniscus: Arthrographic, surgical, and clinical findings. J.Oral Maxillofac.Surg. 22:35, 1984.

59. Westesson, P.L.: Structural hard tissue changes in temporomandibular joints with internal derangement. Oral Surg.Oral Med.Oral Path. 59:220, 1985.

60. Farrar, W.B., and McCarty, W.L.: A clinical outline of temporomandibular joint diagnosis and treatment, ed. 9, Montgomery, 1982, Normandie Publications.

61. Westesson, P.O.: Cineradiographic, arthrographic, and high-speed cinematographic analysis of the mechanisms of clicking temporomandibular joints. In Moffett, B.C., and Westesson, P.L.: Diagnosis of Internal Derangements of the Temporomandibular Joint. Proceedings of a Continuing Dental Education Symposium. Seattle, Feb. 17-18, 1984

62. Katzberg, R.W., Keith, D.A., Guralnick, W.C., and Ten Ewick, W.R.: Correlation of condylar mobility and arthrotomography in patients with internal derangement of the temporomandibular joint. Oral Surg. 54:622, 1982.

63. Farrar, W.: Diagnosis and treatment of anterior dislocation of the articular disc. N.Y.J.Dent. 41:348, 1971.

64. Bell, Y.L., and Kirveskari, P.: Treatment of reciprocal clicking of the temporomandibular joint using a mandibular repositioning splint and occlusal adjustment. Proc.Finn.Dent.Soc. 81:251, 1985.

65. Dolwick, F.M., and Riggs, R.R.: Diagnosis and treatment of internal derangements of the temporomandibular joint. Dent.Clin.North.Am. 27:561, 1983.

66. Clark, G.T.: Treatment of jaw clicking with temporomandibular repositioning: Analysis of 25 cases. J.Craniomand.Pract. 2:263, 1984.

67. Tallents, R.H., Katzberg, R.W., Miller, T.L., Manzione, J., Macher, D.J., and Roberts, C.: Arthrographically assisted splint therapy: Painful clicking with a non reducing meniscus. Oral Surg. 61:2, 1985.

68. Manzione, J.V., Tallents, R.H., Katzberg, R.W., Oster, C., and Miller, T.L.: Arthrographically guided splint therapy for recapturing the temporomandibular joint meniscus. Oral Surg. 57:235, 1984.

69. Leopard, P.J.: Anterior dislocation of the temporomandibular disc. Brit.J.Oral Maxillofac. Surg. 22:9, 1984.

70. Howard, J.: Reductive mobilization of the acute closed lock in the temporomandibular joint. In Moffett, B.C., and Westesson, P. (editors): Diagnosis of Internal Derangements of the Temporomandibular Joint. Proceedings of a Continuing Dental Education Symposium, Seattle, Feb. 17-18, 1984.

71. McCarty, Jr., W.L., and Farrar, W.B.: Surgery for internal derangements of the temporomandibular joint. J.Prosthet.Dent. 42:191, 1979.

72. Westesson, P.L.: Clinical and arthrographic findings in patients with TMJ disorders, in Moffett B.C. (ed.): Diagnosis of Internal Derangements of the Temporomandibular Joint, Vol. 1 Seattle, Univ. of Wash. 1984, pp 59-71.

73. Weinberg, L.A.: Practical evaluation of the lateral temporomandibular joint radiograph. J.Prosthet. Dent. 51:676, 1984.

74. Blaschke, D.D., and Blaschke, J.J.: Normal TMJ bony relationships in centric occlusion. J.Dent.Res. 60:98, 1981.

75. Hansson, L.G., Hansson, T.H., and Peterson, A.: A comparison between clinical and radiologic findings in 259 temporomandibular joint patients. J.Prosthet.Dent. 50:89, 1983.

76. Katzberg, R.W., Keith, D.A., Ten Eick, W.R., and Guralnick, W.C.: Internal derangements of the temporomandibular joint: An assessment of condylar position in centric occlusion. J.Prosthet.Dent. 49:250, 1983.

77. Hatcher, D.C., Blom, R.J., and Baker, C.G.: Temporomandibular joint spacial relationships: Osseous and soft tissues. J.Prosthet.Dent. 56:344, 1986.

78. Gardner, D.L.: The nature and causes of osteoarthrosis Brit.Med.J. 286:418, 1983.

79. Enlow, D.H.: The condyle and facial growth. In Sarnat, B.G., and Laskin, D.M., (ed.): The Temporomandibular Joint. Ed. 3, Springfield, Ill. Charles C. Thomas, 1979.

80. Enlow, D.H.: Handbook of Facial Growth, ed. 2. Philadelphia, W.B. Saunders Co., 1982.

81. Meikle, M.C.: Remodelling, in Sarnat, B.C., and Laskin, D.M. (ed): The Temporomandibular Joint, ed. 3, Springfield, Ill., Charles C. Thomas, Publisher, 1979.

82. Mongini, F.: Influence of function on temporomandibular joint remodelling and degenerative disease. Dent.Clin.N.Am. 27:479, 1983.

83. Hansson, T., Oberg, T., Solberg, W.K.: Anatomic study of the TMJ's of young adults. A pilot investigation. J.Prosthet.Dent. 41:556, 1979.

84. Blackwood, H.J.J.: Cellular remodelling in articular tissue. J.Dent.Res. 45:480, 1966.

85. DeBont, L.G., Boering, G., Havinga, P., and Liem, R.S.: Spatial arrangement of collagen fibrils in the articular cartilage of the human condyle: A light microscopic and scanning microscopic study. J. Oral Maxillofac.Surg. 42:306, 1984.

86. Weis, C., and Mirow, S.: An ultrastructure study of the osteoarthritic changes in the articular structure of knees. J.Bone Joint Surg. 54:954, 1972.

87. Toller, P.A., and Wilcox, J.H.: Ultrastructure of the articular surface of the condyle in temporomandibular arthropathy. Oral Surg. 45:232, 1978.

88. Ogus, H.: Degenerative disease of the temporomandibular joint and pain dysfunction syndrome. J.Roy.Soc.Med. 71:748, 1978.

89. Rasmussen, C.: Temporomandibular arthropathy: Clinical radiographic, and therapeutic aspects with an emphasis on diagnosis. Int.J.Oral Surg. 12:365, 1983.

90. Liem, R.S., DeBont, L.G., and Boering, G.: Ultra structure of human healthy and osteorthritic condyles. J.Oral Rehab. 12:541, 1985.

91. DeBont, L.G., Liem, R.S., and Boering, G.: Articular cartilage matrix changes due to osteoarthrosis of the human mandibular condyle. J.Oral Rehab. 12:549, 1985.

92. Richards, L.C., and Witson, D.F.: Histopathology of the mandibular condyle. J.Oral.Path, 14:624, 1984.

93. Toller, P.A.: Ultrastructure of the condylar articular surface in severe mandibular pain dysfunction syndrome. Int.J.Oral Surg. 6:297, 1977.

94. DeBont, L.G., Boering, G., Liem, R.S., and Havinga,P.: Osteoarthritis of the temporomandibular Joint: A light microscopic study of the articular cartilage of the mandibular condyle. J.Oral Maxillofac.Surg. 43:481, 1985.

95. Sokoloff, L.: The Biology of Degenerative Joint Disease. Chicago: The University of Chicago Press, 1969.

96. Muir, H.: Molecular approach to understanding of osteoarthrosis. Am.Rheum.Dis. 36:199, 1977.

97. Meachim, G., and Brook, G.: The pathology of osteoarthritis. In Moskowitz, R.W. et al (Editors): Osteoarthritis Diagnosis and Management, Philadelphia, 1984, W.B. Saunders Co., pp 29-42.

98. Majersjo, C.: Therapeutic and prognostic considerations in TMJ osteoarthrosis: A literature review and a long term study in 11 subjects. J.Craniomand.Prac. 5:70, 1987.

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

100. Hekneby, M.: The load of the temporomandibular joint. Physical calculations and analysis. J.Prosthet. Dent. 31:303, 1974.

101. Hylander, W.L.: An experimental analysis of temporomandibular joint reaction force in Macaques. Am.J.Phys.Anthrop. 51:433, 1979.

102. Standlee, J.P., Caputo, A.A., and Ralph, J.D. The condyle as a stress bearing component of the temporomandibular joint. J.Oral Rehab. 8:391, 1981.

103. Kirkeskari, P., and Alanen, P.: Association between tooth loss and TMJ dysfunction. J.Oral Rehab. 12:189, 1985.

104. Lieberman, E.G., Fuchs, G.C., and Lilos, P.: Mandibular dysfunction in 10-18 year old school children as related to morphologic occlusion. J.Oral Rehab. 12:209, 1985.

105. Richards, L.C., and Brown, T.: Dental attrition and degenerative arthritis of the temporomandibular joint. J.Oral Rehab. 8:293, 1981.

106. Eversole, L.R., Pappas, J.R. and Graham, R.: Dental occlusal wear and degenerative disease of the temporomandibular joint: A correlational study utilizing skeletal material from a contemporary population. J.Oral Rehab. 12:401, 1985.

107. Dehaat, A., Van Steenberghe, D., and Lesaffre, E.: Occlusal relationships and temporomandibular joint dysfunction. Part II: Correlations between occlusal and articular parameters and symptoms of TMJ dysfunction by means of stepwise logistic regression. J.Prosthet.Dent. 55:116, 1986.

108 Ito, T., Gibbs, C.H., Marguelles-Bonnet, R., Lupkiewicz, S.M., Young, H.M., Lundun, H.C., and Mahan, P.E.: Loading on the temporomandibular joints with five occlusal conditions. J. Prosthet. Dent. 56:478, 1986.

109. McNeil, D.J., and Howell, P.G.T.: Computerized kinesiography in the study of mastication in dentate subjects. J.Prosthet.Dent. 55:629, 1986.

110. Mankin, H.J.: The response of articular tissue to mechanical injury. J.Bone Jt.Surg. 64:460, 1982.

111. Radin, E.L., Parker, H.G., Pugh, J.W., Steinberg, R.S. Paul, I.L., and Rose, R.M.: Response of joints to impact loading III. Relationship between microfracture and cartilage degeneration. J.Biomech. 6:51, 1973.

112. McNamara, J.A., and Carlsson, D:S.: Quantitative analysis of temporomandibular joint adaptations to protrusive function. Am.J.Orthod. 6:593, 1979.

113. Hickory, W., and Nanda, R.: Adaptive changes of temporomandibular joint to maxillary protraction in monkeys. J.Dent.Res. Special Issue A, 60:538, 1981.

114. McNamara, J.A.: Functional adaptations of the temporomandibular joint. Dent.Clin.N.Am. 19:459, 1975.

115. Mongini, F.: Anatomic and clinical evaluation of the relationship between the temporomandibular joint and occlusion. J.Prosthet.Dent. 38:439, 1977.

116. Mongini, F.: Remodelling of the mandibular condyle in the adult and its relation to the condition of the dental arches. Acta Anat. 82:437, 1972.

117. Hansson, T., Oberg, T., Carlsson, G.E., and Kopp, S.: Thickness of the soft tissue layers and the articular disc in the temporomandibular joint. Odont.Scand. 35:77, 1977.

118. Hansson, T.L.: Current concepts about the temporomandibular joint. J.Prosthet.Dent. 55:370, 1986.

119. Hansson, T.L., and Oberg, T.: Arthrosis and deviation in form in the temporomandibular joint. Acta Odontol.Scand. 35:167, 1977.

120. Solberg, W.K., Hansson, T.L., and Nordstrom, B.: The temporomandibular joint in young adults at autopsy: A morphologic classification and evaluation. J. Oral Rehab. 12:303, 1961.

121. Berger, H.: Problems and promises of basilar view cephalograms. Angle Ortho. 31:127, 1961.

122. Letzer, G.M., and Kronman, J.H.: A posteroanterior cephalometric evaluation of craniofacial asymmetry. Angle Ortho. 37:205, 1967.

123. Hansson, T.L., Nordstrom, B., and Solberg, W.K.: Histologic analysis of TMJ's during late growth and maturity. J.Dent.Res. 63:1984 (Abs. No. 519).

124. Blackwood, H.J.J.: Arthritis of the mandibular joint. Br.Dent.J. 115:317, 1963.

125. Kopp, S.: Subjective symptoms in temporomandibular joint osteoarthrosis. Acta.Odont.Scand. 35:207, 1977.

126. DeBont, L.G., Liem, R.S., and Boering, G.: Ultrastructure of the articular cartilage of the mandibular condyle: Aging and degeneration. Oral Surg.Oral Med.Oral Path. 60:631, 1985.

127. Silberman, M., and Livine, E.: Age related degenerative changes in the mouse mandibular joint. J.Anta. 129:507, 1979.

128. Ali, S.Y.: New knowledge of osteoarthrosis. J.Clin.Path. 12:191, 1978.

129. Toller, D.A.: Osteoarthrosis of the mandibular condyle. Br.Dent.J. 134:223, 1973.

130. Westesson, P.L., and Rohlin, M.: Internal derangement related to osteoarthrosis in temporomandibular joint autopsy specimens. Oral Surg. 57:17, 1984.

131. Rasmussen, O.C.: Clinical findings during the course of temporomandibular arthropathy. Scand.J.Dent. Res. 89:283, 1981.

132. Anderson, Q.N., and Katzberg, R.W.: Pathologic evaluation of disc dysfunction and osseous abnormalities of the temporomandibular joint. J. Oral Maxillofac.Surg. 43:947, 1985.

133. Heloc, B., and Heloc, L.A.: Characteristics of a group of patients with temporomandibular joint disorders. Com.Dent.Oral.Epidemiol. 3:72, 1975.

134. Marbach, J.J., and Lipton, J.A.: Treatment of patients with TMJ and other facial pains by otolaryngologists. Arch.Otoloaryngol. 108:102, 1982.

135. Majerso, C., and Hollendergh: Radiography of the temporomandibular joint in female patients with TMJ pain or dysfunction. Acta.Radiologia.Diag. 25:169, 1984.

136. Bean, L.R., Omnell, K.A., and Oberg, T.: Comparison between radiographic observations and macroscopic tissue changes in temporomandibular joints. Dentomaxillofac.Radiol. 6:90, 1977.

137. Berrett, A.: Radiography of the temporomandibular joint. Dent.Clin. of N.Am. 27:527, 1983.

138. Kopp, S., and Rockler, B.: Relationship between clinical and radiographic findings in patients with mandibular pain or dysfunction. Acta Radiologica.Diag. 20:465, 1979.

139. Carlsson, G.E., Lundberg, M., Oberg, T., and Welander, U.: The temporomandibular joint. A comparative anatomic and radiologic study. Odontol.Revy 19:171, 1968.

140. Lindvall, A.M., Hekimo, E., Hollander, L., and Carlsson, G.E.: Radiographic examination of the temporomandibular joint. A comparison between radiographic findings and gross microscopic morphologic observation. Dentomaxillofac. Radiol. 5:24, 1976.

141. Rasmussen, O.C.: Longitudinal study of transpharyngeal radiography in temporomandibular arthropathy. Scand.J.Dent.Res. 88:257, 1980.

142. Kopp, S., and Rockler, B.: Variation in interpretation in radiographs of the temporomandibular and hand joints. Dentomaxillofac. Radiol. 7:95, 1978.

143. Kopp, S.: Clinical findings in temporomandibular joint osteoarthrosis. Scand.J.Dent.Res. 85:434, 1977.

144. Majersjc, C., Hollender, L.: TMJ pain and dysfunction: Clinical and radiographic findings in the short and long term. Scand.J.Dent.Res. 92:241, 1984.

145. Carlsson, G.E.: Mandibular dysfunction and temporomandibular joint pathosis. J. Prosthetic Dent 43:658, 1980

146. Poswillo, D.: Experimental investigation of the effects of intra-articular hydrocortisone and high condylectomy on the mandibular condyle. Oral Surg. Oral Med.Oral Path. 30:161, 1970.

147. Toller, P.: Use and misuse of corticosteroids in the treatment of temporomandibular joint pain. Proc.Roy.Soc.Med. 70:461, 1977.

148. Friedman, D.M., and Moore, M.E.: The efficacy of intra-articular steroids in osteoarthrosis: A double blind study. J.Rheumatol. 7:850, 1980.

149. Rasmussen, O.C.: Treatment of temporomandibular arthropathy. Scand.J.Dent.Res. 89:64, 1981.

150. Kopp, S., Wenneberg, B., Haroldson, T., and Carlsson, G.E.: The short-term effect of intra-articular injections of sodium hyaburonate and corticosteroid on temporomandibular joint pain and dysfunction. J.Oral Maxillofac.Surg. 43:429, 1985.

151. Namiki, O., Tohyshina, H., and Morisaki, N.: Therapeutic effect of intra-articular injestion of high molecular weight hyaluronic acid on osteoarthritis of the knee. Int.J.Clin.Pharmacol.Ther.Toxicol 2:501, 1982.

152. Mongini, F.: Condylar remodelling after occlusal therapy. J.Prosthet.Dent. 43: 568, 1980.

153. Weinberg, L.A.: The etiology, diagnosis, and treatment of TMJ dysfunction-pain-syndrome. J.Prosthet. Dent. 43:186, 1980.

154. Isberg-Holm, A., and Ivarsson, R.: A case of hard tissue reconstruction within the temporomandibular joint. Dentomaxillofac. Radiol. 7:113, 1978.

155. Ito, T., Gibbs, H.C., Maguelles-Bonnet, R., Kupkiewicz, S.M., Young, H.M, Lundeen, H.C., and Mahon, P.E.: Loading on temporomandibular joints with five occlusal conditions. J.Prosthet.Dent. 56:478, 1986.

156. Campbell, J.: Extenuation of the temporomandibular joint space by methods derived from general orthopedic procedures. J.Prosthet.Dent. 7:386, 1957.

157. Brown, K.E.: Dynamic opening device for mandibular trismus. J.Prosthet.Dent. 20:438, 1968.

158. Sears, V.H.: Occlusal pivotal pivots. J.Prosthet. Dent. 6:332, 1956.

159. Berry, D.C.: Occlusal pivots. A case report. Dent. Pract. 12:337, 1962.

160. Lous, I.: Treatment of TMJ syndrome by pivots. J.Prosthet.Dent.

161. Majersjo, C., and Carlsson, G.E.: Analysis of factors influencing the long term effect of treatment of TMJ-pain dysfunction. J.Oral Rehab. 11:289, 1984.

162. Poswillo, D.E.: Conservative management of degenerative joint disease in the elderly. Int.Dent.J. 33:325, 1983.

163. Hansson, L.G., Peterson, A., and Vallon-Christersson, D.V.: A clinical and radiologic six year follow up study of patients with crepitation of the temporomandibular joint. Swed.Dent.J. 8:277, 1984.

164. Greene, C.S., and Markovic, M.A.: Response to non-surgical treatment of patients with positive radiographic findings in the temporomandibular joint. J. Oral Surg. 34:692, 1976

165. Yoshamira, Y., Yoshida, Y., Oka, M., Miyoshi, M. and Uemura, S.: Long term evaluation of nonsurgical treatment of osteoarthrosis of the temporomandibular joint. Int.J.Oral Surg. 11:7, 1982.

166. Carlsson, G.E., Kopp, S., and Lindrom, J.: Surgical treatment of temporomandibular joint disorders. Swed.Dent.J. 5:41, 1981.

167. Agerberg, G, and Lundberg, M.: Changes in the temporomandibular joint after surgical treatment. A radiographic follow up study. Oral Surg. 32:865, 1971.

168. Weinberg, S.: Eminectomy and meniscorrhaphy for internal derangements of the temporomandibular joint. Oral Surg. 57:241, 1984.

169. Eriksson, L., and Westesson, P.L.: Diskectomy in the treatment of anterior disc displacement of the temporomandibular joint. A clinical and radiologic one year follow up study. 55:106, 1986.

170. Eriksson, L., and Westesson, P.L.: Long-term evaluation of menisectomy of the temporomandibular joint. J.Oral Maxillofac.Surg. 43:263, 1985.

171. Madsen, B.: Normal variations in anatomy, movements, and arthrosis frequency of the temporomandibular joints. Acta.Radiol. 4:272, 1966.

172. Updegrave, W.J.: Roentgenographic observations of the functioning temporomandibular joints. J.A.D.A. 54:488, 1957.

173. Lindbroom, G.: On the anatomy and function of the temporomandibular joint. Acta.Odont.Scand. 17 Suppl. #28.

174. Weinberg, L.A.: An evaluation of the Duplicability of the temporomandibular joint radiographs. J.Prosthet.Dent. 24:512, 1970.

175. Weinberg, L.A.: Technique for temporomandibular joint radiographs. J.Prosthet.Dent. 28:284, 1972.

176. Weinberg, L.A.: What we really see in a TMJ radiograph. J.Prosthet.Dent. 30: 898, 1973.

177. Bergarstadt, H., and Wictorin, L.: The influence of angle of projection on the linear error in craniolateral radiography of the temporomandibular joint. Acta Odontolgica Scand. 29:3, 1970.

178. Eckerdal, O., and Lundberg, M.: Periodic roentgenography of the temporomandibular joint. Dentomaxillofac. Radiol. 4:4, 1975.

179. Aquilino, S.A., Matteson, S.R., Holland, G.A., and Philips, C.: Evaluation of condylar position from temporomandibular joint radiographs. J.Prosthet. Dent. 53:88, 1985.

180. Kundert, M.: Limits of perceptibility of condyle displacements on temporomandibular joint radiographs. J.Oral Rehab. 6:375, 1979.

181. Rosenberg, H.M.: Laminography: method and application in oral diagnosis. J.A.D.A. 74:88, 1967.

182. Ricketts, R.M.: Variations of the temporomandibular joint as revealed by cephalometric laminography. Am.J.Orthodont. 36:877, 1950.

183. Ricketts, R.M.: Laminography in diagnosis of temporomandibular joint disorders. J.A.D.A. 620, 1953.

184. Eckerdal, O.: Tomography of the temporomandibular joints: Correlation between tomographic image and histologic sections in a three dimensional system. Acta Radiologica Suppl. #324, 1973.

185. Yale, S.H.: Radiographic evaluation of the temporomandibular joint. J.A.D.A. 79:102, 1969.

186. Preti, G., Bruscagin, C., and Fava, C.: Anatomic and statistical study to determine the inclination of the condylar long axis. J.Prosthet.Dent. 49:572, 1983.

187. Taylor, R., Ware, W., Fowler, D., and Kobayashi, J.: A study of temporomandibular joint morphology and it's relation to the dentition. Oral Surg. 33:1002, 1972.

188. Updegrave, W.J.: Radiology of the temporomandibular joints individualized and simplified. Contin.Ed. Art. #2 Vol IV, No. 1 Jan-Feb. 1983, p 23.

189. Rozencweig, D., and Martin, G.: Selective tomography of the TMJ and myofacial pain-dysfunction syndrome. J.Prosthet.Dent. 40:67, 1978.

190. Preti, G., Fava, C., and Pera, P.: Accuracy and reproducibility of a new procedure for temporomandibular joint radiography. J.Prosthet. Dent. 49:715, 1983.

191. Beckwith, P.J., Monfort, D.R., and Williams, B.J.: Accurate depth of cut in temporomandibular joint laminographs. Angle Orthod. 50:16, 1980.

192. Lysell, L., and Peterson, A.: The submento-vertex projection in radiography of the temporomandibular joint. Dentomaxillofac. Radiol. 9:11, 1980.

193. Omnell, K., and Peterson, A.: Radiology of the temporomndibular joint utilizing oblique transcranial projections. Odont.Revy. 22:77, 1976.

194. Eckerdal, O., and Lundberg, M.: Temporomandibular joint relations as revealed by conventional radiographic techniques. Dentomaxillofac. Radiol. 8:65, 1979.

195. Larheim, T.A., and Tveito, L.: Reproducibility of temporomandibular joint radiographs using oblique lateral transcranial projection and lateral tomographic technique. Dentomaxillofac. Radiol. 9:85, 1980.

196. Fairovich, G., and Omnell, K.: Axial tomography of the temporomandibular joint using hypocycloidal movement of the tube and film. Dentomaxillofac. Radiol. 6:7, 1977.

197. Rosencweiz, D.: Three dimensional tomographic study of the temporomandibular articulation. J.Periodontol. 46:348, 1975.

198. Gushing, A.C.: Frontal tomography of articulating temporomandibular joint surfaces. Angle Orthod. 53:234, 1983.

199. Helkimo, M.: Studies on function and dysfunction of the masticatory system. II Index for anamnestic and clinical dysfunction and occlusal state. Swed.Dent.J. 67:101, 1974.

200. Brewka, R.E.: Pantographic evaluation of cephalometric hinge axis. Am.J.Orthod. 79:1, 1981.

201. Tradowsky, M., Kubicek, W.F.: Method for determining the equilibrium point of the mandible: J.Prosthet.Dent. 45: 558, 1981.

202. Tradowsky, M., Dworkin, J.B.: Determination of the physiologic equilibrium point of the mandible by electronic means. J.Prosthet.Dent. 48: 89, 1982.

203. Serrano, P.T., Nicholls, J.I., Yuodelis, R.A.: Centric relation change during therapy with corrective occlusion prosthesis. J.Prosthet.Dent. 51: 97, 1984.

204. Wolf, B.B.: Measurement of human pain. In Pain, edited by Bonica, J., Raven Press, New York, 1980.

205. Griffiths, R.H.: Report of the President's Conference on the Examination, Diagnosis, and Management of Temporomandibular Disorders. J.A.D.A. 106: 75, 1983.

University of Alberta
Edmonton
Canada T6G 2N8

Appendix 'A'
Faculty of Dentistry
Office of the Dean

3036 Dentistry/Pharmacy Centre, Telephone (403) 432-3312

APPROVAL

FOR

PROPOSAL ON HUMAN RESEARCH

This is to certify that Dr. Paul Major, of the Division of Orthodontics, presented a proposal for a research study "to evaluate the ability of occlusal splint therapy to increase temporomandibular joint space." The Human Ethics Committee is satisfied that the ethical criteria for human research have been met.

August 13, 1986

Human Ethics Committee

Dr. D.M. Collinson, Chairman

Dr. J.A. Hargreaves, Director,
Graduate Studies and Research

Dr. R.L. Ellis, Chairman,
Department of Dental Health Care

Dr. J.H. Placash, Chairman,
Division of Diagnosis

Appendix 'B'
CONSENT FORM

We are conducting a study to evaluate the effectiveness of splint therapy in the treatment of arthritis of the jaw joint (TMJ). All procedures in this study are normal treatment protocol for people like you, who have a diagnosis of arthritis. The difference between your management and those who do not participate in this study is the standardization of records to allow for accurate analysis of treatment results.

The length of the study is eight weeks and will involve taking three sets of dental impressions, several x-rays, and wearing the splint 24 hours a day as instructed for the duration of the study. After the study period you will continue to be evaluated and treated by the TMJ Investigation Unit. Information gained by the study will contribute to our knowledge of TMJ treatment and may be of direct benefit to you.

Your identity as a patient will be kept in strictest confidence. No information which could potentially identify you will be used in reporting or discussing the results of this study. You are under no obligation to comply with this study and are free to discontinue your participation at any time.

We shall be happy to answer any questions or concerns you may have regarding this study. Please contact Dr. Paul Major (432-3065) for further information.

Thank you for your interest and cooperation in this study.

Investigators: Drs. P. Major, D. Hatcher, K. Compton, K. Glover, W. Lobb.

After having had the above study explained by one of the investigators and having read the above letter, I hereby consent to participate in this study to evaluate the effectiveness of occlusal splint therapy.

_____
Signature of Patient

_____ _____
Witness Date

TEMPOROMANDIBULAR JOINT INVESTIGATION UNIT

NEW PATIENT INFORMATION

PATIENT'S LAST name: _____ Occupation: _____

FIRST name: _____

Date of Birth: ___/___/___

Hospital I.D.# _____

Alberta Health Care # _____ Sex: _____ (1=female, 2=male

Address: _____

_____

_____ Postal Code: _____

Phone: _____ (Work) _____ (Home)

Date of Examination: ___/___/___

Referred by: _____

Address: _____

_____

_____

GENERAL SYMPTOMS

1. Your problem is in your: (0=No, 1=right, 2=left, 3=both)

Ear	_____	Back of Head	_____
Jaw Joint	_____	Jaw	_____
Face	_____	Teeth	_____
Eye	_____	Neck	_____

2. What date did your problem start? ___/___ (Month/Year)
3. _____ Which side is worse? (1=right, 2=left, 3=both)
4. _____ Would you call it: (1=pain, 2=uncomfortable, 3=suffering)
5. _____ Does it hurt (1) or is it just uncomfortable (2)?
   0=No, 1=Yes
6. _____ Does the pain or discomfort disturb your sleep?
7. _____ Does the pain or discomfort interfere with daily activities?
8. _____ Is there constant or recurring pain?
9. _____ Is the pain a burning type pain?
10. _____ Is the pain a dull, aching pain?
11. _____ Is the pain a stabbing, severe pain?

COMMENTS:

_____

_____

_____

12. _____ On a scale of 1 to 10 (where 1=no pain and 10=severe pain), what is your current pain level?

0=No, 1=Yes

13. _____ Can you locate a specific site of pain?
14. _____ Has the severity of pain been decreasing?
15. _____ Has the pain severity been increasing?
16. _____ Does it hurt to open your mouth?
17. _____ Does it hurt to chew?

Identify as: (1=right, 2=left, 3=both, 4=neither)

18. _____ Do you hear joint sounds?
19. _____ Do you have any clicking or popping joint sounds?
20. _____ Do you have any grating or grinding type joint sounds?
21. _____ Did your joints have sounds in the past that have now stopped?
22. _____ Do you have difficulty opening your mouth?

0=No, 1=Yes

23. _____ Are your jaws tired after eating a meal?
24. _____ Do you have headaches?
25. _____ Is the condition worse (1=mornings, 2=day, 3=evening, 4=during sleep, 5=after eating, 6=after talking)?
26. _____ Do you prefer to chew (1=right, 2=left, 3=both) sides?
27. _____ Do you prefer to chew on (1=front teeth, 2=back teeth)?
28. _____ Do you chew exclusively on (1=right, 2=left, 3=both) sides?
29. _____ Do you do the following with your teeth? (1=grind, 2=clench, 3=both, 4=not sure)
30. Do you have (0=No, 1=Yes)?
    - _____ Neck pain
    - _____ Shoulder pain
    - _____ Back pain
31. Do you notice (0=No, 1=Yes)?
    - _____ Hearing Loss
    - _____ pain in teeth
    - _____ Headaches
    - _____ stuffiness, plugged ears
    - _____ Ringing in Ears
    - _____ Dizziness
32. _____ Are you currently taking medications for this problem (0=No, 1=Yes)?
33. _____ What type of medications are you taking? (1=painkillers, 2=muscle relaxants, 3=tranquilizers, 4=more than one type, 5=not sure)
35. _____ Would you consider your lifestyle stressful (0=No, 1=Yes)?

COMMENTS

_____

_____

MEDICAL HISTORY

0=No, 1=Yes

1. _____ Have you had a jaw injury?
2. _____ Have you had a whiplash?
3. _____ Was there a strain or stretching of the jaw such as yawning, during dental procedure, while chewing or opening mouth wide?
4. _____ Have you ever had a "nervous stomach" or "ulcers"?
5. _____ Have you ever had digestive problems or colitis?
6. _____ Do you take any medication?
7. _____ Have you had rheumatic fever?
8. _____ Do you have a heart murmur or leaky heart valve?
9. _____ Do you have allergies to any medications?
10. _____ Do you have high blood pressure?
11. _____ Have you ever had hepatitis?
12. _____ Have you had a recent cold?
13. _____ Have you had a recent tooth infection?
14. _____ Have you had a recent throat infection?
15. _____ Have you had a recent ear infection?
16. _____ Do you get migraine headaches?
17. _____ Have you been diagnosed as having Meniere's disease?
18. _____ Have you ever had a general anesthesia?
19. _____ How many times have you had a general anesthesia?
20. _____ Have you ever had a skull surgery?
21. _____ Have you had the surgical placement of metal implants?
22. _____ Are you currently having problems with any other joints in your body?
23. _____ Have you been diagnosed as having rheumatoid arthritis?

Females

1. _____ Are you pregnant?
2. _____ Are currently planning to get pregnant?
3. _____ Are you taking birth control pills?
4. _____ Are you taking any hormones or glandular medication?

Comments

_____

_____

_____

_____

DENTAL HISTORY

0=No, 1=Yes

1. _____ Do you have sore teeth?
2. _____ Do you have badly worn teeth?
3. _____ Are any of your teeth very loose?
4. _____ Have you had teeth extracted?
5. _____ Have you had wisdom teeth extracted?
6. _____ Have you had recent fillings?
7. _____ Have you had recent crowns or bridges place in your mouth?
8. _____ Have you had your teeth straightened or had braces?
9. _____ When were your teeth straightened? Date: _____
10. _____ Have you had your jaw joints x-rayed?
11. _____ Have you had previous jaw joint surgery?
12. _____ Have you had any splint treatment for your jaw joint?
13. _____ Have any of the past treatments for your jaw joints helped?
14. _____ Is there a history of jaw joint problems in your family?
15. _____ Have you had a splint made in the last 12 months?

Comments

_____

_____

_____

_____

TEMPOROMANDIBULAR JOINT INVESTIGATION UNIT

Date _____ OBJECTIVE EXAMINATION PATIENT NAME: _____

MUSCLE TENDERNESS

		Right	Left	(0-3 severity)
1. Temporalis	- Ant.			
	- Post.			
	- Tendon			
2. Masseter	- Sup.			
	- Deep			
3. Sternocleidomastoid				
4. Post. Cervical				
5. Trapezius				
6. Lat. Pterygoid				
7. Med. Pterygoid				

JOINT TENDERNESS

	Right	Left
1. Lat. Palpation		
2. Post. Palpation		

COMMENTS

_____

_____

_____

_____

_____

MANDIBULAR MOVEMENTS

	Right (mm)	Left (mm)	
Vertical Opening			Deviation _____ (1=R, 2=L)
Lateral			
Protrusion			

JOINT SOUNDS

	Right	Left (0=No, 1=Yes)
Click		
Crepitus		
Thud		

JOINT SOUNDS (Con't)

If click then timing (Measure incisal opening)

Right _____ mm.

Left _____ mm.

	Right	Left (0=No, 1=Yes)
Reducable	_____	_____

COMMENTS

_____

_____

_____

_____

_____

OCCLUSION

Angle's Class _____ (Class I=1, II-Div.1=2, II-Div.2=3, Class III=4)

X-bite _____ (0=No, 1=Right, 2=Left)

Open-bite _____ (0=No, 1=Yes)

Incisal Relationship

Overbite _____ mm.

Overjet _____ mm.

Adequate vertical diminsion of occlusion (0=No, 1=Yes) _____

Adequate Tooth Support (0=No, 1=Yes)

_____ overclosed _____ mm.

_____ opened _____ mm.

OCCLUSAL ASSESSMENT

COMMENTS

_____

_____

_____

_____

_____

- 3 -

CLINICAL DIAGNOSIS (Without X-rays)

0=No, 1=Yes		Right	Left
1. Myofacial Pain			
2. Reciprocal Click			
3. Closed Lock	Chronic		
	Acute		
4. Hypomobility			
5. Hypermobility			
6. Condylar Hypoplasia			
7. Condylar Hyperplasia			
8. Facial Asymmetry			
9. Degenerative Joint Disease			

COMMENTS

FINAL DIAGNOSIS (With X-rays)

		Right	Left
1. Myofacial Pain			
2. Reciprocal Click			
3. Closed Lock	Chronic		
	Acute		
4. Hypomobility			
5. Hypermobility			
6. Condylar Hypoplasia			
7. Condylar Hyperplasia			
8. Facial Asymmetry			
9. Degenerative Joint Disease			

COMMENTS

TREATMENT PLAN

Splint Therapy (0=No, 1=Yes)

1. Flat Plane ____
2. Ant. Repositioning ____
3. Off Loading ____

Physiotherapy ____

Medications ____

Consults - surgery ____
- x-rays ____
- arthrogram ____
- other ____

COMMENTS

OBJECTIVES OF THERAPY

RECALL SCHEDULE

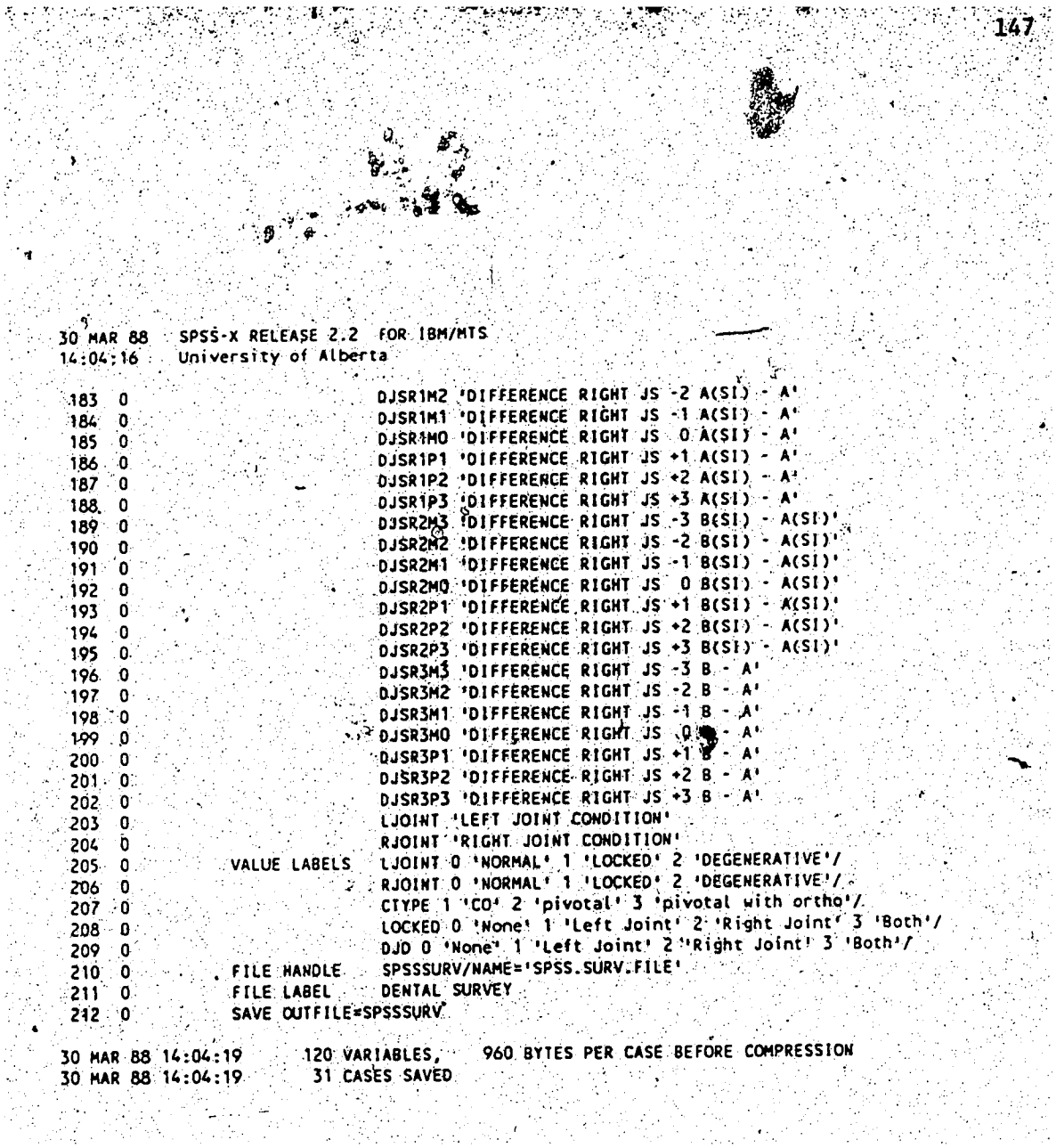
Appendix C

```
30 MAR 88   SPSS-X RELEASE 2.2  FOR IBM/MTS
14:04:16    University of Alberta

  75  0          COMPUTE         DJSR3P1=JSRBP1-JSRAP1
  76  0          COMPUTE         DJSR3P2=JSRBP2-JSRAP2
  77  0          COMPUTE         DJSR3P3=JSRBP3-JSRAP3
  78  0          COMPUTE         LJOINT=0
  79  0          IF              (LOCKED EQ 1 OR LOCKED EQ 3)LJOINT=1
  80  0          IF              (DJD EQ 1 OR DJD EQ 3)LJOINT=2
  81  0          COMPUTE         RJOINT=0
  82  0          IF              (LOCKED EQ 2 OR LOCKED EQ 3)RJOINT=1
  83  0          IF              (DJD EQ 2 OR DJD EQ 3)RJOINT=2
  84  0          VARIABLE LABELS
  85  0                          CTYPE 'CASE TYPE -CO, PIVOTAL, PIVOTAL WITH ORTHO'
  86  0                          MRX 'MODELS - RIGHT X COORDINATE'
  87  0                          MRY 'MODELS - RIGHT Y COORDINATE'
  88  0                          MLX 'MODELS - LEFT X COORDINATE'
  89  0                          MLY 'MODELS - LEFT Y COORDINATE'
  90  0                          PCHANGE 'PAIN DECREASE'
  91  0                          LOCKED 'JOINT LOCKED'
  92  0                          DJD 'DEGENERATIVE JOINT DISEASE PRESENT'
  93  0                          JSLAM3 'JOINT SPACE LEFT A -3 READING'
  94  0                          JSLAM2 'JOINT SPACE LEFT A -2 READING'
  95  0                          JSLAM1 'JOINT SPACE LEFT A -1 READING'
  96  0                          JSLAM0 'JOINT SPACE LEFT A  0 READING'
  97  0                          JSLAP1 'JOINT SPACE LEFT A +1 READING'
  98  0                          JSLAP2 'JOINT SPACE LEFT A +2 READING'
  99  0                          JSLAP3 'JOINT SPACE LEFT A +3 READING'
 100  0                          JSLASIM3 'JOINT SPACE LEFT SI A -3 READING'
 101  0                          JSLASIM2 'JOINT SPACE LEFT SI A -2 READING'
 102  0                          JSLASIM1 'JOINT SPACE LEFT SI A -1 READING'
 103  0                          JSLASIM0 'JOINT SPACE LEFT SI A  0 READING'
 104  0                          JSLASIP1 'JOINT SPACE LEFT SI A +1 READING'
 105  0                          JSLASIP2 'JOINT SPACE LEFT SI A +2 READING'
 106  0                          JSLASIP3 'JOINT SPACE LEFT SI A +3 READING'
 107  0                          JSLBM3 'JOINT SPACE LEFT B -3 READING'
 108  0                          JSLBM2 'JOINT SPACE LEFT B -2 READING'
 109  0                          JSLBM1 'JOINT SPACE LEFT B -1 READING'
 110  0                          JSLBM0 'JOINT SPACE LEFT B  0 READING'
 111  0                          JSLBP1 'JOINT SPACE LEFT B +1 READING'
 112  0                          JSLBP2 'JOINT SPACE LEFT B +2 READING'
 113  0                          JSLBP3 'JOINT SPACE LEFT B +3 READING'
 114  0                          JSLBSIM3 'JOINT SPACE LEFT SI B -3 READING'
 115  0                          JSLBSIM2 'JOINT SPACE LEFT SI B -2 READING'
 116  0                          JSLBSIM1 'JOINT SPACE LEFT SI B -1 READING'
 117  0                          JSLBSIM0 'JOINT SPACE LEFT SI B  0 READING'
 118  0                          JSLBSIP1 'JOINT SPACE LEFT SI B +1 READING'
 119  0                          JSLBSIP2 'JOINT SPACE LEFT SI B +2 READING'
 120  0                          JSLBSIP3 'JOINT SPACE LEFT SI B +3 READING'
 121  0                          JSRAM3 'JOINT SPACE RIGHT A -3 READING'
 122  0                          JSRAM2 'JOINT SPACE RIGHT A -2 READING'
 123  0                          JSRAM1 'JOINT SPACE RIGHT A -1 READING'
 124  0                          JSRAM0 'JOINT SPACE RIGHT A  0 READING'
 125  0                          JSRAP1 'JOINT SPACE RIGHT A +1 READING'
 126  0                          JSRAP2 'JOINT SPACE RIGHT A +2 READING'
 127  0                          JSRAP3 'JOINT SPACE RIGHT A +3 READING'
 128  0                          JSRASIM3 'JOINT SPACE RIGHT SI A -3 READING'
```

30 MAR 88 SPSS-X RELEASE 2.2 FOR IBM/MTS
14:04:16 University of Alberta

```
  129  0                    JSRASIM2 'JOINT SPACE RIGHT SI A -2 READING'
  130  0                    JSRASIM1 'JOINT SPACE RIGHT SI A -1 READING'
  131  0                    JSRASIM0 'JOINT SPACE RIGHT SI A  0 READING'
  132  0                    JSRASIP1 'JOINT SPACE RIGHT SI A +1 READING'
  133  0                    JSRASIP2 'JOINT SPACE RIGHT SI A +2 READING'
  134  0                    JSRASIP3 'JOINT SPACE RIGHT SI A +3 READING'
  135  0                    JSRBM3 'JOINT SPACE RIGHT B -3 READING'
  136  0                    JSRBM2 'JOINT SPACE RIGHT B -2 READING'
  137  0                    JSRBM1 'JOINT SPACE RIGHT B -1 READING'
  138  0                    JSRBM0 'JOINT SPACE RIGHT B  0 READING'
  139  0                    JSRBP1 'JOINT SPACE RIGHT B +1 READING'
  140  0                    JSRBP2 'JOINT SPACE RIGHT B +2 READING'
  141  0                    JSRBP3 'JOINT SPACE RIGHT B +3 READING'
  142  0                    JSRBSIM3 'JOINT SPACE RIGHT SI B -3 READING'
  143  0                    JSRBSIM2 'JOINT SPACE RIGHT SI B -2 READING'
  144  0                    JSRBSIM1 'JOINT SPACE RIGHT SI B -1 READING'
  145  0                    JSRBSIM0 'JOINT SPACE RIGHT SI B  0 READING'
  146  0                    JSRBSIP1 'JOINT SPACE RIGHT SI B +1 READING'
  147  0                    JSRBSIP2 'JOINT SPACE RIGHT SI B +2 READING'
  148  0                    JSRBSIP3 'JOINT SPACE RIGHT SI B +3 READING'
  149  0                    LASIAX 'LEFT A(SI) - A X COORDINATE'
  150  0                    LASIAY 'LEFT A(SI) - A Y COORDINATE'
  151  0                    LASIBSIX 'LEFT A(SI) - B(SI) X COORDINATE'
  152  0                    LASIBSIY 'LEFT A(SI) - B(SI) Y COORDINATE'
  153  0                    LBAX 'LEFT B - A X COORDINATE'
  154  0                    LBAY 'LEFT B - A Y COORDINATE'
  155  0                    RASIAX 'RIGHT A(SI) - A X COORDINATE'
  156  0                    RASIAY 'RIGHT A(SI) - A Y COORDINATE'
  157  0                    RASIBSIX 'RIGHT A(SI) - B(SI) X COORDINATE'
  158  0                    RASIBSIY 'RIGHT A(SI) - B(SI) Y COORDINATE'
  159  0                    RBAX 'RIGHT B - A X COORDINATE'
  160  0                    RBAY 'RIGHT B - A Y COORDINATE'
  161  0                    DJSL1M3 'DIFFERENCE LEFT JS -3 A(SI) - A'
  162  0                    DJSL1M2 'DIFFERENCE LEFT JS -2 A(SI) - A'
  163  0                    DJSL1M1 'DIFFERENCE LEFT JS -1 A(SI) - A'
  164  0                    DJSL1M0 'DIFFERENCE LEFT JS  0 A(SI) - A'
  165  0                    DJSL1P1 'DIFFERENCE LEFT JS +1 A(SI) - A'
  166  0                    DJSL1P2 'DIFFERENCE LEFT JS +2 A(SI) - A'
  167  0                    DJSL1P3 'DIFFERENCE LEFT JS +3 A(SI) - A'
  168  0                    DJSL2M3 'DIFFERENCE LEFT JS -3 B(SI) - A(SI)'
  169  0                    DJSL2M2 'DIFFERENCE LEFT JS -2 B(SI) - A(SI)'
  170  0                    DJSL2M1 'DIFFERENCE LEFT JS -1 B(SI) - A(SI)'
  171  0                    DJSL2M0 'DIFFERENCE LEFT JS  0 B(SI) - A(SI)'
  172  0                    DJSL2P1 'DIFFERENCE LEFT JS +1 B(SI) - A(SI)'
  173  0                    DJSL2P2 'DIFFERENCE LEFT JS +2 B(SI) - A(SI)'
  174  0                    DJSL2P3 'DIFFERENCE LEFT JS +3 B(SI) - A(SI)'
  175  0                    DJSL3M3 'DIFFERENCE LEFT JS -3 B - A'
  176  0                    DJSL3M2 'DIFFERENCE LEFT JS -2 B - A'
  177  0                    DJSL3M1 'DIFFERENCE LEFT JS -1 B - A'
  178  0                    DJSL3M0 'DIFFERENCE LEFT JS  0 B - A'
  179  0                    DJSL3P1 'DIFFERENCE LEFT JS +1 B - A'
  180  0                    DJSL3P2 'DIFFERENCE LEFT JS +2 B - A'
  181  0                    DJSL3P3 'DIFFERENCE LEFT JS +3 B - A'
  182  0                    DJSR1M3 'DIFFERENCE RIGHT JS -3 A(SI) - A'
```



```
30 MAR 88   SPSS-X RELEASE 2.2  FOR IBM/MTS
14:04:16    University of Alberta

 183  0                        DJSR1M2 'DIFFERENCE RIGHT JS -2 A(SI) - A'
 184  0                        DJSR1M1 'DIFFERENCE RIGHT JS -1 A(SI) - A'
 185  0                        DJSR1M0 'DIFFERENCE RIGHT JS  0 A(SI) - A'
 186  0                        DJSR1P1 'DIFFERENCE RIGHT JS +1 A(SI) - A'
 187  0                        DJSR1P2 'DIFFERENCE RIGHT JS +2 A(SI) - A'
 188  0                        DJSR1P3 'DIFFERENCE RIGHT JS +3 A(SI) - A'
 189  0                        DJSR2M3 'DIFFERENCE RIGHT JS -3 B(SI) - A(SI)'
 190  0                        DJSR2M2 'DIFFERENCE RIGHT JS -2 B(SI) - A(SI)'
 191  0                        DJSR2M1 'DIFFERENCE RIGHT JS -1 B(SI) - A(SI)'
 192  0                        DJSR2M0 'DIFFERENCE RIGHT JS  0 B(SI) - A(SI)'
 193  0                        DJSR2P1 'DIFFERENCE RIGHT JS +1 B(SI) - A(SI)'
 194  0                        DJSR2P2 'DIFFERENCE RIGHT JS +2 B(SI) - A(SI)'
 195  0                        DJSR2P3 'DIFFERENCE RIGHT JS +3 B(SI) - A(SI)'
 196  0                        DJSR3M3 'DIFFERENCE RIGHT JS -3 B - A'
 197  0                        DJSR3M2 'DIFFERENCE RIGHT JS -2 B - A'
 198  0                        DJSR3M1 'DIFFERENCE RIGHT JS -1 B - A'
 199  0                        DJSR3M0 'DIFFERENCE RIGHT JS  0 B - A'
 200  0                        DJSR3P1 'DIFFERENCE RIGHT JS +1 B - A'
 201  0                        DJSR3P2 'DIFFERENCE RIGHT JS +2 B - A'
 202  0                        DJSR3P3 'DIFFERENCE RIGHT JS +3 B - A'
 203  0                        LJOINT 'LEFT JOINT CONDITION'
 204  0                        RJOINT 'RIGHT JOINT CONDITION'
 205  0          VALUE LABELS  LJOINT 0 'NORMAL' 1 'LOCKED' 2 'DEGENERATIVE'/
 206  0                        RJOINT 0 'NORMAL' 1 'LOCKED' 2 'DEGENERATIVE'/
 207  0                        CTYPE 1 'CO' 2 'pivotal' 3 'pivotal with ortho'/
 208  0                        LOCKED 0 'None' 1 'Left Joint' 2 'Right Joint' 3 'Both'/
 209  0                        DJD 0 'None' 1 'Left Joint' 2 'Right Joint' 3 'Both'/
 210  0          FILE HANDLE   SPSSSURV/NAME='SPSS.SURV.FILE'
 211  0          FILE LABEL    DENTAL SURVEY
 212  0          SAVE OUTFILE=SPSSSURV

30 MAR 88 14:04:19      120 VARIABLES,    960 BYTES PER CASE BEFORE COMPRESSION
30 MAR 88 14:04:19       31 CASES SAVED
```

Appendix D

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

FILE: DENTAL SURVEY
CTYPE: 1 CO

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 10.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
CTYPE	1.000	.000	1	1	11	CASE TYPE -CO, PIVOTAL, PIVOTAL WITH ORT
MRX	.082	.240	-.50	.60	11	MODELS - RIGHT X COORDINATE
MRY	.245	.423	-.30	1.10	11	MODELS - RIGHT Y COORDINATE
MLX	.155	.501	-.30	1.40	11	MODELS - LEFT X COORDINATE
MLY	.336	.480	-.20	1.50	11	MODELS - LEFT Y COORDINATE
PCHANGE	-1.000	1.483	-3.00	2.00	11	PAIN DECREASE
LOCKED	1.300	.675	1.00	3.00	10	JOINT LOCKED
DJD	.200	.422	.00	1.00	10	DEGENERATIVE JOINT DISEASE PRESENT
JSLAM3	3.255	1.510	1.80	6.40	11	JOINT SPACE LEFT A -3 READING
JSLAM2	2.300	1.553	.70	5.70	11	JOINT SPACE LEFT A -2 READING
JSLAM1	1.818	.811	.80	3.20	11	JOINT SPACE LEFT A -1 READING
JSLAM0	1.900	.583	1.00	3.10	11	JOINT SPACE LEFT A 0 READING
JSLAP1	1.745	.515	1.00	2.40	11	JOINT SPACE LEFT A +1 READING
JSLAP2	1.827	.666	.70	3.00	11	JOINT SPACE LEFT A +2 READING
JSLAP3	2.127	.656	1.50	3.50	11	JOINT SPACE LEFT A +3 READING
JSLASIM3	2.782	1.658	1.00	5.90	11	JOINT SPACE LEFT SI A -3 READING
JSLASIM2	2.273	1.314	.80	4.90	11	JOINT SPACE LEFT SI A -2 READING
JSLASIM1	2.100	.875	1.10	4.00	11	JOINT SPACE LEFT SI A -1 READING
JSLASIM0	2.673	1.018	1.40	4.50	11	JOINT SPACE LEFT SI A 0 READING
JSLASIP1	2.945	1.174	1.50	4.80	11	JOINT SPACE LEFT SI A +1 READING
JSLASIP2	2.773	1.060	1.40	4.80	11	JOINT SPACE LEFT SI A +2 READING
JSLASIP3	3.045	.950	1.80	4.80	11	JOINT SPACE LEFT SI A +3 READING
JSLBM3	3.073	1.710	1.50	6.70	11	JOINT SPACE LEFT B -3 READING
JSLBM2	2.409	1.572	.70	6.00	11	JOINT SPACE LEFT B -2 READING
JSLBM1	2.136	1.062	1.00	3.80	11	JOINT SPACE LEFT B -1 READING
JSLBM0	2.409	.958	1.10	4.20	11	JOINT SPACE LEFT B 0 READING
JSLBP1	2.345	1.136	.20	4.70	11	JOINT SPACE LEFT B +1 READING
JSLBP2	2.336	1.398	.90	5.10	11	JOINT SPACE LEFT B +2 READING
JSLBP3	2.591	1.204	1.40	4.90	11	JOINT SPACE LEFT B +3 READING
JSLBSIM3	2.627	1.415	.90	5.50	11	JOINT SPACE LEFT SI B -3 READING
JSLBSIM2	2.136	1.074	.80	4.10	11	JOINT SPACE LEFT SI B -2 READING
JSLBSIM1	2.209	.854	1.20	4.00	11	JOINT SPACE LEFT SI B -1 READING
JSLBSIM0	2.855	1.066	1.60	5.00	11	JOINT SPACE LEFT SI B 0 READING
JSLBSIP1	3.055	1.131	1.20	4.80	11	JOINT SPACE LEFT SI B +1 READING
JSLBSIP2	3.000	1.111	1.10	4.40	11	JOINT SPACE LEFT SI B +2 READING
JSLBSIP3	3.200	.921	1.90	4.40	11	JOINT SPACE LEFT SI B +3 READING
JSRAM3	3.273	1.029	1.80	4.80	11	JOINT SPACE RIGHT A -3 READING
JSRAM2	2.518	.760	1.40	4.20	11	JOINT SPACE RIGHT A -2 READING
JSRAM1	2.382	.914	1.20	4.10	11	JOINT SPACE RIGHT A -1 READING
JSRAM0	2.282	.858	1.00	3.50	11	JOINT SPACE RIGHT A 0 READING
JSRAP1	2.045	.612	1.40	3.10	11	JOINT SPACE RIGHT A +1 READING
JSRAP2	1.964	.619	.90	2.90	11	JOINT SPACE RIGHT A +2 READING
JSRAP3	2.127	.782	1.00	3.40	11	JOINT SPACE RIGHT A +3 READING
JSRASIM3	2.964	1.176	1.70	5.20	11	JOINT SPACE RIGHT SI A -3 READING
JSRASIM2	2.336	.803	1.50	4.00	11	JOINT SPACE RIGHT SI A -2 READING
JSRASIM1	2.491	.760	1.50	4.00	11	JOINT SPACE RIGHT SI A -1 READING

10 MAR 68 ANALYSIS FOR THE MAJOR STUDY
10:10:14 University of Alberta

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 10.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
JSRASIM0	3.009	1.267	.60	4.80	11	JOINT SPACE RIGHT SI A 0 READING
JSRASIP1	3.082	1.336	.70	4.80	11	JOINT SPACE RIGHT SI A +1 READING
JSRASIP2	3.036	1.229	1.20	4.80	11	JOINT SPACE RIGHT SI A +2 READING
JSRASIP3	3.109	1.228	1.20	4.90	11	JOINT SPACE RIGHT SI A +3 READING
JSRBM3	3.473	1.005	1.70	4.70	11	JOINT SPACE RIGHT B -3 READING
JSRBM2	2.473	.710	.60	4.20	11	JOINT SPACE RIGHT B -2 READING
JSRBM1	2.464	.866	.90	4.30	11	JOINT SPACE RIGHT B -1 READING
JSRBM0	2.345	.906	.90	3.70	11	JOINT SPACE RIGHT B 0 READING
JSRBP1	2.191	.641	1.30	3.20	11	JOINT SPACE RIGHT B +1 READING
JSRBP2	1.991	.686	1.00	3.40	11	JOINT SPACE RIGHT B +2 READING
JSRBP3	2.127	.842	.80	4.00	11	JOINT SPACE RIGHT B +3 READING
JSRBSIM3	3.027	1.130	1.70	4.70	11	JOINT SPACE RIGHT SI B -3 READING
JSRBSIM2	2.291	.797	1.70	4.20	11	JOINT SPACE RIGHT SI B -2 READING
JSRBSIM1	2.455	.899	1.40	4.80	11	JOINT SPACE RIGHT SI B -1 READING
JSRBSIM0	2.945	1.200	1.30	5.30	11	JOINT SPACE RIGHT SI B 0 READING
JSRBSIP1	3.073	1.082	1.80	5.20	11	JOINT SPACE RIGHT SI B +1 READING
JSRBSIP2	3.000	1.017	1.80	4.90	11	JOINT SPACE RIGHT SI B +2 READING
JSRBSIP3	3.109	1.094	1.80	4.50	11	JOINT SPACE RIGHT SI B +3 READING
LASIAX	.618	.624	.00	2.10	11	LEFT A(SI) - A X COORDINATE
LASIAY	.509	.628	-.10	1.90	11	LEFT A(SI) - A Y COORDINATE
LASIBSIX	.027	.287	-.50	.40	11	LEFT A(SI) - B(SI) X COORDINATE
LASIBSIY	.064	.314	-.40	.80	11	LEFT A(SI) - B(SI) Y COORDINATE
LBAX	.118	.477	-.30	1.40	11	LEFT B - A X COORDINATE
LBAY	.300	.492	-.10	1.50	11	LEFT B - A Y COORDINATE
RASIAX	.645	.880	-.80	2.00	11	RIGHT A(SI) - A X COORDINATE
RASIAY	.500	.506	-.20	1.20	11	RIGHT A(SI) - A Y COORDINATE
RASIBSIX	.055	.192	-.20	.50	11	RIGHT A(SI) - B(SI) X COORDINATE
RASIBSIY	-.045	.216	-.50	.30	11	RIGHT A(SI) - B(SI) Y COORDINATE
RBAX	-.045	.175	-.30	.30	11	RIGHT B - A X COORDINATE
RBAY	.100	.210	-.20	.40	11	RIGHT B - A Y COORDINATE
DJSL1M3	-.473	.516	-1.40	.20	11	DIFFERENCE LEFT JS -3 A(SI) - A
DJSL1M2	-.027	.471	-.80	.60	11	DIFFERENCE LEFT JS -2 A(SI) - A
DJSL1M1	.282	.402	-.10	1.10	11	DIFFERENCE LEFT JS -1 A(SI) - A
DJSL1M0	.773	.878	.00	2.70	11	DIFFERENCE LEFT JS 0 A(SI) - A
DJSL1P1	1.200	1.126	.20	3.70	11	DIFFERENCE LEFT JS +1 A(SI) - A
DJSL1P2	.945	.783	-.10	2.40	11	DIFFERENCE LEFT JS +2 A(SI) - A
DJSL1P3	.918	.648	.00	2.10	11	DIFFERENCE LEFT JS +3 A(SI) - A
DJSL2M3	-.155	.592	-1.00	1.00	11	DIFFERENCE LEFT JS -3 B(SI) - A(SI)
DJSL2M2	-.136	.580	-1.40	.90	11	DIFFERENCE LEFT JS -2 B(SI) - A(SI)
DJSL2M1	.109	.437	-.50	1.20	11	DIFFERENCE LEFT JS -1 B(SI) - A(SI)
DJSL2M0	.182	.447	-.40	1.00	11	DIFFERENCE LEFT JS 0 B(SI) - A(SI)
DJSL2P1	.109	.589	-1.00	1.00	11	DIFFERENCE LEFT JS +1 B(SI) - A(SI)
DJSL2P2	.227	.508	-.40	1.10	11	DIFFERENCE LEFT JS +2 B(SI) - A(SI)
DJSL2P3	.155	.468	-.40	1.00	11	DIFFERENCE LEFT JS +3 B(SI) - A(SI)
DJSL3M3	-.182	.512	-1.40	.40	11	DIFFERENCE LEFT JS -3 B - A
DJSL3M2	.109	.239	-.30	.50	11	DIFFERENCE LEFT JS -2 B - A
DJSL3M1	.318	.508	-.30	1.10	11	DIFFERENCE LEFT JS -1 B - A
DJSL3M0	.509	.875	-.10	2.40	11	DIFFERENCE LEFT JS 0 B - A

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 10.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
DJSL3P1	.600	1.018	-.20	2.80	11	DIFFERENCE LEFT JS +1 B - A
DJSL3P2	.509	.944	-.30	2.60	11	DIFFERENCE LEFT JS+2 B - A
DJSL3P3	.464	.749	-.30	2.20	11	DIFFERENCE LEFT JS +3 B - A
DJSR1M3	-.309	.902	-1.70	1.50	11	DIFFERENCE RIGHT JS -3 A(SI) - A
DJSR1M2	-.182	.611	-1.30	1.10	11	DIFFERENCE RIGHT JS -2 A(SI) - A
DJSR1M1	.109	.444	-.60	.90	11	DIFERENCE RIGHT JS -1 A(SI) - A
DJSR1M0	.727	.731	-.40	1.80	11	DIFFERENCE RIGHT JS 0 A(SI) - A
DJSR1P1	1.036	1.210	-1.10	2.80	11	DIFFERENCE RIGHT JS +1 A(SI) - A
DJSR1P2	1.073	1.238	-.80	2.60	11	DIFFERENCE RIGHT JS +2 A(SI) - A
DJSR1P3	.982	1.111	-.90	2.60	11	DIFFERENCE RIGHT JS +3 A(SI) - A
DJSR2M3	.064	.625	-1.50	.80	11	DIFFERENCE RIGHT JS -3 B(SI) - A(SI)
DJSR2M2	-.045	.513	-1.30	.90	11	DIFFERENCE RIGHT JS -2 B(SI) - A(SI)
DJSR2M1	-.036	.364	-.40	.80	11	DIFFERENCE RIGHT JS -1 B(SI) - A(SI)
DJSR2M0	-.064	.388	-.70	.70	11	DIFFERENCE RIGHT JS 0 B(SI) - A(SI)
DJSR2P1	-.009	.517	-.80	1.10	11	DIFFERENCE RIGHT JS +1 B(SI) - A(SI)
DJSR2P2	-.036	.406	-.70	.60	11	DIFFERENCE RIGHT JS +2 B(SI) - A(SI)
DJSR2P3	.000	.326	-.50	.70	11	DIFFERENCE RIGHT JS +3 B(SI) - A(SI)
DJSR3M3	.200	.402	-.60	.70	11	DIFFERENCE RIGHT JS -3 B - A
DJSR3M2	-.045	.345	-.60	.50	11	DIFFERENCE RIGHT JS -2 B - A
DJSR3M1	.082	.343	-.40	.70	11	DIFFERENCE RIGHT JS -1 B - A
DJSR3M0	.064	.294	-.30	.60	11	DIFFERENCE RIGHT JS 0 B - A
DJSR3P1	.145	.362	-.20	.70	11	DIFFERENCE RIGHT JS +1 B - A
DJSR3P2	.027	.307	-.40	.50	11	DIFFERENCE RIGHT JS +2 B - A
DJSR3P3	.000	.379	-.40	.70	11	DIFFERENCE RIGHT JS +3 B - A
LJOINT	1.000	.632	.00	2.00	11	LEFT JOINT CONDITION
RJOINT	.182	.405	.00	1.00	11	RIGHT JOINT CONDITION

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

FILE: DENTAL SURVEY
CTYPE: 2 pivotal

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 9.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
CTYPE	2.000	.000	2	2	10	CASE TYPE -CO, PIVOTAL, PIVOTAL WITH ORT
MRX	-.080	.549	-1.20	.70	10	MODELS - RIGHT X COORDINATE
MRY	.140	.711	-1.50	1.40	10	MODELS - RIGHT Y COORDINATE
MLX	.280	.629	-.60	.90	10	MODELS - LEFT X COORDINATE
MLY	.830	.987	.00	2.60	10	MODELS - LEFT Y COORDINATE
PCHANGE	-3.000	2.357	-6.00	2.00	10	PAIN DECREASE
LOCKED	1.556	.882	1.00	3.00	9	JOINT LOCKED
DJD	.444	1.014	.00	3.00	9	DEGENERATIVE JOINT DISEASE PRESENT
JSLAM3	4.130	1.169	2.50	6.00	10	JOINT SPACE LEFT A -3 READING
JSLAM2	3.700	1.194	2.10	5.90	10	JOINT SPACE LEFT A -2 READING
JSLAM1	3.210	1.125	.60	4.50	10	JOINT SPACE LEFT A -1 READING
JSLAM0	2.470	1.046	.80	4.00	10	JOINT SPACE LEFT A 0 READING
JSLAP1	2.120	.838	1.30	3.40	10	JOINT SPACE LEFT A +1 READING
JSLAP2	2.180	.751	1.00	3.50	10	JOINT SPACE LEFT A +2 READING
JSLAP3	2.140	.755	.60	3.20	10	JOINT SPACE LEFT A +3 READING
JSLASIM3	3.630	1.098	2.30	5.90	10	JOINT SPACE LEFT SI A -3 READING
JSLASIM2	3.310	.924	1.50	4.60	10	JOINT SPACE LEFT SI A -2 READING
JSLASIM1	3.310	1.040	.90	4.90	10	JOINT SPACE LEFT SI A -1 READING
JSLASIM0	3.510	1.202	1.20	5.40	10	JOINT SPACE LEFT SI A 0 READING
JSLASIP1	3.470	1.251	1.40	5.40	10	JOINT SPACE LEFT SI A +1 READING
JSLASIP2	3.330	1.240	1.30	5.10	10	JOINT SPACE LEFT SI A +2 READING
JSLASIP3	3.240	1.292	1.10	5.10	10	JOINT SPACE LEFT SI A +3 READING
JSLBM3	3.940	.999	2.70	5.80	10	JOINT SPACE LEFT B -3 READING
JSLBM2	3.570	.945	1.70	4.80	10	JOINT SPACE LEFT B -2 READING
JSLBM1	3.250	1.066	.80	4.50	10	JOINT SPACE LEFT B -1 READING
JSLBM0	2.900	1.050	1.10	4.80	10	JOINT SPACE LEFT B 0 READING
JSLBP1	2.670	.837	1.50	4.10	10	JOINT SPACE LEFT B +1 READING
JSLBP2	2.550	.943	1.00	4.30	10	JOINT SPACE LEFT B +2 READING
JSLBP3	2.520	1.064	.50	4.30	10	JOINT SPACE LEFT B +3 READING
JSLBSIM3	3.670	.896	2.20	5.20	10	JOINT SPACE LEFT SI B -3 READING
JSLBSIM2	3.310	.677	2.20	4.20	10	JOINT SPACE LEFT SI B -2 READING
JSLBSIM1	3.150	1.015	.80	4.80	10	JOINT SPACE LEFT SI B -1 READING
JSLBSIM0	3.210	.993	1.10	5.10	10	JOINT SPACE LEFT SI B 0 READING
JSLBSIP1	3.210	.919	1.40	5.00	10	JOINT SPACE LEFT SI B +1 READING
JSLBSIP2	2.990	.851	1.60	5.00	10	JOINT SPACE LEFT SI B +2 READING
JSLBSIP3	3.020	.968	1.90	5.00	10	JOINT SPACE LEFT SI B +3 READING
JSRAM3	3.030	1.819	1.10	6.70	10	JOINT SPACE RIGHT A -3 READING
JSRAM2	2.110	1.057	.80	4.00	10	JOINT SPACE RIGHT A -2 READING
JSRAM1	2.190	.746	1.00	3.60	10	JOINT SPACE RIGHT A -1 READING
JSRAM0	2.500	.889	1.00	4.30	10	JOINT SPACE RIGHT A 0 READING
JSRAP1	2.760	1.049	1.00	4.20	10	JOINT SPACE RIGHT A +1 READING
JSRAP2	2.770	1.046	1.40	4.60	10	JOINT SPACE RIGHT A +2 READING
JSRAP3	2.650	.990	1.50	4.60	10	JOINT SPACE RIGHT A +3 READING
JSRASIM3	2.810	1.901	.80	7.10	10	JOINT SPACE RIGHT SI A -3 READING
JSRASIM2	2.690	.872	1.50	4.40	10	JOINT SPACE RIGHT SI A -2 READING
JSRASIM1	2.900	.990	1.80	5.00	10	JOINT SPACE RIGHT SI A -1 READING

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 9.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
JSRASIM0	3.720	1.545	1.90	6.60	10	JOINT SPACE RIGHT SI A 0 READING
JSRASIP1	4.000	1.895	1.90	7.50	10	JOINT SPACE RIGHT SI A +1 READING
JSRASIP2	3.820	1.890	1.60	7.30	10	JOINT SPACE RIGHT SI A +2 READING
JSRASIP3	3.720	1.677	1.70	6.80	10	JOINT SPACE RIGHT SI A +3 READING
JSRBM3	3.070	1.840	1.20	7.10	10	JOINT SPACE RIGHT B -3 READING
JSRBM2	2.220	.868	1.30	4.10	10	JOINT SPACE RIGHT B -2 READING
JSRBM1	2.280	.864	.70	3.80	10	JOINT SPACE RIGHT B -1 READING
JSRBM0	2.830	.883	1.40	4.20	10	JOINT SPACE RIGHT B 0 READING
JSRBP1	3.020	1.006	1.90	4.70	10	JOINT SPACE RIGHT B +1 READING
JSRBP2	2.890	.910	1.60	4.50	10	JOINT SPACE RIGHT B +2 READING
JSRBP3	2.870	.949	1.60	4.50	10	JOINT SPACE RIGHT B +3 READING
JSRBSIM3	2.660	1.143	1.00	4.30	10	JOINT SPACE RIGHT SI B -3 READING
JSRBSIM2	2.230	.742	1.00	3.30	10	JOINT SPACE RIGHT SI B -2 READING
JSRBSIM1	2.850	1.218	1.10	5.20	10	JOINT SPACE RIGHT SI B -1 READING
JSRBSIM0	3.520	1.601	1.30	6.50	10	JOINT SPACE RIGHT SI B 0 READING
JSRBSIP1	3.790	1.828	1.20	7.50	10	JOINT SPACE RIGHT SI B +1 READING
JSRBSIP2	3.550	1.724	1.40	7.30	10	JOINT SPACE RIGHT SI B +2 READING
JSRBSIP3	3.540	1.509	1.40	6.70	10	JOINT SPACE RIGHT SI B +3 READING
LASIAX	.660	.548	.00	1.60	10	LEFT A(SI) - A X COORDINATE
LASIAY	.670	.596	.00	1.70	10	LEFT A(SI) - A Y COORDINATE
LASIBSIX	.150	.502	-.60	1.10	10	LEFT A(SI) - B(SI) X COORDINATE
LASIBSIY	-.210	.441	-1.30	.40	10	LEFT A(SI) - B(SI) Y COORDINATE
LBAX	.180	.270	-.30	.50	10	LEFT B - A X COORDINATE
LBAY	.470	.727	-.10	2.30	10	LEFT B - A Y COORDINATE
RASIAX	.540	.628	-.20	1.80	10	RIGHT A(SI) - A X COORDINATE
RASIAY	.700	.604	.10	2.00	10	RIGHT A(SI) - A Y COORDINATE
RASIBSIX	.080	.569	-.50	1.60	10	RIGHT A(SI) - B(SI) X COORDINATE
RASIBSIY	.100	.427	-.50	.90	10	RIGHT A(SI) - B(SI) Y COORDINATE
RBAX	.000	.452	-1.00	.50	10	RIGHT B - A X COORDINATE
RBAY	.080	.537	-1.20	1.00	10	RIGHT B - A Y COORDINATE
DJSL1M3	-.500	.568	-1.80	.20	10	DIFFERENCE LEFT JS -3 A(SI) - A
DJSL1M2	-.390	.698	-2.10	.40	10	DIFFERENCE LEFT JS -2 A(SI) - A
DJSL1M1	.100	.512	-.60	1.00	10	DIFFERENCE LEFT JS -1 A(SI) - A
DJSL1M0	1.040	.893	.00	2.60	10	DIFFERENCE LEFT JS 0 A(SI) - A
DJSL1P1	1.350	.911	.00	2.70	10	DIFFERENCE LEFT JS +1 A(SI) - A
DJSL1P2	1.150	.788	.00	2.70	10	DIFFERENCE LEFT JS +2 A(SI) - A
DJSL1P3	1.100	.770	.10	2.60	10	DIFFERENCE LEFT JS +3 A(SI) - A
DJSL2M3	.040	.687	-1.60	1.00	10	DIFFERENCE LEFT JS -3 B(SI) - A(SI)
DJSL2M2	.000	.508	-1.10	.80	10	DIFFERENCE LEFT JS -2 B(SI) - A(SI)
DJSL2M1	-.160	.259	-.60	.20	10	DIFFERENCE LEFT JS -1 B(SI) - A(SI)
DJSL2M0	-.300	.643	-1.50	.70	10	DIFFERENCE LEFT JS 0 B(SI) - A(SI)
DJSL2P1	-.260	.838	-1.90	1.80	10	DIFFERENCE LEFT JS +1 B(SI) - A(SI)
DJSL2P2	-.340	.935	-2.00	1.10	10	DIFFERENCE LEFT JS +2 B(SI) - A(SI)
DJSL2P3	-.220	.832	-1.60	1.30	10	DIFFERENCE LEFT JS +3 B(SI) - A(SI)
DJSL3M3	-.190	.528	-1.10	.80	10	DIFFERENCE LEFT JS -3 B - A
DJSL3M2	-.130	.536	-1.40	.60	10	DIFFERENCE LEFT JS -2 B - A
DJSL3M1	.040	.291	-.50	.50	10	DIFFERENCE LEFT JS -1 B - A
DJSL3M0	.430	.677	-.20	2.10	10	DIFFERENCE LEFT JS 0 B - A

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 9.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
DJSL3P1	.550	.638	.00	2.20	10	DIFFERENCE LEFT JS +1 B - A
DJSL3P2	.370	.521	-.20	1.40	10	DIFFERENCE LEFT JS +2 B - A
DJSL3P3	.380	.473	-.20	1.10	10	DIFFERENCE LEFT JS +3 B - A
DJSR1M3	-.220	.382	-1.00	.40	10	DIFFERENCE RIGHT JS -3 A(SI) - A
DJSR1M2	.380	.694	-.80	1.70	10	DIFFERENCE RIGHT JS -2 A(SI) - A
DJSR1M1	.710	.882	-.30	2.50	10	DIFFERENCE RIGHT JS -1 A(SI) - A
DJSR1M0	1.220	1.086	.30	3.50	10	DIFFERENCE RIGHT JS 0 A(SI) - A
DJSR1P1	1.260	1.018	-.10	3.30	10	DIFFERENCE RIGHT JS +1 A(SI) - A
DJSR1P2	1.050	.980	-.10	2.70	10	DIFFERENCE RIGHT JS +2 A(SI) - A
DJSR1P3	1.070	.909	.10	2.90	10	DIFFERENCE RIGHT JS +3 A(SI) - A
DJSR2M3	-.150	1.050	-2.80	.90	10	DIFFERENCE RIGHT JS -3 B(SI) - A(SI)
DJSR2M2	-.260	.659	-1.70	.60	10	DIFFERENCE RIGHT JS -2 B(SI) - A(SI)
DJSR2M1	-.050	.495	-.90	.60	10	DIFFERENCE RIGHT JS -1 B(SI) - A(SI)
DJSR2M0	-.200	.748	-1.80	.90	10	DIFFERENCE RIGHT JS 0 B(SI) - A(SI)
DJSR2P1	-.210	.970	-1.50	1.80	10	DIFFERENCE RIGHT JS +1 B(SI) - A(SI)
DJSR2P2	-.270	.923	-1.70	1.60	10	DIFFERENCE RIGHT JS +2 B(SI) - A(SI)
DJSR2P3	-.180	.742	-1.60	1.30	10	DIFFERENCE RIGHT JS +3 B(SI) - A(SI)
DJSR3M3	.040	.706	-1.00	1.20	10	DIFFERENCE RIGHT JS -3 B - A
DJSR3M2	.110	.491	-1.00	.80	10	DIFFERENCE RIGHT JS -2 B - A
DJSR3M1	.090	.612	-1.10	1.10	10	DIFFERENCE RIGHT JS -1 B - A
DJSR3M0	.330	.879	-1.70	1.60	10	DIFFERENCE RIGHT JS 0 B - A
DJSR3P1	.280	.984	-2.00	1.90	10	DIFFERENCE RIGHT JS +1 B - A
DJSR3P2	.120	.822	-1.60	1.50	10	DIFFERENCE RIGHT JS +2 B - A
DJSR3P3	.220	.851	-1.40	1.50	10	DIFFERENCE RIGHT JS +3 B - A
LJOINT	1.000	.667	.00	2.00	10	LEFT JOINT CONDITION
RJOINT	.400	.699	.00	2.00	10	RIGHT JOINT CONDITION

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

FILE: DENTAL SURVEY
CTYPE: 3 pivotal with ortho

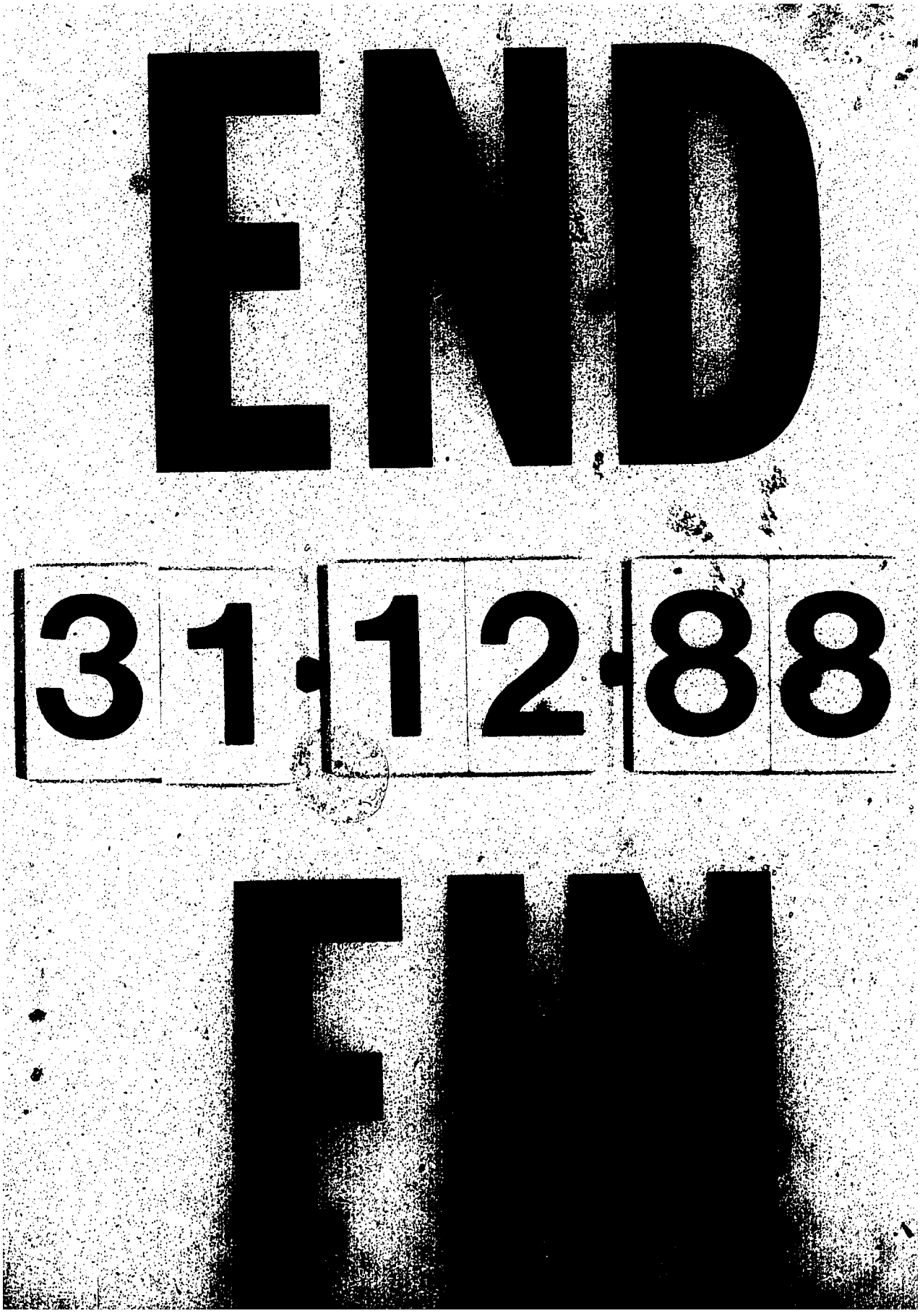
NUMBER OF VALID OBSERVATIONS (LISTWISE) = 10.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
CTYPE	3.000	.000	3	3	10	CASE TYPE -CO, PIVOTAL, PIVOTAL WITH ORT
MRX	-.350	.560	-1.10	.60	10	MODELS - RIGHT X COORDINATE
MRY	1.210	.318	.50	1.50	10	MODELS - RIGHT Y COORDINATE
MLX	-.360	.578	-.90	1.10	10	MODELS - LEFT X COORDINATE
MLY	1.210	.296	.80	1.80	10	MODELS - LEFT Y COORDINATE
PCHANGE	-4.600	1.506	-7.00	-2.00	10	PAIN DECREASE
LOCKED	1.600	.843	1.00	3.00	10	JOINT LOCKED
DJD	.900	1.287	.00	3.00	10	DEGENERATIVE JOINT DISEASE PRESENT
JSLAM3	3.910	1.475	1.70	5.40	10	JOINT SPACE LEFT A -3 READING
JSLAM2	3.020	1.289	1.20	4.80	10	JOINT SPACE LEFT A -2 READING
JSLAM1	2.650	1.052	1.60	4.70	10	JOINT SPACE LEFT A -1 READING
JSLAM0	2.490	.656	1.90	4.10	10	JOINT SPACE LEFT A 0 READING
JSLAP1	2.010	.647	1.20	3.40	10	JOINT SPACE LEFT A +1 READING
JSLAP2	2.040	.718	1.30	3.40	10	JOINT SPACE LEFT A +2 READING
JSLAP3	2.160	.700	1.30	3.50	10	JOINT SPACE LEFT A +3 READING
JSLASIM3	2.610	1.090	1.20	4.60	10	JOINT SPACE LEFT SI A -3 READING
JSLASIM2	2.140	1.073	1.00	4.20	10	JOINT SPACE LEFT SI A -2 READING
JSLASIM1	2.750	1.011	1.60	4.70	10	JOINT SPACE LEFT SI A -1 READING
JSLASIM0	3.320	.555	2.30	4.40	10	JOINT SPACE LEFT SI A 0 READING
JSLASIP1	3.450	.803	2.50	5.10	10	JOINT SPACE LEFT SI A +1 READING
JSLASIP2	3.460	.783	2.70	5.10	10	JOINT SPACE LEFT SI A +2 READING
JSLASIP3	3.550	.711	2.70	4.80	10	JOINT SPACE LEFT SI A +3 READING
JSLBM3	4.090	1.552	2.00	5.90	10	JOINT SPACE LEFT B -3 READING
JSLBM2	3.630	1.363	1.80	5.40	10	JOINT SPACE LEFT B -2 READING
JSLBM1	3.480	.996	2.10	4.90	10	JOINT SPACE LEFT B -1 READING
JSLBM0	3.460	.880	1.90	5.10	10	JOINT SPACE LEFT B 0 READING
JSLBP1	3.100	.883	1.80	5.10	10	JOINT SPACE LEFT B +1 READING
JSLBP2	2.510	.966	1.50	4.70	10	JOINT SPACE LEFT B +2 READING
JSLBP3	2.480	.989	1.20	3.90	10	JOINT SPACE LEFT B +3 READING
JSLBSIM3	3.720	1.428	2.10	5.80	10	JOINT SPACE LEFT SI B -3 READING
JSLBSIM2	3.200	1.145	1.90	5.30	10	JOINT SPACE LEFT SI B -2 READING
JSLBSIM1	3.570	1.049	2.30	5.40	10	JOINT SPACE LEFT SI B -1 READING
JSLBSIM0	4.050	.968	3.20	6.40	10	JOINT SPACE LEFT SI B 0 READING
JSLBSIP1	3.710	1.063	2.30	6.40	10	JOINT SPACE LEFT SI B +1 READING
JSLBSIP2	3.300	1.023	1.90	5.70	10	JOINT SPACE LEFT SI B +2 READING
JSLBSIP3	3.130	.913	1.60	4.60	10	JOINT SPACE LEFT SI B +3 READING
JSRAM3	4.600	1.202	2.00	5.90	10	JOINT SPACE RIGHT A -3 READING
JSRAM2	2.910	1.026	1.00	4.20	10	JOINT SPACE RIGHT A -2 READING
JSRAM1	1.640	.598	.80	2.50	10	JOINT SPACE RIGHT A -1 READING
JSRAM0	1.800	.653	.80	3.00	10	JOINT SPACE RIGHT A 0 READING
JSRAP1	1.890	.962	.80	4.00	10	JOINT SPACE RIGHT A +1 READING
JSRAP2	2.080	1.041	.80	4.20	10	JOINT SPACE RIGHT A +2 READING
JSRAP3	2.150	1.122	.80	4.20	10	JOINT SPACE RIGHT A +3 READING
JSRASIM3	3.540	1.514	1.50	5.90	10	JOINT SPACE RIGHT SI A -3 READING
JSRASIM2	2.270	.907	1.00	3.50	10	JOINT SPACE RIGHT SI A -2 READING
JSRASIM1	1.990	.451	1.30	2.60	10	JOINT SPACE RIGHT SI A -1 READING

30 MAR 88 ANALYSIS FOR THE MAJOR STUDY
15:10:11 University of Alberta

NUMBER OF VALID OBSERVATIONS (LISTWISE) = 10.00

VARIABLE	MEAN	STD DEV	MINIMUM	MAXIMUM	VALID N	LABEL
JSRASIM0	2.510	.666	1.60	3.50	10	JOINT SPACE RIGHT SI A 0 READING
JSRASIP1	3.010	1.045	1.10	4.10	10	JOINT SPACE RIGHT SI A +1 READING
JSRASIP2	3.190	1.230	1.10	4.90	10	JOINT SPACE RIGHT SI A +2 READING
JSRASIP3	3.140	1.329	1.10	5.20	10	JOINT SPACE RIGHT SI A +3 READING
JSRBM3	4.570	1.112	2.90	6.30	10	JOINT SPACE RIGHT B -3 READING
JSRBM2	3.270	1.080	1.40	4.60	10	JOINT SPACE RIGHT B -2 READING
JSRBM1	2.590	.870	1.60	4.40	10	JOINT SPACE RIGHT B -1 READING
JSRBM0	2.860	.955	1.80	5.10	10	JOINT SPACE RIGHT B 0 READING
JSRBP1	2.760	1.109	1.90	5.40	10	JOINT SPACE RIGHT B +1 READING
JSRBP2	2.660	1.303	1.50	5.70	10	JOINT SPACE RIGHT B +2 READING
JSRBP3	2.510	1.435	.80	5.70	10	JOINT SPACE RIGHT B +3 READING
JSRBSIM3	4.130	1.101	2.50	5.40	10	JOINT SPACE RIGHT SI B -3 READING
JSRBSIM2	3.010	.725	2.00	4.20	10	JOINT SPACE RIGHT SI B -2 READING
JSRBSIM1	2.970	.883	1.90	4.50	10	JOINT SPACE RIGHT SI B -1 READING
JSRBSIM0	3.250	.917	1.90	4.90	10	JOINT SPACE RIGHT SI B 0 READING
JSRBSIP1	3.440	.898	2.50	5.20	10	JOINT SPACE RIGHT SI B +1 READING
JSRBSIP2	3.200	1.111	1.90	5.40	10	JOINT SPACE RIGHT SI B +2 READING
JSRBSIP3	3.200	1.349	1.60	5.40	10	JOINT SPACE RIGHT SI B +3 READING
LASIAX	1.080	.349	.60	1.50	10	LEFT A(SI) - A X COORDINATE
LASIAY	.540	.378	.10	1.20	10	LEFT A(SI) - A Y COORDINATE
LASIBSIX	-.530	.437	-1.30	.00	10	LEFT A(SI) - B(SI) X COORDINATE
LASIBSIY	.860	.845	.00	3.10	10	LEFT A(SI) - B(SI) Y COORDINATE
LBAX	-.140	.453	-.70	1.00	10	LEFT B - A X COORDINATE
LBAY	1.140	.259	.90	1.80	10	LEFT B - A Y COORDINATE
RASIAX	.820	.529	.00	1.60	10	RIGHT A(SI) - A X COORDINATE
RASIAY	.590	.451	.00	1.30	10	RIGHT A(SI) - A Y COORDINATE
RASIBSIX	-.320	.452	-1.20	.20	10	RIGHT A(SI) - B(SI) X COORDINATE
RASIBSIY	.790	.640	.00	2.00	10	RIGHT A(SI) - B(SI) Y COORDINATE
RBAX	-.120	.391	-.60	.60	10	RIGHT B - A X COORDINATE
RBAY	.990	.418	.00	1.40	10	RIGHT B - A Y COORDINATE
DJSL1M3	-1.300	.957	-3.30	-.30	10	DIFFERENCE LEFT JS -3 A(SI) - A
DJSL1M2	-.880	1.185	-3.30	.10	10	DIFFERENCE LEFT JS -2 A(SI) - A
DJSL1M1	.100	.337	-.50	.70	10	DIFFERENCE LEFT JS -1 A(SI) - A
DJSL1M0	.830	.579	-.30	1.60	10	DIFFERENCE LEFT JS 0 A(SI) - A
DJSL1P1	1.440	.450	.60	2.00	10	DIFFERENCE LEFT JS +1 A(SI) - A
DJSL1P2	1.420	.509	.70	2.10	10	DIFFERENCE LEFT JS +2 A(SI) - A
DJSL1P3	1.390	.446	.70	2.10	10	DIFFERENCE LEFT JS +3 A(SI) - A
DJSL2M3	1.110	.739	.30	2.50	10	DIFFERENCE LEFT JS -3 B(SI) - A(SI)
DJSL2M2	1.060	.914	.30	3.10	10	DIFFERENCE LEFT JS -2 B(SI) - A(SI)
DJSL2M1	.820	.739	.20	2.50	10	DIFFERENCE LEFT JS -1 B(SI) - A(SI)
DJSL2M0	.730	.558	.10	2.00	10	DIFFERENCE LEFT JS 0 B(SI) - A(SI)
DJSL2P1	.260	.602	-.40	1.30	10	DIFFERENCE LEFT JS +1 B(SI) - A(SI)
DJSL2P2	-.160	.556	-.90	.60	10	DIFFERENCE LEFT JS +2 B(SI) - A(SI)
DJSL2P3	-.420	.442	-1.10	.20	10	DIFFERENCE LEFT JS +3 B(SI) - A(SI)
DJSL3M3	.180	.437	-.60	1.00	10	DIFFERENCE LEFT JS -3 B - A
DJSL3M2	.610	.983	-.60	2.80	10	DIFFERENCE LEFT JS -2 B - A
DJSL3M1	.830	.811	-.10	2.70	10	DIFFERENCE LEFT JS -1 B - A
DJSL3M0	.970	.655	.00	2.30	10	DIFFERENCE LEFT JS 0 B - A


END
3-11-88
FM