"Integrity without knowledge is weak and useless, and knowledge without integrity is dangerous and dreadful."

Samuel Johnson (Sept 18, 1709-Dec. 13, 1874)

University of Alberta

Predictive Value of Magnetic Resonance Imaging of Disc Displacement for Temporomandibular Disorder Signs and Symptoms in a Pre-orthodontic Adolescent Patient Population

by

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DEDICATION

This thesis is lovingly dedicated to my mother Norma Alvaro Moldez, most wonderful and compassionate mother anyone could ever have. Thank you so much Nanay for all the beautiful memories. You are my angel! I love you so much and I truly miss you.

ABSTRACT

The purpose of this retrospective study was to examine the predictive value of quantitative MRI disc displacement for changes of TMD signs and symptoms in a pre-orthodontic adolescent patient sample.

The sample consisted of fifty-five adolescent patients who had baseline temporomandibular joint MRI and longitudinal clinical records of TMD signs and symptoms. Thirty-eight were females and seventeen were males. The mean age was 12.9 ±1.6 years (range 7.4-16.6).

Our analysis revealed that MRI right disc displacement has significant association with changes of ipsilateral joint locking. However, predictive value of disc displacement for changes of joint locking could not be analyzed due to small sample size. Nevertheless, MRI right disc displacement can distinguish subjects with persistent ipsilateral clicking from those who were persistently free of clicking from T1 to T2. These findings indicate that clinical assessment of right joint clicking is consistent with MRI determination of disc displacement.

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LIST OF ABBREVIATIONS

Ach	Acetylcholine
APS	Average Pain Sensitivity
ARS	Anterior Repositioning Splint
BN	Brian Nebbe
CMP	Cervical Muscle Pain
COMT	Catecholamine-O-Methyltransferase
DD	Disc Displacement
DDWR	Disc Displacement With Reduction
DDWOR	Disc Displacement Without Reduction
ECM	Extracellular Matrix
HA	Hyaluronic Acid
HPS	Higher Pain Sensitivity
LDD	Left Disc Displacement
LPS	Low Pain Sensitivity
LTR	Local Twitch Response
MMP	Masticatory Muscle Pain
MRI	Magnetic Resonance Imaging
MTrPs	Myofascial Trigger Points
NIH	National Institute of Health
PLA_2	Phospholipase A_2
RDD	Right Disc Displacement
SCM	Sternocleidomastoid
T1	Time 1 (Initial)
T2	Time 2 (Follow up over 4years)
TMD	Temporomandibular Disorder
TMJ	Temporomandibular Joint

CHAPTER 1

INTRODUCTION

1.1 GENERAL INTRODUCTION

Temporomandibular disorders (TMDs) are medical and dental conditions affecting the temporomandibular joints (TMJs) and/or the masticatory muscles as well as the contiguous tissue structures[1]. TMD pain has a prevalence of about 8% in males and 15% in females of all ages in North American population[2] and is an etiological factor for psychosocial impairment and decreased quality of life in a significant segment of the clinical population[2]. According to the research diagnostic criteria (RDC)[3], the primary subtypes of TMDs are myofascial pain, internal TM joint derangement and osteoarthritis. These conditions produce overlapping TMD signs and symptoms affecting the jaw muscles and the joint in front of the ear, or inside the ear[4-7]. Huang et al[8] interviewed and examined 592 Caucasian adult individuals according to RDC and classified them into the following groups: pain-free (41.6%), myofascial pain only (20.7%), arthralgia only (4.3%), and myofascial pain with arthralgia (33.5%). Based on clinical examination, 31% of the subjects in the three pain groups had diagnosis of disc displacement, as did 12% of the pain-free subjects.

1.1.1 MYOFASCIAL PAIN

Myofascial pain is characterized by tender muscle spots referred to as myofascial trigger points (MTrPs) which can be palpated within taut bands of skeletal muscle as 3–6mm firm and hyperirritable nodules[9]. Trigger points have been associated with sustained and/or repetitive muscle

overload[9,10] which stimulates muscle free nerve endings to release excessive amount of acetylcholine (ACh) at the motor endplate. Consequently, ACh depolarizes the post-junctional membrane of the muscle fiber resulting in sarcomere contraction[10,11]. This contraction increases local energy consumption and reduces local circulation, a condition that leads to ischemia and hypoxia. The local ischemia initiates the release of substances that sensitize nociceptors and in turn, induces the release of neurovasoreactive chemicals such as prostaglandin, bradykinin, capsaicin, serotonin, and histamine that sensitize muscle afferent nerve fibers[10], which in turn, increase ACh release, sustaining the cycle. The affected muscle is painful to stretching which makes the patient protect the muscle through poor posture and sustained contraction[12].

1.1.1.1 Clinical Features of Myofascial Pain

The clinical features of myofascial pain are palpable taut band of muscle with localized tenderness, a characteristic pain referral pattern occurring when pressure is applied and a local twitch response to snapping palpation of the trigger point[9]. Patients who have active trigger points usually report regional and persistent pain that often results in a decreased range of motion while patients with latent trigger points present with hypersensitivity only when subjected to palpation[13,14]. Mechanical stimulation of the ‘taut band’ by needling or rapid transverse pressure often elicit a local twitch response (LTR)[15] defined as brisk contraction by

a group of muscle fibers in a taut band containing a trigger point[16]. The LTR has been described as a spinal reflex[17] indicating that its transmission depends mainly on the central nervous system with a possible minor degree of local transmission.

TMDs may present with trigger points in masseter, pterygoid, upper trapezius and upper sternocleidomastoid muscles[18]. Active myofascial trigger points in the face and neck can produce tension-type headaches[19,20] and migraines[18]. The zone of reference of trigger points in sternocleidomastoid, trapezius, splenius capitis and anterior temporalis are localized in the supraorbital areas and the anterior temporalis, trapezius and splenius capitis are in the temple area[13].

1.1.2 OSTEOARTHRITIS

Osteoarthritis (OA), also referred to as degenerative joint disease (DJD), results from an imbalance between degenerative and reparative processes. This results in the progressive degradation of extracellular matrix (ECM) of cartilage with secondary inflammatory components[21]. If the functional demands of the TMJ complex are exceeded, the collagen fibrils in the articular tissues are fragmented allowing the proteoglycan-water molecules to swell which results in softening of the articular surface, a condition termed as chondromalacia[22]. This condition is reversible. However, if excessive loading continues, regions of fibrillation develops,

resulting in focal roughening of the articular surfaces and breakdown of subchondral tissues[22]. These osteoarthritic changes alter the lubricating properties of the articular surface and may lead to adhesion of the articular surfaces, causing strains on the discal ligaments and eventually lead to disc displacements. De leeuw et al[23] observed that in TMJs with reducing disc displacement, no or mild radiographically visible destructive changes develop, even if this condition persists for several decades. On the other hand, in TMJs with permanent disc displacement, extensive radiographically degenerative changes are frequently visible [23].

1.1.2.1 Clinical Features of Osteoarthritis

Osteoarthritis is diagnosed clinically by the presence of joint pain, tenderness, crepitation and limitation of mandibular movements. Radiographically, it presents with distinct osseous changes such as erosions and osteophytes[24]. Erosions represent the early stage of osteoarthritis and are seen radiographically as decreased density of the cortical and subcortical layers of the condyle[25]. Osteophytes are formed as adaptive response in an attempt to stabilize the joint and withstand undue joint loading[26]. According to the appearance of condyle on transpharyngeal radiographs, osteoarthritis has destructive phase characterized by erosion and a reparative phase by remodeling processes. Bone changes take place within 1-3 years followed by repair. Once repair occurs, the healing is stable[27]. Kurita et al[28] showed that

radiographically visible degenerative changes were arrested or slowed if the symptoms and signs were resolved or reduced.

1.1.3 INTERNAL DERANGEMENT

Disturbance in the normal anatomical disc-condyle relationship results in internal derangements of the TMJ[29]. Internal joint derangement is an orthopedic term defined as a localized mechanical fault interfering with smooth joint movement[30]. Structural surface irregularities, ankylosing conditions (adherence, adhesion, ankylosis), disc-condyle derangements (subluxation, luxation), loose bodies, and disc derangements (disc displacement, disc perforation)[31] are types of internal derangement of the TMJ. Disc displacement is the most common[32,33] and the most frequent subtype is anterior and/or medial displacement[34]. Lateral displacements also occur[33]. A few cases of posterior displacement have also been reported[35].

1.1.3.1 Clinical Features of Internal Derangement

Internal derangement alters the normal function of the TMJ which can be observed from its clinical presentations. The frequent clinical signs and symptoms are joint sounds, mandibular deviation or deflection and limitation of the range of mandibular movements[36], joint pain and masticatory muscle pain[37].

1.2 PROBLEM STATEMENT

Disc displacement of the TMJ may develop early in life[38]. According to Nebbe et al[39], it is present in 46% and 36% symptomatic adolescent males and females respectively. It may produce joint sounds on mouth opening and/or closing[40] and decreased mouth opening[3]. It may progress and cause TMD signs and symptoms that may fluctuate with time[41] and increase in frequency and severity in the second decade of life[42]. Severe TMD signs and symptoms do not improve spontaneously and may be refractory to treatment[43].

Historically, disc displacement of the TMJ was described as a progressive disorder[44-46]. In the 1950s, Ireland[47] believed that disc displacement typically progressed to osteoarthritis. Subsequent publications[48-50] theorized that disc displacement exposed the underlying articular tissues to excessive loading which inevitably led to degenerative changes. On the other hand, longitudinal data revealed that disc displacement may not progress in majority of cases[51]. The signs and symptoms associated with disc displacement with reduction and without reduction often improve over time without therapy[52,53]. Although epidemiological evidence shows that disc displacement is not a progressive condition in the majority of cases[51-53], it is not clear which patients have the greatest risk for progressing to more advanced stages[54]. Therefore, understanding the course of disc

displacement and its associated signs and symptoms is important clinical information.

Several researchers[52,53] examined the natural course of disc displacement and assessed its relationship with TMD signs and symptoms with the use of subjective assessment of TMJ MRI. Whether this method can precisely estimate the severity of disc displacement is subject to debate. The relative position of the disc can be influenced by the depth of the fossa, the slope of the articular eminence, inclination of the head of the condyle and ascending ramus of the mandible[55]. Therefore, quantitative analysis of MRI disc displacements has been suggested in lieu of subjective evaluation[55-58]. To date, there is no published study on this subject in adolescents. This study aims to examine predictive value of quantitative MRI disc displacement for the changes of TMD signs and symptoms in a pre-orthodontic adolescent patient population.

1.3 RESEARCH QUESTION

Are the changes in TMD signs and symptoms over time related to baseline MRI disc displacement?

1.4 NULL HYPOTHESIS

The changes in TMD signs and symptoms over time are not related to baseline MRI disc displacement.

1.5 LITERATURE REVIEW

1.5.1 TMJ ANATOMY

Temporomandibular joint is the articulation formed by the mandibular condyle with the glenoid fossa and articular eminence of the temporal bone. The glenoid fossa is formed by the squamous portion superiorly and the tympanic portion of the temporal bone posteriorly. The articular eminence emanates from the inferior surface of the zygomatic process of the temporal bone. Interposed between the bony structures is the articular disc. The condyle is the part of the mandible which approximates the under-surface of the disc. The part of the temporal bone which approximates the upper surface of the disk is the glenoid (or mandibular) fossa. The articular disc is biconcave with a thin intermediate zone, a thick anterior band and a thicker posterior band. The disc-condyle relationship of the TMJ at maximum inter-occlusal position is considered optimal if the posterior band of the articular disc is directly above the mandibular condyle (i.e. 12 o'clock position) and the intermediate zone is interposed between the head of the mandibular condyle and the posterior slope of the articular eminence of the temporal bone[59-61].

1.5.2 OSSEOUS TMJ ARTICULAR TISSUES

The osseous articular surfaces of the TMJ are covered by a layer of fibrocartilage[62] which contains few chondrocyte-like and fibroblast-like

cells which are responsible for extracellular matrix (ECM) formation and degradation[63]. The ECM contains water, collagen, proteoglycans, structural glycoproteins, and small amounts of lipid and inorganic components[64]. The collagen network provides tensile strength and the proteoglycans provide compressive properties to the articular cartilage[65,66]. The fibrocartilage of the condyle and articular eminence is aneural and avascular. Thus, pain is not perceived directly from these tissues. Nourishment is provided by the synovial fluid which bathes and rinses the articular surfaces[67]. Maintenance of the fibrocartilage layer via continuous turn-over and replacement is essential for function and health of the TMJ throughout life.

1.5.3 TMJ ARTICULAR DISC

The articular disc is biconcave avascular fibrous dense collagenous connective tissue, with little capacity for repair. It is attached to the medial and lateral poles of the condyle, thereby, divides the joint cavity into upper and lower compartments and absorbs the biomechanical stresses associated with jaw movements[68,69]. The condyle closely engages the posterior surface of the articular eminence with the intermediate zone of the disc interposed when the teeth are in contact[22]. The mandibular condyle rotates in the lower joint space beneath the disc and translates in the upper joint space along the temporal fossa and eminence during opening jaw movement through. The TMJ is thus referred to as a

ginglymoarthrodial joint. These movements are carried out by the muscles of mastication and are controlled by the 5th cranial nerve. The temporalis, medial pterygoid and masseter facilitate jaw-closing movement while the lateral pterygoid and suprahyoid muscles, the opening movement. The lateral pterygoid muscle and part of the fibers of the masseter and medial pterygoid muscles aid in the anterior translation of the mandible.

1.5.4 PATHOGENESIS OF DISC DISPLACEMENT

Disc displacement may result from overextension of the jaw during intubation procedures[70], wide opening of the mouth (yawning) [71], third molar extraction and long dental appointment. These events potentially lead to elongation of discal ligaments and consequently, disc displacement.

The most common etiological factor of disc displacement is trauma. There are two types of trauma: macro or micro-trauma. Macro-trauma involves a direct soft tissue overload while the latter represents repetitive cumulative trauma to the bone and soft tissues. A direct blow to the chin when the teeth are separated is a type of macro-trauma which displaces the condyle from the glenoid fossa and causes elongation of discal ligaments and disc displacement.[67].

Clenching and/or bruxing is a repetitive and extended type of micro-trauma which excessively loads the articular cartilage and exhausts its capacity to maintain and regenerate. Clenching produces intra-articular pressure in the TMJ up to >40 mm Hg, which exceeds normal joint capillary perfusion pressure[72]. Consequently, blood flow is transiently disrupted, resulting in tissue hypoxia[73]. When the inter-articular pressure is returned to normal, there is a reperfusion phase. It is theorized that during this phase, free radicals are released into the synovial fluid which rapidly break down the hyaluronic acid that protects the phospholipids that line the joint surfaces and provide lubrication[74,75]. When the phospholipids are lost, the articular surfaces no longer slide smoothly leading to sticking and breakdown of the articular tissues which eventually lead to disc displacement[76]. With loss of normal repair, tissue degradation occurs with articular tissue fibrillation, loss of tissue compessiveness, loss of proteoglycans and formation of micro-adhesion[77,78].

Another type of micro-trauma results from mandibular orthopedic instability. Mandibular orthopedic instability exists when the intercuspal position of the teeth is not in harmony with the musculoskeletally stable position of the condyles[22] when the elevator muscles loads the TMJ. During mandibular loading, unusual movement between the disc and condyle occurs if the condyle is not in stable relationship with the disc and fossa. This movement is often a translatory shift between disc and condyle

and can lead to elongation of the discal ligaments, thinning of the disc and consequent disc displacement[67].

1.5.5 DIAGNOSIS OF DISC DISPLACEMENT

1.5.5.1 RADIOGRAPHIC DIAGNOSIS

Arthrography and magnetic resonance imaging (MRI) have been utilized to determine the presence or absence of disc displacement. Arthrography has a high diagnostic value for abnormal disc positions but it is an invasive procedure. Compared with MRI, it has lower inter-observer agreement in identifying anterior disc displacement[79]. On the other hand, the diagnostic accuracy of MRI acquired from imaging fresh autopsy material using oblique sagittal and oblique coronal sections is 95%[80]. It has high sensitivity and specificity for the diagnosis of disc-condyle malrelationship[81-83]. Therefore, MRI has been considered as the gold standard for the diagnosis of disc displacement of the TMJ. It can provide essential information about the position[84-86], signal intensity[86,87], morphology[87,88] and structure of the disc[85,88]. It is non-invasive, painless, and free of ionizing radiation. However, this procedure is uncomfortable for patients who are obese and claustrophobic or for those who cannot stay motionless during the procedure. It is contraindicated in patients who have pacemakers, intracranial vascular clips, and metal particles in their eyes or other vital structures.

1.5.5.2 CLINICAL DIAGNOSIS

Although MRI is an excellent tool for the diagnosis of TMJ disc displacement, it is expensive and time consuming. Thus, clinical examination and patient's history are often utilized to establish the working diagnosis[89,90]. The quality and quantity of mandibular opening and closing as well as the presence of mouth opening deviation and deflection are assessed. The joints are assessed by digital palpation or with stethoscope to determine the presence of joint sounds. Joints with reciprocal clicking show increased probability of disc displacement with reduction[40]. However, an arthrographic study by Rohlin et al[91] it was revealed that anterior displacement with reduction may exist without joint noises. Tasaki et al[92] examined 57 symptom-free volunteers with the use of TMJ MRI and reported that nearly one third of the volunteers had disc displacement in one or both joints. These studies suggest that disc displacement is not always associated with TMD signs and symptoms. Disc displacement alone is therefore not the only factor in the development of TMJ pain and dysfunction. Westesson and Brooks[93] indicated that the prevalence of magnetic resonance evidence of joint effusion is more strongly associated with pain than disc displacement alone.

The accuracy of diagnosing disc displacement by clinical examination alone is 43% - 75%[89,90]. But, when manipulation (force is applied to direct the condyles supero-anteriorly toward the posterior slope of the

eminences during mouth opening and closing movements) and elimination (patient is directed to open and close the mouth at a protruded jaw position) were performed, the diagnostic accuracy of clinical examination was 90%[94]. However, neither the degree of displacement nor the degree of deformation of the disc could be disclosed by clinical examination alone[40]. This information can only be obtained with diagnostic imaging. Therefore, if a patient's care relies on information that is beyond that which can be gained from a clinical assessment, TMJ imaging like MRI should be utilized[95]. The decision to image is made after considering the patient's history, clinical findings, cost of the examination, and results of prior examinations, treatment planning and expected outcome[96].

1.5.6 SIGNS & SYMPTOMS OF DISC DISPLACEMENT

A displaced disc alters the normal function of the TMJ[22]. Disc displacement with reduction can produce clicking during opening as well as closing of the mouth (reciprocal click)[3]. Mandibular deviation on mouth opening may occur[60] secondary to disc reduction and associated ipsilateral muscle adaptive activity. Isberg et al[97] stated that electromyographic activity of the temporalis and masseter muscles occurred when the condyle slid over the posterior band of the disc and could be interpreted as arthrokinetic reflex. Disappearance of clicking, followed by an onset of limited mouth opening and mandibular deflection may indicate a progression of anterior disc displacement with reduction to

disc displacement without reduction[3]. Anterior disc displacement without reduction may be associated with ipsilateral masseter and temporalis muscle spasm which may hinder condylar movement[97]. Disc displacement with or without reduction may not always be accompanied by pain[22]. Joint clicking, joint pain and masticatory muscle pain are common in adolescent population[37,98-101].

Based on 17 epidemiological studies, the average adult prevalence of TMD with at least one symptom was 41% and with at least one clinical sign presentation was 56%[22]. Solberg et al[102] examined 739 university students (aged 18 to 25) and found that 75% of the students had one or more TMD signs, 10% of the total group had symptoms severe enough to request treatment but only 5% were found to have severe symptoms requiring treatment. The prevalence of TMD in an adult Asian population according to subtypes was 31.4% for muscle disorders, 15.1% and 15.7% for left and right disc displacement respectively, and 12.6% and 13.0% for left and right joint degenerative conditions respectively[103]. This study indicated that muscle disorders were the most prevalent type of TMD. This finding is consistent with similar Swedish and American studies[104].

1.5.7 MALOCCLUSION & TMD SIGNS AND SYMPTOMS

The association between occlusal characteristics and signs and symptoms of TMJ dysfunction have been analyzed in children and young adults[36,101,105,106]. Riolo et al[105] reported that patients with excessive or negative overjet were more likely to have joint tenderness and patients with cusp to cusp or a Class II molar relationships were more likely to have joint and muscle tenderness[105]. TMJ clicking was positively correlated with dental wear and unilateral contact in the retruded contact position but was most influenced by age and sex[36] (i.e., TMJ clicking increased with age and more prevalent in girls than in boys). Based on cross-sectional data obtained from 1,342 subjects 6 to 17 years of age, Riolo et al[105] showed that joint sounds were positively associated with Class II molar relationship in the 6 to 8 and 15 to 17-year age groups. In contrast, Keeling et al[101] using data obtained from 3428 grade school children, showed that the prevalence of joint sounds was not associated with age, gender, or molar class. The seeming contradictory may be due to the multifactorial etiology of disc displacement. Nevertheless, morphologic malocclusion such as Class II and Class III occlusion, anterior open-bite, and cross-bite, when associated with occlusal interference, may create a predisposition to mandibular dysfunction[36].

1.5.8 ORTHODONTICS & TMD SIGNS & SYMPTOMS

Some studies report that adolescents who received orthodontic treatment had less signs and symptoms of TMD than those who did not have the treatment[107]. It is claimed that if malocclusion is corrected to support normal jaw function, TMD symptoms can be treated[108]. While there are clinicians who presented with good treatment results[109-111], some argued that orthodontic treatment can actually increase the risk for developing TMD[112,113]. It was hypothesized that the occlusal scheme brought about by premolar extraction and maxillary incisors retraction lead to posterior displacement of the condyle, thus resulting in internal derangement of the TMJ[114-116]. This theory was invalidated by showing eccentric condylar positions in many asymptomatic patients[117,118]. Subsequent studies showed no significant differences in condylar position before and after orthodontic treatment with premolar extraction[119-121].

Opinions on the relationship between orthodontics and TMD continue to differ between those who have emphasized that orthodontic treatment increases the risk for TMD and those who maintain that it may decrease such risk. Controversy exists because of the following reasons: 1) multi-factorial nature of TMD; 2) the natural course of TMD is not fully known because epidemiological studies examined mostly cross-sectional data; 3) there is no reliable and valid diagnostic classification system for TMD. A recent meta-analysis examined the literature published from 1966 through

September 2000 with regard to the relationship of orthodontics with TMD[122]. The researchers found 960 related articles but only 38 met inclusion criteria. The selected studies indicate that traditional orthodontic treatment did not increase the prevalence of TMD. The evidence shows no definite link between orthodontics and TMD.

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CHAPTER 2

Predictive Value of Magnetic Resonance Imaging of Disc Displacement for Temporomandibular Disorder Signs and Symptoms in a Pre-orthodontic Adolescent Patient Population

2.1 INTRODUCTION

Disruption of the condyle-disc relationship can lead to disc displacement of the TMJ which has been classified into disc displacement with reduction (DDWR) and disc displacement without reduction (DDWOR). DDWR occurs when the mandibular condyle moves over the posterior border and onto the intermediate zone of the displaced articular disc during mouth opening[1]. Joints with a reciprocal or mouth opening/closing click are more consistently correlated clinically with DDWR[2]. DDWOR occurs when the condyle does not assume a normal relationship with the disc during mouth opening[1]. This condition may be associated with a limitation of mouth opening and/or joint pain. Disc displacement without a “click” or crepitation may also be associated with DDWOR[2]. It is possible that the condyle can translate normally and does not produce signs such as limited opening or clicking on mouth opening. However, this type of disc displacement may also be associated with episodic catching on mouth movement or decreased mouth opening.

In the past, clinicians believed that disc displacement and TMD pain have causal relationship. Hence, disc displacement was generally treated by repositioning the disc to a normal position via surgery[3] or with anterior repositioning splints (ARS)[4,5]. Short-term follow-up studies showed that these procedures were effective[6-8]. Moloney and Howard[7] treated 241 patients who had disc displacement with reduction using an anterior

repositioning splint worn 24 hours a day over a four month period and showed 70% of the patients experienced elimination of the click and associated pain. The findings supported the belief that recapturing the disc to normal position must be an essential part of the treatment. However, disc repositioning procedures have been associated with bite changes requiring comprehensive and invasive dental treatment including orthodontics and/or prosthodontics. These procedures, however, have a high degree of relapse[9]. In addition, it appeared that ARS did not recapture the disc or decompress the joint structures when investigated with TMJ imaging[10]. Manco and Messing[11] utilizing computed tomography revealed that 41.8% of the patients did not have disc recapture. Manzione et al[12] found that 46% of the subjects still had disc displacement despite employing arthrographically guided splint therapy to reposition the TMJ articular disc. A study of Kirk[13], using MRI found only 11.1% of disc recapture with the appliance in place. It was concluded that the concept of "disc capture" is only a clinical term, and that the perceived decrease in joint noises following splint insertion may be due to increased joint space allowing improved condylar translation beyond disc surface irregularities and positional abnormalities.

Because disc displacement occurs frequently in non-patient populations[14,15], and its signs and symptoms generally improve over time with little to no therapy[16,17], invasive disc repositioning interventions

appear to be unnecessary in most cases. Reversible and conservative therapies which do not involve disc repositioning techniques are found to obtain symptom improvement[9,18,19] and are now supported. In view of the natural and longitudinal fluctuations of TMJ internal derangement, when there is a demand for treatment, the National Institute of Health (NIH)[20] proposed that reversible treatment should be employed as first line treatment. For example, an anterior repositioning splint can be used to prevent the condyle from articulating on the highly vascularized and well innervated retrodiscal tissues during an acute phase of internal derangement. During the forward position of the mandible, the retrodiscal tissues undergo adaptive and reparative changes[21,22] resulting in a fibrosis of the connective tissues that can be loaded without pain[23]. The patient is weaned off the anterior repositioning splint to prevent irreversible bite changes[23] and the mandible allowed to return to its normal position in the fossa (musculoskeletal stable position). If bruxism is evident, the patient is maintained with a stabilization appliance for sleep time use[23]. In the rare circumstance that a patient has persistent, nonremitting signs and symptoms, a stepwise approach and more non-reversible approach may be required[20].

It remains controversial whether TMJ disc displacement will predispose and/or contribute to TMJ-related pain and dysfunction[24]. Although disc displacement is not always accompanied by pain[1], it may progress and

become symptomatic with time. Therefore, the ability to predict the changes and progression of signs and symptoms associated with TMJ disc displacement is valuable clinical information.

The relationship of the natural course of disc displacement of the TMJ and TMD signs and symptoms has been mostly studied by subjective evaluation of TMJ MRI[16,17]. However precise localization of the disc is not possible with mere subjective evaluation of TMJ MRI[25]. Nebbe et al[26] noted that subjective MRI evaluation can be influenced by contiguous TMJ structures such as the glenoid fossa, articular eminence, head of the condyle and ascending ramus of the mandible. Therefore, quantitative assessment of MRI disc displacement was suggested in lieu of subjective evaluation[25-28]. To date there are no studies that have assessed the association between quantitative MRI disc displacement in adolescent and changes of TMD signs and symptoms over time. The objective of this retrospective study was to examine the predictive value of MRI disc displacement for the changes of TMD signs and symptoms over 3 to 4-year interval.

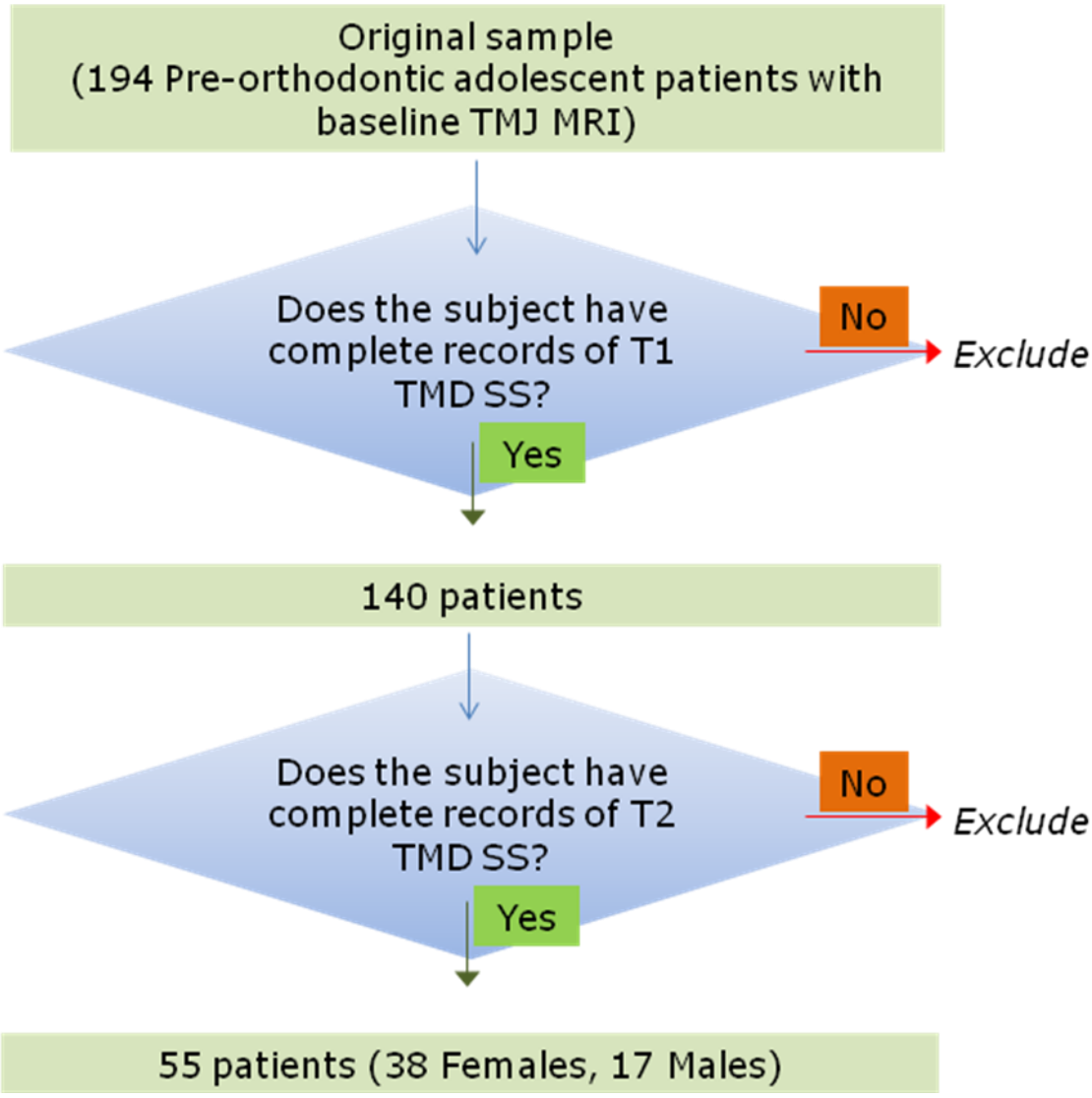
2.2 MATERIALS AND METHODS

2.2.1 SUBJECTS

The scheme for subject selection for this study is presented in Figure 2.0. Subjects in this study were part of the original sample of Dr. Brian Nebbe's (BN) PhD thesis at the University of Alberta, Edmonton, Alberta, Canada. The original samples were recruited among individuals who were seeking orthodontic treatment in either a private orthodontic clinic in Edmonton or the post-graduate orthodontic program at the University of Alberta. Individuals with a history of juvenile rheumatoid arthritis, whiplash injury or previous TMD treatment and/or orthodontic treatment were excluded. This resulted in a study sample which consisted of 194 adolescent subjects with and without clinically detectable TMJ signs and symptoms (capsular pain; joint sounds; masticatory muscle tenderness; limited mandibular range of motion; deviation on opening). Baseline TMJ MRI was taken and clinical examinations were performed for these subjects with the approval from the Joint Dentistry/Pharmacy Human Ethics Committee. Of these subjects, 160 had baseline TMJ MRI as well as initial (T1) clinical records of TMD signs and symptoms. However, 20 subjects were found to have incomplete T1 records. From the remaining 140 subjects, 55 subjects had complete records of both T1 and T2 TMD signs and symptoms. Thirty-eight (69.1%) were females and seventeen (30.9%) were males with a mean age of 12.9 ± 1.6 years (range 7.4-16.6) and born between 1979 and 1988. The mean interval between initial (T1) and follow-up (T2)

clinical records was 4.2 ± 0.4 years (range 3.6 - 4.9) for data collected between November 1995 and January 2000. Possible reasons for lost to follow-up between T1 and T2 were: Not able to be contacted at T2; declined participation at T2, inadequate records at T2 and medical issues.

FIGURE 2.0
SUBJECT INCLUSION-EXCLUSION CRITERIA

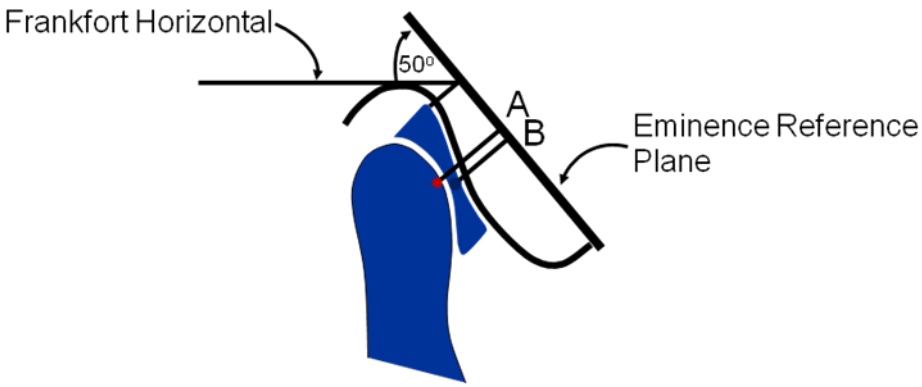


2.2.2 MEASUREMENT OF DISC DISPLACEMENT

Disc displacement (DD) was measured in TMJ MR Images as the total linear distance (in millimeters) between the discal midpoint and the condylar load point parallel to the eminence reference plane. This Eminence Reference Plane of 50 degrees to the Frankfort Horizontal was applied to all traced slices of the TMJ, at a point 10 mm anterior to the maximum height of the glenoid fossa. This measurement scheme by Nebbe et al[26] is presented in figure 2.1. For the present study, measurements were conducted independently for right and left TM joints utilizing a central sagittal MRI slice of the disc at baseline. The complete details of quantitative measurement can be obtained from the previous publication of Nebbe et al[26].

FIGURE 2.1

MEASUREMENT SCHEME FOR MRI DISC DISPLACEMENT



DD was measured from condylar load point (A) to midpoint of intermediate zone (B) along the eminence reference plane. This Eminence Reference Plane of 50 degrees to the Frankfort Horizontal was applied to trace slices of the TMJ, at a point 10 mm anterior to the maximum height of the glenoid fossa. With permission from: Quantitative analysis of temporomandibular joint disk status. Nebbe et al. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998:85:598-607

2.2.3 EXAMINATION OF TMD SIGNS & SYMPTOMS

In this study, TMD signs and symptoms were identified independently for right and left TM joints as cervical muscle pain, masticatory muscle pain, joint clicking, joint locking, crepitus, joint pain and limited mandibular movement. Nominal scores (0=absent, 1=present) were assigned to each sign or symptom at T1 and T2[26] and presented in Table 2.0. Methods for determining TMD signs and symptoms are described below:

2.2.3.1 Cervical muscle pain (CMP)

The presence or absence of tenderness of the superior, middle, inferior sternocleidomastoid (SCM), upper trapezius, trapezius insertion was determined via bilateral digital palpation. CMP was scored present (1) if one or all these muscles were tender.

2.2.3.2 Masticatory muscle pain (MMP)

The presence or absence of tenderness of temporalis and masseter was determined via bilateral digital palpation. MMP was scored present (1) if one or all these muscles were tender.

2.2.3.3 Joint clicking

TMJ clicking (snapping, popping, or cracking sounds of short duration as differentiated from grating or grinding sounds) was determined via stethoscope during mouth opening and closing and scored as either present (1) or absent (0).

2.2.3.4 Joint locking

History of joint locking (previous or current closed joint lock) was determined through subjective questioning and scored as either present (1) or absent (0).

2.2.3.5 Crepitus

Crepitus (defined as multiple grating or grinding sounds during mouth opening and closing as differentiated from reciprocal or non-reciprocal clicking) was determined using a stethoscope and scored as either present (1) or absent (0).

2.2.3.6 Joint pain

Joint pain (tenderness at the superior, posterior and lateral joint regions) was determined via digital palpation and scored as, either present (1) or absent (0).

2.2.3.7 Limited mandibular movements

Limited mandibular movements (assisted/unassisted vertical mouth opening, right/left lateral excursion and protrusion) were determined by making use of a millimeter ruler, accurate to the nearest 0.5 mm and for the purpose of this study were scored as either present (1) or absent (0). A limited mandibular movement was scored 1 when jaw movement was below a normal range. The normal values according to craniomandibular index by Fricton and Schiffman[29] are presented in Table 2.0.

2.2.3.8 Mandibular deviation

Deviation of the jaw on mouth opening was measured from maxillary midline to mandibular midline and scored as either absent (0) or present (1). Deviation greater than 2 mm was scored 1.

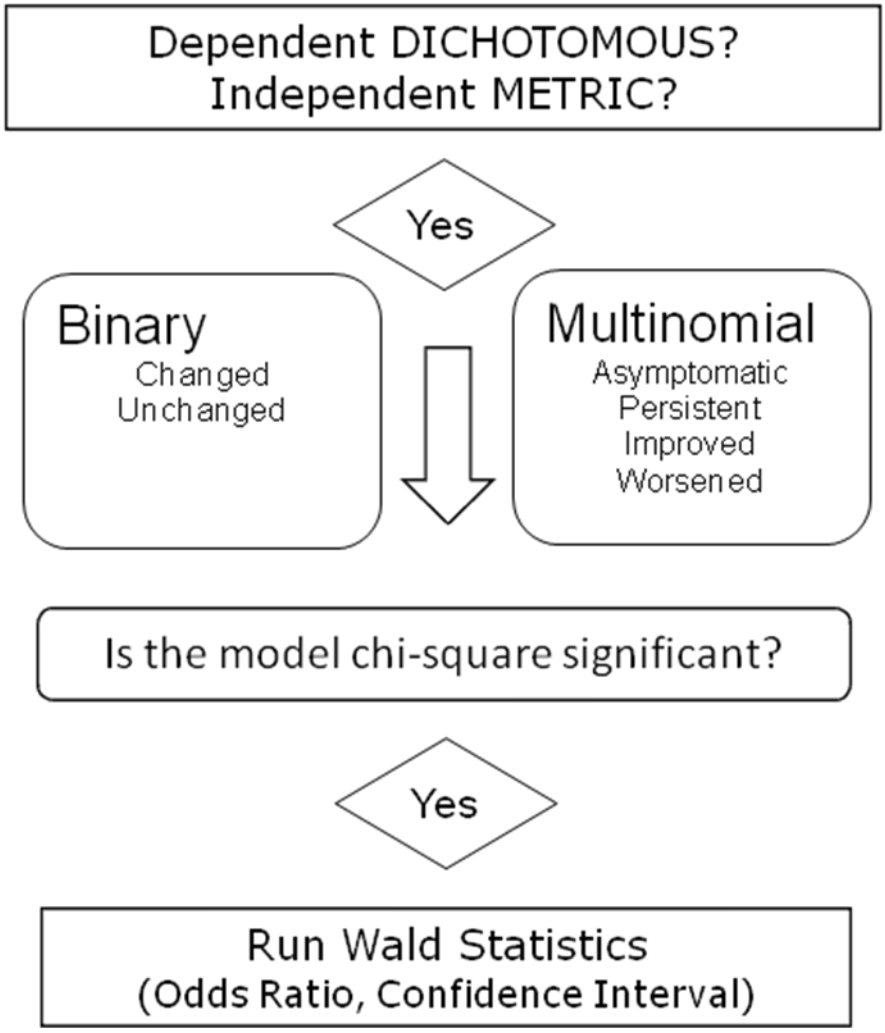
TABLE 2.0
TMD SS NOMINAL SCORES

TMD Signs and Symptoms	Method of Examination	Variable
Cervical Muscle Pain (CMP) Superior SCM Middle SCM Inferior SCM Upper Trapezius Trapezius insertion	Digital Palpation	Tenderness absent (0) Tenderness present (1)
Masticatory Muscle Pain (MMP) Masseter Temporalis	Digital Palpation	Tenderness absent (0) Tenderness present (1)
Joint pain Superior joint Lateral joint Posterior joint	Digital Palpation	Tenderness absent (0) Tenderness present (1)
Joint Clicking Joint Crepitus	Stethoscope	Absent (0) Present (1)
History Joint Locking	Subjective response to questioning	Absent (0) Present (1)
Mandibular Movements (normal values) Unassisted opening (40mm+) Assisted opening (42mm+) Lateral excursion (7mm+) Protrusion (7mm+)	Millimeter ruler	≥ normal value (0) < normal value (1)
Mandibular Deviation (Normal: ≤2mm)		≤ 2mm (0) > 2mm (1)

2.3 Statistics

Fisher's exact tests were performed to examine the frequency of presenting signs and symptoms in males and females. To examine the predictive value of quantitative disc displacement for changes of TMD signs and symptoms from T1 to T2 logistic regressions (binary and multinomial) were performed (Figure 2.2). The first step in both binary and multinomial logistic regressions is to compute the chi-square statistic. The significance test for the chi-square statistic is the statistical evidence of the presence of a relationship between the dependent variables (changes of TMD signs and symptoms) and independent variable (quantitative measures of MRI disc displacement). If the model chi-square test was significant, Wald test was performed to calculate the odds ratio and confidence interval (Figure 2.2). Wald test evaluates whether or not the independent variable is statistically significant in differentiating between the categories defined by the dependent variables. Both binary and multinomial logistic regressions do not make any assumption of normality, linearity and homogeneity of variance for the independent variables[30]. The statistical significance was set at alpha=0.05 with Bonferroni correction.

FIGURE 2.2
STEPS IN LOGISTIC REGRESSION



2.4 Results

Limited mandibular movements and associated right and left mean disc displacement in millimeters are presented in table 2.1A. Patients with limited mandibular movements were not common at T1 and T2. At baseline, clicking was the most common sign while joint pain and masticatory muscle pain were the most common symptoms (Table 2.1B). The signs and symptoms decreased in frequency over time except for right clicking and right crepitus (Table 2.1B). The frequencies of TMD signs and symptoms according to gender are presented in table 2.2. Although females were more likely than males to present with TMD signs and symptoms (Table 2.2), the frequencies of TMD signs and symptoms were not statistically different from expected frequencies at T1 and T2. Therefore, the subjects were not divided according to gender in logistic regression analyses.

TABLE 2.1A

FREQUENCIES OF LIMITED MANDIBULAR MOVEMENTS AND ASSOCIATED MEAN AND SD OF MRI DD AT T1 AND T2

	T1				T2		
		Mean (SD)				Mean (SD)	
Limited Mand. Movts.	n	RDD (mm)	LDD (mm)		n	RDD (mm)	LDD (mm)
Unassisted Opening	2	2.7 (1.3)	6.1 (3.4)		2	3.6 (2.6)	2.4(1.9)
Assisted Opening	2	2.7 (1.3)	6.1 (3.4)		1	5.9	0.7
Right Laterotrusion	4	2.0 (2.3)	2.4 (4.1)		3	4.2 (3.2)	4.8 (3.5)
Left Laterotrusion	1	3.6	8.5		1	1.1	2.8
Protrusion	6	1.1 (1.4)	2.7 (3.5)		5	2.4 (3.0)	0.8 (0.3)
Deviation	4	3.2 (1.7)	4.7 (3.1)		3	3.0 (2.6)	3.1 (2.6)
N=55; RDD=right disc displacement; LDD-left disc displacement							

TABLE 2.1B

FREQUENCIES OF TMD SIGNS AND SYMPTOMS AND ASSOCIATED MEAN AND SD OF DISC DISPLACEMENT

	T1		T2	
	n	Mean (SD)	n	Mean (SD)
RIGHT		RDD (mm)		RDD(mm)
Clicking	16	4.0 (2.6)	20	3.3 (2.5)
Locking	6	3.1 (2.9)	6	3.2 (2.6)
Crepitus	2	2.1 (2.9)	3	2.7 (2.3)
CMP	11	2.0 (2.6)	7	3.1 (3.5)
MMP	20	2.6 (3.5)	14	2.4 (2.4)
Joint Pain	22	2.3 (2.3)	10	2.7 (3.3)
LEFT		LDD (mm)		LDD (mm)
Clicking	22	3.1 (2.7)	15	3.0 (2.8)
Locking	6	2.7 (2.1)	5	3.8 (2.7)
Crepitus	6	2.2 (2.7)	4	2.1 (1.8)
CMP	12	3.2 (3.4)	8	3.0 (3.1)
MMP	20	2.7 (2.8)	13	2.4 (2.2)
Joint Pain	21	1.9 (2.1)	15	2.8 (2.5)
N=55				

TABLE 2.2
FREQUENCY OF TMD SS ACCORDING TO GENDER

	T1			T2	
	Female	Male		Female	Male
RIGHT					
Clicking	14	2		15	5
Locking	5	1		5	1
Crepitus	2	0		1	2
CMP	9	2		5	2
MMP	15	5		12	2
Joint Pain	15	7		8	2
LEFT					
Clicking	20	2		12	3
Locking	5	1		4	1
Crepitus	5	1		2	2
CMP	10	2		5	3
MMP	15	5		10	3
Joint Pain	14	7		13	2
Limited Mandibular Movements					
U. Opening	2	0		2	0
A. Opening	2	0		1	0
R. Laterotrusion	3	1		3	0
L. Laterotrusion	1	0		1	0
Protrusion	5	1		5	0
Md. Deviation	4	0		3	0
Male: n=17; Female: n=38					

Based on nominal scores for TMD signs and symptoms at T1 and T2 (Table 2.0), the TMD signs and symptoms were categorized as either changed (1) or unchanged (0) (Table 2.3). The changed category was consisted of improved (1-0) and worsened (0-1) TMD signs and symptoms while unchanged category was consisted of persistent (1-1) and free (0-0) of TMD signs and symptoms from T1 to T2. The changed category was assigned as the target category and analyzed with binary logistic regression (Table 2.5A). Moreover, the TMD signs and symptoms were categorized as asymptomatic (0-0), persistent (1-1), improved (1-0) and worsened (0-1) (Table 2.4) and analyzed with multinomial logistic regression (Table 2.6A). Because the most frequent category was asymptomatic, it was selected as the reference category. The minimum number of cases per independent variable was set at 5. Since the categories of joint locking, crepitus, cervical muscle pain, joint pain and limited range of mandibular movements were consisted of less than 5 subjects, they could not be analyzed (Table 2.4).

TABLE 2.3

FREQUENCY OF CHANGED/UNCHANGED TMD SS FROM T1 TO T2

	Unchanged	Changed
RIGHT		
Clicking	41	14
Locking	47	8
Crepitus	50	5
CMP	43	12
MMP	39	16
Joint Pain	35	20
LEFT		
Clicking	35	20
Locking	48	7
Crepitus	45	10
CMP	41	14
MMP	36	19
Joint Pain	37	18
ROMMs		
Unassisted Op.	53	2
Assisted Op.	52	3
Right Laterotrusion	48	7
Left Laterotrusion	53	2
Protrusion	46	9
Deviation	48	7

TABLE 2.4
FREQUENCY OF CATEGORIES DEFINED BY NOMINAL SCORES OF TMD SS FROM T1 TO T2

	Asymptomatic	Worsened	Persistent	Improved
RIGHT				
Clicking	30	9	11	5
Locking	45	4	2	4
Crepitus	50	3	0	2
CMP	40	4	3	8
MMP	30	5	9	11
Joint Pain	29	4	6	16
LEFT				
Clicking	25	8	7	15
Locking	46	3	2	4
Crepitus	45	4	0	6
CMP	38	5	3	6
MMP	29	6	7	13
Joint Pain	28	6	9	12
ROMMs				
Unassisted Op.	52	1	1	1
Assisted Op.	52	1	0	2
R.Laterotrusion	48	3	0	4
L.Laterotrusion	53	1	0	1
Protrusion	45	4	1	5
Deviation	48	3	0	4

Binary logistic regression revealed that the chi-square test of RDD for ipsilateral joint locking was significant (p=0.002) (Table 2.5A). To determine whether or not changed joint clicking was significantly related to RDD, Wald statistics was performed (Table 2.5B). The results of Wald statistics revealed that RDD was significantly related (p=0.006) to joint locking (Table 2.6). The odds ratio between changed and unchanged joint locking was 1.691 with 95% confidence interval of 1.162 and 2.461. The classification accuracy rate was 81.8% (Table 2.5C).

TABLE 2.5A
BINARY LOGISTIC REGRESSION
OVERALL TEST OF RELATIONSHIP BETWEEN MRI DD AND CHANGES DEFINED BY TMD SS (CHANGED OR UNCHANGED)

Dependent	Independent	Chi^2	P
RIGHT (Changed/Unchanged)			
CMP	RDD	.281	.596
MMP	RDD	.556	.456
Clicking	RDD	.402	.526
Locking	RDD	10.077	.002
Crepitus	RDD	.039	.844
Joint Pain	RDD	.526	.468
LEFT (Changed/Unchanged)			
CMP	LDD	4.307	0.038
MMP	LDD	0.053	0.819
Clicking	LDD	3.801	0.051
Locking	LDD	4.04	0.044
Crepitus	LDD	0.008	0.931
Joint Pain	LDD	0.169	0.681
Bonferroni corrected α= 0.0083			

TABLE 2.5B
RELATIONSHIP BETWEEN MRI RDD & JOINT LOCKING FROM T1 TO T2

		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
Step 1[a]	RDD	.525	.191	7.539	1	.006	1.691	1.162	2.461
	Constant	-3.480	.898	15.019	1	.000	.031		

TABLE 2.5B

ACCURACY RATE OF MRI RDD IN PREDICTING CHANGES OF IPSILATERAL JOINT LOCKING FROM T1 TO T2

Observed		Predicted		
		LocRCh		Percentage Correct
		Unchanged	Changed	
Right Locking	Unchanged	45	2	95.7
	Changed	8	0	.0
Overall Percentage				81.8

Multinomial logistic regression revealed that the chi-square test of RDD for ipsilateral joint clicking was significant (p=0.003) (Table 2.6A). To determine which category of joint clicking was significantly related to RDD, Wald statistics was performed (Table 2.6B). The results of Wald statistics revealed that RDD was significantly related (p=0.002) to persistent clicking from T1 to T2. The odds ratio between persistent clicking group and reference group (asymptomatic category) was 1.758 with 95% confidence interval of 1.237 and 2.499. The classification accuracy rate was 65.5% (Table 2.6C).

TABLE 2.6A

MULTINOMIAL REGRESSION ANALYSIS

OVERALL TEST OF RELATIONSHIP BETWEEN MRI DD AND CATEGORIES DEFINED BY TMD SS (ASYMPTOMATIC, PERSISTENT, WORSENED AND IMPROVED)

RIGHT	Predictor	Chi^2	P
MMP	RDD	0.908	0.823
Clicking	RDD	13.858	0.003
Bonferroni corrected α= 0.025			
LEFT			
MMP	LDD	1.202	0.753
Clicking	LDD	7.435	0.059
Bonferroni corrected α= 0.025			

TABLE 2.6B

RELATIONSHIP BETWEEN MRI RDD & CATEGORIES OF IPSILATERAL JOINT CLICKING FROM T1 TO T2

Categories of Right Clicking								95% C.I.	
		Coef	SE	Wald	df	P	Odds Ratio	Lower	Upper
Improved (1-0)	Intercept	-2.74	0.808	11.506	1	0.001			
	RDD	0.42	0.214	3.841	1	0.05	1.521	1	2.314
Persistent (1-1)	Intercept	-2.51	0.698	12.927	1	0			
	RDD	0.564	0.179	9.891	1	0.002	1.758	1.237	2.499
Worsened (0-1)	Intercept	-1.52	0.511	8.9	1	0.003			
	RDD	0.191	0.18	1.123	1	0.289	1.21	0.85	1.722
Reference category: Asymptomatic (0-0)									

TABLE 2.6C

ACCURACY RATE OF MRI RDD IN PREDICTING CATEGORIES OF IPSILATERAL CLICKING FROM T1 TO T2

	Predicted				
Categories of R. Clicking	Improved	Persistent	Worsened	Asymptomatic	Correct
Improved	0	2	0	3	0.0%
Persistent	0	7	0	4	63.6%
Worsened	0	2	0	7	0.0%
Asymptomatic	0	1	0	29	96.7%
Overall Percentage	0.0%	21.8%	0.0%	78.2%	65.5%

2.5 Discussion

The range of mandibular movements is an important parameter with which to evaluate dysfunction and pain of the TMJ complex[31]. Reduced jaw movements may indicate intracapsular and/or muscular dysfunction[32]. Based on our data, it appears that there is no association between TMJ disc displacement and reduced mandibular range of mouth opening in an adolescent sample under investigation (Table 2.1A).

Williamson[33] examined 6 to 16-year-old pre-orthodontic patients and found that 35% had muscle tenderness or TMJ clicking. Gazit et al[34] utilizing a sample of 369 Israeli school children, reported that the most common sign of dysfunction were joint sounds (35.8%) which increased with age from 28% in the youngest to 44.3% in the oldest children. They also reported that the second most common symptom was joint sensitivity (30.4%), followed by sensitivity of superficial muscles (20%). Keeling et al[35] evaluated 3428 grade school Caucasian and African children (6-12 years old) and found that 10% of the subjects had TMJ sounds. Joint clicking, joint pain and masticatory muscle pain are also common in our sample (Table 2.1B). Variation in reported prevalences may be related to differences in methods of evaluation, subject characteristics, clinical examination procedures and diagnostic criteria[36].

Several studies suggest that disc displacement generally progresses to joint locking[37]. To test this hypothesis, Könönen et al[38] followed and evaluated by clinical examination 128 young Finnish subjects at ages 14, 15, 18, and 23 years. They found that reported joint clicking increased significantly with age, but no subject developed locking. Our study suggests that clinical evidence of TMJ disc displacement (clicking and locking) can remain unchanged, improve or worsen over a 4-year interval. Approximately half of the subjects without clicking at initial evaluation remained without clicking four years later (Table 2.4). Approximately 20% of subjects with right clicking at initial evaluation still had clicking at follow up and approximately 13% of subjects continued to have left TMJ clicking. Approximately 16% of subjects without right clicking at initial assessment had right clicking at the follow up assessment. Approximately 9% of right TMJs and 27% of left TMJs had resolution of clicking. Approximately 6% of joints developed locking, approximately 4% of joints had continued locking and approximately 7% had resolution of locking over the study period.

Based on binary logistic regression, RDD has significant association with the ipsilateral joint locking. It should be noted however that subjects with improvement and subjects who developed joint locking between T1 and T2 were grouped together under a single category (i.e., changed). Similarly, subjects with persistent joint locking were grouped with subjects

who consistently had no joint locking from T1 to T2 under a single category (i.e., unchanged). In the same manner, this grouping was applied to the rest TMD signs or symptoms under investigation in this study to increase sample size within groups and to assess if disc position had overall predictive value for changes of TMD sign or symptom. Recognizing that subjects who demonstrated improvement may have a different association with disc position than subjects who had worsened signs and/or symptoms, further analysis with multinomial regression was undertaken utilizing sign or symptom change subcategories. For statistical analysis, the minimum number of subjects per group to be included was set at 5 subjects. This prevented subcategories of joint locking, crepitus, joint pain, cervical muscle pain, and limited range of mandibular movements (Table 2.4) from being considered in the multinomial logistic regression. Thus, only the predictive value of disc displacement for clicking and masticatory muscle pain was analyzed (2.6A).

The multinomial logistic regression showed that RDD can distinguish subjects with persistent ipsilateral clicking from those who were persistently free of ipsilateral clicking from T1 to T2 (Table 2.6B). These findings indicate that clinical assessment of right joint clicking, over the study period, is consistent with MRI determination of disc displacement. Left disc displacement was not associated with ipsilateral clicking in our study. This may be related to the preference of the subjects to masticate

on the right side[39]. It has been shown that individuals who prefer to masticate unilaterally tend to develop ipsilateral temporomandibular dysfunction[40]. For instance, Dienberger et al[39] examined 4068 subjects by means of a questionnaire and reported that 1855 (45.4%) exhibited unilateral mastication and 1188 (64%) preferred the right side. They concluded that the masticatory side of preference was significantly associated with some asymmetric factors of the orofacial system such as unilateral pain in facial muscles or the TMJ, subjective unilateral joint clicking and asymmetric tooth loss.

Although this is the first study to evaluate the association of quantitative measurement of disc displacement with the natural course of TMJ signs of symptoms in adolescents, there are definite limitations. Unfortunately missing clinical evaluation data at T2 reduced the sample size, which limited the statistical analysis. To reduce the number of subgroups, muscle tenderness was classified as present or absent. Cases with a single region of muscle tenderness were grouped with cases with multiple regions of muscle tenderness. If sample size were larger, it would have allowed use of a tenderness of palpation index expressed as a ratio of tender sites to total number of sites palpated[29,41].

Disc displacement was assessed only in the sagittal plane using the center MRI slice. Medial disc displacements were not evaluated.

Furthermore disc displacement may occur in a rotational direction with more disc displacement at the medial or lateral pole than at the centre slice. Utilization of just anterior disc displacement at the center of the joint underestimates the true frequency of displacement. The findings of our study suggest that quantitative MRI disc displacement is not predictive of progression of TMD signs and symptoms in adolescents.

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CHAPTER 3

CLINICAL APPLICATION

3.1 INTRODUCTION

Internal derangement of the TMJ is a functional disorder frequently due to anterior and/or medial displacement of the articular disc[1]. Historically, disc displacement of the TMJ was described as a progressive disorder[2-4]. In the 1950s, Ireland[5] believed that disc displacement typically progressed to osteoarthritis. Subsequent publications[6-8] theorized that disc displacement exposes the underlying articular tissues to excessive loading which inevitably lead to degenerative changes. Hence, disc displacement was classified into four consecutive clinical stages[9]: Stage one was described as disc displacement with reduction; stage two as disc displacement with reduction and intermittent locking; stage three as disc displacement without reduction (closed lock); stage four as disc displacement without reduction and perforation of the disc (degenerative joint disease).

However, longitudinal data suggest that disc displacement of the TMJ may not progress in most cases[10]. Moreover, studies show that the signs and symptoms associated with disc displacement with reduction and without reduction often improve over time without therapy[11]. Sato et al[11] showed that reciprocal clicking remained unchanged in 79% and disappeared in 23.8% of their patient population. Four patients (16.7%) in whom clicking disappeared had a normal mouth opening, and only one patient developed locking (4.2%). Moreover, Kurita et al[12] followed 40 patients who had disc

displacement without reduction for 2.5 years without treatment. At the end of the 2.5 year follow-up period, 17 patients (42.5%) were asymptomatic, 13 (32.5%) had improved, and only 10 (25%) continued to be symptomatic or had requested treatment.

Although epidemiological evidence has shown that disc displacement is not a progressive condition in the majority of cases, it is not clear which patients have the greatest risk for progressing to more advanced stages. Therefore, understanding the natural course of disc displacement and its associated signs and symptoms is useful in diagnosis, treatment decisions and prognosis.

3.2 STUDY LIMITATIONS

The first limitation of the study pertains to sample size. The authors of Applied Logistic Regression[13], suggested 10 subjects as the minimum number of dependent variables per independent variable in logistic regression. Since most of the subjects were asymptomatic at T1 and T2, worsened, persistent and improved categories consisted of subjects less than 10 (Table 2.4).

The second limitation of the study was that the subjects were not obtained through random sampling. Randomization is important for two reasons: First, it provides a sample that is not biased, and second, it meets the

requirements for statistical validity[14]. Because the subjects were recruited through convenience sampling (i.e., from a pool of patients who were waiting for orthodontic treatment at the graduate orthodontic clinic in University of Alberta and in private clinics in Edmonton), our study sample might be biased. It is unlikely that the subjects selected had similar distribution of TMD signs and symptoms as the target population. Therefore we cannot generalize the results to the population and any statement generalizing the results beyond the actual sample tested must be stated with caution. The results may be extrapolated only to a targeted and narrowly defined population (i.e., preorthodontic adolescent patients).

The third limitation of this study was that disc displacement was analyzed in one dimension only (i.e., sagittal). Since the articular disc may also be displaced in latero-medially direction[15], evaluation of disc displacement in transverse direction[16] is equally important. Complementary coronal plane MR images have been suggested for optimum TMJ evaluation of rotational disc displacements[17]. This information was available on the MRI for the Edmonton sample and available for future study.

The fourth limitation of this study relates to rater reliability. Although a standardized examination chart was used to help obtain reproducible records of TMD signs and symptoms, the consistency of the rater in

acquiring this data was not tested for reliability or drift of clinical examination skill.

Lastly, limitations in mandibular movements were analyzed as dichotomous rather than continuous variables. There seems to be a general need in clinical practice to label individuals as having or not having an attribute of a disease. The most common argument seems to be simplicity. A binary split leads to a simple comparison of groups of individuals with high or low values of the measurement. Fricton[19] determined the optimal cut point for assisted mouth opening to be 42 mm, but this does not mean that 40 mm is abnormal. This dichotomization may yield misleading results since not everyone below the normal value is abnormal and subjects who have abnormal mandibular opening may also exhibit normal value. Various perceived advantages of dichotomizing continuous variables generally cannot be supported[18].

3.3 CLINICAL IMPLICATIONS

The data currently available from our study indicates poor association between clinical signs and symptoms and quantitative determination of internal derangement by MRI in the adolescent sample. Sample size may be the greatest limiting factor in this regard. Our analysis, however, showed that quantitative MRI disc displacement can distinguish subjects with persistent right clicking from those who were persistently free of

ipsilateral clicking from T1 to T2 (table 2.6). This finding suggests persistent relationship between the right clinical sign of joint sounds over time and initial determination of ipsilateral disc displacement by MRI. Based on our results, it appears that TMJ disc displacement does not necessary cause limitation of mandibular movements in majority of cases.

3.4 DIRECTIONS FOR FUTURE STUDIES

Pain is a protective biological system essential for survival[20]. Individuals who are unable to perceive pain are prone to injury due to the absence of self-preserving responses to everyday noxious events[21]. However, when pain persists beyond healing time, it becomes an unpleasant and emotionally arousing sensory experience requiring medical and psychological attention.

TMD pain is an etiological factor for psychosocial impairment and decreased quality of life in a significant segment of the clinical population[22]. It has a prevalence of about 8% in males and 15% in females of all ages in North American population[22]. Huang et al[23] examined 469 Caucasian individuals according to RDC and found that 20.7% of the subjects had myofascial pain, 4.3% had arthralgia, and 33.5% had myofascial pain with arthralgia. 31% of the subjects in the three pain groups had diagnoses of disc displacement. Our data showed that masticatory muscle pain and TM joint pain are the most common

TMD symptoms. The prevalence pattern reported in previous studies and observed in present study suggests that investigations should be directed at managing pain associated with TM joint dysfunction. Currently, our ability to satisfactorily manage persistent TMD pain is inadequate[24].

Identifying specific genetic elements of pain perception is emerging as key for creating new and individualized pharmacotherapy for chronic pain. For example, recognition of the role of voltage-gated sodium channel in pain perception[25-27] stimulated the search for analgesics that selectively target this sodium channel subunit[25]. In 1976, Marbach and Levitt[28] reported that patients with facial pain conditions had increased urinary levels of catecholamine metabolites and diminished erythrocytic catecholamine-O-methyltransferase (COMT) activity. Recent study[29] identified three major COMT variants (haplotypes) associated with myogenous pain in subjects with TMD: low pain sensitivity (LPS), average pain sensitivity (APS) and high pain sensitivity (HPS). The LPS haplotype was associated with low pain sensitivity; APS was associated with higher pain sensitivity and HPS with the highest pain sensitivity. Collectively, these three haplotypes account for 11% of the variability in pain perception. Further research should focus on the application of these findings into clinical practice. Further in the future, gene therapy might be employed for chronic pain refractory to pharmacotherapy. The challenge will be to understand the

interactions between genes, hormones, cytokines, and between genes and environmental risk factors for TMDs[30].

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