The Findings and Natural History of Radiographic Signs of Temporomandibular Osseous Changes in an Orthodontic Population

A THESIS
SUBMITTED TO THE FACULTY
OF UNIVERSITY OF MINNESOTA
BY

Brian Craig Anderson

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
MASTER OF SCIENCE

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August 2013

Acknowledgments

I feel very appreciative to have had the chance to study orthodontics and to complete a Master of Science degree at the University of Minnesota. Many people have made meaningful contributions to help make this all possible; a special 'thanks' to:

- My wife Mindy, and my daughters Chloe, Brielle, and Kylie, for your love and support and for making me truly happy.
- My family through blood and law, for your examples and for your support.
- Good teachers, friends, and neighbors throughout my life, for your influences.
- Dr. Nanci De Felippe, for your mentorship, encouragement, time, and thoughtful feedback.
- Dr. Mansur Ahmad, for your expertise, training, time, and support.
- Dr. Brent Larson, for believing in me and for all you've taught me.
- Dr. Philippe Gaillard and Andrew Wey, for your statistical analyses.
- Dr. Vladimir Leon, for your time, ideas and collaboration.
- Dr. Jeremy Goodson, for being a true friend and for your technical expertise.
- Farah Farnaz and Enoch Ng, for your time and help collecting data.
- My co-residents, for your friendship, support, and the good times we've shared.

Dedication

I dedicate this thesis to my wife, Mindy, who has been an amazing friend and companion as we've journeyed through our married life together. I love you infinitely; thank you for being you—you are truly wonderful.

To my parents, who instilled in me a passion for learning and a strong desire to seek truth and goodness. You are amazing people and great examples.

To the rest of my family, who supported me, Mindy, and our young family in many ways. We could not have done this without you.

To my children, who inspire me to be a better person, who make me laugh, and make me smile—I love you.

Abstract

AIMS: Evaluate the natural history of radiographic temporomandibular joint (TMJ) findings in an orthodontic sample population by interpreting cone-beam CT (CBCT) scans, made before and after comprehensive orthodontic treatment, according to Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) imaging criteria.

METHODS: 348 subjects were included in the study. Pre-treatment CBCTs were interpreted and screening diagnoses were given. Subjects were categorized: control (normal), indeterminate (remodeling), and case (degenerative joint disease [DJD]); these were matched for age and gender. The pre-treatment and post-treatment CBCTs of 76 matched subjects (152 joints) were interpreted and definitive diagnoses were given for each joint.

RESULTS AND CONCLUSIONS: There was no statistical evidence that, on average, a worse joint diagnosis existed pre-treatment or post-treatment. The likelihood of having a worse diagnosis post-treatment was statistically significant given a pre-treatment diagnosis of DJD or indeterminate. From pre-treatment to post-treatment 52.6% of diagnoses were unchanged, 25% worsened, and 22.4% improved.

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INTRODUCTION

Symptoms of temporomandibular disorders (TMD) are most prevalent among individuals between 15 to 25 years of age.[1] Since orthodontists often provide treatment for people in this age group, some of their patients may report symptoms of TMD before orthodontic treatment or may experience symptoms during or after treatment. TMDs are often associated with degenerative osseous changes in the temporomandibular joints (TMJs)[2] that can create crippling morphological and functional deformities.[3] In fact, osteoarthritis (OA) is the most common pathologic condition affecting the TMJ.[3,4] Therefore, it is important that orthodontists understand TMD and OA and their implications on clinical practice.

Several previous studies have used radiography to evaluate osseous changes in the TMJs in orthodontically treated patients,[5-7] including before and after orthodontic treatment.[8-11] However, after a search of the published literature, no studies were found that evaluated the TMJs before and after orthodontic treatment either with cone-beam computed tomography (CBCT) or the validated imaging criteria belonging to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).

The aims of this study were to (1) use CBCT and the RDC/TMD imaging criteria to assess the osseous components of the TMJs of a pre-orthodontic population and to evaluate for remodeling and degenerative changes, and (2) evaluate the natural history of radiographic TMJ findings and diagnoses in an orthodontic sample population by interpreting CBCT scans, made before and after comprehensive orthodontic treatment, according to the RDC/TMD imaging criteria.

LITERATURE REVIEW

TEMPOROMANDIBULAR DISORDER

TMD is a term that encompasses a group of complex conditions with varied, and often, multifactorial etiologies. TMD is primarily non-inflammatory in origin.[3] The pathologic process includes wear and deterioration of articular cartilage and local thickening and remodeling of underlying bone, often with superimposition of secondary inflammatory changes.[3]

TMD can affect muscles, bone, or the soft tissue components (including cartilage, the articular disc, and ligaments) of the TMJ.[3,12] TMD is the most common cause of non-dental pain in the orofacial region,[2,12,13] and is often localized to muscles of mastication or to the TMJ and/or preauricular area.[12] The most common clinical signs and symptoms include fairly localized pain of the joint or masticatory muscles, limitations in mouth-opening capacity or asymmetric mandibular movements, and joint noises (clicking or crepitation).[2,14] Signs and symptoms may appear and disappear within a single individual.[15] In 1996, the National Institute of Health (NIH) concluded that most TMD symptoms are self-limiting, can recur, and may fluctuate over time.[1]

TMD CLASSIFICATION

Historically, TMD has been difficult to define and measure, and there has been confusion concerning the definition of TMD; when considering whether an individual has TMD, the diagnosis may differ depending on which classification scheme is used.[15]

Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) was proposed in 1992 as a new system for classifying TMD. The objective of this new

diagnostic system was to maximize the reliability and minimize the variability of the examination methods and clinical judgment.[16] RDC/TMD has two axes: Axis I, which is concerned with clinical aspects of TMD and aims to differentiate (1) muscle disorders, (2) disc displacements, and (3) arthralgia, arthritis, and arthrosis; and Axis II, which considers pain disability and psychological condition of the patient.[16,17]

TMD EPIDEMIOLOGY

The literature reports a great range in the prevalence of clinical signs (0-93%) and symptoms (6-93%) of TMD.[16] Luther[14] notes that the reported prevalence ranges from 7-84% with an age range of 3-74 years. Multiple authors have cited prevalence studies showing that up to 75% of the population have at least one sign of TMD (abnormal jaw movement, joint noises, tenderness on palpation, etc.) and 33% have at least one symptom (facial pain, joint pain, etc.).[18-20] Tanaka et al[3] refer to a summary of prevalence studies by Carlsson and LeResche showing a range of 33-86% for signs and 16-59% for symptoms. Roda et al[16] suggested that the considerable variation is probably not a result of strikingly different study populations, but is more likely due to differing clinical criteria to define TMD. Luther[14] concluded that the magnitude of the variation in reported prevalence and affected ages might result from differences in methods of assessing TMD and procedures for recruiting and selecting study participants. Regardless of the true prevalence, about 3-7% of the population seeks treatment for TMD.[16]

TMD ETIOLOGY AND RISK FACTORS

Much of the etiology and pathophysiology of TMD is not well understood and remains controversial.[1,3] Proposed etiologic factors include inflammatory processes, effects of age on the body's tissue composition, bruxim, and gender or genetic factors (including changes in hormone levels, pain receptors and pain perception, and central factors).[18] Psychological factors, such as stress, anxiety, and depression are considered significant risk factors.[16] Several occlusal factors, such as discrepancies between centric relation (CR) and maximum intercuspation (MI), and unilateral crossbite, have also demonstrated a predictive potential for development of TMD.[16]

Age

Elderly patients have greater clinical and radiographic signs of TMD, although they report fewer symptoms and seek treatment less often than younger adults.[16] According to Luther,[18] TMD is rare in children, increases in late adolescence, and seems to peak between 35-45 years of age. Nilsson[21] found the overall annual incidence of TMD pain among Swedish adolescents was 2.9% (4.5% for girls and 1.3% for boys, a statistically significant difference), and TMD pain increased with age (especially for girls). However, the estimated prevalence of TMD in children and adolescents, like the general population, varies significantly from 6-68%[16] (depending on the different diagnostic criteria and examination methods used), and has even been reported to occur in as high as 30-70% of adolescents.[22]

Gender

There tends to be no significant difference in TMD signs between the genders,[15] but symptoms are significantly more common among women.[15,20] The female to male

ratio is at least 2:1 for TMD symptoms,[18] and may be as high as four times[14] to nine times[23] more frequent in females than males. Females tend to seek treatment for TMD three times more often than males,[16] and it has been reported that most individuals seeking treatment for TMD are women between 20 and 40 years of age.[24]

The difference in symptoms between the genders may be associated with genetic variations of pain perception.[20] Circulating hormones have been linked to TMD pain, which has been shown to increase about 30% in postmenopausal females receiving estrogen hormone replacement therapy and approximately 20% among women using oral contraceptives.[16] Low estrogen levels or rapidly changing estrogen levels have also been associated with higher levels of pain.[20]

Genetic factors

Although genetics have been cited as a potential risk factor for TMD, results from studies of monozygous and dizygous twins suggest that genetic factors and the family environment do not significantly affect TMD signs or symptoms.[16]

Parafunction/bruxism

Parafunctional habits and bruxism reportedly carry odds ratios of up to 4.8 as risk factors for TMD,[16] and parafunction has been associated with TMD signs and symptoms in adolescents.[25] However, pain does not always follow bruxism—Luther's[18] literature review noted that some studies found no pain in nightly bruxers; therefore, bruxism is not universally associated with TMD.

Despite being cited as etiologic factors for TMD, bruxism and clenching—especially bruxism—appear to be endemic in the general population.[26] The prevalence of bruxism in adults and children is approximately 20%, although parent interviews suggest

the prevalence might be as high as 38% in children (however, only 5% of these parents reported subjective TMD symptoms in their children).[16] Furthermore, some studies suggest that the prevalence of bruxism is highest in children and declines into adulthood, which is at odds with the known prevalence of TMD.[18]

Psychological factors

Stress, anxiety, and depression have all been suggested as risk factors for TMD symptoms, as depression has been associated with TMD myofascial pain.[16] TMD has a varying degree of psychogenic influence (i.e., no physical cause can be identified).[20] Psychosocial factors may play a major role in the development of TMD, influencing how the central nervous system processes pain and affecting how pain is interpreted.[20]

Occlusion

It has been estimated that occlusal factors may contribute about 10-20% to the multifactorial characterization of TMD.[26] Five occlusal factors have been associated with specific diagnostic groups of TMD conditions: (1) skeletal anterior open bite, (2) overjets of greater than six to seven millimeters, (3) slides greater than four millimeters from retruded cuspal position to intercuspal position, (4) unilateral lingual crossbite, and (5) five or more missing posterior teeth.[26] Also, de Santis et al[27] have recently shown that children with TMD have significantly fewer occlusal contact points than children without TMD.

However, it is unclear if malocclusion is a true etiologic factor for TMD.[19,20] Correlation is not causation; moreover, the evidence linking TMD with occlusal factors and malocclusion is relatively weak,[18,28] and several studies have suggested that some

malocclusions may be a consequence, and not a cause, of TMD,[16,28] or that the growth patterns that lead to the malocclusions may play a role in the development of TMD.[28]

Although some studies have reported relationships between various malocclusions and TMD signs and symptoms, these studies are difficult to perform and study designs have generally been flawed, subjecting the results to substantial criticism.[14] TMD experts agree that occlusion has a relatively small role in the development of a complex disorder that is etiologically diverse and multifactorial in origin.[26] Neither functional nor static occlusal factors can be called etiologic factors for TMD due to a lack of evidence supporting such claims.[18] Luther et al[20] concluded that no evidence from trials has shown that orthodontic treatment can prevent or relieve TMD, which supports the belief that teeth are not part of its cause (they suspect that the real cause of TMD remains unknown).

Evidence from systematic reviews seems to indicate that occlusal equilibration is not helpful for treating or preventing TMD.[29] Studies that show occlusal equilibration to have a TMJ benefit have been excluded from systematic reviews due to poor study design.[29] However, randomized controlled trials that were included in a systematic review that did not show a benefit were judged to be of low to fair quality; therefore, the conclusion has generally been that there is insufficient evidence to claim that occlusal adjustment is effective in treating or preventing TMD.[19,20,29] According to a Cochrane Systematic Review on the subject, occlusal adjustment cannot be recommended for TMD treatment or prevention.[19]

According to a systematic review by Forssell et al,[13] splint therapy may be of some benefit in treating TMD because randomized controlled trials have sometimes shown superior outcomes, but generally comparable outcomes, of splints to control

treatments. In general, studies on occlusal splints have yielded indeterminate results, and the clinical effectiveness of splints in relieving pain seems modest to other pain-management modalities.[30] However, due to generally low-quality studies on splint therapy, no firm conclusions have been reached.[13]

TREATMENT FOR TMD

Treatment options for TMD include reassurance (patient education, behavior therapy, and self-care instructions) at one end of the spectrum to surgery at the other end.[20] Physiotherapy (e.g., acupuncture, ultrasound, heat exercises, biofeedback, etc.), splint therapy, pharmaceutical therapy, occlusal adjustment, and combined treatments are additional treatment modalities.[20]

The need for treatment has been debated. Luther et al[20] report that LeResche suggested only 10% of the population over 18 years of age are likely to have symptoms that require treatment, and that other experts estimated 3.6% to 7% of the population need treatment.

TMD AND ORTHODONTIC TREATMENT

In response to growing interest in the potential relationship between TMD and orthodontic treatment since the late 1980s, many studies have been conducted.[1] In 1992, the entire January issue of the *American Journal of Orthodontics and Dentofacial Orthopedics* was devoted to the subject.[14,26]

Gnathologists assert that failure to treat orthodontic patients to gnathologic standards may lead to TMD.[26] This rationale is based on the hypothesis that a

malocclusion can restrict TMJ function and predispose the joint to pathological deterioration; therefore, correcting the malocclusion will allow the TMJ to remodel to the overriding new functional needs, thereby treating any disease process or malfunction of joint integrity.[20]

Based on the body of available evidence, however, it is generally accepted that orthodontic treatment is neither a cause nor a cure of TMD.[14-16] The proposed causal link between orthodontic treatment and TMD has been addressed considerably in the scientific literature, and the vast majority of studies conclude that orthodontic treatment does not make TMD better or worse.[16] Most studies have found little or no relationship between orthodontic treatment and TMD.[1]

A meta-analysis of published longitudinal data did not indicate that traditional orthodontic treatment increased the prevalence of TMD,[14] and overwhelming evidence supports the conclusion that people who had orthodontic treatment as adolescents are not at greater risk for developing TMD than those who did not have treatment.[31] Sadowsky[31] pointed out that orthodontic treatment produces relatively slow changes in an environment that typically has considerable adaptive capacity. However, McNamara[26] noted that some occlusal variables may increase stress on the TMJ system, and while most individuals adapt without problems, adaptation in others may increase the risk of dysfunction.

REMODELING OF THE TMJ

Joint remodeling is a reversible biologic adaptation to altered environmental circumstances.[32] TMJ remodeling is an essential physiologic response to normal

functional demands; the joint morphology changes in response to applied mechanical forces, thereby ensuring homeostasis of joint form, function, and occlusal relationships.[3,12]

Signs of TMJ remodeling can be seen in load-bearing areas of the condyle or articular eminence, and include flattening of curved joint surfaces and subcortical sclerosis (thickening of articular cortical surfaces).[12,17] Signs of remodeling are often detected in asymptomatic subjects, and remodeling is not considered abnormal unless seen in conjunction with signs and symptoms of pain or dysfunction, or if the extent of radiographic remodeling is deemed severe.[12]

In an autopsy study of young adults, Hansson et al[24] found that deviations in form, which include flattening (indicative of remodeling), are common in young people. Solberg et al,[33] who scored even minor changes to be deviations in form, reported similar findings—the observed frequency of deviations in form was 85% among young adults being autopsied. Solberg et al[33] concluded that " . . . the most apt characterization is that the TMJ changes in this age group [26.4 +/- 6.8 years] are adaptive phenomena occurring in order to cope with the details of articular fit and function."[33] (1985, p. 303)

In an epidemiologic study using cephalometrically corrected hypocycloidal tomography, Brooks et al[34] found signs of minimal flattening in 35% of TMJs of asymptomatic patients with no internal derangement (internal derangement refers to an aberrant position of the articular disc relative to the mandibular condyle and articular eminence). Barghan et al[12] cited the sensitivity of tomography in the range of 53% to 90%; using this data, one can conclude that the 35% detected in the study by Brooks et al[34] likely underestimated the true value, and perhaps only around half of the true

flattening was detected. Brooks et al[34] concluded that minimal flattening is probably of no clinical significance because arthrography and MRI showed no soft tissue abnormalities in the subjects that were studied, and they suggested that minimal flattening in asymptomatic patients without internal derangement should be regarded as being within normal limits. Brooks et al[34] contrasted their findings with those of Muir and Goss (who reportedly found osseous TMJ changes in more than 90% of their asymptomatic subjects), and explained that differences in findings may result from different patient selection, different radiographic techniques, and different radiographic interpretation.[34]

Skeletal and histologic studies by Muir and Goss[35] demonstrated that remodeling occurs in all joints throughout adult life. As they discussed, remodeling appears to increase with age; and, although most studies indicate that the correlation between remodeling and age is not direct, there has been disagreement. Using panoramic radiography to study adult females, Muir and Goss[35] found condylar flattening, condylar sclerosis, and condylar osteophytes in 70%, 73%, and 21% of asymptomatic, dentate subjects, respectively. Flattening and sclerosis were the most common findings; all findings were found more commonly on the condyle than the articular eminence (consistent with the findings of dos Anjos Pontual[2] and Campos et al[36]). Muir and Goss[35] concluded that it is a common misconception that asymptomatic TMJs are radiographically normal.[35]

However, distinguishing remodeling from osteoarthritic changes can be difficult.[32] In a study of TMD patients with signs of osseous change, dos Anjos Pontual et al[2] reported 59% showed flattening and no other sign, and flattening alone was the most

prevalent of any sign of osseous change. They surmised that the high prevalence of flattening might be explained in part because this bony change represents an adaptive response.[2] Based on a TMJ study using MRI, Campos et al[36] found no significant differences in condylar flattening between TMJs with and without degenerative bony changes, concluding that flattening may or may not be the result of condylar destruction. However, they suggested that flattening might be the first change of a progressive disease.[36]

DEGENERATIVE CHANGES OF THE TMJ

TMDs are often associated with degenerative osseous changes in the TMJ[2] that can create crippling morphological and functional deformities.[3] Low, or secondary, inflammatory conditions with degenerative osseous TMJ changes include osteoarthritis and osteoarthrosis. These are technically distinct entities with pain as a distinguishing feature (present in osteoarthritis but absent in osteoarthrosis).[17] However, because it is difficult to draw distinctions in etiology, pathology, and management, the terms are often used synonymously.[3] Although the term osteoarthritis has been widely adopted, Ahmad et al[17] suggested that, technically, degenerative joint disease (DJD) might be the best term when the diagnosis is made radiographically and no clinical information is available. In this paper, the terms OA and DJD will be used synonymously.

TMJ OSTEOARTHRITIS EPIDEMIOLOGY

Osteoarthritis (OA) is the most common pathologic condition affecting the TMJ.[3,4] It has been reported that 11% of individuals with TMDs have symptoms of OA.[3]

The prevalence has been reported to be 22-38% of the population within the age range of 20 to 90 years.[37] However, as with TMD, the reported prevalence varies greatly. Epidemiological studies of morphological TMJ alterations, identified from radiographic analysis, have been unable to identify a standardized pattern in the distribution of TMJ osteoarthritis (TMJ-OA).[16] Radiographic signs of TMJ-OA have been found in 14-44% of individuals, far exceeding the 1-24% of patients who demonstrate certain clinical signs, such as crepitus with palpation or auscultation.[16]

OA of the TMJ is often called an age-related disease due to increased frequency of degenerative changes with age.[2,4,23,38] Up to 80% of people between 60 and 80 years of age may be affected.[37] Tanaka et al[3] report that both the frequency and severity of degenerative joint changes increase with age; for example, the articular disc tends to calcify with age, becoming more stiff and fragile, thereby reducing its ability to appropriately withstand loading forces; and, hyaluronic acid (which is essential for maintaining the proper viscosity of articular cartilage) decreases dramatically with aging.[3] However, despite an increase in morphologic TMJ changes with age, symptoms and other signs appear to decrease with age.[23]

Autopsy studies have shown that TMJ-OA is common in elderly people,[32,39,40] and uncommon in younger people.[24,33] Pereira[41] found degenerative TMJ changes in 28% of patients aged 16-39 years and in 50% of patients aged 55-78 years. Wiberg and Wanman[37] cited one autopsy study that found degenerative changes in 22% of the TMJs of older adults, but only in 4% of the TMJs of young adults.

However, Zhao et al[42] concluded that although TMJ-OA is age-related, age is not the critical determinant in the pathologic development of OA. They found that

radiographic signs of osteoarthritis of the TMJs were common in adolescent and young adult patients with TMD, with a prevalence that increased sharply from ages 11-19 and a maximum frequency in the age range of 15-19 years.[42] As Wiberg and Wanman[37] recounted, Dibbetts found that 16% of children referred for orthodontic treatment showed signs of deformative changes of the condyle classified as arthrosis deformans juvenalis (ADJ) using the Parma radiographic method, and 24% had signs of ADJ after a two year follow-up. Cho and Jung[43] also found that radiographic signs of TMJ-OA were relatively common in children and adolescents. Pliska et al[44] found, in a group of pre-orthodontic patients with an average age of 13.0 years, 18.0% had significant TMJ findings that required immediate follow-up (osteophyte, subcortical cyst, degenerative changes, and anteriorly displaced disc).

Many studies have found OA to be more common in females than males.[2,4,12,22,42] Akerman et al[40] found a difference between the genders in degenerative TMJ changes and deviations in form; in their autopsy study of elderly subjects (60-89 years old), they found the most advanced changes in the joints of women.

Petrikowski and Grace[22] found, from analysis of corrected sagittal tomographs, that adolescent girls had significantly higher frequencies of certain TMJ radiographic abnormalities than adolescent boys. The difference between the genders was even more significant in older adolescents.[22]

However, the findings of some studies do not support that OA is greater in females than males. Cho and Jung[43] found no significant radiographic difference between asymptomatic child and adolescent males and females, and actually found that osteoarthritic changes of the TMJs were significantly more common among symptomatic

males than females. Autopsy studies by Widmalm et al[23] demonstrated no significant difference between elderly men and women with regard to morphologic changes to the TMJs, and they concluded that gender is not a major factor for the development TMJ pathosis in elderly individuals.

TMJ OSTEOARTHRITIS ETIOLOGY

Unlike high inflammatory conditions (such as rheumatoid arthritis, lupus erythematosus, etc.), OA is a secondary inflammatory condition.[12] Following degeneration of joint cartilage, degraded proteoglycans and proteolytic enzymes are released into synovial fluid, triggering a secondary inflammatory response and further degradation.[12] Loading beyond the adaptive capacity of the joint, parafunction, and loss of molar support may be etiologic factors of TMJ-OA; however, the cause or causes are not fully understood.[37] The etiologic factors given by Barghan et al[12] include biomechanical overloading and microtrauma (resulting from parafunction, such as bruxism and unilateral chewing), attrition and loss of molar support, and internal derangement of the joint. According to Tanaka et al,[3] it is believed that degenerative changes in the joint result from either dysfunctional remodeling (secondary to a host's decreased adaptive capacity), or from excessive or sustained physical stresses (which exceed the normal adaptive capacity of the joint), or from a combination of both. Factors affecting host-adaptive capacities include age, systemic illness, and hormonal factors; factors contributing to joint stresses include macrotrauma, parafunction, and functional overloading and increased joint friction.[3]

Milam et al[45] proposed the Oxidative Stress Model for the development of degenerative joint disease. Both mechanical injury and excessive intraarticular pressure (which occurs with unfavorable joint loading) can cause oxidative stress via the formation of free radicals (highly reactive molecules that can cause significant tissue damage).[3,45] Parafunction, clenching, and bruxing are behaviors that produce mechanical stresses and can result in functional overloading.[3,45] With unfavorable loading, intraarticular pressures exceed the end-capillary perfusion pressure of TMJ tissues, resulting in a brief period of hypoxia, thereby altering local cellular metabolism and leading to the production of free radicals when perfusion is reestablished.[45]

Internal derangement appears to be associated with OA—in an autopsy study by de Bont et al,[32] degenerative changes of the condyle and articular eminence were highly correlated with disc displacement (either partial or complete). They concluded that TMJ internal derangement is likely an accompanying sign of osteoarthritis.[32] Tanaka et al[3] reported that internal derangement might be a predisposing factor to joint degeneration, or vice versa.

TMJ IMAGING

Imaging, in conjunction with clinical examination, plays a critical role in the diagnostic process of TMD, and radiographic findings have been shown to influence prescribed treatment modalities for patients with TMD.[12] Several imaging modalities have been used to evaluate the TMJs for clinical and research purposes, including panoramic radiography, conventional linear or complex motion tomography, and

computed tomography (CT) for evaluation of osseous TMJ tissues, and magnetic resonance imaging (MRI) for evaluation of the soft-tissues of the joints.[12]

Panoramic radiography

Panoramic radiography can be used to screen for gross TMJ osseous pathology,[12] but inherent limitations (including distortions, two-dimensional superimpositions, a limited view of the joint,[12] nonoptimal imaging geometry,[46] etc.) minimize its value for TMJ assessment[12] and make it unsuitable for detailed visualization of TMJ structures.[46] Panoramic radiography has poor reliability and low sensitivity for detecting osseous TMJ changes.[12,17]

Tomography

For several years, linear or complex motion tomography was the radiographic modality of choice for examination of osseous components of the TMJs because it overcame inherent distortion and superimposition problems with panoramic radiographs.[12] Ludlow et al[46] demonstrated that significantly more accurate detection of simulated condylar lesions was obtained using biplanar tomography than with biplanar panoramic radiographs. The reported sensitivity and specificity of tomography for detecting TMJ bony changes range from 53% to 90% and from 73% to 95%, respectively.[12] However, Flygare et al[47] demonstrated in an autopsy study that tomography underestimates both the existence and the size of small bony lesions of the TMJ, thereby limiting its diagnostic accuracy.[12,47]

Computed tomography

Computed tomographic units can be divided into two types according to the acquisition x-ray geometry—fan beam and cone beam.[48] Fan-beam CT is also known as

medical CT, helical CT, or multidetector CT (MDCT). A collimated, fan-shaped x-ray beam characterizes fan-beam scanners.[49] The x-ray source and detector are mounted on a rotating gantry and data are acquired via a thin, broad beam that is transmitted through an object.[48] Individual image slices of the object are stacked to produce its three-dimensional representation, with each slice requiring a separate scan and separate two-dimensional reconstruction.[48] Cone-beam CT (CBCT) imaging, sometimes called volumetric CT,[50-52] uses a three-dimensional, cone-shaped x-ray beam with an extended two-dimensional detector.[48] The tube detector system rotates 360 degrees around the patient's head with a constant beam angle.[49] Some units use an image intensifier tube/charge coupled device (IIT/CCD), while others use a flat-panel imager (e.g., i-CAT scanners); systems that use IIT/CCD generally produce images with more noise and measurement inaccuracies than images from flat-panel devices[48] During the scan, many 2D snapshot images are acquired (the initial rotation produces the raw data), and specialized reconstruction algorithms can then be used to compile the images into a 3D dataset.[52] The time required for CBCT data acquisition is considerably less than that for MDCT because a single rotation of the gantry can generate a scan of the entire region of interest.[48] Several options for layer thickness in the reconstruction include 0.3 mm, 1.0 mm, and 3.0 mm.[50] CBCT software can manipulate the data to allow visualization of anatomy and to accurately show relationships within the craniofacial complex.[52]

CT provides critical information for diagnosing OA.[12,50,53] According to Honey et al[48] and Zain-Alabdeen and Alsadahn,[49] MDCT has a reported accuracy of 87.5% to 96% in detecting TMJ degenerative arthritic changes,[48,49] and an autopsy study by Westesson et al[54] showed MDCT to have a sensitivity of 75%, a specificity of 100%, and a

diagnostic accuracy of 87% in detecting TMJ osseous changes. MDCT is superior to MRI and panoramic radiography in diagnosing TMA-OA,[17] and is better than MRI and conventional tomography for assessing osseous components of TMJ.[53] Indeed, MDCT has historically been the gold standard for the diagnosis of TMJ pathosis and RDC/TMD guidelines recommend CT as the imaging modality of choice for detection of TMJ osseous changes.[12] However the relative high cost, inaccessibility, and high radiation dose of MDCT have limited its use in dentistry.[52]

Honey et al[48] reported the diagnostic accuracy of CBCT is significantly better than corrected angle linear tomography, normal panoramic radiography, or TMJ-specific panoramic radiography in detecting simulated condylar lesions. Ahmad et al[17] cited a systematic review indicating that CBCT might be superior to MDCT, and Honda et al[53] showed similar sensitivity, specificity, and diagnostic accuracy for CBCT and MDCT in diagnosing TMJ abnormalities in a radiographic study using macroscopically evaluated autopsy specimens. Zain-Alabdeen and Alsadhan[49] compared CBCT and MDCT and found no difference in the accuracy of detecting surface osseous changes of the TMJ, and they reported comparable intraoberver reliability between the two modalities. They also cited two studies that showed CBCT was superior to MDCT in displaying hard tissue of the maxillofacial region with significantly less radiation to the patient.[49] Advantages of CBCT over MDCT include lower radiation dose,[12,48-50] (the range of radiation exposure from CBCT is within a similar range of other dental radiographic modalities and is generally significantly less than MDCT devices),[52] shorter examination time,[48,50] lower cost,[12] generally superior spatial resolution of the image,[48,49] less space required for equipment, and often easier access to CBCT equipment.[12] Therefore, CBCT may be

considered the imaging technique of choice to evaluate the osseous components of the TMJ.[50]

RESEARCH DIAGNOSTIC CRITERIA FOR TEMPOROMANDIBULAR DISORDERS (RDC/TMD)

Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) is a widely used diagnostic system for TMD.[17] Image analysis criteria for disc displacement (using arthrography and MRI) and OA (based on tomography) were briefly described in the original Axis I protocol of RDC/TMD;[17,55] however, criteria for image interpretation were not specified.[55] RDC/TMD is currently used by dozens of research groups and has been translated into at least 18 languages.[17] Despite the robust scope of the RDC/TMD, its original lack of well-defined diagnostic criteria limited the usefulness of its application in image interpretation; and, as Ahmad et al[17] noted, reliable criteria for image analysis are essential for both research and clinical endeavors.[17]

In 2001, the National Institute of Dental and Craniofacial Research (NIDCR) funded a comprehensive characterization of the reliability and the criterion validity of the RDC/TMD called 'Research Diagnostic Criteria: Reliability and Validity', which has since been referred to in the literature as the RDC/TMD Validation Project.[17,55] Due to increasing use of CT and MRI, comprehensive TMJ diagnostic criteria were developed by Ahmad et al[17] for image analysis using these techniques as a part of RDC/TMD, and criteria for panoramic radiography were also developed (panoramic radiography, which has been recommended as a screening tool for TMJ pathology, was not included as an imaging option in the original RDC/TMD).[17] Panoramic radiography, CT, and MRI were

used for the radiologic diagnosis of OA, and interexaminer reliability was estimated using the kappa (κ) statistic.[17] Ahmad et al[17] and Look et al[55] cited study guidelines for classifying kappa statistics: $\kappa > 0.75$ is considered excellent reliability, κ of 0.4—0.75 is considered fair to good reliability, and $\kappa < 0.4$ is considered poor reliability. The Validation Project found that interexaminer reliability was poor for panoramic radiography ($\kappa = 0.16$), fair for MRI ($\kappa = 0.46$), and near the excellent threshold for CT ($\kappa = 0.71$).[17,55] Positive percent agreement among examiners was 19% for panoramic radiography, 59% for MRI, and 84% for CT.[17] The CT-based diagnosis of OA was used as a reference standard to assess the validity of the other modalities; the sensitivity for OA using panoramic radiography was very low at 0.26, but the specificity was excellent at 0.99; the sensitivity of MRI for OA was 0.59, and the specificity 0.98.[55] CBCT was not used in the Validation Project because when the study was conducted CBCT was not widely available and was not accessible to the participating radiologists.[17] Ahmad et al[17] concluded that the image analysis criteria that were developed for the Validation Project are reliable and should be used in both clinical and research settings.

MORPHOLOGIC TMJ CHANGES AND ORTHODONTIC TREATMENT

Since orthodontic treatment has not been shown to cause or cure TMD,[14-16] it might be assumed that it does not affect the development of TMJ-OA. However, several radiographic studies have been completed to help answer the question: is orthodontic treatment associated with morphologic TMJ changes? Hansen et al[5] used lateral tomography to evaluate the TMJs of 19 consecutive male subjects who were treated with the Herbst appliance, an average of 7.5 years after Herbst treatment was completed, and

concluded that the treatment did not seem to have any long-term adverse effects on the craniomandibular system. Dibbetts and van der Weele[9] reported that the first radiographic appearance of structural bony changes in the mandibular condyles was between 12 and 16 years of age. According to Peltola et al,[8] Dibbetts and van der Weele found a slight increase in the frequency of condylar findings during the first two years of orthodontic treatment, but concluded that the increase was associated with age and not with orthodontic treatment. Peltola et al[6-8,56] did several of their own studies using panoramic radiography to investigate if condylar changes and orthodontic treatment are associated. Prior to orthodontic treatment, they found no significant difference with regard to condylar findings between pre-orthodontic patients and normal population controls, but discovered that condylar findings increased with age in the subjects treated orthodontically but not in population controls.[56] In another study, they found significantly more condylar findings in subjects who had been treated orthodontically than in untreated control subjects.[6] In that study, flattening and subcortical sclerosis were significantly more common in subjects who had experienced orthodontic treatment.[6] In a different study, they evaluated radiographs of subjects before and after orthodontic treatment, and discovered that condylar findings, including flattening, subcortical sclerosis, osteophyte, microcyst, marginal erosion, periarticular ossicle, and other (including deformities in size and form), increased significantly in the orthodontic treatment group (present in 2% of subjects before orthodontic treatment, and 9% of subjects after orthodontic treatment) but not in the untreated population controls.[8] Among subjects with condylar findings, "flattening only" was observed in 50% of the post-orthodontic subjects and in 35% of controls, and the most common combination of findings was flattening and subcortical

sclerosis.[8] Of the 14 orthodontic subjects who initially had findings, 6 had normal condyles after orthodontic treatment, 3 were unchanged, and 5 had findings that had become more severe.[8] In a follow-up to a previous study, they made panoramic radiographs about 12 years after the post-orthodontic radiographic exam and found that, in most subjects, the condylar findings remained constant or became more severe, but disappeared in 28% of subjects.[7]

RATIONALE FOR THE STUDY

In 1995, Peltola et al[8] noted that the number of published radiographic studies on the subject is small. This still seems to be true—in studies published in 2012, dos Anjos Pontual et al[2] reported that there are few studies on the prevalence of TMJ osseous changes (particularly studies using CT), and Cho and Jung[43] noted that radiographic studies examining the TMJs of children and adolescents have rarely been carried out. At the time the current study began, no other studies of which the author is aware, had used CBCT to analyze the TMJs before and after orthodontic treatment.

This study attempts to address a void in the literature by using CBCT to assess TMJ osseous changes in a relatively young population (generally adolescents), and to evaluate how the joints change during orthodontic treatment to answer the questions: Do normal joints remain normal? Do remodeling joints get better or worse? Can degenerative joints get better?

The aims of this study were to (1) use CBCT and the RDC/TMD imaging criteria to assess the osseous components of the TMJs of a pre-orthodontic population and to evaluate for remodeling and degenerative changes, and (2) evaluate the natural history of

radiographic TMJ findings and diagnoses in an orthodontic sample population by interpreting CBCT scans, made before and after comprehensive orthodontic treatment, according to the RDC/TMD imaging criteria.

MATERIALS AND METHODS

IRB INFORMATION

The IRB for the current study was approved on October 27, 2011. The IRB code number is 1110M05804.

STUDY DESIGN AND SAMPLE

All consecutively treated patients who had CBCT scans before and after fixed, comprehensive orthodontic treatment at the Graduate Orthodontic Department of the University of Minnesota School of Dentistry between July 1, 2008, and November 30, 2011 were identified, totaling 381 patients (note: in the orthodontic department, it is standard protocol for patients to have CBCT scans both before and after orthodontic treatment). The CBCT scans were made with a Next Generation i-CAT scanner (Imaging Sciences International, LLC, Hatfield, PA) in maximum intercuspation for orthodontic diagnostic records and following the completion of each patient's comprehensive orthodontic treatment. The machine was set at 120 kVp and 37.10 mA, with resolution set at 0.3 mm voxels enhanced scan; scan acquisition time was 17.8 s. The patients' digital records (photographs and radiographs) were stored and organized by timepoint in Dolphin Imaging (Dolphin Imaging and Management Solutions, Chatsworth, CA). After a brief review of each patient's digital records, 33 were excluded from this study for the following reasons: (1) one of the CBCT files (pre-treatment or post-treatment) was

unavailable for analysis (n = 19); (2) digital records showed phase I orthodontic treatment had occurred prior to fixed, comprehensive treatment (n = 7); (3) the pre-treatment radiographic file was corrupted or incomplete (n = 3); (4) full fixed appliances were present at pre-treatment scan (n = 2); (5) the pre-treatment scan was made more than one year before beginning orthodontic treatment (n = 1); and (6) the pre-treatment scan was not of diagnostic quality for TMJ evaluation (n = 1). The final population consisted of 348 subjects (N = 348).

METHODS

An orthodontic resident (B. A.) was trained by a board-certified oral and maxillofacial radiologist (M. A.) to interpret CT images of TMJs using the validated RDC/TMD image analysis criteria. Following initial training, the resident was calibrated using 58 static, digital CT images of TMJs, from which the resident and oral and maxillofacial radiologist independently made diagnoses of each joint. Interexaminer agreement was 93.1% with a kappa statistic of 0.88 (in the excellent range) with a 95% confidence interval of (0.78, 0.99).

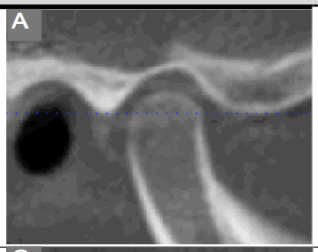
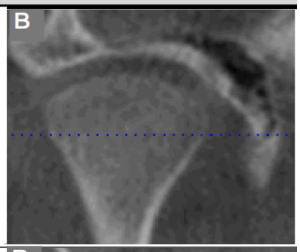
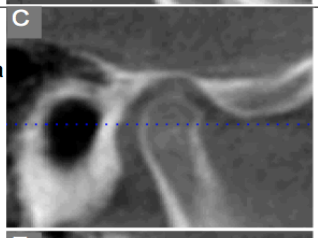
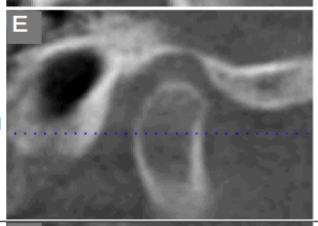
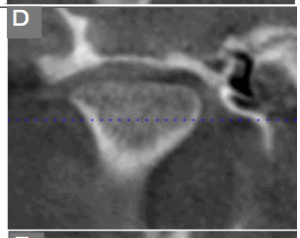
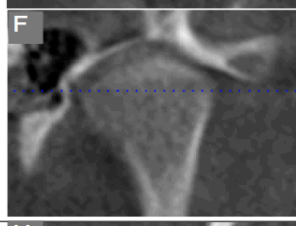
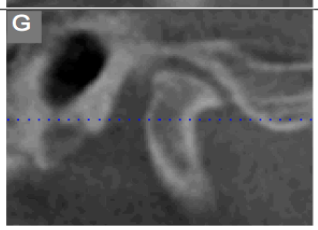
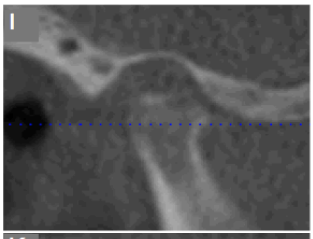
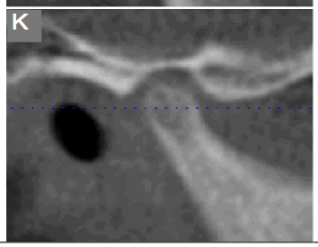
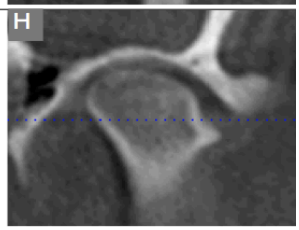
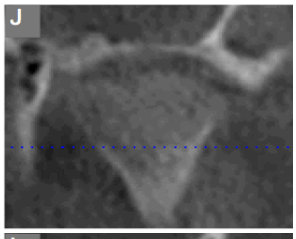
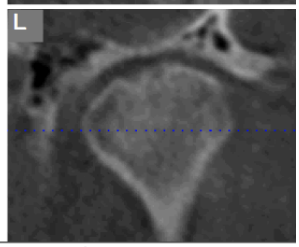
The population was randomized in Microsoft Excel 2008 for Mac, Version 12.3.4 (Microsoft Corp, Redmond, WA), and the orthodontic resident used i-CAT Vision (Imaging Sciences International, LLC, Hatfield, PA) to interpret the pre-treatment CBCT scans of the randomized population, which allowed the examiner to interactively scroll sagittal and axially corrected coronal image slices and to adjust the contrast, sharpness, and magnification. No demographic or clinical information was available to the examiner

during CBCT interpretation, and both the condyle and the temporal component of the TMJ were evaluated.

The radiographic osseous changes that were recorded were (1) articular surface flattening (a loss of the rounded contour of the surface); (2) subcortical sclerosis (any increased thickness of the cortical plate in the load bearing areas relative to the adjacent non-load bearing areas); (3) osteophyte (marginal hypertrophy with sclerotic borders and exophytic angular formation of osseous tissue arising from the surface); (4) surface erosion (loss of continuity of articular cortex); and (5) subcortical cyst (a cavity below the articular surface that deviates from normal marrow pattern).

The diagnostic criteria for TMJ DJD that were used were: (1) normal: no subcortical sclerosis or articular surface flattening, and no deformation due to subcortical cyst, surface erosion, or osteophyte, (2) indeterminate/remodeling (these terms are used synonymously in this paper): localized sclerosis and/or flattening, (3) grade I DJD (aka DJD): osteophyte, or surface erosion, or subcortical cyst, and (4) grade II DJD (aka DJD II): osteophyte, and/or surface erosion, and/or subcortical cyst (i.e., $\geq$ 2 signs of grade I DJD; size of the degenerative changes were not measured in this study)—Table I.

Table I. Diagnostic criteria and image examples from matched subjects in the study. **A, B,** No findings. **C,** Slight flattening of anterior slope. **D,** Flattening of superior margin. **E, F,** Sclerosis of superior margin. **G,** Osteophyte at anterior margin. **H,** Osteophyte at lateral margin. **I,** Surface erosion. **J,** Surface erosion. **K,** Cyst below posterosuperior margin. **L,** Cyst below superior margin (note: in this left joint, surface erosion present medially to cyst).

Diagnosis	Finding	Example: Sagittal	Example: Coronal
Normal	No subcortical sclerosis or articular surface flattening, and no deformation due to subcortical cyst, surface erosion, or osteophyte		
Remodeling	**Articular surface flattening**—a loss of the rounded contour of the surface *and/or* **Subcortical sclerosis**—any increased thickness of the cortical plate in the load bearing areas relative to the adjacent non-load bearing areas	 	 
DJD (grade I and grade II)	**Osteophyte**—marginal hypertrophy with sclerotic borders and exophytic angular formation of osseous tissue arising from the surface *or (grade I)/and/or (grade II)* **Surface erosion**—loss of continuity of articular cortex *or (grade I)/and/or (grade II)* **Subcortical cyst**—a cavity below the articular surface that deviates from normal marrow pattern	  	  

Principles used in interpretation were as follows: (1) the most advanced finding must be present in at least two views to arrive at the diagnosis; and (2) if surface erosion and subcortical cyst are continuous, then the diagnosis is surface erosion.

Screening diagnoses of pre-treatment scans were given for each subject, but not for both joints (i.e., the worst of the two joints determined the diagnosis the subject received). Subjects with unclear diagnoses (i.e., with questionable radiographic findings) were given the diagnosis that the examiner judged was most representative; in addition, these diagnoses were differentiated with a "question mark" following the diagnosis to distinguish a level of uncertainty.

Following a screening diagnosis for each subject's pre-treatment CBCT scan, digital images of the pre-treatment TMJs in the sagittal and axially corrected coronal views were captured with PrintKey 2000, Version 5.05 FreeWare (Nuerensdorf, Switzerland); these images did not contain any subject identifiers.

The percentage of each diagnosis was calculated, after which subjects were assigned into groups based on diagnosis—0: Exclude (questionable diagnoses), 1: Control (normal diagnoses), 2: Indeterminate (remodeling diagnoses), and 3: Case (grade I and grade II DJD)—Fig. 1. After excluding subjects with questionable diagnoses, 277 subjects remained (N = 277; 171 females and 106 males)—Fig. 2. This population was randomized in Microsoft Excel 2008 for Mac, Version 12.3.4 (Microsoft Corp, Redmond, WA), and the subjects' digital radiographic files were copied and stored electronically following the order of the randomized list. Finally, 26 subjects from each group were matched for age (+/- 2 years) and gender (N = 78), with a total of 57 females and 21 males.

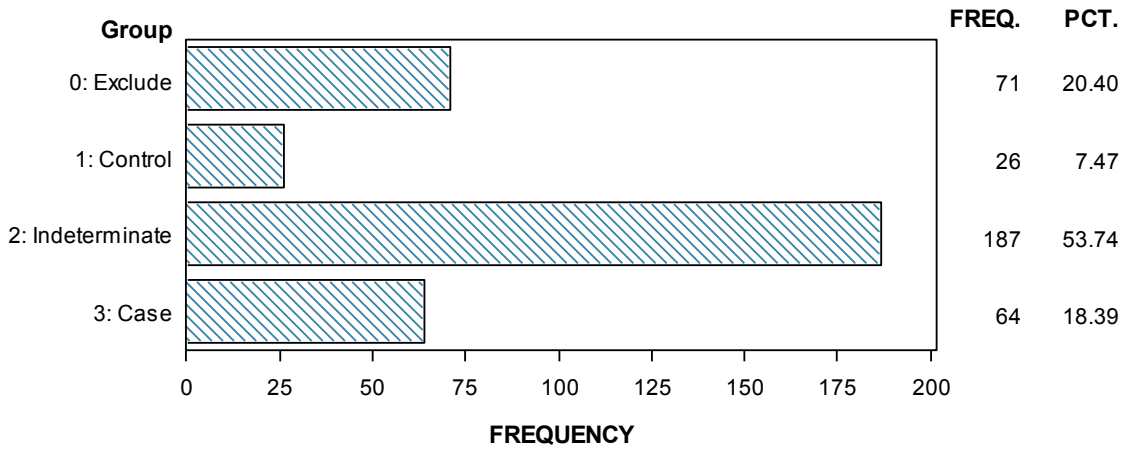


Fig. 1. Histogram of groups in the pre-orthodontic screening population (N = 348). **0: Exclude** = questionable; **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

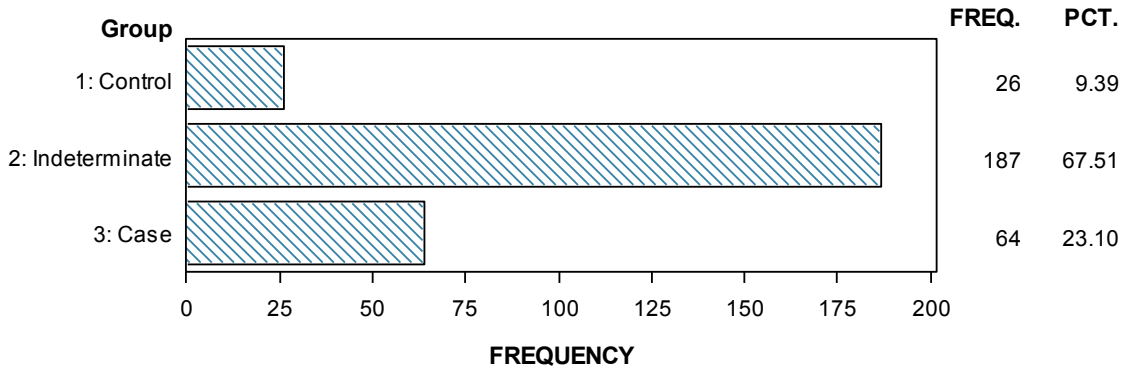


Fig. 2. Histogram of groups in the pre-orthodontic screening population (after exclusion of questionable diagnoses; N = 277). **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Approximately one month after completion of screening diagnoses of pre-treatment CBCT scans, the orthodontic resident used the previously captured, static pre-treatment CBCT images to make definitive diagnoses for the right and left joints of each matched subject (N = 78 x 2 = 156). An independent party randomized the matched subset in Microsoft Excel 2008 for Mac, Version 12.3.4 (Microsoft Corp, Redmond, WA), and assigned numbers to the subjects in ascending order (1-78), and then renamed the subjects' corresponding pre-treatment digital image files accordingly.

Approximately 10 days after making definitive diagnoses for all matched subjects, the orthodontic resident blindly rediagnosed the right and left joints of the first one-third of the previously randomized matched subjects (subjects 1-26; N = 52 joints) using the previously captured, static pre-treatment CBCT images. The intraexaminer reliability is presented in Table II for three different analyses, which consider different diagnostic group combinations.

Table II. Analyses of intraexaminer reliability. **Analysis 1,** Fair to good reliability. **Analysis 2,** At threshold of excellent reliability. **Analysis 3,** Excellent reliability.

Analysis	Diagnostic Groups	Kappa	95% Confidence Interval
1. Normal vs. Remodeling vs. DJDs	1. Normal 2. Remodeling 3. DJD + DJD II	0.618	(0.415, 0.798)
2. Non-DJDs vs. DJDs	1. Normal + Remodeling 2. DJD + DJD II	0.7425	(0.4767, 0.9221)
3. Non-DJDs vs. grade I DJD vs. grade II DJD	1. Normal + Remodeling 2. DJD 3. DJD II	0.7652	(0.5219, 0.9413)

As was done by Ahmad et al,[17] to account for the dependence of right and left joints from a single individual, 95% confidence intervals (CIs) for kappa statistics were calculated using the bootstrap method with 5,000 replications.[57] The kappa statistic for the first analysis was 0.618, in the range of fair to good reliability; the kappa statistic for the second analysis was 0.7425, at the threshold of excellent reliability; the kappa statistic for the third analysis was 0.7652, in the range of excellent reliability. All of the kappa statistics were statistically significantly larger than zero. The most relevant analysis is

likely the second, which considers degenerative joint disease as either present (DJD or DJD II) versus absent (normal or remodeling), because, as Ahmad et al[17] noted, the RDC/TMD requires that dichotomous radiologic ratings be used in the clinical TMD diagnosis algorithm.

Last, post-treatment CBCTs for the matched subset were loaded into i-CAT Vision (Imaging Sciences International, LLC, Hatfield, PA) and PrintKey 2000, Version 5.05 FreeWare (Nuerensdorf, Switzerland), was used to capture digital images of sagittal and axially corrected coronal views of each subject in the matched subset at post-treatment. An independent party re-randomized the matched subset in Microsoft Excel 2008 for Mac, Version 12.3.4 (Microsoft Corp, Redmond, WA), and assigned numbers to the subjects in ascending order (1-78), and then renamed the subjects' corresponding post-treatment digital image files accordingly. The orthodontic resident blindly diagnosed the right and left joints of 76 of 78 matched subjects using the previously captured, static post-treatment CBCT images. The post-treatment CBCT images for two subjects were not of diagnostic quality, and these two subjects (four joints) were removed from the study; hence, no diagnoses or findings for these four joints were included in the pre-treatment or post-treatment statistical analyses. Therefore, the final number of pre-treatment and post-treatment joints that were analyzed was N = 152 (76 subjects x 2 = 152 joints).

A summary of the study design is presented in Fig. 3.

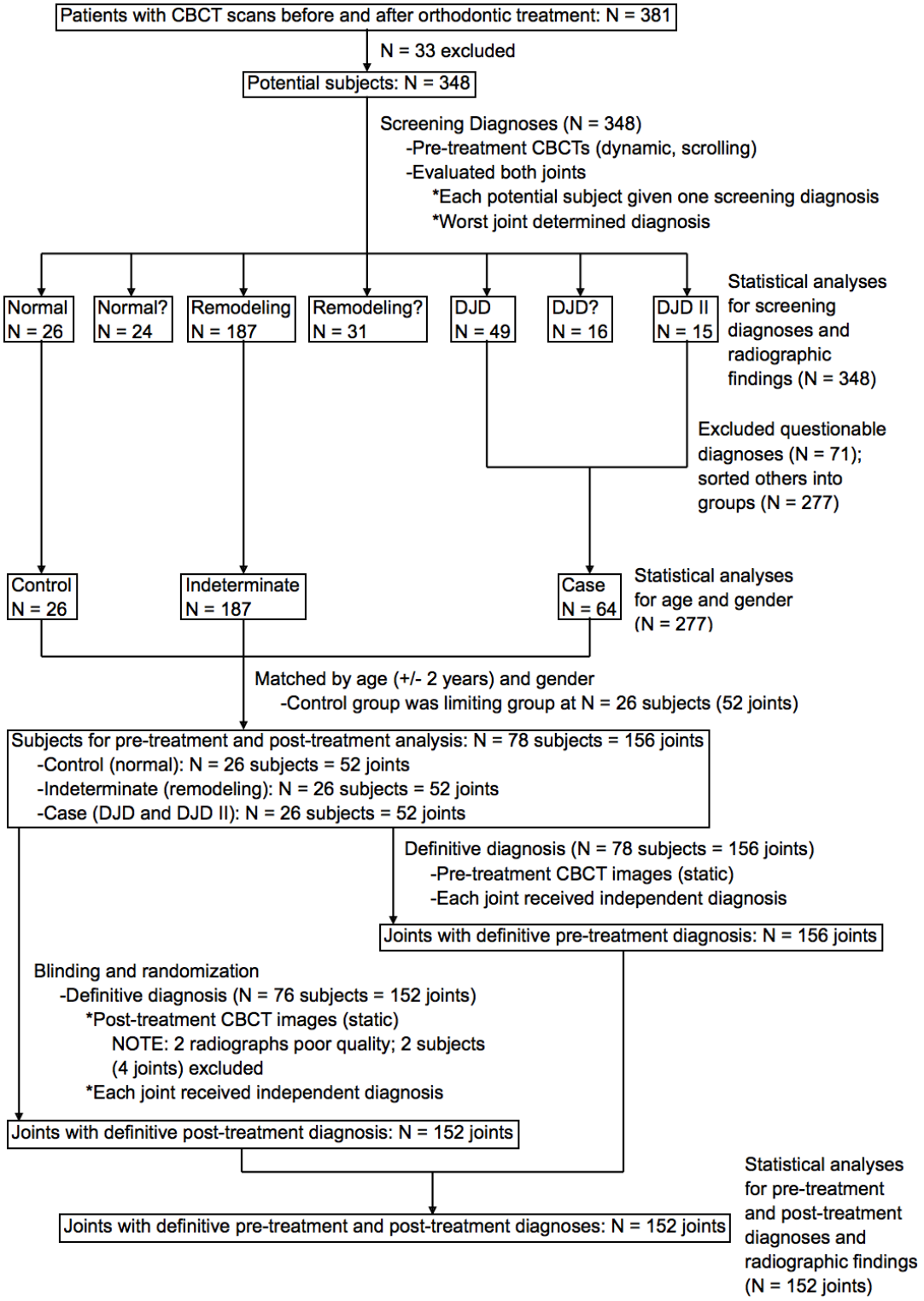


Fig. 3. Flow chart summarizing study design.

RESULTS

SCREENING POPULATION (N = 348 SUBJECTS))

The screening diagnoses of the original pre-orthodontic population are shown in Fig. 4. Considering subjects with screening diagnoses of certainty, remodeling was by far the most common (53.7%), followed by DJD (14.1%), normal (7.5%), and DJD II (4.3%). If the certainty aspect is ignored and questionable diagnoses are combined with their certain, sibling diagnoses, and all DJD diagnoses are combined, remodeling diagnoses (Remodeling + Remodeling?) constitute 62.6% of the total, DJD diagnoses (DJD I + DJD II? +DJD II) constitute 23.0%, and normal diagnoses (Normal + Normal?) constitute 14.4%. Considering radiographic findings of certainty (and ignoring questionable findings), the vast majority of subjects had flattening (78.2%) or sclerosis (58.6%) in at least one joint (this includes subjects with DJD); 16.1% had osteophyte, 4.0% had erosion, and 3.7% had subcortical cyst (Table III). The most common combination of findings was flattening and sclerosis at 38.8% (Table IV); 15.5% had zero total certain findings, 27.3% had one total certain finding, 42.8% had two total certain findings, etc (Table V).

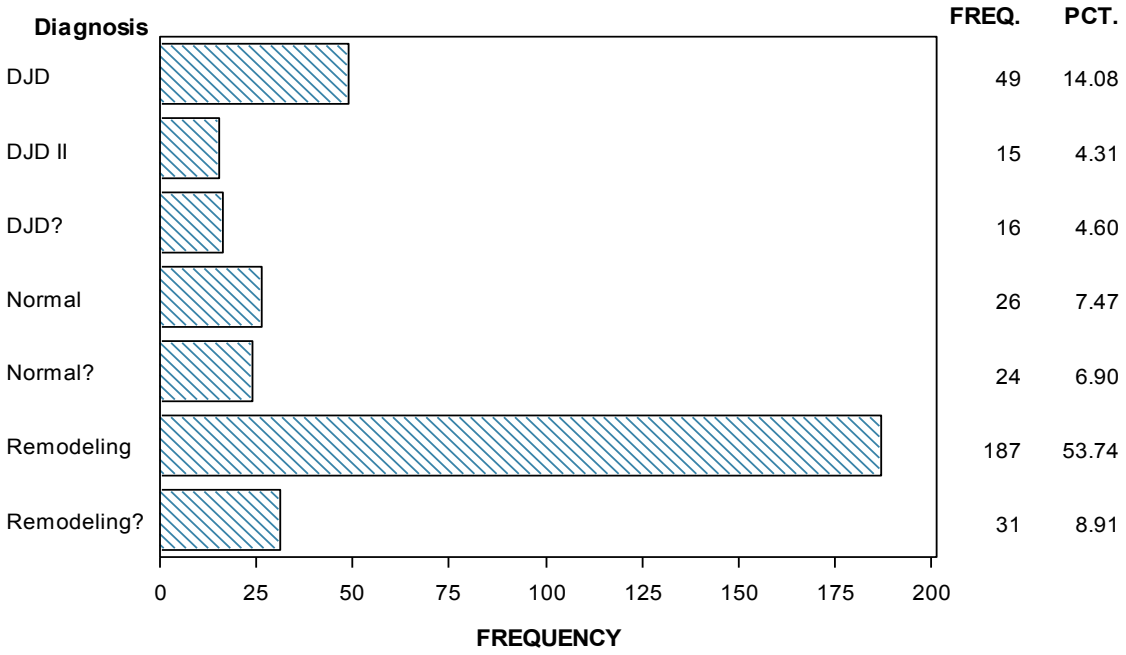


Fig. 4. Histogram showing the frequency of screening diagnoses in the pre-orthodontic screening population (prior to exclusion of questionable diagnoses;
N = 348).

Table III. Radiographic findings in the pre-orthodontic screening population (N = 348).

Radiographic Findings	% of Total
Sclerosis	58.6
Flattening	78.2
Osteophyte	16.1
Erosion	4.0
Cyst	3.7
Total	100

Table IV. Combinations of radiographic findings in the pre-orthodontic screening population (N = 348).

No finding = 0 Sclerosis = 1 Flattening = 2 Osteophyte = 3 Erosion = 4 Cyst = 5		
Radiographic Combination	Total Number of Radiographic Combinations	% of Total
1,2	135	38.8
2	76	21.8
0	54	15.5
1,2,3	28	8.1
1	19	5.5
2,3	11	3.2
1,2,3,4	8	2.3
1,2,5	5	1.4
1,2,3,4,5	3	0.9
2,5	2	0.6
1,2,3,5	2	0.6
1,3	1	0.3
1,2,4	1	0.3
1,3,4	1	0.3
1,3,5	1	0.3
2,3,4	1	0.3
Total	348	100

Table V. Radiographic findings in the pre-orthodontic screening population by total number.

Number of Radiographic Findings	%
0 Total	15.5
1 Total	27.3
2 Total	42.8
3 Total	10.6
4 Total	2.9
5 Total	0.9
Total	100

SCREENING POPULATION AFTER EXCLUSION OF QUESTIONABLE DIAGNOSES AND AFTER ASSIGNMENT INTO GROUPS (N = 277 SUBJECTS)

When subjects with questionable diagnoses were excluded (N = 71), the remaining subjects were assigned into one of three groups: 1: Control (N = 26—those with a screening diagnosis of normal), 2: Indeterminate (N = 187—those with a screening diagnosis of remodeling), and 3: Case (N = 64—those with a screening diagnosis of DJD and DJD II) (Table VI, Fig. 2). The control group constituted 9.4% of the total, the indeterminate group constituted 67.5%, and the case group constituted 23.1% (Fig. 2). This population included 277 subjects—177 females, and 106 males; the relationships between group assignment and gender are shown in Table VII and Table VIII. Females were more likely to have a screening diagnosis of normal or DJD than males (11.1% vs. 6.6%, and 25.1% vs. 19.8%, respectively), and males were more likely to have a screening diagnosis of remodeling than females (73.6% vs. 63.7%, respectively); however, none of the differences between genders were statistically significant ($p = 0.2113$, using Fisher's exact test).

Table VI. Assignment of screening diagnoses into groups. **0: Exclude** = questionable; **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Diagnosis	Group				
Frequency Percent	0: Exclude	1: Control	2: Indeterminate	3: Case	Total
DJD	0 0.00	0 0.00	0 0.00	49 14.08	49 14.08
DJD II	0 0.00	0 0.00	0 0.00	15 4.31	15 4.31
DJD?	16 4.60	0 0.00	0 0.00	0 0.00	16 4.60
Normal	0 0.00	26 7.47	0 0.00	0 0.00	26 7.47
Normal?	24 6.90	0 0.00	0 0.00	0 0.00	24 6.90
Remodeling	0 0.00	0 0.00	187 53.74	0 0.00	187 53.74
Remodeling ?	31 8.91	0 0.00	0 0.00	0 0.00	31 8.91
Total	71 20.40	26 7.47	187 53.74	64 18.39	348 100.00

Table VII. Group assignment by gender with row percentages (after exclusion of questionable diagnoses; N = 277).

Group	Gender		
Frequency Row %	F	M	Total
1: Control	19 73.08	7 26.92	26
2: Indeterminate	109 58.29	78 41.71	187
3: Case	43 67.19	21 32.81	64
Total	171	106	277

Table VIII. Group assignment by gender with column percentages (after exclusion of questionable diagnoses; N = 277).

Group	Gender		
Frequency Column %	F	M	Total
1: Control	19 11.11	7 6.60	26
2: Indeterminate	109 63.74	78 73.58	187
3: Case	43 25.15	21 19.81	64
Total	171	106	277

The population with screening diagnoses after exclusion of subjects with questionable diagnoses (N = 277) had a mean age of 18.1 years, with a minimum of 10.1 years and a maximum of 66.8 years (Fig. 5). The vast majority of subjects were adolescents—209 of the 277 subjects (75.5%) were < 17.5 years of age at the time of pre-treatment CBCT. The relationships between group assignment and age are shown in Fig. 6 and Table IX. The control group had the lowest mean age at 14.5 years, followed by the indeterminate group with a mean age of 17.5 years, and the case group had the highest mean age at 21.5 years. The case group had a significantly higher mean age than the indeterminate group and the control group ($p = 0.0024$, using a 1-way ANOVA).

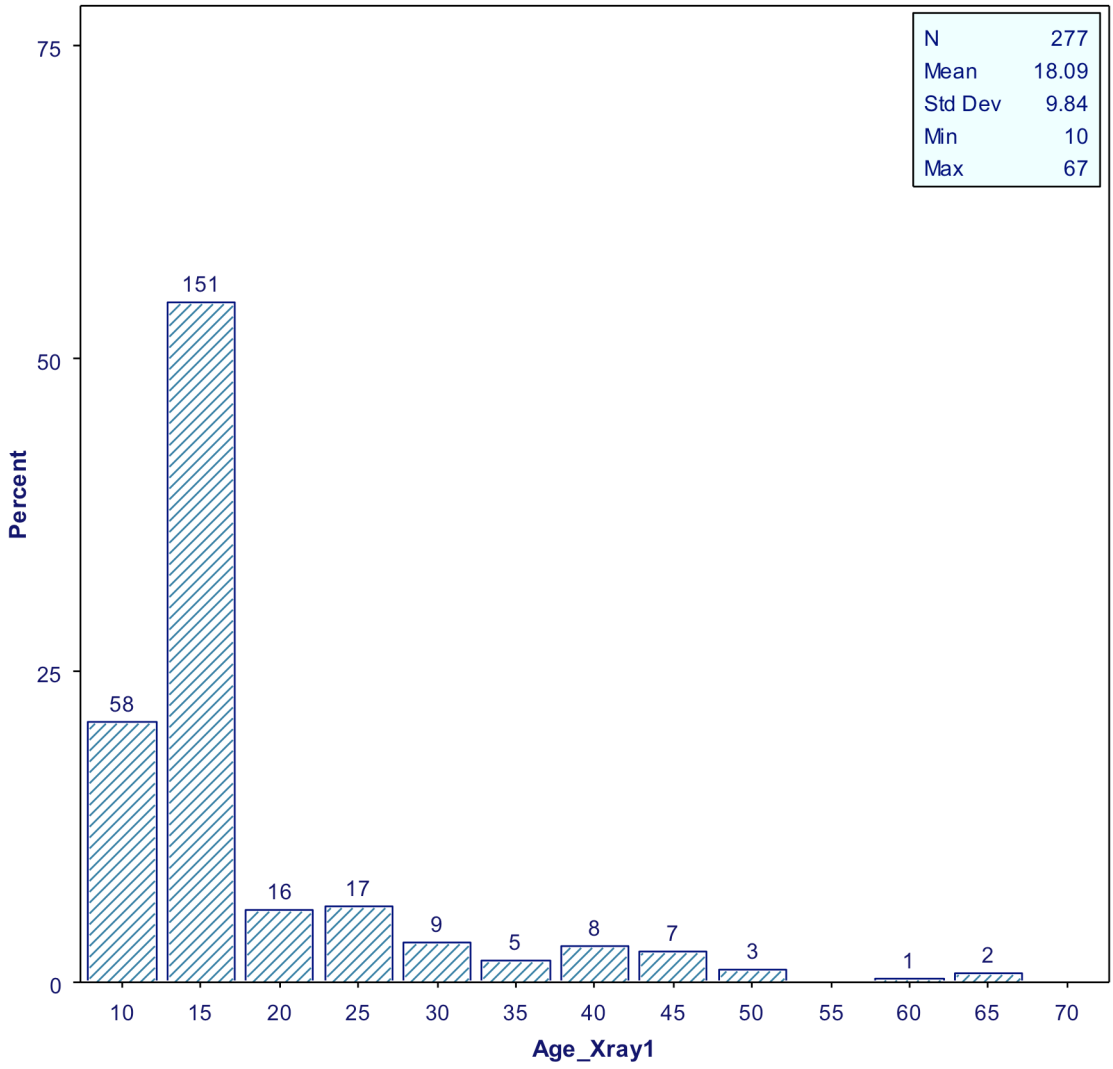


Fig. 5. Histogram of screening population showing age at pre-treatment CBCT (after exclusion of questionable diagnoses; N = 277).

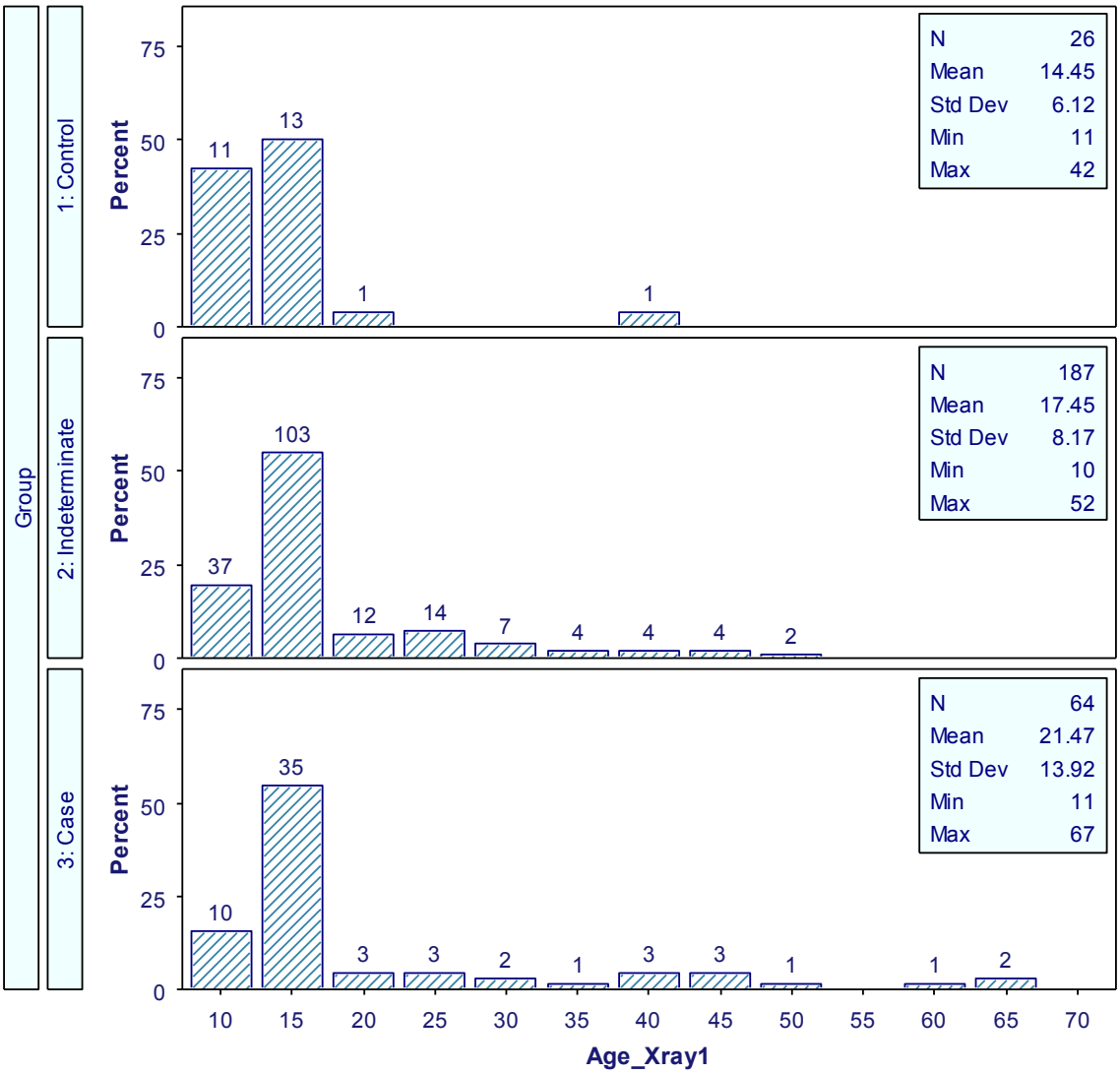


Fig. 6. Multiple histogram of age by group at pre-treatment CBCT (after exclusion of questionable diagnoses; N = 277). **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Table IX. Statistics for age by group at pre-treatment CBCT (after exclusion of questionable diagnoses; N = 277). **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Group	N Obs	Mean	Median	Std Dev	Minimum	Maximum
1: Control	26	14.45	12.94	6.12	10.59	41.86
2: Indeterminate	187	17.45	14.19	8.17	10.15	51.50
3: Case	64	21.47	15.02	13.92	10.56	66.84

MATCHED POPULATION (N = 78 SUBJECTS)

The control group, consisting of those who had both condyles radiographically normal, was the smallest of the three groups, with 26 subjects. These subjects were matched by gender and age (+/- 2 years) with subjects in the indeterminate group and subjects in the case group (matched subjects in the two latter groups were randomly selected), making a sample population of 78 subjects. Of those, 57 were females (19 in each category: case, control, and indeterminate) and 21 were males (7 in each category: case, control, and indeterminate).

The mean age of the matched subset at pre-treatment CBCT scan was 14.7 years, with a minimum of 10.6 years and a maximum of 41.9 years. The vast majority of matched subjects were adolescents—72 of the 78 subjects (92.3%) were < 17.5 years of age at the time of pre-treatment CBCT (Fig. 7).

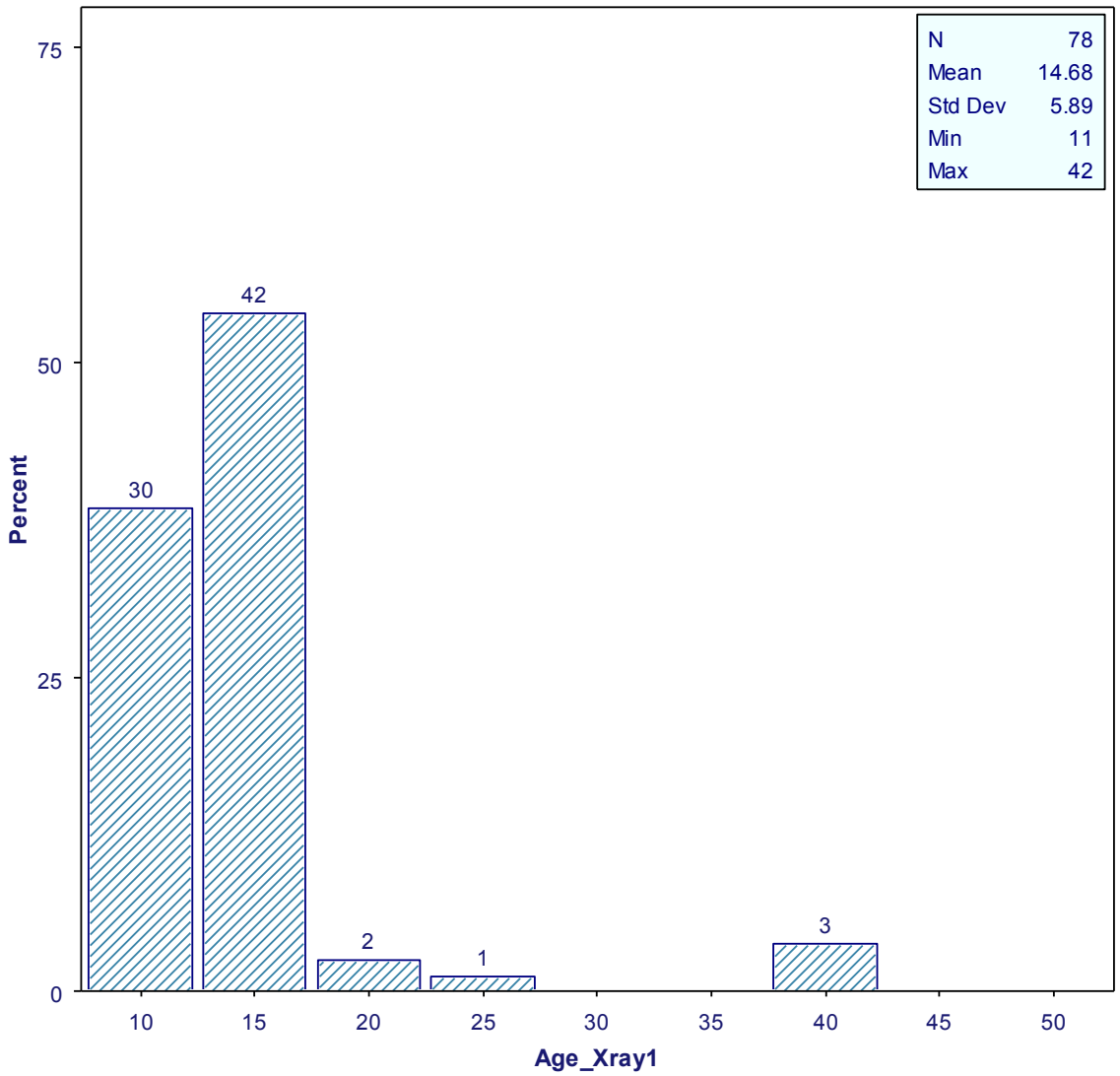


Fig. 7. Histogram of age (all three groups combined) for the matched subset at pre-treatment CBCT (N = 78).

The relationships between group assignment and age at pre-treatment CBCT are shown in Fig. 8. The matched control group had the lowest mean age at 14.5 years, followed by the matched indeterminate group with a mean age of 14.6 years, and the case

group had the highest mean age at 15.0 years. The mean age of the entire subset at post-treatment CBCT scan was 16.8 years, with a minimum of 12.2 years and a maximum of 43.7 years (Table X).

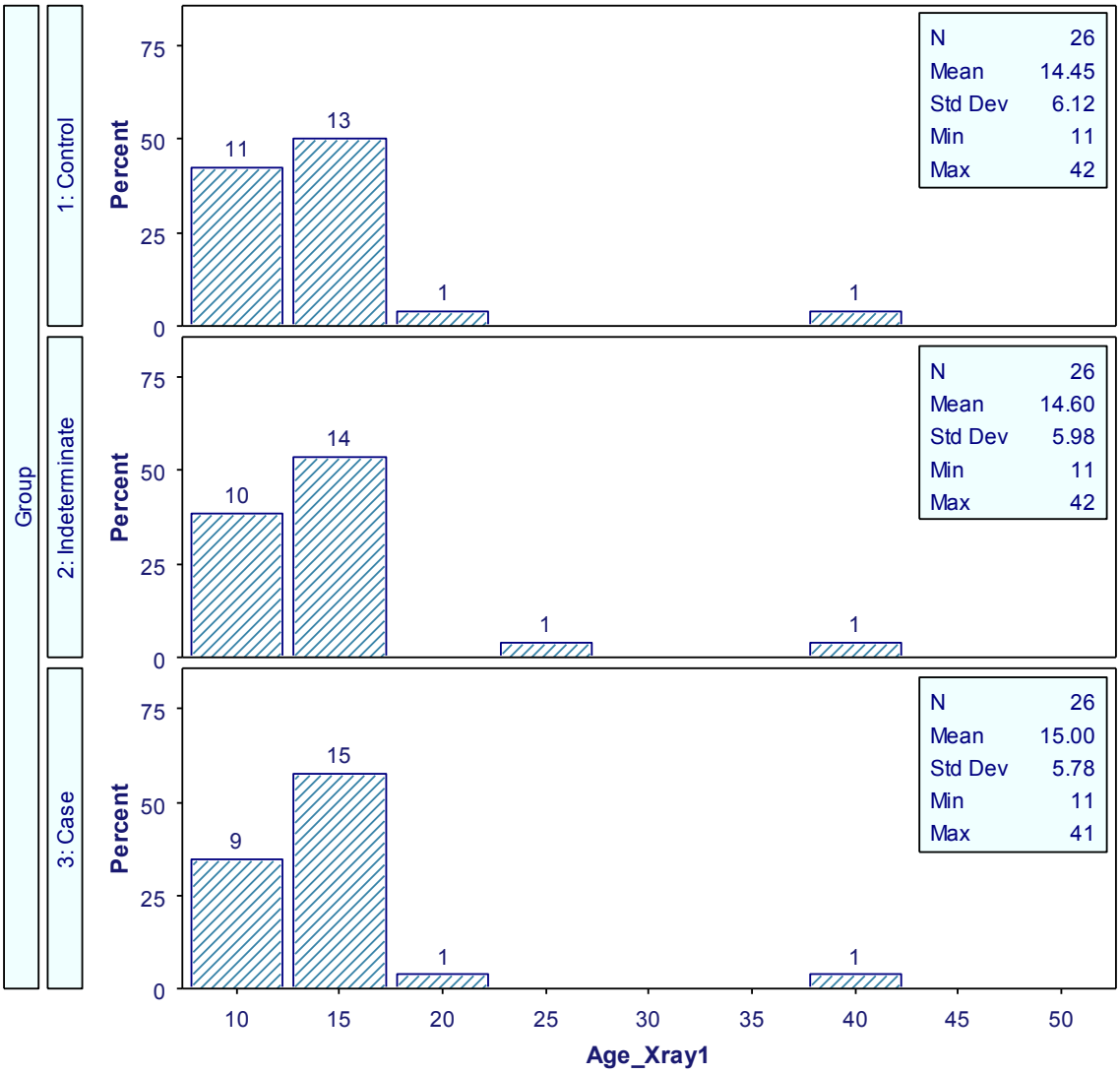


Fig. 8. Multiple histogram of age by group for the matched subset at pre-treatment CBCT (N = 78). **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Table X. Statistics for age (all three groups combined) for the matched subset at post-treatment CBCT (N = 78). **1: Control** = normal; **2: Indeterminate** = remodeling; **3: Case** = DJD + DJD II.

Age at Post-treatment CBCT (N = 78 subjects)					
N	Mean	Median	Std Dev	Minimum	Maximum
78	16.75	15.48	5.90	12.19	43.73

JOINTS FROM THE MATCHED POPULATION WITH DEFINITIVE PRE-TREATMENT AND POST-TREATMENT DIAGNOSES (N = 152 JOINTS)

The pre-treatment and post-treatment CBCT images were used to make definitive diagnoses of 152 joints (N = 152) from the matched subset (as noted earlier, 2 subjects = 4 joints, were excluded due to poor-quality CBCTs); the aspect of certainty was ignored. 'Marginal' and 'conditional' models were used to explore how diagnoses changed after treatment using a proportional odds regression. The marginal model answers the question: what is the average difference in diagnosis severity between pre-treatment and post-treatment? The conditional model, on the other hand, seeks a more specific answer: how does post-treatment severity depend on the pre-treatment severity? Changes in radiographic findings were measured with a marginal model (similar to what was done for joint diagnoses). The conditional model used for the analysis is a proportional odds regression,[58] with the post-treatment diagnosis and the pre-treatment diagnosis as the dependent and independent variables, respectively. The marginal model uses both the pre-treatment and post-treatment diagnosis as outcomes with the time as the predictor of interest (i.e., pre-treatment verses post-treatment). The correlation induced by multiple measurements within a person was accounted by generalized linear mixed models.[59]

Table XI presents the results of the marginal model. The odds were 20% less that a joint would have a worse diagnosis before orthodontic treatment than after orthodontic treatment, but the effect was not statistically significant (p = 0.378).

Table XI. Marginal model for changes in joint diagnosis, pre-treatment/post-treatment, for the matched subset (after exclusion of 4 joints; N = 152 joints).

Time	Odds Ratio	95% Confidence Interval	P-Value
Pre-treatment Diagnosis	0.80	(0.50, 1.31)	0.378

The conditional model results are presented in Table XII. This analysis was designed to determine whether a joint was more likely to have a worse post-treatment diagnosis given a particular pre-treatment diagnosis. For example, a joint with a diagnosis of DJD before orthodontic treatment had, on average, approximately 42 times (4225%) higher odds of a worse diagnosis after orthodontic treatment than a joint with a normal diagnosis before orthodontic treatment; a joint with a diagnosis of indeterminate (remodeling) before orthodontic treatment had, on average, about 6 times (602%) higher odds of a worse diagnosis after orthodontic treatment than a joint with a normal diagnosis before orthodontic treatment. The likelihood of having a worse diagnosis post-treatment was statistically significant given a pre-treatment diagnosis of DJD or indeterminate ($p < 0.001$ and $p = 0.001$, respectively).

Table XII. The results for the conditional model on post-treatment joint diagnosis for the matched subset (after exclusion of 4 joints; N = 152 joints).

Initial Diagnosis	Odds Ratio	95% Confidence Interval	P-Value
DJD	42.25	(10.63, 167.93)	< 0.001
Indeterminate	6.02	(2.09, 17.38)	0.001

Tables XIII-XV show how joint diagnoses for the sample changed from pre-treatment to post-treatment. The number of joints with normal diagnoses decreased after orthodontic treatment from 54 to 43, the number of joints with remodeling increased after orthodontic treatment from 62 to 76, and the number of joints with DJD (DJD I or DJD II) decreased after orthodontic treatment from 36 to 33 (Table XIV). At post-treatment, 52.6% of joints had the same diagnosis, 25% had a worse diagnosis, and 22.4% had a better diagnosis (Table XV).

Table XIII. Changes in joint diagnoses from pre-treatment to post-treatment in the matched subset, considering all four diagnoses (after exclusion of 4 joints; N = 152 joints).

Pre-Treatment	Post-Treatment	Total	% Change
Normal	Normal	29	53.7
Normal	Remodeling	24	44.4
Normal	DJD	1	1.9
Normal	DJD II	0	0.0
Pre-Treatment Normal		54	100
Remodeling	Normal	13	21.0
Remodeling	Remodeling	38	61.3
Remodeling	DJD	9	14.5
Remodeling	DJD II	2	3.2
Pre-Treatment Remodeling		62	100
DJD	Normal	1	4.2
DJD	Remodeling	12	50.0
DJD	DJD	9	37.5
DJD	DJD II	2	8.3
Pre-Treatment DJD		24	100
DJD II	Normal	0	0.0
DJD II	Remodeling	2	16.7
DJD II	DJD	6	50.0
DJD II	DJD II	4	33.3
Pre-Treatment DJD II		12	100

Table XIV. Overall changes in joint diagnoses from pre-treatment to post-treatment in the matched subset, considering all four diagnoses (after exclusion of 4 joints; N = 152 joints).

Diagnosis	Total Pre-Treatment	Total Post-Treatment	% Change
Normal	54	43	20.4
Remodeling	62	76	22.6
DJD	24	25	4.2
DJD II	12	8	33.3
Total	152	152	

Table XV. Changes in joint diagnoses by scale and overall from pre-treatment to post-treatment in the matched subset (after exclusion of 4 joints; N = 152 joints).

Change by (-) = better diagnosis at post-treatment than pre-treatment Change by (+) = worse diagnosis at post-treatment than pre-treatment			
Changes in Diagnoses by Scale		Overall Changes	%
Change by -3	0	Better = 34	22.4
Change by -2	3		
Change by -1	31		
Change by 0	80	Same = 80	52.6
Change by +1	35	Worse = 38	25.0
Change by +2	3		
Change by +3	0		
Total	152	152	100%

Table XVI presents the results for the analysis of radiographic TMJ findings in the joints of the matched subset. The odds of sclerosis being present in a joint were 5% higher prior to orthodontic treatment than after orthodontic treatment, the odds of flattening being present in a joint were 30% lower prior to orthodontic treatment than after orthodontic treatment, the odds of an osteophyte being present in a joint were 3% higher prior to orthodontic treatment than after orthodontic treatment, etc. None of the differences in radiographic findings from pre-treatment to post-treatment were statistically significant.

Table XVI. Changes in radiographic findings from pre-treatment to post-treatment in the matched subset (after exclusion of 4 joints; N = 152 joints).

Finding	Time	Odds Ratio	95% Confidence Interval	P-Value
Sclerosis	Pre-Treatment	1.05	(0.62, 1.77)	0.870
Flattening	Pre-Treatment	0.70	(0.41, 1.18)	0.176
Osteophyte	Pre-Treatment	1.03	(0.55, 1.92)	0.929
Erosion	Pre-Treatment	3.08	(0.83, 11.41)	0.091
Cyst	Pre-Treatment	1.14	(0.39, 3.32)	0.814

Tables XVII-XIX summarize the radiographic findings in the matched subset at pre-treatment and post-treatment. Flattening and sclerosis were the most common findings at pre-treatment and at post-treatment (Table XVII); cyst was the least common finding at pre-treatment, and erosion was the least common finding at post-treatment. When considering all possible radiographic findings (individual and combined), the most common was zero findings at pre-treatment and at post-treatment; however, the most common combination of findings was flattening and sclerosis at pre-treatment and at post-treatment (Table XVIII). At pre-treatment, 35.5% had zero total findings, 21.7% had one total findings, 27.0% had two total findings, etc.; at post-treatment, 29.6% had zero total findings, 34.2% had one total findings, 19.7% had two total findings, etc. (Table XIX).

Table XVII. Radiographic findings in the matched subset at pre-treatment CBCT and post-treatment CBCT (after exclusion of 4 joints; N = 152 joints).

Radiographic Findings	% of Total
Pre-Treatment CBCT	
Sclerosis	38.8
Flattening	57.9
Osteophyte	20.4
Erosion	6.6
Cyst	5.9
Post-Treatment CBCT	
Sclerosis	37.5
Flattening	63.8
Osteophyte	19.7
Erosion	2.6
Cyst	5.3

Table XVIII. Combinations of radiographic findings in the matched group at pre-treatment CBCT and post-treatment CBCT (after exclusion of 4 joints; N = 152 joints).

No finding = 0
Sclerosis = 1
Flattening = 2
Osteophyte = 3
Erosion = 4
Cyst = 5

Pre-Treatment CBCT			Post-Treatment CBCT		
Radiographic Combination	Total Number of Radiographic Combinations	% of Total	Radiographic Combination	Total Number of Radiographic Combinations	% of Total
0	54	35.5	0	45	29.6
1,2	29	19.1	2	39	25.7
2	28	18.4	1,2	22	14.5
1,2,3	10	6.6	1,2,3	15	9.9
2,3	8	5.3	2,3	10	6.6
1	5	3.3	1	7	4.6
1,2,3,4	4	2.6	1,2,3,5	4	2.6
1,2,3,5	3	2.0	1,2,5	3	2.0
1,3	2	1.3	1,3	2	1.3
2,5	1	0.7	1,2,4	2	1.3
3,5	1	0.7	1,2,3,4	1	0.7
1,2,4	1	0.7	1,2,3,4,5	1	0.7
1,2,5	1	0.7	3	1	0.7
1,3,4	1	0.7	Total	152	100
1,4,5	1	0.7			
2,3,4	1	0.7			
1,2,4,5	1	0.7			
1,2,3,4,5	1	0.7			
Total	152	100			

Table XIX. Radiographic findings in the matched subset at pre-treatment CBCT and post-treatment CBCT by total number (after exclusion of 4 joints; N = 152 joints).

Number of Radiographic Findings	%
Pre-Treatment	
0 Total	35.5
1 Total	21.7
2 Total	27.0
3 Total	9.9
4 Total	5.3
5 Total	0.7
Total	100
Post-Treatment	
0 Total	29.6
1 Total	34.2
2 Total	19.7
3 Total	11.2
4 Total	4.6
5 Total	0.7
Total	100

DISCUSSION

The interexaminer kappa statistic and the intraexaminer kappa statistics for the second and third analyses, in which diagnoses of normal and remodeling were combined and separated from diagnoses of degenerative joint disease, were at or above the excellent threshold. This indicates that the orthodontic resident was able to reliably differentiate the presence and absence of degenerative joint disease according to his training in the RDC/TMD imaging criteria. Interestingly, the kappa statistic for the third analysis (Non-DJDs vs. grade I DJD vs. grade II DJD) was slightly higher, at 0.7652, than the kappa statistic for the second analysis (Non-DJDs vs DJDs), at 0.7425. This may be explained because grade II DJD was slightly easier to differentiate from Non-DJDs and grade I DJD due to the severity of findings found in grade II DJD. The kappa statistic for the first analysis, in which diagnoses of normal and remodeling were separated from each other and from degenerative joint disease (Normal vs. Remodeling vs. DJDs), was lower than the other analyses (in the range of fair to good reliability), suggesting that it is more difficult to differentiate normal from remodeling joints.

It was somewhat surprising to find so few subjects in the original pre-orthodontic screening population with both joints radiographically normal (7.5%), and so many subjects with radiographic signs indicating at least one remodeling joint (53.7%) or one osteoarthritic joint (18.4%). Furthermore, it was unexpected that so many subjects would have signs of remodeling (78.2% flattening, 58.6% sclerosis, 38.8% flattening and sclerosis), although many of the signs were very minor. The number of findings detected in the current study was much higher than that reported by Cho and Jung,[43] who evaluated CBCT scans of Korean children and adolescents in the age range of 10-18

years. In an asymptomatic group consisting of 101 individuals with no TMD signs or symptoms who had CBCT scans for examination of tooth impaction or for orthodontic evaluation, osseous changes were detected in 9.9% of joints; 2.5% had flattening, 5.4% had sclerosis, 2.5% had osteophyte, and 1% had erosion (note: the percentages of individual findings don't add up to 9.9% because some findings were found together in some joints).[43] In a symptomatic group consisting of 181 individuals who sought treatment for TMD at the hospital, osseous changes were present in 26.8% of joints; 5.8% had flattening, 13% had sclerosis, 6.6% had osteophyte, and 15.6% had erosion.[43] They concluded that TMJ-OA is not uncommon in children and adolescents, regardless of the presence of symptoms.[43] However, their definition of OA differs from that described in the RDC/TMD imaging guidelines—Cho and Jung[43] defined OA as one or more osteoarthritic changes of flattening, sclerosis, osteophyte, or erosion (calling flattening and sclerosis signs of OA rather than remodeling); also, they did not evaluate for subcortical cyst.

The magnitude of osseous TMJ changes detected in the current study was also considerably higher than the condylar changes reported by Peltola et al,[8] who detected condylar findings in only 2% of pre-orthodontic subjects and 9% of post-orthodontic subjects. However, the validity of studies using panoramic radiography to assess TMJs are questionable due to inherent limitations with panoramic radiography;[48] also, as Ahmad et al[17] discovered, approximately 75% of CT-based OA is not detected when using panoramic radiography, and the interexaminer reliability is poor ($\kappa = 0.16$). Furthermore, at the time Peltola et al[8] did the study examining the TMJs of subjects prior to and after orthodontic treatment, panoramic radiography had not been included as an

imaging option in the original RDC/TMD);[17] therefore, no RDC/TMD imaging criteria were available.

The number of findings detected in the original pre-orthodontic screening population of the current study are more similar to those reported by Pliska et al,[44] who evaluated the radiology reports from CBCT scans of patients presenting for diagnostic records at the University of Minnesota Graduate Orthodontic Department from July of 2008 to July of 2009. The radiology reports were made by one of two board certified oral and maxillofacial radiologists at the University of Minnesota School of Dentistry, one of whom was M. A. Pliska et al[44] found that 34.5% of 194 total patients had incidental findings (findings that required continued monitoring) of the TMJ, including flat condylar margin, irregular margin, subcortical sclerosis, and bifid condyle; the most prevalent incidental findings was flat condylar margin. They found that 18% of patients had significant TMJ findings (findings that required immediate follow-up), including osteophyte, subcortical cyst, degenerative changes, and anteriorly displaced disc.[44] It is interesting to note that some of the patients in the study by Pliska et al[44] must have also been subjects in the current study (the studies had inclusion periods that overlapped); however, the primary investigator of the current study (B. A.) was not aware of the existence of the former study until after data collection was complete. The relative similarity in findings is not entirely surprising, since the populations had some of the same subjects and M. A.'s diagnostics affected both studies (directly for Pliska et al[44], and indirectly for the current study).

In the pre-orthodontic screening population of the current study, females were more likely to have a screening diagnosis of DJD than males, but the difference was not

statistically significant. However, multiple studies have shown that OA is more common females than males.[2,4,12,22,42] It is interesting that in the current study, females not only were more likely to have a screening diagnosis of DJD than males, but they were also more likely to have a screening diagnosis of normal than males, while males were more likely to have a screening diagnosis of remodeling than females; however, none of the differences were statistically significant.

Subjects with a screening diagnosis of DJD were significantly older than subjects with a screening diagnosis of remodeling or normal, consistent with the reports of others that the frequency of degenerative changes increases with age.[2,4,23,38] It was therefore not unexpected that subjects with a screening diagnosis of remodeling were significantly older than subjects with a screening diagnosis of normal.

There was no statistical evidence that, on average, a worse joint diagnosis existed pre-treatment or post-treatment. However, the likelihood of having a worse diagnosis post-treatment was statistically significant given a pre-treatment diagnosis of DJD or indeterminate. Consistent with the findings of other studies,[7,8,42,60] individual joint diagnoses in the current study tended to stay the same (52.6%) or get worse (25%) with time, but some (22.4%) got better. In a study using transcranial projection coupled with either transpharyngeal or panoramic projection to assess the TMJs of adolescents and young adults who had initial radiographic signs of TMJ-OA, Zhao et al[42] found at follow-up radiographic examination that the joints of 56.4% of patients remained unchanged, 23.1% showed signs of progression (i.e., radiographic signs of OA were more severe), and 20.5% showed signs of improvement (i.e., radiographic signs of OA were less severe). These percentages are remarkably similar to those in the current study. However,

it is important to note that the classification of TMJ-OA used by Zhao et al[42] was not based on the RDC/TMD imaging criteria. Peltola et al[8] found that, of 14 orthodontic subjects who initially had condylar findings in panoramic radiographs, 3 were unchanged (21.4%), 5 had become more severe (35.7%), and 6 had become normal (42.9%) after orthodontic treatment. Koyama et al[60] used MDCT to reevaluate the TMJs of 51 TMD subjects; the mean age of these subjects was 29.4 years, and the mean interval between initial MDCT and follow up MDCT was 13.4 months.[60] They found that although condylar osseous changes tended to stay the same or worsen with time, a minority of joints did transition from showing signs of remodeling (flattening) or osteoarthritis (erosion) to normal. Of initial remodeling joints with flattening, 8% were normal at follow-up; of initial osteoarthritic joints with erosion, 14% were normal at follow-up.[60] The current study found that, of pre-treatment joints with diagnoses of remodeling, about 21% (13/62) were normal after orthodontic treatment; of pre-treatment joints with diagnoses of DJD, about 4% (1/24) were normal after orthodontic treatment. Considering the 4 possible diagnoses in the current study—normal, remodeling, DJD and DJD II—individual joints tended to stay the same (80/152—52.6%) or progress by one diagnosis or improve by one diagnosis (66/152—43.4%); very few changed by 2 diagnoses (6/152—4%), and zero joints changed by 3 diagnoses. Only one joint with a pre-treatment diagnosis of normal had progressed to DJD at post-treatment, and only one joint with a pre-treatment diagnosis of DJD had improved to normal at post-treatment. In light of the variability in joint diagnoses at pre-treatment and post-treatment, the clinical significance of minor signs of DJD, particularly in young, growing patients, is questionable. Perhaps what appear to be minor signs of DJD in adolescents may actually

be the result of normal joint development and remodeling. Furthermore, the results of the current study can be interpreted in a positive light. Rather than saying that joint diagnoses tended to stay the same (52.6%) or get worse (25%) with time, it can also be said that many joint diagnoses stayed the same (52.6%) or got better (22.4%); or, in other words, 75% of joint diagnoses were either the same or better following treatment.

Considering signs of remodeling, our results were consistent with those of other studies that have found TMJ flattening and sclerosis as the most common or among the most common individual findings,[4,6,8,35,61] and flattening and sclerosis as the most common combination of findings.[8] However, the general findings of remodeling in the current study are considerably higher than several others,[8,42,43] but are more consistent with what was discovered by Brooks et al,[34] Hansson et al,[24] and Muir and Goss.[35] Brooks et al[34] detected minimal flattening via tomography in 35% of joints in asymptomatic subjects with no internal derangement. Hansson et al[24] found adaptive TMJ changes in 85% of young adult autopsy specimens. Muir and Goss[35] found condylar flattening and sclerosis in 70% and 73% of asymptomatic subjects, respectively, using panoramic radiography. Therefore, with potentially so many of the population with signs of TMJ remodeling, the question begs to be asked—is a radiographically "normal" TMJ actually the norm? Orthodontists make a living because normal occlusion—teeth that interdigitate perfectly and are arranged along a perfectly regular line of occlusion—is actually quite rare.[62] Perhaps the same is true with regard to the morphology of the TMJs. Perhaps perfectly round condyles and eminences that show no signs of flattening or sclerosis (i.e., "normal" joints) are a minority. Perhaps, like the presence of mammelons on the maxillary and mandibular incisors, when signs of joint remodeling are absent, the

joint is either so immature that it has not been exposed to functional stresses requiring it to remodel, or normal function is lacking or absent.

EXPLANATIONS FOR DIFFERENCES IN FINDGINS AMONG STUDIES

Potential explanations for differences in radiographic findings between the current study and others include the use of different radiographic modalities, different diagnostic criteria, examiner variation, population differences, and the existence of relatively few studies.

Different Radiographic Modalities

It seems logical that findings should be more common when using MDCT or CBCT than other radiographic modalities because, not only is there is so much more information—multiple views and multiple slices in all three dimensions—in which findings can be made, but the TMJ anatomy can also be examined without superimposition and distortion.[12]

Different Diagnostic Criteria

As Roda et al[16] and Luther[14] suggested in their TMD studies, application of different diagnostic criteria can also lead to considerable variability in results found in the published literature. And, after reviewing the published literature, the principal investigator of the current study did not find any studies that evaluated the TMJs of an orthodontic population—either before or after orthodontic treatment—that followed the guidelines of the RDC/TMD Validation Project. Therefore, it is difficult to compare results with other studies that have used different radiographic modalities and/or different diagnostic criteria, and the validity of such comparisons is questionable.

Examiner Variation

Even when the same diagnostic criteria are applied among studies, examiner variation may account for differences in results. As Muir and Goss[35] noted, even with training, there is significant variation in interexaminer interpretation. Variation in intraexaminer interpretation was noticed in the current study; when making definitive diagnoses of the joints of the matched subjects it was noted that several subjects would have been given different screening diagnoses if screening diagnoses had been given at that time. However, joint diagnoses in the matched subset were considered definitive, which is supported by research; regarding intraexaminer reliability, Ludlow et al[46] found that when examiners were given a second viewing session, all were more accurate (demonstrating improved detection of simulated condylar lesions) at the second session. Therefore, even when applying the same diagnostic criteria, interpretation variation exists among examiners, and reliability is never perfect within examiners. The root of variation likely lies in the fact that there is still quite an element of subjectivity when applying the same diagnostic criteria; for example, will miniscule signs of flattening or sclerosis be scored, even if the joint looks very nearly normal? In the current study, even very minor signs of radiographic findings were scored.

Population Differences

Differences in study populations are also an important consideration when comparing results. For example, the population of the current study, which consisted mostly of adolescents, may have something to do with the relatively high prevalence of sclerosis that was detected. Peltola et al[8] discussed that cortical outlines of the condyles are difficult to observe radiographically in children and that autopsy studies have shown

that sclerosis is often a false positive. Furthermore, Lei et al[63] used CBCT to evaluate the TMJs of 1438 subjects between the ages of 10 and 30 (who had no signs or symptoms of TMD), and found that subchondral formation of condylar cortical bone was not seen until 12-14 years of age, and cortical bone formation begins around the periphery of the condyles; a continuous, uniform, compact layer of cortical bone is not established until after 21-22 years of age.[63] Therefore, it is possible that what the orthodontic resident in the current study interpreted as sclerosis may have simply been, in some instances, partial formation of cortical bone. The RDC/TMD guideline that was used for the current study to identify subcortical sclerosis —"Any increased thickness of the cortical plate in the load-bearing areas relative to the adjacent nonload-bearing areas"[17] (2009, p. 846)—may need to be modified for adolescents, since they will likely show partial formation of cortical bone, which may be misinterpreted as sclerosis since the cortical plate is not homogeneous. Furthermore, It is not clear how signs of remodeling and OA differ between older and younger populations. Peltola et al[8] discussed that Akerman et al[40] interpreted flattening, minor osteophytes, and sclerosis in elderly people to be signs of remodeling, but commented that it is open to question whether osteophytes and combined lesions are normal in young adolescents. Therefore, there seems to be no validated consensus in the scientific community as to how the TMJs of young, growing individuals should appear radiographically. Consequently, it seems reasonable that more information is needed to determine the appropriateness of applying the current RDC/TMD imaging criteria to adolescents.

Few Studies

Last, it will be reiterated that there are few studies on the prevalence of TMJ osseous changes (particularly studies using CT),[2] and that radiographic studies examining the TMJs of children and adolescents have rarely been carried out.[43] Furthermore, with relatively few similar studies, a wide variation in results does not seem unusual. However, with increasing use of CBCT in adolescent patients for orthodontic records, it is important that orthodontists understand what is normal and what is abnormal so they can counsel with their patients and patients' families appropriately.

LIMITATIONS OF THIS STUDY

A major limitation of this study was the absence of a true control group (one that did not have orthodontic treatment); therefore, it can't be determined whether orthodontic treatment had any effect on the natural history of joint diagnoses or radiographic findings. Other limitations include failures to account for growth, treatment mechanics, and the presence of TMD symptoms.

CONCLUSIONS

The aims of this study were to (1) use CBCT and the RDC/TMD imaging criteria to assess the osseous components of the TMJs of a pre-orthodontic population and to evaluate for remodeling and degenerative changes, and (2) evaluate the natural history of radiographic TMJ diagnoses and findings in an orthodontic sample population by interpreting CBCT scans, made before and after comprehensive orthodontic treatment,

according to the RDC/TMD imaging criteria. The findings pertinent to the sample studied are summarized below:

1. The majority of pre-orthodontic subjects had radiographic TMJ findings indicative of remodeling in at least one joint; subjects with a diagnosis of DJD in at least one joint were next most common, and subjects with both joints radiographically normal were least common.
2. Females were more likely to have a screening diagnosis of normal or DJD than males, and males were more likely to have a screening diagnosis of remodeling than females; however, the differences between the genders were not statistically significant.
3. Subjects with a screening diagnosis of DJD were significantly older than subjects with a screening diagnosis of remodeling or normal, and subjects with a screening diagnosis of remodeling were significantly older than subjects with a screening diagnosis of normal.
4. There was no statistical evidence that, on average, a worse joint diagnosis existed pre-treatment or post-treatment.
5. The likelihood of having a worse diagnosis post-treatment was statistically significant given a pre-treatment diagnosis of DJD or indeterminate. Joint diagnoses tended to stay the same (52.6%) or worsen (25%) from pre-treatment to post-treatment, although a considerable number improved (22.4%). Viewed optimistically, however, 75% of joint diagnoses were either the same or better following treatment.

FUTURE DIRECTIONS

Future studies might involve 3-D surface mapping to quantify morphologic changes to TMJs before and after orthodontic treatment. Using this technology, Cevidanes et al [64] recently demonstrated profound morphologic differences between human condyles with OA and asymptomatic condyles. Also, future studies should address TMJ changes with orthodontic treatment mechanics, race, malocclusions prior to treatment, status of growth, etc.

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