

Correlating Cone Beam CT Results with Temporomandibular joint pain of Osteoarthritic origin: A Retrospective study

Ginalyn Palconet

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Approved by:

Dr. John Ludlow

Dr. Pei Feng Lim

Dr. Donald Tyndall

ABSTRACT

GINALYN PALCONET: Correlating Cone Beam CT Results with Temporomandibular joint pain of Osteoarthritic origin: A Retrospective Study
(Under the direction of John Ludlow)

Purpose:

To determine if Temporomandibular Joint bony changes are correlated with self-reported pain, limitation in range of motion, crepitation, pain on palpation and jaw use.

Materials and Methods:

Clinical data and Cone Beam Computed Tomography images of 30 patients with TMJ osteoarthritis at the UNC School of Dentistry Orofacial Pain Clinic were analyzed.

Koyama's¹ and Ahmad's² criteria were used to classify the condylar bony changes. Clinical measures included pain rating and mandibular range of motion. Generalized Linear Modeling was used to correlate the clinical and radiographic findings. Spearman's Rho was used to correlate Koyama's with Ahmad's classifications.

Results:

Correlation between the maximum condyle change and verbal pain rating and mandibular range of motion was poor. However both the Koyama and RDC/TMD classifications were highly correlated for average and maximum bony change.

Conclusion:

The findings of this study do not support the use of radiographs alone for diagnosis and treatment planning.

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DEDICATIONS

I would like to dedicate this project to my inspiration for the last 22 years, my daughter Angel, for always being with me especially in the most difficult days of the last 3 years.

TABLE OF CONTENTS

LIST OF TABLES	vii
LIST OF FIGURES	viii
ABBREVIATIONS	ix
CHAPTER 1	1
INTRODUCTION.....	1
TEMPOROMANDIBULAR DISORDERS.....	2
OSTEOARTHRITIS	2
PREVIOUS STUDIES CORRELATING TMJ OSTEOARTHRITIS WITH RADIOGRAPHIC FINDINGS	5
PAIN.....	7
HYPOTHESIS	8
CHAPTER 2	9
MATERIALS AND METHODS	9
STATISTICAL ANALYSIS	12
CHAPTER 3	12
RESULTS.....	12
DATA ANALYSIS FOR RELIABILITY STUDIES.....	18
CHAPTER 4.....	20
DISCUSSION	20

CHAPTER 5	29
CONCLUSION	29
REFERENCES.....	26

LIST OF TABLES

Table

1. Univariate table showing the descriptive statistics of subjects 14
2. Table of descriptive statistics of Right and Left condyle changes with Koyama and RDC/TMD classification 15
3. Table of descriptive statistics of Right and Left glenoid changes with RDC/TMD classification 15
4. Maximum bony change of the right and left condyle analyzed with verbal pain score and ranges of motion..... 16
5. Table correlating the average and maximum changes for condyle and glenoid fossa..... 18
6. Intraobserver and Interobserver analysis 19

LIST OF FIGURES

Figure

1. CBCT condylar bony changes adapting Koyama's classifications 11
2. Condylar bony changes adapting RDC/TMD classification..... 12
3. Verbal pain rating score correlated with Koyama & RDC/TMD classifications of bony changes..... 17
4. Ranges of motion correlated with Koyama & RDC/Ranges of motion correlated with Koyama & RDC/TMD classification of bony changes 17

ABBREVIATIONS

3D	three-dimensional
AAOMR	American Academy of Oral and Maxillofacial Radiology
ACST	Axially Corrected Sagittal Tomography
Ave	average
CBCT	Cone beam Computed tomography
CT	Computed Tomography
DICOM	Digital Communications in Medicine
DVT	Digital Volume Tomography
GLM	Generalized Linear Model
Glen	glenoid
HCT	Helical Computed Tomography
HPA	Hypothalamic Pituitary Axis
IndetOA	Indeterminate for Osteoarthritis
L lateral	left lateral
marg prolif	marginal proliferation
Max	maximum
mm	millimeter
MRI	Magnetic Resonance Imaging
NGF	Nerve Growth Factor
OA	Osteoarthritis
open	opening
osteoph	osteophyte

PGE2	Prostaglandin E2
RDC/TMD	Research Diagnostic Criteria for Temporomandibular Disorders
R lateral	right lateral
SAS	Statistical Analysis System
TMJ	Temporomandibular joints

CHAPTER 1

INTRODUCTION

Temporomandibular joint (TMJ) imaging is very challenging because the bony components are small and superimpositions from the base of the skull often result in a lack of clear delineation of the joint.^{3,4} Different imaging modalities have been used for diagnosing TMJ osteoarthritis. These include plain film radiographs, panoramic radiographs, conventional and Computed Tomography (CT) and Magnetic Resonance Imaging (MRI). Some problems associated with these modalities include anatomic superimpositions with plain films and panoramic radiography; high radiation doses with computed tomography; and long scanning time and in some cases restricted availability MRI.⁴ According to Westesson et al, CT has a greater sensitivity and specificity than MRI in detecting changes in the bone, thereby concluding that CT is superior in depicting osseous abnormalities.⁵

Due to the increasing demand for 3-dimensional information in activities such as implant treatment planning, a compact low-dose machine was developed specifically designed for maxillofacial imaging. This is known as the Cone Beam Computed Tomography (CBCT) or Digital volume tomography (DVT). CBCT is a fairly new imaging modality for the TMJ. Its use in dentistry started only in 1998.⁴ The use of CBCT has gained increasing popularity among dentists because it produces images of high diagnostic quality, using generally less radiation dose than medical CT, is more readily accessible by the dental patient, and costs less than comparable medical CT examinations.^{5,6,7} Also, CBCT completely eliminates the

superimpositions of bony structures in the TMJ seen in conventional and panoramic radiographs although visualization of the disc is not possible.

TEMPOROMANDIBULAR DISORDERS

Temporomandibular Disorders (TMD) are disorders affecting the TMJ masticatory muscles and/or associated structures.⁸ TMD is a common disorder with approximately 33% of the population showing at least one TMD symptom and 3.6% to 7% of the population are afflicted with TMD severe enough to cause them to seek treatment.⁹ TMD symptoms are usually associated with parafunctional activities, including clenching and bruxism causing masticatory muscle tension.⁸ Symptoms are also commonly seen with an increase of psychosocial factors such as stress, frustration and depression.⁹

OSTEOARTHRITIS

Osteoarthritis , also known as degenerative joint disease (DJD)¹⁰ is an age-related disorder and the most common pathological condition affecting the TMJ. It is a destructive process of the bony articular surfaces of the mandibular condyle and glenoid fossa often brought about by increased loading of the joint. Continuous loading results in resorption of the subarticular bone. TMJ osteoarthritis is characterized by a gradual progressive destruction of articular tissues. With advanced degeneration, the subchondral cortical layer is lost and erosion and other radiographic signs of osteoarthritis appears.¹¹⁻¹³ Patients suffering from TMJ osteoarthritis usually experience pain of different intensity, limited mandibular movement and crepitations.¹⁴

Normally, the TMJ has a capability to adapt to functional demands as a result of continuous remodeling so that there will be balance between form and function. In cases where there is joint overloading, the ability of the joint to remodel may be exceeded and the articular surface is unable to remodel and bring about a breakdown of the articular tissues.¹⁵

Previous histologic and biomechanical studies have proven that there is the loss or breakage of fibrocartilage covering the mandibular condyle and temporal component in osteoarthritis.^{14,16,17} The disruption of collagen followed by loss of proteoglycans and glycosaminoglycans are one of the earliest molecular processes that take place in osteoarthritis.¹⁴ Because of loss of these molecules, the articular tissues began to lose resiliency to compressive and shearing forces during mandibular movements making the affected joint more exposed to structural damage from repetitive joint loading. The damaged articular tissues further induce advancement of the disease by stimulating inflammatory responses.¹⁸ Mild overloading of the joint may result to remodeling but excessive pressure on the TMJ could lead to degeneration of the fibrocartilage covering the condyle. The elastic capacity of the fibrocartilage is most often reduced causing increased stress to the underlying bone. As the degenerative process advances, osteoclastic activity may result to thinning and eventual cracking of the articular surface. This could be followed by the development of subarticular cystic areas in the medullary portion of the condyle. The bone may eventually become exposed causing the breakdown of the cyst and eventually leading to erosion and change in the shape of the condyle. Osteophytes generally form as a result of proliferation of the bones at the margins of the condyle.¹⁵

Two important radiographic hallmarks of degenerative joint disease are the presence of erosions and osteophytes.¹⁹ Erosion is an area of decreased bone density of the cortical

bone. It represents the early stage of degenerative changes suggesting that the TMJ is unstable and there is alteration of bony surfaces. Radiographically, it appears as a local area in the condyle showing decreased density of the cortical and adjacent subcortical bone. Osteophyte is described as marginal bony outgrowth of the condyle.²⁰ They usually appear in the later part of the degenerative changes when the body is adapting and trying to repair the joint.^{19,20}

In osteoarthritis, there is secondary inflammation of the synovium that causes pain. There are some patients that tend to experience inflammatory reaction to overloading of the TMJ. This may be part of an “arthritic event” which indicates that the inflammatory process is producing pain during remodeling.²¹

The chief complaint of the patient is usually pain that is constant and localized to the area. As a result of pain, there is also a limitation of mandibular movement and sometimes deflection to the ipsilateral side and more limited movement to the contralateral side. The painful osteoarthritic TMJ is often tender to palpation and the pain exacerbated by movements.²⁰ Pain is experienced everytime the joint is loaded. Patients may also experience myalgia and spasm of the masticatory muscle as the muscles try to guard against movement. Usually osteoarthritis has a gradual onset and self-limiting.¹⁵

Numerous imaging modalities claimed to be able to image osteophytes and erosions but there is no consensus on which imaging modality should be considered as the “gold standard” for detecting these TMJ bony changes.¹⁹ Ludlow et al reported that axially corrected sagittal tomography (ACST) can detect osteophytes better than panoramic in the TMJ.³ According to Honda, there was no significant difference in detecting erosions and osteophytes between CBCT and helical computed tomography (HCT).²²

Increasing morphologic radiographic changes was also observed with increasing age and for females.²³ Alexiou et al²⁰ reported that degenerative joint disease is an age-related disorder where the development and severity of osseous changes in mandibular condyle and glenoid fossa are increased with age. Patients in the older age group are anticipated to have more frequent and advanced degenerative osseous changes compared to the younger age group. The TMJ compared with other load bearing synovial joints has a very remarkable adaptive capacity. The articular surfaces of the TMJ are composed of fibrocartilages. There is also proof that the molecular events occurring in the TMJ that control the development and healing responses is different from those of other load bearing joints.²⁴ Because of the regenerative capacity of the TMJ in younger individuals, osteoarthritis is not as common as in older age groups.¹⁸

Epidemiologic studies proved that osteoarthritis has a female predisposition than males.^{25,26} Clinical investigations showed that there is an association between estrogens and development of temporomandibular disorders. It was shown that estrogens could increase vulnerability to TMJ osteoarthritis because of 2 mechanisms: (1) by altering the generation of matrix-degrading enzymes (as a response to hormonal stimulation) and (2) causing joint hypermobility leading to damaging biomechanics.¹⁸

PREVIOUS STUDIES CORRELATING TMJ OSTEOARTHRITIS WITH RADIOGRAPHIC FINDINGS

Previous studies attempting to correlate pain intensity levels with the quality of bony changes in TMJ osteoarthritis using different imaging modalities were equivocal. Prediction of radiographic findings is typically challenging because the association between signs and clinical presentation is not well-founded.^{23,27-29}

Using lateral tomography, Kurita et al found a significant relationship between pain on function with radiographic evidence of bony changes of the articular surface. Similarly they reported that condylar resorption observed from plain film radiographs were significantly associated with palpation pain.³⁰ This is consistent with the study of Hatcher et al showing that erosive condyle change was accompanied with severe symptoms.³¹ A significant correlation between the clinical finding of TMJ pain and MR imaging diagnosis of TMJ osteoarthritis was reported by Emshoff et al.³² However they also mentioned that clinical pain was not predictable of the presence of TMJ osteoarthritis or effusion.

Other studies reported varying results in their investigations. Poveda-Roda et al showed that while patients showing osteoarthritic clinical signs and symptoms did not have radiologic manifestations, a surprisingly high percentage of healthy subjects have radiologic affirmation of osteoarthritis.³³ The study by Yamada et al demonstrated that whereas some patients with erosive bony change had severe TMD symptoms such as TMJ pain and difficulty in mouth opening, others with erosive change of the condyle did not manifest clinical symptoms.³⁴ Because the results in their study showed that the presence of condylar changes were associated with higher prevalence of pain and also MRI findings of degenerative changes without pain, Campos et al suggested that MRI findings of osteoarthritis are not always important factors in the development of TMJ pain.³⁵

Some studies showed poor correlation between TMJ pain and radiologic bony changes.²⁷⁻²⁹ Weise et al reported in their study that none of the pain related variables were associated with radiographic changes.²³ This is in agreement with Sato et al's study where they concluded that there was a poor association between abnormal radiographic findings and TMD signs and symptoms in the elderly.³⁶ Ohlmann et al also reported that MRI-depicted

images of bony changes in the TMJ did not correlate with pain.³⁷ Similarly, Crow et al reported the presence of condylar osteophytic changes as well as mild and severe flattening without TMD symptoms.³⁸ Larheim's study also demonstrated that remodeling of bone may occur from mild flattening, osteophyte formations, sclerosis to extensive abnormalities without evidence of clinical symptoms.³⁹ These findings reveal that substantial inconsistency exists between findings by imaging versus the patient clinical symptoms creating a significant problem in clinical diagnosis and treatment.⁴⁰

A position paper released by the American Academy of Oral and Maxillofacial Radiology (AAOMR) in 1997 stated that while some patients with radiographically normal TMJs report clinical pain complaints, other patients with radiographic evidence of degenerative joint disease may not experience any pain.⁴¹ This radiographic evidence of degenerative joint disease may be due to remodeling that took place wherein the bony morphology became altered but the condition has stabilized.²⁰

PAIN

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage.⁴² The unpleasantness associated with pain pushes patients to seek relief. Mechanical, thermal or chemical (noxious) stimuli from trauma, infection or surgery cause varying degrees of tissue injury, thereby producing different pain intensity levels.⁴³

Pain is a multidimensional experience where there is a complex interaction of somatic, sensory, sympathetic and thalamocortical systems.⁴⁴ TMD patients usually complain of pain of musculoskeletal origin that is either localized or diffuse and aggravated by jaw function. Distinguishing TMD pains from other craniofacial pain conditions is quite difficult

because the TMJ is located in the head, which is the most highly innervated part of the human body.⁴⁵ For TMD patients, pain is not fully apprehended and may be caused by other conditions aside from the inflammatory reactions³⁹

Numerous studies on TMD patients have reported the fluctuating nature of pain reported over time and the influence of various environmental, psychological and psychosocial measures on self-reported pain.⁴⁶⁻⁴⁸ Acute pain is believed to be biologically useful because it alerts the individual to the presence of a possible noxious stimulus. Thus, the presence of acute pain signals an actual or potential tissue injury. On the other hand, the cause of chronic pain may not necessarily be biological and may not be cured.⁴⁹

Musculoskeletal pain is most often localized and characterized by deep ache and recurring in scattered and irregular episodes.⁵⁰ Visceral pain is a poorly localized pain coming from the internal organs often accompanied by autonomic reflexes.⁵¹ Cutaneous pain is a pain sensation arising from the skin.⁵²

HYPOTHESIS

This study aims to revisit the relationship between the bony changes in the TMJ and self-reported pain. We hypothesize that the quality of the bony changes seen in CBCT images is related to the patients' pain symptoms as reported by the verbal pain score and pain on palpation. To test this hypothesis, we reviewed and quantified the condylar changes using CBCT images and examined their relationship with clinical measures.

CHAPTER 2

MATERIALS AND METHODS

This study received approval from the Institutional Review Board of the University of North Carolina at Chapel Hill. CBCT images and clinical records of 30 consecutive patients with osteoarthritis of the TMJ who sought treatment at University of North Carolina Chapel Hill Orofacial Pain Clinic from January 2007 to August 2008 were reviewed in this study. Study inclusion included meeting the Research Diagnostic Criteria for TMD (RDC/TMD) Group IIIb - Osteoarthritis of the TMJ defined by the presence of arthralgia and either TMJ crepitations or CBCT bony changes including erosion, sclerosis, flattening of joint surfaces or osteophyte formation. Exclusion criteria included a history of TMJ surgery, condylar fracture, jaw trauma, and polyarthritis (such as rheumatoid arthritis, gout arthritis, psoriatic arthritis). Subjects with missing data were also excluded.

A detailed history taking and clinical assessment was performed on all subjects by the Orofacial Pain Specialist. Self-reported average pain intensity level was rated on a 0 to 10 Verbal Rating Scale where “0” was no pain and “10” was the worst pain possible. Clinical assessments included mandibular range of motion (maximum mouth opening, right and left lateral range of motion and protrusion), joint pain on palpation and on jaw functions, and the presence of crepitations.

The CBCT images were taken with Galileos (Sirona Dental Systems Inc., Bersheim, Germany) with voltage set at 85 kV and current at 7mA. The effective dose is approximately

70 uSv (ICRP 2007) and the field of view is 6 inches.⁵³ Reconstructed three-dimensional (3D) data were saved in a proprietary data format file and multiplanar images were exported in Digital Communications in Medicine (DICOM) format files.

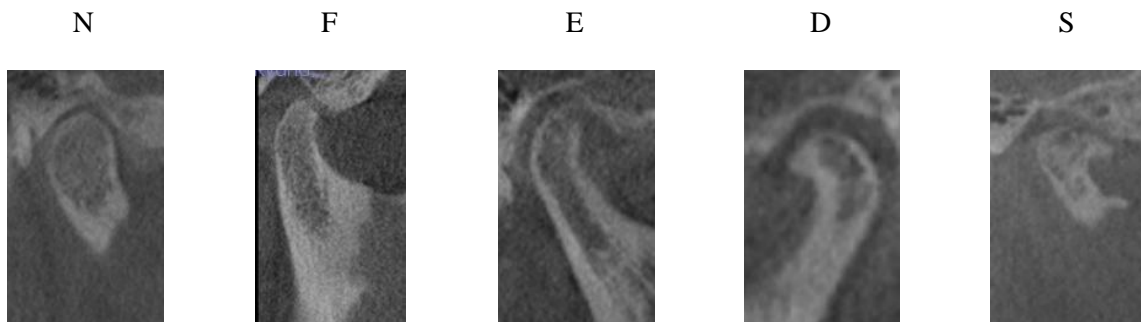
Invivo Dental (Anatomage, Inc. San Jose, CA, USA) software was used to view the DICOM volumes. Images were viewed in the axial, coronal and sagittal planes in the software's multiplanar reformatted view. Corrected axis cross sections of the joint were also viewed. All images were interpreted by 3 Oral and Maxillofacial Radiologists who have more than 20 years of experience and routinely interpret TMJ pathology from CBCT. Interpretations and illustrative sample views were recorded in the Electronic Patient Record (EPR).

The radiographic interpretations in the EPR were retrieved and reviewed by the PI. Additionally, using Invivo Dental software, each subjects' volume images were re-opened to visualize the findings reported in the EPR. A Lenovo (Lenovo, Morrisville, NC, USA) T60p monitor with 1024 X 768 resolution was used. Each subject's TMJ bony changes were eventually classified using Koyama's criteria.¹ If there was doubt on which classification should be assigned, the volume was revisited with the Radiologist who interpreted the image until an agreement was reached.

The criteria for determination of the type of condylar bony changes according to Koyama et al¹ were as follows: N (No proliferation or thickening on the cortical surface of the condyle displaying typical morphology or **normal**; F (Flattened contour at the anteroposterior and/or posterosuperior portions of the Condyle or **flattening**; E (Proliferation or partial hypodense change with or without roughening of the cortical surface of the condyle or **erosion**; D (condyle has a deformed contour, like a beak, without proliferation nor partial

hypodense change on the condylar surface or **deformity, marginal proliferation, osteophyte**) and **S** (Type D accompanied by Type E (**erosion, deformity, osteophyte, marginal proliferation**)). Koyama et al did not include the glenoid fossa changes in their classification.¹ This present study classified the bony changes of the glenoid fossa as “positive” in the presence of flattening, erosion, and/or sclerosis; or “negative” when the glenoid fossa appeared normal. Additionally, sclerosis was also not included in the Koyama classification of condylar bony changes.¹ According to Uemura, sclerosis was defined as hardening of trabecular bone.⁵⁴ Since there were few joints with bone change limited to trabecular bone, Koyama et al decided that including trabecular bone would complicate the classification of the condylar bony change.¹

Figure 1 Sample CBCT images of condylar bony changes according to Koyama’s classifications

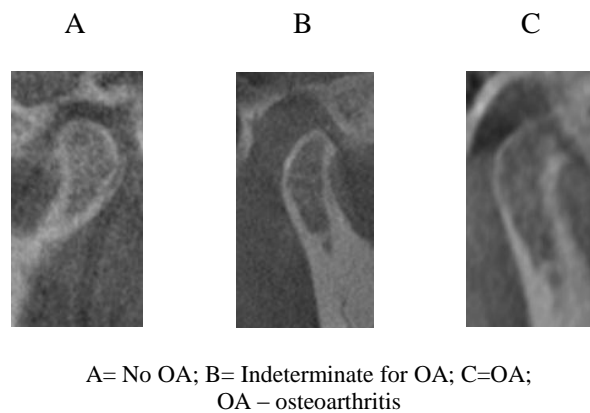


N.Normal; F.Flattening; E.Erosion; D.Deformity; Marginal proliferation, Osteophyte; S.Erosion, Deformity, Osteophyte, Marginal proliferation. Images were taken from subjects in this study.

The type of condylar bony change was also classified using the image analysis criteria developed recently by Ahmad et al² (Fig 2). These criteria include: **A** (No osteoarthritis – normal relative size of the condylar head, no subcortical sclerosis or surface flattening and no deformation due to subcortical cyst, surface erosion, osteophyte or

generalized sclerosis); **B** (Indeterminate for osteoarthritis - normal relative size of the condylar head, subcortical sclerosis with/without articular surface flattening or articular surface flattening with/without subcortical sclerosis and no deformation due to subcortical cyst, surface erosion, osteophytes or generalized sclerosis); **C** (Osteoarthritis - deformation due to subcortical cyst, surface erosion, osteophyte, or generalized sclerosis).

Figure 2. Sample images of Condylar bony changes according to Ahmad's classification



STATISTICAL ANALYSIS

All data were entered into Excel 2007 (Microsoft, Richmond, WA, USA). SAS version 9.1 (SAS Institute Inc., Cary, NC, USA) was used for statistical testing. Only the maximum bony changes of the condyle was used as covariate. The GLM (Generalized Linear Modeling) procedure analyzed the correlation between the maximum condyle changes with Verbal pain score and the four mandibular ranges of motion. The F statistic and the p-value obtained from it measured the part of variation in the dependent variable that was explained by the maximum bony changes. Spearman's rho correlation was used to correlate the average and maximum condyle changes for the Koyama and Ahmad classifications as well as the glenoid fossa changes.

CHAPTER 3

RESULTS

A total of 30 patients fulfilled the inclusion and exclusion criteria and their data were analyzed. The age of the patients ranged from 16 to 71 years (mean 41 years/ SD19.4). These patients reported pain rating scores ranging from 2 to 8 (mean 5.7/ SD1.8). Also, the clinical assessments of these patients showed mean range of motion in opening (46.7 mm/SD 10.1), protrusion (6.7 mm/SD 2.1), right lateral (8.5 mm/SD 2.1) and left lateral (9.0 mm/SD 2.4) motion.

Bilateral joint crepitations was infrequently detected clinically showing negative (24 patients/80%) and positive (6 patients/20%) results for the right TMJ. The left TMJ also showed almost similar results revealing negative (22 patients/73%) and positive (8 patients/27%) crepitations. Even when crepitation in either joint was considered, the data was too sparse for reliable computational use.

Due to the sparseness of the various clinical measures in the dataset, the only outcomes that were modeled were verbal pain score and the four mandibular range of motion measures. The maximum condyle change was used as explanatory variable.

Table 1. Univariate table showing the descriptive statistics of subjects

	N	Percent	Mean	Std Dev
Age	30		41.0*	19.4*
Gender				
Male	4	13		
Female	26	87		
Verbal pain rating score				
2	2	7		
3	3	10		
4	2	7		
5	4	13		
6	6	20		
7	10	33		
8	3	10		
Left lateral capsule palpation pain				
Negative	6	20		
Positive	24	80		
Right lateral capsule pain				
Negative	9	30		
Positive	21	70		
Left external auditory meatus palpation pain				
Negative	23	77		
Positive	67	23		
Right external auditory meatus palpation pain				
Negative	24	80		
Positive	6	20		
Pain on jaw use				
Negative	3	10		
Positive	27	90		
Right crepitation				
Negative	24	80		
Positive	6	20		
Left crepitation				
Negative	22	73		
Positive	8	27		
Opening ROM	30		46.7**	10.1**
Protrusion	28		6.7**	2.1**
Right lateral ROM	29		8.5**	2.1**
Left lateral ROM	29		9.0**	2.4**

ROM – range of motion; Std Dev – Standard deviation; * - years; ** - millimeters

Table 2. Table of descriptive statistics of Right and Left condyle changes with Koyama's & Ahmad's classification

Right Condyle					Left condyle			
N=30	Ahmad's Classification				Ahmad's Classification			
Koyama's classification	No OA (%)	IndetOA (%)	OA (%)	Total (%)	No OA (%)	IndetOA (%)	OA (%)	Total (%)
Normal	4 (13)	0 (0)	0 (0)	4 (13)	1 (3)	0 (0)	0 (0)	1 (3)
Flattening	0 (0)	5 (17)	0 (0)	5 (17)	2 (6)	11 (37)	0 (0)	13 (43)
Erosion	1 (3)	4 (13)	6 (20)	11 (37)	0 (0)	1 (3)	4 (13)	5 (17)
Deformity, osteophyte, marg prolif	0 (0)	0 (0)	4 (13)	4 (13)	0 (0)	0 (0)	5 (17)	5 (17)
Deformity, osteophyte, marg prolif, erosion	0 (0)	0 (0)	6 (20)	6 (20)	0 (0)	0 (0)	6 (20)	6 (20)
Total	5 (17)	9 (30)	16 (53)	30 (100)	3 (10)	12 (40)	15 (50)	30 (100)

OA – osteoarthritis; marg prolif – marginal proliferation; IndetOA – Indeterminate for osteoarthritis

Table 3. Table of descriptive statistics of Right and Left glenoid changes with Ahmad's classification

Right glenoid					Left glenoid			
N=30	Ahmad's Classification				Ahmad's Classification			
	No OA (%)	IndetOA (%)	OA (%)	Total (%)	No OA (%)	IndetOA (%)	OA (%)	Total (%)
Negative	21 (70)	2 (7)	0 (0)	23 (77)	21 (70)	0 (0)	1 (3)	22 (73)
Positive	1 (3)	4 (13)	2 (7)	7 (23)	1 (3)	2 (7)	5 (17)	8 (27)
Total	22 (73)	6 (20)	2 (7)	30 (100)	22 (73)	2 (7)	6 (20)	30 (100)

OA - Osteoarthritis; IndetOA – Indeterminate for osteoarthritis

The relationships between maximum condylar bony change with verbal pain score, ranges of motion in maximum opening, protrusion, right and left lateral range of motion are shown in Table 4. There was a poor correlation between maximum condyle bony change and Verbal pain score producing a $p=0.3995$ (Koyama) and $p=0.9490$ (Ahmad). Also, there was

no statistically significant relationship between maximum condyle changes and opening range of motion yielding $p=0.0629$ (Koyama) and $p=0.0951$ (Ahmad). With protrusion, the relation was also not statistically significant with $p=0.7001$ (Koyama) and $p=0.3612$ (Ahmad) respectively.

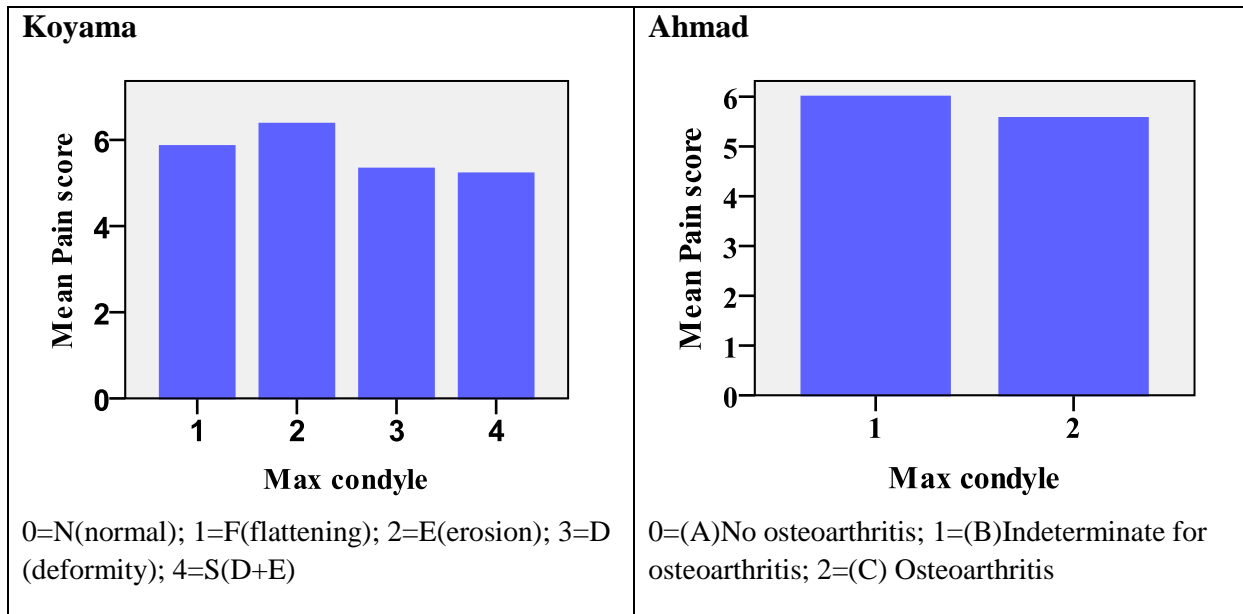
The maximum condyle bony change was not significantly correlated with right lateral range of motion resulting in a $p=0.9093$ (Koyama) and $p=0.6877$ (Ahmad). With left lateral range of motion, it resulted in a $p=0.6494$ (Koyama) and $p=0.3236$ (Ahmad). (Fig. 3 and 4).

Table 4. Maximum bony change of the right and left condyle analyzed with verbal pain score and ranges of motion

	Koyama p-value	F-value	Ahmad p-value	F-value
Verbal painscore	0.3995	1.05	0.9490	0.18
Opening	0.0629	2.57	0.0951	2.23
Protrusion	0.7001	0.55	0.3612	1.14
R lateral ROM	0.9093	0.25	0.6877	0.57
L lateral ROM	0.6494	0.62	0.3236	1.23

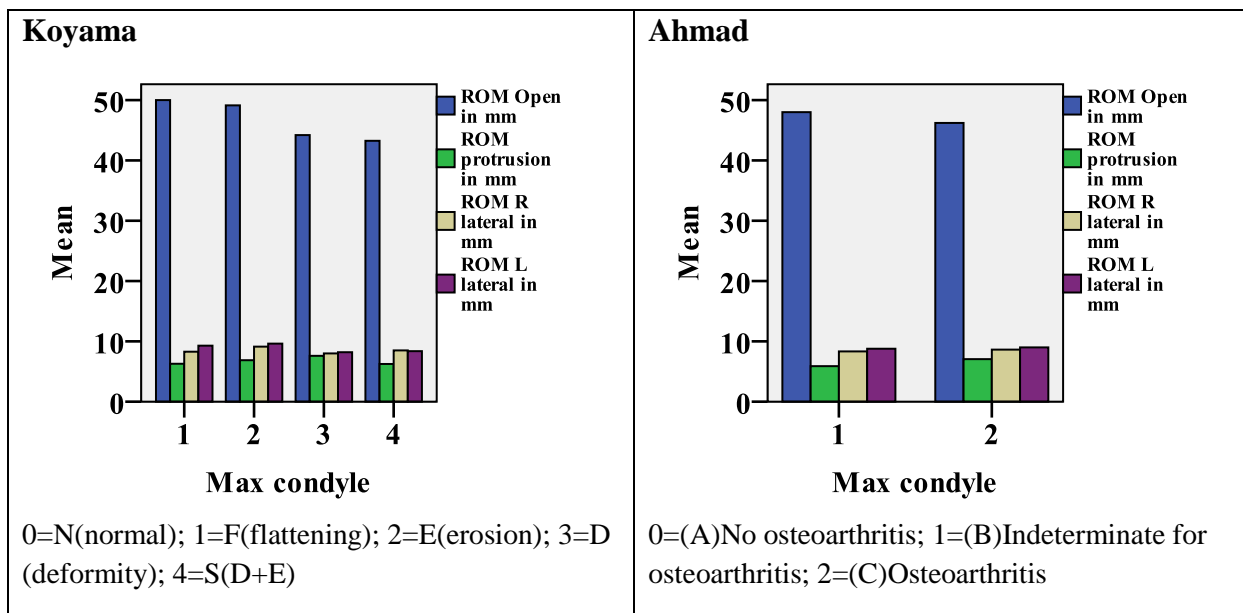
There was no statistically significant relationship between maximum condylar bony changes and verbal pain score producing $p=0.3995$ (Koyama) and $p=0.9490$ (Ahmad); opening $p=0.0629$ (Koyama) and $p=0.0951$ (Ahmad); protrusion $p=0.7001$ (Koyama) and $p=0.3612$ (Ahmad); right lateral ROM $p=0.9093$ (Koyama) and $p=0.6877$ (Ahmad); left lateral ROM $p=0.6494$ (Koyama) and $p=0.3236$ (Ahmad); ROM – range of motion; R lateral– right lateral; L lateral – left lateral. GLM (Generalized linear modeling) procedure was used.

Figure 3. Verbal pain score correlated with maximum condyle change (Koyama and Ahmad)



There was no statistically significant correlation between the verbal pain score and maximum condyle change (Koyama and Ahmad). Max condyle – maximum condyle

Figure 4. Ranges of motion correlated with maximum condyle change (Koyama and Ahmad)



There was no statistically significant correlation between the ranges of motion in mm (opening, protrusion, right lateral, left lateral) and maximum condyle change (Koyama and Ahmad). Max condyle – maximum condyle; ROM – range of motion; R lateral – right lateral; L lateral – left lateral; Open – opening; mm - millimeter

The relationship between Koyama's and Ahmad's classification is shown in Table 5. There was a high correlation between the average and maximum condyle changes ($p < .0001$). Similarly, the average and maximum glenoid fossa changes also showed statistically significant correlation between Ahmad's classification and the positive/negative responses ($p < .0001$) that was used in this study.

Table 5. Table correlating the average and maximum changes for condyle and glenoid fossa

N=30	KOYAMA							
Ahmad	Ave condyle	p-value	Max condyle	p-value	Ave glenoid	p-value	Max Glenoid	p-value
Ave condyle	0.9341**	<.0001						
Max condyle			0.7694**	<.0001				
Ave Glenoid					0.8308**	<.0001		
Max Glenoid							0.7754**	<.0001

There was statistically significant correlations between the average and maximum changes of condyle and glenoid fossa. Ave condyle – average condyle; Max condyle – maximum condyle; **Spearman's correlation coefficient

DATA ANALYSIS FOR RELIABILITY STUDIES

To determine interexaminer reliability in assigning a classification, a set of 9 subjects were randomly selected and their radiologic interpretations were reviewed by a second observer (Senior Radiology resident). The volume images were also re-opened under standardized conditions to visualize the findings in the EPR. The observer was given a visual instruction sheet with sample images of bony changes to serve as a guide in designating a classification.

To establish intraexaminer reliability, another set of 9 subjects were randomly selected 2 weeks after the initial review. The radiologic reports were reviewed again and the

volume images were re-opened under the same standardized conditions to envision the findings in the radiographic interpretations. The observers then designated a classification using Koyama's & Ahmad's criteria.

The kappa values were computed as a measure of intra and interobserver variability (Table 6). It was interpreted based on the criteria by Landis and Koch (1977): 0.81-1.00 (almost perfect), 0.61-0.80 (substantial), 0.41-0.60 (moderate), 0.21-0.40 (Fair) and 0.01-0.20 (slight), <0.00 (poor) agreement.⁵⁵

Table 6. Intraobserver and Interobserver analysis

Measured Variable	Intra-rater Analysis		Inter-rater analysis	
	Kappa Coefficient	P-value	Kappa Coefficient	P-value
Right Condyle	0.8205	<.0001	0.8170	<.0001
Left Condyle	0.8235	<.0005	0.7724	<.0001
Right Glen Fossa	1.0000	<.0001	1.0000	<.0001
Left Glen Fossa	0.7273	<.0233	1.0000	<.0001

Right Glen Fossa – right glenoid fossa; Left Glen Fossa – left glenoid fossa; Level of significance $p < .05$

As seen in the tables above (Table 6), strong inter-rater and intra-rater agreement was observed on all variables. Perfect agreement was obtained for inter-rater analysis on right and left glenoid fossa and intra-rater analysis on right glenoid fossa.

CHAPTER 4

DISCUSSION

The results in this study where the radiographic findings did not find significant correlation with clinical findings is consistent with previous studies.^{10,23,27-29,36-39} In the present study, CBCT was used to examine the TMJ. Previous studies have proven its usefulness in depicting bony changes accurately while imparting a relatively low radiation dose to the patient.^{4,20,22,53,56,57} Similarly, Honda et al concluded in their study that CBCT is helpful for TMJ diagnosis because of its accuracy of measurements and lower radiation dose compared with helical CT.⁵⁸

From the images in this study, it was possible to see a clear delineation of the joint in 3 dimensions without the problem of superimposition. Nevertheless, this study found no significant relationship between maximum bony changes with self-reported pain and ranges of motion. We proposed that one of the reasons for the lack of correlation was related to the multidimensional experience of pain. Besides, patients are unable to distinguish masticatory muscle pain from pain of osteoarthritic origin. Localized muscle pain often accompany TMJ inflammation. Patients may complain of tenderness to palpation that may be equal or greater than the degree of pain in the joint.⁵⁹

The study by Weise et al²³ did not find any association between degenerative bony changes in TMJ tomograms and any pain-related variables such as muscle and TMJ pain on palpation, duration of pain somatization scores, and graded chronic pain, including disability points and characteristic pain intensity. Also there was no significant association with

depression score, jaw disability score, and presence or absence of arthritic disease. They explained that this non-association may be due to the differences in the onset of pain and detectable radiographic bony changes because radiographs do not depict ongoing process but the effect of a previous process.

In this present study, the relation of maximum condyle change with verbal pain rating score and ranges of motion was evaluated. The result showing a very poor correlation strongly suggests that pain reported may be due to other causes other than temporomandibular joint pain. Other conditions such as synovitis or inflammation of the capsular tissues may be possible causes of joint pain. Degenerative changes that are not evident on radiographs are also significant factors in joint pain.³⁰

Chronic psychological stress may add to symptoms of TMJ osteoarthritis.¹⁸ An increased incidence of parafunctional habits like clenching and bruxism that increase joint loading may increase the pain associated with TMJ osteoarthritis.¹⁸ Parafunctional activity most often causes sustained muscle contraction for long periods of time. This isometric activity decreases blood flow within the muscle tissue resulting to accumulation of metabolic by-products that lead to symptoms of fatigue, pain, and spasm.¹⁰ There are at least 2 mechanisms in which mechanical stimuli can activate molecular events that could result to osteoarthritis. Highly responsive molecules known as free radicals that can damage essential molecules of the articular tissues as well as synovial fluid can stimulate cellular responses that could induce degenerative joint disease as a result of mechanical load.^{18,60} Also, sensory neurons are stimulated causing the release of neuropeptides and other molecules like nitric oxide that could lead to osteoarthritis by starting neurogenic inflammation.⁶¹ The release of potent biochemicals like nerve growth factor (NGF) may add to the production of pain. There

is also the ongoing activation of the hypothalamic pituitary axis (HPA) that lead to complex systemic effects that is essential in the development of TMD and associated sequelae.⁴⁹

Stress has been regarded as an important factor in the onset of TMD.⁶² It does not cause pain directly but may indirectly exacerbate pain and lessen an individual's tolerance to pain.⁶³ It is worth mentioning that measurement of stress is challenging because what may be minor stress for one patient may be regarded as major stress to another. The study by Filho et al concluded that the absence of stress is a strong factor for non-development of TMD.⁶²

Patients may experience symptoms for as long as 6 months before bony changes can be seen radiographically. This explains why in early stages of osteoarthritis, radiographs may appear normal and may not be helpful in validating the diagnosis.⁸ Radiographic changes such as flattening, osteophytes, cystic formation and decreased articular space typically appear in the later stages of the disease.¹⁵ For osteoarthritic patients, clinical symptoms may be present without radiographic degenerative changes because of the difference in the appearance of pain and development of degenerative changes that may be visible on radiographs.⁶⁴

Some joints that show radiographic evidence of DJD may be due to remodeling that took place but where the condition has already stabilized. When the radiographic images confirm the structural changes in the subarticular bone but the patient no longer has pain symptoms the condition may be termed **osteoarthrosis**.⁸ The degenerative course seems to burn-out within a 3-year period so that the inflammatory process has subsided, pain has gradually diminished, adequate range of motion is restored and there is decrease in joint sounds but the remodeling that has taken place in the condyle and fossa remains.⁶⁴ Osteoarthrosis is a non-inflammatory condition representing a subacute or chronic phase

where there is an inflammatory component identified in fluid and tissue samples but the TMJ does not appear clinically inflamed.¹⁸ In osteoarthritis, there is usually the absence of pain and no point tenderness to palpation. Additionally, limitations in mandibular motion as well as crepitus, are often present. This is more obvious during the later part of the disease. Imaging often disclose indications of structural changes in the subarticular bone of the condyle that substantiate a diagnosis. These radiographic changes may not be manifested in the early part of the degenerative process.¹⁵

We used the 2 classification systems in this study in order to verify that the results can be duplicated by the other classification system. Both Koyama's and Ahmad's criteria yielded no correlation between maximum condyle changes and verbal pain rating score as well as ranges of motions. This study has shown that results will not be very different no matter what classification scheme was used. It is noteworthy that both classification systems when correlated for average and maximum bony changes yielded very significant correlations.

The study by Koyama et al was aimed at establishing criteria for evaluation of condylar bony change using Helical CT¹. Likewise Ahmad et al developed the RDC/TMD² image analysis criteria, a comprehensive classification system for assessing osteoarthritic changes using CT. Both Koyama and Ahmad recommended that to reduce radiation dose, a dental CT or CBCT can be used which can provide diagnostic input similar to multidetector CT^{1,2}.

The criteria developed by Koyama et al and Ahmad et al will be useful for research purposes as well as TMJ assessments by TMD clinicians. Clinicians generally order

radiographs to either support their clinical expectations and to rule out alternative differentials if there is some uncertainty from the clinical presentation.⁶⁶

The Koyama classification¹ consists of 5 criteria. It was released in 2007 and was aimed at establishing criteria for evaluation of condylar bony change. Definite criteria for osseous change of the TMJ using multiplanar reconstructed (MPR) images have not yet been established when this classification was released.

The RDC/TMD comprise of an acknowledged diagnostic system for the diagnosis and treatment of TMD.⁶⁷⁻⁶⁹ It is the most universally used TMD diagnostic system for clinical studies and it also permit multiple comparison of clinical findings.⁷⁰ Because of the increasing demand for the use of CT and MRI, it was essential to develop an encompassing criteria for image analysis using these modalities as part of the RDC/TMD. In order to complement the operational requirements of the RDC/TMD, this classification system was developed for acquiring and analyzing panoramic, MRI and CT images to evaluate the TMJ.² The classification developed by Ahmad et al was released in 2009.

A limitation of this study is its retrospective nature. We could not design the study to get measures other than those included in the patient's dental/medical record. Future prospective studies should utilize multidimensional instruments to measure pain including the cognitive, motivational and evaluative components instead of just the sensory discriminative aspect.⁷¹ Prospective cohorts may capture clinically relevant variables missing from retrospective data sets such as pain intensity levels at different occasions throughout the course of the study instead of a generalized pain reporting during the initial clinical examination.

CHAPTER 5

CONCLUSION

While the results of this study showed a high correlation between Koyama's & Ahmad's classification on Average and Maximum condylar bony changes in TMJ osteoarthritis, both classification schemes did not correlate pain intensity and mandibular ranges of motion with maximum condylar bony change. The findings of this study do not support the use of radiographs alone for diagnosis and treatment planning. Factors mitigating this lack of correlation warrants further investigation.

REFERENCES

1. Koyama J, Nishiyama H, Hayashi T. Follow-up study of condylar bony changes using helical computed tomography in patients with temporomandibular disorder. *Dentomaxillofac Radiol.* 2007; 36:472-477.
2. Ahmad M, Hollender L, Anderson Q, Kartha K, Ohrbach R, Truelove EL, et al. Research diagnostic criteria for temporomandibular disorders (RDC/TMD): development of image analysis criteria and examiner reliability for image analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2009; 107:844-860.
3. Ludlow JB, Davies KL, Tyndall DA. Temporomandibular joint imaging: a comparative study of diagnostic accuracy for the detection of bone change with biplanar multidirectional tomography and panoramic images. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1995; 80:735-743.
4. Tsiklakis K, Syriopoulos K, Stamatakis HC. Radiographic examination of the temporomandibular joint using cone beam computed tomography. *Dentomaxillofac Radiol.* 2004; 33:196-201.
5. Westesson PL, Katzberg RW, Tallents RH, Sanchez-Woodworth RE, Svensson SA. CT and MR of the temporomandibular joint: comparison with autopsy specimens. *Am J Roentgenol.* 1987; 148:1165-1171.
6. Mischkowski RA, Scherer P, Ritter L, Neugebauer J, Keeve E, Zöller JE. Diagnostic quality of multiplanar reformations obtained with a newly developed cone beam device for maxillofacial imaging. *Dentomaxillofac Radiol.* 2008; 37:1-9.
7. Hintze H, Wiese M, Wenzel A. Cone beam CT and conventional tomography for the detection of morphological temporomandibular joint changes. *Dentomaxillofac Radiol.* 2007; 36:192-197.
8. Okeson JP. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management.* Chicago, IL: Quintessence Publishing Co, 1996.
9. Wright EF. *Manual of Temporomandibular Disorders.* Ames, IA: Wiley-Blackwell, 2005.
10. Okeson JP. *Management of Temporomandibular Disorders and Occlusion.* 5th ed. New York, NY: Mosby, 2002.
11. Stegenga B, de Bont LG, Boering G. Osteoarthritis as the cause of craniomandibular pain and dysfunction: a unifying concept. *J. Oral Maxillofac. Surg.* 1989; 47:249-256.
12. Stegenga B, de Bont LG, Boering G, van Willigen JD. Tissue responses to degenerative changes in the temporomandibular joint: a review. *J. Oral Maxillofac. Surg.* 1991;

49:1079-1088.

13. de Bont LG, Stegenga B. Pathology of temporomandibular joint internal derangement and osteoarthritis. *Int J Oral Maxillofac Surg.* 1993; 22:71-74.
14. Israel HA, Saed-Nejad F, Ratcliffe A. Early diagnosis of osteoarthritis of the temporomandibular joint: correlation between arthroscopic diagnosis and keratan sulfate levels in the synovial fluid. *J. Oral Maxillofac. Surg.* 1991; 49:708-711; discussion 712.
15. Pertes, R and Gross, S. Disorders of the temporomandibular joints. In: *Clinical Management of Temporomandibular Disorders and Orofacial Pain.* Carol Stream, IL: Quintessence Publishing Co, 1995. pp 69-89.
16. Ma XC, Zou ZJ, Zhang ZK, Wu QG. Radiographic, pathological and operative observations of cases with TMJ disturbance syndrome. *Int J Oral Surg.* 1983; 12:299-308.
17. Haskin CL, Milam SB, Cameron IL. Pathogenesis of degenerative joint disease in the human temporomandibular joint. *Crit. Rev. Oral Biol. Med.* 1995; 6:248-277.
18. Milam SB. TMJ osteoarthritis. In: *Temporomandibular Disorders: An Evidenced-Based Approach to Diagnosis And Treatment.* Hanover Park, IL: Quintessence Publishing Co, 2006. pp 105-123.
19. Hussain AM, Packota G, Major PW, Flores-Mir C. Role of different imaging modalities in assessment of temporomandibular joint erosions and osteophytes: a systematic review. *Dentomaxillofac Radiol.* 2008; 37:63-71.
20. Alexiou K, Stamatakis H, Tsiklakis K. Evaluation of the severity of temporomandibular joint osteoarthritic changes related to age using cone beam computed tomography. *Dentomaxillofac Radiol.* 2009; 38:141-147.
21. Nickerson JW, Boering G. Natural course of osteoarthritis as it relates to internal derangement of the temporomandibular joint. *Oral Maxillofac Surg Clin North Am.* 1989; 1:27-45.
22. Honda K, Larheim TA, Maruhashi K, Matsumoto K, Iwai K. Osseous abnormalities of the mandibular condyle: diagnostic reliability of cone beam computed tomography compared with helical computed tomography based on an autopsy material. *Dentomaxillofac Radiol.* 2006; 35:152-157.
23. Wiese M, Svensson P, Bakke M, List T, Hintze H, Petersson A, et al. Association between temporomandibular joint symptoms, signs, and clinical diagnosis using the RDC/TMD and radiographic findings in temporomandibular joint tomograms. *J Orofac Pain.* 2008; 22:239-251.

24. Luyten FP. A scientific basis for the biologic regeneration of synovial joints. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997; 83:167-169.
25. Milam SB. Pathophysiology and epidemiology of TMJ. *J Musculoskelet Neuronal Interact.* 2003; 3:382-390; discussion 406-407.
26. LeResche L, Saunders K, Von Korff MR, Barlow W, Dworkin SF. Use of exogenous hormones and risk of temporomandibular disorder pain. *Pain.* 1997; 69:153-160.
27. Kopp S, Rockler B. Relationship between clinical and radiographic findings in patients with mandibular pain or dysfunction. *Acta Radiol Diagn.* 1979; 20:465-477.
28. Madsen B. Normal variations in anatomy, condylar movements, and arthrosis frequency of the temporomandibular joints. *Acta Radiol Diagn.* 1966; 4:273-288.
29. Hansson LG, Hansson T, Petersson A. A comparison between clinical and radiologic findings in 259 temporomandibular joint patients. *J Prosthet Dent.* 1983; 50:89-94.
30. Kurita H, Kojima Y, Nakatsuka A, Koike T, Kobayashi H, Kurashina K. Relationship between temporomandibular joint (TMJ)-related pain and morphological changes of the TMJ condyle in patients with temporomandibular disorders. *Dentomaxillofac Radiol.* 2004; 33:329-333.
31. Hatcher DC, McEvoy SP, Mah RT, Faulkner MG. Distribution of local and general stresses in the stomatognathic system. In: McNeil C (ed). *Science and practice of occlusion.* Chicago, IL: Quintessence Publishing Co, 1997. pp 259.
32. Emshoff R, Brandimaier I, Bertram S, Rudisch A. Magnetic resonance imaging findings of osteoarthritis and effusion in patients with unilateral temporomandibular joint pain. *Int J Oral Maxillofac Surg.* 2002; 31:598-602.
33. Poveda-Roda R, Bagán JV, Jiménez-Soriano Y, Fons-Font A. Retrospective study of a series of 850 patients with temporomandibular dysfunction (TMD). Clinical and radiological findings. *Med Oral Patol Oral Cir Bucal.* 2009; 14:e628-634.
34. Yamada K, Saito I, Hanada K, Hayashi T. Observation of three cases of temporomandibular joint osteoarthritis and mandibular morphology during adolescence using helical CT. *J Oral Rehabil.* 2004; 31:298-305.
35. Campos MIG, Campos PSF, Cangussu MCT, Guimarães RC, Line SRP. Analysis of magnetic resonance imaging characteristics and pain in temporomandibular joints with and without degenerative changes of the condyle. *Int J Oral Maxillofac Surg.* 2008; 37:529-534.
36. Sato H, Osterberg T, Ahlqvist M, Carlsson GE, Gröndahl HG, Rubinstein B. Association between radiographic findings in the mandibular condyle and temporomandibular

- dysfunction in an elderly population. *Acta Odontol. Scand.* 1996; 54:384-390.
37. Ohlmann B, Rammelsberg P, Henschel V, Kress B, Gabbert O, Schmitter M. Prediction of TMJ arthralgia according to clinical diagnosis and MRI findings. *Int J Prosthodont.* 2006; 19:333-338.
 38. Crow HC, Parks E, Campbell JH, Stucki DS, Daggy J. The utility of panoramic radiography in temporomandibular joint assessment. *Dentomaxillofac Radiol.* 2005; 34:91-95.
 39. Larheim TA. Current trends in temporomandibular joint imaging. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1995; 80:555-576.
 40. Sano T, Otonari-Yamamoto M, Otonari T, Yajima A. Osseous abnormalities related to the temporomandibular joint. *Semin. Ultrasound CT MR.* 2007; 28:213-221.
 41. Brooks SL, Brand JW, Gibbs SJ, Hollender L, Lurie AG, Omnell KA, et al. Imaging of the temporomandibular joint: a position paper of the American Academy of Oral and Maxillofacial Radiology. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997; 83:609-618.
 42. Loeser JD, Treede R. The Kyoto protocol of IASP Basic Pain Terminology. *Pain.* 2008 31; 137:473-477.
 43. Merskey H, Bogduk N. Classification of chronic pain: description of chronic pain syndromes and definition of pain terms. In: Task force on taxonomy of the International Association for the Study of Pain: classification of chronic pain. Description of pain syndromes and definitions of pain terms. Seattle: IASP Press;1994. XI, p. 210.
 44. Graff-Radford SB. Orofacial pain of neurogenous origin. In: Clinical management of temporomandibular disorders and orofacial pain. Carol Stream, IL: Quintessence Publishing Co, 1995. pp 329-341.
 45. Greene CS. The Role of technology in TMD diagnosis. In: Temporomandibular Disorders: An Evidenced-Based Approach to Diagnosis And Treatment. Hanover Park, IL: Quintessence Publishing Co, 2006. p. 193-202.
 46. van Selms MKA, Lobbezoo F, Naeije M. Time courses of myofascial temporomandibular disorder complaints during a 12-month follow-up period. *J Orofac Pain.* 2009; 23:345-352.
 47. Turner JA, Brister H, Huggins K, Mancl L, Aaron LA, Truelove EL. Catastrophizing is associated with clinical examination findings, activity interference, and health care use among patients with temporomandibular disorders. *J Orofac Pain.* 2005; 19:291-300.
 48. Weingarten TN, Iverson BC, Shi Y, Schroeder DR, Warner DO, Reid KI. Impact of

tobacco use on the symptoms of painful temporomandibular joint disorders. *Pain*. 2009 15; 147:67-71.

49. Grzesiak R and Ciccone D. Understanding the Pain patient. In: *Clinical Management of Temporomandibular Disorders and Orofacial Pain*. Carol Stream, IL: Quintessence Publishing Co, 1995, pp 45-57.
50. Dunn J and Mannheimer J. The Cervical Spine. In: *Clinical Management of Temporomandibular Disorders and Orofacial Pain*. Carol Stream, IL: Quintessence Publishing Co, 1995. pp 13-33.
51. Cervero F, Laird JM. Visceral pain. *Lancet*. 1999. 353:2145-2148.
52. Weissman-Fogel I, Sprecher E, Granovsky Y, Yarnitsky D. Repeated noxious stimulation of the skin enhances pain perception of migraine patients in between attacks: clinical evidence for continuous subthreshold increase in membrane excitability of central trigeminovascular neurons. *Pain*. 2003. 104:693-700.
53. Ludlow JB, Ivanovic M. Comparative dosimetry of dental CBCT devices and 64-slice CT for oral and maxillofacial radiology. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2008; 106:106-114.
54. Uemura S, Nakamura M, Iwasaki H, Fuchimata H. A roentgenological study on temporomandibular joint disorders. Morphological changes of TMJ in arthrosis. *Dent Radiol*. 1979; 19:224-237.
55. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics*. 1977; 33:159-174.
56. Ludlow JB, Davies-Ludlow LE, Brooks SL. Dosimetry of two extraoral direct digital imaging devices: NewTom cone beam CT and Orthophos Plus DS panoramic unit. *Dentomaxillofac Radiol*. 2003; 32:229-234.
57. Tsiklakis K, Donta C, Gavala S, Karayianni K, Kamenopoulou V, Hourdakakis CJ. Dose reduction in maxillofacial imaging using low dose cone beam CT. *Eur J Radiol*. 2005; 56:413-417.
58. Honda K, Arai Y, Kashima M, Takano Y, Sawada K, Ejima K, et al. Evaluation of the usefulness of the limited cone-beam CT (3DX) in the assessment of the thickness of the roof of the glenoid fossa of the temporomandibular joint. *Dentomaxillofac Radiol*. 2004; 33:391-395.
59. Clark GT. Treatment of Myogenous Pain and Dysfunction. In: *Temporomandibular Disorders: An Evidenced-Based Approach to Diagnosis And Treatment*. Hanover Park, IL: Quintessence Publishing Co, 2006. pp 483-500.

60. Fukuoka Y, Hagihara M, Nagatsu T, Kaneda T. The relationship between collagen metabolism and temporomandibular joint osteoarthritis in mice. *J. Oral Maxillofac. Surg.* 1993; 51:288-291.
61. Milam SB, Schmitz JP. Molecular biology of temporomandibular joint disorders: proposed mechanisms of disease. *J. Oral Maxillofac. Surg.* 1995; 53:1448-1454.
62. Filho J, Manzi FR, de Freitas DQ, Bóscolo FN, de Almeida SM. Evaluation of temporomandibular joint in stress-free patients. *Dentomaxillofac Radiol.* 2007; 36:336-340.
63. Hathaway K. Behavioral and Psychosocial Management. In: *Clinical management of temporomandibular disorders and orofacial pain*. Carol Stream, IL: Quintessence Publishing Co, 1995. pp 245-252.
64. Wiese M, Wenzel A, Hintze H, Petersson A, Knutsson K, Bakke M, et al. Osseous changes and condyle position in TMJ tomograms: impact of RDC/TMD clinical diagnoses on agreement between expected and actual findings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2008; 106:e52-63.
65. Kaplan AS, Buchbinder D. Arthritis. In: *Temporomandibular disorders: diagnosis and treatment*. Philadelphia, PA: WB Saunders, 1991. pp 165-89.
66. Pullinger AG, White SC. Efficacy of TMJ radiographs in terms of expected versus actual findings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1995; 79:367-374.
67. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord.* 1992; 6:301-355.
68. John MT, Dworkin SF, Mancl LA. Reliability of clinical temporomandibular disorder diagnoses. *Pain.* 2005; 118:61-69.
69. Dworkin SF, Sherman J, Mancl L, Ohrbach R, LeResche L, Truelove E. Reliability, validity, and clinical utility of the research diagnostic criteria for Temporomandibular Disorders Axis II Scales: depression, non-specific physical symptoms, and graded chronic pain. *J Orofac Pain.* 2002; 16:207-220.
70. List T, Dworkin SF. Comparing TMD diagnoses and clinical findings at Swedish and US TMD centers using research diagnostic criteria for temporomandibular disorders. *J Orofac Pain.* 1996; 10:240-253.
71. Jamison R, Rudy T, Penzien D, Mosley T. Cognitive-behavioral classifications of chronic pain: replication and extension of empirically derived patient profile. *Pain.* 1994; 57:277-292.