CLINICAL CORRELATION OF OSSEOUS CHANGES IN CT FOR PATIENTS WITH TEMPOROMANDIBULAR JOINT DISORDERS – A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that this dissertation titled **"CLINICAL CORRELATION OF OSSEOUS CHANGES IN CT FOR PATIENTS WITH TEMPOROMANDIBULAR JOINT DISORDERS – A PROSPECTIVE STUDY"** is a bonafide record of work done by **Dr. A. E. Malarvizhi** under my guidance during her postgraduate study period **2010-2013.**

This dissertation is submitted to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,** in partial fulfillment for the degree of **MASTER OF DENTAL SURGERY, BRANCH IX – Oral Medicine & Radiology.**

It has not been submitted (partial or full) for the award of any other degree or diploma.

Guided by:

RAGAS DENTAL COLLEGE & HOSPITAL CHENNAI

S. Manojkumar

Dr. (Capt) S. Manojkumar, M.D.S.,
Professor
Department of Oral Medicine & Radiology
Ragas Dental College & Hospital
Chennai – 600 119.

Dr.S.MANOJ KUMAR, M.D.S.,
PROFESSOR
DEPT.OF ORAL MEDICINE & RADIOLOGY
RAGAS DENTAL COLLEGE & HOSPITAL
2/102, EAST COAST ROAD,
UTHANDI, CHENNAI - 600 119.

S. Kailasam
24/09/12

Dr. S. Kailasam, B.Sc., M.D.S.,
Professor & Head
Department of Oral Medicine & Radiology
Ragas Dental College & Hospital
Chennai – 600 119.

Dr. S. KAILASAM, B.Sc., M.D.S.,
PROFESSOR & HEAD
DEPT. OF ORAL MEDICINE & RADIOLOGY
RAGAS DENTAL COLLEGE & HOSPITAL
2/102, East Coast Road,
Uthandi, Chennai - 600 119

Dr. S. Ramachandran, M.D.S.,
Principal,
Ragas Dental College & Hospital
Chennai – 600 119.

PRINCIPAL
RAGAS DENTAL COLLEGE & HOSPITAL
CHENNAI

Date: 21/12/12

Place: Chennai-600 119.

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LIST OF ABBREVIATIONS

S.NO	ABBREVIATION	EXPANSION
1.	AELT	Articular Eminence In Left TMJ
2.	AERT	Articular Eminence In Right TMJ
3.	CCLT	Condylar changes In Left TMJ
4.	CCRT	Condylar changes In Right TMJ
5.	CT	Computed Tomography
6.	GTLT	Glenoid Thickness – Left Side
7.	GTRT	Glenoid Thickness – Right Side
8.	MRI	Magnetic Resonance Imaging
9.	TMD	Temperomandibular Joint Disorder
10.	TMJ	Temperomandibular Joint

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ABSTRACT

Background: Temporomandibular joint disorder, as suggested by Bell, which constitutes joint and masticatory system has heterogenous collection of signs and symptoms. Multiple imaging avenues are available to evaluate TMJ disorders. It is extremely useful for revealing the skeletal abnormalities of the TMJ and has excellent predictive value.

Aim: The aim of this study is to correlate the clinical characteristics of temporomandibular joint (TMJ) disorder with osseous changes in CT.

Methodology: A Prospective analytical study was conducted among 15 patients with symptomatic temporomandibular joint disorders. Patients were clinically examined and parameters were recorded. CT images were taken and interpreted for articular eminence morphology, condylar changes and glenoid thickness. Clinical parameters were then correlated with CT findings. Data was analysed using SPSS software.

Results: Sigmoid was the most prevalent articular eminence morphology on both right (80.0%) and left (60.0%) TMJ. Normal condyle was predominantly found in both TMJ (80.0%) followed by flattened condyle (20.0%). The glenoid fossa thickness showed variation of 0.1 and 0.2 mm respectively in the right and left side between normal and flattened condyle.

Conclusion: Though we have correlated numerous clinical and radiographic features we couldn't staunchly correlate osseous changes in CT for patients with temporomandibular joint disorders which may be due to smaller sample size.

Key words: Temporomandibular joint disorders, CT, Articular eminence morphology, Condylar changes, Glenoid fossa thickness

The TMJ articulation is classified as *ginglymodiarthrodial joint*, namely a joint that is capable of hinge type movements and gliding movements, with the bony components enclosed and connected by a fibrous capsule. The mandibular condyle forms the lower part of the bony joint and is generally elliptical. The articulation is formed by the mandibular condyle occupying a hollow in the temporal bone (mandibular or glenoid fossa) [1].

TMJ begins to develop by the 10th week of gestation from two separate blastemas – one for the temporal bone component and one for the condyle. Superior to the condylar blastema, a band of mesenchymal cells develops that will eventually differentiate into the disk. All the components of the mature joint of TMJ can be seen at the 14th week of gestation[1].

The components of TMJ constitutes, capsule, extracapsular ligaments, articular eminence, glenoid fossa, condyle, disk, disk ligaments and synovial membrane[1].

The structure and biochemical composition of contacting surface of TMJ may be altered by articular disk displacements. Disk deformation and/or perforation, atypical cellular architecture, osteophyte formation, subchondral bone resorption, disruption of the physical continuity of the articular surface of the mandibular condyle, and adhesion formation have all been observed in TMJs with articular disk displacement[1].

The term *temporomandibular joint disorder* (TMD) was first suggested by *Bell,* which includes disorders of the joint as well as masticatory system. TMD is a heterogenous collection of signs and symptoms that can be generally characterized by the presence of pain, TM joint noise and limitation of jaw motion. They can be broadly grouped as structural/organic or functional disorders[2].

Multiple imaging avenues are available to evaluate TMJ disorders. Accurate assessment of skeletal and soft tissue abnormalities should be obtained before treatment planning.

CT examination of the TMJs began in the early 1970s. it has the advantages of extremely fast slice acquisition, generates a volume data set rather than simple slice thickness and data can be reformatted into multiple planes of imaging using computer techniques. It is extremely useful for revealing the skeletal abnormalities of the TMJ and has excellent predictive value[3].

CT images of the TMJ are generally obtained in axial and/or coronal projections. Both projections allow simultaneous bilateral TMJ depiction. The axial projection considered to be most useful in demonstrating osseous abnormalities. This projection is also easiest to achieve considering the construction of the CT machine. Examinations performed in the coronal projection can add information, but may be difficult to perform on patients with neck stiffness. Sagittal view can also be achieved as reconstruction from the axial or coronal images. These reconstructions can be valuable complements to the axial images in demonstrating larger bone changes such as osteophytes[3].

Retrospective studies support the general idea that TMJ internal derangement is likely to progress to osteoarthritis. Katzberg et al suggested that obstruction by disc displacement without reduction produced compressive forces that impaired contacting structures in the joint[3].

The purpose of the study is to correlate the clinical characteristics of temporomandibular joint (TMJ) disorder patients with osseous changes.

AIM OF THE STUDY:

The aim of this study is to correlate the clinical characteristics of temporomandibular joint (TMJ) disorder patients with osseous changes in CT.

OBJECTIVES OF THE STUDY:

1. To correlate the clinical characteristics of temporomandibular joint (TMJ) disorder patients with osseous changes in CT.

2. To evaluate the types of osseous changes that occurs in temporomandibular joint apparatus in symptomatic temporomandibular disorder patients.

3. To evaluate the prevalence of various types of osseous changes that occurs in temporomandibular joint apparatus due to symptomatic temperomandibular disorder.

Temporomandibular joint disorder (TMJD) is a global common disease, which generally includes a number of separate entities and multiple etiologies, whose clinical signs or symptoms are almost always clustered into muscle disorders, intracapsular derangements of the components of the temporomandibular joint (TMJ), and degenerative changes in the bony components of the joint itself. Imaging plays a vital role in the diagnosis of TMDs. This study aims to elucidate the osseous changes in CT of patients with temporomandibular disorders.

FUNCTIONAL ANATOMY[4]

The temporomandibular joint or craniomandibular articulation is a ginglymoid-arthrodial joint. Each joint is an articulation between the articular tubercle eminence of the squamous portion of the temporal bone (the mandibular fossa or glenoid fossa) and the mandibular condyle. A fibrous disc, which acts as a third bone, is interposed between the condyle and the fossa formed by the temporal bone. These paired joints and the mandible, a single bone that crosses the skeletal midline, function together since neither joint is capable of independent movement. That is, one temporomandibular joint cannot possibly move without producing movement in the opposite joint.

The human mandible is the first bone of the body to demonstrate an ossification center. At approximately six weeks in utero, developing from the mandibular process of the first branchial arch, the mandible is seen as a thin plate of bone in close association to the lateral side of the anterior region of Meckel's cartilage on both sides of the developing face. Although Meckel's cartilage does not contribute much to mandibular development, it does to the incus, malleus, sphenomandibular and malleo-mandibular ligaments. All major portions of the mandible (the body, ramus, coronoid and condylar processes), develop by intramembranous ossification. Only the articular surface of the condyle and the tip of the coronoid process develop by endochondral ossification. The articular eminence of the temporal bone is composed of compact bone overlying trabecular bone with marrow spaces. Both the articular eminence and the articulating surface of the condyle are covered with fibrocartilage, not hyaline cartilage, as in most other articulations of the body.

The temporomandibular joint is richly innervated by three different branches of the third division of the trigeminal nerve. The auriculotemporal nerve, providing innervation to the posterior, lateral and some medial portions of the joint, contributes approximately 75% of the total sensory supply to the joint. Anterior and medial innervation of the temporomandibular joints is provided by the masseteric nerve, giving about 15% of the total innervation. The posterior deep temporal nerve, supplying about 10% of the innervation, furnishes sensory innervation to a small area in the anterolateral portion of the joint.

Blood flow to the temporomandibular joints is also abundant and from many sources. The principle blood supply comes from the superficial temporal artery and branches of the maxillary artery, both of which are the terminal branches of the external carotid artery. Venous drainage is provided by companion veins, all of which contribute to the retromandibular vein, and by the facial vein, which contributes to the anterior jugular vein.

TERMINOLOGY

Over the years functional disturbances of the masticatory system have been identified by a variety of terms. In 1934, James Costen described a group of symptoms that centered on the ears and TMJ. Because of his work the term Costen Syndrome developed. Later the term Temporomandibular joint disturbances became popular. In 1959, Shore introduced the term Temporomandibular joint dysfunctional syndrome. Later came the term Functional Temporomandibular joint disturbances coined by Ash and Ramfjord. Some terms described the suggested cause such as, Occlusomandibular disturbances and myoarthropathy of the temporomandibular joint. Others stressed pain such as pain dysfunction syndrome, myofacial pain dysfunction syndrome and temporomandibular pain dysfunction syndrome. Because the symptoms are not always isolated to the TMJ, some authors believe that the foregoing terms are too limited and that a broader, collective term should be used, such as Craniomandibular disorders. Bell suggested the term **Temporomandibular**

disorders which has gained popularity. This term does not suggest merely problems that are isolated to the joint, that includes all disturbances associated with the function of the masticatory system.

DEFINITION[5]

According to the American Association of Orofacial Pain (AAOP) definition, a temporomandibular disorder (TMD) is: "a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint and associated structures, or both."

Internal derangement of the temporomandibular joint (TMJ) may be defined as a disruption within the internal aspects of the TMJ in which there is a displacement of the disc from its normal functional relationship with the mandibular condyle and the articular portion of the temporal bone

Disk displacement with reduction: disk is displaced from its position between the condyle and eminence to an anterior and medial or lateral position but is reduced in full opening, usually resulting in a noise

Disk displacement without reduction with limited opening: disk is displaced from normal position between condyle and fossa to an anterior and medial or lateral position, associated with limited opening

Disk displacement without reduction without limited opening: disk is displaced from its position between condyle and eminence to an anterior and medial or lateral position, not associated with limited opening

CLASSIFICATION

The research diagnostic criteria (RDC) developed by Dworkin and LeResche (1992), established a dual diagnosis that recognizes not only the physical conditions (axis I), including muscle disorders, disc displacements and other types of joint conditions that may contribute to the pain disorder, but also the psychosocial issues (axis II) that contribute to the suffering, pain behavior, and disability associated with the patient's pain experience.

TMD's RDC groups: Classification of temporomandibular joint disorders. Axis I. (Dworkin and LeResche, 1992 axis I)[6]

I GROUP I: Muscle disorders:

- Ia. Myofascial pain
- Ib. Myofascial pain with limited opening

II GROUP II: Disc Displacements (DD):

- IIa. DD with reduction
- IIb. DD without reduction with limited opening
- IIc. DD without reduction without limited opening

III GROUP III: Other common Joint disorders:

IIIa. Arthralgia

IIIb. Osteoarthritis

IIIc. Osteoarthrosis

The subtype classification of temporomandibular joint disorder established by the Japanese Society for the Temporomandibular Joint in 2001[7]

Type I: Masticatory muscle disorder

There is jaw movement pain in the muscle whose region can be identified.

Type II: Capsule-ligament disorder

There is movement pain in the TMJ with palpation tenderness. (This category includes chronic and traumatic diseases of either the retrodiscal tissue, joint capsule or ligament)

Type III: Disc disorder

Type IIIa: Disc displacement with reduction

There is a clicking sound or temporal sticking motion when opening and closing the mouth.

Type IIIb: Disc displacement without reduction

There is trismus and jaw opening pain or clenching pain after the disappearance of clicking. A protrusive slide of the mandibular condyle is usually disturbed on the problem side.

Type IV: Degenerative joint diseases, osteoarthritis, osteoarthrosis

There is at least one of joint pain, a trismus or a joint sound. A picture image reveals marginal proliferation (osteophyte), erosion or a deformity of the mandibular condyle.

Type V: Cases not included type I-IV

WILKES CLASSIFICATION OF INTERNAL DERANGEMENT [3]

Early Stage

Clinical: No significant mechanical symptoms other than reciprocal clicking (early in opening movement, late in closing movement, and soft in intensity); no pain or limitation in opening motion

Radiologic: Slight forward displacement; good anatomic contour of disk; normal tomograms

Surgical: Normal anatomic form; slight anterior displacement; passive incoordination (clicking) demonstrable

Early-Intermediate Stage

Clinical: First few episodes of pain; occasional joint tenderness and related temporal headaches; beginning of major mechanical problems; increase in intensity of clicking sounds; joint sounds later in opening movement; and beginning transient subluxations or joint catching and locking

Radiologic: Slight forward displacement; slight thickening of posterior edge or beginning of anatomic deformity of disk; normal tomograms

Surgical: Anterior displacement; early anatomic deformity (slight to mild thickening of posterior edge); well-defined central articulating area

Intermediate Stage

Clinical: Multiple episodes of pain, joint tenderness, temporal headaches, major mechanical symptoms: transient catching, locking, and sustained locking (closed locks); restriction of motion; difficulty (pain) with function

Radiologic: Anterior displacement with significant anatomic deformity or prolapse of disk (moderate to marked thickening of posterior edge); normal tomograms

Surgical: Marked anatomic deformity with displacement; variable adhesions (anterior, lateral, and posterior recesses); no hard-tissue changes

Intermediate-Late Stage

Clinical: Characterized by chronicity with variable and episodic pain, headaches, variable restriction of motion; undulating course

Radiologic: Increase in severity over intermediate stage; abnormal tomograms; early to moderate degenerative remodeling; hard-tissue changes

Surgical: Increase in severity over intermediate stage; hard-tissue degenerative remodeling changes of both bearing surfaces; osteophytic projections; multiple adhesions (lateral, anterior, and posterior recesses); no perforation of disk or attachment

Late Stage

Clinical: Characterized by crepitus on examination; scraping, grating, grinding; variable and episodic pain; chronic restriction of motion; and difficulty with function

Radiologic: Anterior displacement; perforation with simultaneous filling of upper and lower compartments; filling defects; gross anatomic deformity of disk and hard tissues; abnormal tomograms; essentially degenerative arthritic changes

Surgical: Gross degenerative changes of disk and hard tissues; perforation of posterior attachments; erosions of bearing surfaces; multiple adhesions equivalent to degenerative arthritis (sclerosis, flattening, and anvil-shaped condyle, osteophytic projections, and subcortical cystic formation).

CLINICAL STAGES[5]

Anatomical, epidemiological and clinical studies have shed some light upon the ultimate fate of the displaced disc. Traditionally, internal derangement of the TMJ has been described as a progressive disorder with a natural history that may be classified into four consecutive clinical stages[1,5,6]: stage one has been described as disc displacement with reduction, stage two as disc displacement with reduction and intermittent locking, stage three as disc displacement without reduction (closed lock), and stage four as disc displacement without reduction and with perforation of the disc or posterior attachment tissue (degenerative joint disease).

Stage One

Stage one is characterized clinically by reciprocal clicking as a result of anterior disc displacement with reduction. Although it has been stated that the later the opening click occurs, the more advanced the disc displacement, diagnostic assignment based on joint sounds has recently come under question. The fifth World Congress on Pain determined that “Clinic cases cannot be distinguished from controls on the basis of clinically detectable joint sounds.” This concept is further emphasized by Rohlin and others, who showed in an arthrographic study that anterior displacement with reduction can exist without joint noises (i.e., false negative).

The clinical hallmark of disc displacement with reduction is limited mouth opening, usually accompanied by deviation of the mandible to the involved side, until a pop or click (reduction) occurs. After the pop, the patient is able to open the mouth fully with a midline position of the mandible. Arthrograms show anterior disc displacement in centric occlusion, but the disc is normally located in the open-mouth position.

Stage Two

Stage two features all the aforementioned characteristics, plus additional episodes of limited mouth opening, which can last for various lengths of time. Patients may describe it as "hitting an obstruction" when opening is attempted. The "obstruction" may disappear spontaneously or the patient may be able to manipulate the mandible beyond the interference. Arthrographically, stage two is similar to stage one.

Stage Three

Closed lock (disc displacement without reduction) occurs when clicking noises disappear but limited opening persists. The patient complains of TMJ pain and chronic limited opening, with the opening usually less than 30 mm. Examination will reveal preauricular tenderness and deviation of the mandible to the affected side with mouth opening and protrusive movements.

TMJ pain may accompany border movement. Interestingly, arthrocentesis and arthroscopic surgery have documented consistently high success rates in relieving this particular pattern of internal derangement. Arthrographic examination and magnetic resonance imaging show anterior disc displacement in both centric occlusion and maximal mouth open positions. Limited condylar translation may also be evident.

In chronic closed lock episodes, if the condition progresses, the condyle may steadily push the disc forward to achieve almost normal ranges of mouth opening, in spite of the presence of a non-reducing disc.

Stage Four

With continued mandibular function, the stretched posterior attachment slowly loses its elasticity, and the patient begins to regain some of the lost range of motion. As retrodiscal tissue continues to be stretched and loaded, it becomes subject to thinning and perforation. Anatomic studies have shown that this tissue may remodel before it succumbs, ill-adapted to the functional load, and perforates. In addition, arthrograms have shown joint crepitus to be highly suggestive of but clearly not pathognomic of disc perforation.

Although often classified as characteristic of a separate final stage, hard tissue remodelling probably occurs throughout all stages. Clinically, osteoarthrosis may be diagnosed because the remodelling often occurs unilaterally, the symptoms appear to worsen as the day goes on, crepitation as distinct from clicking is often present and radiographic evidence is frequent (e.g., flattening, sclerosis, osteophytes, erosion).

The Progressive Nature of Internal Derangement

Although in many patients internal derangement undergoes the progressive changes just described, it is still not clear whether this progression happens in all cases. In fact, longitudinal epidemiological studies do not seem to support the idea of progression. For 10 years, Magnusson and others studied 293 subjects with clicking. At the five-year follow-up, clicking had not changed to locking in any of the subjects[8]. At the 10-year follow-up, only one of the 293 subjects reported intermittent locking[9].

Additionally, the authors reported that half the patients who exhibited clicking at age 15 no longer did so at age 20, and about half of those who did not exhibit clicking at age 15 went on to develop clicking. Thus, the probability that TMJ clicking would disappear in a symptomatic individual was equal to the probability of it appearing in an asymptomatic individual. This lack of progression of internal derangement from a reducing disc to a non-reducing disc condition was confirmed in studies by Greene and Laskin, Laskin and Lundh and others[10].

Sato and others[11] studied the natural course of anterior disc displacement without reduction in 44 subjects who agreed to observation without treatment. The incidence of successful resolution of the condition was

68% at 18 months. This finding suggests that the signs and symptoms of anterior disc displacement without reduction tend to be alleviated during the natural course of the condition. The authors failed to mention what happened to the anteriorly displaced disc. They noted, however, that the maximal mouth opening increased from 29.7 mm to 38 mm and concluded that it was unlikely that the disc became self-reducing; rather, it was more plausible that there was some stretching and remodelling of the retrodiscal tissues, enabling the disc to be displaced more anteriorly by the translating condyle.

Thus, although clinical evidence does support progressive worsening of the condition in some patients, important clinical questions remain. It is not clear what the progression rate is, nor is it clear which patients have the greatest risk of progressing to more advanced stages. Consequently, clinicians who justify aggressive treatment of asymptomatic TMJ clicking based on their belief in a high progression rate to a non-reducing state should instead exercise patience and clinical vigilance in their management of this condition.

ETIOLOGY

Etiological Concepts

The etiological concepts in its earlier days of inception, were purely mechanistic; attributing the various signs and symptoms to derangement of a particular anatomical region (temporomandibular joint, muscles of mastication or the occlusion). The earlier theories were based on a biomedical model comprising

- The mechanical displacement theory
- The trauma theory
- The biomedical theory
- The osteoarthritic theory
- The muscle theory

The mechanical displacement theory[12] hypothesized that the lack of molar support or functional occlusal prematurities caused a direct eccentric positioning of the condyle in the glenoid fossa, leading to pain, dysfunction and ear symptoms. The faulty condylar position led directly to adverse muscle activity. This theory gained momentum after Costen published his article focusing on occlusion as the most important causative factor for TMD. He proposed that due to the absence of molar support, the powerful elevating muscles of the mandible could press the condyles upward and backward causing damage to nerves and vessels including chorda tympani.

The trauma theory[13] proposed by Zarb and Speck considered micro-/macrotrauma as a principal factor that initiated pathologic processes and dysfunction in different parts of the stomatognathic system thus leading to the symptoms of TMD. According to this theory any trauma which can cause structural alteration to the joint or the muscles is considered Macrotrauma. Microtrauma refers to any small force that is repeatedly applied to the joint structures over a long period of time. Consequently, even though the etiological premise of this theory was related to trauma, it was actually an earlier multidimensional etiological model. However, no critical appraisal for the multitude of factors involved was given in the causation of TMD.

The biomedical theory[14] by Reade also supported the role of trauma in the initiation of the disorder. Once initiated, the condition will either resolve or in presence of certain factors like disrupted occlusion, parafunctional habits (particularly bruxism) and occupational activities, will progress further. Apart from factors causing increase or adverse functional loading, psychological elements were recognized as important maintaining influences. According to Reade (1984) “this theory would explain why similar occlusal interferences do not cause similar symptoms in different individuals and why all individuals with stress do not develop TMD.

The osteoarthritic theory[15] by Stegenga proposed osteoarthrosis as the causative factor for TMD. According to this theory muscular symptoms and internal derangement were secondary to joint pathology Pathological changes in the TMJ could be induced by absolute or relative overloading. Absolute overloading of the joint can occur at the time of trauma. Relative overloading could happen if the adaptive capacity of the joint structures is reduced by inflammation and ageing. This theory can explain some subcategories of TMD, but lacks in its ability to explain all the other disorders under the TMD’s.

The muscle theory[16] supported by Travell and Rinzler, suggested that the primary etiologic factor was in the masticatory muscles themselves. It suggests that myalgia of masticatory muscles can refer pain to TMJ. The myalgia in the facial region is caused by chronic myospasm which is secondary to parafunctional habits. This theory placed the temporomandibular pain in the context of a wider general muscle disorder and denied any influence of the occlusion.

The neuromuscular theory[17] supported by Ramjford proposed that the occlusal interferences were the causative factor for the disorder. He noted that regional pain associated with bruxism and myalgia was completely eliminated in subjects after occlusal equilibration. This theory proposed that the occlusal interferences caused an altered proprioceptive feedback, leading to incoordination and spasm of some of the masticatory muscles. Slowly the idea of TMD's occurring outside the realm of physical factors started percolating through. Perhaps the very first attempt in this direction was made by Schwartz.

The psychophysiological theory[2] by Schwartz and Laskin, suggested that the psychological factors are more important than the occlusal disturbances in initiating and perpetuating TMD. Spasm of the masticatory muscles, caused by overextension, overcontraction or muscle fatigue due to parafunctions was used by patients as a means to relieve stress. According to this theory it is the interaction between physiological predisposition, and psychological stress which causes TMD. The effect on the individual depended on their ability to cope with stress. Later several theories emerged based on the psychological and psychosocial factors. There is currently considerable evidence that psychological and psychosocial factors are of importance in the understanding of TMD as with other chronic pain disorders.

The psychological theory proposed that emotional disturbances initiating centrally, induced muscular hyperactivity which led to parafunctional habits and so indirectly to occlusal abnormalities. It emphasizes emotional factors, particularly stress, whereby tense individuals clench their teeth creating a state of muscle contractility that leads to pain. In TMD patient the behavioural aspect of the patient needs to be studied. Several authors have confirmed the role of psychological factors in TMD[18,19].

Various researchers have talked about the influence of personality, mental attitude and behavioral pattern of the patient on TMD[20-22].

Despite ample support concerning the relevance of emotional and affective factors in TMD, it is still not clear whether they are the cause or the consequence of pain. Of importance is the recognition of somatization in the assessment and management of TMD, wherein there is a preoccupation with physical symptoms disproportionate to actual physical disturbance. Scientific literature confirms at least the following psychological and psychosocial dimensions as important in the assessment and management of TMD: affective disturbance (depression and/or anxiety), somatization and psychosocial dysfunction. Also poor correspondence between objective signs (peripheral dysfunctional aspects) and subjective symptoms (intrinsic and extrinsic central aspects of pain perception), maladaptive coping resources and excessive use of the health care system are considered important. There is now general agreement that all patients with TMD should be screened for psychological and psychosocial dysfunction[23].

Gradually, concepts based on a single factor lost their scientific and clinical credibility. As it became more and more apparent that the etiology was multifactorial and that none of these theories in isolation could explain the etiologic mechanisms in TMD patients. The theories advanced from a pure mechanistic view, and expanded to a wider arena inclusive of psychological and behavioral factors. This development also led to the conclusion that temporomandibular disorders were not a single disease but a collection of structural and/ or functional disorders resulting clinically in comparable and analogue complaints. It also became evident that, with respect to the multifactorial etiology, the same factor wielded a different importance in the etiologic process, by playing a role in initiation, precipitation or perpetuation of the symptoms[24].

The Multifactorial Concept

The TMJ and the stomatognathic system in general are affected by a large variety of pathological conditions with different prognosis. They often overlap with respect to their signs and symptoms thus making the differential diagnosis in the individual patient difficult resulting in diagnostic errors. It is now generally accepted that the etiology is multifactorial for TMD even though finding the primary etiologic factor can be difficult for the individual patient[25, 26].

1. Age

The estimated prevalence of TMD in children and adolescents varies from 6-68%, depending on the different diagnostic criteria used and on the differences in clinical examination. In a study published by List et al. in adolescents between 12 and 18 years of age, 7% were diagnosed with temporomandibular pain-dysfunction, the prevalence being significantly higher in females than in males. Clicks were recorded 11% of the study population, with stiffness and mandibular fatigue in 3% and limitations in aperture in 1%[27].

Schmitter et al. reported that geriatric patients experience joint sounds in 38% of the cases and muscle pain in 12%, though without resting pain or joint pain. This contrasts with the group of young patients – with joint sounds in only 7% of cases, but with a much higher incidence of symptoms: facial pain in 7%, joint pain in 16%, and muscle pain in 25%[28].

2. **Genetic factors**[29]

Michalowicz et al. evaluated the hypothesis that signs and symptoms of TMD may be hereditary. To this effect they collected information by means of a questionnaire administered to a group of 494 monozygous and dizygous twins. The monozygous twins showed no greater similarities than in the case of the dizygous twins, and the homozygous twins that grew up together showed no greater similarities than those that grew up separately. The authors concluded that genetic factors and the family environment exert no relevant effect upon the presence of symptoms and signs of the TMJ.

3. **Sex**[30]

Epidemiological studies generally document a greater frequency and severity of TMD in females than in males. In effect, TMD is seen to be up to four times more frequent in women, and these tend to seek treatment for their TMJ problems three times more often than males. Attempts have been made to explain these differences in terms of behavioral, psychosocial, hormonal and constitutional differences, though no conclusive results have been drawn to date. It has been suggested that the presence of estrogen receptors in the TMJ of women modulates metabolic functions in relation to laxity of the ligaments, and this could be relevant in TMD. Estrogens would act by increasing vigilance in relation to pain stimuli, modulating the activity of the limbic system neurons. Although not all authors coincide, studies in humans have shown that the appearance of pain in the context of TMD increases approximately 30% in patients receiving hormone replacement therapy (HRT) in postmenopause (estrogens), and approximately 20% among women who use oral contraceptives.

4. Occlusion

Alterations in occlusion such as Angle malocclusions, crossbite, open bite, occlusal interferences, prominent overjet and overbite, crowding, midline discrepancies and missing teeth have been identified in different studies as predisposing, triggering or perpetuating factors. However, on one hand a relatively weak association is observed between occlusal factors and TMD, and on the other hand most studies published in the literature are of a cross-sectional design; as a result, few firm conclusions can be drawn regarding a possible causal relationship.

Donald Selligman and Andrew Pullinger, of the University of California, are probably the authors who have shown the greatest rigor in studying the relationship between occlusion and TMD. In their study published in the year 2000 comparisons were made of a group of women with internal TMJ derangement versus asymptomatic control women[31].

The patients with disc displacement were mainly characterized by unilateral posterior crossbite and long displacement of centric relation to the position of maximum intercuspidation. The patients with osteoarthrosis in turn associated an increased distance between centric relation and maximum intercuspidation, greater overjet and a reduction in overbite. The authors concluded that occlusal alterations may act as cofactors in the identification of patients with TMD and that some occlusal variables may be a consequence rather than a cause of TMD.

The results of this study are partially refuted by Hirsch et al., who after studying 3033 subjects concluded that greater or lesser overjet or overbite – even at extreme values – does not constitute a risk factor for the appearance of joint sounds (reciprocal clicks and crepitation)[32].

In the work published by Magnusson et al., involving the follow-up of 402 patients during 20 years, it was concluded that occlusal factors are weakly associated to TMD, though forced laterality between centric relation and maximum intercuspidation, and unilateral crossbite deserve consideration as possible local risk factors in the appearance of TMD[33].

In view of the information provided by the literature, the precise role of occlusion in TMJ pathology does not seem to be clearly defined. In contrast, and as has been pointed out by Koh et al. in an analysis of the published randomized and quasi-randomized trials on the subject, there appears to be no evidence that occlusal fit treats or prevents TMD, and that it therefore cannot be recommended for the management or prevention of such disorders[34].

5. Hyperlaxity

Kavuncu et al. evaluated the risk of TMD in patients with systemic and TMJ hypermobility. Local hypermobility was diagnosed in the presence of condylar subluxation, while systemic hypermobility was assessed by means of the Beighton test. The authors found that both local and general hypermobility are more frequently detected in patients with TMD than in the controls, and that the risk of TMJ dysfunction is greater if the patient presents both alterations simultaneously. The investigators concluded that both situations may play a role in the etiology of TMD[35].

The study by de Coster et al[36] likewise supports the hypothesis that hyperlaxity could cause TMD, since in a series of 31 subjects with Ehler-Danlos disease, all presented symptoms of temporomandibular dysfunction and suffered recurrent temporomandibular dislocations. These results are in contrast to those previously reported by Conti et al., [37] who compared a group of 60 patients with mandibular sounds, pain or block versus a group of 60 asymptomatic patients. No association was found between the intraarticular disorders and systemic hyperlaxity or between TMJ mobility and systemic hypermobility.

6. Antecedents of acute trauma

The possibility that acute trauma may induce histological alterations of the TMJ has been evidenced by studies in rats in which joint synovitis was generated by forcing condylar mobility. Improvement in synovitis or its total disappearance 20 weeks later was also observed. There are no conclusive results regarding whether acute trauma (whiplash in traffic accidents being the most extensively studied example) acts as a triggering factor of chronic TMD. Klobas et al.,[38] found that patients with antecedents of whiplash showed significant differences versus patients without such antecedents, with more frequent severe TMJ symptoms (89% versus 18%) and also more clinical signs. Likewise, maximum oral aperture was smaller (54 mm versus 48 mm). Pain in response to the palpation of muscles and joints was more common, as was pain in response to mobilization. The authors concluded that the prevalence of TMD is greater among individuals with chronic whiplash injury than in the controls, and that neck injuries can affect TMJ function.

Different results have been published by Probert et al. in a retrospective study in Australia, involving 20,673 traffic accident victims. They documented 28 patients with TMD and only one of the 237 patients that suffered mandibular fracture required posterior treatment for TMD. They concluded that the incidence of TMD after whiplash is very small and that this mechanism of trauma alone is unable to account for TMD. Ferrari et al. postulated that a series of cultural and psychosocial factors could in fact be more relevant than whiplash in explaining why some patients in certain societies refer chronic symptoms[39].

7. Parafunctional Habits

Dorland's Medical dictionary defines parafunction as disorderly or perverted function. Although the relationship between parafunction and muscle pain is biologically plausible, and there is some evidence to suggest a chronological relationship between the two, the fact is that controversy exists regarding this purported causal relationship.

Chewing gum has been used in a number of studies to evaluate the appearance of muscle pain with overfunction.

Karibell et al.[40] after inducing the chewing of gum for 6 minutes, found pain to increase in both males and females in the patient group, though unexpectedly it also increased among the women in the control group – thus supporting the hypothesis of increased female susceptibility.

Miyake et al.[41] in a group of 3557 university students, found that chewing gum on one side of the mouth only, and tooth clenching, increased the risk of TMD – though the corresponding odds ratio (OR) only reached 2 for limitation in oral aperture among the subjects that chewed gum on one side only.

In a study published by Winocur et al.[42] in Tel Aviv (Israel) among 323 females aged 15-16, it was seen that those individuals with an intense habit of chewing gum (more than 4 hours a day) associated pain in the ear region at rest and during movement, as well as a greater prevalence of joint sounds. What the authors referred to as “jaw play” (the habit of forced mandibular lateralization or protrusion movements without occlusal contact) appeared less often

a. Bruxism

The prevalence of bruxism in the adult population is around 20%, and is similar to that recorded in children. In a recent study conducted in Boston by Cheifetz et al., parent interviewing revealed that 38% of the children (in a group of 854 with a mean age of 8.1 years) presented bruxism. However, only 5% of the parents reported subjective symptoms of TMD in their offspring[43].

The greatest incidence of bruxism is between 20 and 50 years of age, after which the habit progressively decreases. Regarding the etiology of bruxism, the intervention of occlusal interferences was initially postulated, though at present emotional stress is considered to be the principal triggering factor. Other factors that have been related to the origin of bruxism are certain drugs, central nervous system disorders, and a certain genetic and/or familial predisposition. Magnusson et al.[44] in a longitudinal study of 420 individuals followed-up on for 20 years, reported a significant correlation between bruxism and TMD. Dental crowding at the start of the study was seen to be a predictor of TMD.

Huang et al.,[45] in a study of 274 patients diagnosed with myofascial pain (n=97), arthralgia (n=20), and myofascial pain plus arthralgia (n=157), found the diagnosis of myofascial pain to be significantly associated to tooth clenching (OR=4.8). In the group of patients with myofascial pain plus arthralgia, the odds ratio was 3.3 versus the control group.

8. Stress, anxiety and other psychological factors [46]

In 1955, Laszlo Schwartz et al. reported that a group of patients within the population classified as presenting "TMJ syndrome" could be characterized by painful limitation of mandibular movement caused by masticatory muscle spasm, and that this syndrome (known as mandibular pain dysfunction) was probably of myofascial origin. Emphasis was placed on psychological stress rather than on occlusal disharmony, as primary cause of the problem.

In 1969, Daniel Laskin proposed the psychophysiological theory of myofascial pain, where stress is defined as a major causal factor. According to this theory, stress induces muscle hyperactivity. Fatigue resulting from such hyperactivity in turn would cause muscle spasms, with the following consequences: contracture, occlusal disharmony, internal derangement and degenerative arthritis. These factors would be able to alter the occlusion pattern during mastication, and this alteration therefore would be the effect rather than the cause of the pain-dysfunction syndrome.

Different studies have confirmed that patients with myofascial pain and with myofascial pain associated to arthralgia, arthritis or arthrosis suffer increased levels of depression and somatization than those diagnosed only with disc displacement.

9. Orthodontic treatment

The possibility that orthodontic treatment could cause TMJ pathology has been extensively dealt with in the scientific literature. Despite the diverse methodological approaches involved, the great majority of studies conclude that orthodontic treatment neither improves nor worsens TMD.

Kim[47] reviewed 31 publications on orthodontics and TMD. He drew attention to the heterogeneity of the methodologies involved in these studies, and pointed out that only one of the reviewed articles found tooth extraction during orthodontic treatment to change the prevalence of TMD. The author concluded that orthodontic treatment does not increase the prevalence of TMD. Mohlin et al[48] are of the same opinion. In a study conducted in Gothenburg (Sweden) involving 337 patients followed-up on between 11 and 30 years of age, they found that orthodontic treatment neither prevents nor improves dysfunction of the TMJ.

EPIDEMIOLOGY

In the National Oral Health Survey conducted in Spain in 1994, in accordance with the criteria for epidemiological studies on oral health auspiced by the World Health Organization (WHO), it was seen that at 12 years of age 6.3% of the population presented clicks – a figure that increased to 9.4% in those aged 15 years, 14.70% in the 35-44 years age range, and 23% in the 65-74 years age group. Limitation of oral aperture was seen to affect 2.2% at 12 years of age, 4.5% in the 35-44 years interval, and 3.5% in the 65-74 years age group. Pain in turn affected 0.2% of the population aged 15 years, 3.4% of those in the 35-44 years age group, and 1.3% of the subjects aged 65-74 years[49].

In the following survey carried out at national level in the year 2000, it was seen that 17.6% of the population aged 35-44 years presented clicks, while 1.8% suffered pain in response to palpation, and 1.8% had limited mobility. Symptoms were detected in 10.8% of the population. In the 65-74 years group, clicks were present in 15.5% of subjects, pain in response to palpation in 2.5%, and reduced mobility in 2.9%. Symptoms were present in 11.2% of the population[50].

In the studies of prevalence of the disease, the variability is extreme – ranging from 6% to 93% when based on patient-contributed information, and from 0% to 93% when based on clinical evaluation[51].

The epidemiological studies of TMJ alterations based on imaging analyses likewise have been unable to define a standardized pattern in the distribution of the disease. Radiographic changes corresponding to osteoarthrosis appear in 14-44% of the individuals – a figure far from the 1-24% of patients who showcrepitants in response to palpation or to auscultation of the TMJ (crepitation being considered a clinical sign of osteoarthrosis). In contrast to what might be expected, there is a poor correlation between the magnetic resonance imaging (MRI) findings in relation to the alterations of the intraarticular meniscus and the corresponding clinical findings[51].

The sample comprised 196 subjects, aged 18-25 years. According to our results, 50% of the subjects had TMD, but it was of moderate or severe degree in only 9.18% of them. No statistically significant association could be found between TMD and gender or occlusion. TMD was found to have statistically significant association with HADSa but not with HADSd[52].

A total of 240 subjects (103 males, 127 females, mean age 35.7 ± 12.5 years) participated. The prevalence of individuals with at least one TMD symptom was 37%, and no gender differences were found. However, significant differences were found between the levels of psychological factors among females and males who did not suffer from chronic pain[53].

TMD SYMPTOMS [4]

1. The most common symptoms of a temporomandibular disorder are:

- Ear symptoms.
- Headache.
- Neck and upper shoulder muscle pain.
- Jaw pain.
- Temporomandibular joint noise (clicking, grating) with mandibular movement. (This is only a symptom if it is painful or associated with dysfunction)
- Limited mouth opening and/or disturbances in capacity for mandibular movement.
- Dizziness.
- Pain and paresthesia in the upper extremities.
- Difficulty in swallowing.

TMD EXAMINATION[4]

The six parts of the TMD examination include:

1. Case history.
2. Range of motion.
3. Mandibular tracking.

4. Palpation.

5. Auscultation.

6. Joint/muscle challenges (provocations).

IMAGING STUDIES[54]

- Conventional radiography is the most utilized imaging study. It is simple, evaluates bony structures, and in most cases is sufficient.

- Dynamic high-resolution ultrasonography allows for visualization of the morphological elements and the functions of the TMJ, articular disk, mandibular condyle, and lateral pterygoid muscle. It is useful in the evaluation of internal derangements of the TMJ.

- CT scans can explore both bony structures and muscular soft tissues. Of interest, there is utility with cone beam computed tomography (CBCT). The patient is scanned with the mouth open and closed. Specifically, CBCT can aid in the diagnosis of osteoarthritis, rheumatoid arthritis, synovial chondromatosis, and neoplastic disorders

- MRI should be used as the study of choice if an articular or meniscal pathology is suspected and an endoscopic or surgical procedure is contemplated, or in the case of traumatic TMD.

MANAGEMENT[54]

- Most temporomandibular disorders (TMDs) are self-limiting and do not get worse. Simple treatment, involving self-care practices, rehabilitation aimed at eliminating muscle spasms, and restoring correct coordination, is all that is required. Nonsteroidal anti-inflammatory analgesics (NSAIDs) should be used on a short-term, regular basis and not on an as needed basis.

- On the other hand, treatment of chronic TMD can be difficult and the condition is best managed by a team approach; the team consists of a primary care physician, a dentist, a physiotherapist, a psychologist, a pharmacologist, and in small number of cases, a surgeon. The different modalities include patient education and self-care practices, medication, physical therapy, splints, psychological counseling, relaxation techniques, biofeedback, hypnotherapy, acupuncture, and arthrocentesis.

COMPUTED TOMOGRAPHY

X-ray computed tomography, also **Computed Tomography** (**CT**) or **Computed Axial Tomography** (**CAT**), used for medical imaging. Digital geometry processing is used to generate a three-dimensional image of the inside of an object from a large series of two-dimensional X-ray images taken around a single axis of rotation[55].

Definition

Computed tomography (CT) scanning is a valuable diagnostic tool that provides physicians with views of internal body structures. During a CT scan, multiple x rays are passed through the body, producing cross-sectional images, or "slices," on a cathode-ray tube (CRT), a device resembling a television screen. These images can then be preserved on film for examination.

Etymology[56]

1917	Johann Radon demonstrated that the image of a 3-dimensional object can be recostructed from an infinite number of 2-dimsneisonal projections of the object
1956	Ronald Bracewell publishes paper mapping sunspots using a series of one-dimensional images to reconstruct a two-dimensional image using Fourier transform
1958	William Oldendorf builds a model CT scanner without a computer
1960	Oldendorf applies for a patent for his model
1963	Alan Cormack publishes results from experimental scanner using a computer to reconstruct images from data
1966	David Kuhl publishes paper with the transmission images of a subject's thorax

1967	Bracewell reconstructs lunar images without using Fourier transforms
1968	EMI patents Godfrey Hounsfieild's method apparatus and the apparatus for scanning the body with X-rays
1971	The first CT scanner, limited to the head, demonstrated by EMI at Atkinson Morley's hospital in London
1972	The first CT scanner demonstrated in the United States
1973	Robert Ledley markets ACTA, a whole body CT scanner
1975	Second gerneration Delta CT scanners are marketed GEs third generation CT scanners are marketed
1979	Cormack and Hounsfield are awarded the Nobel Prize in Medicine for the invention of CT
1985	Superfast CT is developed by Douglas Boyd
1989	First spiral CT enters the market

The word "tomography" is derived from the Greek tomos (slice) and graphein (to write). Computed tomography was originally known as the "EMI scan" as it was developed at a research branch of EMI, a company best known today for its music and recording business. It was later known as **computed axial tomography** (CAT or CT scan) and **body section röntgenography**

CT History & Development [57]

Types of CT Machine

Spinning tube, commonly called spiral CT, in which an entire X-ray tube is spun around the central axis of the area being scanned. The main limitation of this type is the bulk and inertia of the equipment (X-ray tube assembly and detector array on the opposite side of the circle) which limits the speed at which the equipment can spin.

Electron beam tomography is a specific form of CT in which a large enough X-ray tube is constructed so that only the path of the electrons, traveling between the cathode and anode of the X-ray tube, are spun using deflection coils. This type has a major advantage since sweep speeds can be much faster, allowing for less blurry imaging of moving structures, such as the heart and arteries. However, far fewer CTs of this design have been produced, mainly due to the higher cost associated with building a much larger X-ray tube and detector array[58].

Principle[58, 59]

CT produces a volume of data that can be manipulated, through a process known as “windowing”. It is a method used to vary the density and contrast. The window width is the range of CT numbers we select for display and the window level is usually but not always the central CT number about which the window is chosen.

Process [60, 61]

X-ray slice data is generated using an X-ray source that rotates around the object; X-ray sensors are positioned on the opposite side of the circle from the X-ray source. The earliest sensors were scintillation detectors, with photomultiplier tubes excited by (typically) cesium iodide crystals. Cesium iodide was replaced during the 1980s by ion chambers containing high-pressure Xenon gas. These systems were in turn replaced by scintillation systems based on photodiodes instead of photomultipliers and modern scintillation materials with more desirable characteristics. Many data scans are progressively taken as the object is gradually passed through the gantry.

Newer machines with faster computer systems and newer software strategies can process not only individual cross sections but continuously changing cross sections as the gantry, with the object to be imaged slowly and smoothly slid through the X-ray circle. These are called helical or spiral CT machines. Their computer systems integrate the data of the moving individual slices to generate three dimensional volumetric information (3D-CT scan), in turn viewable from multiple different perspectives on attached CT workstation monitors. This type of data acquisition requires enormous processing power, as the data are arriving in a continuous stream and must be processed in real-time.

In conventional CT machines, an X-ray tube and detector are physically rotated behind a circular shroud in the electron beam tomography

(EBT), the tube is far larger and higher power to support the high temporal resolution. The electron beam is deflected in a hollow funnel-shaped vacuum chamber. X-rays are generated when the beam hits the stationary target. The detector is also stationary. This arrangement can result in very fast scans, but is extremely expensive.

Once the scan data has been acquired, the data must be processed using a form of tomographic reconstruction, which produces a series of cross-sectional images. The most common technique in general use is filtered back projection, which is straightforward to implement and can be computed rapidly. In terms of mathematics, this method is based on the Radon transform. However, this is not the only technique available: the original EMI scanner solved the tomographic reconstruction problem by linear algebra, but this approach was limited by its high computational complexity, especially given the computer technology available at the time. More recently, manufacturers have developed iterative physical model-based expectation-maximization techniques. These techniques are advantageous because they use an internal model of the scanner's physical properties and of the physical laws of X-ray interactions. By contrast, earlier methods have assumed a perfect scanner and highly simplified physics, which leads to a number of artifacts and reduced resolution - the result is images with improved resolution, reduced noise and fewer artifacts, as well as the ability to greatly reduce the radiation dose in certain circumstances. The disadvantage is a very high computational requirement, which is at the limits of practicality for current scan protocols.

Pixels in an image obtained by CT scanning are displayed in terms of relative radiodensity. The pixel itself is displayed according to the mean attenuation of the tissue(s) that it corresponds to on a scale from +3071 (most attenuating) to -1024 (least attenuating) on the Hounsfield scale. Pixel is a two dimensional unit based on the matrix size and the field of view. When the CT slice thickness is also factored in, the unit is known as a Voxel, which is a three-dimensional unit. The phenomenon that one part of the detector cannot differentiate between different tissues is called the "Partial Volume Effect". That means that a big amount of cartilage and a thin layer of compact bone can cause the same attenuation in a voxel as hyperdense cartilage alone. Water has an attenuation of 0 Hounsfield units (HU), while air is -1000 HU, cancellous bone is typically +400 HU, cranial bone can reach 2000 HU or more (os temporale) and can cause artifacts. The attenuation of metallic implants depends on atomic number of the element used: Titanium usually has an amount of +1000 HU, iron steel can completely extinguish the X-ray and is, therefore, responsible for well-known line-artifacts in computed tomograms. Artifacts are caused by abrupt transitions between low- and high-density materials, which results in data values that exceed the dynamic range of the processing electronics.

Contrast mediums used for X-ray CT, as well as for plain film X-ray, are called radiocontrasts. Radiocontrasts for X-ray CT are, in general, iodine-based.[28] Often, images are taken both with and without radiocontrast. CT images are called precontrast or native-phase images before any radiocontrast has been administrated, and postcontrast after radiocontrast administration.

Three-dimensional reconstruction[62]

Because contemporary CT scanners offer isotropic or near isotropic, resolution, display of images does not need to be restricted to the conventional axial images. Instead, it is possible for a software program to build a volume by "stacking" the individual slices one on top of the other. The program may then display the volume in an alternative manner.

Multiplanar reconstruction[62]

Multiplanar reconstruction (MPR) is the simplest method of reconstruction. A volume is built by stacking the axial slices. The software then cuts slices through the volume in a different plane (usually orthogonal). As an option, a special projection method, such as maximum-intensity projection (MIP) or minimum-intensity projection (mIP), can be used to build the reconstructed slices.

MPR is frequently used for examining the spine. Axial images through the spine will only show one vertebral body at a time and cannot reliably show the intervertebral discs. By reformatting the volume, it becomes much easier to visualise the position of one vertebral body in relation to the others.

Modern software allows reconstruction in non-orthogonal (oblique) planes so that the optimal plane can be chosen to display an anatomical structure. This may be particularly useful for visualising the structure of the bronchi as these do not lie orthogonal to the direction of the scan.

For vascular imaging, curved-plane reconstruction can be performed. This allows bends in a vessel to be "straightened" so that the entire length can be visualised on one image, or a short series of images. Once a vessel has been "straightened" in this way, quantitative measurements of length and cross sectional area can be made, so that surgery or interventional treatment can be planned.

MIP reconstructions enhance areas of high radiodensity and so are useful for angiographic studies. MIP reconstructions tend to enhance air spaces so are useful for assessing lung structure.

3D rendering techniques [62]

Surface rendering

A threshold value of radiodensity is set by the operator (e.g., a level that corresponds to bone). From this, a three-dimensional model can be constructed using edge detection image processing algorithms and displayed on screen. Multiple models can be constructed from various thresholds, allowing different colors to represent each anatomical component such as bone, muscle, and cartilage. However, the interior structure of each element is not visible in this mode of operation.

Volume rendering

Surface rendering is limited in that it will display only surfaces that meet a threshold density, and will display only the surface that is closest to the imaginary viewer. In volume rendering, transparency and colors are used to allow a better representation of the volume to be shown in a single image. For example, the bones of the pelvis could be displayed as semi-transparent, so that, even at an oblique angle, one part of the image does not conceal another.

Image segmentation

Where different structures have similar radiodensity, it can become impossible to separate them simply by adjusting volume rendering parameters. The solution is called segmentation, a manual or automatic procedure that can remove the unwanted structures from the image.

GENERATIONS [63]

GENERATION	CONFIGURATION	DETECTORS	BEAM	MINIMUM SCAN TIME
FIRST	Rotate - Translate	1-2	Pencil thin	2.5 min
SECOND	Rotate - Translate	3-52	Narrow fan	10 sec
THIRD	Rotate - Rotate	256-1000	Wide fan	0.5 sec
FOURTH	Rotate – Fixed	600-4800	Wide fan	1 sec
FIFTH	Electron beam	1284	Wide fan electron beam	33 ms

INDICATION IN HEAD AND NECK [64]

1. Intracranial disease and trauma
2. Malignancy of jaws
3. Investigation of TMJ
4. Investigation of intrinsic and extrinsic swelling of salivary gland
5. Evaluation of bone for implant placement
6. Fracture of facial bones
7. Post-irradiation assessment
8. Foreign body assessment

ADVANTAGES OF CT [64]

1. It provides axial, coronal and sagittal view of a tissue.
2. It shows anatomically precise location of the lesion and extent.
3. It provides greater geometric precision.
4. CT allows reconstruction of cross sectional images of the entire maxilla or mandible or both from a single imaging procedure.
5. It helps in distinguishing between benign and malignant lesions.
6. The structures of the soft tissues both normal and pathological are clearly displayed.
7. A clearer picture is obtained as compared to conventional tomography

which is often blurred due to the superimposition of surrounding structures.

8. Due to inherent high contrast resolution of CT, difference between the tissues that differs by less than 1% in their physical densities can be made.

9. Sensitivity of the detector is more so that larger amount of information can be obtained from relatively small amount of radiation exposure to the patient.

10. Image can be manipulated.

11. Image can be enhanced by the use of IV contrast media.

DISADVANTAGES OF CT [64]

1. CT scan is sophisticated, costly and expensive to maintain.

2. Very high density materials like metal bullets and dental restorations produce severe artifact on CT scan which makes the interpretation difficult.

3. Very thin contiguous or overlapping slices may result in a high dose of radiation.

4. There is a inherent risk associated with the contrast medium.

REVIEW OF STUDIES IN CT

A Tsuruta et al in 2003 conducted a study in 37 orthodontic patients with temporomandibular disorders to investigate the relationship between the thickness of the roof of the glenoid fossa in the temporomandibular joint (TMJ) and the existence and types of condylar bone change. The roof of the glenoid fossa was significantly thicker in joints with bone change than in joints with no bone change .There was also a significant difference in relation to the type of condylar bone change: the thickness of the roof of the glenoid fossa in the erosion group was significantly greater than in the no bone change, flattening and osteophyte formation[65].

J Koyama et al in 2007 conducted a follow up study in 1032 joints from 516 subjects in order to clarify the incidence and type of bone changes in the temporomandibular joint (TMJ), and alteration of the change during follow-up, in patients with temporomandibular disorders (TMD). Condylar bone change was seen in 617 (63.7%) of 1032 joints and in 70 (68.6%) of 102 follow-up joints. The number of joints of Types D- deformity; and S deformity accompanied by erosion with or without roughening. - increased at follow-up, but those of Types N- no bone change , F- flattening and E-erosion decreased. The main direction of transition of condylar bone change in joints with TMD was absorptive bone change to absorptive with sclerotic (proliferative) bone change and further to sclerotic (proliferative) bone change[66].

Uekiet al in 2008 conducted a study in 47 Japanese patients with mandibular prognathism, 24 underwent SSRO and 23 underwent SSRO in combination with a Le Fort I osteotomy to evaluate the horizontal changes in the condylar head with bent plate fixation after sagittal split ramus osteotomy (SSRO) with and without a Le Fort I osteotomy. There was no significant difference in reduction in mandibular length between SSRO alone and SSRO with Le Fort I on the axial view of a 3D CT. There were no significant differences between pre- and postoperative horizontal changes in the condylar long axis or in the antero-posterior and medio-lateral displacement of the condylar head, although the length of the proximal segment in SSRO with Le Fort I osteotomy was significantly shorter than in SSRO alone[67].

Linda Z. Arvidsson et al in 2010 conducted a study in Forty-seven patients with JIA to assess the long-term temporomandibular joint (TMJ) manifestations of juvenile idiopathic arthritis (JIA), as depicted at computed tomography (CT) and magnetic resonance (MR) imaging, in 47 adult patients. The TMJs were involved in 33 (70%) of the 47 patients with JIA, with bilateral involvement in 29 patients. Slight to moderate contrast enhancement was observed on the images obtained in 14 (42%) of the 33 patients with TMJ JIA abnormalities. All main joint components were abnormal in 28 of the 33 patients, mainly showing flat deformed condyles, wide flat fossae, and thin or perforated disks in the normal position, or absent disks. Condylar concavity or bifidity, and secondary osteoarthritis were found in approximately half of the abnormal joints[68].

REVIEW OF STUDIES IN CBCT

Ji-Un Lee et al in 2007 conducted a study in 314 temporomandibular joints (TMJs) images of 163 TMD patients to assess bone changes of mandibular condyle using cone beam computed tomography (CBCT) in temporomandibular disorder (TMD) patients. Osteophyte (2.9%) was found more frequently on anterior surface of the mandibular condyle. Erosion (31.8%) was found more frequently on anterior and medial surfaces of the mandibular condyle[69].

H Hintze et al in 2007 conducted a study in 80 dry human skulls to compare the diagnostic accuracy of cone beam CT images with conventional omographic images for the detection of morphological temporomandibular joint (TMJ) changes. Detection of the various types of morphological changes in relation to the condyle and the articular tubercle assessed separately resulted in no significant differences between the two radiographic modalities, with the exception of bone defects in the articular tubercle examined on frontal views alone where the specificity with tomography was significantly higher than with cone beam CT. Detection of all morphological changes in relation to both the condyle and the articular tubercle showed a significantly higher accuracy with tomography than with cone beam CT using lateral views alone, but there was no significant difference between the two modalities using frontal views alone and lateral and frontal views in combination[70].

Alexandre Perez Marques et al in 2010 conducted a study in 30 dry mandibular condyles to analyze two protocols of cone beam computed tomography for the evaluation of simulated mandibular condyle bone lesions 1) axial, coronal and sagittal multiplanar reconstruction (MPR); and 2) sagittal plus coronal slices throughout the longitudinal axis of the mandibular condyles. The results showed there were no statistically significant differences between the 2 protocols[71].

José Valladares Neto et al in 2010 conducted a cross-sectional study in 36 condyles of 18 subjects from 3 to 20 years of age to investigate morphological changes of the mandibular condyle from childhood to adulthood using CBCT. The linear dimension of the mandibular condyle on the lateral section varied little with growth and seemed to be established early, while the dimension of the frontal section increased. Small asymmetries between left and right condyles were common but without statistical significance for both lateral (P=0.815) and frontal (P=0.374) dimensions[72].

ML dos Anjos Pontual et al in 2012 conducted a study in patients treated by a radiologist at a private dental radiology service over a period of 1 year to assess bone changes and mobility in temporomandibular joints (TMJs) using cone beam CT (CBCT) in a population sample in Recife, PE, Brazil. Bone changes were present in 227 (71%) patients. Age group and gender showed a statistically significant association with presence of bone changes (p # 0.05). There was no significant difference between the right and left sides (p 5 0.556) and in condylar mobility (p 5 0.925) with regard to the presence of degenerative bone changes[73].

Study Topic: Clinical correlation of osseous changes in CT for patients with temporomandibular joint disorders – A Prospective study

Study Design: The present study is a prospective analytical study.

Study Duration: This study was conducted between March 2012 to July 2012 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Saravana Scans, Anna Salai, Chennai.

Study Population:

A total number of 15 patients were involved in the study.

Obtaining approval from the authorities:

Permission from the ethical committee of **Ragas Dental College and Hospital**, Chennai was obtained before starting the study.

Due consent to participate in the study was obtained from the Subjects in letter format both in Tamil and English.

MATERIALS

Examination of the patient

INSTRUMENTS USED:

1. Dental chair with halogen lamp
2. Disposable latex gloves
3. Mouth mask
4. Plain mouth mirror
5. Dental probe
6. Metallic scale
7. Divider

RADIOGRAPHIC INVESTIGATION

CT machine model: Siemens Emotion 6 - Spiral CT scan

METHODOLOGY:

Inclusion criteria:

1. Patients with symptomatic TMJ disorder

Exclusion criteria:

1. Patients with TMJ changes due to developmental anamolies, age changes, trauma, infections, systemic diseases and tumours.
2. Patients with history of previous surgery in TMJ region

The patients included in the study were made to sit in the dental chair. They were interrogated to collect information regarding name, age, sex, address and chief complaint. They were examined clinically under the following headings

Pain / Tenderness :

- Character :
- Duration :
- Frequency:

- Functional disruption :

Mouth opening :

Deviation :

TMJ sounds :

Palpation :

Auscultation:

The findings were recorded on the proforma made for the study after getting signature from the patient in the letter of consent.

The patients were then subjected to CT investigation in Saravana scans, Anna salai, Chennai.

Preparation of the patient prior to examination:

The patients were advised to wear comfortable, loose-fitting clothing. Metal objects including jewelry, eyeglasses, dentures and hairpins were removed prior to the examination. The patients were then made to lie flat on their back in the CT examination table. Straps and pillows were used to help maintain the correct position and to hold still during the examination.

Once the scanning procedure is done, the images were obtained and evaluated for osseous changes. The type of bone change was determined on the sagittal and coronal CT images depicting the median portion of the condyle in closed-mouth position.

Condylar bone changes were determined under the following criteria:(Koyama et al [66])

Type N - No proliferation or thickening on the cortical surface of the condyle; displaying typical morphology.

Type F- Flattened contour at the anterosuperior and/or posterosuperior portions of the condyle.

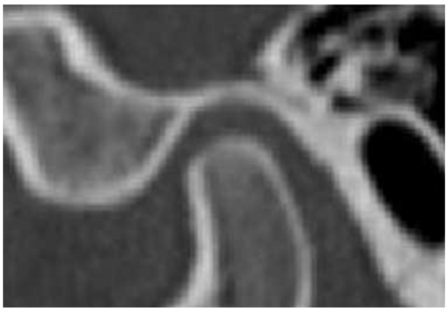
Type E - Proliferation or partial hypodense change with or without roughening on the cortical surface of the condyle.

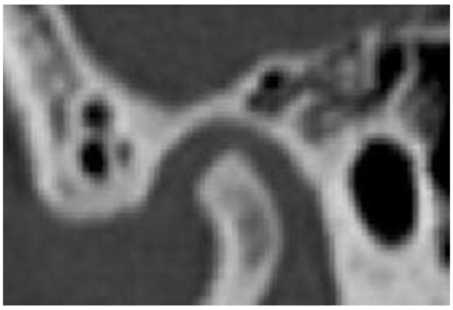
Type D- The condyle has a deformed contour, like a beak, without proliferation nor partial hypodense change on the condylar surface.

Type S- Type D accompanied by Type E.

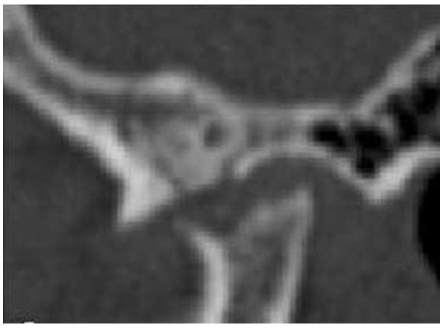
Fig.1: Condylar Bone Changes

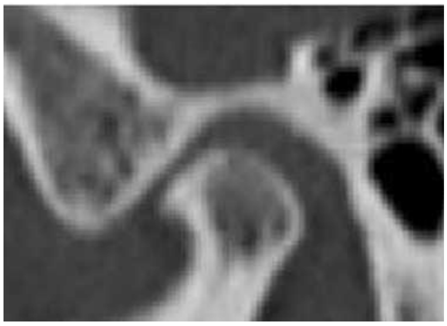
A. Normal **B. Flattened**





C. Erosion **D.Deformed**





Articular eminence morphology were classified into four types, according to the criteria set by Kurita *et al*.[74] as box, sigmoid, flattened or deformed.

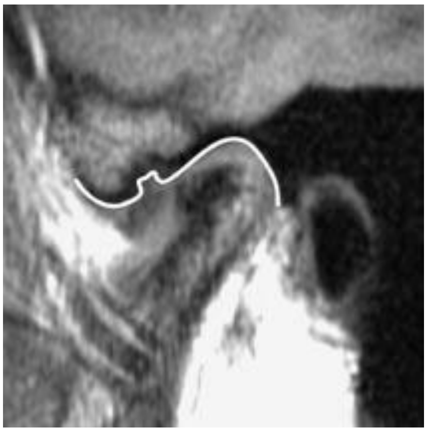
Fig.2: Articular Eminence Morphology

A. Box

B. Sigmoid

C. Flattened

D. Deformed



These findings were then correlated with the clinical characteristics of the patient and subjected to statistical analysis.

Scanning cone from base of the skull to mandible and 1.2mm slice reconstructed. Scan data were reformatted into 0.6 mm interval axial images at 4-fold magnification using the software included on the Xvigor Real CT, and were transferred to a Medical Viewer INTAGE RV version 1.3 workstation. The thinnest area of the glenoid fossa was identified among the multiple slices on the monitor and measured in DICOM viewer. (A Tsuruta et al [65])

The findings were then recorded on the proforma and subjected to statistical analysis.

STATISTICAL ANALYSIS:

Mean and standard deviation were estimated in the sample for each study group. Mean values were compared by using one-way ANOVA followed by multiple range tests by SPSS Software.

In the present study $P < 0.05$ was considered as the level of significance.

$$\text{Mean }(X) = \frac{\sum \overline{Xi}}{n}$$

$$\text{Standard Deviation} = \sqrt{\frac{\sum (Xi - X)^2}{n-1}}$$

Where Xi is the individual observation and n is the sample size.

Fig.3: CT Machine

Fig.4: Patient Positioning

Fig.5: Workstation

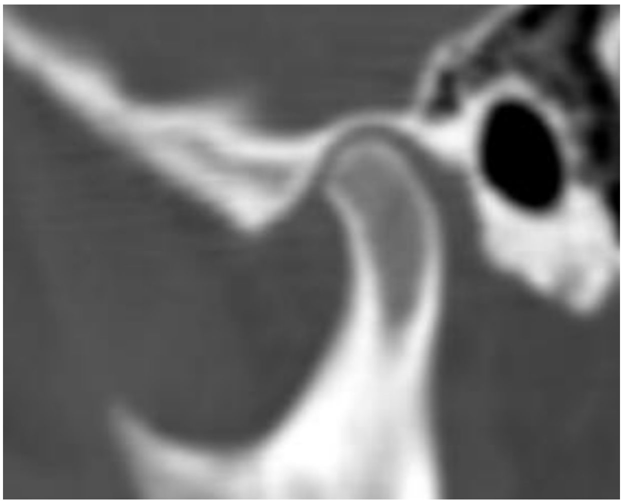


Fig.6a **Fig.6b**

Fig.6a: Showing extra oral picture of the patient and **Fig.6b**: Showing the CT picture demonstrating normal condyle

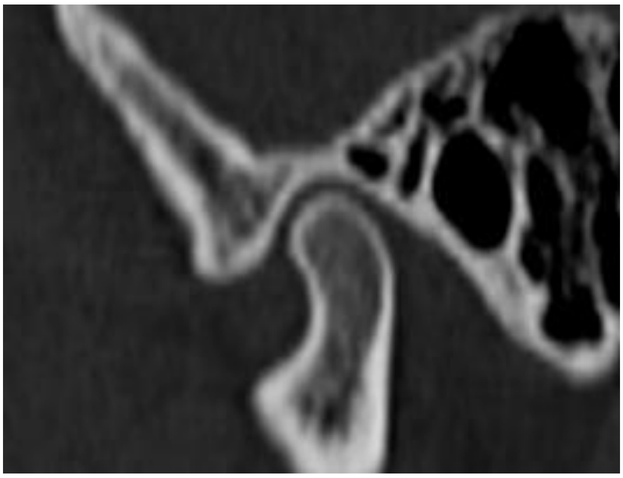


Fig. 7a **Fig. 7b**

Fig. 7a: Showing extra oral picture of the patient and **Fig.7b:** Showing the CT picture demonstrating normal condyle

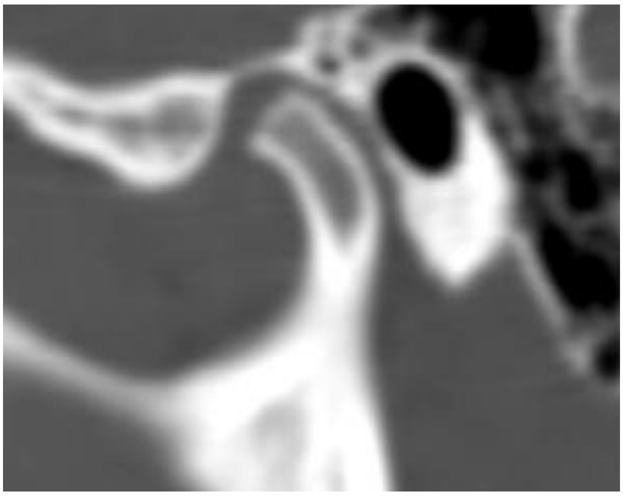


Fig.8a **Fig.8b**

Fig.8a: Showing extra oral picture of the patient and **Fig.8b:** Showing the CT picture demonstrating flattened condyle

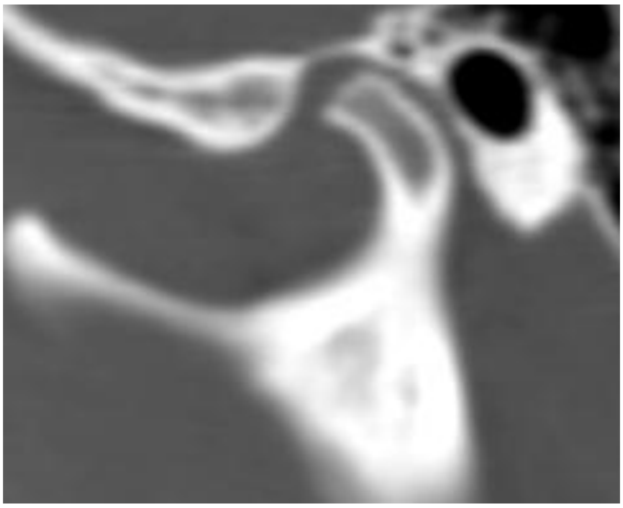


Fig.9a **Fig.9b**

Fig.9a: Showing extra oral picture of the patient and **Fig.9b:** Showing the CT picture demonstrating flattened condyle

Table-1 and Graph-1 shows the distribution of subjects according to sex:

A total of 15(100%) subjects were interrogated and examined in this study. Among the 15 subjects, 3(20%) were males and 12(80%) were females.

Table-2 and Graph-2 shows the distribution of subjects according to chief complaint:

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region, 1(6.7%) had pain in the right and left TMJ region, 2(13.3%) had clicking in left TMJ region, 2(13.3%) had clicking in right TMJ region, 1(6.7%) had clicking in both right and left TMJ region, 1(6.7%) had pain and clicking in the right TMJ region, 1(6.7%) had lock jaw.

Table-3 and Graph-3 shows the distribution of subjects according to mouth opening:

In the total of 15(100%) subjects, 7(46.7%) had mouth opening between 30 and 40 mm, 8(53.3%) had mouth opening above 40mm.

Table-4 and Graph-4 shows the distribution of subjects according to deviation:

In the total of 15(100%) subjects, 9(60.0%) did not have deviation, 2(13.3%) had deviation to right, 4(26.7%) had deviation to left.

Table-5 and Graph-5 shows the distribution of subjects according to palpatory findings:

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, 1(6.7%) had pain in right TMJ, 1(6.7%) had pain in right and left TMJ, 6(40.0%) had pain and clicking in the left TMJ, 2(13.3%) had pain and clicking in the right TMJ, 1(6.7%) had pain and clicking in the left and right TMJ, 1(6.7%) had pain and crepitus in the left TMJ, 1(6.7%) had pain and crepitus in the right TMJ.

Table-6 and Graph-6 shows the distribution of subjects according to auscultatory findings:

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, 6(40.0%) had clicking in left TMJ, 2(13.3%) had clicking in right TMJ, 1(6.7%) had clicking in left and right TMJ, 1(6.7%) had crepitus in left TMJ, 1(6.7%) had crepitus in right TMJ.

Table-7 and Graph-7 shows the distribution of articular eminence morphology in right TMJ

In the total of 15(100%) CT images examined for articular eminence morphology, 12(80.0%) were sigmoid, 1(6.7%) was flattened, 1(6.7%) was box and 1(6.7%) was deformed in shape.

Table-8 and Graph-8 shows the distribution of articular eminence morphology in left TMJ

In the total of 15(100%) CT images examined for articular eminence morphology, 9(60.0%) were sigmoid, 3(20.0%) was flattened, 3(20.0%) was box and none was deformed in shape.

Table-9 and Graph-9 shows the distribution of condylar changes in right TMJ

In the total of 15(100%) CT images examined for condylar changes, 12(80.0%) were normal and 3(20.0%) were flattened.

Table-10 and Graph-10 shows the distribution of condylar changes in left TMJ

In the total of 15(100%) CT images examined for condylar changes, 12(80.0%) were normal and 3(20.0%) were flattened.

Table-11 and Graph-11 shows the Correlation between chief complaint and mouth opening

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region,in which 5(71.4%) had mouth opening between 30 and 40mm, 2(28.6%) had mouth opening above 40mm. 1(6.7%) had pain in the right and left TMJ region, had mouth opening above 40mm(100.0%). 2(13.3%) had clicking in left TMJ region, in which both (100.0%) had mouth opening above 40mm. 2(13.3%) had clicking in right TMJ region, in which both (100.0%) had mouth opening above 40mm. 1(6.7%) had clicking in both right and left TMJ region, had mouth opening between 30 and 40mm, (100.0%). 1(6.7%) had pain and clicking in the right TMJ region, had mouth opening above 40mm(100.0%). 1(6.7%) had lock jaw, had mouth opening between 30 and

40mm(100.0%). The Correlation between chief complaint and amount of mouth opening was insignificant with a P value of 0.15.

Table-12 and Graph-12 shows the Correlation between chief complaint and palpatory findings

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region, in which 2(28.6%) had pain in the left TMJ, 4(57.1%) had pain and clicking in left TMJ, 1(14.3%) had pain and crepitus in left TMJ on palpation. 1(6.7%) had pain in the right and left TMJ region, had pain in the right and left TMJ region on palpation(100.0%). 2(13.3%) had clicking in left TMJ region, in which both(100.0%) had pain and clicking in left TMJ region on palpation. 2(13.3%) had clicking in right TMJ region, both(100.0%) had pain and clicking in right TMJ region on palpation.1(6.7%) had clicking in both right and left TMJ region, who had pain and clicking in both right and left TMJ region on palpation. 1(6.7%) had pain and clicking in the right TMJ region, had pain and crepitus in right TMJ region(100.0%) on palpation. 1(6.7%) had lock jaw, had pain in right TMJ region(100.0%) on palpation. The Correlation between chief complaint and palpatory findings was significant with a P value of 0.001.

Table-13 and Graph-13 shows the Correlation between chief complaint and auscultatory findings

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region, 2(28.6%) did not have any sound, 4(57.1%) had clicking in left, 1(14.3%) had crepitus in left on auscultation. 1(6.7%) had pain in the right and left TMJ region, did not have any sounds in auscultation. 2(13.3%) had clicking in left TMJ region, in which both(100.0%) had clicking in left on auscultation. 2(13.3%) had clicking in right TMJ region, in which both(100.0%) had clicking in right on auscultation. 1(6.7%) had clicking in both right and left TMJ region, had clicking in both right and left TMJ region on auscultation. 1(6.7%) had pain and clicking in the right TMJ region, had crepitus in right TMJ region(100.0%). 1(6.7%) had lock jaw had no sounds(100.0%). The Correlation between chief complaint and auscultatory findings was significant with a P value of 0.007.

Table-14 and Graph-14 shows the Correlation between chief complaint and AERT

In the total of 15(100%) CT images examined for articular eminence morphology, 7(46.7%) had pain in the left TMJ region, in which all 7(100.0%) subjects had sigmoid shape, 1(6.7%) had pain in the right and left TMJ region, who also had sigmoid shape, 2(13.3%) had clicking in left TMJ region, in which 1(50.0%) had sigmoid and 1(50.0%) had box shape, 2(13.3%) had clicking in right TMJ region, in which both had sigmoid shape, 1(6.7%) had clicking in both right and left TMJ region, who also had sigmoid shape 1(6.7%) had pain and clicking in the right TMJ region, who had flattened shape 1(6.7%) had lock jaw had deformed shape. The Correlation between chief complaint and morphology of articular eminence in right TMJ was highly significant with a P value of 0.005.

Table-15 and Graph-15 shows the Correlation between chief complaint and AELT

In the total of 15(100%) CT images examined for articular eminence morphology, 7(46.7%) had pain in the left TMJ region, 3(42.9%) subjects had sigmoid, 3(42.9%) had flattened, 1(14.3%) had box shape, 1(6.7%) had pain in the right and left TMJ region, who had sigmoid shape, 2(13.3%) had clicking in left TMJ region, both (100.0%) had sigmoid shape, 2(13.3%) had clicking in right TMJ region, in which 1(50.0%) had sigmoid shape 1(50.0%) had box shape and 1(6.7%) had clicking in both right and left TMJ region, who had sigmoid shape 1(6.7%) had pain and clicking in the right TMJ region, who also had sigmoid shape 1(6.7%) had lock jaw, had box shape. The Correlation between chief complaint and morphology of articular eminence in left TMJ was insignificant with a P value of 0.53.

Table-16 and Graph-16 shows the Correlation between chief complaint and CCRT

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region, in which all 7 (100%) of them had normal condylar morphology,1(6.7%) had pain in the right and left TMJ region who had normal condylar morphology(100%) , 2(13.3%) had clicking in left TMJ region in which 1(50%) had normal condylar morphology and 1(50%) had flattened condylar morphology , 2(13.3%) had clicking in right TMJ region in which both of them had normal condylar morphology(100%),1(6.7%) had clicking in both right and left TMJ region who had normal condylar morphology(100%), 1(6.7%) had pain and clicking in the right TMJ region who had flattened condylar morphology(100%), 1(6.7%) had lock jaw and had flattened condylar morphology(100%). The Correlation between chief complaint and condylar change in right TMJ was insignificant with a P value of 0.065.

Table-17 and Graph-17 shows the Correlation between chief complaint and CCLT

In the total of 15(100%) subjects, 7(46.7%) had pain in the left TMJ region in which 6(85.7%) had normal condylar morphology and 1(14.3%) had flattened condylar morphology, 1(6.7%) had pain in the right and left TMJ region who had flattened condylar morphology(100%), 2(13.3%) had clicking in left TMJ region in which 1(100%) had normal condylar morphology and 1(100%) had flattened condylar morphology, 2(13.3%) had clicking in right TMJ region in which 2(100%) of them had normal condylar morphology and, 1(6.7%) had clicking in both right and left TMJ region who had normal condylar morphology(100%), 1(6.7%) had pain and clicking in the right TMJ region who had normal condylar morphology(100%) and 1(6.7%) had lock jaw who had normal condylar morphology(100%). The Correlation between chief complaint and condylar change in right TMJ was insignificant with a P value of 0.368.

Table-18 and Graph-18 shows the Correlation between mouth opening and CCLT

In the total of 15(100%) subjects, 7(46.7%) had mouth opening between 30 and 40 mm, in which all(100%) of them had normal condylar morphology. 8(53.3%) had mouth opening above 40mm in which 6(75%) had normal condylar morphology and 2(25%) had flattened condylar morphology. The Correlation between mouth opening and condylar change in left TMJ was insignificant with a P value of 0.155.

Table-19 and Graph-19 shows the Correlation between mouth opening and CCRT

In the total of 15(100%) subjects, 7(46.7%) had mouth opening between 30 and 40 mm, in which 6(85.7%) had normal condylar morphology and 1(14.3%) had flattened condylar morphology, 8(53.3%) had mouth opening above 40mm in which 6(75%) had normal condylar morphology and 2(25%) had flattened condylar morphology. The Correlation between mouth opening and condylar change in right TMJ was insignificant with a P value of 0.605.

Table-20 and Graph-20 shows the Correlation between deviation and CCLT

In the total of 15(100%) subjects, 9(60.0%) did not have deviation, in which 7(77.8%) had normal condylar morphology and 2(22.2%) had flattened condylar morphology. 2(13.3%) had deviation to right, in which both(100%) had normal condylar morphology, 4(26.7%) had deviation to left, in which all of them(100%) had normal condylar morphology. The Correlation between deviation and condylar change in left TMJ was insignificant with a P value of 0.463.

Table-21 and Graph-21 shows the Correlation between deviation and CCRT

In the total of 15(100%) subjects, 9(60.0%) did not have deviation, in which 8(88.9%) had normal condylar morphology and 1(11.1%) had flattened condylar morphology. 2(13.3%) had deviation to right, in which both(100%) had normal condylar morphology, 4(26.7%) had deviation to left, in which 2(50%) had normal condylar morphology and 2(50%) had flattened condylar morphology. The Correlation between deviation and condylar change in right TMJ was insignificant with a P value of 0.202.

Table-22 and Graph-22 shows the Correlation between palpation and AERT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which both(100.0%) of them had sigmoid shape. 1(6.7%) had pain in right TMJ, had deformed shape(100.0%). 1(6.7%) had pain in right and left TMJ, had sigmoid shape(100.0%). 6(40.0%) had pain and clicking in the left TMJ, in which 5(83.3%) had sigmoid shape and 1(16.7%) had box shape. 2(13.3%) had pain and clicking in the right TMJ, in which both(100.0%) of them had sigmoid shape. 1(6.7%) had pain and clicking in the left and right TMJ, had sigmoid shape(100.0%). 1(6.7%) had pain and crepitus in the left TMJ, had sigmoid shape(100.0%). 1(6.7%) had pain and crepitus in the right TMJ had flattened shape(100.0%). The Correlation between palpation and articular eminence in right TMJ was insignificant with a P value of 0.066.

Table-23 and Graph-23 shows the Correlation between palpation and AELT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which 1(50.0%) had sigmoid and 1(50.0%) had box shape. 1(6.7%) had pain in right TMJ, who had box shape(100.0%) 1(6.7%) had pain in right and left TMJ, had sigmoid shape(100.0%). 6(40.0%) had pain and clicking in the left TMJ, in which 4(66.7%) had sigmoid shape and 2(33.3%) had flattened shape. 2(13.3%) had pain and clicking in the right TMJ, in which 1(50.0%) had sigmoid and 1(50.0%) had box shape. 1(6.7%) had pain and clicking in the left and right TMJ, had sigmoid shape(100.0%). 1(6.7%) had pain and crepitus in the left TMJ, had flattened shape(100.0%). 1(6.7%) had pain and crepitus in the right TMJ had sigmoid shape(100.0%). The Correlation between palpation and articular eminence in left TMJ was insignificant with a P value of 0.417.

Table-24 and Graph-24 shows the Correlation between palpation and CCRT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which both(100.0%) of them had normal condylar morphology. 1(6.7%) had pain in right TMJ, had flattened condylar morphology(100.0%). 1(6.7%) had pain in right and left TMJ, had normal condylar morphology(100.0%). 6(40.0%) had pain and clicking in the left TMJ, in which 5(83.3%) had normal condylar morphology, and 1(16.7%) had flattened condylar morphology. 2(13.3%) had pain and clicking in the right TMJ, in which both(100.0%) of them had normal condylar morphology. 1(6.7%) had pain and clicking in the left and right TMJ, had normal condylar morphology(100.0%). 1(6.7%) had pain and crepitus in the left TMJ, had normal condylar morphology(100.0%). 1(6.7%) had pain and crepitus in the right TMJ had flattened condylar morphology(100.0%). The Correlation between palpation and condylar changes in right TMJ was insignificant with a P value of 0.201.

Table-25 and Graph-25 shows the Correlation between palpation and CCLT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which both(100.0%) of them had normal condylar morphology. 1(6.7%) had pain in right TMJ, had normal condylar morphology(100.0%). 1(6.7%) had pain in right and left TMJ, had flattened condylar morphology(100.0%). 6(40.0%) had pain and clicking in the left TMJ, in which 5(83.3%) had normal condylar morphology, and 1(16.7%) had flattened condylar morphology. 2(13.3%) had pain and clicking in the right TMJ, in which both(100.0%) of them had normal condylar morphology. 1(6.7%) had pain and clicking in the left and right TMJ, had normal condylar morphology(100.0%). 1(6.7%) had pain and crepitus in the left TMJ, had normal condylar morphology(100.0%). 1(6.7%) had pain and crepitus in the right TMJ had normal condylar morphology(100.0%). The Correlation between palpation and condylar changes in left TMJ was insignificant with a P value of 0.352.

Table-26 and Graph-26 shows the Correlation between palpation and GTRT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which 1(50%) had glenoid thickness of 1.9mm and 1(50%) had glenoid thickness of 2.0mm in the right side. 1(6.7%) had pain in right TMJ, who(100%) had glenoid thickness of 1.9mm in the right side. 1(6.7%) had pain in right and left TMJ, who(100%) had glenoid thickness of 1.0mm in the right side. 6(40.0%) had pain and clicking in the left TMJ, in which 1(16.7%) had glenoid thickness of 1.2mm, 2(33.3%) had glenoid thickness of 1.7mm, 3(50%) had glenoid thickness of 1.8mm in the right side. 2(13.3%) had pain and clicking in the right TMJ, in which 1(50%) had glenoid thickness of 1.3mm and 1(50%) had glenoid thickness of 1.5mm in the right side. 1(6.7%) had pain and clicking in the left and right TMJ, who(100%) had glenoid thickness of 2.0mm in the right side. 1(6.7%) had pain and crepitus in the left TMJ, who(100%) had glenoid thickness of 1.3mm in the right side. 1(6.7%) had pain and crepitus in the right TMJ who(100%) had glenoid thickness of

1.0mm in the right side. The Correlation between palpation and glenoid thickness in right TMJ was insignificant with a P value of 0.222.

Table-27 and Graph-27 shows the Correlation between palpation and GTLT

In the total of 15(100%) subjects, 2(13.3%) had pain in left TMJ, in which 1(50%) had glenoid thickness of 1.8mm and 1(50%) had glenoid thickness of 2.3mm in the left side. 1(6.7%) had pain in right TMJ, who(100%) had glenoid thickness of 1.5mm in the left side. 1(6.7%) had pain in right and left TMJ, who(100%) had glenoid thickness of 1.5mm in the left side. 6(40.0%) had pain and clicking in the left TMJ, in which 1(16.7%) had glenoid thickness of 1.3mm, 1(16.7%) had glenoid thickness of 1.4mm, 1(16.7%) had glenoid thickness of 1.5mm and 3(50%) had glenoid thickness of 1.7mm in the left side. 2(13.3%) had pain and clicking in the right TMJ, in which 1(50%) had glenoid thickness of 1.6mm and 1(50%) had glenoid thickness of 1.8mm in the left side. 1(6.7%) had pain and clicking in the left and right TMJ, who(100%) had glenoid thickness of 1.7mm in the left side. 1(6.7%) had pain and crepitus in the left TMJ, who(100%) had glenoid thickness of 1.7mm in the left side. 1(6.7%) had pain and crepitus in the right TMJ who(100%) had glenoid thickness of 1.3mm in the left side. The Correlation between palpation and glenoid thickness in left TMJ was insignificant with a P value of 0.522.

Table-28 and Graph-28 shows the Correlation between auscultation and AERT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 3(75.0%) had sigmoid and 1(25.0%) had deformed shape. 6(40.0%) had clicking in left TMJ, in which 5(83.3%) had sigmoid and 1(16.7%) had box shape. 2(13.3%) had clicking in right TMJ, in which both(100.0%) had sigmoid shape. 1(6.7%) had clicking in left and right TMJ, had sigmoid shape(100.0%). 1(6.7%) had crepitus in left TMJ, had sigmoid shape(100.0%). 1(6.7%) had crepitus in right TMJ, had flattened shape(100.0%). The Correlation between auscultation and articular eminence morphology in right TMJ was insignificant with a P value of 0.202.

Table-29 and Graph-29 shows the Correlation between auscultation and AELT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 2(50.0%) had sigmoid and 2(50.0%) had box shape, 6(40.0%) had clicking in left TMJ, in which 4(66.7%) had sigmoid and 2(33.3%) had flattened shape, 2(13.3%) had clicking in right TMJ, in which 1(50.0%) had sigmoid and 1(50.0%) had box shape, 1(6.7%) had clicking in left and right TMJ, had sigmoid shape(100%), 1(6.7%) had crepitus in left TMJ, had flattened shape(100%) 1(6.7%) had crepitus in right TMJ had sigmoid shape(100%). The Correlation between auscultation and articular eminence morphology in left TMJ was insignificant with a P value of 0.349.

Table-30 and Graph- 30 shows the Correlation between auscultation and CCRT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 3(75%) had normal and 1(25%) had flattened condylar morphology, 6(40.0%) had clicking in left TMJ, in which 5(83.3%) had normal and 1(16.7%) had flattened condylar morphology, 2(13.3%) had clicking in right

TMJ, in which both(100%) of them had normal condylar morphlogy, 1(6.7%) had clicking in left and right TMJ, had normal condylar morphology(100%), 1(6.7%) had crepitus in left TMJ, had normal condylar morphology(100%), 1(6.7%) had crepitus in right TMJ, had flattened condylar morphology(100%). The Correlation between auscultation and condylar morphology in right TMJ was insignificant with a P value of 0.403.

Table-31 and Graph-31 shows the Correlation between auscultation and CCLT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 3(75%) had normal and 1(25%) had flattened condylar morphology, 6(40.0%) had clicking in left TMJ, in which 5(83.3%) had normal and 1(16.7%) had flattened condylar morphology, 2(13.3%) had clicking in right TMJ, in which both(100%) of them had normal condylar morphlogy, 1(6.7%) had clicking in left and right TMJ, had normal condylar morphology(100%), 1(6.7%) had crepitus in left TMJ, had normal condylar morphology(100%), 1(6.7%) had crepitus in right TMJ, had normal condylar morphology(100%).

The Correlation between auscultation and condylar morphology in left TMJ was insignificant with a P value of 0.935.

Table-32 and Graph- 32 shows the Correlation between auscultation and GTRT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 1(25%) had glenoid thickness of 1.0mm, 2(50%) had glenoid thickness of 1.9mm and 1(25%) had glenoid thickness of 2.0mm in right side. 6(40.0%) had clicking in left TMJ, 1(16.7%) had glenoid thickness of 1.2mm, 2(33.3%) had glenoid thickness of 1.7mm and 3(50%) had glenoid thickness of 1.8mm in right side. 2(13.3%) had clicking in right TMJ, in which 1(50%) had glenoid thickness of 1.3mm and in which 1(50%) had glenoid thickness of 1.5mm in right side. 1(6.7%) had clicking in left and right TMJ, who(100%) had glenoid thickness of 2.0mm in right side. 1(6.7%) had crepitus in left TMJ, who(100%) had glenoid thickness of 1.3mm in right side. 1(6.7%) had crepitus in right TMJ who(100%) had glenoid thickness of 1.0mm in right side. The Correlation between auscultation and glenoid thickness in right TMJ was insignificant with a P value of 0.120.

Table-33 and Graph- 33 shows the Correlation between auscultation and GTLT

In the total of 15(100%) subjects, 4(26.7%) did not have any sounds, in which 2(50%) had glenoid thickness of 1.5mm, 1(25%) had glenoid thickness of 1.8mm 1(25%) had glenoid thickness of 2.3mm in left side. 6(40.0%) had clicking in left TMJ, in which 1(16.7%) had glenoid thickness of 1.3mm 1(16.7%) had glenoid thickness of 1.4mm 1(16.7%) had glenoid thickness of 1.5mm and 3(50%) had glenoid thickness of 1.7mm in left side. 2(13.3%) had clicking in right TMJ, 1(50%) had glenoid thickness of 1.8mm 1(50%) had glenoid thickness of 1.6mm in left side. 1(6.7%) had clicking in left and right TMJ, who(100%) had glenoid thickness of 1.8mm in left side. 1(6.7%) had crepitus in left TMJ, who(100%) had glenoid thickness of 1.8mm in left side. 1(6.7%) had crepitus in right TMJ who(100%) had glenoid thickness of 1.3mm in left side. The Correlation between auscultation and glenoid thickness in left TMJ was insignificant with a P value of 0.408.

Table-34 and Graph-34 shows the Correlation between GTRT and GTLT

In the total of 15(100%) subjects, 2(13.3%) had right side glenoid thickness of 1.0mm in which, 1(50%) had glenoid thickness of 1.3mm and 1(50%) had glenoid thickness of 1.5mm in left side. 1(6.7%) had right side glenoid thickness of 1.2mm who (100%) had glenoid thickness of 1.3mm in left side. 2(13.3%) had right side glenoid thickness of 1.3mm in which both(100%) had glenoid thickness of 1.8mm in left side. 1(6.7%) had right side glenoid thickness of 1.5mm who (100%) had glenoid thickness of 1.6mm in left side. 2(13.3%) had right side glenoid thickness of 1.7mm in which, 1(50%) had glenoid thickness of 1.5mm and 1(50%) had glenoid thickness of 1.7mm in left side. 3(20%) had right side glenoid thickness of 1.8mm in which, 1(33.3%) had glenoid thickness of 1.4mm and 2(66.7%) had glenoid thickness of 1.7mm in left side. 2(13.3%) had right side glenoid thickness of 1.9mm in which, 1(50%) had glenoid thickness of 1.5mm and 1(50%) had glenoid thickness of 2.3mm in left side. 2(13.3%) had right side glenoid thickness of 2.0mm in which both(100%) had glenoid thickness of 1.8mm in left side. The Correlation between glenoid thickness in right TMJ and glenoid thickness in left TMJ was insignificant with a P value of 0.08.

Table-35 and Graph-35 shows the Correlation between CCRT and GTRT

In the total of 15(100%) subjects, 12 had normal condylar morphology of the right TMJ who had mean glenoid thickness of 1.6 and standard deviation of 0.33. The Correlation between condylar morphology and glenoid thickness in right TMJ was insignificant with a P value of 0.778.

Table-36 and Graph-36 shows the Correlation between CCLT and GTLT

In the total of 15(100%) subjects, 12 had normal condylar morphology of the left TMJ who had mean glenoid thickness of 1.69 and standard deviation of 0.26. The Correlation between condylar morphology and glenoid thickness in left TMJ was insignificant with a P value of 0.261.

Table 1: Distribution of subjects according to sex

Sex	Frequency	Percent
Male	3	20.0
Female	12	80.0
Total	15	100.0

Table 2: Distribution of subjects according to chief complaint

Chief complaint	Frequency	Percent
Pain left	7	46.7
Pain RT LT	1	6.7
Clicking left	2	13.3
Clicking right	2	13.3
Clicking RT LT	1	6.7
Pain & click right	1	6.7
Lock jaw	1	6.7
Total	15	100.0

Table 3: Distribution of subjects according to mouth opening

Mouth opening	Frequency	Percent
30-40	7	46.7
Above 40	8	53.3
Total	15	100.0

Table 4: Distribution of subjects according to deviation

Deviation	Frequency	Percent
Absent	9	60.0
Deviation right	2	13.3
Deviation left	4	26.7
Total	15	100.0

Table 5: Distribution of subjects according to palpatory findings

Palpation	Frequency	Percent
Pain left	2	13.3
Pain right	1	6.7
Pain right and left	1	6.7
Pain & Clicking left	6	40.0
Pain & Clicking right	2	13.3
Pain & Clicking right and left	1	6.7
Pain & Crepitus left	1	6.7
Pain & Crepitus right	1	6.7
Total	15	100.0

Table 6: Distribution of subjects according to auscultatory findings

Auscultation	Frequency	Percent
No sounds	4	26.7
Clicking left	6	40.0
Clicking right	2	13.3
Clicking right and left	1	6.7
Crepitus left	1	6.7
Crepitus right	1	6.7
Total	15	100.0

Table 7: Distribution of articular eminence morphology in right TMJ

AERT	Frequency	Percent
Sigmoid	12	80.0
Flattened	1	6.7
Box	1	6.7
Deformed	1	6.7
Total	15	100.0

Table 8: Distribution of articular eminence morphology in left TMJ

AELT	Frequency	Percent
Sigmoid	9	60.0
Flattened	3	20.0
Box	3	20.0
Total	15	100.0

Table 9: Distribution of condylar changes in right TMJ

CCRT	Frequency	Percent
Normal	12	80.0
Fattened	3	20.0
Total	15	100.0

Table 10: Distribution of condylar changes in left TMJ

CCLT	Frequency	Percent
Normal	12	80.0
Fattened	3	20.0
Total	15	100.0

Table 11: Correlation between chief complaint and mouth opening

Chief complaint		**Mouth opening**		**Total**
		30-40	Above 40	
Pain left	Count	5	2	7
	% within Chief complaint	71.4%	28.6%	100.0 %
	% within Mouth opening	71.4%	25.0%	46.7%
Pain RT LT	Count	0	1	1
	% within Chief complaint	.0%	100.0%	100.0 %
	% within Mouth opening	.0%	12.5%	6.7%
Clicking left	Count	0	2	2
	% within Chief complaint	.0%	100.0%	100.0 %
	% within Mouth opening	.0%	25.0%	13.3%
Clicking right	Count	0	2	2
	% within Chief complaint	.0%	100.0%	100.0 %
	% within Mouth opening	.0%	25.0%	13.3%
Clicking RT LT	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0 %
	% within Mouth opening	14.3%	.0%	6.7%
Pain & click right	Count	0	1	1
	% within Chief complaint	.0%	100.0%	100.0 %
	% within Mouth opening	.0%	12.5%	6.7%
Lock jaw	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0 %
	% within Mouth opening	14.3%	.0%	6.7%
Total	Count	7	8	15
	% within Chief complaint	46.7%	53.3%	100.0 %
	% within Mouth opening	100.0%	100.0%	100.0 %

P - 0.15

Table 12: Correlation between chief complaint and palpatory findings

Chief complaint	Palpation								Total
	Pain left	Pain right	Pain right & left	Pain & Click left	Pain & Click right	Pain & Click RT LT	Pain & Crepitus left	Pain & Crepitus right	
Pain left	2	0	0	4	0	0	1	0	7
	28.6%	.0%	.0%	57.1%	.0%	.0%	14.3%	.0%	100.0%
	100.0%	.0%	.0%	66.7%	.0%	.0%	100.0%	.0%	46.7%
Pain RT LT	0	0	1	0	0	0	0	0	1
	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	6.7%
Clicking left	0	0	0	2	0	0	0	0	2
	.0%	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	100.0%
	.0%	.0%	.0%	33.3%	.0%	.0%	.0%	.0%	13.3%
Clicking right	0	0	0	0	2	0	0	0	2
	.0%	.0%	.0%	.0%	100.0%	.0%	.0%	.0%	100.0%
	.0%	.0%	.0%	.0%	100.0%	.0%	.0%	.0%	13.3%
Clicking RT LT	0	0	0	0	0	1	0	0	1
	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	.0%	100.0%
	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	.0%	6.7%
Pain & click right	0	0	0	0	0	0	0	1	1
	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%	100.0%
	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%	6.7%
Lock jaw	0	1	0	0	0	0	0	0	1
	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
Total	2	1	1	6	2	1	1	1	15
	13.3%	6.7%	6.7%	40.0%	13.3%	6.7%	6.7%	6.7%	100.0%
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

P – 0.001

Table 13: Correlation between chief complaint and auscultatory findings

Chief complaint	Auscultation						Total
	No sounds	Clicking left	Clicking right	Clicking right & left	Crepitu s left	Crepit us right	
Pain left	2	4	0	0	1	0	7
	28.6%	57.1%	.0%	.0%	14.3%	.0%	100.0%
	50.0%	66.7%	.0%	.0%	100.0%	.0%	46.7%
Pain RT LT	1	0	0	0	0	0	1
	100.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	25.0%	.0%	.0%	.0%	.0%	.0%	6.7%
Clicking left	0	2	0	0	0	0	2
	.0%	100.0%	.0%	.0%	.0%	.0%	100.0%
	.0%	33.3%	.0%	.0%	.0%	.0%	13.3%
Clicking right	0	0	2	0	0	0	2
	.0%	.0%	100.0%	.0%	.0%	.0%	100.0%
	.0%	.0%	100.0%	.0%	.0%	.0%	13.3%
Clicking RT LT	0	0	0	1	0	0	1
	.0%	.0%	.0%	100.0%	.0%	.0%	100.0%
	.0%	.0%	.0%	100.0%	.0%	.0%	6.7%
Pain & click right	0	0	0	0	0	1	1
	.0%	.0%	.0%	.0%	.0%	100.0 %	100.0%
	.0%	.0%	.0%	.0%	.0%	100.0 %	6.7%
Lock jaw	1	0	0	0	0	0	1
	100.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	25.0%	.0%	.0%	.0%	.0%	.0%	6.7%
Total	4	6	2	1	1	1	15
	26.7%	40.0%	13.3%	6.7%	6.7%	6.7%	100.0%
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0 %	100.0%

P - 0.007

Table 14: Correlation between chief complaint and AERT

Chief complaint		AE- RT				Total
		Sigmoid	**Flattened**	**Box**	**Deformed**	
Pain left	Count	7	0	0	0	7
	% within Chief complaint	100.0%	.0%	.0%	.0%	100.0%
	% within AE- RT	58.3%	.0%	.0%	.0%	46.7%
Pain RT LT	Count	1	0	0	0	1
	% within Chief complaint	100.0%	.0%	.0%	.0%	100.0%
	% within AE- RT	8.3%	.0%	.0%	.0%	6.7%
Clicking left	Count	1	0	1	0	2
	% within Chief complaint	50.0%	.0%	50.0%	.0%	100.0%
	% within AE- RT	8.3%	.0%	100.0%	.0%	13.3%
Clicking right	Count	2	0	0	0	2
	% within Chief complaint	100.0%	.0%	.0%	.0%	100.0%
	% within AE- RT	16.7%	.0%	.0%	.0%	13.3%
Clicking RT LT	Count	1	0	0	0	1
	% within Chief complaint	100.0%	.0%	.0%	.0%	100.0%
	% within AE- RT	8.3%	.0%	.0%	.0%	6.7%
Pain & click right	Count	0	1	0	0	1
	% within Chief complaint	.0%	100.0%	.0%	.0%	100.0%
	% within AE- RT	.0%	100.0%	.0%	.0%	6.7%
Lock jaw	Count	0	0	0	1	1
	% within Chief complaint	.0%	.0%	.0%	100.0%	100.0%
	% within AE- RT	.0%	.0%	.0%	100.0%	6.7%
Total	Count	12	1	1	1	15
	% within Chief complaint	80.0%	6.7%	6.7%	6.7%	100.0%
	% within AE- RT	100.0%	100.0%	100.0%	100.0%	100.0%

P – 0.005

Table 15: Correlation between chief complaint and AELT

Chief complaint		**AE-LT**			**Total**
		Sigmoid	**Flattened**	**Box**	
Pain left	Count	3	3	1	7
	% within Chief complaint	42.9%	42.9%	14.3%	100.0%
	% within AE-LT	33.3%	100.0%	33.3%	46.7%
Pain RT LT	Count	1	0	0	1
	% within Chief complaint	100.0%	.0%	.0%	100.0%
	% within AE-LT	11.1%	.0%	.0%	6.7%
Clicking left	Count	2	0	0	2
	% within Chief complaint	100.0%	.0%	.0%	100.0%
	% within AE-LT	22.2%	.0%	.0%	13.3%
Clicking right	Count	1	0	1	2
	% within Chief complaint	50.0%	.0%	50.0%	100.0%
	% within AE-LT	11.1%	.0%	33.3%	13.3%
Clicking RT LT	Count	1	0	0	1
	% within Chief complaint	100.0%	.0%	.0%	100.0%
	% within AE-LT	11.1%	.0%	.0%	6.7%
Pain & click right	Count	1	0	0	1
	% within Chief complaint	100.0%	.0%	.0%	100.0%
	% within AE-LT	11.1%	.0%	.0%	6.7%
Lock jaw	Count	0	0	1	1
	% within Chief complaint	.0%	.0%	100.0%	100.0%
	% within AE-LT	.0%	.0%	33.3%	6.7%
Total	Count	9	3	3	15
	% within Chief complaint	60.0%	20.0%	20.0%	100.0%
	% within AE-LT	100.0%	100.0%	100.0%	100.0%

P – 0.53

Table 16: Correlation between chief complaint and CCRT

Chief complaint		CC-RT		Total
		Normal	Fattened	
Pain left	Count	7	0	7
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-RT	58.3%	.0%	46.7%
Pain RT LT	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-RT	8.3%	.0%	6.7%
Clicking left	Count	1	1	2
	% within Chief complaint	50.0%	50.0%	100.0%
	% within CC-RT	8.3%	33.3%	13.3%
Clicking right	Count	2	0	2
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-RT	16.7%	.0%	13.3%
Clicking RT LT	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-RT	8.3%	.0%	6.7%
Pain & click right	Count	0	1	1
	% within Chief complaint	.0%	100.0%	100.0%
	% within CC-RT	.0%	33.3%	6.7%
Lock jaw	Count	0	1	1
	% within Chief complaint	.0%	100.0%	100.0%
	% within CC-RT	.0%	33.3%	6.7%
Total	Count	12	3	15
	% within Chief complaint	80.0%	20.0%	100.0%
	% within CC-RT	100.0%	100.0%	100.0%

P – 0.065

Table 17: Correlation between chief complaint and CCLT

Chief complaint		CC-LT		Total
		Normal	**Fattened**	
Pain left	Count	6	1	7
	% within Chief complaint	85.7%	14.3%	100.0%
	% within CC-LT	50.0%	33.3%	46.7%
Pain RT LT	Count	0	1	1
	% within Chief complaint	.0%	100.0%	100.0%
	% within CC-LT	.0%	33.3%	6.7%
Clicking left	Count	1	1	2
	% within Chief complaint	50.0%	50.0%	100.0%
	% within CC-LT	8.3%	33.3%	13.3%
Clicking right	Count	2	0	2
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-LT	16.7%	.0%	13.3%
Clicking RT LT	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-LT	8.3%	.0%	6.7%
Pain & click right	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-LT	8.3%	.0%	6.7%
Lock jaw	Count	1	0	1
	% within Chief complaint	100.0%	.0%	100.0%
	% within CC-LT	8.3%	.0%	6.7%
Total	Count	12	3	15
	% within Chief complaint	80.0%	20.0%	100.0%
	% within CC-LT	100.0%	100.0%	100.0%

P – 0.368

Table 18: Correlation between mouth opening and CCLT

Mouthopening		CCLT Normal	CCLT Flattened	Total
31-40	Count	7	0	7
	% within mouthopening	100.0%	.0%	100.0%
	% within CCLT	53.8%	.0%	46.7%
above 40	Count	6	2	8
	% within mouthopening	75.0%	25.0%	100.0%
	% within CCLT	46.2%	100.0%	53.3%
Total	Count	13	2	15
	% within mouthopening	86.7%	13.3%	100.0%
	% within CCLT	100.0%	100.0%	100.0%

P - .267

Table 19: Correlation between mouth opening and CCRT

Mouthopening		CCRT Normal	CCRT Flattened	Total
31-40	Count	6	1	7
	% within mouthopening	85.7%	14.3%	100.0%
	% within CCRT	50.0%	33.3%	46.7%
above 40	Count	6	2	8
	% within mouthopening	75.0%	25.0%	100.0%
	% within CCRT	50.0%	66.7%	53.3%
Total	Count	12	3	15
	% within mouthopening	80.0%	20.0%	100.0%
	% within CCRT	100.0%	100.0%	100.0%

P - .554

Table 20: Correlation between deviation and CCLT

Deviation		CCRT		Total
		Normal	Flattened	
no sound	Count	8	1	9
	% within deviation	88.9%	11.1%	100.0%
	% within CCRT	66.7%	33.3%	60.0%
deviation rt	Count	2	0	2
	% within deviation	100.0%	.0%	100.0%
	% within CCRT	16.7%	.0%	13.3%
deviation lt	Count	2	2	4
	% within deviation	50.0%	50.0%	100.0%
	% within CCRT	16.7%	66.7%	26.7%
Total	Count	12	3	15
	% within deviation	80.0%	20.0%	100.0%
	% within CCRT	100.0%	100.0%	100.0%

P - .202

Table 21: Correlation between deviation and CCRT

Deviation		**CCLT**		**Total**
		Normal	**Flattened**	
no sound	Count	7	2	9
	% within deviation	77.8%	22.2%	100.0%
	% within CCLT	53.8%	100.0%	60.0%
deviation rt	Count	2	0	2
	% within deviation	100.0%	.0%	100.0%
	% within CCLT	15.4%	.0%	13.3%
deviation lt	Count	4	0	4
	% within deviation	100.0%	.0%	100.0%
	% within CCLT	30.8%	.0%	26.7%
Total	Count	13	2	15
	% within deviation	86.7%	13.3%	100.0%
	% within CCLT	100.0%	100.0%	100.0%

P - .463

Table 22: Correlation between palpation and AERT

Palpation		AERT				Total
		Sigmoid	**Flattened**	**Box**	**Deformed**	
pain lt	Count	2	0	0	0	2
	% within palpation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	16.7%	.0%	.0%	.0%	13.3%
pain rt	Count	0	0	0	1	1
	% within palpation	.0%	.0%	.0%	100.0%	100.0%
	% within AERT	.0%	.0%	.0%	100.0%	6.7%
pain rt, lt	Count	1	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	8.3%	.0%	.0%	.0%	6.7%
pain, clicking lt	Count	5	0	1	0	6
	% within palpation	83.3%	.0%	16.7%	.0%	100.0%
	% within AERT	41.7%	.0%	100.0%	.0%	40.0%
pain,clicking rt	Count	2	0	0	0	2
	% within palpation	100.0%	.0%	.0%	.0%	100.0%
	% within aert	16.7%	.0%	.0%	.0%	13.3%
pain,clickingrt,lt	Count	1	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	8.3%	.0%	.0%	.0%	6.7%
pain,crepitus lt	Count	1	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	8.3%	.0%	.0%	.0%	6.7%
pain,crepitus rt	Count	0	1	0	0	1
	% within palpation	.0%	100.0%	.0%	.0%	100.0%
	% within AERT	.0%	100.0%	.0%	.0%	6.7%
Total	Count	12	1	1	1	15
	% within palpation	80.0%	6.7%	6.7%	6.7%	100.0%
	% within AERT	100.0%	100.0%	100.0%	100.0%	100.0%

P - .066

Table 23: Correlation between palpation and AELT

Palpation		AELT			Total
		Sigmoid	Flattened	Box	
pain lt	Count	1	0	1	2
	% within palpation	50.0%	.0%	50.0%	100.0%
	% within AELT	11.1%	.0%	33.3%	13.3%
pain rt	Count	0	0	1	1
	% within palpation	.0%	.0%	100.0%	100.0%
	% within AELT	.0%	.0%	33.3%	6.7%
pain rt, lt	Count	1	0	0	1
	% within palpation	100.0%	.0%	.0%	100.0%
	% within AELT	11.1%	.0%	.0%	6.7%
pain, clicking lt	Count	4	2	0	6
	% within palpation	66.7%	33.3%	.0%	100.0%
	% within AELT	44.4%	66.7%	.0%	40.0%
pain,clicking rt	Count	1	0	1	2
	% within palpation	50.0%	.0%	50.0%	100.0%
	% within AELT	11.1%	.0%	33.3%	13.3%
pain,clickingrt, lt	Count	1	0	0	1
	% within palpation	100.0%	.0%	.0%	100.0%
	% within AELT	11.1%	.0%	.0%	6.7%
pain,crepitus lt	Count	0	1	0	1
	% within palpation	.0%	100.0%	.0%	100.0%
	% within AELT	.0%	33.3%	.0%	6.7%
pain,crepitus rt	Count	1	0	0	1
	% within palpation	100.0%	.0%	.0%	100.0%
	% within AELT	11.1%	.0%	.0%	6.7%
Total	Count	9	3	3	15
	% within palpation	60.0%	20.0%	20.0%	100.0%
	% within AELT	100.0%	100.0%	100.0%	100.0%

P - .417

Table 24: Correlation between palpation and CCRT

Palpation		CCRT		Total
		Normal	Flattened	
pain lt	Count	2	0	2
	% within palpation	100.0%	.0%	100.0%
	% within CCRT	16.7%	.0%	13.3%
pain rt	Count	0	1	1
	% within palpation	.0%	100.0%	100.0%
	% within CCRT	.0%	33.3%	6.7%
pain rt, lt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCRT	8.3%	.0%	6.7%
pain, clicking lt	Count	5	1	6
	% within palpation	83.3%	16.7%	100.0%
	% within CCRT	41.7%	33.3%	40.0%
pain,clicking rt	Count	2	0	2
	% within palpation	100.0%	.0%	100.0%
	% within CCRT	16.7%	.0%	13.3%
pain,clickingrt,lt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCRT	8.3%	.0%	6.7%
pain,crepitus lt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCRT	8.3%	.0%	6.7%
pain,crepitus rt	Count	0	1	1
	% within palpation	.0%	100.0%	100.0%
	% within CCRT	.0%	33.3%	6.7%
Total	Count	12	3	15
	% within palpation	80.0%	20.0%	100.0%
	% within CCRT	100.0%	100.0%	100.0%

P - .201

Table 25: Correlation between palpation and CCLT

Palpation		CCLT		Total
		Normal	**Flattened**	
pain lt	Count	2	0	2
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	15.4%	.0%	13.3%
pain rt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
pain rt, lt	Count	0	1	1
	% within palpation	.0%	100.0%	100.0%
	% within CCLT	.0%	50.0%	6.7%
pain, clicking lt	Count	5	1	6
	% within palpation	83.3%	16.7%	100.0%
	% within CCLT	38.5%	50.0%	40.0%
pain,clicking rt	Count	2	0	2
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	15.4%	.0%	13.3%
pain,clickingrt,lt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
pain,crepitus lt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
pain,crepitus rt	Count	1	0	1
	% within palpation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
Total	Count	13	2	15
	% within palpation	86.7%	13.3%	100.0%
	% within CCLT	100.0%	100.0%	100.0%

P - .352

Table 26: Correlation between palpation and GTRT

Palpation		GTRT								Total
		1.0	1.2	1.3	1.5	1.7	1.8	1.9	2.0	
pain lt	Count	0	0	0	0	0	0	1	1	2
	% within palpation	.0%	.0%	.0%	.0%	.0%	.0%	50.0%	50.0%	100.0%
	% within ggrt	.0%	.0%	.0%	.0%	.0%	.0%	50.0%	50.0%	13.3%
pain rt	Count	0	0	0	0	0	0	1	0	1
	% within palpation	.0%	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	100.0%
	% within ggrt	.0%	.0%	.0%	.0%	.0%	.0%	50.0%	.0%	6.7%
pain rt, lt	Count	1	0	0	0	0	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
pain, clicking lt	Count	0	1	0	0	2	3	0	0	6
	% within palpation	.0%	16.7%	.0%	.0%	33.3%	50.0%	.0%	.0%	100.0%
	% within ggrt	.0%	100.0%	.0%	.0%	100.0%	100.0%	.0%	.0%	40.0%
pain,clicking rt	Count	0	0	1	1	0	0	0	0	2
	% within palpation	.0%	.0%	50.0%	50.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	.0%	.0%	50.0%	100.0%	.0%	.0%	.0%	.0%	13.3%
pain,clicking rt,lt	Count	0	0	0	0	0	0	0	1	1
	% within palpation	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%	100.0%
	% within ggrt	.0%	.0%	.0%	.0%	.0%	.0%	.0%	50.0%	6.7%
pain,crepitus lt	Count	0	0	1	0	0	0	0	0	1
	% within palpation	.0%	.0%	100.0 %	.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	.0%	.0%	50.0%	.0%	.0%	.0%	.0%	.0%	6.7%
pain,crepitus rt	Count	1	0	0	0	0	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
total	Count	2	1	2	1	2	3	2	2	15
	% within palpation	13.3%	6.7%	13.3%	6.7%	13.3%	20.0%	13.3%	13.3%	100.0%
	% within ggrt	100.0%	100.0%	100.0 %	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

P - .222

Table 27: Correlation between palpation and GTLT

Palpation		GTLT							Total
		1.3	1.4	1.5	1.6	1.7	1.8	2.3	
pain lt	Count	0	0	0	0	0	1	1	2
	% within palpation	.0%	.0%	.0%	.0%	.0%	50.0%	50.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	25.0%	100.0 %	13.3%
pain rt	Count	0	0	1	0	0	0	0	1
	% within palpation	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	.0%	.0%	33.3%	.0%	.0%	.0%	.0%	6.7%
pain rt, lt	Count	0	0	1	0	0	0	0	1
	% within palpation	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	.0%	.0%	33.3%	.0%	.0%	.0%	.0%	6.7%
pain, clicking lt	Count	1	1	1	0	3	0	0	6
	% within palpation	16.7%	16.7%	16.7%	.0%	50.0%	.0%	.0%	100.0%
	% within gglt	50.0%	100.0%	33.3%	.0%	100.0%	.0%	.0%	40.0%
pain,clicking rt	Count	0	0	0	1	0	1	0	2
	% within palpation	.0%	.0%	.0%	50.0%	.0%	50.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	100.0%	.0%	25.0%	.0%	13.3%
pain,clicking rt,lt	Count	0	0	0	0	0	1	0	1
	% within palpation	.0%	.0%	.0%	.0%	.0%	100.0 %	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	25.0%	.0%	6.7%
pain,crepitus lt	Count	0	0	0	0	0	1	0	1
	% within palpation	.0%	.0%	.0%	.0%	.0%	100.0 %	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	25.0%	.0%	6.7%
pain,crepitus rt	Count	1	0	0	0	0	0	0	1
	% within palpation	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
Total	Count	2	1	3	1	3	4	1	15
	% within palpation	13.3%	6.7%	20.0%	6.7%	20.0%	26.7%	6.7%	100.0%
	% within gglt	100.0%	100.0%	100.0%	100.0%	100.0%	100.0 %	100.0 %	100.0%

P - .522

Table 28: Correlation between auscultation and AERT

Auscultation		AERT				Total
		Sigmoid	Flattened	Box	Deformed	
no sound	Count	3	0	0	1	4
	% within auscultation	75.0%	.0%	.0%	25.0%	100.0%
	% within AERT	25.0%	.0%	.0%	100.0%	26.7%
clicking lt	Count	5	0	1	0	6
	% within auscultation	83.3%	.0%	16.7%	.0%	100.0%
	% within AERT	41.7%	.0%	100.0%	.0%	40.0%
clicking rt	Count	2	0	0	0	2
	% within auscultation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	16.7%	.0%	.0%	.0%	13.3%
clicking rt,lt	Count	1	0	0	0	1
	% within auscultation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	8.3%	.0%	.0%	.0%	6.7%
crepitus lt	Count	1	0	0	0	1
	% within auscultation	100.0%	.0%	.0%	.0%	100.0%
	% within AERT	8.3%	.0%	.0%	.0%	6.7%
crepitus rt	Count	0	1	0	0	1
	% within auscultation	.0%	100.0%	.0%	.0%	100.0%
	% within AERT	.0%	100.0%	.0%	.0%	6.7%
Total	Count	12	1	1	1	15
	% within auscultation	80.0%	6.7%	6.7%	6.7%	100.0%
	% within AERT	100.0%	100.0%	100.0%	100.0%	100.0%

P - .202

Table 29: Correlation between auscultation and AELT

Auscultation		AELT			Total
		Sigmoid	**Flattened**	**Box**	
no sound	Count	2	0	2	4
	% within auscultation	50.0%	.0%	50.0%	100.0%
	% within AELT	22.2%	.0%	66.7%	26.7%
clicking lt	Count	4	2	0	6
	% within auscultation	66.7%	33.3%	.0%	100.0%
	% within AELT	44.4%	66.7%	.0%	40.0%
clicking rt	Count	1	0	1	2
	% within auscultation	50.0%	.0%	50.0%	100.0%
	% within AELT	11.1%	.0%	33.3%	13.3%
clicking rt,lt	Count	1	0	0	1
	% within auscultation	100.0%	.0%	.0%	100.0%
	% within AELT	11.1%	.0%	.0%	6.7%
crepitus lt	Count	0	1	0	1
	% within auscultation	.0%	100.0%	.0%	100.0%
	% within AELT	.0%	33.3%	.0%	6.7%
crepitus rt	Count	1	0	0	1
	% within auscultation	100.0%	.0%	.0%	100.0%
	% within AELT	11.1%	.0%	.0%	6.7%
Total	Count	9	3	3	15
	% within auscultation	60.0%	20.0%	20.0%	100.0%
	% within AELT	100.0%	100.0%	100.0%	100.0%

P - .349

Table 30: Correlation between auscultation and CCRT

Auscultation		CCRT		Total
		Normal	Flattened	
no sound	Count	3	1	4
	% within auscultation	75.0%	25.0%	100.0%
	% within CCRT	25.0%	33.3%	26.7%
clicking lt	Count	5	1	6
	% within auscultation	83.3%	16.7%	100.0%
	% within CCRT	41.7%	33.3%	40.0%
clicking rt	Count	2	0	2
	% within auscultation	100.0%	.0%	100.0%
	% within CCRT	16.7%	.0%	13.3%
clicking rt,lt	Count	1	0	1
	% within auscultation	100.0%	.0%	100.0%
	% within CCRT	8.3%	.0%	6.7%
crepitus lt	Count	1	0	1
	% within auscultation	100.0%	.0%	100.0%
	% within CCRT	8.3%	.0%	6.7%
crepitus rt	Count	0	1	1
	% within auscultation	.0%	100.0%	100.0%
	% within CCRT	.0%	33.3%	6.7%
Total	Count	12	3	15
	% within auscultation	80.0%	20.0%	100.0%
	% within CCRT	100.0%	100.0%	100.0%

P - .403

Table 31: Correlation between auscultation and CCLT

Auscultation		CCLT		Total
		Normal	Flattened	
no sound	Count	3	1	4
	% within auscultation	75.0%	25.0%	100.0%
	% within CCLT	23.1%	50.0%	26.7%
clicking lt	Count	5	1	6
	% within auscultation	83.3%	16.7%	100.0%
	% within CCLT	38.5%	50.0%	40.0%
clicking rt	Count	2	0	2
	% within auscultation	100.0%	.0%	100.0%
	% within CCLT	15.4%	.0%	13.3%
clicking rt,lt	Count	1	0	1
	% within auscultation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
crepitus lt	Count	1	0	1
	% within auscultation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
crepitus rt	Count	1	0	1
	% within auscultation	100.0%	.0%	100.0%
	% within CCLT	7.7%	.0%	6.7%
Total	Count	13	2	15
	% within auscultation	86.7%	13.3%	100.0%
	% within CCLT	100.0%	100.0%	100.0%

P - .935

Table 32: Correlation between auscultation and GTRT

Auscultation		GTRT								Total
		1.0	**1.2**	**1.3**	**1.5**	**1.7**	**1.8**	**1.9**	**2.0**	
no sound	Count	1	0	0	0	0	0	2	1	4
	% within auscultation	25.0%	.0%	.0%	.0%	.0%	.0%	50.0%	25.0%	100.0%
	% within ggrt	50.0%	.0%	.0%	.0%	.0%	.0%	100.0%	50.0%	26.7%
clicking lt	Count	0	1	0	0	2	3	0	0	6
	% within auscultation	.0%	16.7 %	.0%	.0%	33.3%	50.0%	.0%	.0%	100.0%
	% within ggrt	.0%	100. 0%	.0%	.0%	100.0%	100.0%	.0%	.0%	40.0%
clicking rt	Count	0	0	1	1	0	0	0	0	2
	% within auscultation	.0%	.0%	50.0%	50.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	.0%	.0%	50.0%	100.0 %	.0%	.0%	.0%	.0%	13.3%
clicking rt,lt	Count	0	0	0	0	0	0	0	1	1
	% within auscultation	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0 %	100.0%
	% within ggrt	.0%	.0%	.0%	.0%	.0%	.0%	.0%	50.0%	6.7%
crepitus lt	Count	0	0	1	0	0	0	0	0	1
	% within auscultation	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	.0%	.0%	50.0%	.0%	.0%	.0%	.0%	.0%	6.7%
crepitus rt	Count	1	0	0	0	0	0	0	0	1
	% within auscultation	100.0 %	.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within ggrt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
total	Count	2	1	2	1	2	3	2	2	15
	% within auscultation	13.3%	6.7%	13.3%	6.7%	13.3%	20.0%	13.3%	13.3%	100.0%
	% within ggrt	100.0 %	100. 0%	100.0%	100.0 %	100.0%	100.0%	100.0%	100.0 %	100.0%

P - .120

Table 33: Correlation between auscultation and GTLT

Auscultation		GTLT							Total
		1.3	**1.4**	**1.5**	**1.6**	**1.7**	**1.8**	**2.3**	
no sound	Count	0	0	2	0	0	1	1	4
	% within auscultation	.0%	.0%	50.0%	.0%	.0%	25.0%	25.0%	100.0%
	% within gglt	.0%	.0%	66.7%	.0%	.0%	25.0%	100.0%	26.7%
clicking lt	Count	1	1	1	0	3	0	0	6
	% within auscultation	16.7%	16.7%	16.7%	.0%	50.0%	.0%	.0%	100.0%
	% within gglt	50.0%	100.0%	33.3%	.0%	100.0%	.0%	.0%	40.0%
clicking rt	Count	0	0	0	1	0	1	0	2
	% within auscultation	.0%	.0%	.0%	50.0%	.0%	50.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	100.0%	.0%	25.0%	.0%	13.3%
clicking rt,lt	Count	0	0	0	0	0	1	0	1
	% within auscultation	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	25.0%	.0%	6.7%
crepitus lt	Count	0	0	0	0	0	1	0	1
	% within auscultation	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	25.0%	.0%	6.7%
crepitus rt	Count	1	0	0	0	0	0	0	1
	% within auscultation	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
total	Count	2	1	3	1	3	4	1	15
	% within auscultation	13.3%	6.7%	20.0%	6.7%	20.0%	26.7%	6.7%	100.0%
	% within gglt	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

P - .408

Table 34: Correlation between GGRT and GTLT

GTRT		GTLT							Total
		1.3	**1.4**	**1.5**	**1.6**	**1.7**	**1.8**	**2.3**	
1.0	Count	1	0	1	0	0	0	0	2
	% within ggrt	50.0%	.0%	50.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	50.0%	.0%	33.3%	.0%	.0%	.0%	.0%	13.3%
1.2	Count	1	0	0	0	0	0	0	1
	% within ggrt	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	% within gglt	50.0%	.0%	.0%	.0%	.0%	.0%	.0%	6.7%
1.3	Count	0	0	0	0	0	2	0	2
	% within ggrt	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	50.0%	.0%	13.3%
1.5	Count	0	0	0	1	0	0	0	1
	% within ggrt	.0%	.0%	.0%	100.0%	.0%	.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	100.0%	.0%	.0%	.0%	6.7%
1.7	Count	0	0	1	0	1	0	0	2
	% within ggrt	.0%	.0%	50.0%	.0%	50.0%	.0%	.0%	100.0%
	% within gglt	.0%	.0%	33.3%	.0%	33.3%	.0%	.0%	13.3%
1.8	Count	0	1	0	0	2	0	0	3
	% within ggrt	.0%	33.3%	.0%	.0%	66.7%	.0%	.0%	100.0%
	% within gglt	.0%	100.0%	.0%	.0%	66.7%	.0%	.0%	20.0%
1.9	Count	0	0	1	0	0	0	1	2
	% within ggrt	.0%	.0%	50.0%	.0%	.0%	.0%	50.0%	100.0%
	% within gglt	.0%	.0%	33.3%	.0%	.0%	.0%	100.0%	13.3%
2.0	Count	0	0	0	0	0	2	0	2
	% within ggrt	.0%	.0%	.0%	.0%	.0%	100.0%	.0%	100.0%
	% within gglt	.0%	.0%	.0%	.0%	.0%	50.0%	.0%	13.3%
Total	Count	2	1	3	1	3	4	1	15
	% within ggrt	13.3%	6.7%	20.0%	6.7%	20.0%	26.7%	6.7%	100.0%
	% within gglt	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

P – 0.008

Table 35: Correlation between CCRT and GTRT

	CC-RT	N	Mean	Std, Deviation
GT-RT	Normal	12	1.600	.3330
	Fattened	3	1.533	.4726

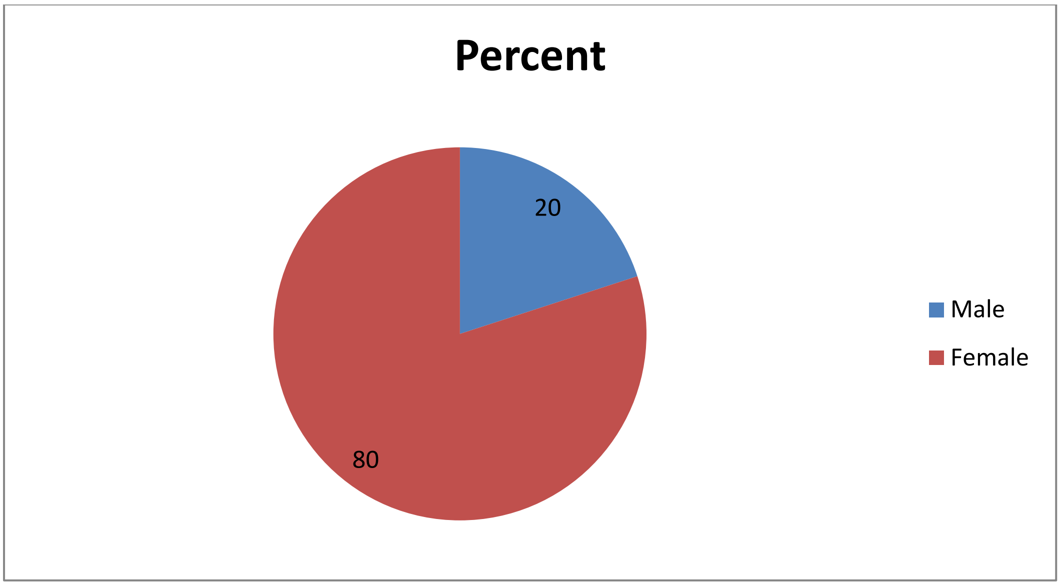
P – 0.778

Table 36: Correlation between CCLT and GTLT

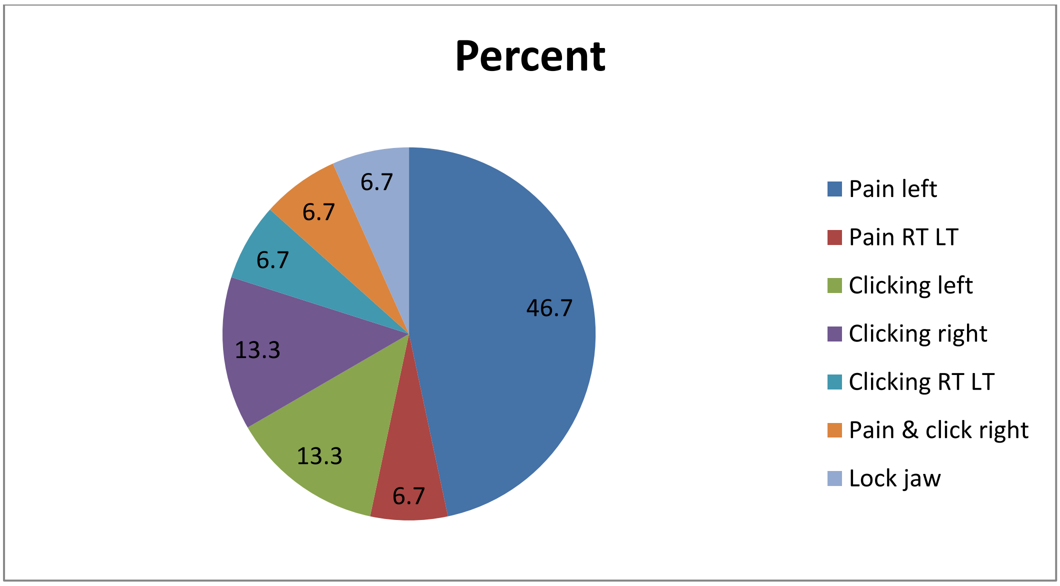
	CC-RT	N	Mean	Std, Deviation
GT-RT	Normal	12	1.692	.2610
	Fattened	3	1.500	.2000

P– 0.261

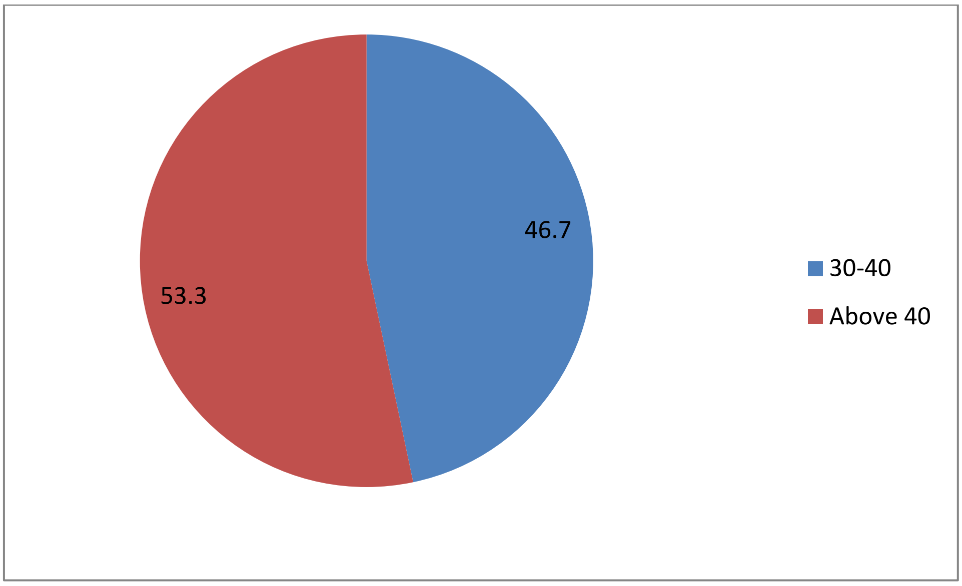
Graph-1: Distribution of subjects according to sex



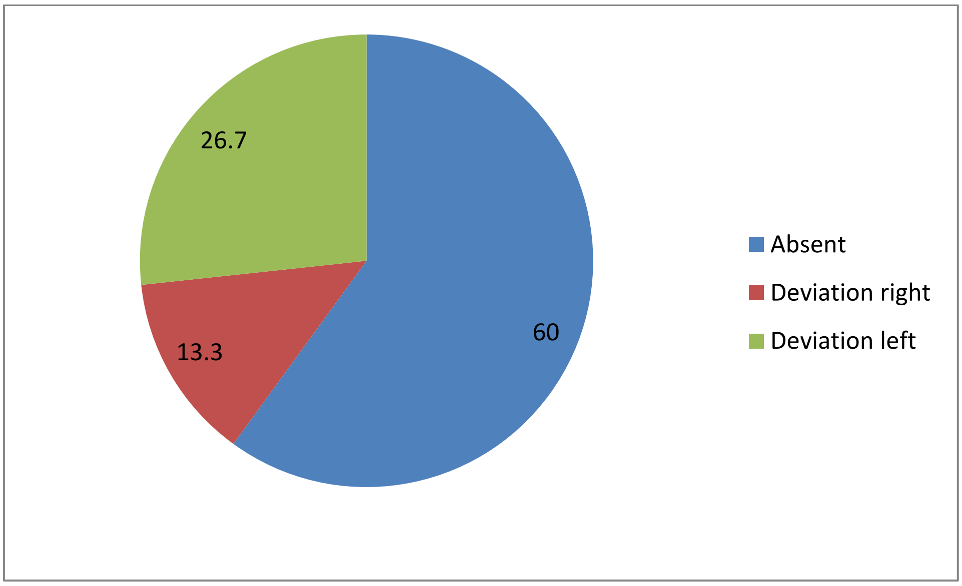
Graph-2: Distribution of subjects according to chief complaint



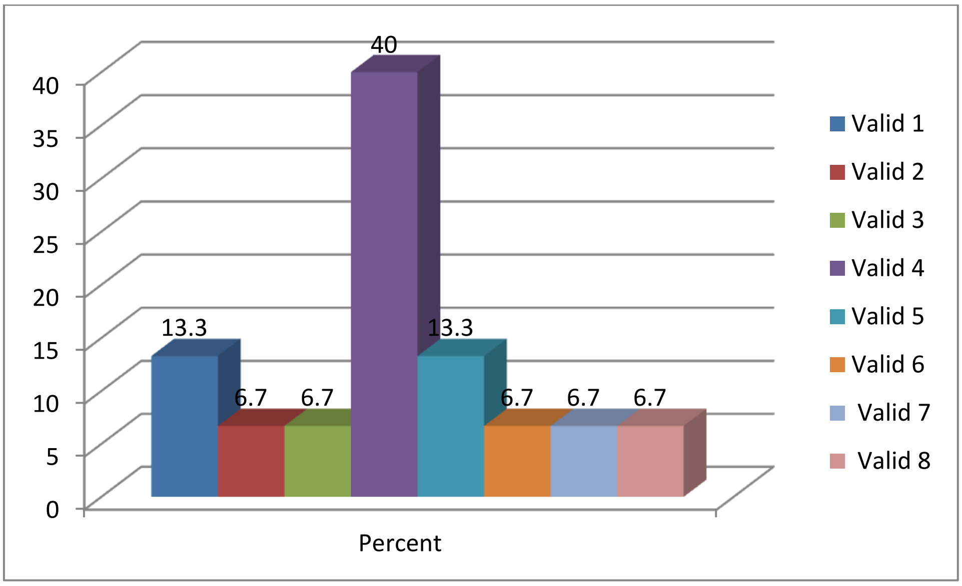
Graph-3: Distribution of subjects according to mouth opening



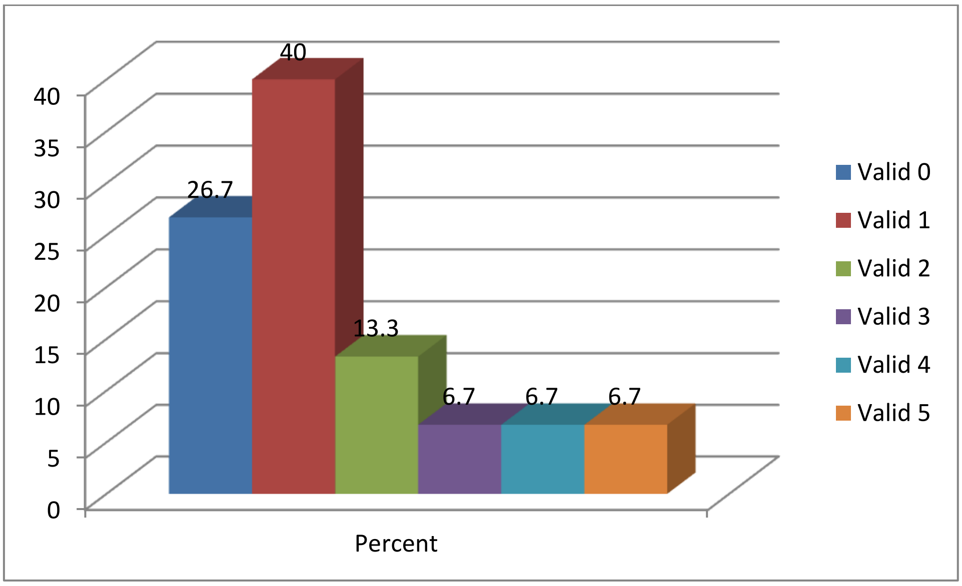
Graph-4: Distribution of subjects according to deviation



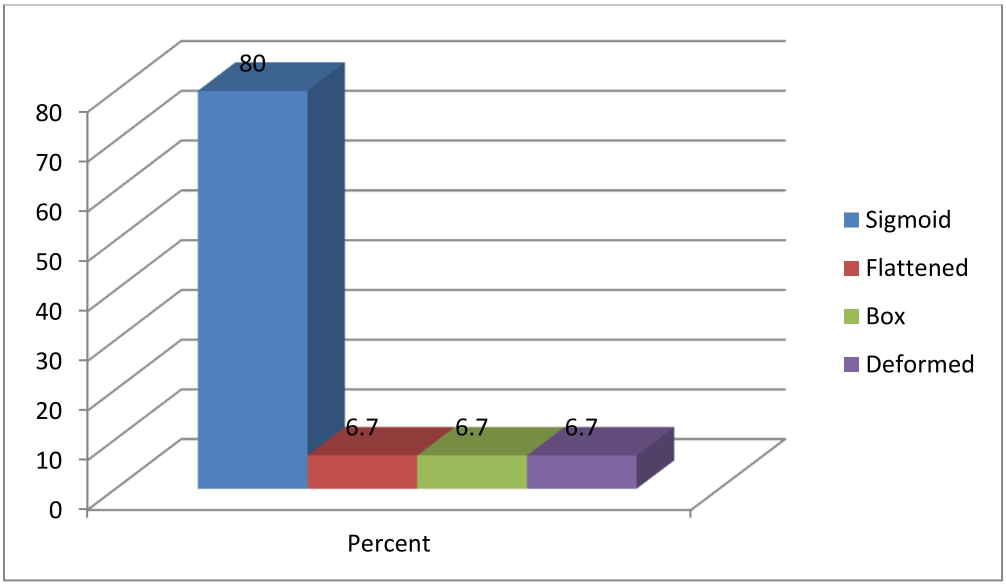
Graph-5: Distribution of subjects according to palpatory findings



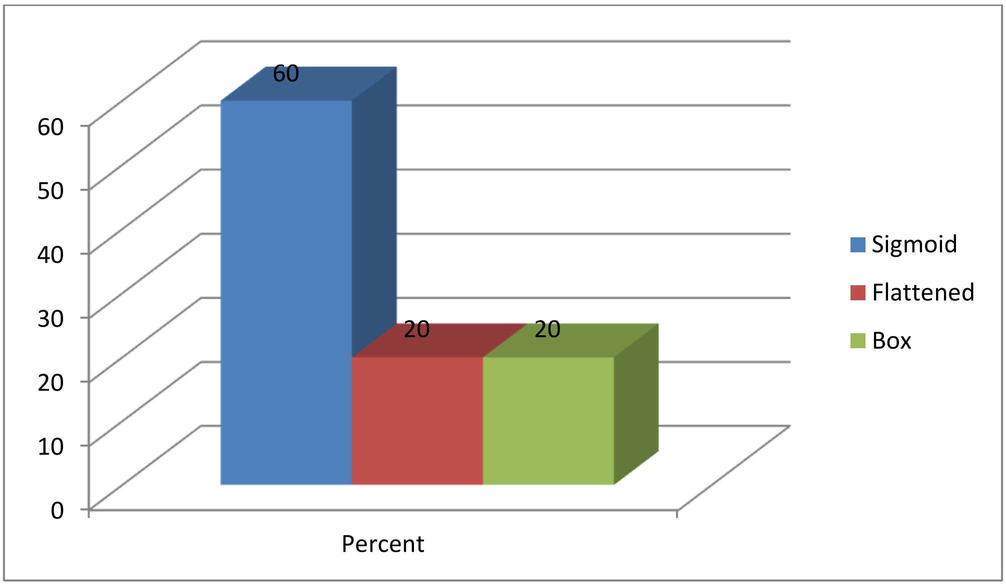
Graph-6: Distribution of subjects according to auscultatory findings



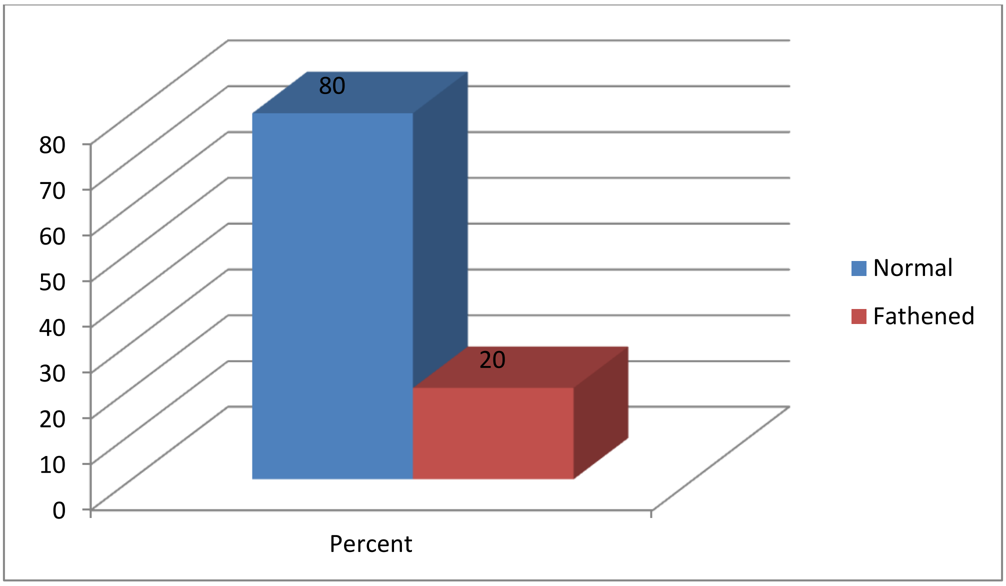
Graph-7: Distribution of articular eminence morphology in right TMJ



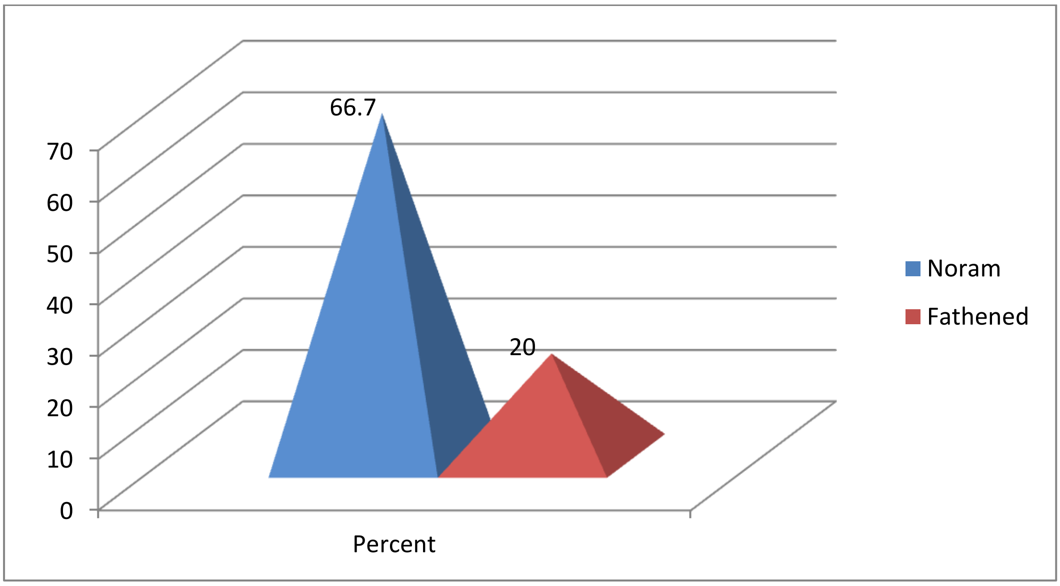
Graph-8: Distribution of articular eminence morphology in left TMJ



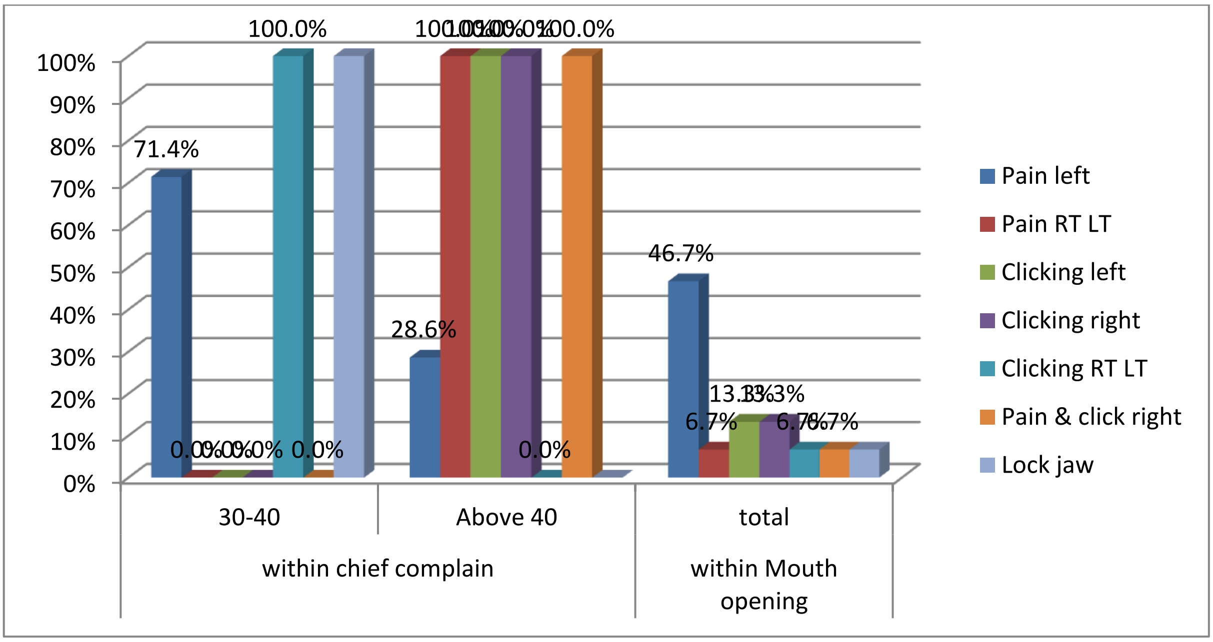
Graph-9: Distribution of condylar changes in right TMJ



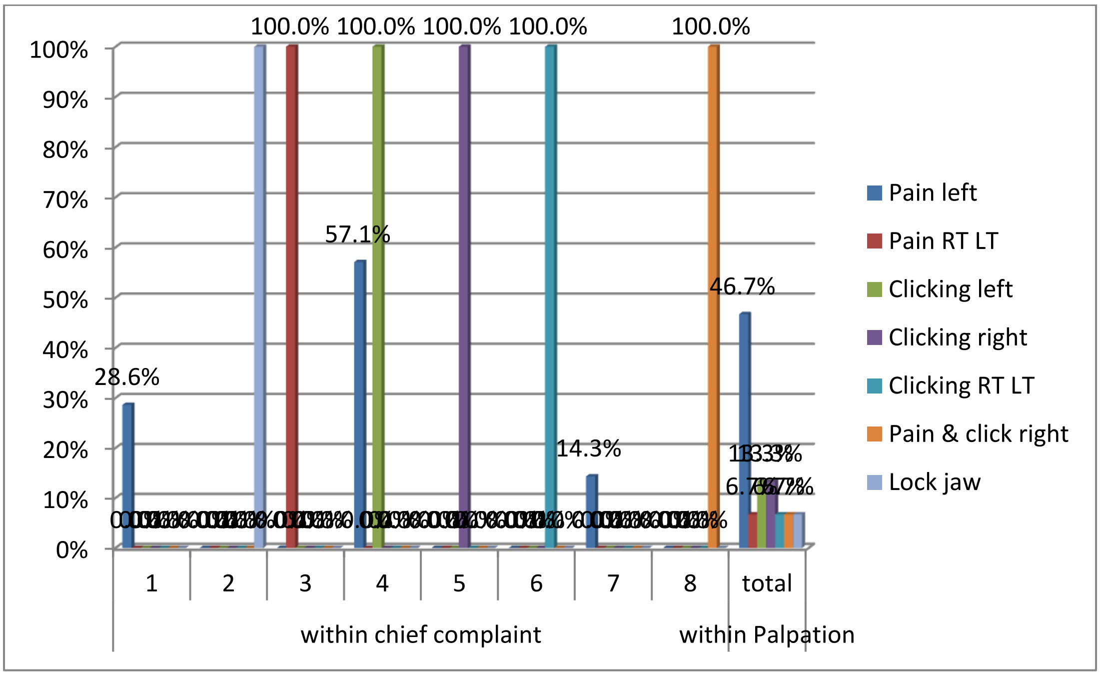
Graph-10: Distribution of condylar changes in left TMJ



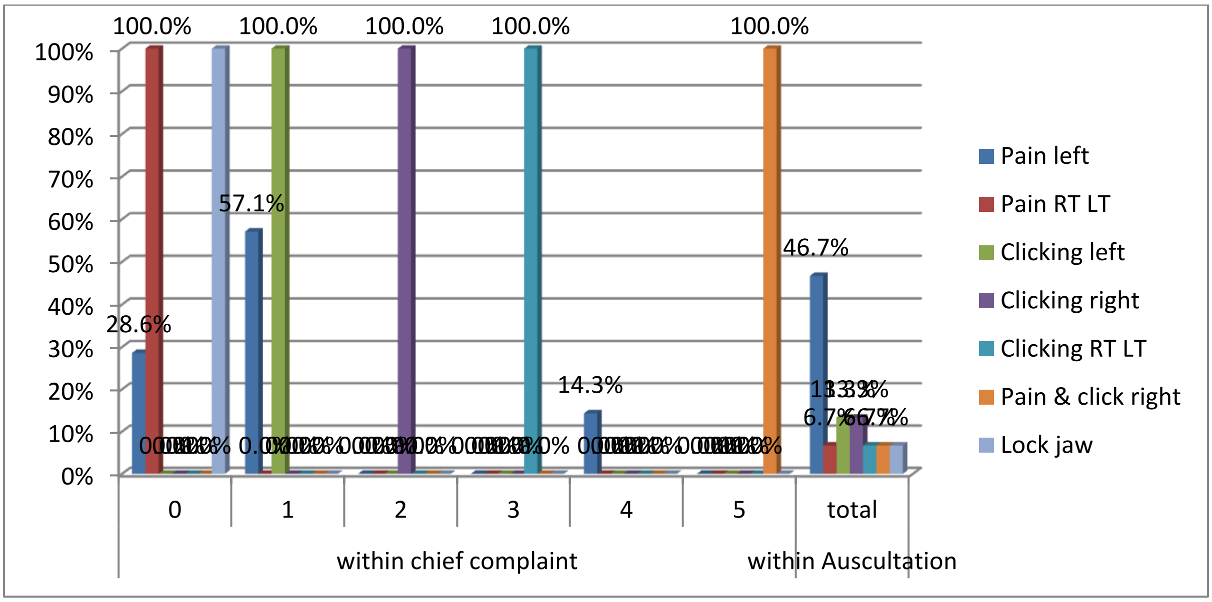
Graph-11: Correlation between chief complaint and mouth opening



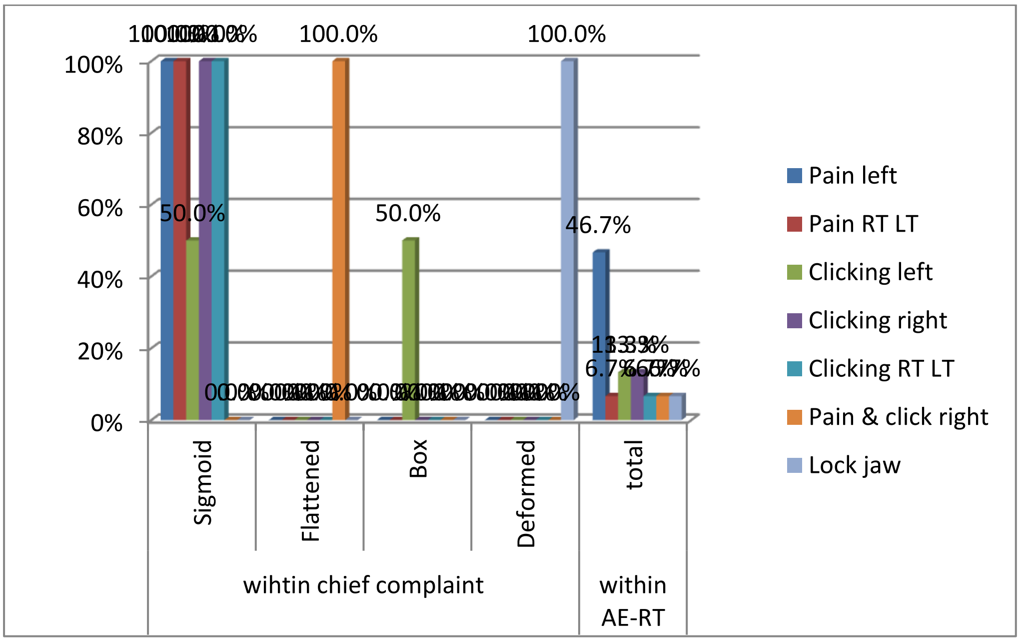
Graph-12: Correlation between chief complaint and palpatory findings



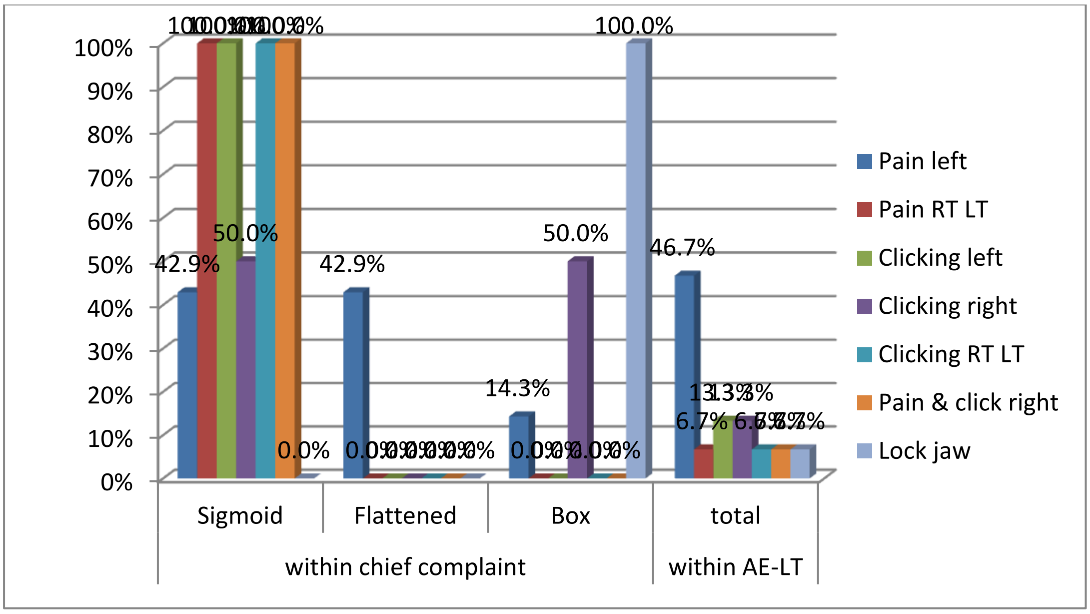
Graph-13: Correlation between chief complaint and auscultatory findings



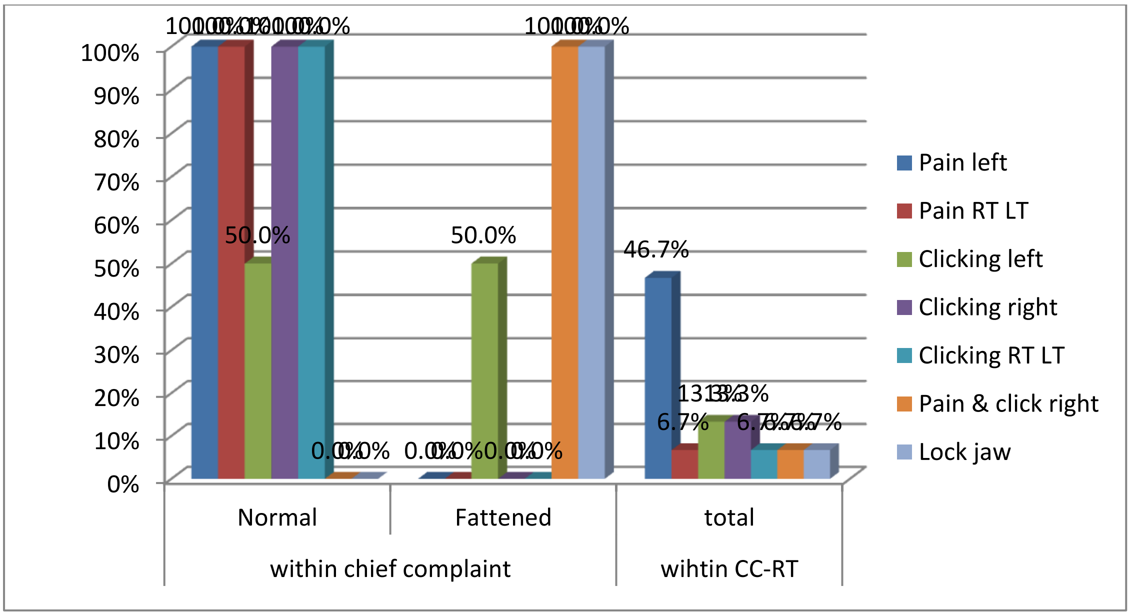
Graph-14: Correlation between chief complaint and AERT



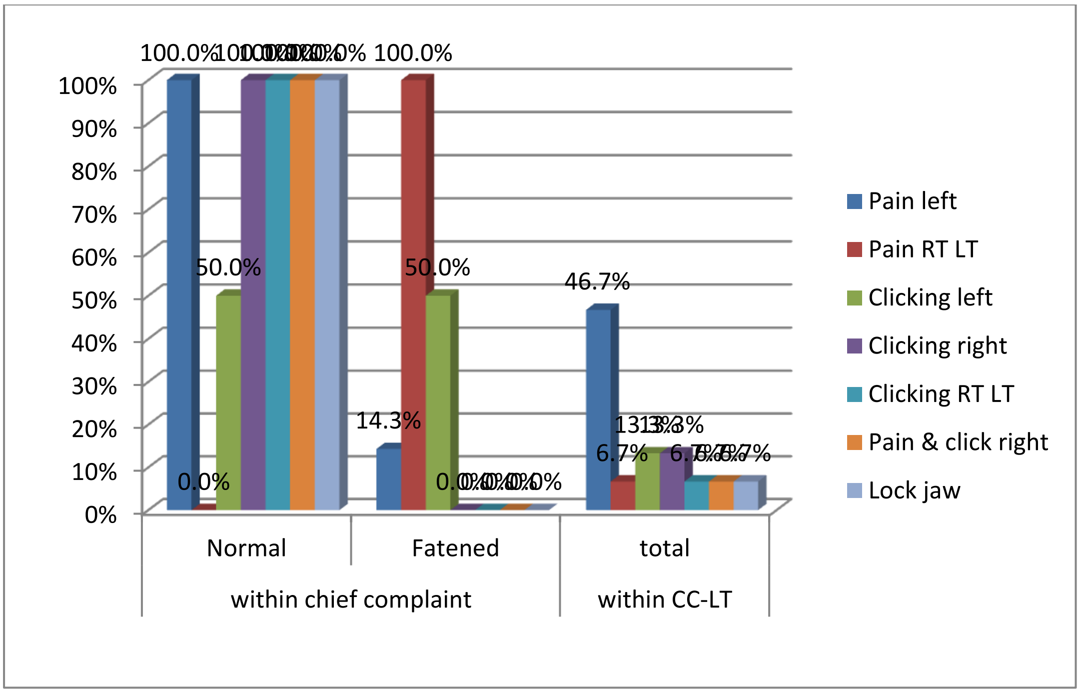
Graph-15: Correlation between chief complaint and AELT



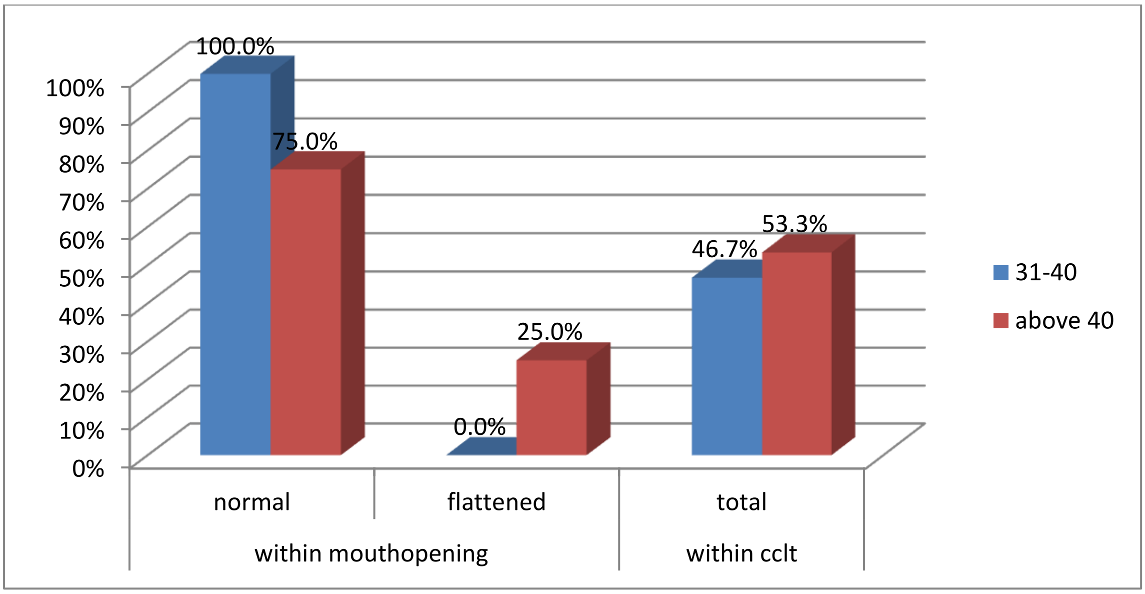
Graph-16: Correlation between chief complaint and CCRT



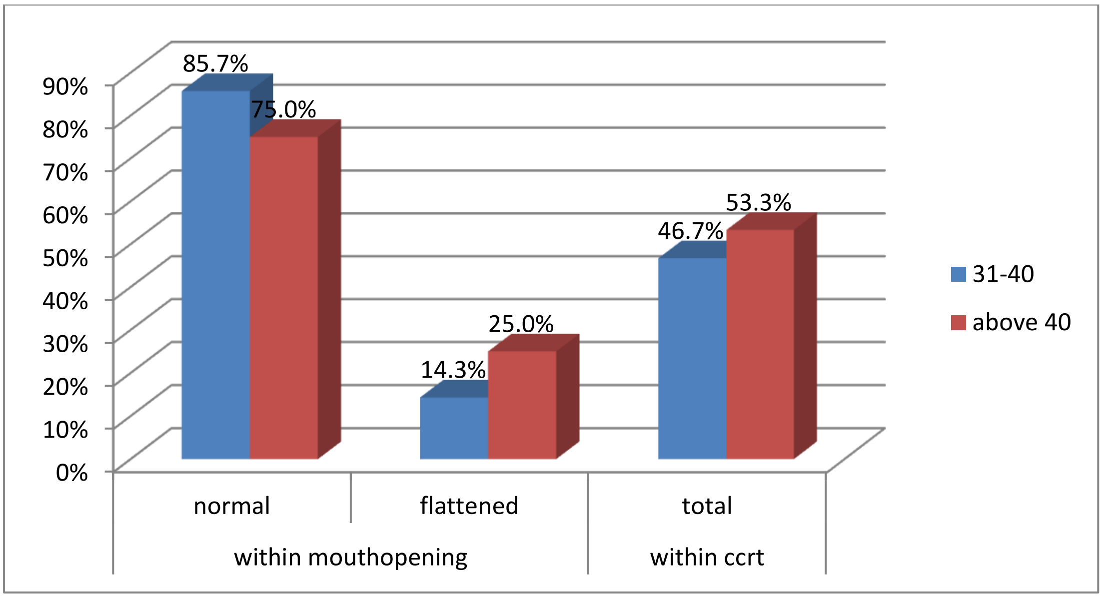
Graph-17: Correlation between chief complaint and CCLT



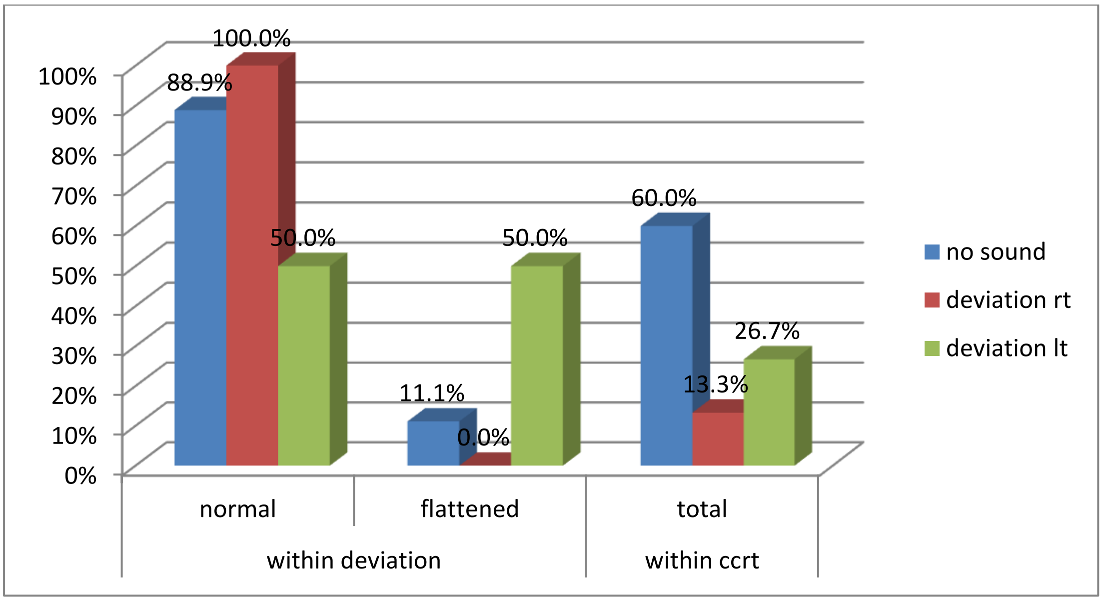
Graph-18: Correlation between mouth opening and CCLT



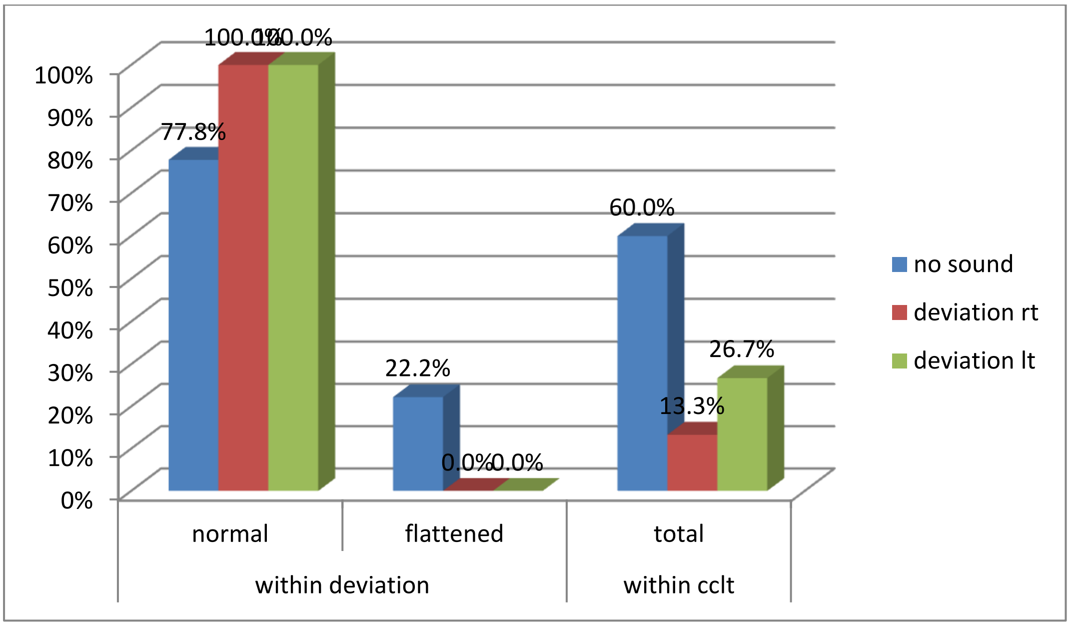
Graph-19: Correlation between mouth opening and CCRT



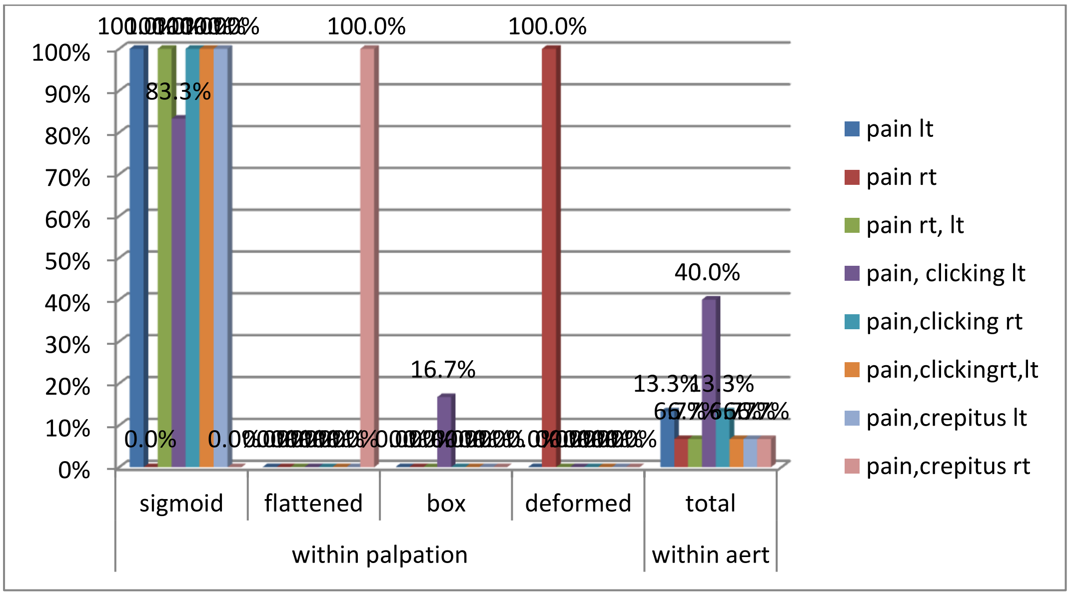
Graph-20: Correlation between deviation and CCLT



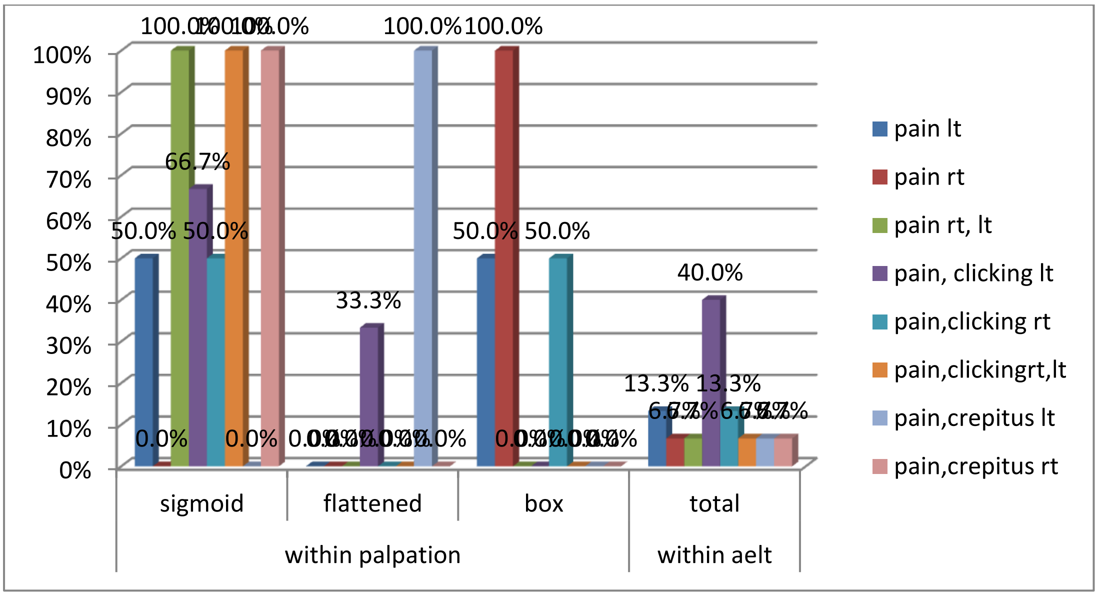
Graph-21: Correlation between deviation and CCRT



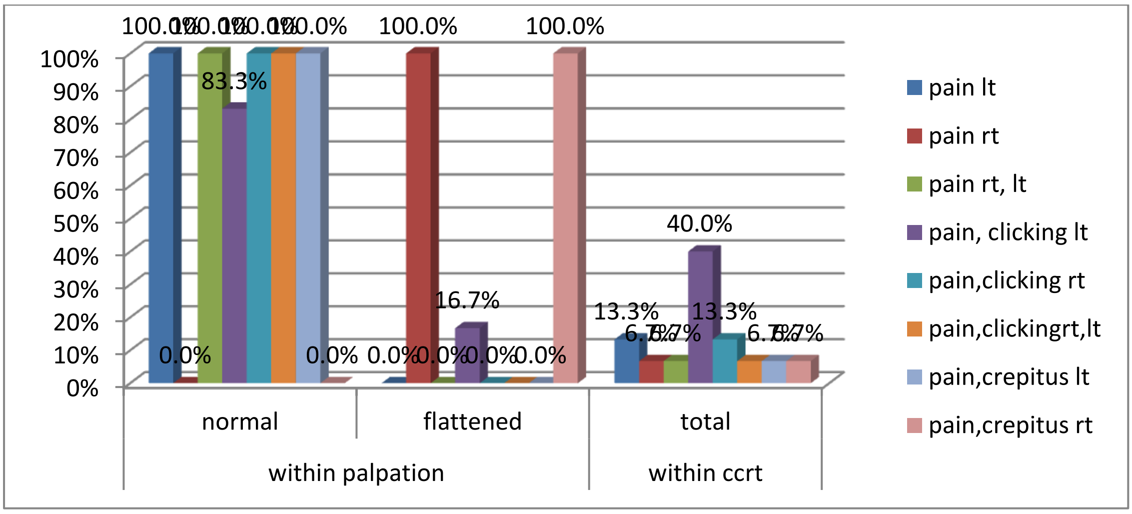
Graph-22: Correlation between palpation and AERT



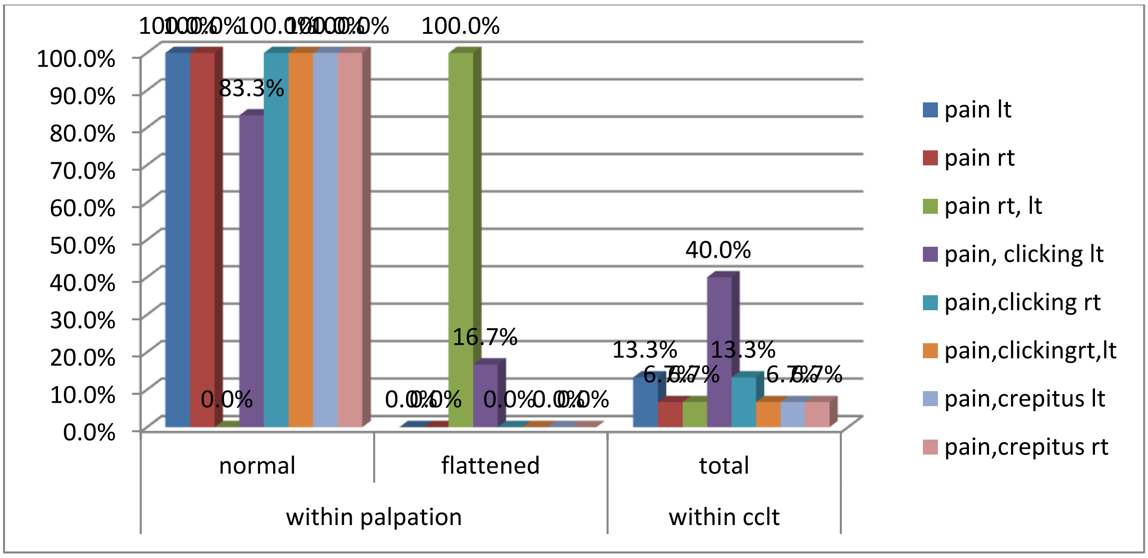
Graph-23: Correlation between palpation and AELT



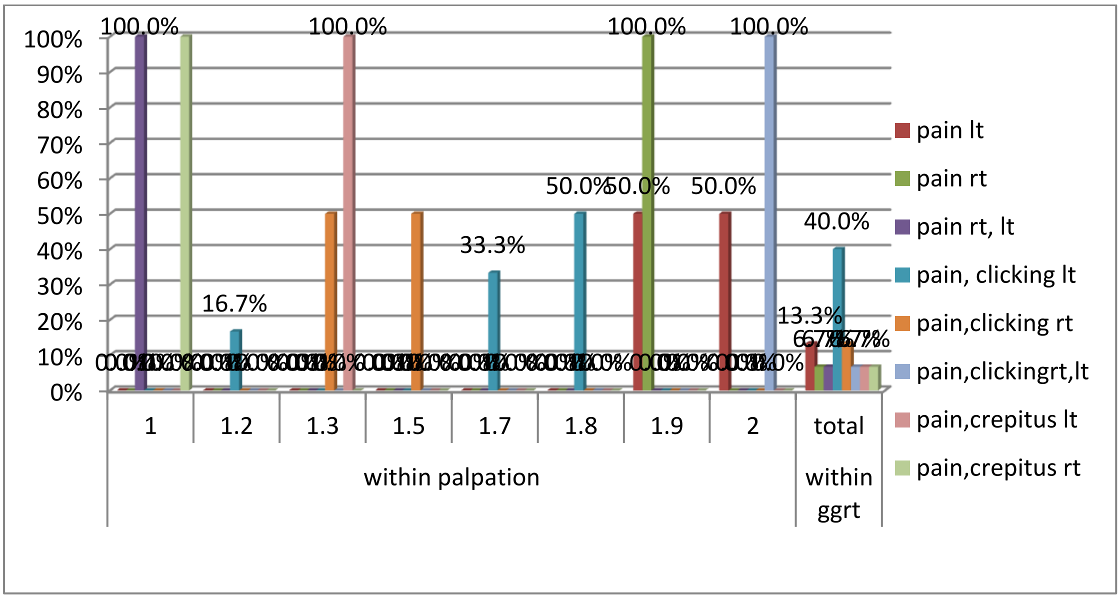
Graph-24: Correlation between palpation and CCRT



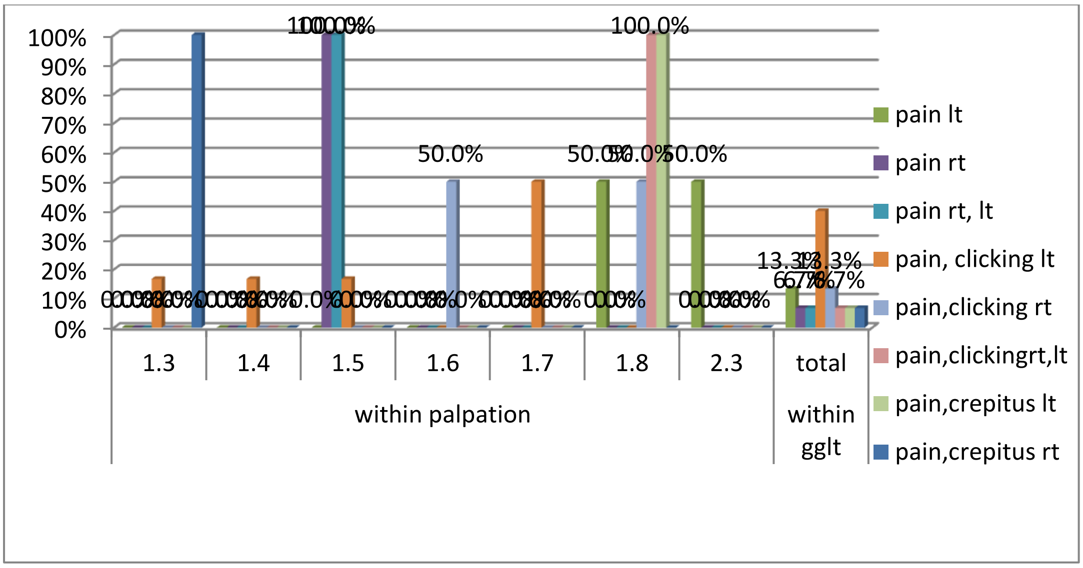
Graph-25: Correlation between palpation and CCLT



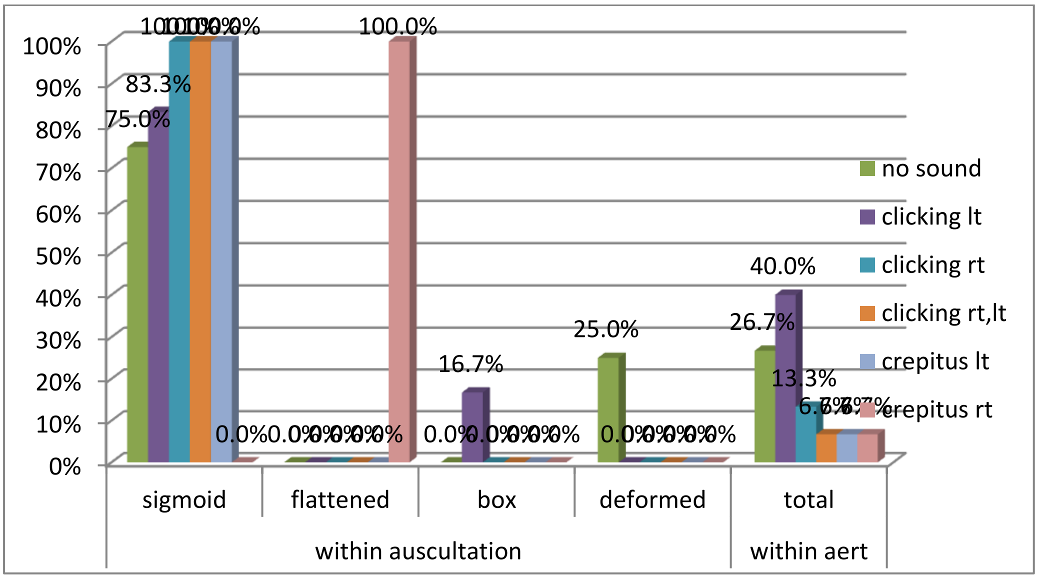
Graph-26: Correlation between palpation and GTRT



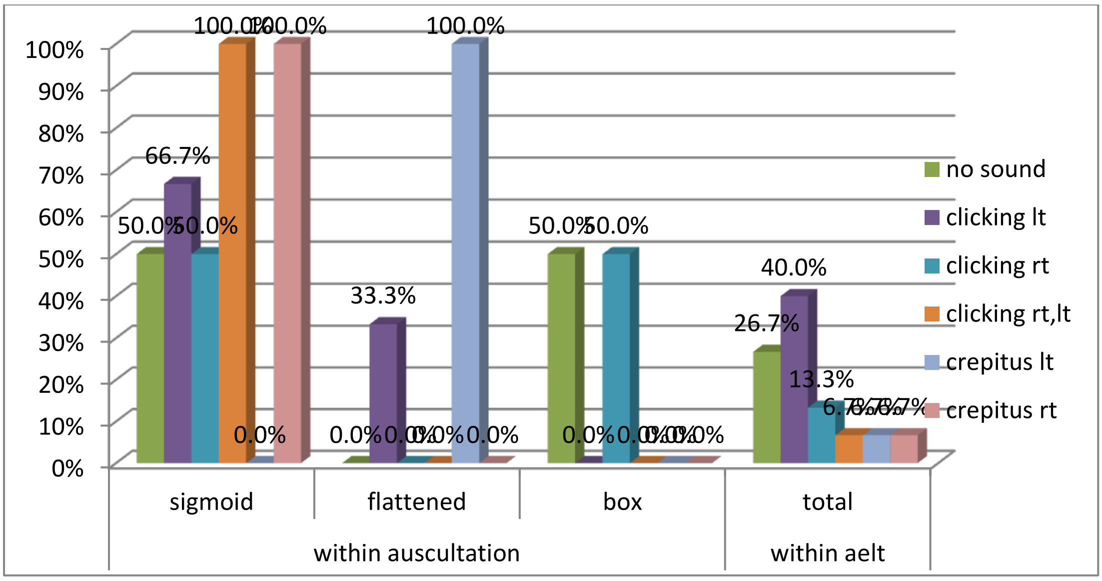
Graph-27: Correlation between palpation and GTLT



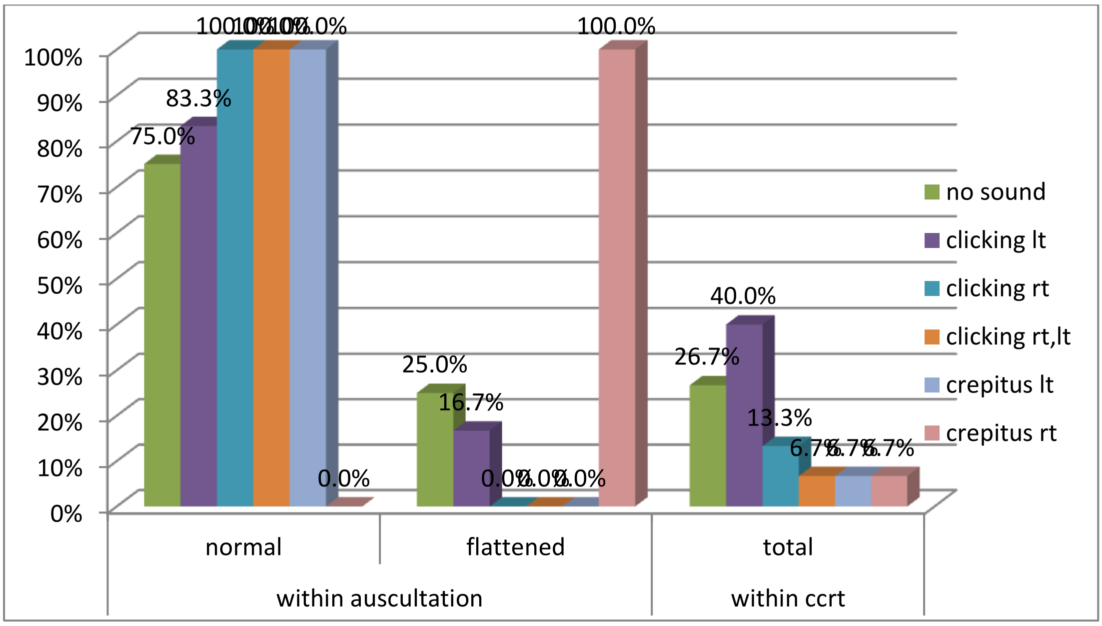
Graph-28: Correlation between auscultation and AERT



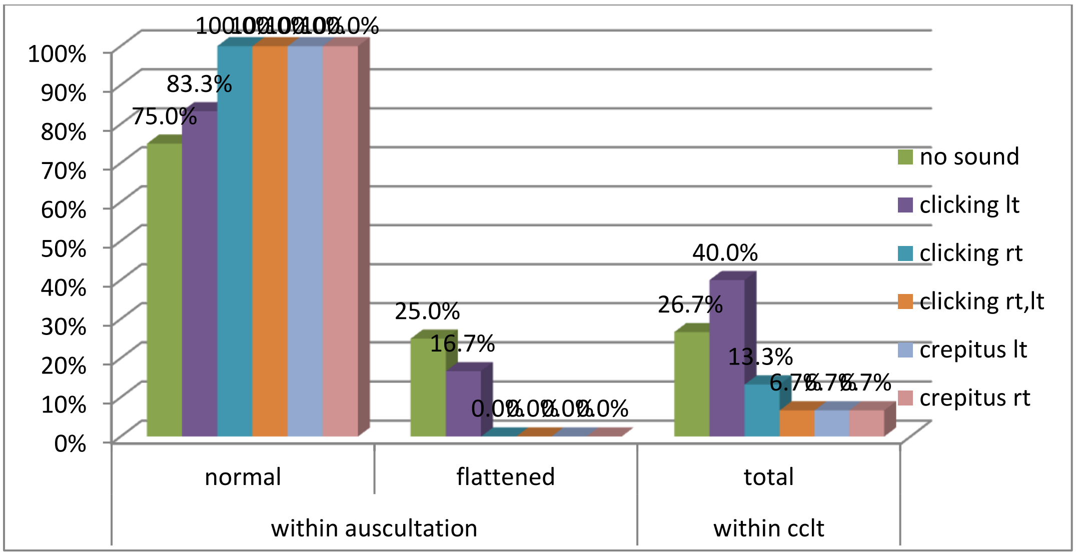
Graph-29: Correlation between auscultation and AELT



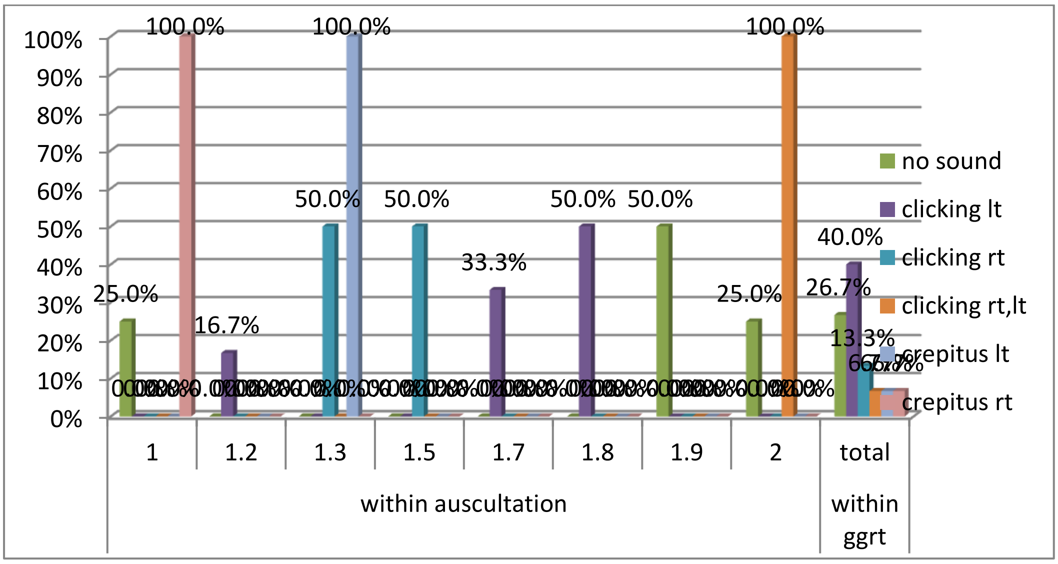
Graph-30: Correlation between auscultation and CCRT



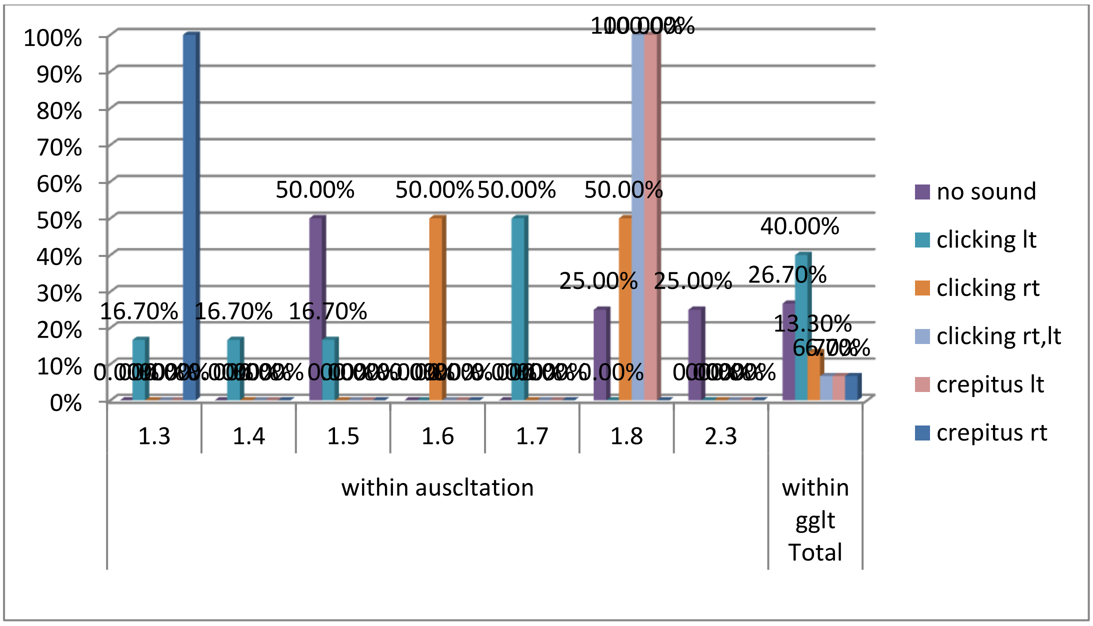
Graph-31: Correlation between auscultation and CCLT



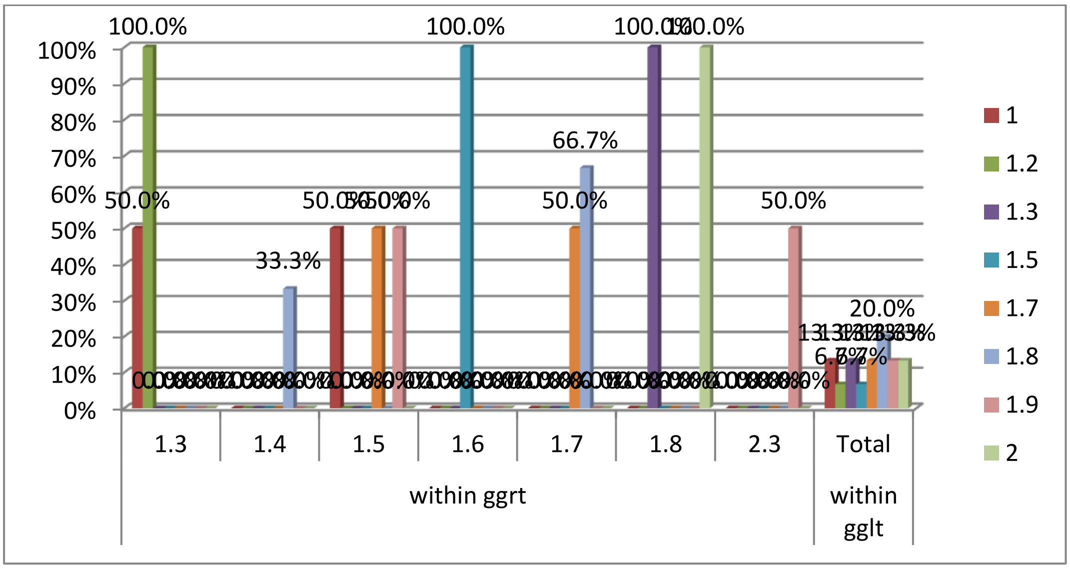
Graph-32: Correlation between auscultation and GTRT



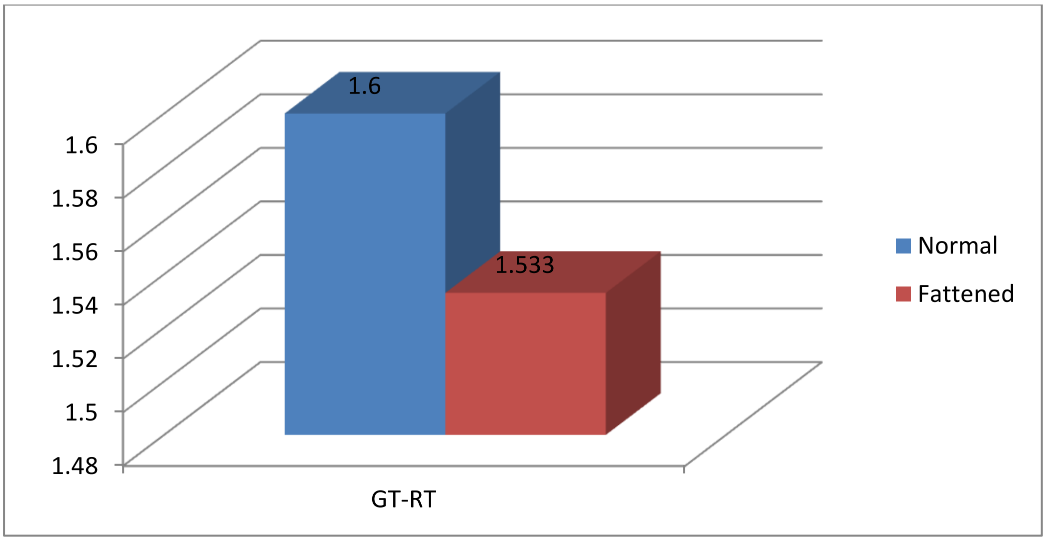
Graph-33: Correlation between auscultation and GTLT



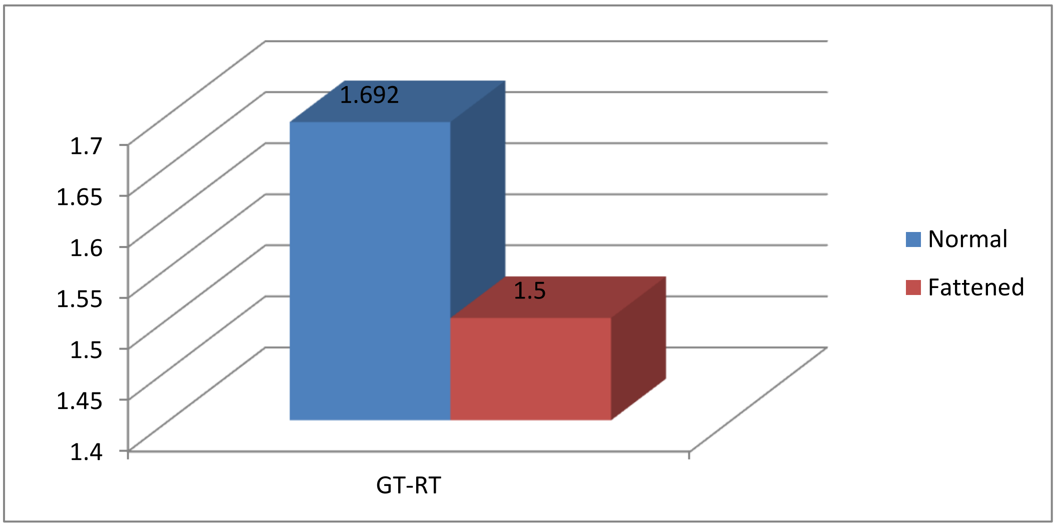
Graph-34: Correlation between GGRT and GTLT



Graph-35: Correlation between CCRT and GTRT



Graph-36: Correlation between CCLT and GTLT



The term temporomandibular joint disorder (TMD) was first suggested by Bell, which includes disorders of the joint as well as masticatory system. TMD is a heterogenous collection of signs and symptoms that can be generally characterized by the presence of pain, TM joint noise and limitation of jaw motion. Various etiological concepts have been related to temperomandibular joint disorders.

The structure and biochemical composition of contacting surface of TMJ may be altered by articular disk displacements. Disk deformation and/or perforation, atypical cellular architecture, osteophyte formation, subchondral bone resorption, disruption of the physical continuity of the articular surface of the mandibular condyle, and adhesion formation have all been observed in TMJs with articular disk displacement.

The purpose of the study is to correlate the clinical characteristics of temporomandibular joint (TMJ) disorder patients with osseous changes using Computed Tomography.

This study was conducted between March 2012 to July 2012 in the Department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Saravana Scans, Chennai.

A total number of 15 patients with symptomatic TMJ disorder were involved in the study.

Patients with TMJ changes due to developmental anamolies, age changes, trauma, infections, systemic diseases and tumours, patients with history of previous surgery in TMJ region and Patients with internal (implanted) defibrillator or pacemaker, cochlear (ear) implant, clips used on brain aneurysms, metal coils placed within blood vessels were excluded from the study.

In the total of 15 subjects involved in our study, the incidence of TMJ disorder was found to be more common in females than in males. Most of them had pain/clicking or both on the left side which may be related to predominant chewing habit. Deviation was present in 6 patients, which were all on the same side. On palpation, pain was commonly present in all the patients and all other findings such as clicking and crepitus were consistent with their chief complaint. Auscultatory findings were consistent with the palpatory findings.

The broad discussion about the predisposing factors for the development of internal TMJ disorders led to the development of diverse models to assess the association of anatomical structures with these disorders. It was important to divide these structures into groups in order to compare both sides and have a functional view of the TMJ, considering that most studies did not take that into account.

Articular eminence morphology

In this study, the articular eminence shape was classified into four groups according to the criteria of Kurita et al.[74]

The box shape represents a larger articular eminence or a deeper articular fossa than found in the sigmoid and flattened shapes. The sigmoid shape is more likely to have a larger articular eminence or a deeper articular fossa in the articular eminence than the flattened shape. The flattened shape is the shallowest.

On examining the articular eminence morphology in CT, sigmoid shape was most commonly observed which was in accordance with Fabrique et al.[74] One patient with the chief complaint of lock jaw had deformed shape on right side and box shape on left side. One patient with the chief complaint of pain in the left and one patient with clicking in right had box shape in the right side. Three patients with pain in the left had flattened shape in the left side. One patient with the pain and clicking in the right had flattened shape in the right side.

Yale and his coworkers,[66] considered the pioneers of this type of research, classified the shape of the condyle into five types: flat, convex, angled, round, others at its coronal view.

MRI generally has the disadvantage against CT for the display of the detailed contour of the condyle because of the limited spatial resolution and the magnetic susceptibility of bone

Previous radiological investigation using conventional tomography or MR has revealed that advanced osseous changes of the condyle, such as erosive osseous change, osteophyte and sclerosis, have not often been detected.

On examining the condylar changes in CT, normal condyle was the predominant finding followed by flattened condyle which was present in 5 patients which was in accordance with J Koyama et al.[66] Erosion and deformed condylar changes were not found in any of the patient.

On examining the glenoid thickness in CT, the mean thickness of glenoid fossa with normal condyle on the right side was 1.6 mm and the mean thickness of glenoid fossa with flattened condyle on the right side was 1.5 mm. The mean thickness of glenoid fossa with normal condyle on the left side was 1.7 mm and the mean thickness of glenoid fossa with flattened condyle on the right side was 1.5 mm.

With regard to the thickness of the roof of the glenoid fossa, an autopsy study showed that the minimum thickness of the roof of the glenoid fossa varied between 0.5 mm and 1.5 mm, with an average of 0.9 mm. Eckerdal and Ahlqvist[65] reported that the minimum thickness was on average 1.1 mm (range 0.1–3.6 mm) in a microradiography study.

In the present study, the average thickness of the roof of the glenoid fossa in joints with no condylar bone change was 0.7 mm, in agreement with a previous study.

As for the reaction of the TMJ to mechanical stress, a study of biomechanical simulation in the TMJ showed that morphological changes in the condyle and glenoid fossa altered the stress distribution, suggesting the existence of a mechanism for maintaining or changing condylar morphology in response to the stress distribution in the area. A study using strain gauge measuring techniques showed that during mandibular movements, a buffer effect on the forces is produced not only by the articular disc but also by the bone of the upper wall of the glenoid fossa. Honda et al[65] reported that mechanical stimulation may cause an increase in bone thickness in the glenoid fossa because of an incomplete shock absorption function resulting from a perforation of the disc or retrodiscal connective tissue.

Though we have correlated numerous clinical and radiographic features like age, sex, chief complaint, duration, mouth opening, deviation, palpation, auscultation, articular eminence morphology, condylar change and thickness of glenoid fossa in the above given sample size we couldn’t staunchly correlate osseous changes in CT for patients with temporomandibular joint disorders which may be due to smaller sample size. Hence further exploration in the above topic is required with larger sample size.

Temporomandibular joint disorder, as suggested by Bell, which constitutes joint and masticatory system has heterogenous collection of signs and symptoms.

History revealed, various studies which showed the etiopathogenensis being considered intially as a single cause but later turned out to be multifactorial. This complex joint which has structure and biochemical composition requires more exploration to identify and understand the cause and effect relation. Hence this study is framed to correlate the clinical characteristics of TMD patients with osseous changes using an advanced imaging modality, the CT.

The study was conducted between March 2012 and July 2012 in our department taking a total number of 15 symptomatic TMD patients.

The results showed a female preponderance predominately on the left side with deviation and clicking which were confirmed with palpation and auscultation.

The morphological variability of articular eminence were of the following types, sigmoid, flattened, box, deformed, among which sigmoid was the most common morphology found.

The morphological alteration of condyle due to TMD were catagorised as, normal, flattened, erosion, deformed by which is used for standardization in our study, among which most symptomatic patients had a normal condyle, few had flattened condyle whereas erosion and deformed condyles were never found. This may be due to smaller sample size and not standardizing the stage of the disease in the patient.

The glenoid fossa thickness showed variation of 0.1 and 0.2 mm respectively in the right and left side between normal and flattened condyle. Even this need a further exploration with larger sample size to confirm the variation.

To conclude, certain osseous changes like erosion, deformed and osteophyte formation were not appreciated much in our study. These changes are usually found in chronic stage of the disorder and hence can be achieved through a longitudinal study done with follow up.

TMD is a multifactorial disorder which has various clinical and radiological characterizations. Though it is difficult to standardize the criteria for diagnosis, a meticulous case history with detailed questionnaire and continuous follow up and investigation helps us categorize the stage of the disease and the investigation required at various stages for effective management. Hence these are to be done in large scale with effective training to the practitioners for successful management of the patients.

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S. No	Age	Sex	Chief Complaint	Duration (Months)	Mouth Opening (mm)	Deviation	Palpation	Auscultation	AE RT	AE LT	CC RT	CC LT	GT RT	GT LT
													GT	RT
1	32	F	Lock jaw	60	-35	Right	Pain Right	No Sound	Deformed	Box	Flattened	Normal	1.9	1.5
2	19	F	Pain left	6	38	Deviates to left	pain & clicking left	Clicking left	sigmoid	sigmoid	Normal	Normal	1.8	1.4
3	18	F	Pain left	4	40	Absent	pain clicking left	crepitus left	sigmoid	flattened	Normal	Normal	1.3	1.8
4	23	F	clicking left	10	44	left	pain & clicking left	Clicking left	sigmoid	sigmoid	Flattened	Normal	1.7	1.7
5	27	M	Pain left	18	38	Absent	Pain left	No Sound	sigmoid	Box	Normal	Normal	2.0	1.8
6	28	M	Pain left	24	42	Absent	pain clicking left	Clicking left	sigmoid	sigmoid	Normal	Normal	1.8	1.7
7	46	F	Pain left	36	35	Absent	pain clicking left	Clicking left	sigmoid	flattened	Normal	Normal	1.8	1.7
8	46	F	Pain left	24	36	Absent	pain clicking left	Clicking left	sigmoid	flattened	Normal	Normal	1.7	1.5
9	19	F	clicking left	6	44	left	pain clicking left	Clicking left	sigmoid	Box	Normal	Normal	1.3	1.8
10	23	F	clicking left	12	38	Absent	pain clicking left	Clicking left	sigmoid	sigmoid	Normal	Normal	2.0	1.8
11	27	F	clicking left	30	42	Absent	pain clicking left	Clicking left	Box	sigmoid	Normal	Flattene d	1.2	1.3
12	26	M	Pain left	12	44	Absent	Pain left	No Sound	sigmoid	sigmoid	Normal	Normal	1.9	2.3
13	20	F	clicking left	6	45	Absent	pain clicking left	Clicking left	sigmoid	sigmoid	Normal	Normal	1.5	1.6
14	35	F	pain right & left	12	41	Right	pain right & left	No Sound	sigmoid	sigmoid	Normal	flattened	1.0	1.5
15	30	F	clicking left	12	42	left	pain clicking left	crepitus left	flattened	sigmoid	Flattened	Normal	1.0	1.3

RAGAS DENTAL COLLEGE AND HOSPITALS

Department of Oral Medicine Diagnosis and Radiology

CASE SHEET PROFOMA

Clinical correlation of Osseous Changes in CT for patients with temporomandibular joint disorders – A Prospective study

Serial No. OP No: Date :

Name: Age/ Sex:

Address :

Chief complaint with duration:

TMJ Examination data:

Pain / Tenderness:

- Character :
- Duration :
- Frequency:
- Functional disruption :

Mouth opening:

Deviation:

Palpation:

Auscultation:

Condylar changes

N	F	E	D	S
no bone change	flattening	erosion with or without roughening	deformity	deformity accompanied by erosion with or without roughening

Articular eminence morphology:

Box	Sigmoid	Flattened	Deformed

Thickness of the roof of the glenoid fossa (measured at the thinnest part)

CONSENT LETTER

I ,the under signed hereby give my consent for the performance of taking **CT** on myself for the study titled Clinical correlation of osseous changes in CT for patients with temperomandibular joint disorder , conducted by **Dr.A.E.Malarvizhi**, under the guidance of **Dr. S. Manojkumar, MDS,** Professor, Department of Oral Medicine and Radiology, Ragas Dental College and Hospital, Chennai. I have been informed and explained about the evaluation procedure, risk involved and likelihood of successes. I also understand and accept this as a part of study protocol, thereby voluntarily, unconditionally freely give my consent without any fear or pressure in mentally sound, conscious state to participate in the study.

Witness/Representative	Patient Signature
(If any)	Date:

ஒப்புதல் படிவம்

நான் என்னுடைய முழு ஒத்துழைப்பை மருத்துவர் **அ.இ. மலர்விழி** அவர்கள் நடத்தும் தாடை மூட்டு பகுதியை **CT** மூலம் கண்டறிதல் என்ற ஆராய்ச்சி கட்டுரைக்கு வழி நடத்தும் மருத்துவர் **எஸ். மனோஜ் குமார்** பேராசிரியர் வாய் மருத்துவம், நோய் அறிதல் மற்றும் ஊடுகதிர் பிரிவு, ராகாஸ் பல் மருத்துவமனை, அவர்களுக்கு அளிக்கிறேன். ஆய்வின் பற்றிய தன்மையும், அதைச் சார்ந்த நடத்தும் பரிசோதனைக்கும், அதனால் ஏற்படும் பின்விளைவுகள் மற்றும் அதன் முக்கியத்துவத்தையும் எனக்கு விளக்கிக் கூறப்பட்டது. இதை அறிந்து செயல்முறையை முழுவதும் நடத்தி முடிக்க நானாக வேறொருவர்தூண்டுதல் இன்றி முழு சுயநினைவோடு எந்த வித அச்சமும் இன்றி இந்த ஆய்வுக்கு பூரண ஒத்துழைப்பு அளிக்க ஒப்புதல் அளிக்கின்றேன்.

தேதி : **கையொப்பம்**