Republic of Iraq

Ministry of Higher Education and Scientific Research

Babylon University

College of Dentistry

جمهورية العراق

وزارة التعليم العالي والبحث العلمي

جامعة بابل

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Severity index in adults patients with Temporomandibular Disorders

A project

Submitted to College of Dentistry in Partial Fulfillment of the Requirements for the Degree of Bachelor of Dentistry

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2024 A.D. 1445 H.D.

بسم الله الرحمن الرحيم

(وَلَسَوْفَ يُعْطِيكَ رَبُّكَ فَتَرْضَى)

صدق الله العظيم

سورة الضحى/ الآية 5

الإهداء :

."من قال أنا لها "نالها

. وأنا لها وإن أبت رغُماً عنها أتيتُ بِها

لم تكُن الرحلة قصيرة ولا ينبغي لها أن تكون ،

. لم يكن الحلم قريباً ولا الطريقُ كانَ محفوفاً بالتسهيلات لكنني فعلتُها ونِلتُها

الحمدُ لله حُباً وشكراً وأمتناناً ،الذي بفضله ها أنا اليوم أنظر إلى حُلماً طال أنتظاره وقد أصبح . واقعاً أفتخر به

أهدي بكل حب بحث تخرجي

. إلى نفسي العظيمة القوية التي تحملت كل العثرات وأكملت رغم الصعوبات

" إلى ملاكي الطاهر وقوتي بعد الله داعمتي الأولى والأبدية "أمي

أهديكِ هذا الأنجاز الذي لولا تضحياتكِ لما كان له وجود ،ممتنه لأن الله قد أصطفاكِ لي من البشر . أُماً يا خيرَ سندٍ وعوضٍ

إلى من دعمني بِلا حدود وأعطاني بِلا مقابل

"أبي"

: إلى من قيل فيهم

(سَنَشُدُّ عَضُدَكَ بِأَخِيكَ)

إلى خيرة أيامي وصفوتها ،إلى من مُدت لي أياديهم وقت ضعفي وأمنو بقدراتي ،إلى ضلعي الثابت "وأمان قلبي "اخوتي

وفي الختام أقف اليوم أمامكم ، قلبي مملوء بمزيج من المشاعر المتناقضة ،فرحة الأنجاز ،وحزن . الفراق ،مشاعر ممزوجة بذكريات رحلة تعليمية طويلة ومليئة بالتحديات والأنجازات

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CBCT	Cone beam computerized tomography
CT	Computed tomography
ECA	The external carotid artery
LM	Left masseter muscle
LT	Left temporalis muscle
LTMJ	Left temporomandibular joint
MRI	magnetic resonance imaging
NSAIDs	Non-steroidal anti inflammatory drug
RM	Right masseter muscle
RT	Right temporalis muscle
RTMJ	Right temporomandibular joint
TMD	Temporomandibular disorders
TMJ	temporomandibular joint

Chapter One
Introduction

1.1: Anatomy of TMJ

The temporomandibular articulation is composed of bilateral, temporomandibular joints (TMJs) [1] . Each joint is formed by a mandibular condyle and its corresponding temporal cavity (glenoid fossa and articular eminence) [2] . The TMJ and its associated structures play an essential role in guiding mandibular motion and distributing stresses produced by everyday tasks, such as chewing, swallowing, and speaking [3] . Temporomandibular disorders (TMD) is an umbrella term for pain and dysfunction involving the masticatory muscles and the temporomandibular joints[4] . The American Association for Dental Research (AADR) has defined these disorders as "a group that involves musculoskeletal and neuromuscular conditions where apart from the TMJ, the muscles of mastication and other surrounding structures are also involved [5] . Its prominent features include regional pain in the face and preauricular area, limitations in jaw movement, and noise from the TMJs during jaw movements [6] . Individuals with TMD commonly also have other painful and non-painful comorbidities including headaches, fibromyalgia, irritable bowel syndrome, tinnitus, chronic fatigue syndrome, depression and sleep disturbance [7] . Majority of people who suffer from temporomandibular disorders (TMD) opt for medical help when they encounter severe chronic pain in the form of headaches or facial pain [8] It is reported that more than half of the patients who are diagnosed with TMD suffer from temporomandibular myofascial pain [9] . It affects 5% to 10% of the population, with the majority being the females [10] . Early diagnosis and management of TMD can greatly improve prognosis and quality of life for patients [11] . The anatomy of the temporomandibular joint (TMJ) is an inimitable considering it has a complex structural architecture with its associated muscles, cartilage, ligaments, vessels and the neural supply to the joint [12].

1.1.1:Temporal fossa (glenoid fossa)

The cranial surface of TMJ consists of the squamous area of the temporal bone; it takes the name of glenoid fossa and welcomes the condyle of the jaw [13]. The posterior area of the fossa is known as posterior articular ridge; sideways to the latter,

we find a bone portion called postglenoid process [14]. The postglenoid process area contributes to forming the upper wall of the external acoustic meatus [15]. The anterior limit of the glenoid fossa of the temporal bone constitutes the articular eminence, which forms a medial bone prominence at the posterior border of the zygomatic bone [16]. The preglenoid plane is slightly inclined, which leads into the articular eminence; the latter is anterior to the fossa, along with the base of the skull [17]. This area allows and facilitates the movements of the articular disk and the condyle [18] . On the lateral surface of the articular eminence, there is a bone ridge, known as the articular tubercle, near the root of the zygomatic process [19]. The glenoid fossa is wider in its mediolateral portion, compared to the anteroposterior area [20]. The inferior articular surface of the glenoid fossa represents the superior area of the mandible [21]. It consists of the condyle of the mandible with a transverse diameter of about 15 to 20 mm and a measurement of about 8 to 10 mm in the anteroposterior direction [22].

1.1.2: Articular disc :

The articular disc that covers the condyle and interposes below the glenoid fossa has a biconcave or oval shape; the cartilaginous disc has an anterior (about 2 mm) and posterior (about 3 mm) portion, with a thinner diameter in the middle [23]. The anterior portion of the disk consists of a layer of fibroelastic fascia (above) and a fibrous layer (inferiorly) [24]. The upper portion is in contact with the postglenoid process, with the function of preventing the disc from slipping during the opening of the mouth ([25]. The lower portion of the disk has the task of avoiding excessive rotational movements of the disk relative to the mandibular condyle [26].

- The anterior portion of the articular disk is in contact with the joint capsule , articular eminence condyle , the upper area of the lateral pterygoid muscle [27].
- The posterior portion of the articular disk relates to: bilateral retro-disc tissue (behind the condyle), glenoid fossa; condyle; temporal bone. The medial and lateral aspect of the cartilaginous disc isattached to the condylar formation of

the mandible. The edges of the disc partly fuse with the fibrous capsule surrounding the joint [28].

1.1.3: Ligaments :

Several ligaments manage the TMJ forces and send multiple proprioceptive afferents such as

1. Capsular ligment : The capsular ligament is a thin elastic fibrous connective tissue envelope that attaches to the margins of the articular surfaces [29].

2. Lateral temporomandibular Ligament : The lateral temporomandibular ligament is the main ligament of the joint, lateral to the capsule but not easily separated from it [30].

3. Accessory Ligaments

- The sphenomandibular ligament
- The stylomandibular ligament

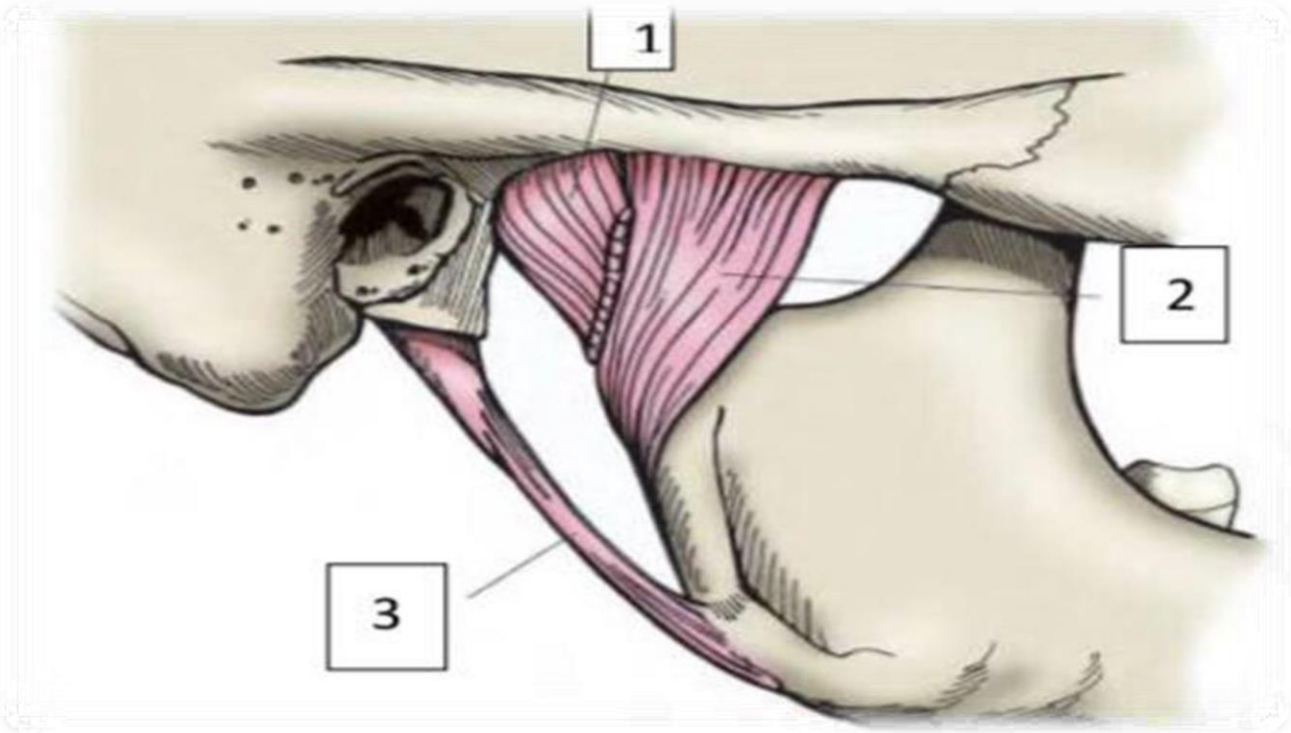

Figure 1.1 : anatomy of TMJ

1.1.4: Muscles of mastication

The primary muscles of mastication are the paired masseter, medial and lateral pterygoids, and temporalis muscle [31]. Mandibular movements toward the tooth contact position are performed by contraction of the masseter, temporalis, and medial pterygoid muscles[32]. The lateral pterygoid is the main protrusive and opening muscle of the mandible [33].

The accessory muscles of mastication are relatively smaller, the digastric muscle and hyoid muscles (mainly omohyoid muscles) with platysmas lowers the jaw when active and is not active in a jaw resting position [34].

1.1.5: Vascular Supply of Masticatory System Structures

The external carotid artery (ECA) is the main blood supply for the structures of the masticatory system. It bifurcates into the superficial temporal artery and the internal maxillary artery [35]. These two arteries supply the muscles of mastication and the TMJ. Arteries within the temporal bone and mandible also send branches to the capsule [36].

1.1.6: Nerve Supply of Masticatory System Structures

The masticatory structures are innervated primarily by the trigeminal nerve, but cranial nerves VII, IX, X, and XI and cervical nerves 2 and 3 also contribute [37].

1.2; General etiology of TMJ disorders

There is many factor that my effect on tmj such as .

1- Occlusal factors :

Occlusion is the first and probably the most controversial etiologic factor of TMD [38]. Costen was the one who first established with certainty the involvement of occlusion in the development of TMD [39]. Nowadays, most researchers include occlusion among all the factors related to TMD, having a possible role in both susceptibility and onset or perpetuation of TMD [40].

2- Trauma :

injury to the head or neck is often considered a significant risk factor for development of TMDs, and has been proposed to produce internal derangements of the TMJ3- Psychological factors :

The role of stress and personality in the etiology of the temporomandibular pain dysfunction syndrome has undergone extensive scrutiny [41]. Psychological studies have shown that patients with TMD have similar psychological profiles and psychological dysfunction as other chronic musculoskeletal pain disorders, such as tension type headache and back or arthritic pain [42]. There is considerable evidence that psychological and psychosocial factors are of great importance in the understanding of TMD, but there is less evidence that these factors are etiologic [43] .

Nowadays the association between depression and stress and different physical symptoms of TMD is widely acknowledged [44]. TMD symptoms, especially pain, are also discussed as being a causative or intensifying factor in the development of depression and psychic diseases [45].

Stress, anxiety and other psychological factors induce muscle hyperactivity and muscle fatigue with the appearance of muscle spasms and the following consequences: contracture, occlusal disharmony, internal disturbances and degenerative arthritis [46]. These factors can alter the occlusal scheme of the masticatory cycle, so that these alterations are more a result of TMD and not a triggering factor [47]. Various studies have confirmed that patients with myofascial pain or myofascial pain associated with arthralgia, arthritis or osteoarthritis present more advanced stages of depression and somatization than those diagnosed with disc displacement [48].

4- Hormonal factors :

Signs and symptoms of TMD are four times more common among women, who seek specialized treatment for this disease three times more frequently than men [49]. Despite the fact that the low prevalence of TMD in men has not been completely

elucidated yet, the presence of higher testosterone levels may be a plausible explanation5- Parafunctions:

Parafunctions are defined as impaired or altered functions of TMJ [50]. Of these, excessive gum chewing, teeth clenching and bruxism have been extensively studied as possible risk factors for TMD [51].

6- Joint hyperlaxity and joint hypermobility :

The relationship between hypermobility and TMD has also been studied [52]. Some authors have reported no association between TMD and systemic hyperlaxity or between TMJ mobility and systemic hypermobility, while others found a positive relationship between generalized joint hypermobility and TMD [53]. Kavuncu et al. assessed the risk for TMD in patients with systemic hypermobility and TMJ hypermobility [54]. They found that both local and general hypermobility were more frequently detected in patients with TMD than in the controls, and that the risk of TMJ dysfunction was greater if the patient presented both alterations simultaneously [55].

1.1.4: Diagnosis of TMJ disorders :

The signs and symptoms of TMJ may mimic other orofacial pain conditions. Although precise physical diagnosis into the type of TMJ is helpful in developing an appropriate treatment plan, it might not be straight forward in every case.

- Taking a patients' history is an important part of diagnosing the TMJ condition. The acquisition of history follows the usual format.[56] Apart from the chief complaint, inquiries should be made regarding any history of trauma or previous episodes, aggravating factors, such as eating, talking, yawning or spontaneous background pain, and any previous investigations or treatmen.[57] The clinician should note any habits such as smoking, drinking and recreational drug use, and any

history of clenching or bruxism as complained by the patients' bed partner.[58] Additionally, the clinician should ask questions regarding stress and level of life satisfaction, and whether there are any recent life events, such as change of job or loss of a loved one.[59] Although most clinicians treating TMJ may be experienced with acquiring a clinical history, some may not be comfortable with taking a psychological history.[60] If desired, the clinician may employ the numerous psychosocial instruments available to aid in their diagnosis, such as those in Axis-II of DC/TMJ .[61] Most clinicians who treat orofacial pain believe clinical examination is the most crucial process of diagnosing TMJ. [62]

- The location of pain, and whether the pain is localized, remains within or spreads beyond the confines of the muscle, should be confirmed with palpation, which is done at rest and during mandibular function.[63]
- Clicking or crepitus upon mandibular function might be quite obvious in some cases, and the detection might be aided by the use of a stethoscope.[64] the presence or location of clicking detected by the clinician might be different from that reported by the patient, and this should be documented. [65]The range of mouth opening measured should include pain-free maximum mouth opening, maximumunassisted mouth opening, and maximum assisted mouth opening.[66] Any deviation of the mandible may indicate differential obstruction of the movement of the mandibular condyle in rotation and/or translation.[67] An intra-oral examination is performed to rule out any mucosal pathologies of the oral cavity and oropharyngeal region, as well as to assess the state of the dentition.[68]
- Imaging is considered to be a useful adjunct in the diagnosis of TMJ . [69]Although the diagnostic information provided by plain radiographs like orthopantomogram is limited, they are convenient, simple and

serve to rule out some of the differential diagnoses of the bony TMJ, such as fractures, ankylosis, growth disturbances, as well as neoplasms when further information is desired, magnetic resonance imaging (MRI) is the gold standard for TMJ imaging, and is useful in assessing the status of the osseous, as well as the non-osseous structures of the TMJ, such as the masticatory muscles, ligaments and the cartilaginous disc.[70]

- while MRI is the most commonly used diagnostic imaging for the common diagnoses of TMJ , other imaging modalities are also employed for specific indications.[71] Cone-beam computed tomography (CBCT) has been used to further assess the osseous structure of the TMJ .[72]This may be desirable in cases of TMJ Ankylosis, benign bony neoplasms or overgrowth, or for the planning of osseous surgery, such as for eminectomy for recurrence TMJ dislocation. However, for most other diagnoses of TMJ, the value of CBCT is not well-established since the information provided in terms of soft tissues is limite.[73] Moreover, the use of ultrasound as a diagnostic tool for TMJ has been suggested.[74] Ultrasound has the advantages of being non-invasive, cheap, and widely available in many health institutions, yet the effectiveness as a diagnostic method remains to be confirmed .[75] For some inflammatory conditions of the TMJ, such as osteoarthritis and joint inflammation, bone scintigraphy may be of value as a diagnostic tool.[76] Moreover, bone scintigraphy has been proposed as a method for the evaluation of active TMJ condylar growth, but it has been shown that both the sensitivity and specificity are low for this indication .[78] Apart from the different imaging modalities available, other investigations are not commonly done for most diagnoses ofTMJ , except in specific indications.[79] For example, blood investigations may be done for TMD related to systemic conditions, such as rheumatoid

arthritis or gout.[80] In the case of uncertain diagnoses of rare diseases or neoplasms, tissue biopsies might be taken, which may be done by fine-needle aspiration, arthroscopic or open joint approach.[81]

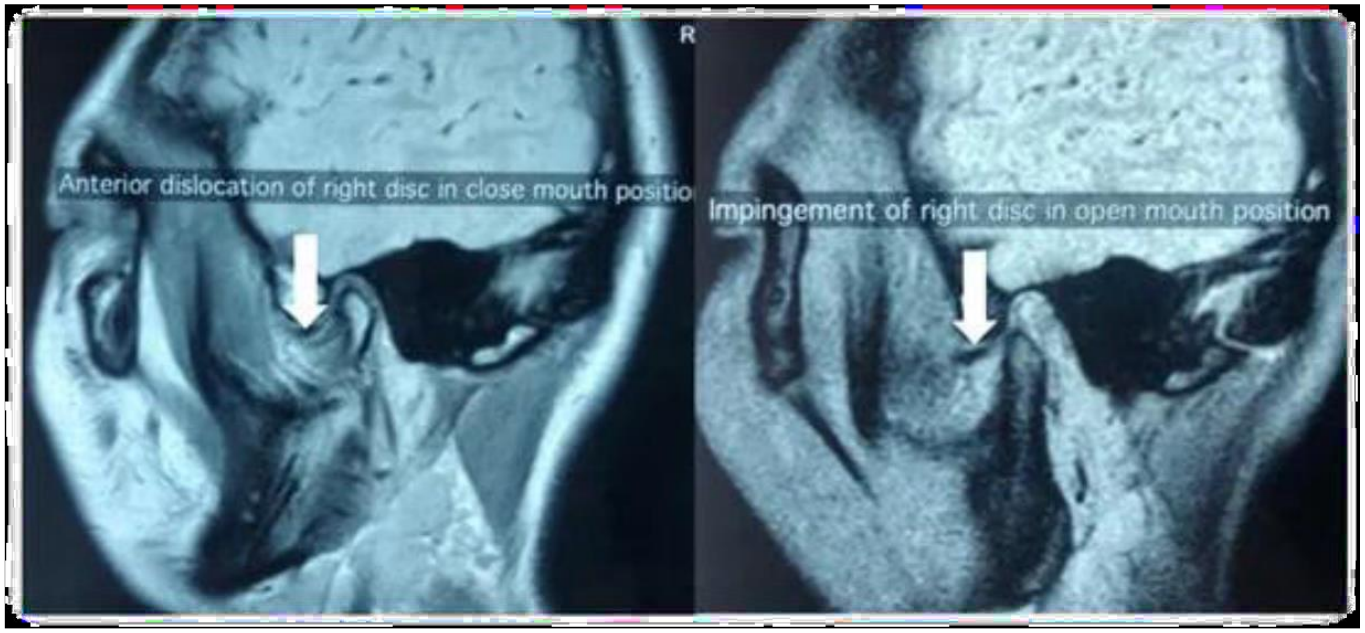

Figure 1.2 : MRI are showing anterior dislocation of right disc on open and close mouth

1.1.5: Type of TMJ disorders

1.1.5.1: Disc disorders

Internal Derangement OF The TMJ:

Wilkes Classifcation of Internal Derangements Disc- Condyle disorders can be staged based on the characteristics of the pain, amount of mouth opening, disc Location/condition, and altered joint anatomy, as observed from physical examination, MRI, and arthroscopy .

a) Disc Displacement with Reduction:

A diagnosis of disc displacement with reduction is made when the patient presents with a history of a click or pop and it can be felt when the patient moves the mandible. As the patient opens the mouth, the condyle translates forwardand moves on to an intermediate zone of the disc (reduced position) that may cause the opening click or

pop. As the mouth continues to open, the condyle continues to translate forward with the disc and remains in the intermediate zone of the disc. As the patient closes the mouth, the condyle retrudes and moves back under the posterior band onto the retrodiscal tissue, which may again cause the closing click or pop. As the mouth continues to close, the condyle remains on the retrodiscal tissue. If both opening and closing click/pop is present, then the opening click/pop occurs at a wider opening than the closing click/pop. Rather, it should be heard by the patient at least once in the last 30 days and by the examining dentist during at least a third of the mandibular movements. Because the disc reduces during condylar translation, range of motion is not limited. However, movements may not be as smooth as a normal TMJ because of the momentary sliding of the condyle on and off of the disc .[83]

b) Disc Displacement with Reduction with Intermittent Locking :

A diagnosis of disc displacement with reduction with intermittent locking is made when the patient has a disc displacement with reduction and reports that occasionally the TMJ structure that normally causes the click blocks the condyle's movement, inhibiting the mouth for obtaining its normal opening. This lock suddenly occurs, may last for seconds to days, and then suddenly releases. When the limited opening occurs, a maneuver may be needed to unlock

the TMJ. History is positive for any noises present with jaw movement or function in the last 30 days or during the examination itself and report of intermittent locking with limited opening in the last 30 days or evidence of intermittent locking during clinical examination .[84]

c) Disc Displacement without Reduction with Limited Opening:

A diagnosis of disc displacement without reduction with limited opening (closed lock) is made when a patient has a suddenly occurring continuous marked limited opening (less than 40 mm). The patients themselves are usually aware that the TMJ structure that normally caused the click is now blocking them from obtaining their normal opening. They may also report of their TMJ catching at that locationor

intermittently having had this problem (lasting seconds to days), which suddenly released and allowed them to regain their normal opening. As the mouth opens, the condyle first rotates and then attempts to translate forward, but the condyle cannot slide under the disc's posterior band to reduce onto the intermediate zone of the disc. The translation is limited by the disc, and typically, the patient is initially able to open only between 20 and 30 mm .[85]

d) Disc Displacement without Reduction without Limited Opening:

A diagnosis of disc displacement without reduction without limited opening is made when the patient has a history of sudden-onset limited opening that gradually increased to 40 mm or greater. This suggests that the patient had a disc displacement without reduction with limited opening and over time, the retrodiscal tissue stretched and enabled the disc to move forward, thereby allowing the condyle to translate further and permitting the patient to open wider. History is the same as defined for disc displacement without reduction with limited opening, .

e) Posterior Disc Displacement :

Posterior disc displacement has been described as the condyle slipping over the anterior rim of the disc during opening, with the disc being caught and brought backward in an abnormal relationship to the condyle when the mouth is closed. The disc is folded in the dorsal part of the joint space, preventing full mouth closure. The Clinical features are [86]

(1) a sudden inability to bring the upper and lower teeth together in maximal occlusion.

(2) pain in the affected joint when trying to bring the teeth firmly together.

(3) displacement anteriorly of the mandible on the affected side.

(4) restricted lateral movement to the affected side.

(5) no restriction of mouth opening.

1.1.5.2.: Hypomobility disorders other than disc disorders:-

a) Adherence/Adhesions:

Adherence refers to a transient sticking of the articular surfaces. However, prolonged periods of adherence may result in true adhesions, wherein fibrous bands of connective tissue form between the articulating surfaces of the condyle or mandibular fossa, the disc, or surrounding tissues. Adhesions occur secondary to prolonged static loading of the TMJ's surfaces (for example, jaw-clenching during sleep). The patient presents with history of loss of jaw mobility and no history of TMJ clicking (historically to differentiate from disc displacement without reduction with limited opening .[87]

b) Ankylosis :

Ankylosis is the firm restriction of the condyle due to fibrous bands or osseous union within the TMJ, most commonly resulting from trauma to the mandible and/or TMJ. It is a Chronic, painless,limitation jaw motion. The involved condyle may not be able to translate and may have limited rotation, causing the patient to have a very limited opening, depending on the type and extent of the ankylosis. Ankylosis of the temporomandibular joint (TMJ) most often results from trauma or infection, but it may be congenital or a result of rheumatoid arthritis. When ankylosis leads to arrest of condylar growth, facial asymmetry is common. Intra-articular (true) ankylosis must be distinguished from extra-articular (false) ankylosis, which may be caused by enlargement of the coronoid process, depressed fracture of the zygomatic arch, or scarring resulting from surgery, irradiation, or infection.

In most cases of true ankylosis, x-rays of the joint show loss of normal bony architecture. A late and rare finding; in some cases, it affects both. In severe cases, there is a loss of mandibular condylar support with resultant retrognathia. Congenital temporomandibular joint ankylosis is a rare maxillofacial disorder characterized by significant reduction in mouth opening (i.e. from a fewmillimeters to a few centimeters) in the absence of acquired factors (e.g. trauma, infection) contributing to the ankylosis. It is associated with variable degrees of facial dysmorphism (i.e. lateral

deviation of the mandible and chin, lower facial asymmetry, retrognathia, micrognathia, dental malocclusion) and patients typically present with feeding and breathing difficulties,also developmental delay, hypotonia, seizures, and additional dysmorphic feature.Clinical differential diagnosis of ankylosis should include pseudoankylosis of extra-articular pathology occurring in hypomobility of the joint due to coronoid hyperplasia (Jacob disease), fbrous adhesions between coronoid and tuberosity of maxilla or zygoma, depressed zygomatic arch fracture, dislocated zygomatic complex fracture, temporalis muscle scarring, or myositis ossifcans,.

Ankylosis may be fibrous or osseous.[88].

- ❖ Fibrous Ankylosis:-

Fibrous ankylosis results when fibrotic tissue forms between the articulating surfaces of the condyle or mandibular fossa, the disc, or surrounding tissues. There are no gross bony changes and no radiographic findings other than absence of ipsilateral condylar translation on opening. Patient findings include history of progressive loss of jaw mobility; positive findings for severe limited range of motion on opening, uncorrected jaw deviation to the affected side, marked limited laterotrusion to the contralateral side; and positive CT/CBCT imaging finding of decreased ipsilateral condylar translation on opening and a joint space between ipsilateral condyle and eminence .[89]

- ❖ Osseous Ankylosis:-

Bone formation between the condyle and fossa usually results in osseous ankylosis, the patient has a more restricted opening than with fibrous ankylosis or even complete immobility of the joint. The characteristic findingsinclude radiographic evidence of bone proliferation with marked defection to the affected side and marked limited laterotrusion to the contralateral side. Patient findings include history of progressive loss of jaw mobility, positive examination findings such as absence of or severely

limited jaw mobility with all movements and CT/CBCT being positive for imaging-based evidence of bone proliferation, with obliteration of part or all of the joint space.

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1.1.5.3: Hypermobility disorders :

a) Subluxation (Partial Dislocation):

This is a condition involving the disc-condyle complex and the articular eminence. A diagnosis of subluxation is made when in the opened mouth , the disc-condyle complex is positioned anterior to the articular eminence and is unable to return to normal closed-mouth position without a manipulative maneuver by the patient. Causes of subluxation include looseness of the joint capsule and ligaments, as in overextension injury, following dental procedures that require prolonged mouth opening or excessive yawning, extrinsic trauma (intubation, endoscopy), and connective tissue disorders (Ehlers-Danlos syndrome, Marfan syndrome). History is positive for jaw locking or catching in a wide-opening mouth position, even for a moment, so the patient could not close from the wide-open position in the last 30 days and for inability to close the mouth from wide opening without a self-maneuver. No examination findings are required .[90]

b) Luxation (Dislocation, Open Lock) :

This is a condition in which the disc-condyle complex is positioned anterior to the articular eminence and is unable to return to the fossa without a specifc manipulative maneuver by a clinician. This is also referred to as open lock. Causes of luxation include post-traumatic capsular loosening, prolonged wide mouth opening, chronic subluxation, seizure disorders, Parkinsonism, drug- induced tardive dyskinesia (neuroleptics like phenothiazines), defects in the bony surface (shallow articular eminence), or a genetic predisposition (Ehlers-Danlos syndrome, Marfan syndrome). Patient reports of inability to close from wideopening and that mouth closing can be achieved only with a specifc mandibular maneuver by the clinician. Examination is positive for wide opening mouth,

protruded jaw position, and lateral position to the contralateral side if unilateral. 91_(okeson and leeuw 2011).

1.2.Joint diseases (Degenerative joint disease)

-Osteoarthrosis

-osteoarthritis

1.2.1 : Osteoarthrosis:

Osteoarthrosis is a multifactorial disease associated with TMJ overloading.

Though it is synonymous with osteoarthritis in medical orthopedic literature , in dental TMJ literature, it has been recently identifed as a chronic low infammatory degenerative progressive loss of articular cartilage in the TMJ resulting from an imbalance between predominantly chondrocyte-controlled reparative and degradative processes. Patient generally presents with no symptoms. The past history may reveal a period of time when symptoms were present (osteoarthritis) that can only be confrmed through radiographs. Crepitation is a common finding. In the absence of clinical symptoms like joint pain, treatment of this arthritides is contraindicated. The only treatment that may need to be considered is if the bony changes in the condyle have been signifcant enough to alter the occlusal condition and, in such cases, dental therapy may need to be considered .[92]_(

1.2.2 : Osteoarthritis(Degenerative Joint Disease, DJD)

Degenerative joint disease (DJD), is primarily a disorder of articular cartilage and subchondral bone, with secondary minimal inflammation of the synovial membrane. It is a localized joint disease without systemic manifestations. The process begins in loaded articular cartilage that thins, clefts (fibrillation), and then fragments leading to sclerosis of underlying bone, subchondral cysts,

and osteophyte formtion. The articular changes are essentially a response of the joint to chronic microtrauma or pressure. Microtrauma may be in the form of continuous abrasion of the articular surfaces as in natural wear associated with age or due to increased loading related to chronic parafunctional activity. The fibrous tissue covering in patients with degenerative disease is preserved. This may be a factor in remodeling and the recovery that is usually expected in osteoarthrosis and osteoarthritis of the TMJs.[93]

Osteoarthritis presentation:-

The patients who develop (OA) present with a variety of symptoms including pain on opening, limited movement to the opposite side, coarse grinding noise on function, history of clicking that has now stopped, and deviation on opening to the affected side. An unusually large percentage of those diagnosed are women around the age of 35. In addition patients have had a macrotrauma usually from a maximal voluntary contraction (MVC) force or even a blow to the mandible. The clinical findings are pain on palpation of lateral pole, decreased range of motion findings , heavy occlusion on second molar on the affected side, facial asymmetry, and tipped Curve of Wilson. Some other indicators include loss of condylar bone which traumatizes the posterior molar on the same side, pain referral pattern to the ear, pain on eating, talking, or function of the jaw joint, jaw locking, and pain in the front tooth of a bridge (due to torque forces on two molars). In summary, a picture of pain, dysfunction, and disability is involved in osteoarthritis of jaw joint. with flattened condyle, osteophytes on condyle(could be noticed by Xray findings,[94]

1.2.3 : Rheumatoid arthritis (RA)

Is a chronic, systemic, autoimmune inflammatory disorder that is characterized by joint inflammation, erosive properties and symmetric multiple joint

involvement. Temporomandibular joint (TMJ) is very rare to be affected in the early phase of the disease, thus posing diagnostic challenges for the dentist. Conventional radiographs fail to show the early lesions due to its limitations. More recently cone-

beam computed tomography (CBCT) has been found to diagnose the early degenerative changes of TMJ and hence ,aid in the diagnosis of the lesions more accurately.

Some common clinical symptoms of Rheumatoid arthritis include TMJ sounds/noises, TMJ pain, facial pain, headaches, limited range of mandibular movement, change in occlusion, masticatory difficulty, earaches, tinnitus, vertigo, and neck, shoulder, and back pain. Some patients may have pathological internal derangement of the TMJ, however, are asymptomatic or have relatively innocuous clinical symptoms,[95]

1.2.4: Infection arthritis:-

Infection arthritis of the temporomandibular joint (TMJ) may result from direct extension of adjacent infection or hematogenous spread of bloodborne organism. The area is inflamed, and jaw movement is limited and painful. Local signs of infection associated with evidence of a systemic disease or with an adjacent infection suggest the diagnosis. X-Ray results are negative in the early stages but may show bone destruction later. If suppurative arthritis is suspected, the joint is aspirated to confirm the diagnosis and to identify the causative organism(diagnosis must be made rapidly to prevent permanent joint damage). Treatment includes antibiotics, proper hydration, pain control, and motion restriction. Parenteral penicillin G is the drug of choice until a specific bacteriologic diagnosis can be made on the basis of culture and sensitivity testing. For methicillin-resistant Staphylococcus aureus (MRSA) infections of the oral structures, IV vancomycin is the antibiotic of choice. Suppurative infections are aspirated or incised. Once the infection is controlled, passive jaw-

opening exercises help prevent scarring and limitation of motion. The Most common symptoms include , difficulty in mouth opening due to pain, fibrous adhesions, anterior disc displacement, muscle contracture, inflammation, or more severe degeneration,[96]

1.2.5 : Traumatic arthritis:

Rarely, acute injury (eg, due to difficult tooth extraction or endotracheal intubation) may lead to arthritis of the TMJ. Pain, tenderness, and limitation of motion occur. Diagnosis is based primarily on history. X-ray results are negative except when intra-articular edema or hemorrhage widens the joint space. Treatment includes NSAIDs, application of heat, a soft diet, and restriction of jaw movement.[97]_

1.2.6: Osteochondrosis Dissecans:

Osteochondritis dissecans is a disorder of unclear pathophysiology wherein fragments of articular cartilage and bone freely move within the Synovial fluid ("joint mice"). It usually occurs in the knee and elbow and is often related to sports. Reports have described this condition in the TMJ but little is known about the signs and symptoms. History is positive for arthralgia as previously defined and joint noises with mandibular movement or swelling. Examination is positive for similar clinical findings as operationalized for arthralgia, or crepitus detected by the examiner during palpation or reported by patient during mandibular movements or maximum assisted opening with vertical overlap ,[98].

1.3.Neoplasm:-

A neoplasm is new, often uncontrolled growth of abnormal tissue arising or involving the TMJ or supporting structures. Tumors of the TMJ are rare, can be malignant or benign, and present with symptoms similar to intra-articular disorders. Occasionally metastatic tumors have also been reported. Presenting symptoms include reduced mouth opening which is progressive, joint pain, malocclusion, swelling in the TMJ region, skin reactions in the TMJ region,

lymphadenopathy, and crepitus. If the condyle is involved, there is frequently development of a facial asymmetry with a midline shift as that noticed in condylar hyperplasia. Diagnostic imaging and biopsy are essential when a neoplasm is suspected. Treatment options include surgery, radiotherapy, and chemotherapy.[99]_

1.4. Congenital/developmental disorders

14.1. Hyperplasia

Is the overdevelopment of the mandible or cranial bones that occurs unilaterally or bilaterally as a localized enlargement such as condylar hyperplasia or as an overdevelopment of the entire mandible or side of the face. Hyperplasia normally occurs during adolescence, leading to facial asymmetry, mandibular deviation, and a malocclusion . Facial asymmetry resulting from excessive condylar growth is of two types :

- Type I: Hemimandibular hyperplasia (HH).
- Type II: Hemimandibular elongation (HE).

To diagnose hyperplasia, the history must be positive for progressive development of mandibular or facial asymmetry, and the examination must confirm this history. Imaging using panoramic radiography and/or CT/CBCT and single-photon emission CT is positive for asymmetry in mandibular ramus height and there is an increased uptake of technetium-99 m hydroxy diphosphonate on bone scintigraphy scan (nuclear imaging).[100]

1.4.2. Hypoplasia :-

An incomplete development or underdevelopment of the cranial bones or the mandible occurs often secondary to trauma during adolescence. It may result in asymmetric growth of the mandible, and may be associated with malocclusion that includes open bite. History must be positive for progressive development of mandibular asymmetry or micrognathia from birth or early childhood and development of malocclusion, which may include posterior open bite. Examination

must confirm this history. Imaging using CT/CBCT will show at least one of the following:

hypoplasia of the fossa, hypoplasia of the condyle, or shortened mandibular ramus height.[101]

1.4.3. Aplasia :-

A failure of the condyle to develop or incomplete development of the articular fossa and eminence is usually associated with congenital anomalies such as oculo-auriculo-vertebral spectrum (Goldenhar syndrome), hemifacial microsomia, and mandibulofacial dysostosis (Treacher Collins syndrome). Such aplasia is unilateral, causing facial asymmetries, and might cause a malocclusion. In rare occasions, it may be bilateral, without facial asymmetry, but with a definitive micrognathia and open bite. History must be positive for progressive development of mandibular asymmetry or micrognathia from birth or early childhood and development of malocclusion, which may include posterior open bite. Examination reveals mandibular asymmetry, with deviation of the chin to the affected side or micrognathia and inability to detect the condyle upon palpation during mandibular movements. Imaging will show severe hypoplasia of the fossa and eminence and aplasia of the condyle ,[102].

1.5 : Masticatory Muscle Disorders:-

1.5.1: Trismus :-

Also called locked jaw, is reduced opening of the jaws (limited jaw range of motion). It may be caused by spasm of the muscles of mastication or a variety of other causes. Usually temporary trismus occurs much more frequently than permanent trismus. It is known to interfere with eating, speaking, and maintaining proper oral hygiene. This interference, specifically with the patient's ability to swallow properly, results in an increased risk of aspiration. In some instances, trismus presents with altered facial appearance. The condition may be distressing and painful for the patient. Examination and treatments requiring access to the oral cavity can be limited, or in some cases impossible,due to the nature of the condition itself,[103]

1.5.2: Myalgia :-

Myofascial pain or myalgia is the most common muscle disorder characterized by pain and dysfunction that arises from pathologic and functional processes in the masticatory muscles. It is diagnosed when the patient's muscle pain is aggravated by mandibular movement, function, or parafunction and can be reproduced by palpating the painful muscles such as temporalis or masseter. Types: Myalgias can be acute or chronic and are of three subtypes,1[04]

- Local myalgia.
- Myofascial pain with spreading.
- Myofascial pain with referral.

1.5.3: Tendonitis

Tendonitis involves pain of tendon origin aggravated by mandibular movement, function, or parafunction, and it can be reproduced by provocation testing of the painful tendon. Limitation of mandibular movements secondary to pain may be present. The only masticatory muscle tendon that can be palpated separately from the muscle is the temporalis muscle tendon, which can be palpated intraorally. Also, the temporalis tendon is a common site of tendonitis with referred pain to the teeth or other structures,105_(Nakano et al., 2009;Fricton et al.,1985).

1.5.4: Spasm

Spasm refers to the sudden, involuntary, reversible tonic contraction of a muscle that is diagnosed when the muscle meets the criteria for myalgia, it causes a limited range of motion. The pain and limited range of motion had an immediate onset. Acute malocclusion may be present. Certain local muscle conditions known to predispose to muscle spasm include muscle fatigue, alteration in local electrolyte balance, and deep

pain. The patient usually complains of inability to put the ipsilateral posterior teeth together without excruciating pain (the first tooth contact is in the area of the contralateral canine) and a difficulty in translating the condyle forward leading to a marked limited opening. To diagnose a spasm, the patient must report immediate onset of muscle pain modifed by function and parafunction as operationalized in myalgia and immediate report of limited range of jaw motion,[106]

1.5.5: Myositis

Myositis is diagnosed when the muscle meets the criteria for myalgia and has clinical characteristics of infammation or infection: edema, erythema, and/or increased temperature. Onset of symptoms is usually acute, related to direct

trauma to the muscle or infection of the muscle from orodental causes such as pericoronitis or cellulitis, or it can occur chronically from an autoimmune disease. To diagnose myositis, the patient must have local myalgia, and the examination of the temporalis or masseter muscle must confrm both of the following:

- Local myalgia.
- Presence of edema, erythema, and/or increased temperature over the muscle,[107]

15.6. Myofascial Pain with Referral:

Myofascial pain with referral is diagnosed when the disorder meets the criteria for myalgia and the referral of pain beyond the boundary of the masticatory muscles being palpated. To diagnose myofascial pain with referral, the patient must have myalgia, and the examination of the temporalis or masseter muscle must confrm both of the following:

- Familiar muscle pain with palpation.
- Pain with muscle palpation beyond the boundary of the muscle.

Other masticatory muscles may be examined as required. Though it is not signifcant to differentiate between local myalgia and myofascial pain with spreading, when a

patient’s pain is due to referred pain from a muscle, it should be diagnosed as myofascial pain with referral. [108]

1.6: Management of the TMD

1.6.1 : Non-pharmacologic Management:

Patient education is the recommended initial treatment for TMD. Adjunctive measures include jaw rest, soft diet, moist warm compresses and passive stretching exercise . On the other hand TMJ immobilization has shown no benefit and may worsen symptoms as a result of muscle contractures,

Muscle fatigue, and reduced synovial fluid production. Patients should be counseled on behavior modifications such as stress reduction,elimination of parafunctional habits (e.g., teeth Grinding, pencil or ice chewing, teeth clenching), and avoidance of extreme mandibular movement (e.g., excessive opening during yawning, tooth brushing, and flossing)[109],(Bordoni et al.,2019

a)Physical Therapy:

There is evidence that supports the use of physical therapy for improving symptoms associated with TMD. Techniques may be Active or passive (e.g., scissor opening with fingers, use of medical devices) with the goal of improving muscle strength, coordination, relaxation, and range of motion. Specialized physical therapy options such as ultrasound, , electrotherapy, or low-level laser therapy have been used in the management of TMD[110] .(Bender et al.,2018)

b)Acupuncture:

Acupuncture is a commonly used strategy for pain relief in which an acupuncture needle or, more often acupuncture needles are inserted around the ear, face and jaw, and the trigger points are the masseter, the lateral pterygoid, the medial pterygoid, and

the temporalis muscles. Acupuncture can help with muscle relaxation and reduce muscle spasms of the TMJ[111] .(Lee & Ernst, 2011)

1.6.2: Pharmacological Management :

Pharmacologic treatments for TMD are largely based on expert opinion. Several classes of medication are used to treat the underlying pain associated with TMD. Nonsteroidal anti-inflammatory drugs (NSAIDs; including salicylates and Cyclooxygenase inhibitors), benzodiazepines, anti-epileptic agents, and muscle relaxant. Despite the multiple choices of NSAIDs available, only naproxen (Naprosyn) has proven benefit in reduction of pain ,also muscle relaxants can be

prescribed with NSAIDs if there is evidence of a muscular component to TMD .

Tricyclic antidepressants—most commonly amitriptyline, desipramine (Norpramin), doxepin, and nortriptyline (Pamelor)—are used for the management of chronic TMD pain. Benzodiazepines are also used, but are generally limited to two to four weeks in the initial phase of treatment ,diazepam [Valium], Clonazepam [Klonopin], gabapentin [Neurontin] may provide more benefit than Shorteracting agents. Opioids are not recommended and, if prescribed, should be used for a short period in the setting of severe pain[112](Kalaykova et al.,2011;Michael et al.,2021

1.6.3: Dental occlusal splinting and permanent occlusal adjustment :

Occlusal splint has been the mainstays of TMJ disorder treatment. It can be defined as “the art and science of establishing neuromuscular harmony in the masticatory system by creating a mechanical disadvantage for parafunctional forces with removable Appliances.” . Other goals of treatment are to improve jaw-muscle function and to relieve associated pain by creating a stable balanced occlusion. Two main types of splinting are available occluding and non- occluding Occluding splints, also called stabilization splints are specially fabricated to improve the alignment of the upper and lower teeth. Non-Occluding splints ,also called simple splints ,primarily open the jaw to release muscle tension ,and prevent the teeth clenching, usually made of a soft –

vinyl and are easier and cheaper to fabricate[113].(Bender et al.,2018;Michael et al.,2021)

1.6.4: Surgical intervention :

Surgical intervention is appropriate only when[114]: (Michael et al.,2021)

a) There is identifiable pathology amenable to surgical intervention.

b) There is resultant loss of mechanical function

c) There is pain related to joint pathology

1.7: Aim of study:

are to determine the severity of TMD according to demographic data in a group of Dental clinics patients at the Faculty of Dentistry /university of Babylon.

Chapter Two
Patients and Methods

2.1: Study design and setting

This is a cross sectional study was conducted in the maxillofacial department in college of dentistry during the period from first of December 2023 to first of April 2024

2.2: Ethical approval:

Ethical approval according to standard criteria of Ministry of Higher Education and Scientific Research. Main objectives of current study were described and explained, and verbal consent was taken from all participants.

Table 2.1: Instruments, equipment, and their producing-company and original country.

NO.	Instruments	Company	Source
1-	mirror	winway	China
2-	towle	winway	china
3-	ruler	winway	china

2.3: Patient selection

the Study involved 60 Referred patients in Dental clinics, their age ranged from(20-37 years) , those patients were presented with lower TMJ pain .

All cases were assessed by history taking and physical examination. History taken according to well-structured questionnaire according to Fonseca anamnestic index

2.4: Examination of TMJ disorders :

1. Extra oral examination :

- Assymetery
- Color of the face
- Presence of scar…etc
- Palpation of the muscles of mastication
- Digital examination of the TMJ
- Auscultation of the Joint

2. Intra oral examination :

- Soft tissue condition
- Teeth and jaws relation
- Mouth opening and jaw movements

2.4.1: Examination componnet of masticatory system of TMD and Observations:

- Inspection : Facial asymmetry, swelling, and masseter and temporal muscle hypertrophy Opening pattern (corrected and uncorrected deviations, uncoordinated movements,
- General palpation : Parotid and submandibular areas Lymph nodes. Mandibular range of motion (Vertical jaw movements: pain-free opening, maximal opening with pain, and maximal assisted opening Horizontal jaw movements:

lateral and protrusive movements Pain provocation, location, and replication are assessed with each movement.

- TMJ noises : Any noise produced by vertical or horizontal movements, as reported by the patient and as identified as to Type by the examiner (e.g., click vs. crepitus), any pain and replication with noise, and any locking
- Palpation for pain : Masticatory muscles ,Temporomandibular joints
- movement to pressure)Dynamic pain test (active mandibular movement Additional provocation Tests as indicated Static pain test (no mandibular against resistance)Pain in the joints or muscles with tooth clenching or unilateral biting Reproduction of symptoms with chewing (wax, sugarless gum)
- Other systems :Cervical ROM Palpation for pain of neck muscles and accessory muscles of mastication ,Neurologic screening, sensory testing
- Intraoral examination :Signs of parafunction: cheek or lip biting, accentuated linea alba, occlusal wear ,Dental pathology: tooth mobility, percussion, thermal testing.

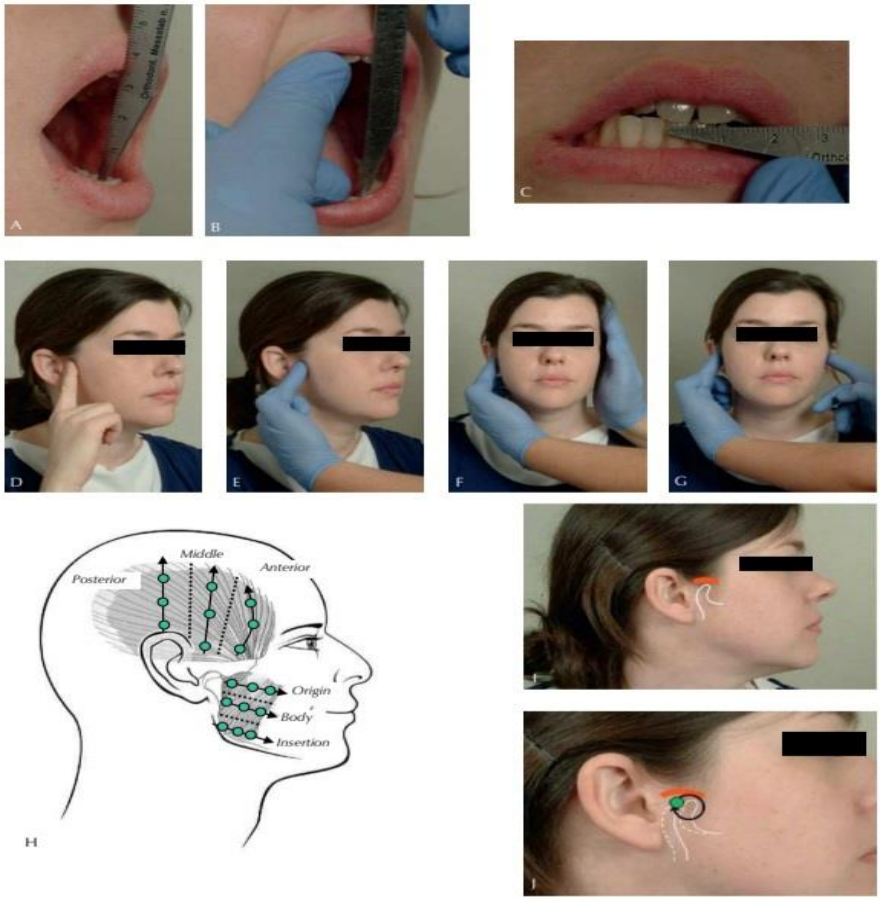

Figure 2.1 The core components of the clinical examination. (a) Measuring pain-free opening and maximal unassisted (active)Opening. (b) Measuring maximal assisted (passive) opening. (c) Measuring right lateral movement of the mandible; this is repeated forMovement of the mandible to the left, and then in protrusive. (d) Patient points to location of pain, which is asked after each of maximalUnassisted and maximal assisted opening, right and left lateral movement, and protrusive. € Examiner confirms structure that thePatient had pointed to as painful from the range of motion procedure. (f) Position of hands for palpation of single TMJ during range of Motion. (g) Position of hands for simultaneous bilateral palpation of the TMJ during range of motion. (h) Location of palpation sites for palpation for pain of the temporalis and masseter muscles. (i) Visualization of lateral pole for palpation for pain of the lateral pole; finger is placed directly on the pole. (j) Visualization of starting finger position (green dot) for circumpolar palpation for pain of the TMJ;the finger rotates around the condyle, as shown by the black arrow. Source: Illustrations and photos are adapted from Ohrbach et al,.Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) Clinical Examination Protocol

2.5: Diagnosis of TMJ severity

Diagnosis of TMJ disorder were depended on Fonseca anamnestic index (FAI)

The FAI was used to determine the severity of signs and symptoms of TMD.The FAI comprised 10 questions concerning pain (TMJ, masticatory muscle, neck pain, and headache) and function-related (TMJ sounds, opening and jaw movement difficulties) TMD symptoms as well as risk factors (teeth clenching, perception of malocclusion [poor bite], and emotional stress). The items were scored on a 3-point response scale with no = 0 points, sometimes= 5 points, and yes =10 points. Total scores for all 10 items were calculated, and subjects were allocated afterward into the ‘‘no TMDs’’ (NT; total scores ≤15 points) and ‘‘with TMDs’’ (WT; total scores ≥ 20 points) groups. The WT group was further categorized into ‘‘mild’’ (20 to 40 points), ‘‘moderate’’ (45 to 65 points), and ‘‘severe’’ (70 to 100 points) TMD . The sum of points allows the classification of TMD severity as absent (0–15 points), mild (20–45 points), moderate (50–65 points), or severe (70–100 points) An examiner instructed the volunteers how to fill out the questionnaire. Each volunteer answered the questionnaire independently in a well-lit, climate-controlled room with no time constraint.

2.6: Statistical analysis

Statistical analysis of data are calculated by using SPSS version 26 software. Continuous data were expressed as means±SD as the median and .compared their significant level by using one-way analysis of

variance (ANOVA) for normally distributed data for 4 categories groups. Categorical variables are presented as absolute numbers and percentages and compared their significant level by Pearson's chi-squared test . Kolmogorov-Smirnov test is used to determine the normality distribution of data. P value of less than 0.05 was considered significant.

Chapter Three

Results

A cross sectional research which included 42 participants.

In analysis of demographic data (as seen in table 3.1), 10-20 age group exhibited 100% in no , moderate and sever TMD and 96.2% in mild TMD with no statistical significant difference (p value= 0.889)

Similarities , sex group showed insignificant association (p value = 0.824) , with regarding more presenting female percentage 54% and 75% mild and sever TMD groups respectively while male showed more percentage 46% and 67% in mild and no TMD.

Table 3.1: General demographic descriptive data in patients with TMD regarding its severity.

parameters		patients group with TMD (n = 42)				
		No TMDs (n =6)	mild TMDs (n =26)	moderate TMDs (n =6)	severe TMDs (n =4)	*p value
		no. (%)	no. (%)	no. (%)	no. (%)	
Age /years	10-20	6(100%)	25(96.2%)	6(100%)	4(100%)	0.889
	21-40	0	1(3.8%)	0	0	
Sex	Male	4(67%)	12(46%)	3(50%)	1(25%)	0.824
	Female	2(33%)	14(54%)	3(50%)	3(75%)	

*Chi-Square Test

The mean age group (25.5±6.38) showed more value moderate TMD with insignificant association (p value = 0.561) as presenting in table 3.2

Table 3.2: Comparison age factor and Fonseca anamnestic index (FAI) score according to TMD.

parameters	patients group with TMD (n = 42)				
	No TMDs (n =6)	mild TMDs (n =26)	moderate TMDs (n =6)	severe TMDs (n =4)	*P value
	mean±SD	mean±SD	mean±SD	mean±SD	
Age /years	22.5±4.183	24.65±5.396	25.5±6.38	21.75±2.36	0.561

*One way anova test

However, Evaluation of TMD site , No TMDs were more in right and left temporalis muscle (83% and 17% respectively), mild TMDs were higher presenting in both right and left masseter (17%) and right and left temporomandibular joint (24% and 17% respectively), moderate TMDs were more left and right temporalis (28.5%) and right masseter (28.5%), and finally , sever TMD had more in left temporomandibular joint (40%) as illustrated in table 3.3.

Table 3.3: Comparison clinical data between variable severity level of patients with site of TMD .

parameters	No TMDs (n =6)	mild TMDs (n =29)	moderate TMDs (n =14)	severe TMDs (n =5)
	no. (%)	no. (%)	no. (%)	no. (%)
LM	0	5(17%)	2(15.5%)	0
RM	0	5(17%)	4(28.5%)	0
LT	1(17%)	3(11%)	4(28.5%)	1(20%)
RT	5(83%)	4(14%)	4(28.5%)	1(20%)
LTMJ	0	5(17%)	0	2(40%)
RTMJ	0	7(24%)	0	1(20%)

LM: left masseter; RM: right masseter; LT:left temporalis muscle; RT: right temporalis muscle; LTMJ: left temporomandibular joint; RTMJ: right temporomandibular joint.

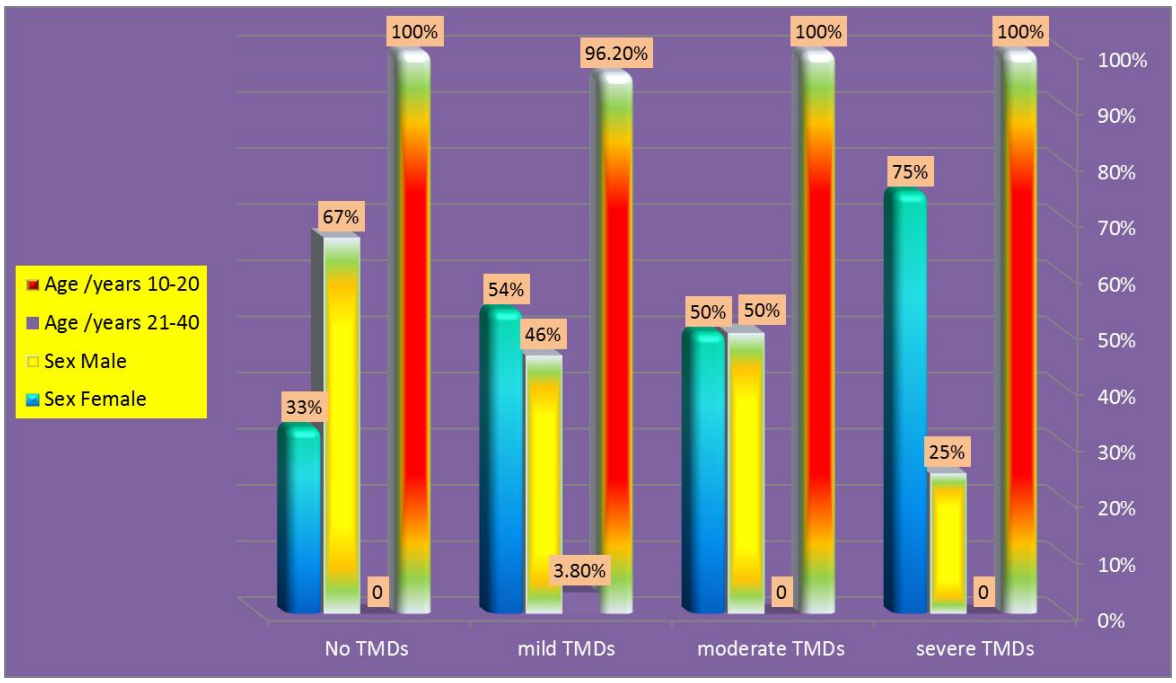

Figure 3.1; Percentage of patients with TMD regarding its severity according to age and sex.

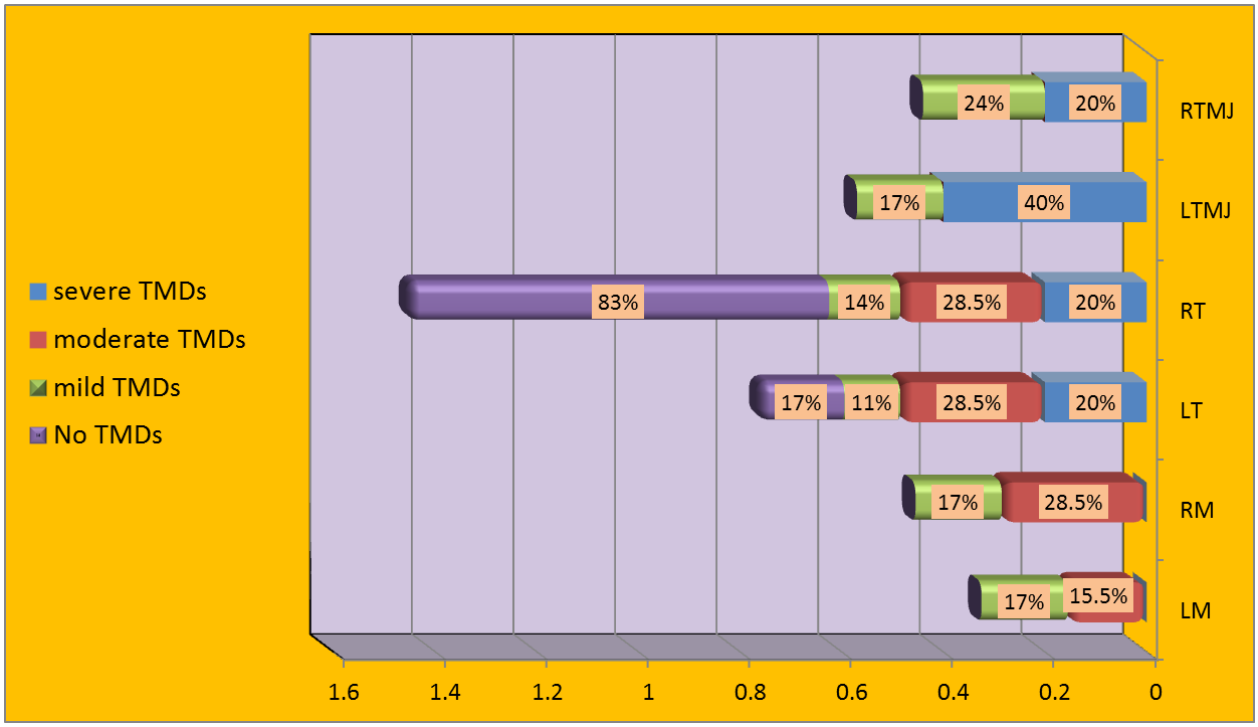

Figure 3.2: distribution of variable severity level of patients according to site of TMD .

Chapter Four
Discussion

Discussion

Temporomandibular disorder (TMD) is characterized by myofascial and/or joint pain, joint noises, lack of motor coordination, and a limited range of mandibular motion [115]. Conceptually, TMD is a complex pathological condition that can affect different structures of the stomatognathic system, such as the temporomandibular joint (TMJ) and masticatory muscles[116].

the present study. The literature contains not significant findings regarding association between degree of severity of temporomandibular disorder and sex and age groups, in which, there were mostly all ranges of FAI score presented in age group ranging between 10-20 years old , however sex male group were mostly presented in no TMD score while female sex group were presented in mild and sever TMD score

this finding were agree with Adrian Ujin Yap et al study [117]

in which, there were Chi-square test revealed no significant difference in sex or age distribution , also were mostly no TMD and sever TMD presented in age group ranging between 10-20 years old , however sex female group were mostly presented all ranging of TMD score;

in current study, mean age group were increasing slightly with increasing severity of TMD score with no significant association

However, this were compatible with Almir Vieira et al study[118] were also showing mean age group were mean age group were increasing slightly with increasing severity of TMD score .

Lastly in this study showed distribution of pain sites, in which, the masseter muscle were finding in mild and moderate TMD score,

temporalis muscle were presented in all ranging of severity score, and temporomandibular joint had finding in mild and sever TMD score.

In conclusion, The present findings demonstrate an no association between FAI score and demographic data over the TMJ, as determined , there were a divergence findings regarding distribution of sex and age in comparison to degree of severity of FAI score.
severe TMD exhibited mostly in younger and female sex group.
Regarding distribution of TMD sites , temporalis and masseter muscles were more presented in mild and moderate TMD score, while sever TMD score were presented in sever TMJ

References

References:

1. Suvinen TI, Reade PC. Temporomandibular disorders: a critical review of the nature of pain and its assessment. J Orofac Pain. 1995;9:317–339.
2. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? J Prosthet Dent. 1994;72:29–38.
3. Lupton DE. Psychological aspects of temporomandibular joint dysfunction. J Am Dent Assoc. 1969;79:131–136.
4. Yap AU, Dworkin SF, Chua EK, List T, Tan KB, Tan HH. Prevalence of temporomandibular disorder subtypes, psychologic distress, and psychosocial dysfunction in Asian patients. J Orofac Pain. 2003;17:21–28.
5. Yap AU, Tan KB, Chua EK, Tan HH. Depression and somatization in patients with temporomandibular disorders. J Prosthet Dent. 2002;88:479–484.
6. Steed PA, Wexler GB. Temporomandibular disorders—traumatic etiology vs. nontraumatic etiology: a clinical and methodological inquiry into symptomatology and treatment outcomes. Cranio. 2001;19:188–194. [PubMed]
7. Ferrando M, Andreu Y, Galdon MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98:153–60.
8. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007;8:437–442.
9. errando M, Andreu Y, Galdon MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98:153–60.
10. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007;8:437–442.

11. De Coster PJ, Van den Berghe LI, Martens LC. Generalized joint hypermobility and temporomandibular disorders: inherited connective tissue disease as a model with maximum expression. J Orofac Pain. 2005;19:47–57.
12. Kavuncu V, Sahin S, Kamanli A, Karan A, Aksoy C. The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome. Rheumatol Int. 2006;26:257–260.
13. List T, Dworkin SF. Comparing TMD diagnoses and clinical findings at Swedish and US TMD centers using research diagnostic criteria for temporomandibular disorders. J Orofac Pain 1996; 10: 240–253.
14. Schiffman EL, Truelove EL, Ohrbach R, et al. The research diagnostic criteria for temporomandibular disorders. I: Overview and methodology for assessment of validity. J Orofac Pain 2010; 24: 7–24.
15. Anastassaki Kohler A, Hugoson A, Magnusson T. Prevalence of symptoms indicative of temporomandibular disorders in adults: Cross-sectional epidemiological investigations covering two decades. Acta Odontol Scand 2012; 70: 213–223.
16. List T, Wahlund K, Wenneberg B, et al. TMD in children and adolescents: Prevalence of pain, gender differences, and perceived treatment need. J Orofac Pain 1999; 13: 9–20.
17. Ahmad M, Hollender L, Anderson Q, et al. Research diagnostic criteria for temporomandibular disorders (RDC/TMD): Development of image analysis criteria and examiner reliability for image analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009; 107: 844–860.
18. Slade GD, Ohrbach R, Greenspan JD, et al. Painful temporomandibular disorder: Decade of discovery from OPPERA studies. J Dental Res 2016; 95: 1084–1092–1084–1092.
19. Velly AM, List T and Lobbezoo F. Comorbid pain and psychological conditions in patients with orofacial pain. In: Sessle BJ (ed) Orofacial pain. Recent advances in assessment, management, and understanding of mechanisms.Washington, DC: IASP Press, 2014, pp.53–73.

20. Drangsholt M Temporomandibular Pain. In: Crombie I, Croft P, Linton S, et al. (eds). Epidemiology of pain, Seattle: IASP Press, 1999, pp. 203–234.
21. Nilsson IM, List T, Drangsholt M. Prevalence of temporomandibular pain and subsequent dental treatment in Swedish adolescents. J Orofac Pain 2005; 19: 144–150.
22. Macfarlane TV, Glenny AM, Worthington HV. Systematic review of population-based epidemiological studies of oro-facial pain. J Dentistry 2001; 29: 451–467.
23. Nilsson IM, List T, Drangsholt M. Incidence and temporal patterns of temporomandibular disorder pain among Swedish adolescents. J Orofac Pain 2007; 21: 127–132.
24. Slade GD, Bair E, Greenspan JD, et al. Signs and symptoms of first-onset TMD and sociodemographic predictors of its development: The OPPERA prospective cohort study. J Pain 2013; 14: T20–32 e1-3.
25. LeResche L, Mancl LA, Drangsholt MT, et al. Predictors of onset of facial pain and temporomandibular disorders in early adolescence. Pain 2007; 129: 269–278.
26. Slade GD, Ohrbach R, Greenspan JD, et al. Painful temporomandibular disorder: Decade of discovery from OPPERA studies. J Dent Res 2016; 95: 1084–1092.
27. Nilsson IM and List T. 10-year longitudinal follow-up of 2,255 adolescents screened for TMD pain. Int Ass Dent Res, Seoul 2016; abstr nr 1458.
28. Maixner W, Diatchenko L, Dubner R, et al. Orofacial pain prospective evaluation and risk assessment study – the OPPERA study. J Pain 2011; 12: T4–11 e1-2.
29. Smith SB, Maixner DW, Greenspan JD, et al. Potential genetic risk factors for chronic TMD: Genetic associations from the OPPERA case control study. J Pain 2011; 12: T92–101.
30. Smith SB, Mir E, Bair E, et al. Genetic variants associated with development of TMD and its intermediate phenotypes: The genetic architecture of TMD in the OPPERA prospective cohort study. J Pain 2013; 14: T91–101 e1-3.
31. Visscher CM, Lobbezoo F. TMD pain is partly heritable. A systematic review of family studies and genetic association studies. J Oral Rehab 2015; 42: 386–399.

32. LeResche L, Mancl L, Sherman JJ, et al. Changes in temporomandibular pain and other symptoms across the menstrual cycle. Pain 2003; 106: 253–261.
33. Wang J, Chao Y, Wan Q, et al. The possible role of estrogen in the incidence of temporomandibular disorders. Med Hypotheses 2008; 71: 564–567.
34. Berger M, Szalewski L, Bakalczuk M, et al. Association between estrogen levels and temporomandibular disorders: A systematic literature review. Prz Menopauzalny 2015; 14: 260–270.
35. Plesh O, Adams SH, Gansky SA. Temporomandibular joint and muscle disorder-type pain and comorbid pains in a national US sample. J Orofac Pain 2011; 25: 190–198.
36. Dominick CH, Blyth FM, Nicholas MK. Unpacking the burden: Understanding the relationships between chronic pain and comorbidity in the general population. Pain 2012; 153: 293–304.
37. Ciancaglini R, Radaelli G. The relationship between headache and symptoms of temporomandibular disorder in the general population. J Dent 2001; 29: 93–98
38. McNeill C. Craniomandibular Disorders: Guidelines for Evaluation, Diagnosis and Management. Chicago: Quintessence; 1990. pp. 25–39.
39. Almăşan OC, Băciuţ M, Almăşan HA, Bran S, Lascu L, Iancu M, et al. Skeletal pattern in subjects with temporomandibular joint disorders. Arch Med Sci. 2013;9(1):118–126.
40. Almășan OC, Băciuț M, Băciuț G. Influența disfuncției temporomandibulare asupra tiparului scheletic la subiecți cu anomalie de clasa a III-a scheletică [The influence of temporomandibular dysfunction on the skeletal pattern in patients with class 3 skeletal abnormality] Clujul Medical. 2012;85(S1):47–50.
41. Suvinen TI, Reade PC. Temporomandibular disorders: a critical review of the nature of pain and its assessment. J Orofac Pain. 1995;9:317–339.
42. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? J Prosthet Dent. 1994;72:29–38.
43. Lupton DE. Psychological aspects of temporomandibular joint dysfunction. J Am Dent Assoc. 1969;79:131–136.

44. Yap AU, Dworkin SF, Chua EK, List T, Tan KB, Tan HH. Prevalence of temporomandibular disorder subtypes, psychologic distress, and psychosocial dysfunction in Asian patients. J Orofac Pain. 2003;17:21–28.
45. Yap AU, Tan KB, Chua EK, Tan HH. Depression and somatization in patients with temporomandibular disorders. J Prosthet Dent. 2002;88:479–484.
46. Steed PA, Wexler GB. Temporomandibular disorders—traumatic etiology vs. nontraumatic etiology: a clinical and methodological inquiry into symptomatology and treatment outcomes. Cranio. 2001;19:188–194. [PubMed]
47. Ferrando M, Andreu Y, Galdon MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98:153–60.
48. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007;8:437–442.
49. De Coster PJ, Van den Berghe LI, Martens LC. Generalized joint hypermobility and temporomandibular disorders: inherited connective tissue disease as a model with maximum expression. J Orofac Pain. 2005;19:47–57.
50. Kavuncu V, Sahin S, Kamanli A, Karan A, Aksoy C. The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome. Rheumatol Int. 2006;26:257–260.
51. Le Bell Y, Jämsä T, Korri S, Niemi PM, Alanen P. Effect of artificial occlusal interferences depends on previous experience of temporomandibular disorders. Acta Odontol Scand. 2002;60:219–222.
52. Landi N, Manfredini D, Tognini F, Romagnoli M, Bosco M. Quantification of the relative risk of multiple occlusal variables for muscle disorders of the stomatognathic system. J Prosthet Dent. 2004;92:190–195.
53. McNamara JA, Jr, Seligman DA, Okeson JP. Occlusion, Orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain. 1995;9:73–90
54. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. J Prosthet Dent. 1992;67:692–696.

55. Shiau YY, Chang C. An epidemiological study of temporomandibular disorders in university students of Taiwan. Community Dent Oral Epidemiol. 1992;20:43–47
56. Connelly, S.T.; Myung, J.; Gupta, R.; Tartaglia, G.M.; Gizdulich, A.; Yang, J.; Silva, R. Clinical outcomes of Botox injections for chronic temporomandibular disorders: Do we understand how Botox works on muscle, pain, and the brain? *Int. J. Oral Maxillofac. Surg.* **2017**, *46*, 322–327. [**Google Scholar**] [**CrossRef**]

57. Kim, Y.H.; Bang, J.I.; Son, H.J.; Kim, Y.; Kim, J.H.; Bae, H.; Han, S.J.; Yoon, H.J.; Kim, B.S. Protective effects of extracorporeal shockwave on rat chondrocytes and temporomandibular joint osteoarthritis; preclinical evaluation with in vivo(99m)Tc-HDP SPECT and ex vivo micro-CT. *Osteoarthr. Cartil.* **2019**, *27*, 1692–1701. [**Google Scholar**] [**CrossRef**]

58. Schenk, I.; Vesper, M.; Nam, V.C. Initial results using extracorporeal low energy shockwave therapy ESWT in muscle reflex-induced lock jaw. *Mund Kiefer Gesichtschir.* **2002**, *6*, 351–355. [**Google Scholar**] [**CrossRef**] [**PubMed**]

59. Dworkin, S.F.; Turner, J.A.; Mancl, L.; Wilson, L.; Massoth, D.; Huggins, K.H.; LeResche, L.; Truelove, E. A randomized clinical trial of a tailored comprehensive care treatment program for temporomandibular disorders. *J. Orofac. Pain* **2002**, *16*, 259–276. [**Google Scholar**]

60. Türp, J.C.; Jokstad, A.; Motschall, E.; Schindler, H.J.; Windecker-Gétaz, I.; Ettlin, D.A. Is there a superiority of multimodal as opposed to simple therapy in patients with temporomandibular disorders? A qualitative systematic review of the literature. *Clin. Oral Implant. Res.* **2007**, *18* (Suppl. 3), 138–150. [**Google Scholar**] [**CrossRef**]

61. Conti, P.C.; Correa, A.S.; Lauris, J.R.; Stuginski-Barbosa, J. Management of painful temporomandibular joint clicking with different intraoral devices and counseling: A

controlled study. *J. Appl. Oral Sci.* **2015**, *23*, 529–535. [**Google Scholar**] [**CrossRef**] [**Green Version**]

62. de Resende, C.; de Oliveira Medeiros, F.G.L.; de Figueiredo Rego, C.R.; Bispo, A.S.L.; Barbosa, G.A.S.; de Almeida, E.O. Short-term effectiveness of conservative therapies in pain, quality of life, and sleep in patients with temporomandibular disorders: A randomized clinical trial. *Cranio* **2019**, 1–9. [**Google Scholar**] [**CrossRef**]

63. de Barros Pascoal, A.L.; de Freitas, R.; da Silva, L.F.G.; Oliveira, A.; Dos Santos Calderon, P. Effectiveness of Counseling on Chronic Pain Management in Patients with Temporomandibular Disorders. *J. Oral Facial Pain Headache* **2020**, *34*, 77–82. [**Google Scholar**] [**CrossRef**]

64. Delgado-Delgado, R.; Iriarte-Álvarez, N.; Valera-Calero, J.A.; Centenera-Centenera, M.B.; Garnacho-Garnacho, V.E.; Gallego-Sendarrubias, G.M. Association between temporomandibular disorders with clinical and sociodemographic features: An observational study. *Int. J. Clin. Pract* **2021**, e13961. [**Google Scholar**] [**CrossRef**]

65. Al-Ani, Z. Occlusion and Temporomandibular Disorders: A Long-Standing Controversy in Dentistry. *Prim. Dent. J.* **2020**, *9*, 43–48. [**Google Scholar**] [**CrossRef**]

66. Manfredini, D.; Lombardo, L.; Siciliani, G. Temporomandibular disorders and dental occlusion. A systematic review of association studies: End of an era? *J. Oral Rehabil.* **2017**, *44*, 908–923. [**Google Scholar**] [**CrossRef**]

67. Kakudate, N.; Yokoyama, Y.; Sumida, F.; Matsumoto, Y.; Gordan, V.V.; Gilbert, G.H.; Velly, A.M.; Schiffman, E.L. Dentist Practice Patterns and Therapeutic Confidence in the Treatment of Pain Related to Temporomandibular Disorders in a Dental Practice-Based Research Network. *J. Oral Facial Pain Headache* **2017**, *31*, 152–158. [**Google Scholar**] [**CrossRef**] [**PubMed**]

68. Onishi, M. Arthroscopy of the temporomandibular joint (author's transl). *Kokubyo Gakkai Zasshi* **1975**, *42*, 207–213. [**Google Scholar**] [**CrossRef**] [**PubMed**] [**Green Version**]

69. Murakami, K.; Ono, T. Temporomandibular joint arthroscopy by inferolateral approach. *Int. J. Oral Maxillofac. Surg.* **1986**, *15*, 410–417. [**Google Scholar**] [**CrossRef**]

70. Sanders, B. Arthroscopic surgery of the temporomandibular joint: Treatment of internal derangement with persistent closed lock. *Oral Surg. Oral Med. Oral Pathol.* **1986**, *62*, 361–372. [**Google Scholar**] [**CrossRef**]

71. Sanders, B.; Buoncristiani, R. Diagnostic and surgical arthroscopy of the temporomandibular joint: Clinical experience with 137 procedures over a 2-year period. *J. Craniomandib. Disord.: Facial Oral Pain* **1987**, *1*, 202–213. [**Google Scholar**]

72. McCain, J.P. Arthroscopy of the human temporomandibular joint. *J. Oral Maxillofac. Surg.* **1988**, *46*, 648–655. [**Google Scholar**] [**CrossRef**

73. McCain, J.P.; Sanders, B.; Koslin, M.G.; Quinn, J.H.; Peters, P.B.; Indresano, A.T. Temporomandibular joint arthroscopy: A 6-year multicenter retrospective study of 4,831 joints. *J. Oral Maxillofac. Surg.* **1992**, *50*, 926–930. [**Google Scholar**] [**CrossRef**]

74. Reston, J.T.; Turkelson, C.M. Meta-analysis of surgical treatments for temporomandibular articular disorders. *J. Oral Maxillofac. Surg.* **2003**, *61*, 3–10. [**Google Scholar**] [**CrossRef**

75. Schiffman, E.L.; Velly, A.M.; Look, J.O.; Hodges, J.S.; Swift, J.Q.; Decker, K.L.; Anderson, Q.N.; Templeton, R.B.; Lenton, P.A.; Kang, W.; et al. Effects of four treatment strategies for temporomandibular joint closed lock. *Int. J. Oral Maxillofac. Surg.* **2014**, *43*, 217–226. [**Google Scholar**] [**CrossRef**] [**Green Version**]

76. Dimitroulis, G. Outcomes of temporomandibular joint arthroscopy in patients with painful but otherwise normal joints. *J. Craniomaxillofac. Surg.* **2015**, *43*, 940–943. [**Google Scholar**] [**CrossRef**]

77. McCain, J.P.; Hossameldin, R.H.; Srouji, S.; Maher, A. Arthroscopic discopexy is effective in managing temporomandibular joint internal derangement in patients with Wilkes stage II and III. *J. Oral Maxillofac. Surg.* **2015**, *73*, 391–401. [**Google Scholar**] [**CrossRef**]

78. Al-Moraissi, E.A. Arthroscopy versus arthrocentesis in the management of internal derangement of the temporomandibular joint: A systematic review and meta-analysis. *Int. J. Oral Maxillofac. Surg.* **2015**, *44*, 104–112. [**Google Scholar**] [**CrossRef**]

79. Liu, X.; Zheng, J.; Cai, X.; Abdelrehem, A.; Yang, C. Techniques of Yang's arthroscopic discopexy for temporomandibular joint rotational anterior disc displacement. *Int. J. Oral Maxillofac. Surg.* **2019**, *48*, 769–778. [**Google Scholar**] [**CrossRef**]

80. Machoň, V.; Levorová, J.; Hirjak, D.; Beňo, M.; Drahoš, M.; Foltán, R. Does arthroscopic lysis and lavage in subjects with Wilkes III internal derangement reduce pain? *Oral Maxillofac. Surg.* **2021**. [**Google Scholar**] [**CrossRef**] [**PubMed**]

81. Nitzan, D.W.; Dolwick, M.F.; Martinez, G.A. Temporomandibular joint arthrocentesis: A simplified treatment for

82. severe, limited mouth opening. *J. Oral Maxillofac. Surg.* **1991**, *49,* 1163–1167. [**Google Scholar**] [**CrossRef**

83. De Leeuw, R., & Klasser, G. D. (Eds.). (2018). Orofacial pain: guidelines for assessment, diagnosis, and management. Hanover Park, IL: Quintessence Publishing Company, Incorporated.

-Demir, E., Cary, M. P., Paley, S., Fukuda, K., Lemer, C., Vastrik, I., … & Bader, G. D. (2010). The BioPAX community standard for pathway data sharing. Nature biotechnology, 28(9), 935-942.

-Detamore, M. S., & Athanasiou, K. A. (2003). Motivation, characterization, and strategy for tissue engineering the temporomandibular joint disc. Tissue engineering, 9(6), 1065-1087

84-Rinchuse, D. J., & Kandasamy, S. (2010). Orthodontics and TMD management. In Current concepts on temporomandibular disrorders (pp. 429-446). Quintessence, Chicago.

85_0Kalaykova, S. I., Lobbezoo, F., & Naeije, M. (2011). Risk factors for anterior disc displacement with reduction and intermittent locking in adolescents. Journal of orofacial pain, 25(2), 153.

86_Kalaykova, S. I., Lobbezoo, F., & Naeije, M. (2011). Risk factors for anterior disc displacement with reduction and intermittent locking in adolescents. Journal of orofacial pain, 25(2), 153.

87_Okeson, J. P., & de Leeuw, R. (2011). Differential diagnosis of temporomandibular disorders and other orofacial pain disorders. Dental Clinics, 55(1), 105-120.

88-Zhi, K., Ren, W., Zhou, H., Gao, L., Zhao, L., Hou, C., & Zhang, Y. (2009). Management of temporomandibular joint ankylosis: 11 years' clinical experience. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 108(5), 687-692

.89_ Okeson, J. P. (2019). Management of temporomandibular disorders and occlusion-E-book. Elsevier Health Sciences.

90_Okeson, J. P., & de Leeuw, R. (2011). Differential diagnosis of Temporomandibular disorders and other orofacial pain disorders. Dental Clinics, 55(1), 105-120.

91_Okeson, J. P., & de Leeuw, R. (2011). Differential diagnosis of temporomandibular disorders and other orofacial pain disorders. Dental Clinics, 55(1), 105-120.

92_Okeson, J. P. (2008). The classification of orofacial pains. Oral and maxillofacial surgery clinics of North America, 20(2), 133-144.

93_Morel, K. L., Sheahan, A. V., Burkhart, D. L., Baca, S. C., Boufaied, N., Liu, Y., … & Ellis, L. (2021). EZH2 inhibition activates a dsRNA–STING–interferon stress axis that potentiates response to PD-1 checkpoint blockade in prostate cancer. Nature cancer, 2(4), 444-456

94_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach. Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

94_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach.

Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

95_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach. Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

96_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach. Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

97_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach. Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

98_Aiyer, A. A., Granger, C. J., McCormick, K. L., Cipriano, C. A., Kaplan, J. R., Varacallo, M. A., … & Levine, W. N. (2020). The impact of COVID-19 on the orthopaedic surgery residency application process. The Journal of the American Academy of Orthopaedic Surgeons .

99_Mehrotra, D., Dhasmana, S., Kamboj, M., & Gambhir, G. (2011). Condylar hyperplasia and facial asymmetry: report of five cases. Journal of maxillofacial and oral surgery, 10(1), 50-56.

100_Mehrotra, D., Dhasmana, S., Kamboj, M., & Gambhir, G. (2011). Condylar

hyperplasia and facial asymmetry: report of five cases. Journal of maxillofacial and oral surgery, 10(1), 50-56.

101_Nakano, M., Fujita, T., Ohtani, J., Kawata, T., Kaku, M., Motokawa, M., … & Tanne, K. (2009). Effects of mandibular advancement on growth after condylectomy. Journal of dental research, 88(3), 261-265.

102_Nakano, M., Fujita, T., Ohtani, J., Kawata, T., Kaku, M., Motokawa, M., … & Tanne, K. (2009). Effects of mandibular advancement on growth after condylectomy. Journal of dental research, 88(3), 261-265.

103_Perrott, D. H. (2003). Axel Bumann, Ulrich Lotzmann, TMJ disorders and orofacial pain. The role of dentistry in a multidisciplinary diagnostic approach. Theime, Stuttgart, 1304 illustrations in colour, ISBN: 3-13-127161-2 (GTV), 1-58890-111-4 (TNY).

104_Fricton, J. R., Kroening, R., Haley, D., & Siegert, R. (1985). Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral surgery, oral medicine, oral pathology, 60(6), 615-623.

105_Nakano, M., Fujita, T., Ohtani, J., Kawata, T., Kaku, M., Motokawa, M., … & Tanne, K. (2009). Effects of mandibular advancement on growth after Condylectomy. Journal of dental research, 88(3), 261-265.

106_Fricton, J. R., Kroening, R., Haley, D., & Siegert, R. (1985). Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral surgery, oral medicine, oral pathology, 60(6), 615-623.

107_Fricton, J. R., Kroening, R., Haley, D., & Siegert, R. (1985). Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral surgery, oral medicine, oral pathology, 60(6), 615-623.

108_Fricton, J. R., Kroening, R., Haley, D., & Siegert, R. (1985). Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral surgery, oral medicine, oral pathology, 60(6), 615-623.

109-Bordoni, B., & Varacallo, M. (2019). Anatomy, head and neck, temporomandibular joint

110_-Bender, M. E., Lipin, R. B., & Goudy, S. L. (2018). Development of the pediatric temporomandibular joint. Oral and Maxillofacial Surgery Clinics, 30(1), 1-9

111-Lee MS, Ernst E. 2011. Acupuncture for pain: an overview of Cochrane reviews. Chin J Integr Med ;17:187–9

112-Kalaykova, S. I., Lobbezoo, F., & Naeije, M. (2011). Risk factors for anterior disc displacement with reduction and intermittent locking in adolescents. Journal of orofacial pain

113-Bender, M. E., Lipin, R. B., & Goudy, S. L. (2018). Development of the pediatric temporomandibular joint. Oral and Maxillofacial Surgery Clinics, 30(1), 1-9

114--Michael Glick, DMD, Martin S. Greenberg, Peter B. Lockhart, Stephen J. Challacombe,13 Ed.2021

115- (Manfredini, D., Guarda-Nardini, L., Winocur, E., et al, 2011. Research diagnostic criteria for temporomandibular disorders: a systematic review of axis I epidemiologic findings. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 112 (4), 453–462.).

116 - Leeuw, R., 2008. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management, fourth ed. Quintessence, Chicago).

117 - Yap, A. U., Chen, C., Wong, H. C., Yow, M., & Tan, E. (2021). Temporomandibular disorders in prospective orthodontic patients: Their association with malocclusion severity and impact on oral health–related quality of life. The Angle Orthodontist, 91(3), 377-383..)

118 - Dibai-Filho, A. V., de Souza Costa, A. C., Packer, A. C., de Castro, E. M., & Rodrigues-Bigaton, D. (2015). Women with more severe degrees of temporomandibular disorder exhibit an increase in temperature over the temporomandibular joint. The Saudi Dental Journal, 27(1), 44-49.)