INTRODUCTION

Temporomandibular joint disorder (TMD) is a generic term to describe a group of disorders or diseases affecting masticatory muscles, the Temporomandibular joint (TMJ) and associated structures [1]. Epidemiologic studies show that approximately 75% of population have some TMD sign while 33% have at least one symptom. [2]

The temporomandibular joints are complex structures containing muscles, tendons, and bones. Injury to or disorders of these structures can all result in pain in the jaw area. Jaw pain may occur on one side or on both sides, depending upon the cause.

The temporomandibular joint is different from the body's other joints. The combination of hinge and sliding motions makes this joint among the most complicated in the body.

Temporomandibular Disorders (TMD) is a collective term embracing all the problems relating to Temporomandibular joint (TMJ) and related musculoskeletal masticatory structures [3]. It refers to a cluster of disorders characterized by pain in the preauricular region, pain in TMJ, or the masticatory muscles, limitation or deviations in mandibular range of motion and noises in the TMJ during mandibular function. TMJ disorders are also sometimes referred to as Myofascial pain dysfunction, Craniomandibular Disorder and Costen's syndrome. [4]

The Temporomandibular Joint (TMJ) is the site of articulation between the mandible and the skull, specifically the area about the articular eminence of the temporal bone.

The articulation consists of parts of the mandible and temporal bones, which are covered by dense, fibrous connective tissue and are surrounded by several ligaments. The TMJ is a synovial, condylar and hinge-type joint with fibro cartilaginous surfaces rather than hyaline cartilage and an articular disc. The disc completely divides each joint into two cavities. The joint connects the lower jaw (mandible) to the temporal bone of the skull. [5]

The joints are flexible, allowing the jaw to move smoothly up and down and side to side and enabling one to talk, chew and yawn. Muscles attach to and surround the jaw joint to control the position and movement of the jaw. The muscles include masseter, temporalis, medial pterygoid and lateral pterygoid. [6]

TMDs can present with a multitude of signs and symptoms, which can sometimes be confusing to both the patient and clinician. While up to 25% of the population may experience symptoms of TMD, [7] only a small percentage of afflicted individuals seek treatment. For instance, studies in the 1980s detected TMD symptoms in 16% to 59% of the population [8] although only 3% to 7% of the adult population actually sought care for pain and dysfunction associated with TMD. [9] Furthermore, TMD symptoms occur disproportionately between the sexes with a much higher incidence reported in females; female to male ratios range between 2:1–8:1. [7, 10–12] Most patients presenting symptoms are between 20 and 50 years of age, [12-13].

It is important to understand that many other reasons exist for pain in the head and neck area, outside of TMJ problems. The site of the pain is not always the source of the pain. Pain originating from nerves, skeletal muscles, blood vessels, glands, skin, or teeth can all refer pain to the face.

Causes of pain may be infection, tissue trauma, nerve disease, musculoskeletal problems, psychophysiological effects, autoimmune phenomena, or even cancer. Sometimes these conditions go undetected due to inadequate examination or diagnostics. It is important to get to the root of the pain, particularly when multiple overlapping conditions confound the condition.

Disruption to the anatomy of this joint from inflammatory changes and trauma could lead to a poor quality of life mainly through pain and functional deficit.

Several studies have pointed out that persistent and recurrent pain has a potential impact on daily life- mainly in the areas of psychological discomfort, physical disability and functional limitations- which leads to limitations in quality of life.

Because muscles and joints work together, a problem with either one can lead to TMJ disorders. Teeth grinding and teeth clenching (bruxism) increase the wear on the cartilage lining of the TMJ. Many patients awaken in the morning with jaw or ear pain.

Habitual gum chewing or fingernail biting, misalignment of the teeth (malocclusion), trauma to the jaws: Previous fractures in the jaw or facial bones can lead to TMJ disorders, problematic relation between the jaw and malocclusions with deflective contacts in the teeth, stress frequently leads to unreleased nervous energy. It is very common for people under stress to release this nervous energy by either consciously or unconsciously grinding and clenching their teeth, Occupational tasks or habits such as holding the telephone between the head and shoulder may contribute to TMJ disorders. [14]

Symptoms associated with TMD are: pain or tenderness in and around the ear, difficulty chewing or discomfort while chewing, aching facial pain, locking of the joint, difficultly with opening or closing of the mouth, headaches, uncomfortable bite, or uneven bite. Clicking, popping or grating sounds in the joint with opening and closing of the mouth. Other common symptoms include toothaches, neck pain, dizziness, earaches and hearing problems. [15]

TMD handling may be simple or may require a multidisciplinary approach. Dentists, physicians, psychologists and physical therapists work together to cope with such condition afflicting patients. Clinical treatment is considered the first choice [16]. Numerous physical therapy methods are effective to treat TMD, such as moist heat, ultrasound, TENS, microwaves, laser, exercises and manual therapy techniques [17]. These methods aim at decreasing musculoskeletal load and pain effects, at decreasing inflammation, at restoring normal joint function (strength, movement and resistance) and at helping return to daily activities. [18]

The identification of an unambiguous, universal cause of TMDs is lacking due to their complex and multifactorial. Factors that increase the risk of TMDs are called predisposing factors, factors that cause the onset of TMDs are called initiating factors, and factors that interfere with healing or enhance the progression of TMDs are called

perpetuating factors. There is not a single etiologic factor or a unique theoretical model that can interpret the onset of TMDs.

There is no widely accepted, standard test now available to correctly diagnose TMJ disorders. Because the exact causes and symptoms are not clear, identifying these disorders can be difficult and confusing.

Currently, health care providers note the patient's description of symptoms, take a detailed medical and dental history, and examine problem areas, including the head, neck, face, and jaw. Imaging studies may also be recommended.

Because more studies are needed on the safety and effectiveness of most treatments for jaw joint and muscle disorders, experts strongly recommend using the most conservative, reversible treatments possible. Conservative treatments do not invade the tissues of the face, jaw, or joint, or involve surgery. Reversible treatments do not cause permanent changes in the structure or position of the jaw or teeth. Even when TMJ disorders have become persistent, most patients still do not need aggressive types of treatment.

REVIEW OF LITERATURE

1. **Zhang et al** [19] in **2017,** evaluated the prognosis of using standard alloplastic TMJ prostheses for the treatment of TMJ ankylosis in Chinese patients with severe mandibular deficiency. Patients treated from 2013 to 2015 were reviewed. The computer-aided design and manufacture (CAD/CAM) technique was used to guide bony mass removal and locate the TMJ prosthesis. Eleven patients were included in this study. All prostheses were successfully installed and stabilized intraoperatively. In 4 patients with severe mandibular deficiency, their mandibular ramus was elongated by the TMJ prosthesis and 2 patients were combined with Le Fort I osteotomy guided by digital templates. Their mean chin advancement was 10.19 mm. Their SNB and ramus heights were also significantly improved after operation ($P < 0.05$). There was no prosthesis loosening, breakage, or infection leading to removal after a mean follow-up period of 22 months (range, 12-31mos.). Mouth opening was significantly improved from 5.5 mm preoperatively to 31.5 mm postoperatively. TMJ reconstruction with standard alloplastic prosthesis is a reliable treatment for ankylosis, especially in recurrent cases. By CAD/CAM technique, it can correct jaw deformities simultaneously and produce stable results.

2. **Pahwa et al** [20] in **2015** describes the technique of multi-detector computed tomography (MDCT) of the TMJ, and describes in detail various osseous pathologic afflictions affecting the joint. Traumatic injuries affecting the mandibular condyle are most common, followed by joint ankylosis as a sequel to arthritis.

   The congenital anomalies are less frequent, hemifacial microsomia being the most commonly encountered anomaly involving the TMJ. Neoplastic afflictions of TMJ are distinctly uncommon, osteochondroma being one of the most common lesions. MDCT enables comprehensive evaluation of osseous afflictions of TMJ, and is a valuable tool for surgical planning. Sagittal, coronal and 3D reformatted images well depict osseous TMJ lesions, and their relationship to adjacent structures.

3. **Asim K Bag et al** [21] in **2014** stated that Imaging of TMJ should be performed on a case by case basis depending upon clinical signs and symptoms. MRI is the diagnostic study of choice for evaluation of disk position and internal derangement of the joint. CT scan for evaluation of TMJ is indicated if bony involvement is suspected and should be judiciously considered because of radiation risk. Understanding of the TMJ anatomy, biomechanics, and the imaging manifestations of diseases is important to accurately recognize and manage these various pathologies.

4. **Eduardo Grossmann, et al** [22] **;(2012).** This article aimed at reviewing the scientific literature on the use of TENS in Temporomandibular joint patients. TMD has different aetiologies and specific treatments, among them the transcutaneous electrical nerve stimulation (TENS) is used which administers electrical current to the skin surface, to relax hyperactive muscles and promote pain relief. And further it was concluded that although there are controversies about the use of TENS to control chronic pain, its use for masticatory muscle pain is still relevant. However, an accurate diagnosis is needed to prevent its inadequate use. There is still need of controlled randomized studies including selected samples to homogenize the use of TENS in TMD patients.

5. **Shalender Sharma, et al** [23]**; (2011).** This study concluded that there is successful management of temporomandibular disorders and is dependent on identifying and controlling the contributing factors. The temporomandibular disorders are more common in females, the reason is not clearly known.

6. **Edward F. Wright, Sarah L. North** [24]**; (2009).** This study concluded that routinely managed by medical and dental practitioners, TMD may be more effectively cared for when physical therapists are involved in the treatment process. Hence, a listing of situations when practitioners should consider referring TMD patients to a physical therapist can be provided to the practitioners in each physical therapist's region.

7. **Milan Knezvic, et al** [25] **(2008).** This study deals with conservative non-surgical treatment of painful temporomandibular joint (TMJ) syndrome administered in thirty patients. The treatment involved TENS applications, and particularly extension exercises of the masseter muscle, temporalis and pterygoid muscles, as well as the local application of ultrasound. The result was an evident improvement in a significant number of cases. Physical exercises represent a useful treatment modality for TMPDS patients. TMPDS treatment should be organized in a multidisciplinary way, with dentists, physiotherapists and psychologists being all equally involved.

8. **Waseem et al** [26] in **2007** aimed to develop a simple, cost-effective management programme for TMDs using CD-ROM. 41 patients (age 18–70) participated in this study, patients were divided into three groups: the 1st group were involved in an attention placebo CD-ROM (contain anatomical information about the temporomandibular system), the 2nd group received information on CD-ROM designed to increase their control and self-efficacy, while the 3rd group received the same programme of the 2nd group added to it an introduction to self-relaxing techniques followed by audio tape of progressive muscle relaxation exercises. Each of the groups was asked to complete a number of questionnaires on the day of initial consultation and six weeks afterwards. Results: The two experimental groups (2nd & 3rd) were equally effective in reducing pain, disability and distress, and both were more effective than the attention placebo group (1st), however the experimental groups appeared to have improved at follow-up relative to the placebo-group in terms of disability, pain and depressed mood.

9. **Turp JC et al** [27] in **2007** suggested that individuals without major psychological symptoms do not require more than simple therapy. In contrast, patients with major psychological involvement need multimodal, interdisciplinary therapeutic strategies. The clinician's acceptance of the importance of psychological factors in TMD pain forms the platform for convincingly educating patients about the need for multimodal management.

10. **Melissa Kato et al** [28]**; (2006**). This research compared two treatments in a sample of 18 patients with chronic TMD of muscular origin, divided into two groups (LASER and TENS). Pain relief and reestablishment of normal jaw function are the main goals of conservative management of Temporomandibular Disorders (TMD).And it was concluded that within the limitations of this study, both therapies were effective for decreasing the symptoms of TMD patients, regardless of the type of device used. The cumulative effect may be responsible for this improvement, since it is just observed after several sessions, whereas the immediate effect was not significant. Further clinical studies must be performed to evaluate the real efficacy of physical therapy modalities

11. **Marega S Medlicott and Susan R Harris** [29] **;(2006).** This systematic review analysed studies examining the effectiveness of various physical therapy interventions for Temporomandibular disorder and it was concluded that the following active exercises and manual mobilizations postural training, mid-laser therapy, relaxation techniques and biofeedback, electromyography training, and proprioceptive re-education and combinations may be effective

12. **Allan Kalamir et al** [30] in **2006** stated that the contemporary biopsychosocial health paradigm emphasizes a reversible and conservative approach to chronic pain management. Manual therapy for temporomandibular disorders (TMDs) claims to fulfil these criteria. The results of manual therapy trials for this condition suggest that manual therapy is a viable and useful approach in the management of TMD. Manual therapy has also been shown to be more cost effective and less prone to side effects than dental treatment

13. **Debora Grossi, Thais Cristina Chaves** [31] **;(2004).** This systemic review focuses on to a correct approach for effective physiotherapeutic intervention for treatment of temporomandibular joint intervention. And it was concluded that Manual therapy, therapeutically resources (like as ultra-sound, TENS) and postural re-education must

be applied in a physical therapy treatment for TMD patients and appropriate intervention should be related not only to symptoms relief, but also TMD´s etiology.

14. **Wahlund K** [32] in **2003** investigated different aspects of temporomandibular disorders (TMD) and pain such as prevalence, diagnostics, and treatment among adolescents. The reliability of a questionnaire and the clinical examination and diagnoses according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) were assessed. The prevalence of TMD pain, gender differences, and the need for treatment were investigated among 864 adolescents from a Public Dental Service clinic. Seven percent of the subjects received a pain diagnosis according to the RDC/TMD, and the prevalence was higher among girls than boys. More subjects with TMD pain reported school absence and analgesic consumption compared to controls. Approximately every second subject who reported TMD pain also perceived a need for treatment. In an evaluation of psychosocial and dental factors, the following were found to play an important role in adolescents with TMD: stress, somatic complaints, and emotional problems. Three treatment methods were compared in a randomized controlled trial: brief information only, brief information and occlusal appliance, and brief information and relaxation therapy.

    In the brief information and occlusal appliance group, 60%--significantly more than in the other two groups--experienced a reduction of at least 50% in TMD pain. The influence of somatic and emotional stimuli was evaluated, and we found that adolescents with TMD pain were significantly more sensitive to not only aversive somatic but also pleasant somatic stimuli compared with healthy controls. The results suggest that not only nociceptive but also cognitive processes are implicated in chronic pain states in young TMD subjects. In conclusion, TMD pain is more common in girls than in boys and affects daily life. TMD pain in adolescents can best be improved by traditional treatment with occlusal appliance combined with brief information.

15. **P Nicolakis, et al** [33] **(2002).** The objective of this study is to find out effectiveness of exercise therapy in patients with myofascial pain dysfunction syndrome, the treatment protocol consisted of active passive jaw movement exercises, correction of body

postures and relaxation techniques and further it was concluded that exercise therapy seems to be useful in the treatment of MPD syndrome.

16. **Tsukiyama et al** [34] in **2001** assessed occlusal adjustment as a treatment for temporomandibular disorders. Three experiments evaluated the relationship between occlusal adjustment and bruxism.

    Six experiments evaluated occlusal adjustment therapy as a treatment for patients with primary temporomandibular disorders. One experiment looked at occlusal adjustment effect on headache/temporomandibular disorder symptoms; another looked at its effect on chronic neck pain. Most of these experiments used a mock adjustment or a comparison treatment as the control condition in adults who had an existing nonacute general temporomandibular disorder. Overall, the data from these experiments did not demonstrate elevated therapeutic efficacy for occlusal adjustment over the control or the contrasting therapy. The experimental evidence reviewed was neither convincing nor powerful enough to support the performance of occlusal therapy as a general method for treating a nonacute temporomandibular disorder, bruxism, or headache

17. **Forsell et al** [35] in **1999** evaluated the occlusal treatment and their superior effects over each other. He found out that the use of occlusal splints may be of some benefit in the treatment of TMD. Evidence for the use of occlusal adjustment is lacking.

18. **Huddleston Slater** [36] in **1997** stated that although patients often report painless clicking of their temporomandibular joint, treatment is not required. However, sometimes a clicking joint leads to a painful limitation of the movements of this joint. Why this happens is unknown. It is therefore recommended to monitor the clicking joint over time. Opto-electronic recording of condylar movements might offer additional possibilities in the assessment of clicking joints

19. **Hobo** [37] in **1996** evaluated that the concept of using the condylar path as the reference for occlusion is questionable for the patient whose temporomandibular joint has

pathological changes because the condylar path of TMD patient deviates greatly. After occlusal splint therapy it is suggested that the patient's occlusion be treated using the Twin-Stage Procedure which does not require measurement of the condylar path. The research findings that occlusion controls the condylar path seems to support the concept that if the dentist creates the occlusion properly, the condylar path may be corrected and thereby minimise the micro-trauma which causes TMD

20. **S.E.Widmalm** [38] in **1996** came about with the classification of temporomandibular joint sounds based upon their reduced interference distribution. A new method for time-frequency analysis, the reduced interference distribution (RID), was used to classify the electronic sound recordings into five subclasses, RID types 1–5, based upon location and number of their energy peaks

21. **R.Celikar et al** [39] in **1995** correlated the relationship of TMJ disorders on rheumatoid arthritis and concluded that TMJ involvement may be detected even in asymptomatic patients with RA and there is a positive correlation between the severity of disease and involvement of TMJ.

22. **R J Gray et al** [40] **;(1994).** This study aims a comparative evaluation of four different physiotherapy treatments and placebo in the management of TMJPDS and comments on their cost benefit aspects compared with that of splint therapy. The four methods of physiotherapy tested were short-wave diathermy, megapulse, ultrasound and soft laser. And it was concluded that there was no statistically significant difference in success rate between any of the four tested (range 70.4–77.7%) although each individually was significantly better than placebo treatment.

23. **P.-L. Westesson** [41] in **1993** did a study on Reliability and Validity of Imaging Diagnosis of Temporomandibular Joint Disorder and concluded that Plain-film and tomography are basic imaging techniques for assessment of the temporomandibular joint. Arthrography, computed tomography, and magnetic resonance imaging have all been used for evaluation of the soft-tissue components of the joints. Accuracy studies

of these techniques have demonstrated the highest diagnostic accuracy for magnetic resonance imaging. Arthrography is relatively insensitive for detection of medial and lateral displacements. Magnetic resonance imaging accurately depicts both hard and soft tissues, and this technique is emerging as the prime diagnostic imaging technique in patients presenting with clinical signs and symptoms of a disorder of the temporomandibular joint.

24. **Douglas H Morgan** [42] in **1992** studied the relationship between tinnitus and TMJ dysfunction and found out that individuals who have tinnitus with no apparent otologic basis for this symptom should have a careful evaluation of the temporomandibular apparatus. A temporomandibular disorder may be one of the primary causes of this symptom.

25. **Dorrit W. Nitzan** [43] in **1991** introduced a simplified treatment for severe, limited mouth: Temporomandibular joint arthrocentesis. Seventeen patients complaining of suddenly occurring, severe, and persistent limited mouth opening were treated by irrigation of the upper compartment of the affected temporomandibular joint with lactated Ringer's solution. This simple treatment was found to be highly effective in reestablishing normal opening and relieving pain for a follow-up period of 4 to 14 months.

26. **Kaban et al** [44] in **1990** introduced the protocol for treatment of TMJ ankyloses which includes 1) aggressive resection, 2) ipsilateral coronoidectomy, 3) contralateral coronoidectomy when necessary, 4) lining of the TMJ with temporalis fascia or cartilage, 5) reconstruction of the ramus with a costochondral graft, 6) rigid fixation, and 7) early mobilization and aggressive physiotherapy

27. **Andrew G. Pullinger et al** [45] in **1988** did a study to find out: Occlusal factors associated with temporomandibular joint tenderness and dysfunction and concluded that TMJ tenderness was more frequent in class II, division 2 than in class I, but overall

was not associated with occlusal factors such as deep overbites, length of a symmetric RCP-ICP slide, and unilateral contact in RCP.

28. **L.F.Greenwood** [46] in **1987** did a study to find the relationship between TMJ dysfunction and generalized joint hypermobility and concluded that generalized joint hypermobility is not associated with increased mobility of the TMJ, and further that generalized joint hypermobility has no relationship with TMJ dysfunction.

29. **Aune M Raustia** [47] in **1985** did a randomized study to compare Acupuncture with stomatognathic treatment for TMJ dysfunction. Acupuncture may prove to be ideal early therapy for some patients and complemented later with stomatognathic methods.

30. **Marc W. Heft** [48] in **1984** studied about the prevalence of temporomandibular joint disorder and found out that the majority of the reports profiling TMJ patients suggest that most are females (80%) in the 20–40 year age group

31. **Alfred C. Griffin** [49] in **1983** did A Laminagraphic Study to determine the Temporomandibular Joint Morphology as Related to Anterior Occlusion and Vertical Facial Height and concluded that the correlative comparison between anterior and posterior guidance showed a substantial tendency for subjects with increased overbite to have greater articular eminence heights and a greater inclination to the posterior guidance slope

32. **David A Keith** [50] in **1982** stated that the TMJ develops later than most other human joints. Unlike other joints which develop from a single blastema, the TMJ forms from two blastemata which grow toward one another. Most joints are covered by hyaline cartilage while the articulating surfaces of the TMJ are covered by fibrous tissue consisting of both elastic and collagen fibres. Finally, the process of bone formation in the condyle is different from that seen in growth plates.

33.M. J. Sbuttoni [51] conducted a study in **1981** to determine the effect of Bite Blocks on the Temporomandibular Joint and concluded that large variations in condylar angles (9.5 to 44 degrees) and significant variations from one side to the other on the same patient were observed. The results emphasize the need for individualized appliance design for very specific therapeutic objectives for TMJ disorders.

TERMINOLOGY

Over the years functional disturbances of the masticatory system has been identified by a variety of terms. In **1934**, **James Costen** described a group of symptoms centering around the ear and TMJ. Because of his work the term "**Costen syndrome**" developed [52]. In **1952 Milton R Beyers** and **Samuel Teich** introduced the word "**Mandibular joint neuralgia".**

Mandibular joint neuralgia is accepted by Otorhinolaryngologists and members of the dental profession as a definite clinical entity. It should be considered in every differential diagnosis of recurring facial pain.

The pathology is either malocclusion from any cause or destructive changes of one or both mandibular joints. The resultant abnormal pressure in the mandibular fossa causes partial or complete closure of the internal auditory canal, accounting for the "stuffy deaf" sensation so common to the syndrome. The diverse and seemingly unconnected neuralgic pains are due to either direct nerve compression within the abnormal joint or reflex irritation of the nerves lying in close association with the joint

During the 1950s, **Schwartz** coined the term "**Temporomandibular joint pain dysfunction syndrome**" [53]. Later the term "**TMJ disturbances**" became popular and then in **1959**, **Shore** introduced the term "**TMJ dysfunction syndrome**" [54]. Dysfunction of the temporomandibular joint is due to abnormal dental occlusions in more than 90 per cent of the cases. The clinical manifestations of this functional disorder are multiple and often involve the entire neuromuscular system.

Although the range of symptoms varies with each patient, the neuromuscular symptoms that usually manifest themselves are (1) temporomandibular joint clicking accompanied by crepitation, (2) irregular opening and closing of the mandible, (3) limited or excessive mandibular movement, (4) mandibular subluxation, (5) condylar hypermobility, and (6) a vicious and, frequently, exquisitely painful cycle of muscle spasm-pain-spasm that is often the precipitating factor in the patient's desire for medical assistance.

Later came the term "**Functional TMJ disturbances**" coined by **Ramjford and Ash** [55]. Some terms like "**Occlusomandibular disturbances**" [56] and "**Myoarthropathy of TMJ**" [57] described the suggested etiological factors.

Subsequently, as a result of clinical observation and a variety of research studies, **Laskin** proposed the term "**Myofacial Pain Dysfunction Syndrome (MPDS)**" [58]. He attributed the features of pain, joint sounds and limited mandibular movement to multiple causes and provided experimental evidence for the concept of a psychophysiologic disorder.

Others stressed pain such as "**Pain dysfunction syndrome**", "**Myofascial pain dysfunction syndrome**" and "**TM pain dysfunction syndrome**". The signs associated with functional disorders of the TMJ are probably the most common findings when examining a patient for masticatory dysfunction.

Many of these signs do not produce painful symptoms and therefore, the patient may not seek treatment. When present, however, they generally fall into three broad categories: derangements of the condyle-disc complex, structural incompatibility of the articular surfaces, and inflammatory joint disorders. The first two categories have been collectively referred to as "**Disc-interference disorders**".

The term disc-interference disorder was first introduced by **Welden Bell** to describe a category of functional disorders that arises from problems with the condyle-disc complex

The symptoms are not always confined to the temporomandibular joint. Some authors believe that the forgoing is too limited and that a broader more collective term should be used, such as "**Craniomandibular disorders**"**.** The term craniomandibular disorders is used synonymously with the term temporomandibular disorders and is considered a major cause of nondental pain in the orofacial pain region.

Bell suggested the term "**TM disorders**" [59] which gained popularity. The term does not suggest merely problems that are associated to the TMJs but includes all disturbances associated with the function of masticatory system.

The wide variety of terms used has contributed to the great amount of confusion that exists in this already complicated field. Therefore, the **American Dental Association** has adopted the term "**Temporomandibular disorders" [**[60]

Fascialarthromyalgia, mandibular dysfunction, myofascial pain, masticatory myalgia syndrome and **primary myalgia** affecting the masticatory musculature are also used synonymously.

Frequently, TMD has been treated as a single syndrome, but the prevailing modern view is that TMD is a cluster of related disorders with many common features. Indeed, some have suggested that in the future the term TMD may be discarded as the different causes are fully identified and separated into different conditions.

Sometimes, "**Temporomandibular joint dysfunction**" is described as the most common form of temporomandibular disorder, whereas many other sources use the term temporomandibular disorder synonymously, or instead of the term temporomandibular joint dysfunction. In turn, the term temporomandibular disorder is described as "a clinical term [referring to] musculoskeletal disorders affecting the temporomandibular joints and their associated musculature.

It is a collective term which represents a diverse group of pathologies involving the temporomandibular joint, the muscles of mastication, or both. Another definition of temporomandibular disorders is "a group of conditions with similar signs and symptoms that affect the temporomandibular joints, the muscles of mastication, or both.

"**Temporomandibular disorder**" is a term that creates confusion since it refers to a group of similarly symptomatic conditions, whilst many sources use the term temporomandibular disorders as a vague description rather than a specific syndrome, and refer to any condition which may affect the temporomandibular joint. The temporomandibular joint is susceptible to a huge range of diseases, some rarer than others, and there is no implication that all of these will cause any symptoms or limitation in function at all.

The preferred terms in medical publications is to an extent influenced by geographic location, e.g. in the United Kingdom, the term "Pain dysfunction syndrome" is in common use, and in other countries different terms are used.

In the United States, the term "**Temporomandibular disorder**" is generally favored. The American Academy of Orofacial Pain uses temporomandibular disorder, whilst the National Institute of Dental and Craniofacial Research uses **Temporomandibular joint disorder.**

A more complete list of synonyms for this topic is extensive, with some being more commonly used than others. In addition to those already mentioned, examples include "temporomandibular joint pain dysfunction syndrome", "temporomandibular pain dysfunction syndrome", "temporomandibular joint syndrome", "temporomandibular dysfunction syndrome", "temporomandibular dysfunction", "temporomandibular disorder", "temporomandibular syndrome", "facial arthromyalgia", "myofacial pain dysfunction syndrome", "craniomandibular dysfunction" (CMD), "myofacial pain dysfunction", "masticatory myalgia", "mandibular dysfunction", and "Costen's syndrome".

The lack of standardization in terms is not restricted to medical papers. Notable internationally recognized sources vary in both their preferred term, and their offered definition, e.g. "Temporomandibular Pain and Dysfunction Syndrome – Aching in the muscles of mastication, sometimes with an occasional brief severe pain on chewing, often associated with restricted jaw movement and clicking or popping sounds." (Classification of Chronic Pain, International Association for the Study of Pain).

"Headache or facial pain attributed to temporomandibular joint disorder." (International Classification of Headache Disorders 2nd edition (ICHD-2), International Headache Society).

"Temporomandibular joint-pain-dysfunction syndrome" listed in turn under "Temporomandibular joint disorders" (International Classification of Diseases 10th revision, World Health Organization).

However there is no single, globally accepted term or definition concerning this topic. Today, a much more comprehensive view of this condition exists, and the term "**Temporomandibular disorder"** (TMD) is the preferred term according to the **American Academy of Orofacial Pain (AAOP)** and most other groups who sponsor studies into its origins and treatment. Interestingly, the National Institute of Dental and Craniofacial Research (NIDCR) puts TMJ and TMD together and refers to them as "**Temporomandibular joint disorder"** (TMJD). However, the term TMD is preferred.

HISTORY OF TEMPOROMANDIBULAR DISORDERS

The origin and homologies of the jaws in all vertebrates were clearly outlined in the last century by Gegenbauer [61], later a definitive statement of this was made by Reichert in 1837 known as the Reichert Theory. Fawcett has shown that in man, dermal bones (as the name implies they directly arise in the dermal tissue) cover the cranium, house the facial organs and form the adult jaws.[62] Remnants of the cartilaginous upper jaw have been described in the human embryo and Meckel's cartilage is the well-known embryonic vestige of the lower jaw. This dermal bone is one of the earliest bones to appear in the fetus.

The dental profession was generally first drawn into the area of TMDs with an article written by Dr. James Costen, an otolaryngologist, in 1934. On the basis of 11 cases, Costen first suggested to the profession that changes in the dental condition were responsible for various ear symptoms.

Listed in the Quarterly Cumulative Index Medicus (QCIM) for 1939, under "Jaws, diseases," is the first article with "Costen's syndrome" in the title.[63] ' Costen's original article, which appeared in 1934, also listed under "Jaws, diseases," was titled "A Syndrome of Ear and Sinus Symptoms Dependent Upon Disturbed Function of the Temporomandibular Joint."[64] He included ear symptoms such as impaired hearing, stuffy sensation in the ears, tinnitus, ear pain, and sometimes severe dizziness relieved by eustachian tube inflation, "sinus" symptoms such as headache at the vertex and occiput, and burning sensations in the throat and tongue. When causes in the sinuses and ears could be ruled out, he attributed all these symptoms to dental malocclusion. He followed up his 1934 publication in the next 5 years with 11 other articles on the same subject

Costen was not the first to point out otolaryngological symptoms of abnormalities of mandibular articulation, but he made an impact in his 1934 article "With its directness and unequivocation" [65], in spite of its anatomic naivete. He had become interested in referred pain after seeing many patients for head pain that had been unrelieved by ethmoid or sphenoid sinus surgery. Costen thought the pain was probably related to the

temporomandibular joint (TMJ). His work focused attention on pain in the region of the ear and the TMJ.

Stressing occlusal derangement with consequent disturbance in the function of the joint, he publicized to his fellow otolaryngologists an important diagnostic advance: to look beyond the sinuses for a source of head pain.

Costen's son [William S. Costen, MD, an orthopedic surgeon] recalls his father laying out charts in his study at home when he began collecting data on the TMJ and on pain fibers in the chorda tympani. In 1957 William noted a description of the possible anatomic basis for referred pain in Hilton's "On Rest and Pain" [66] [1879] and brought it to his father's attention. Subsequently, his father wrote a monograph giving Hilton credit for describing the anatomic basis for pain referral.

Almost as soon as the term Costen's syndrome came into general use, questions as to its validity began to surface. Over the years, various authors have disagreed with the idea that this sweeping panoply of symptoms could be attributable to such a simple cause as malocclusion.

Sicher, [67] in 1948, wrote that Costen's syndrome should be replaced by the diagnosis "temporomandibular deforming arthritis from mandibular overclosure or displacement."

He rejected Costen's anatomic explanations for the pain in this symptom complex, but seemed to accept the grouping of symptoms. As early as 1943, Shapiro and Truex were doubtful that TMJ problems could be responsible for deafness, except for possible eustachian tube impingement, and even this was considered unlikely. Questions were also raised about tinnitus, vertigo, neuralgias similar to trigeminal, and headaches as being of TMJ dysfunction origin. On the other hand, writers defending the use of Costen's syndrome as a diagnosis suggested that not all the symptoms listed in Costen's original work were required to be present to make the diagnosis. In more than one study, tinnitus and vertigo were found to be statistically more frequent in TMJ disorder patients than in normal controls. [68]

In the late 1930s and through the 1940s, only a few dentists became interested in managing these pain problems. The most common therapies provided at that time were bite-raising appliances, which were first suggested and described by Costen himself.

In the late 1940s and into the 1950s, the dental profession began to question bite-raising appliances as the therapy of choice for mandibular dysfunction. It was at this time that the occlusal interferences began to be looked upon as the major etiologic factor in TMD complaints. [69]

Even after "Costen's syndrome" became a widely used term, the QCIM continued listing it under "TMJ syndrome." By 1966, however, it was listed as a separate entry. In 1969, there were 59 entries in the category "Costen's syndrome," and 'TMJ syndrome" was listed under "Costen's syndrome." However, just 4 years later, "Costen's syndrome" was back under 'TMJ syndrome."

This rise and subsequent fall in the use of the term Costen's syndrome to classify all TMJ syndrome references can be attributed to several factors. Although universally acclaimed for bringing the significant role of the TMJ into the realm of awareness of physicians and dentists alike, Costen was judged by many to have been naive in his espousal of the range of symptoms arising from TMJ disorders, in his anatomic explanations, and especially in his view of the pivotal role of malocclusion.

Some authors recommended that the term Costen's syndrome be dropped, as it was too inclusive, and replaced with "osteoarthritis of the temporomandibular joint." Others began using "myofascial pain," by definition a muscular pain in numerous body regions produced by pressure on trigger points, with TMJ syndrome being the prime example. Proponents viewed the TMJ syndrome as a cascade of events, from psycho logic stress to clenching and bruxism, resulting in occlusal problems, spasm, and pain. "Myofascial pain syndrome" has gradually become the more popular diagnosis. Nowadays, it is more acceptable in medical diagnosis to include the etiologic effects of tension and anxiety. Nevertheless, many cases of TMJ disorders have continued to be reported as "Costen's syndrome," even in recent times. The etiology and treatment offered by Costen were not always correct, yet the term Costen's syndrome may still be useful, perhaps with a new,

more inclusive etiology. Costen rendered a great service in pointing out the relationship between malocclusion, TMJ dysfunction, and facial pain. Otolaryngology owes a debt to Costen for bringing out these points.

Scientific investigation of TMDs first began in the 1950s. Early scientific studies suggested that the occlusal condition could influence masticatory muscle function. Electromyographic studies were used to correlate such relationships. [70] In the late 1950s the first textbooks were written describing masticatory dysfunctions.

The most common conditions described at that time were masticatory muscle pain disorders. The cause of these disorders was generally thought to be occlusal disharmony. Occlusion and later, emotional stress were accepted as the major etiologic factors of functional disorders of the masticatory system through the 1960s and into the 1970s.

Then in the 1970s an explosion of interest in TMDs took place. Also at this time information reached the profession concerning pain disorders arising from intracapsular sources. It was not until the 1980s that the profession began to recognize fully and appreciate the complexity of TMDs. When the dental community began examining and seeking to understand TMJD in the 1980's, research and technology helped uncover some science behind the temporomandibular joint and its role in the complex workings of the face, jaw and neck.

As studies were increasingly run on volunteer sufferers of TMJD, it soon came to light that there was a notable connection between arthritis and TMJD. From this discovery came the first of more effective treatment plans for those with TMJD. Rather than only providing pain relief, healthcare professionals began to treat the causes of some forms of the condition.

The National Institute of Dental and Craniofacial Research (NIDCR), among other groups, is conducting research into TMJD. A significant study seeks to identify contributing risk factors in order to better understand the underlying cause and developmental course of TMJ disease and disorders. It is hoped that, better understanding of cause can lead to better diagnosis and treatment.

The National Institutes of Health (NIH) are conducting ongoing studies about pain sensation and processing, including research about:

- How men and women may respond differently to pain and pain medications.
- What TMJD may have in common with pain conditions such as myofascial pain and head ache pain.
- Medications and treatments for the effective relief of chronic pain.
- How environmental stressors including noise, temperature, or physical strain can affect pain symptoms in TMJD patients and how lifestyle changes may relieve pain.

Long-term research is currently aimed at the replacement of injured or irregular parts of the temporomandibular joint, discs and chewing muscles such as implants or laboratory grown human tissue.

SURFACE ANATOMY

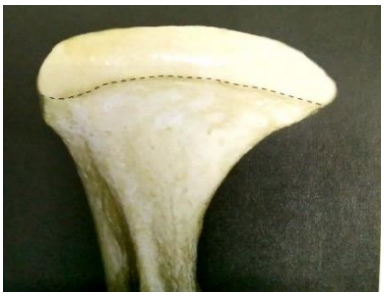
The area where the mandible articulates with the cranium, the TMJ, is one of the most complex joints in the body. It provides for hinging movement in one plane and therefore can be considered a ginglymoid joint. However, at the same time it also provides for gliding movements, which classifies it as an arthrodial joint. Thus it has been technically considered a ginglymoarthrodial joint [71].

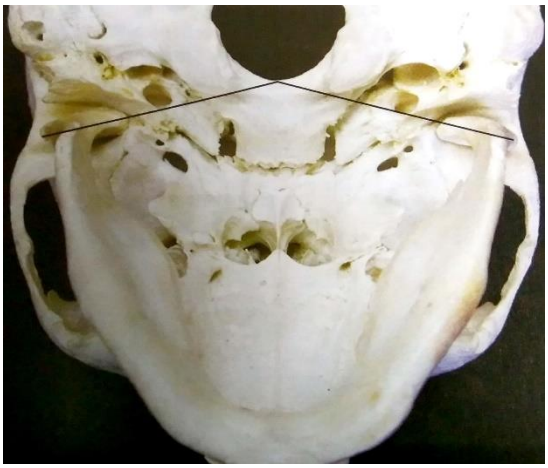
The TMJ is quite superficial and is situated below the posterior end of the zygomatic arch just in front of the external acoustic meatus. Its position is noted by defining the condyle of the mandible; when the mouth opens, the condyles move out of the mandibular fossa into the articular tubercle and a depression is noted at the location of the joint. According to Guralnick [72] et al, it is the posterior lateral aspect of the condyle that may be palpated at this location.

The TMJ is formed by the mandibular condyle fitting into the mandibular fossa of the temporal bone. Separating these two bones from direct articulation is the articular disc. The TMJ is classified as a compound joint. By definition, a compound joint requires the presence of at least three bones, yet the TMJ is made up of only two bones.

CONDYLE

The condyle is the portion of the mandible that articulates with the cranium, around which movement occurs. From the anterior view it has medial and lateral projections, called poles. The medial pole is generally more prominent than the lateral.





A line drawn through the centers of the poles of the condyle will usually extend medially and posteriorly toward the anterior border of the foramen magnum.

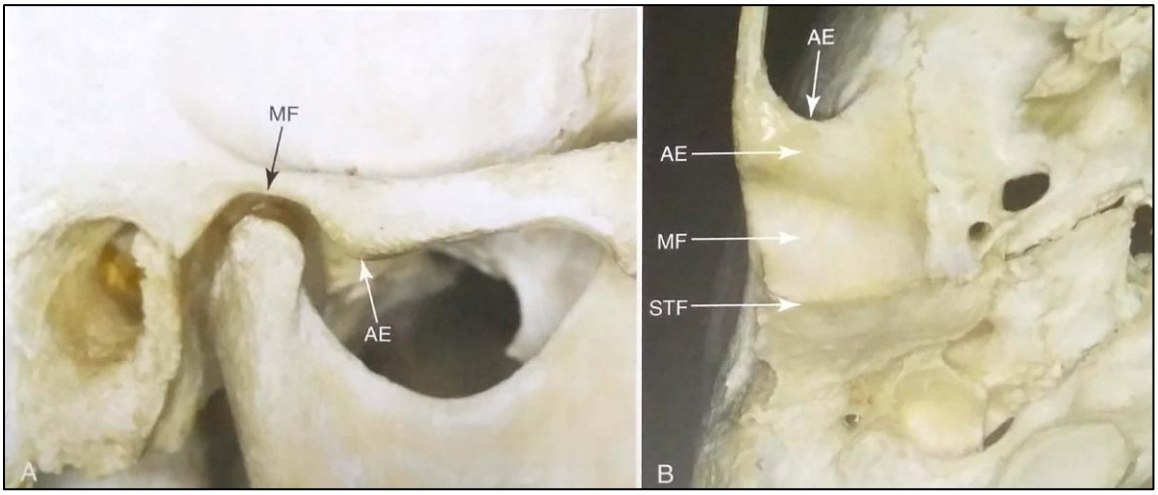
The total mediolateral length of the condyle is between 18 and 23mm, and the anteroposterior width is between 8 and 10 mm. The actual articulating surface of the condyle extends both anteriorly and posteriorly to the most superior aspect of the condyle. The posterior articulating surface is greater than the anterior surface.

Immediately anterior to the fossa is a convex bony prominence called the articular eminence. The degree of convexity of the articular eminence is highly variable but important because the steepness of this surface dictates the pathway of the condyle when the mandible is positioned anteriorly. The posterior roof of the mandibular fossa is quite thin, indicating that this area of the temporal bone is not designed to sustain heavy forces. The articular eminence, however, consists of thick dense bone and is more likely to tolerate such forces.

GLENOID FOSSA/MANDIBULAR FOSSA/ARTICULAR FOSSA

The mandibular condyle articulates at the base of the cranium with the squamous portion of the temporal bone. This portion of the temporal bone is made up of a concave mandibular fossa, in which the condyle is situated and which has also been called the articular or glenoid fossa. Posterior to the mandibular fossa is the squamotympanic fissure, which extends mediolaterally. As this fissure extends medially, it divides into the petrosquamous fissure anteriorly and the petrotympanic fissure (glaserian fissure) posteriorly. [73]

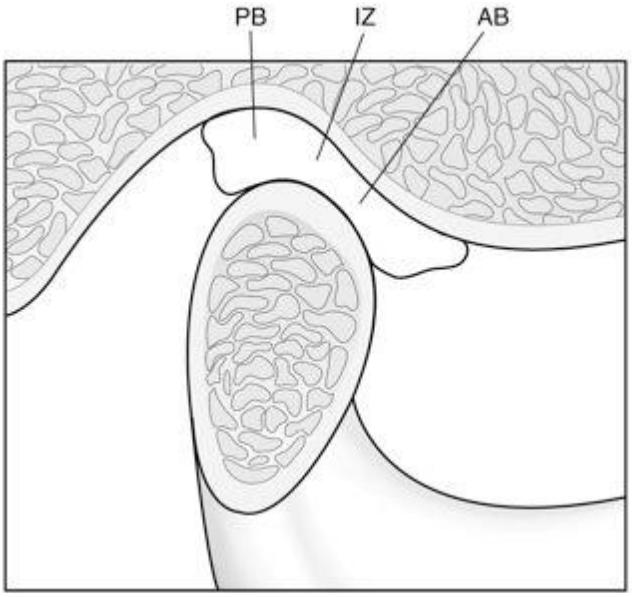
The glenoid fossa accepts the condylar process of the mandible and is formed by the articular tubercle anteriorly and by the postglenoid tubercle posteriorly of the zygoma portion of the temporal bone. The articular surface is smooth, oval, and deeply concave containing the articular disc or meniscus. According to Gray's Anatomy," there is a nonarticular portion of the fossa in which a small part of the parotid gland is found. The fossa is composed of a thin compact bone, whereas the articular tubercle and condylar process is of a spongy bone under a thin compact bone. [73]



A, Bony structures of the temporomandibular joint (lateral view). **B**, Articular fossa (inferior view). AE, Articular eminence; MF, mandibular fossa; STF, squamotympanic fissure.

ARTICULAR DISC

Functionally, the articular disc serves as a nonossified bone that permits the complex movements of the joint. Because the articular disc functions as a third bone, the craniomandibular articulation is considered a compound joint. The articular disc is composed of dense fibrous connective tissue, for the most part devoid of any blood vessels or nerve fibers. The extreme periphery of the disc, however, is slightly innervated. In the sagittal plane it can be divided into three regions according to thickness. The central area is the thinnest and is called the intermediate zone. The disc becomes considerably thicker both anterior and posterior to the intermediate zone. The posterior border is generally slightly thicker than the anterior border. In the normal joint the articular surface of the condyle is located on the intermediate zone of the disc, bordered by the thicker anterior and posterior regions.



The condyle is normally situated on the thinner intermediate zone (IZ) of the disc. The anterior border of the disc (AB) is considerably thicker than the intermediate zone, and the posterior border (PB) is even thicker.

The articular disc is attached posteriorly to a region of loose connective tissue that is highly vascularized and innervated. This is known as the retrodiscal tissue or posterior attachment. Superiorly, it is bordered by a lamina of connective tissue that contains many elastic fibers, the superior retrodiscal lamina. The superior retrodiscal lamina attaches the articular disc posteriorly to the tympanic plate.

At the lower border of the retrodiscal tissues is the inferior retrodiscal lamina, which attaches the inferior border of the posterior edge of the disc to the posterior margin of the articular surface of the condyle. The inferior retrodiscal lamina is composed chiefly of collagenous fibers, not elastic fibers like the superior retrodiscal lamina.

The remaining body of the retrodiscal tissue is attached posteriorly to a large venous plexus, which fills with blood as the condyle moves forward. The superior and inferior attachments of the anterior region of the disc are to the capsular ligament, which surrounds most of the joint. The superior attachment is to the anterior margin of the articular surface of the temporal bone. The inferior attachment is to the anterior margin of the articular surface of the condyle. Both these anterior attachments are composed of collagenous fibers. Anteriorly, between the attachments of the capsular ligament, the disc is also attached by tendinous fibers to the superior lateral pterygoid muscle.

The articular disc is attached to the capsular ligament not only anteriorly and posteriorly but also medially and laterally. This divides the joint into two distinct cavities. The upper or superior cavity is bordered by the mandibular fossa and the superior surface of the disc. The lower or inferior cavity is bordered by the mandibular condyle and the inferior surface of the disc.

The internal surfaces of the cavities are surrounded by specialized endothelial cells that form a synovial lining. This lining, along with a specialized synovial fringe located at the anterior border of the retrodiscal tissues, produces synovial fluid, which fills both joint cavities. Thus the TMJ is referred to as a synovial joint. This synovial fluid serves two purposes. Because the articular surfaces of the joint are nonvascular, the synovial fluid acts as a medium for providing metabolic requirements to these tissues. Free and rapid exchange exists between the vessels of the capsule, the synovial fluid, and the articular

tissues. The synovial fluid also serves as a lubricant between articular surfaces during function. The articular surfaces of the disc, condyle, and fossa are very smooth, so friction during movement is minimized.

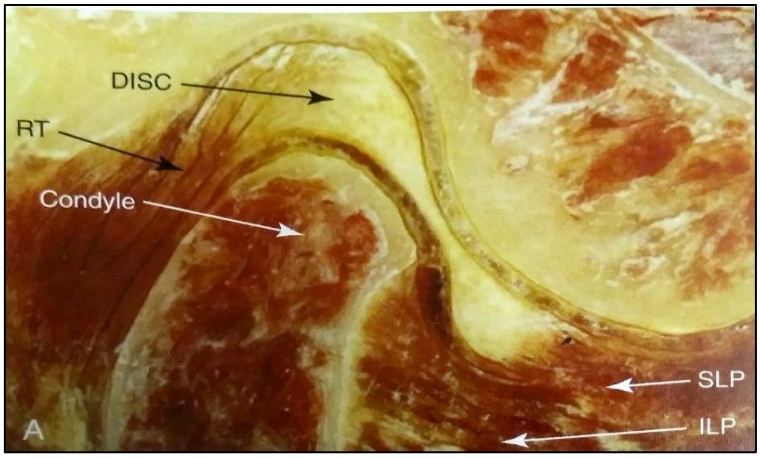
The synovial fluid helps to minimize this friction further. Synovial fluid lubricates the articular surfaces by way of two mechanisms.

1) The first is called **boundary lubrication**, which occurs when the joint is moved and the synovial fluid is forced from one area of the cavity into another. The synovial fluid located in the border or recess areas is forced on the articular surface, thus providing lubrication. Boundary lubrication prevents friction in the moving joint and is the primary mechanism of joint lubrication.

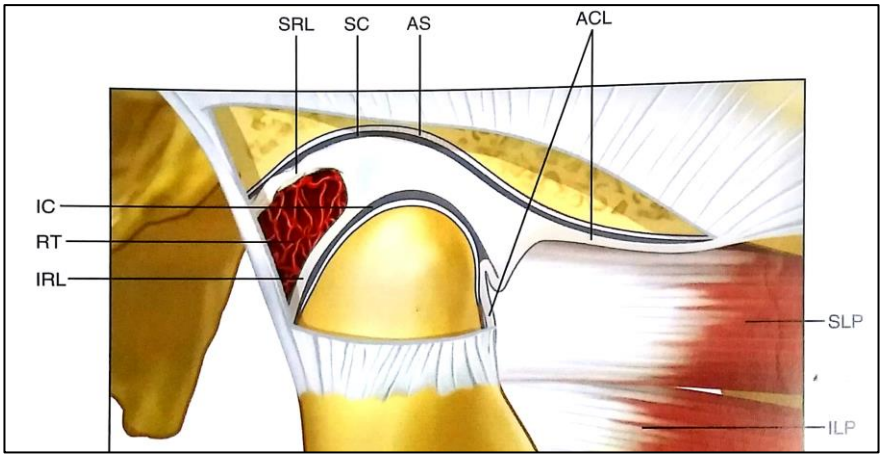
2) A second lubricating mechanism is called **weeping lubrication**. This refers to the ability of the articular surfaces to absorb a small amount of synovial fluid [74].

During function of a joint, forces are created between the articular surfaces. These forces drive a small amount of synovial fluid in and out of the articular tissues. This is the mechanism by which metabolic exchange occurs.

Under compressive forces, therefore, a small amount of synovial fluid is released. This synovial fluid acts as a lubricant between articular tissues to prevent sticking. Weeping lubrication helps eliminate friction in the compressed but not moving joint. Only a small amount of friction is eliminated as a result of weeping lubrication; therefore prolonged compressive forces to the articular surfaces will exhaust this supply.



A



B

A, Lateral view. **B**, Diagram showing the anatomic components. ACL, Anterior capsular ligament (collagenous); AS, articular surface; IC, inferior joint cavity; ILP, inferior lateral pterygoid muscles; IRL, inferior retrodiscal lamina (collagenous); RT, retrodiscal tissues; SC,superior joint cavity; SLP, superior lateral pterygoid muscles; SRL, superior retrodiscal lamina (elastic).

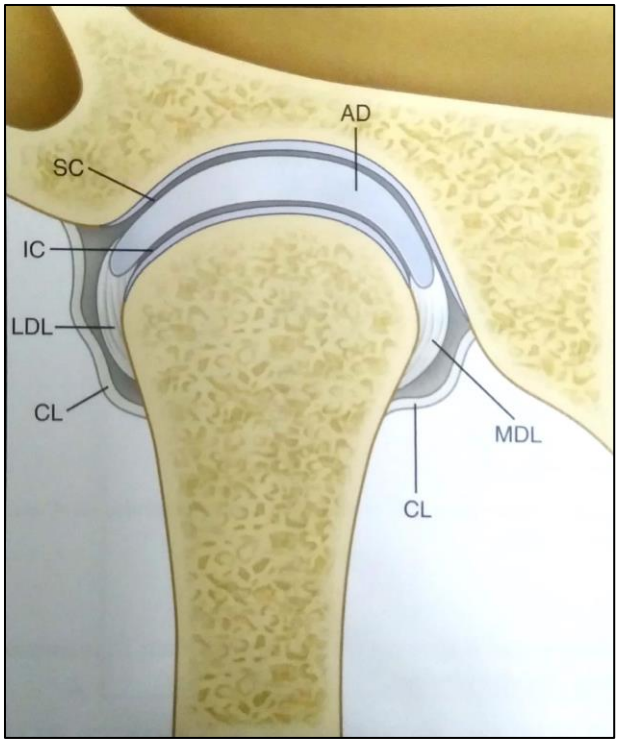
LIGAMENTS

As with any joint system, ligaments play an important role in protecting the structures. The ligaments of the joint are composed of collagenous tissues that have particular lengths. They do not stretch. However, if extensive forces are applied to a ligament, whether suddenly or over a prolonged period of time, the ligament can be elongated. When this occurs, the function of the ligament is compromised, thereby altering joint function. Ligaments do not enter actively into joint function but instead act as passive restraining devices to limit and restrict border movements. Three functional ligaments [75] support the TMJ: (1) the collateral ligaments, (2) the capsular ligament, and (3) the temporomandibular (TM) ligament. Two accessory ligaments also exist: (4) the sphenomandibular and (5) the stylomandibular.

(1) COLLATERAL LIGAMENTS

The collateral ligaments attach the medial and lateral borders of the articular disc to the poles of the condyle. They are commonly called the discal ligaments, and there are two. The medial discal ligament attaches the medial edge of the disc to the medial pole of the condyle. The lateral discal ligament attaches the lateral edge of the disc to the lateral pole of the condyle. These ligaments are responsible for dividing the joint mediolaterally into the superior and inferior joint cavities. The discal ligaments are true ligaments, composed of collagenous connective tissue fibers; therefore they do not stretch. They function to restrict movement of the disc away from the condyle.

In other words, they allow the disc to move passively with the condyle as it glides anteriorly and posteriorly. The attachments of the discal ligaments permit the disc to be rotated anteriorly and posteriorly on the articular surface of the condyle. Thus these ligaments are responsible for the hinging movement of the TMJ, which occurs between the condyle and the articular disc. The discal ligaments have a vascular supply and are innervated. Their innervation provides information regarding joint position and movement. Strain on these ligaments produces pain.



AD, Articular disc; CL, capsular ligament; IC, inferior joint cavity; LDL, lateral discal ligament; MDL, medial discal ligament; SC, superior joint cavity.

(2) CAPSULAR LIGAMENT

The entire TMJ is surrounded and encompassed by the capsular ligament. The fibers of the capsular ligament are attached superiorly to the temporal bone along the borders of the articular surfaces of the mandibular fossa and articular eminence. The fibers of the capsular ligament are attached superiorly to the temporal bone along the borders of the articular surfaces of the mandibular fossa and articular eminence. Inferiorly, the fibers of the capsular ligament attach to the neck of the condyle. The capsular ligament acts to resist any medial, lateral, or inferior forces that tend to separate or dislocate the articular surfaces. A significant function of the capsular ligament is to encompass the joint, thus retaining the synovial fluid. The capsular ligament is well innervated and provides proprioceptive feedback regarding position and movement of the joint.

(3) TEMPOROMANDIBULAR LIGAMENT

The lateral aspect of the capsular ligament is reinforced by strong, tight fibers that make up the lateral ligament, or TM ligament. The TM ligament is composed of two parts, an outer oblique portion and an inner horizontal portion. The outer portion extends from the outer surface of the articular tubercle and zygomatic process posteroinferiorly to the outer surface of the condylar neck. The inner horizontal portion extends from the outer surface of the articular tubercle and zygomatic process posteriorly and horizontally to the lateral pole of the condyle and posterior part of the articular disc.

The oblique portion of the TM ligament resists excessive dropping of the condyle, therefore limiting the extent of mouth opening. This portion of the ligament also influences the normal opening movement of the mandible.

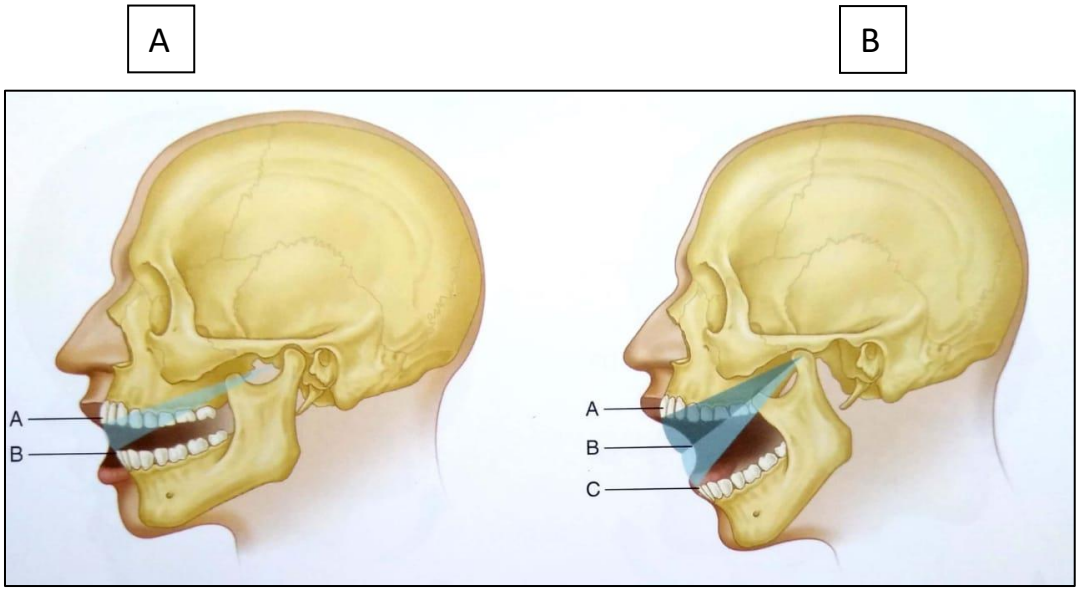
During the initial phase of opening, the condyle can rotate around a fixed point until the TM ligament becomes tight as its point of insertion on the neck of the condyle is rotated posteriorly. When the ligament is taut, the neck of the condyle cannot rotate further. If the mouth were to be opened wider, the condyle would need to move downward and forward across the articular eminence. This effect can be demonstrated clinically by closing the mouth and applying mild posterior force to the chin. With this force applied, the patient should be asked to open the mouth. The jaw will easily rotate open until the teeth are 20 to 25 mm apart. At this point, resistance will be felt when the jaw is opened wider. If the jaw is opened still wider, a distinct change in the opening movement will occur, representing the change from rotation of the condyle around a fixed point to movement forward and down the articular eminence.

This change in opening movement is brought about by the tightening of the TM ligament. This unique feature of the TM ligament, which limits rotational opening, is found only in humans. In the erect postural position and with a vertically placed vertebral column, continued rotational opening movement would cause the mandible to impinge on the vital submandibular and retromandibular structures of the neck. The outer oblique portion of the TM ligament functions to resist this impingement.

The inner horizontal portion of the TM ligament limits posterior movement of the condyle and disc.

When force applied to the mandible displaces the condyle posteriorly, this portion of the ligament becomes tight and prevents the condyle from moving into the posterior region of the mandibular fossa. The TM ligament therefore protects the retrodiscal tissues from trauma created by the posterior displacement of the condyle.

The inner horizontal portion also protects the lateral pterygoid muscle from overlengthening or extension. The effectiveness of this ligament is demonstrated during cases of extreme trauma to the mandible. In such cases, the neck of the condyle will be seen to fracture before the retrodiscal tissues are severed or the condyle enters the middle cranial fossa.



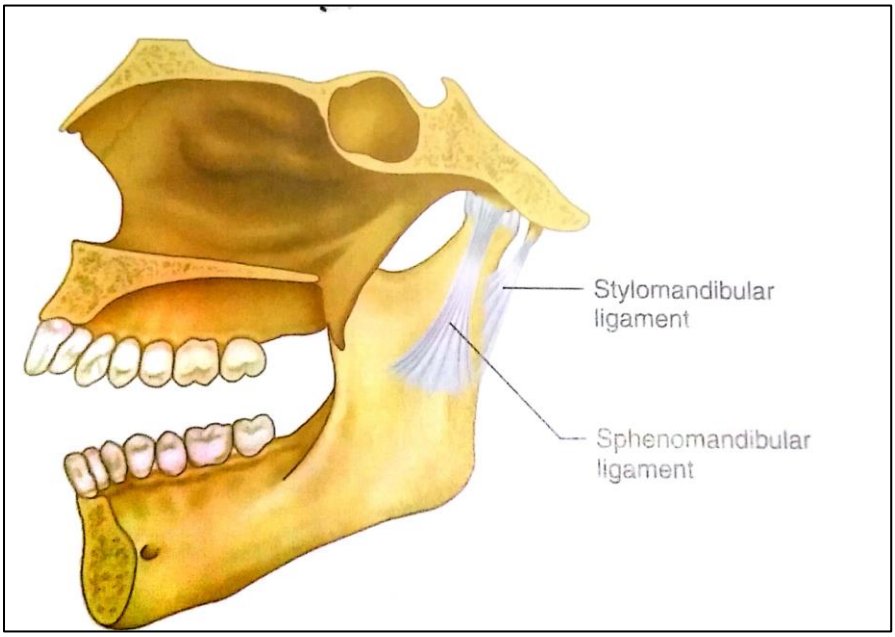
A, As the mouth opens, the teeth can be separated about 20 to 25 mm (from A to B)without the condyles moving from the fossae. **B**, TM ligaments are fully extended. As the mouth opens wider, they force the condyles to move downward and forward out of the fossae. This creates a second arc of opening (from B to C).

(4) SPHENOMANDIBULAR LIGAMENT

The sphenomandibular ligament is one of two TMJ accessory ligaments. It arises from the spine of the sphenoid bone and extends downward to a small bony prominence on the medial surface of the ramus of the mandible, which is called the lingula. It does not have any significant limiting effects on mandibular movement.

(5) STYLOMANDIBULAR LIGAMENT

The second accessory ligament is the stylomandibular ligament. It arises from the styloid process and extends downward and forward to the angle and posterior border of the ramus of the mandible. It becomes taut when the mandible is protruded but is most relaxed when the mandible is opened. The stylomandibular ligament therefore limits excessive protrusive movements of the mandible

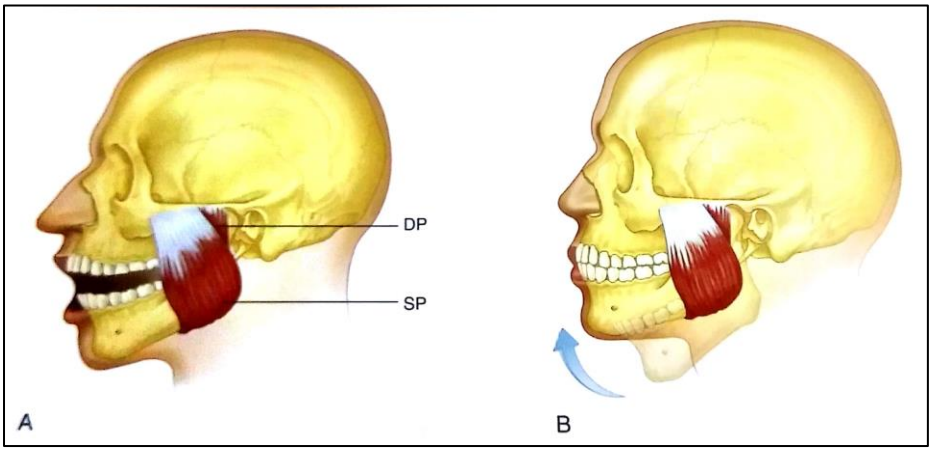


MUSCLES OF MASTICATION

Four pairs of muscles make up a group called the muscles of mastication: the masseter, temporalis, medial pterygoid, and lateral pterygoid [76]. Although not considered to be muscles of mastication, the digastrics also play an important role in mandibular function

MASSETER

The masseter is a rectangular muscle that originates from the zygomatic arch and extends downward to the lateral aspect of the lower border of the ramus of the mandible. Its insertion on the mandible extends from the region of the second molar at the inferior border posteriorly to include the angle. It has two portions, or heads: (1) The superficial portion consists of fibers that run downward and slightly backward, and (2) the deep portion consists of fibers that run in a predominantly vertical direction. As fibers of the masseter contract, the mandible is elevated and the teeth are brought into contact. The masseter is a powerful muscle that provides the force necessary to chew efficiently. Its superficial portion may also aid in protruding the mandible. When the mandible is protruded and biting force is applied, the fibers of the deep portion stabilize the condyle against the articular eminence.



A, Masseter muscle. DP, Deep portion; SP, superficial portion. **B**, Function: elevation of the mandible.

TEMPORALIS

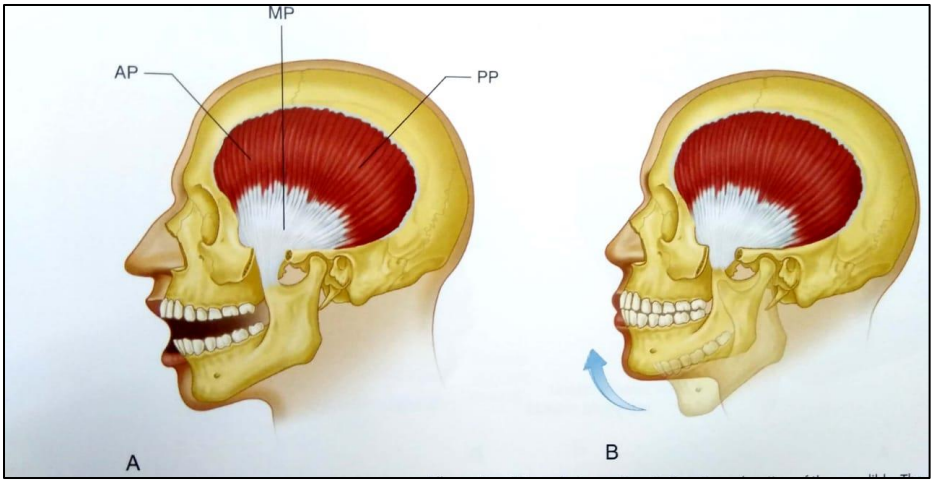
The temporalis is a large, fan-shaped muscle that originates from the temporal fossa and the lateral surface of the skull. Its fibers come together as they extend downward between the zygomatic arch and the lateral surface of the skull to form a tendon that inserts on the coronoid process and anterior border of the ascending ramus.

It can be divided into three distinct areas according to fiber direction and ultimate function. The anterior portion consists of fibers that are directed almost vertically. The middle portion contains fibers that run obliquely across the lateral aspect of the skull (slightly forward as they pass downward).

The posterior portion consists of fibers that are aligned almost horizontally, coming forward above the ear to join other temporalis fibers as they pass under the zygomatic arch.

When the temporal muscle contracts, it elevates the mandible and the teeth are brought into contact. If only portions contract, the mandible is moved according to the direction of those fibers that are activated. When the anterior portion contracts, the mandible is raised vertically. Contraction of the middle portion will elevate and retrude the mandible.

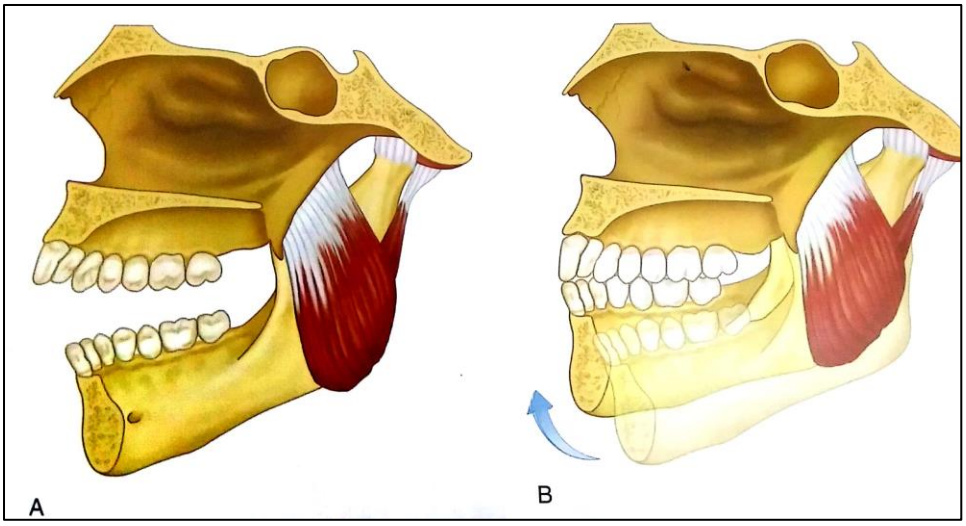
Function of the posterior portion is somewhat controversial. Although it would appear that contraction of this portion will retrude the mandible, DuBrul [77] suggests that the fibers below the root of the zygomatic process are the only significant ones and therefore contraction will cause elevation and only slight retrusion. Because the angulation of its muscle fibers varies, the temporalis is capable of coordinating closing movements. Thus it is a significant positioning muscle of the mandible.



A, Temporal muscle. AP, Anterior portion; MP, middle portion; PP, posterior portion. **B**, Function: elevation of the mandible. The exact movement by the location of the fibers or portion being activated.

PTERYGOIDEUS MEDIALIS

The medial (internal) pterygoid originates from the pterygoid fossa and extends downward, backward, and outward to insert along the medial surface of the mandibular angle. Along with the masseter, it forms a muscular sling that supports the mandible at the mandibular angle. When its fibers contract, the mandible is elevated and the teeth are brought into contact. This muscle is also active in protruding the mandible. Unilateral contraction will bring about a mediotrusive movement of the mandible.



A, Medial pterygoid muscle. **B**, Function: elevation of the mandible.

PTERYGOIDEUS LATERALIS

For many years the lateral (external) pterygoid was described as having two distinct portions or bellies: (1) an inferior and (2) a superior. The muscles are described as the inferior lateral and the superior lateral pterygoid**.**

INFERIOR LATERAL PTERYGOID

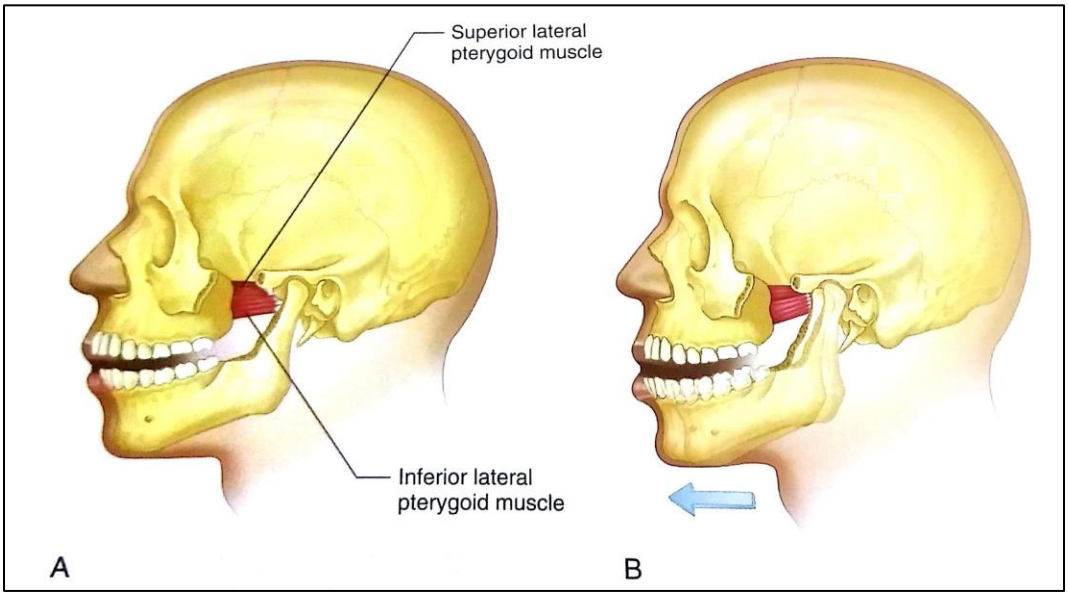
The inferiorlateral pterygoid originates at the outer surface of the lateral pterygoid plate and extends backward, upward, and outward to its insertion primarily on the neck of the condyle. When the right and left inferior lateral pterygoids contract simultaneously, the condyles are pulled down the articular eminences and the mandible is protruded. Unilateral contraction creates a mediotrusive movement of that condyle and causes a lateral movement of the mandible to the opposite side.

When this muscle functions with the mandibular depressors, the mandible is lowered and the condyles glide forward and downward on the articular eminences.

SUPERIOR LATERAL PTERYGOID

The superior lateral pterygoid is considerably smaller than the inferior and originates at the infratemporal surface of the greater sphenoid wing, extending almost horizontally, backward, and outward to insert on the articular capsule, the disc, and the neck of the condyle. The majority of the fibers of the superior lateral pterygoid (60% to 70%) attach to the neck of the condyle, with only 30% to 40% attaching to the disc.

Importantly, the attachments are more predominant on the medial aspect than on the lateral. Although the inferior lateral pterygoid is active during opening, the superior remains inactive, becoming active only in conjunction with the elevator muscles. The superior lateral pterygoid is especially active during the power stroke and when the teeth are held together. The power stroke refers to movements that involve closure of the mandible against resistance, such as in chewing or clenching the teeth together.



A, Inferior and superior lateral pterygoid muscles. **B**, Function of the inferior lateral pterygoid: protrusion of the mandible.

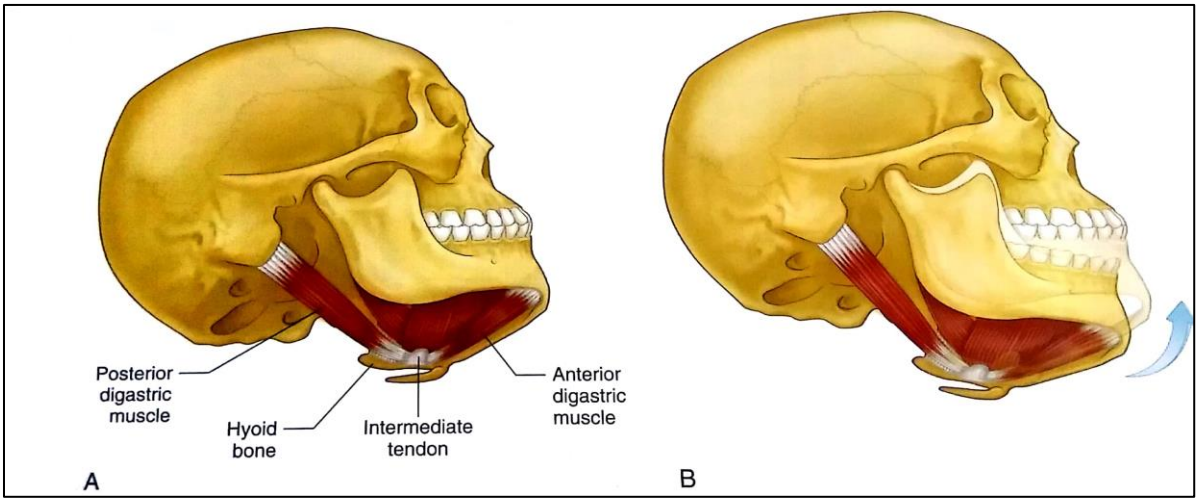
DIGASTRICUS

Although the digastric is not generally considered a muscle of mastication, it does have an important influence on the function of the mandible. It is divided into two portions, or bellies

1. The posterior belly originates from the mastoid notch, just medial to the mastoid process; its fibers run forward, downward, and inward to the intermediate tendon attached to the hyoid bone.

2. The anterior belly originates at a fossa on the lingual surface of the mandible, just above the lower border and close to the midline; its fibers extend downward and backward to insert at the same intermediate tendon as does the posterior belly.

When the right and left digastrics contract and the hyoid bone is fixed by the suprahyoid and infrahyoid muscles, the mandible is depressed and pulled backward and the teeth are brought out of contact. When the mandible is stabilized, the digastric muscles with the suprahyoid and infrahyoid muscles elevate the hyoid bone, which is a necessary function for swallowing.



A, Digastric muscle. **B**, Function: depression of the mandible.

UNIQUE CHARACTERISTICS OF TMJ

It becomes apparent that the TMJ articulation has some unique characteristics [78]. The following points agree with the conclusions presented by Sarnat, to which the joint evidences a pressure bearing adaption:

1) The roof of the fossa is thin and translucent and thinly covered with fibrocartilage.

2) The articular tubercle and post glenoid tubercle with its interposed disc is the craniomandibular articulation

3) The fibrous cartilage lining both mandible and temporal surfaces are avascular, as is the meniscus

4) The fiber bundles of cartilage and of meniscus show deep layers at right angles to bony surfaces and superficial layers are parallel

5) Deep layers are adapted to pressure and superficial layers to gliding under pressure

6) Presence of loose anterior and posterior capsule structure

7) Fan-shaped fibers of capsule restrict retrusal compression.

The TMJ is then actually two joints in one, and they act synchronously- The upper joint, between articular eminence and disc; the lower joint, between condyle and disc.

ETIOLOGY

The etiology of TMDs is complex and multifactorial. Numerous factors can contribute to TMDs. Factors that increase the risk of TMDs are called predisposing factors. Factors that cause the onset of TMDs are called initiating factors, and factors that interfere with healing or enhance the progression of TMDs are called perpetuating factors. In some instances a single factor may serve one or all of these roles. The successful management of TMDs depends on identifying and controlling these contributing factors.

For the dentist attempting to manage a TMD patient, it is critical to appreciate the major etiologic factors that may be associated with the condition. This is essential for selecting proper and effective therapy. Therefore it is appropriate to begin a thorough discussion of the major etiologic factors that lead to TMDs. Proper identification of the correct factor is basic for therapeutic success. A review of the scientific literature reveals five major factors associated with TMDs. These factors are the **occlusal condition, trauma, emotional stress, deep pain input, and parafunctional activities**. The importance of any of these factors varies greatly from patient to patient.

OCCLUSAL CONDITION

One contributing factor to TMDs that has been strongly debated for years is the occlusal condition. Early in the development of this field, the profession was convinced that occlusion was the most important contributing factor in TMD. More recently, many researchers argued that it may or may play a pivotal role.

When evaluating the relationship between occlusal factors and TMDs, the occlusal condition should be considered both statically and dynamically. To date, most occlusal studies have assessed the static relationship of the teeth. The studies cited previously considered the significance or no significance of occlusal factors relative to the TMDs as isolated static factors.

A single factor alone is not associated with TMD but a combination of factors in any given patient.

Pullinger, Seligman, and Gornbein attempted to do this by using a blinded multifactorial analysis to determine the weighted influence of each factor acting in combination with the other factors. The interaction of 11 occlusal factors was considered in randomly collected but strictly defined diagnostic groups compared with asymptomatic controls. Pullinger et al [79] concluded that no single occlusal factor was able to differentiate patients from healthy subjects. Four occlusal features, however, occurred mainly in TMD patients and were rare in normal subjects: (1) the presence of a skeletal anterior open bite, (2), retruded contact position (RCP)/ ICP slides of greater than 2 mm, (3) overjets of greater than 4 mm, and (4) five or more missing and unreplaced posterior teeth. Unfortunately, all of these signs are rare not only in healthy individuals but also in patient populations as well, indicating limited diagnostic usefulness of these features. The multifactorial analysis of Pullinger et al suggests that, except for a few defined occlusal conditions, there is a relatively minor relationship between occlusal factors and TMDs.

Dynamic functional relationships between occlusion and temporomandibular disorders

When considering the dynamic functional relationship between the mandible and the cranium, it appears that the occlusal condition can affect some TMDs in at least two ways [80]. The first relates to how the occlusal condition affects orthopedic stability of the mandible as it loads against the cranium. The second is how acute changes in the occlusal condition can influence mandibular function, thus leading to TMD symptoms.

Effects of Occlusal Factors on Orthopedic Stability

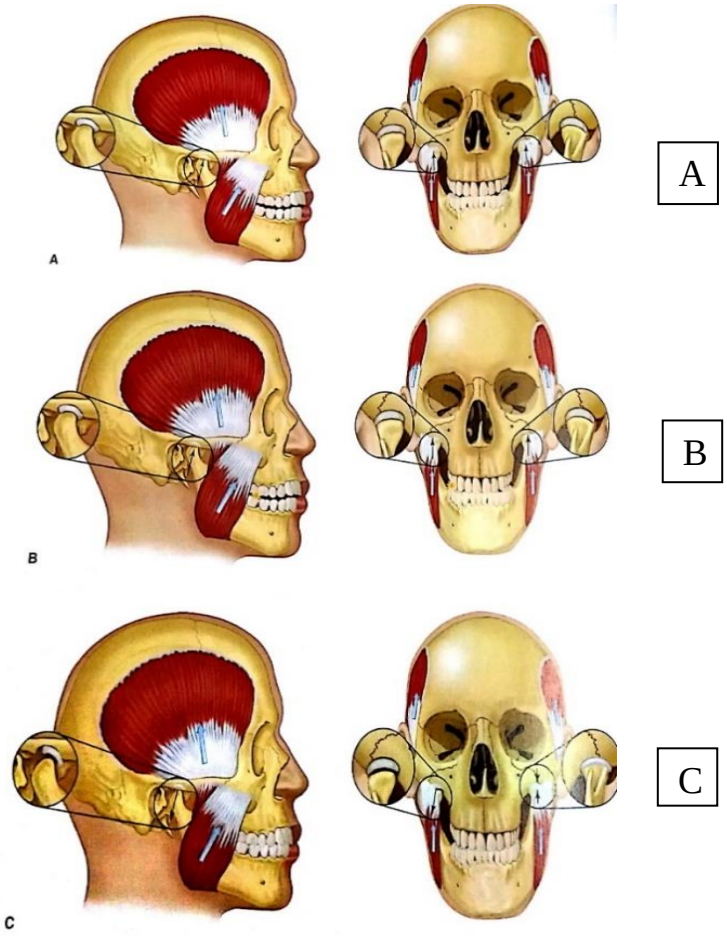
Orthopedic stability exists when the stable ICP (intercuspal position) of the teeth is in harmony with the Musculoskeletal (MS) position of the condyles in the fossae. When this condition exists, functional forces can be applied to the teeth and joints without tissue injury. However, when this condition does not exist, opportunities for overloading and

injury can be present. When orthopedic instability exists and the teeth are not in occlusion, the condyles are maintained in their MS positions by the elevator muscles. However, when orthopedic instability exists and the teeth are brought into contact, only one tooth may contact. This represents an unstable occlusal position, even though each condyle remains in a stable joint position. The individual must both maintain the stable joint position and only occlude on one tooth or bring the teeth into a more stable occlusal position, which may compromise joint stability. Because occlusal stability is basic to function, the priority is to achieve occlusal stability and the mandible is shifted to a position that maximizes occlusal contacts (the ICP).

When this occurs, the shift can force one or both condyles from its MS position, resulting in orthopedic instability. What this means is that when the teeth are in a stable position for loading, the condyles are not (or vice versa).

When orthopedic instability exists, however, merely bringing the teeth into occlusion may not create a problem because loading forces are minimal.

Problems arise when such an orthopedically unstable condition is loaded by the elevator muscles or by extrinsic forces (trauma). Because the ICP represents the most stable position for the teeth, loading is accepted by the teeth without consequence. If the condyles are also in a stable relationship in the fossae, loading occurs with no adverse effect to the joint structures. If, however, loading occurs when a joint is not in a stable relationship with the disc and fossa, unusual movement can occur in an attempt to gain stability. This movement, although small, is often a translatory shift between disc and condyle. Movement such as this can lead to strain to the discal ligaments and eventually elongation of the discal ligaments and thinning of the disc. These changes can lead to a group of intracapsular disorders.



A, With the teeth apart, the elevator muscles maintain the condyles in their musculoskeletally stable positions (i.e., superoanterior resting against the posterior slopes of the articular eminences). In this situation there is joint stability.

B, When the mouth is closed, a single tooth contact does not allow the entire dental arch to gain full intercuspation. At this moment there is occlusal instability but still joint stability. Because the condyles and teeth do not fit in a stable relationship at the same time, this is orthopedic instability.

C, To gain the occlusal stability necessary for functional activities, the mandible is shifted and the intercuspal position is achieved. At this moment the patient achieves occlusal stability, but the condyles may no longer be orthopedically stable.

This orthopedic instability may not pose a problem unless unusual loading occurs. If loading begins, the condyles will seek out stability and the unusual movement can lead to strains on the condyle/disc complex, resulting in an intracapsular disorder.

Two factors determine whether an intracapsular disorder will develop:

(1) The degree of orthopedic instability and
(2) The amount of loading.

Orthopedic instabilities with discrepancies of 1 or 2 mm are not likely significant enough to create a problem. However, as the discrepancy between the MS position of the condyles and the maximum intercuspation of the teeth becomes greater, the risk of intracapsular disorders increases.

The second factor that determines whether the patient will develop a TMD is the amount of loading. Therefore bruxing patients with orthopedic instability represent a higher risk for developing problems than nonbruxers with the same orthopedic instability. Also, forceful unilateral chewing can provide the mechanics that lead to sudden intracapsular disorders.

These variables may help explain why patients with similar occlusal conditions may not develop similar disorders. In fact, when the static occlusal relationships of two patients are compared, the patient with the more significant malocclusion may not always be the patient who develops the disorder.

The term dental malocclusion refers to the specific relationship of the teeth to each other, but it does not necessarily reflect any risk factors for the development of functional disturbances in the masticatory system (i.e., TMDs).

Dentists have observed dental malocclusions for years (e.g., an open bite, an Angle Class II). However, this dental malocclusion does not relate well to TMDs, as depicted by the literature. These dental malocclusions are only important when viewed in relationship to the joint position. Therefore merely looking in the mouth or viewing hand-held study casts does not provide insight as to the relative risk factor for TMDs. Only by observing

the occlusal relationship with respect to the stable joint position can one appreciate the degree of orthopedic instability that is present. Orthopedic instability is the critical factor that needs to be considered when accessing relative risk factors for TMDs.

Effects of Acute Changes in the Occlusal Condition and Temporomandibular Disorder

A second manner by which the occlusal condition can affect TMD symptoms is through a sudden or acute change.

Activities of the Masticatory System

Activities of the masticatory muscles can be divided into two basic types:

(1) Functional which include chewing, speaking, and swallowing; and

(2) Parafunctional (i.e., nonfunctional), which include clenching or grinding of the teeth (i.e., bruxism) and various oral habits.

The term muscle hyperactivity has also been used to describe any increased muscular activity over and above that necessary for function. Muscle hyperactivity thus includes not only the parafunctional activities of clenching, bruxing, and other oral habits but also any general increase in the level of muscle tonus.

Some muscle hyperactivity may not even involve tooth contact or jaw movement but merely represent an increase in the static tonic contraction of the muscle. Functional and parafunctional activities are quite different clinical entities. The former are controlled muscle activities that allow the masticatory system to perform necessary functions with minimum damage to any structure. Protective reflexes are constantly present, guarding against potential damaging tooth contacts. Interfering tooth contacts during function have

inhibitory effects on functional muscle activity. Therefore functional activities are directly influenced by the occlusal condition

Occlusal Contacts and Muscle Hyperactivity

Muscle hyperactivity is an inclusive term referring to any increased level of muscle activity that is not associated with a functional activity. This includes not only bruxism and clenching but also any increase in muscle tonicity related to habits, posture, or increased emotional stress

In a study by Le Bell et al [81] artificial occlusal interferences were placed in normal healthy subjects and in subjects with a past history of TMD symptoms. The TMD subjects had a history of TMD symptoms but none present at the time of the study. The artificial occlusal interferences were placed for 2 weeks and then removed. The results showed that the normal subjects reported some initial symptoms, which resolved in a few days. The prior-TMD subjects reported significantly greater symptoms at the 2 week session, the point at which the interference was removed. This suggests that the adaptability of TMD patients may be less than that of normal subjects and that they may therefore be more vulnerable to the development.

Rugh et al. [82] decided to challenge the concept that a premature occlusal contact could cause bruxism. They deliberately placed a high crown in 10 subjects and observed its effects on nocturnal bruxism. Although much of the dental profession was certain that this would lead to increased levels of bruxism, it did not. In fact, most of the subjects had a significant reduction in bruxism during the first two to four nights, followed by a return to normal bruxing levels. The conclusion from this study and from others would suggest that premature occlusal contacts do not increase bruxing activity. In other words, a high posterior occlusal contact does not necessarily increase muscle activity.

Closer evaluation of the study by Rugh et al also reveals that a significant percentage of the subjects wearing the poorly fitting crown reported an increase in muscle pain. This was not associated with increased bruxism, as many would have predicted. Rather, it was more likely produced by increased tonus of the elevator muscles in their attempt to protect the mandible from closing on the poorly fitting crown. In other words, a sudden occlusal change that disrupts the ICP can lead to a protective response of the elevator muscles known as protective co-contraction. If this response is maintained, it can result in pain.

TRAUMA

Certainly, trauma to the facial structures can lead to functional disturbances in the masticatory system. Ample evidence supports this concept. Trauma seems to have a greater impact on intracapsular disorder than muscular disorders. Trauma can be divided into two general types: macrotrauma and microtrauma. Macrotrauma is considered any sudden force that can result in structural alterations, such as a direct blow to the face. Microtrauma refers to any small force that is repeatedly applied to the structures over a long period of time.

Activities such as bruxism or clenching can produce microtrauma to the tissues that are being loaded (i.e., teeth, joints, or muscles).

EMOTIONAL STRESS

A common systemic event that can influence masticatory function is an increase in the level of emotional stress experienced by the patient. The emotional centers of the brain influence muscle function. The hypothalamus, the reticular system, and particularly the limbic system are primarily responsible for the emotional state of the individual. These centers influence muscle activity in many ways, one of which is through the gamma efferent pathways. Stress can affect the body by activating the hypothalamus, which in turn prepares the body to respond (the autonomic nervous system). The hypothalamus, through

complex neural pathways, increases the activity of the gamma efferents, which cause the intrafusal fibers of the muscle spindles to contract. This sensitizes the spindle so that any slight stretching of the muscle will cause a reflex contraction. The overall effect is an increase in tonicity of the muscle.

The therapist must understand and appreciate emotional stress because it commonly plays an important role in TMD. The patient's emotional state is largely dependent on the psychologic stress being experienced. Stress is described by Hans Selye [83] as "the nonspecific response of the body to any demand made upon it."

The important fact to remember is that the body reacts to the stressor by creating certain demands for readjustment or adaptation (the fight-or flight response). These demands are related in degree to the intensity of the stressor.

A simple way of describing stress is to consider it as a type of energy. When a stressful situation is encountered, energy is generated within the body and must be released in some way. Two types of releasing mechanisms exist: (1) external and (2) internal.

The external stress-releasing mechanism is represented by activities such as shouting, cursing, hitting, or throwing objects. Physical exercise is a healthy way of energy release to deal with stress.

The internal stress-releasing mechanism is used when a person releases the stress internally and develops a psychophysiologic disorder such as irritable bowel syndrome, hypertension, certain cardiac arrhythmia disorder, asthma, or an increase in the tonicity of the head and neck musculature. As accurate documentation regarding the prevalence of increased muscle tension is accumulated, it may be learned that this type of stress-releasing mechanism is by far the most common. The clinician should remember that the perception of the stressor, in both type and intensity, varies greatly from person to person.

What may be stressful for one person quite possibly represents no stress for another. Therefore it is difficult to judge the intensity of a given stressor on a given patient.

Increased levels of emotional stress experienced by the patient increase not only the tonicity of head and neck muscles but also the levels of nonfunctional muscle activity such as bruxism or tooth clenching.

Another systemic factor that can influence an individual's physiologic tolerance to certain events is his or her sympathetic activity or tone. The autonomic nervous system constantly monitors and regulates numerous subconscious systems that maintain homeostasis.

One of the functions of the autonomic system is to regulate blood flow within the body. The sympathetic nervous system is closely related to the fight-or-flight reflex activated by stressors. Therefore in the presence of stress the capillary blood flow in the outer tissues is constricted, permitting increased blood flow to the more important musculoskeletal structures and internal organs. The result is a cooling of the skin such as the hands. Prolonged activity of the sympathetic nervous system can affect certain tissues such as the muscles. It has been suggested that sympathetic activity can increase muscle tone, thereby producing a painful muscle condition. Increased sympathetic activity or tone therefore represents an etiologic factor that can influence TMD symptoms.

Emotional stress can also influence TMD symptoms by reducing the patient's physiologic tolerance. This is likely to occur because of increased sympathetic tone. This effect often represents the individual's learned response to various stressors.

DEEP PAIN INPUT

Although often overlooked, a common concept is that sources of deep pain input can cause altered muscle function. Deep pain input can centrally excite the brainstem, producing protective co-contraction. This represents a normal healthy manner in which the body responds to injury or threat of injury. Therefore it is reasonable to find a patient who is suffering with pain, such as toothache (i.e., necrotic pulp), to have limited mouth opening. This represents the body's response to protect the injured part by limiting its use.

This clinical finding is common in many toothache patients. Once the tooth pain is resolved, normal mouth opening returns.

The limited mouth opening is merely a secondary response to the experience of the deep pain. If the clinician does not recognize this phenomenon, however, he or she may conclude that the limited mouth opening is a primary TMD problem and treatment would be misdirected. Any source of constant deep pain input can represent an etiologic factor that may lead to limited mouth opening and therefore clinically present as TMD. Tooth pain, sinus pain, and ear pain can create this response.

Even pain sources remote to the face, such as cervical pain input, can lead to this condition. Too often, dentists do not appreciate this phenomenon and begin treating a patient for TMD complaints. Only after treatment failure is the cervical pain condition identified as being responsible for creating the facial pain and limited mouth opening. Understanding how this occurs is basic to treatment and emphasizes the importance of making the correct diagnosis

PARAFUNCTIONAL ACTIVITIES

As previously discussed, parafunctional activity refers to any activity that is not considered functional (chewing, speaking, and swallowing) [84]. This includes bruxing, clenching, and certain oral habits. Some of these activities may be responsible for creating TMD symptoms parafunctional activity can be subdivided into two general types:

(1) That which occurs through the day (diurnal) and

(2) That which occurs at night (nocturnal).

Diurnal Activity

Parafunctional activity during the day consists of clenching and grinding, as well as many oral habits that are often performed without the individual even being aware of them (e.g., cheek and tongue biting; finger and thumb sucking; unusual postural habits;

occupation-related activities such as biting on pencils, pins, or nails or holding objects under the chin [a telephone or violin]).

During daily activities, individuals commonly place their teeth together and apply force. This type of diurnal activity may be seen in someone who is concentrating on a task or performing a strenuous physical chore. The masseter muscle contracts periodically in a manner that is totally irrelevant to the task at hand. Such irrelevant activity, is commonly associated with many daytime tasks (e.g., driving a car, reading, writing, typing, lifting heavy objects). Some diurnal activities are closely related to the task being accomplished, such as an underwater diver biting on the mouth piece or a musician playing certain musical instruments.

The clinician must recognize that most parafunctional activities occur at a subconscious level. In other words, individuals are often not even aware of their clenching or cheek-biting habits.

Therefore merely questioning the patient is not a reliable way to assess the presence or absence of these activities. In many instances, once the clinician makes the patient aware of the possibility of these diurnal activities, he or she will recognize them and can then decrease them.

Nocturnal Activity

Data from various sources have suggested that parafunctional activity during sleep is quite common and seems to take the form of single episodes (referred to as clenching) and rhythmic contractions (known as bruxing).

Whether these activities result from different etiologic factors or are the same phenomenon in two different presentations is not known. In many patients both activities occur and are sometimes difficult to separate. For that reason clenching and bruxism are often referred to as bruxing events

Sleep

To best understand nocturnal bruxism, the clinician should first have an appreciation of the sleep process. Sleep is investigated by monitoring the brain wave activity (electroencephalogram) of an individual during sleep. This monitoring is called a polysomnogram. A polysomnogram reveals two basic types of brain wave activities that appear to cycle during a night of sleep: (1) alpha and (2) delta. Alpha waves are relatively fast (about 10 waves per second) and are the predominant waves observed during the early stages of sleep or light sleep. Delta waves are slower waves (0.5 to 4 waves per second) and are observed during the deeper stages of sleep.

The sleep cyclic is divided into four stages that are free of rapid eye movement (non-REM) followed by a period of REM. Stages 1 and 2 represent the early phases of light sleep and are made up of groups of fast alpha waves along with a few beta waves and "sleep spindles." Stages 3 and 4 represent the deeper stages of sleep with the predominance of the slower beta waves. During a normal cycle of sleep, a subject will pass from the light stages of 1 and 2 into the deeper stages of 3 and 4. The subject will then pass through a stage of sleep that is quite different from the others. This stage appears as a desynchronized activity in which other physiologic events occur, such as muscle twitching of the extremities and facial muscles, alterations in heart and breathing rates, and rapid movement of the eyes beneath the eyelids.

Because of this last characteristic, this phase has been called REM sleep. Dreaming occurs most commonly during REM sleep. After the REM period the person typically moves back into a lighter stage, and the cycle repeats itself throughout the night. Each complete cycle of sleep takes from 60 to 90 minutes, resulting in an average of 4 to 6 cycles of sleep per night.

A REM phase usually occurs following stage 4 sleep and lasts from 5 to 15 minutes. Non-REM sleep is thought to be important in restoring function of the body systems. During this phase of sleep there is an increase in synthesis of vital macromolecules (e.g., proteins, RNA). REM sleep, on the other hand, seems to be important in restoring function of the cortex and brainstem activities.

During this phase of sleep, emotions are dealt with and smoothed out. It is a time at which recent experiences are brought into perspective with old pathways.

The importance of these two types of sleep is evident from studies that attempt to deprive individuals of one or the other. When an individual is experimentally deprived of REM sleep, certain emotional states become predominant. Individuals show greater anxiety and irritability. They also have difficulty concentrating. It would appear that REM sleep is important for psychic rest. A different finding is revealed when an individual is deprived of non-REM sleep. When a normal subject is experimentally deprived of non-REM sleep for several nights, the subject will often begin to complain of musculoskeletal tenderness, aching, and stiffness. This may result from the individual's inability to restore metabolic requirements. In other words, non-REM sleep is important for physical rest.

Forces of Tooth Contacts

In evaluating the effect of tooth contacts on the structures of the masticatory system, two factors must be considered: the magnitude and duration of the contacts.

A reasonable way to compare the effects of functional and parafunctional contacts is to evaluate the amount of force placed on the teeth in pounds per second per day for each activity.

Comparison of Functional and Parafunctional Activities Using Five Common Factors

Factor	Functional activity	Para functional activity
Forces of tooth contacts	17,200 lb/sec/day	57,600 lb/sec/day, possibly more
Direction of applied forces to teeth	Vertical (well tolerated)	Horizontal (not well tolerated)
Mandibular position	Centric occlusion (relatively stable)	Eccentric movements (relatively unstable)
Type of muscle contraction Reflexes	Isotonic (physiologic)	Isometric (nonphysiologic)
Influence of protective reflexes	Present	Obtunded
Pathologic effects	Unlikely	Very likely

It can easily be appreciated that force and duration of tooth contacts during parafunctional activity pose a much more serious consequence to the structures of the masticatory system than do those of functional activity.

Direction of Applied Forces

During chewing and swallowing, the mandible is moving primarily in a vertical direction. As it closes and tooth contacts occur, the predominant forces applied to the teeth are also in a vertical direction. Vertical forces are accepted well by the supportive structures

of the teeth. During parafunctional activities, however (e.g., bruxism), heavy forces are applied to the teeth as the mandible shifts from side to side. This shifting causes horizontal forces, which are not well accepted and increase the likelihood of damage to the teeth and/or supportive structures.

Mandibular Position

Most functional activity occurs at or near the ICP. Although the ICP may not always be the most MS position for the condyles, it is stable for the occlusion because of the maximum number of tooth contacts it provides. Therefore the forces of functional activity are distributed to many teeth, minimizing potential damage to an individual tooth. Tooth wear patterns suggest that most parafunctional activity occurs in eccentric positions. Few tooth contacts occur during this activity, and often the condyles are translated far from a stable position.

Activity in this type of mandibular position places more strain on the masticatory system, rendering it more susceptible to breakdown.

Type of Muscle Contraction

Most functional activity consists of well-controlled and rhythmic contraction and relaxation of the muscles involved during jaw function. This isotonic activity permits adequate blood flow to oxygenate the tissues and eliminate by products accumulated at the cellular level. Therefore functional activity is a physiologic muscle activity. Parafunctional activity, by contrast, often results in sustained muscle contraction over long periods. This type of isometric activity inhibits normal blood flow within the muscle tissues.

As a result, there is an increase in metabolic byproducts within the muscle tissues, creating the symptoms of fatigue, pain, and spasms.

Influences of Protective Reflexes

Neuromuscular reflexes are present during functional activities, protecting the dental structures from damage. During parafunctional activities, however, the neuromuscular protecting mechanisms appear to be somewhat obtunded, resulting in less influence over muscle activity. This allows parafunctional activity to increase and eventually reach levels high enough to create breakdown of the structures involved.

After considering these factors, it becomes apparent that parafunctional activity is more likely to be responsible for structural breakdown of the masticatory system and TMDs than functional activity. This is an important concept to remember because many patients come to the dental office complaining of functional disturbances such as difficulty in eating or pain during speaking.

The clinician should remember that functional activities often bring to the patient's awareness the symptoms that have been created by parafunctional activities. In these instances treatment should be primarily directed toward controlling parafunctional activity. Altering the functional activity of which the patient is complaining can be helpful in reducing symptoms, but alone it is not sufficient treatment to resolve the disorder.

Another concept to remember is that parafunctional activities occur almost entirely subconsciously. Much of this damaging activity occurs during sleep in the form of bruxism and clenching. Often patients awake with no awareness of the activity that has occurred during sleep. They may even awake with TMD symptoms but not relate this discomfort to any causative factor. When they are questioned regarding bruxism, most will deny such activity.

PATIENT ADAPTABILITY

The etiology of TMD is not simple but multifactorial. Five well defined etiologic factors are presented. Most clinicians overlook the factor of adaptability of the patient. Each patient has a biological system that can tolerate a certain degree of variation from ideal. Therefore some malocclusion, minor trauma, some emotional stress and presence of deep pain and parafunction do not always lead to TMD symptoms. Often these fall within the adaptability of the patient's musculoskeletal system. Patients who are fortunate to have significant adaptability may present with more striking etiological factors and yet not exhibit any TMD symptoms.

CLASSIFICATION

Throughout the years the terminology and classification of many disorder affecting the temporomandibular joint (TMJ) have been based on etiologic assumptions. A variety of concepts have been suggested, and their proponents introduced their own terminology for the same syndrome.

Since Costen's syndrome, the conviction that malocclusion is the major etiologic factor in disorders of the masticatory system has dominated dental literature for many years. In 1959, Schwartz [53] described the TMJ pain-dysfunction syndrome (PDS) as a basically functional problem of the masticatory musculature. One or more symptoms of the classical triad (pain in the region of the temporomandibular joints and/or the masticatory muscles, joint noises during condylar movement and limitation of mandibular movement) are required for the diagnosis. However, the presence of only clicking or crepitation is not considered to be sufficient.

This concept was expanded by Laskin [58] in 1969, who introduced the "myofascial pain-dysfunction syndrome" (MPD) as a subgroup of PDS. It requires absence of clinical or radiological evidence of organic joint changes and lack of joint tenderness when palpated through the external auditory meatus. The significance of the joint itself, and the articular disk in particular, was reestablished by Farrar in 1971 when he introduced the concept of interferences during mandibular movement due to articular disk displacement or internal derangements [85].

At present, the label pain-dysfunction syndrome is used in many varieties as a diagnostic entity, thus contributing to the existing controversy about TMJ problems. This may explain why there is no generally accepted classification available.

Redefinition and systematic classification of TMJ disorders was recommended by Farrar in 1983 as a first step toward resolution of the controversies [86]. Although several important features about joint disorders in general and TMJ disorders in particular, especially those related to etiology, are still subjects of research and discussion, a

considerable amount of information useful for classifying temporomandibular (TM) disorders is now available in literature.

In dental literature, four main groups of TMJ disorders may be distinguished. The pain-dysfunction syndrome is generally referred to as the major disorder affecting the TMJ, which was first considered to be occlusion-related. Since the late 1960's, emotional stress has been increasingly regarded as the major etiologic factor [87]. The other groups include disorders due to trauma, both acute and chronic, arthritis, including organic disorders of inflammatory, infectious or degenerative origin, and congenital and developmental disorders. During the past decade, several approaches to classification have been proposed. In 1980 Scheman distinguished local, referred, and psychogenic disorders [88]. Ogus and Toiler in 1980 separated common from uncommon disorders [89].

Farrar and McCarty in 1982 described three groups of TMJ disorders: diseases, dysfunctions, and developmental disorders [90]. In an attempt to achieve general agreement, several consensus conferences were dedicated to the classification of TM disorders.

The classification suggested by the American Academy of Craniomandibular Disorders separates articular from extra-articular, and, within these groups, structural from functional disorders.

Classifications Suggested at Dental Conferences AMERICAN SOCIETY OF CRANIOMANDIBULAR DISORDERS (McNeill et al. 1980) [91]

I Articular noninflammatory disorders

1. Degenerative joint disease
2. Traumatic disorders: Fractures
3. Mechanical:
    a) Disk dysfunction
    b) Disk displacement
    c) Disk dyscrasias
    d) Condylar displacement

4. Congenital disorders:
    a) Agenesis
    b) Hypoplasia
    c) Hyperplasia
    d) Osteochondromatosis
5. Neoplasia:
    a) Chondroma
    b) Osteoma
    c) Primary malignant
    d) Metastatic

II Articular inflammatory disorders

1. Synovitis
2. Diskitis
3. Capsulitis
4. Osteochondritis
5. Contusion
6. Arthritides (local osteoarthritis, traumatic, rheumatoid, infectious arthritis, polyarthritis (including gout, lupus), rheumatoid variants)

III Other Arthropathies

1. Ankylosis
    a) Fibrous
    b) Osseous
2. Chronic hypomobilities
3. Capsular fibrosis

IV Nonarticular disorders

1. Neuromuscular -myofascitis, contracture, trismus/spasm -myofascial trigger points, myofascial paindysfunction syndrome -bruxism
2. Occlusal conditions -structural imbalance, premature posterior contacts, lack of posterior support
3. Psychogenic syndromes -occlusal neurosis, conversion hysteria, atypical facial pain

AMERICAN DENTAL ASSOCIATION (Modification according to Bell, 1985) [92]

I. Disk interference disorders:

1. Class I interference
2. Class 11 interference (initial opening)
3. Class Ill interference (translation)
4. Class IV interference (hypermobility)
5. Class V interference (spontaneous. dislocation)

II. Growth disorders:

1. Developmental abberations
2. Acquired changes in structure
3. Neoplasia

III. Articular inflammatory disorders

1. Synovitis/capsulitis
2. Retrodiscitis
3. Inflammatory arthritis (traumatic, degenerative, infectious, rheumatoid, hyperuricemia)

IV. Other Arthropathies

1. Chronic hypomobilities
2. Capsular fibrosis
3. Ankylosis (fibrous, bony)

V. Non articular disorders

1. Acute muscle disorders:
    a) Protective Muscle Splinting spasm (MPD)
    b) Myositis

In 2013, the International Research Diagnostic Criteria for Temporomandibular Dysfunction Consortium Network published an updated classification for TMDs [93].

I. Articular disorders (intra articular)

1. Congenital or developmental disorders
    a) Condylar hyperplasia
    b) First and second branchial arch disorders
    c) Idiopathic condylar resorption
2. Degenerative joint disorders
    a) Inflammatory: capsulitis, synovitis, polyarthritides (rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Reiter syndrome, gout)
    b) Noninflammatory: osteoarthritis
3. Disk derangement disorders
    a) Displacement with reduction
    b) Displacement without reduction (closed lock)
    c) Perforation
4. Infection
5. Neoplasia
6. Temporomandibular hypermobility
    a) Dislocation
    b) Joint laxity
    c) Subluxation
7. Temporomandibular hypomobility
    a) Ankylosis: true ankylosis (bony or fibrous) or pseudoankylosis
    b) Postradiation fibrosis
    c) Trismus

8. Trauma
    a) Contusion
    b) Fracture
    c) Intracapsular hemorrhage

II. Masticatory muscle disorders (extra-articular)

1. Local myalgia
2. Myofascial pain disorder
3. Myofibrotic contracture
4. Myositis
5. Myospasm
6. Neoplasia

Classification System Used for Diagnosing Temporomandibular Disorders (According to Okeson JP)[94]

I. Masticatory muscle disorders

1. Protective co-contraction
2. Local muscle soreness
3. Myofascial pain
4. Myospasm
5. Centrally mediated myalgia

II. Temporomandibular joint (TMJ) disorders

1. Derangement of the condyle-disc complex
    a) Disc displacements
    b) Disc dislocation with reduction
    c) Disc dislocation without reduction
2. Structural incompatibility of the articular surfaces
    a) Deviation in form
        i. Disc
        ii. Condyle

iii. Fossa

b) Adhesions

i. Disc to condyle

ii. Disc to fossa

c) Subluxation (hypermobility)

d) Spontaneous dislocation

3. Inflammatory disorders of the TMJ

a) Synovitis/capsulitis

b) Retrodiscitis

c) Arthritides

i. Osteoarthritis

ii. Osteoarthrosis

iii. Polyarthritides

d) Inflammatory disorders of associated structures

i. Temporal tendonitis

ii. Stylomandibular ligament inflammation

III. Chronic mandibular hypomobility

1. Ankylosis

a) Fibrous

b) Bony

2. Muscle contracture

a) Myostatic

b) Myofibrotic

3. Coronoid impedance

IV. Growth disorders

1.Congenital and developmental bone disorders

a) Agenesis

b) Hypoplasia

c) Hyperplasia

d) Neoplasia

2.Congenital and developmental muscle disorders

a) Hypotrophy
b) Hypertrophy
c) Neoplasia

SIGNS AND SYMPTOMS OF TEMPOROMANDIBULAR DISORDERS

A sign is an objective clinical finding that the clinician uncovers during a clinical examination. A symptom is a description or complaint reported by the patient. Patients are acutely aware of their symptoms, yet they may not be aware of their clinical signs. When evaluating a patient, it is important to identify both signs and symptoms clearly.

The clinical signs and symptoms of masticatory dysfunction can be grouped into categories according to structures that are affected:

1. The muscles,
2. The temporomandibular joints (TMJs),
3. The dentition.

Muscle and TMJ disorders make up the group of conditions known as temporomandibular disorders (TMDs).

Functional disorders of masticatory muscles are probably the most common TMD complaint of patients seeking treatment in the dental office. They are generally grouped in a large category known as masticatory muscle disorders.

As with any pathologic state, two major symptoms can be observed [95]:

(1) Pain

(2) Dysfunction.

PAIN

Certainly the most common complaint of patients with masticatory muscle disorders is muscle pain, which may range from slight tenderness to extreme discomfort. Pain felt in muscle tissue is called myalgia. Myalgia can arise from increased levels of muscular use. The symptoms are often associated with a feeling of muscle fatigue and tightness.

The severity of muscle pain is directly related to the functional activity of the muscle involved. Therefore patients often report that the pain affects their functional activity. When a patient reports pain during chewing or speaking, these functional activities are not usually the cause of the disorder. Instead they heighten the patient's awareness of it. More likely some type of activity or central nervous system (CNS) effect has led to the muscle pain, and thus treatment directed toward the functional activity will not be appropriate or successful; rather, treatment needs to be directed toward diminishing the CNS effects or possibly muscle hyperactivity.

Another common symptom associated with masticatory muscle pain is headache.

DYSFUNCTION

Dysfunction is a common clinical symptom associated with masticatory muscle disorders. Usually it is seen as a decrease in the range of mandibular movement. When muscle tissues have been compromised by overuse, any contraction or stretching increases the pain. Therefore to maintain comfort, the patient restricts movement within a range that does not increase pain levels.

Clinically this is seen as an inability to open widely. The restriction may be at any degree of opening depending on where discomfort is felt. In some myalgic disorders the patient can slowly open wider, but the pain is still present and may even become worse.

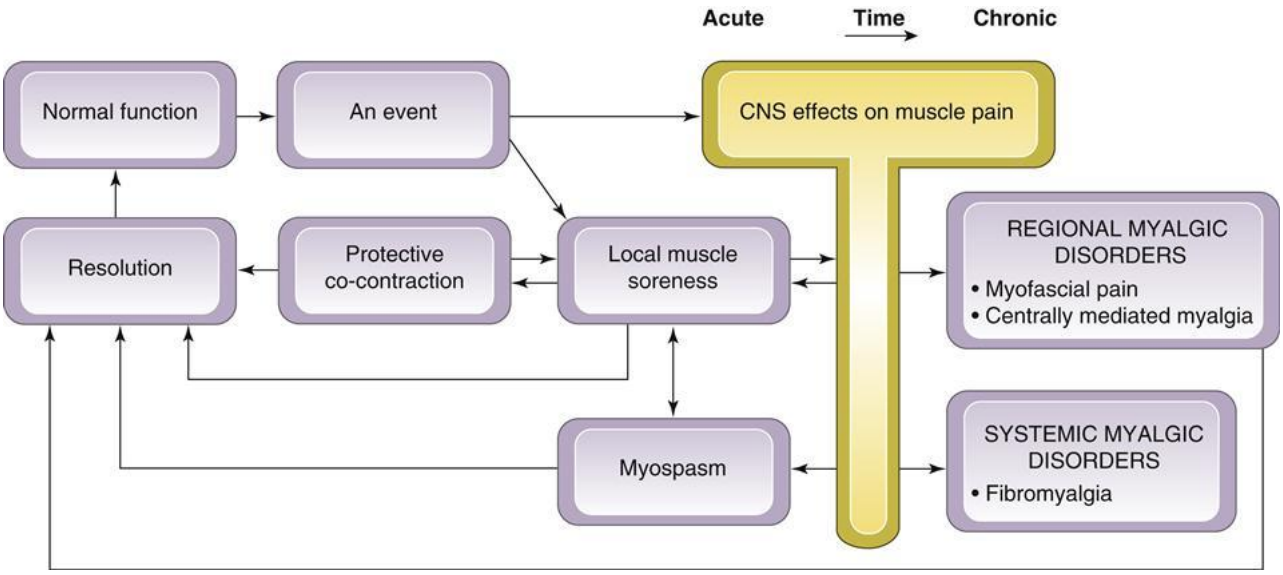
Acute malocclusion is another type of dysfunction. Acute malocclusion refers to any sudden change in the occlusal condition that has been created by a disorder. An acute

malocclusion may result from a sudden change in the resting length of a muscle that controls jaw position. When this occurs the patient describes a change in the occlusal contact of the teeth. The mandibular position and resultant alteration in occlusal relationships depend on the muscles involved.

CLINICAL MASTICATORY MUSCLE PAIN MODEL

This model depicts the relationship among various clinically identifiable muscle pain disorders along with some etiologic considerations.[96] The model begins with the assumption that the muscles of mastication are healthy and functioning normally. Normal muscle function can be interrupted by certain types of events. If an event is significant, a muscle response known as protective co-contraction (muscles splinting) occurs.

In many instances the consequence of the event is minor and co-contraction resolves quickly, allowing muscle function to return to normal. If, however, protective co-contraction is prolonged, local biochemical and later structural changes can occur, creating a condition known as local muscle soreness. This condition may resolve spontaneously with rest or may need the assistance of treatment.



EVENTS

Normal muscle function can be interrupted by various types of events. These events can arise from either local or systemic factors. Local factors represent any events that acutely alter sensory or proprioceptive input in the masticatory structures (e.g., the fracture of a tooth or the placement of a restoration in supraocclusion). Trauma to local structures such as tissue damage caused by a dental injection represents another type of local event.

Trauma might also arise from excessive or unaccustomed use of masticatory structures, such as chewing unusually hard food or chewing for a long period of time (i.e., gum chewing). Opening too wide may produce a strain to ligaments supporting the joint and/or muscles. This may occur as a result of a long dental procedure or even by simply opening too wide (i.e., yawning)

Any source of constant deep pain input may also represent a local factor that alters muscle function. This pain input may have its source in local structures such as the teeth, joints, or even the muscles themselves. The source of the pain, however, is not significant because any constant deep pain, even idiopathic pain, may create a muscle response.

Systemic factors may also represent events that can interrupt normal muscle function. One of the most commonly recognized systemic factors is emotional stress. Other systemic factors can influence muscle function and are less understood, such as acute illness or viral infections. Likewise, a broad category of poorly understood to each patient exists. Such factors include immunologic resistance and autonomic balance of the patient. These factors seem to reduce the individual's ability to resist or combat the challenge or demand created by the event. Constitutional factors are likely to be influenced by age, gender, diet, and perhaps even genetic predisposition.

HISTORY TAKING

The signs and symptoms of temporomandibular disorders (TMDs) are extremely common. Some of these appear as significant symptoms that motivate the patient to seek treatment. Signs of which the patient is unaware are said to be subclinical. Some subclinical signs can later become apparent and represent more significant functional disturbances if left unattended. Therefore it is important to identify any and all signs and symptoms of functional disturbances in each patient.

This is not to suggest that all signs indicate a need for treatment. The significance of the sign and the etiology, as well as the prognosis of the disorder, are factors that determine the need for treatment. The effectiveness and success of treatment lie in the ability of the clinician to establish the proper diagnosis. This can be established only after a thorough examination of the patient for the signs and symptoms of functional disturbances. Each sign represents a portion of information needed to establish a proper diagnosis. Therefore it is extremely important that each sign and symptom be identified by means of a thorough history and examination procedure.

The purpose of a history and examination is to identify any area or structure of the masticatory system that shows breakdown or pathologic change. To be effective, the examiner must have a sound understanding of the clinical appearance and function of the healthy masticatory system. Breakdown in the masticatory system is generally signified by pain and/or dysfunction. History and examination procedures should therefore be directed toward the identification of masticatory pain and dysfunction.

Masticatory structures are the teeth, periodontium, supporting structures of the teeth, temporomandibular joints (TMJs), and muscles that move the mandible.

Unfortunately, however, disorders of the head and neck can frequently lead to heterotopic pains that are felt in the masticatory structures but do not have their sources within masticatory structures. These types of pains must be properly identified during an examination so that an accurate diagnosis can be established. To be effective, treatment must be directed toward the source of pain and not toward the site. For dental treatment to

be effective, the pain must be of masticatory origin. A general rule in identifying masticatory pain is that jaw function usually aggravates or accentuates the problem.

Screening history and examination

The purpose of the screening history and examination is to identify patients with subclinical signs, as well as symptoms that the patient may not relate but are commonly associated with functional disturbances of the masticatory system (e.g., headaches, ear symptoms). The screening history consists of several questions that will help orient the clinician to any TMDs. These can be asked personally by the clinician or may be included in a general health and dental questionnaire that the patient completes before being seen by the dentist.

Accompanying the screening history is a short screening examination. This should be brief and is an attempt to identify any variation from normal anatomy and function. It begins with an inspection of the facial symmetry. Any variation from the general bilateral symmetry should raise suspicion and indicate the need for further examination.

The screening examination also includes observations of jaw movement. Restriction or irregular mandibular movements are indications for a more thorough examination. Several important structures of the masticatory system are palpated for pain or tenderness during the screening examination.

If the screening history and examination reveal positive findings, a more thorough history and examination for TMDs is completed. Three basic structures should be examined for pain and/or dysfunction: the muscles, TMJs, and dentition. Before the examination, a complete history of the problem, both past and present, is obtained from the patient.

HISTORY TAKING FOR TEMPOROMANDIBULAR DISORDERS

The history can be obtained by one of two manners. Some clinicians prefer to converse directly with the patient concerning the history of the problem. This allows them to direct questions that appropriately follow the patient's previous response. A more thorough and consistent history can be taken by a written questionnaire that includes all areas of concern.

The history begins with a complete medical questionnaire identifying any major medical problems of the patient. Major medical problems can play an important role in functional disturbances. For example, a patient's generalized arthritic condition can also affect the TMJ.

An effective history centers on the patient's chief complaint. This is a good starting point in obtaining needed information. The patient is allowed to describe, in his or her own words, the chief complaint. A complete history obtains information in the following specific areas [97].

Features to Be Included in a Thorough Orofacial Pain History

I Chief complaint (may be more than one).

A. Location of pain

B. Onset of pain

1. Associated with other factors

2. Progression

C. Characteristics of pain

1. Quality of pain

2. Behavior of pain

a. Temporal

b. Duration

c. Localization

3. Intensity of pain

4. Concomitant symptoms

5. Flow of pain

D. Aggravating and alleviating factors

1. Function and parafunction

2. Physical modalities

3. Medications

4. Emotional stress

5. Sleep disturbances

6. Litigation

E. Past consultations and/or treatments

F. Relationship to other pain complaints

II. Past medical history

III. Review of systems

IV. Psychologic assessment

CLINICAL EXAMINATION

Once the history has been obtained and thoroughly discussed with the patient, a clinical examination is performed. It should identify any variations from the normal health and function of the masticatory system.

Even before one examines the masticatory structures, it is important to evaluate gross function of the cranial nerves and the eyes, ears, and neck. If abnormal findings are identified, an immediate referral to the appropriate specialist is indicated.

MUSCLE EXAMINATION

A frequent clinical sign of compromised muscle tissue is pain. The condition that brings about compromise or unhealthy muscle tissue may be muscle overuse or physical trauma such as overstretching or receiving a blow to the muscle tissue itself.

Most often the muscles of mastication become compromised through increased activity. As the number and duration of contractions increase, so also do the physiologic needs of the muscle tissues. Increased muscle tonicity or hyperactivity, however, can lead to a decrease in blood flow to the muscle tissues, lowering the inflow of nutrient substances needed for normal cell function while accumulating metabolic waste products

Muscle Palpation

A widely accepted method of determining muscle tenderness and pain is by digital palpation. A healthy muscle does not elicit sensations of tenderness or pain when palpated. Therefore if a patient reports discomfort during palpation of a specific muscle, it can be deduced that the muscle tissue has been compromised by either trauma or fatigue.

Palpation of the muscle is accomplished mainly by the palmar surface of the middle finger, with the index finger and forefinger testing the adjacent areas. Soft but firm pressure

is applied to the designated muscles, with the fingers compressing the adjacent tissues in a small circular motion.

A single firm thrust of 1 or 2 seconds duration is usually better than several light thrusts. During palpation the patient is asked whether it hurts or is just uncomfortable. When trigger points are located, an attempt should be made to determine whether there is any pattern of pain referral. Pressure should be applied to the trigger point for 4 to 5 seconds, and the patient is asked whether the pain is felt to radiate in any direction.

If a pattern of referred pain is reported, it should be noted on a drawing of the face for future reference. Patterns of referred pain are often helpful in identifying and diagnosing certain pain conditions.

A routine muscle examination includes palpation of the following muscles or muscle groups: temporalis, masseter, sternocleidomastoideus, and posterior cervical (e.g., the splenius capitis and trapezius) [98].

The muscle examination also includes evaluation of the medial and lateral pterygoids by functional manipulation. This technique is used for muscles that are impossible or nearly impossible to palpate manually.

Temporalis

The temporalis is divided into three functional areas, and therefore each area is independently palpated. The anterior region is palpated above the zygomatic arch and anterior to the TMJ. Fibers of this region run essentially in a vertical direction.

The middle region is palpated directly above the TMJ and superior to the zygomatic arch. Fibers in this region run in an oblique direction across the lateral aspect of the skull.

The posterior region is palpated above and behind the ear. These fibers run in an essentially horizontal direction.

If uncertainty arises regarding the proper finger placement, the patient is asked to clench the teeth together. The temporalis will contract, and the fibers should be felt beneath the fingertips.

Masseter

The masseter is palpated bilaterally at its superior and inferior attachments. First, the fingers are placed on each zygomatic arch (just anterior to the TMJ). They are then dropped down slightly to the portion of the masseter attached to the zygomatic arch, just anterior to the joint. Once this portion (the deep masseter) has been palpated, the fingers drop to the inferior attachment on the inferior border of the ramus. The area of palpation is directly above the attachment of the body of the masseter (i.e., the superficial masseter)

Sternocleidomastoideus

Although the SCM does not function directly in moving the mandible, it is specifically mentioned because it often becomes symptomatic with TMDs and it is easily palpated. The palpation is done bilaterally near its insertion on the outer surface of the mastoid fossa, behind the ear. The entire length of the muscle is palpated, down to its origin near the clavicle. The patient is asked to report any discomfort during the procedure.

Posterior Cervical Muscles

The posterior cervical muscles (trapezius, longissimus [capitis and cervicis], splenius [capitis and cervicis], and levator scapulae) do not directly affect mandibular movement; however, they do become symptomatic during certain TMDs and therefore are routinely palpated. They originate at the posterior occipital area and extend inferiorly along the cervicospinal region.

In palpating these muscles, the examiner's fingers slip behind the patient's head. Those of the right hand palpate the right occipital area, and those of the left hand palpate the left at the origins of the muscles. The patient is questioned regarding any discomfort. The fingers move down the length of the neck muscles through the cervical area and any patient discomfort is recorded.

Functional Manipulation

Three muscles that are basic to jaw movement but impossible or nearly impossible to palpate are the inferior lateral pterygoid, superior lateral pterygoid, and medial pterygoid.

The inferior and superior lateral pterygoids reside deep within the skull, originating on the lateral wing of the sphenoid bone and the maxillary tuberosity and inserting on the neck of the mandibular condyle and the TMJ capsule.

The medial pterygoid has a similar origin, but it extends downward and laterally to insert on the medial surface of the angle of the mandible. Although the medial pterygoid can be palpated by placing the finger in the lateral aspect of the pharyngeal wall of the throat, this palpation is difficult and sometimes uncomfortable for the patient.

All three muscles receive their innervation from the mandibular branch of the trigeminal (V) nerve. During functional manipulation each muscle is contracted and then stretched. If the muscle is a true source of pain, both activities will increase the pain.

Action	**Medial Pterygoid Muscle**	**Inferior Lateral Pterygoid Muscle**	**Superior Lateral Pterygoid Muscle**	**Intracapsular Disorder**
Opening widely	Pain ↑	Pain ↑ slightly	No pain	Pain ↑
Protruding against resistance	Pain ↑ slightly	Pain ↑	No pain	Pain ↑
Clenching on teeth	Pain ↑	Pain ↑	Pain ↑	Pain ↑
Clenching on separator (unilaterally)	Pain ↑		Pain ↑	No pain
Protruding against resistance with unilateral separator	Pain ↑ slightly	Pain ↑	Pain ↑ slightly (if clenching on unilateral separator)	No pain

TEMPOROMANDIBULAR JOINT EXAMINATION

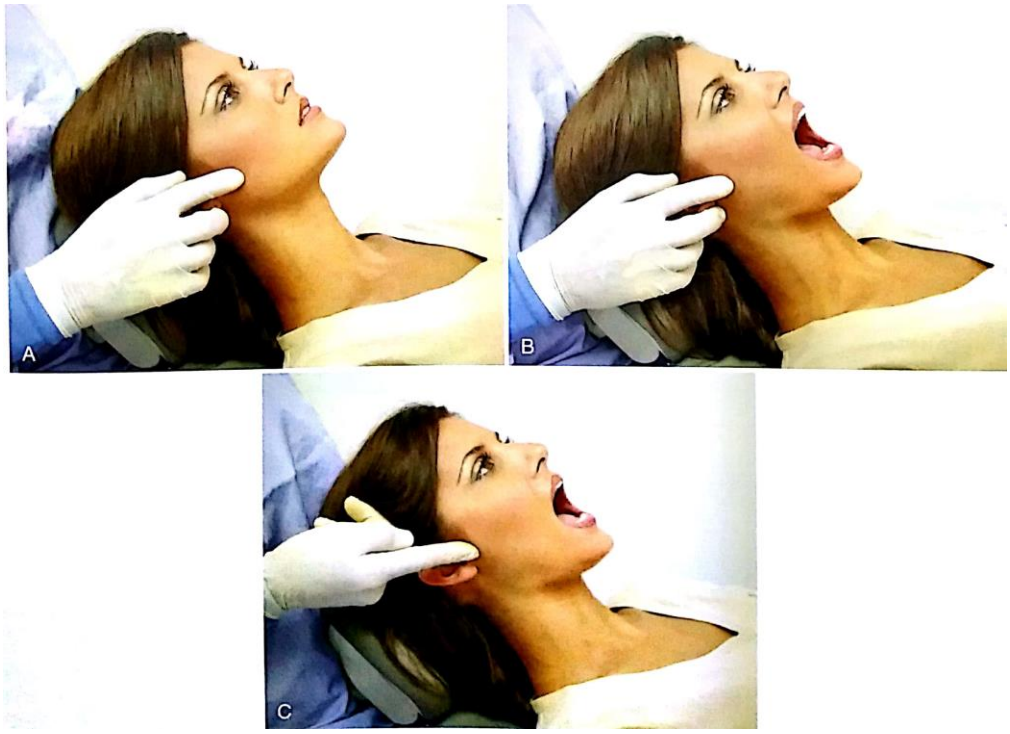
The TMJs are examined for any signs or symptoms associated with pain and dysfunction. Radiographs and other imaging techniques can also be useful

Temporomandibular Joint Pain

Pain or tenderness of the TMJs is determined by digital palpation of the joints when the mandible is both stationary and during dynamic movement. The fingertips are placed over the lateral aspects of both joint areas simultaneous. The fingertips should feel the lateral poles of the condyles passing downward and forward across the articular eminences. Once the position of the fingers over the joints has been verified, the patient relaxes and medial force is applied to the joint areas. Once the symptoms are recorded in a static position, the patient opens and closes, and any symptoms associated with this movement are recorded. As the patient opens maximally, the fingers should be rotated slightly posteriorly to apply force to the posterior aspect of the condyle. Posterior capsulitis and retrodiscitis are clinically evaluated in this manner.

When the fingers are placed properly over the lateral poles of the condyles and the patient is asked to clench, little to no movement is felt. However, if the fingers are misplaced only 1 cm anterior to the lateral pole and the patient is asked to clench, the deep portion of the masseter can be felt contracting.

The clinician must also be aware that a portion of the parotid gland extends to the region of the joint and parotid symptoms can arise from this area. The examiner must be astute in identifying whether the symptoms are originating from joint, muscle, or gland.



A, Lateral aspect of the joint with the mouth closed.

B, Lateral aspect of the joint during opening and closing.

C, With the mouth fully open, the finger is moved behind the condyle to palpate the posterior aspect of the joint.

Temporomandibular Joint Dysfunction

Dysfunction of the TMJs can be separated into two types: joint sounds and joint restrictions.

Joint Sounds

Joint sounds are either clicks or crepitation. A click is a single sound of short duration. If it is relatively loud, it is sometimes referred to as a pop. Crepitation is a multiple gravel-like sound described as grating and complicated. Crepitation is most commonly associated with osteoarthritic changes of the articular surfaces of the joint. Joint sounds can be perceived by placing the fingertips over the lateral surfaces of the joint and having the patient open and close. Often they may be felt by the fingertips.

A more careful examination can be performed by placing a stethoscope over the joint area. Not only should the character of any joint sounds be recorded (e.g., clicking, crepitation), but also the degree of mandibular opening (i.e., interincisal distance) associated with the sound. Of equal importance is whether the sound occurs during opening or closing or can be heard (or felt) during both these movements.

The dental examination begins with inspection of the teeth and their supportive structures for any indications of breakdown. Common signs and symptoms are tooth mobility, pulpitis, and tooth wear.

Joint restrictions

The dynamic movements of the mandible are observed for ant irregularities or restrictions.

Locating the centric relation position

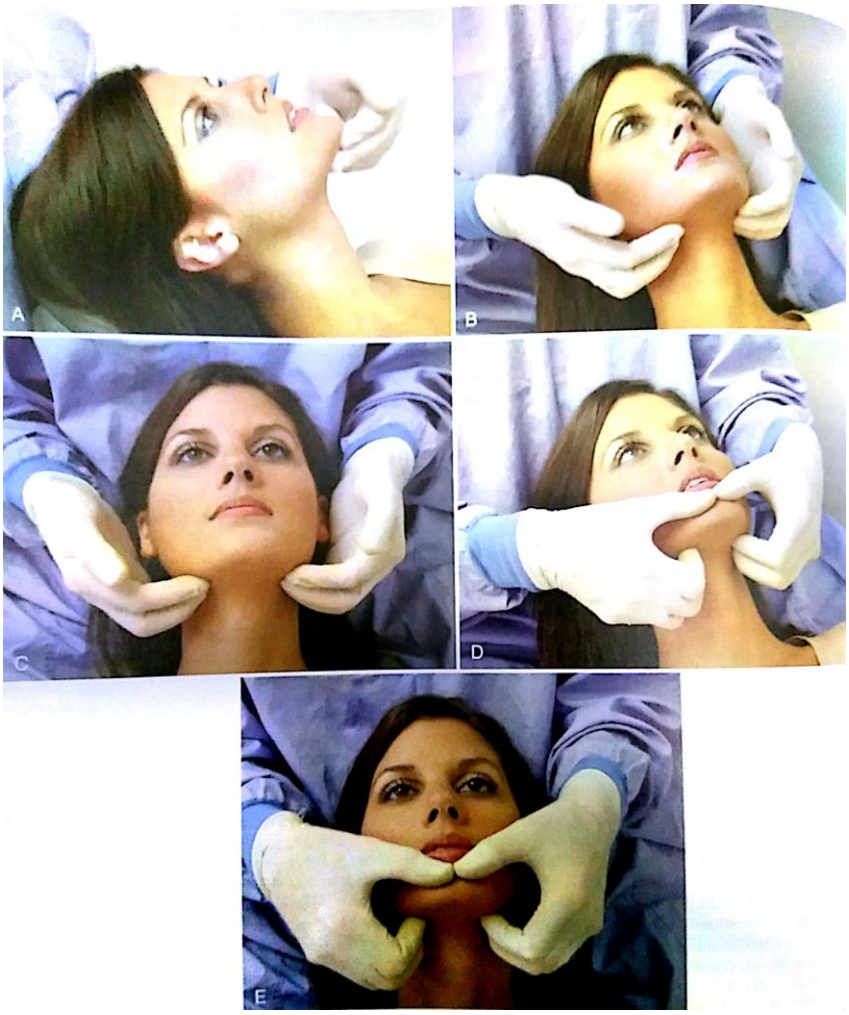
Locating the CR position can sometimes be difficult. To guide the mandible into this position, one must first understand that the neuromuscular control system governs all movement. Therefore care must be taken in positioning the mandible to assure the patient's neuromuscular system that damage will not occur. In attempting to locate CR, it is important that the patient be relaxed. This can be aided by having the patient recline comfortably in the dental chair.

Dawson [99] has described an effective technique for guiding the mandible into CR. It begins with the patient lying back with the chin pointed upward. Lifting the chin upward places the head in an easier position to locate the condyles near the CR position. The dentist sits behind the patient, and the four fingers of each hand are placed on the lower border of the mandible with the smallest finger behind the angle of the mandible.

Next, both thumbs are placed over the symphysis of the chin so that they touch each other between the chin and the lower lip. When the hands are in this position, the mandible is guided by upward force placed on its lower border and angle with the fingers, while at the same time the thumbs press downward and backward on the chin.

The overall force on the mandible is directed so that the condyles will be seated in their most superoanterior position braced against the posterior slopes of the eminences.

When such a relationship exists, guiding the mandible to CR creates no pain. If pain is produced, it is likely that an intracapsular disorder exists. TMJ symptoms during bilateral manual manipulation are likely the results of loading the retrodiscal tissues secondary to a functionally displaced or dislocated disc. Inflammatory disorders of the TMJ can also elicit discomfort when, in guiding the mandible, force is applied to inflamed structures. If either of these conditions exists, an accurate reproducible CR position will not likely be achieved.



A, Successfully guiding the mandible into centric relation begins with having the patient recline and directing the chin upward.

B and **C,** The four fingers of each hand are placed along the lower border of the mandible with the small finger behind the mandibular angle. They should be positioned on the bone and not in the soft tissues of the neck.

D and **E,** The thumbs meet over the symphysis of the chin.

DIAGNOSIS

To manage masticatory disorders effectively, one must understand the numerous types of problems that can exist and the variety of etiologies that cause them. Separating these disorders into common groups of symptoms and etiologies is a process called diagnosis. The clinician must keep in mind that for each diagnosis there is an appropriate treatment. No single treatment is appropriate for all temporomandibular disorders (TMDs). Therefore making a proper diagnosis becomes an extremely important part of managing the patient disorder.

In many instances the success of the therapy depends not on how well the treatment is performed but instead on how appropriate the therapy is for the disorder. In other words, proper diagnosis is the key to successful treatment.

A diagnosis is achieved by careful evaluation of information derived through the history and examination procedures. This information should lead to the identification of a specific disorder. If a person has a single disorder, diagnosis becomes a relatively routine procedure. The clinician should remember, however, that there are no rules limiting an individual to just one disorder at a time. In fact, many persons who have suffered for more than several months are likely to present with more than one disorder. It is the clinician's responsibility to identify each disorder and then (when possible) prioritize them in order of their significance.

Suppose a person falls and sustains a blow to the chin and jaw. A disc derangement disorder can develop. After several days of joint pain, the muscles become secondarily involved as a mechanism of restricting jaw movement (protective co-contraction). When the person reports to the office, both joint pain (i.e., pain in the intracapsular tissues) and muscle pain are present. The information received during the history and examination should assist in determining that the patient has a primary problem with the joint and a secondary problem with the muscles.

Once proper treatment is extended to the joint, the joint symptoms will resolve and so will the secondary muscle pain. If in this instance the muscle pain is treated but not the joint pain, the treatment will likely fail because the primary disorder has not been managed.

The same problem can occur, in reverse, with a masticatory muscle disorder that increases the clinical symptom of clicking in the joint. The person reports with muscle pain and joint clicking. If the clicking alone is treated, the painful muscle will remain. Treatment should be directed toward the primary, not secondary, diagnosis. The history and examination must assist the clinician in determining this order. The clinician should also realize that the person may be suffering from both a muscle disorder and a joint disorder that are unrelated to each other. Generally, in this instance, the chief complaint should be addressed first.

DIAGNOSING PAIN DISORDERS

In disorders that have pain as a primary symptom, it is imperative that the source of the pain be identified.

If it is primary pain, this will not be difficult because the source and the site are in the same location. With primary pain the patient is pointing directly to the source of pain. However, if the pain is heterotopic, the patient will be directing attention to the site of pain, which may be quite remote from the actual source of the pain. Remember, treatment is effective only if it is directed at the source, not at the site of pain.

One key in locating the source of pain is that local provocation should accentuate it. Although this rule does not always apply, one should be suspicious that when local provocation does not increase the pain it may be heterotopic. In other words, if a patient is complaining of temporo-mandibular joint (TMJ) area pain, he or she should also complain that it hurts to open and chew (local provocation). If the patient does not report any functional problem with jaw movement, the TMJ may be merely a site of pain and not pathologically involved. It is the clinician's job in this instance to continue to examine the patient for the source of the pain.

When pain symptoms become complex, it is sometimes necessary to use selective local anesthetic blockade of tissues to help differentiate the site from the source. Anesthetic blocking should be a routine diagnostic procedure for the clinician.

Local anesthetic blockade of the source of pain will at least temporarily eliminate the symptoms because it blocks the nociceptive input originating from the true source of the pain. Local anesthetic blockade of the site will have no effect because there is no nociceptive input coming from that site.

Heterotopic pains are the result of the central excitatory effect in the brainstem produced by a distance source of nociception.

An example is a patient whose chief complaint is a tension-type headache in the temporal region. The clinician may discover that it is produced by central excitatory effects from a trigger point in the trapezius. The patient points to the temple as the chief complaint, but palpation of this area (local provocation) does not accentuate the headache. Local anesthetic blockade of the temporal muscle fails to reduce the pain because this is a site and not the source. As the clinician continues the examination, an active trigger point is found in the trapezius. Local provocation of the trigger point not only increases the pain felt in the trapezius but also increases the temporal headache (site of referred pain). Local anesthetic blockade of the trapezius trigger point eliminates not only the trigger point pain but also the temporal headache (the referred pain is wholly dependent on the source of pain). The clinician has now identified the source of the headache and thus made a diagnosis. Diagnostic blocking of a muscle trigger point may be extremely helpful when myofascial trigger point pain is suspected.

Compared with referred pain, secondary hyper-algesia responds differently to local anesthetic blockade. When the original source of pain is blocked, referred pain resolves immediately but secondary hyperalgesia may remain for hours. Therefore the effect of a local anesthetic injection on secondary hyperalgesia should not be evaluated until the following day.

The following four rules summarize the examination techniques used to differentiate primary pain from referred pain [100]

1. Local provocation of the site of pain does not increase the pain.

2. Local provocation at the source of pain increases the pain not only at the source but also increases the pain at the site.

3. Local anesthetic blocking of the site of pain does not decrease the pain.

4. Local anesthetic blocking of the source of the pain decreases the pain at the source, as well as the site.

DIAGNOSTIC ANALGESIC BLOCKING

Indications for analgesic blocking

The value of local anesthetic injections and application of topical anesthetics to identify and localize pain cannot be overemphasized. It is essential when differentiating primary from secondary pains. It is equally useful to identify the pathways that mediate peripheral pain and to localize pain sources. Often when the source of pain is difficult to identify, local anesthetic blocking of related tissues is the key to making the proper diagnosis.

The examiner should therefore become skilled in the use of this valuable diagnostic tool. Muscle injections can also be useful for diagnostic purposes, as well as for therapy. Local anesthetic blocking not only provides valuable diagnostic information but in some pain disorders can also provide therapeutic value. This is especially true for myofascial pain and myospasm.

Another indication for analgesic blocking is to help educate the patient to the source of his or her pain problem. Often patients do not appreciate the concept of pain referral, and it can be quite convincing to the patient when blocking a remote site reduces or even eliminates the chief complaint. This can be a valuable educational tool.

Armamentarium

The armamentarium needed to provide anesthetic blocks is already present in most dental offices [101]. It begins with an aspirating syringe and both short and long 27-gauge needles. The length needed depends on the structure that is targeted. Alcohol and/or Betadine swipes are also required to clean the site to be injected. Sterile 2 × 2 gauzes should also be available to apply to the injection site to control bleeding. Clean disposable gloves are also necessary.

The type of local anesthetic used may vary according to the type and purpose of the particular injection. When only diagnostic information is necessary, the use of short-acting drugs is most desirable. Usually a solution without a vasoconstricting agent is best. Good anesthesia for skeletal muscle requires a non vasoconstricting solution because of the vasodilating effect of epinephrine like substances on such tissue. This reverse effect on muscle tissue is sometimes forgotten and may account for the transient anesthesia of poor quality sometimes obtained when muscles are injected for diagnostic purposes.

Local anesthetics have been demonstrated to have a measure of myotoxicity. Procaine appears to be the least myotoxic of the local anesthetics in common use. Mild inflammatory reactions follow the injection of 1% and 2% procaine hydrochloride, as well as isotonic sodium chloride. Single injections of either procaine or isotonic saline cause no muscle necrosis.

The longer-acting and stronger anesthetics induce more severe inflammation and occasional coagulation necrosis of muscle tissue. Regeneration takes place in approximately 7 days. Solutions containing epinephrine cause greater muscle damage. To minimize the danger of muscle damage in analgesic blocking for both diagnostic and therapeutic purposes, low concentrations of procaine are advisable and such injections should be spaced at least 7 days apart. Because procaine is not available in dental carpules, the dentist may select 2% lidocaine (Xylocaine) or 3% mepivacaine (Carbocaine) without a vasoconstrictor.

When a longer-acting anesthetic is indicated, 0.5% bupivacaine (Marcaine) may be used. Although bupivacaine is sometimes indicated for joint pain (auriculotemporal nerve block), it should not be routinely used with muscle injections because of its myotoxicity.

General rules to follow

When any injection is to be performed, the clinician should observe the following fundamental rules:

1. The clinician should have a sound knowledge of the anatomy of all structures in the region that is to be injected. The purpose of an injection is to isolate the particular structure that is to be blocked. Therefore the clinician must know the precise location and appropriate technique used to get the needle tip to the desired structure. Equally important is that the clinician should have a sound understanding of all the important structures in the area that should be avoided during the injection.
2. The clinician should have a sound knowledge of the pharmacology of all solutions that will be used.
3. The clinician should avoid injecting into inflamed or diseased tissues.
4. The clinician should maintain strict asepsis at all times.
5. The clinician should always aspirate before injecting solution so as to be sure the needle is not in a blood vessel.

TYPES OF INJECTIONS

Diagnostic and therapeutic anesthetic blocks are divided into three types according to the structures that are targeted [102]: muscle injections, nerve block injections, and intracapsular injections.

Muscle Injections

Injecting a muscle can be valuable in determining the source of a pain disorder. In some instances muscle injections can provide therapeutic value. For example, the injection of local anesthetic into a myofascial trigger point can result in significant pain reduction long after the anesthetic has been metabolized. In myofascial pain the patient presents with a firm, taut band of muscle tissue that is quite painful to palpate. This is known as a trigger point and is often responsible for producing a pattern of pain referral. When this is suspected, the trigger point can be injected with local anesthetic and the resulting pattern of pain referral is shut down.

When it has been determined that injection of the trigger point is indicated, the following sequence should be followed:

• The trigger point is located by placing the finger over the muscle, and firm pressure is applied to locate the tight band. The finger is moved across the band so that it can be felt to "snap" under the pressure of the finger. Once the band is identified, the finger is moved up and down the band until the most painful area is located

• Once the trigger point is located, the tissue over the trigger point is cleaned with alcohol. The trigger point is then trapped between two fingers so that when the needle is placed into the area, the tight band will not move away.

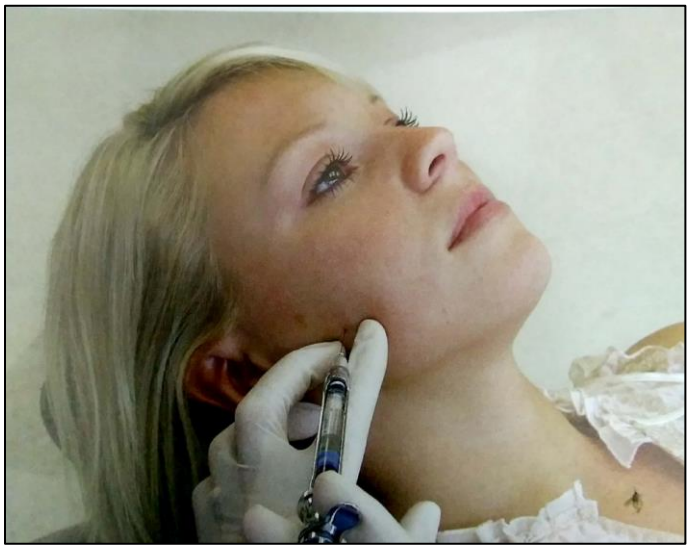
• The needle tip is then inserted into the tissue superficial to the trigger point and is penetrated to the depth of the tight band. Receiving feedback from the patient regarding the accuracy of needle placement is often helpful. Usually the patient can tell immediately when the clinician has entered the trigger point. Once the needle tip is at the proper depth, the syringe is aspirated to ensure it is not in located in a vessel. Then a small amount of anesthetic is deposited in the area (one quarter of a carpule).

• Once the initial anesthetic is deposited, it is useful to "fan" the needle tip slightly. This is done by withdrawing the needle halfway, changing the needle direction slightly, and reentering into the firm band to the same depth. The needle tip should not be completely removed from the tissue. Once the injection is completed, the needle is fully withdrawn

and a sterile gauze is held over the injection site with slight pressure for 20 to 30 seconds to ensure good hemostasis

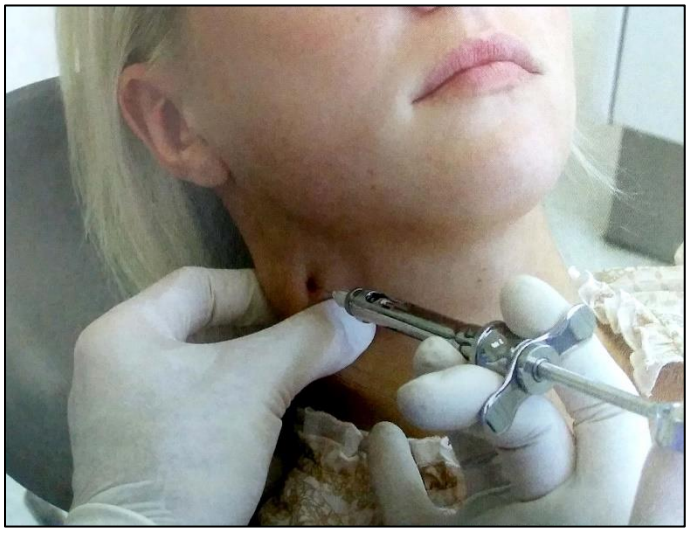
This general technique is used for most muscle injections; however, the unique anatomy of each muscle may demand slight variations. Extremely important is that the clinician is familiar with the anatomy of the muscle to be injected so as to avoid disturbing any neighboring structures.

Some of the muscles that can be easily injected are the masseter, temporalis, sternocleidomastoid, splenius capitis, posterior occipital, and trapezius.

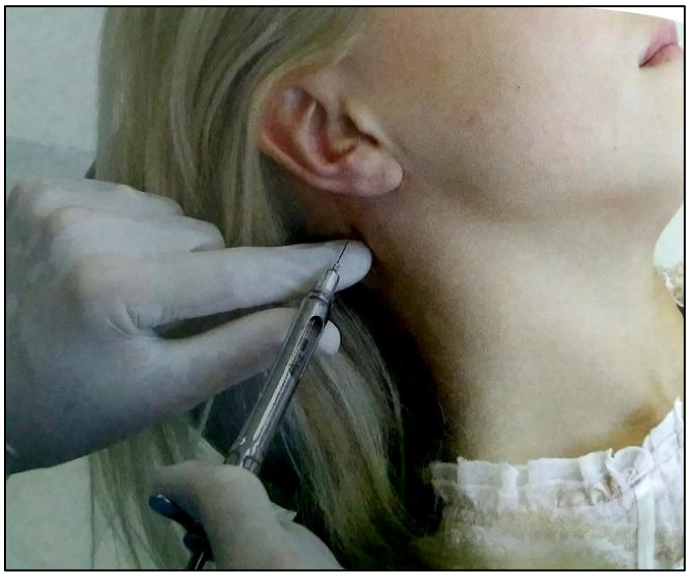


Injection of the masseter muscle.

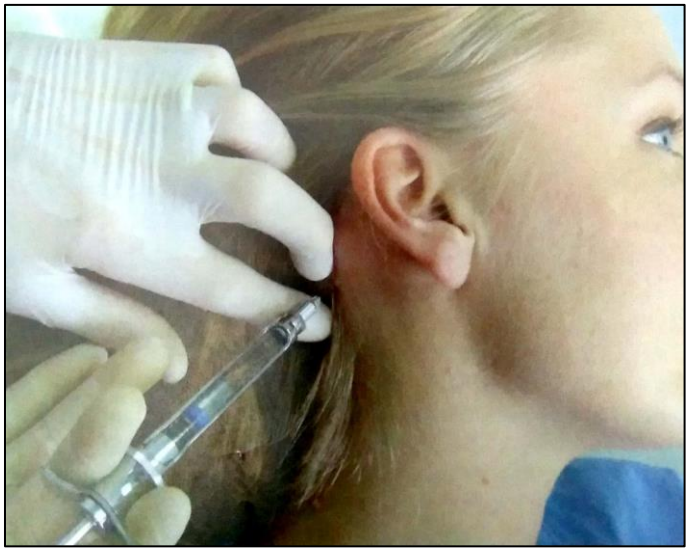
Injection of the temporalis muscle



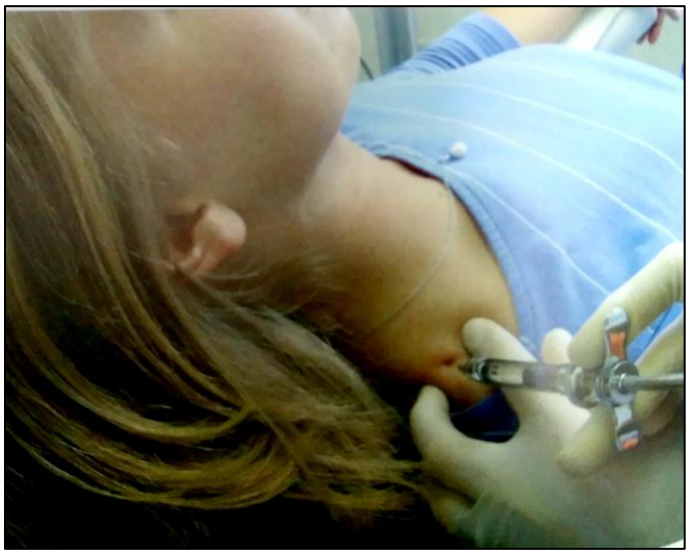
Injection of the sternocleidomastoid muscle from an anterior approach so as to avoid vital deep structures.



Injection of the splenius capitis muscle at its attachment to the skull slightly distal to the mastoid process.



Injection of the posterior occipital muscles at their attachments to the skull



Injection of a common site for a trigger point in the trapezius muscle.

Nerve block injections

Diagnostic nerve blocks can be useful in identifying whether a painful structure is actually a site or source of pain. When diagnosis is the primary purpose for the injection, a short-acting local anesthetic should be used without a vasoconstrictor. In some instances long-term pain relief maybe therapeutically indicated. This may be appropriate for certain chronic pain conditions when prolonged relief of pain can be used to interrupt pain cycling and hopefully reduce central sensitization. When long-term anesthesia is indicated, a long-term local anesthetic such as bupivacaine with a vasoconstrictor may be a better choice.

Dental Blocks

The practicing dentist uses nerve blocks routinely in dental treatments. The clinician should remember that these same injections can provide valuable diagnostic information. The common nerve blocks used are the inferior alveolar nerve block, the posterior superior nerve block, the mental nerve block, and infiltration blocks often administered in various areas of the maxillary arch

An inferior alveolar nerve block will completely eliminate any source of pain coming the mandibular teeth on the side of the injection. This block is useful in separating dental pain from muscle or joint pain because it only blocks the dental structures. This is important diagnostic information, especially when a patient's chief complaint is toothache. If a mandibular toothache is truly of dental origin, an inferior alveolar nerve block will eliminate the pain. If, however, the toothache is actually a referred pain to the tooth, the block will not change the pain.

When attempting to localize a particular tooth as a source of pain, it is important to consider local infiltration of the anesthetic first before a total nerve block (i.e., mandibular block).

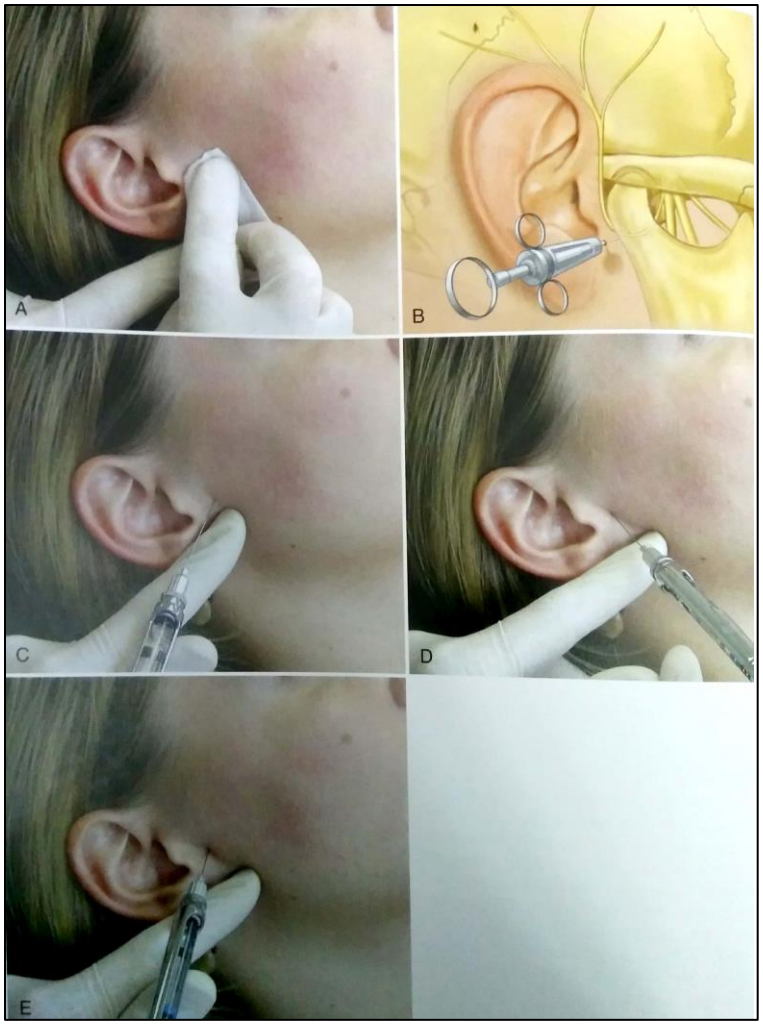
Isolating a single tooth with local anesthetic is far more specific than blocking an entire quadrant of teeth. Once the entire nerve has been blocked, it may be difficult to

identify the specific tooth until the anesthesia has been metabolized. As a general rule for diagnosing tooth pain, begin by using localized areas of anesthesia and moving to broader areas as needed.

Auriculotemporal Nerve Block

This nerve block has significant diagnostic value. The primary innervation of the TMJ is from the auriculotemporal nerve, with secondary innervation coming from the masseteric and posterior deep temporal nerves. Therefore if the TMJ is a source of pain, this nerve block will quickly eliminate the pain.

Some clinicians anesthetize the TMJ by injecting directly into the joint or into the retrodiscal structures. The auriculotemporal nerve can be blocked by first cleaning the tissue and then passing a 27-gauge needle through the skin just anterior to and slightly above the junction of the tragus and the earlobe. The needle is then advanced until it touches the posterior neck of the condyle. The needle is then repositioned in a more posterior direction until the tip of the needle is able to pass behind the posterior neck of the condyle. Once the neck of the condyle is felt, the tip of the needle is carefully moved slightly behind the posterior aspect of the condyle in an anteromedial direction to a depth of 1 cm. The syringe is then aspirated and if no blood is seen, the solution is deposited. If the true source of pain is the joint, the pain should be eliminated or certainly significantly decreased in 4 to 5 minutes.



A, The tissue at the site of the injection is thoroughly cleaned.

B, This drawing shows the position of the auriculotemporal nerve as it trans-verses around the posterior aspect of the condyle.

C, The needle is placed slightly anterior to the junction of the tragus and ear lobule and is penetrated until the posterior neck of the condyle is felt.

D, The needle is then repositioned in a more posterior direction until the tip of the needle is able to pass behind the posterior neck of the condyle.

E, Once the needle tip passes beyond the neck of the condyle, the syringe is inserted behind the neck of the condyle.

Intracapsular Injections

On occasion it is indicated to inject directly into the TMJ. This type of injection would be indicated for therapeutic, not diagnostic, reasons [104] Diagnostic information is derived from performing the auriculotemporal nerve block. A therapeutic injection would be indicated when it is appropriate to introduce some medication to the joint structures.

Normally the superior joint space is the target for an intracapsular (intraarticular) injection because it is the largest joint space and is the simplest to consistently locate. The joint can be entered by first locating the lateral pole of the condyle. This can be assisted by asking the patient to open and close the mouth. Once the pole is located, the clinician should ask the patient to open slightly and palpate directly above to locate the zygomatic arch.

The tissue is cleaned, and the tip of the needle is placed just below the zygomatic arch and slightly behind the posterior and superior aspect of the condyle. The needle is angulated slightly anterior superiorly to avoid the retrodiscal tissues. Once the capsule is penetrated, the tip of the needle will be in the superior joint space. The solution is then deposited and the needle removed. A sterile gauze is held over the injection site for a few seconds to ensure hemostasis. The patient is then asked to open and close the mouth a few times to distributed the solution throughout the joint space.

Often a successful intraarticular injection will leave the patient with an immediate acute malocclusion on the ipsilateral side. Because there is little area in the superior joint space, the introduction of additional fluid will temporally cause an increase in the joint space that leads to the separation of the posterior teeth on the same side of the injection. This will resolve in a few hours.

Infraorbital Nerve Block

The infraorbital nerve transverses below the eye to exit from the infraorbital foramen located in the inferior border of the orbit. This nerve goes on to innervate the facial structures below the eye and some of the lateral aspects of the nose. In cases of trauma to

the face this nerve can be injured, resulting in a continuous neuropathic pain. Blocking this nerve may have some therapeutic value. The nerve can be blocked by either an extraoral or intraoral approach.

When the extraoral approached is used the foramen is identified by palpation of the inferior border of the orbit, feeling for a slight notch. The notch represents the exit of the infraorbital nerve. Once the notch is located, the tissue is cleaned and the needle is place to the depth of the notch and into the foreman when possible. When the intraoral approach is used, the notch is found in the same manner as previously described. The middle finger is used to maintain the position of the notch, while the index finger and thumb are used to retract the lip. The needle is placed into the mouth, and the tip is inserted into the vestibule and directed upward to the notch.

Keys in making a differential diagnosis

Although muscle and joint disorders have some common clinical findings, seven areas of information acquired during the history and examination will assist in separating them. These keys in diagnosis are the following [104]: (1) history, (2) mandibular restriction, (3) mandibular interference, (4) acute mal occlusion, (5) loading of the joint, (6) functional manipulation, and (7) diagnostic anesthetic blockade.

1. History.

The history is always helpful in distinguishing joint from muscle disorders. When a joint is traumatized, the symptoms are likely to begin in association with the trauma and be relatively constant or worsen from that time forward. Muscle disorders, on the other hand, appear to fluctuate and cycle from severe to mild with no apparent initiating event. Muscle problems are more closely related to changes in levels of emotional stress, and therefore periods of total remission are not uncommon when stress is low.

2. Mandibular restriction.

Restriction of mouth opening and eccentric movements are common findings with both joint disorders and muscle disorders. The character of the restrictions, however, can be quite different. Restriction in mouth opening because of intracapsular problems (e.g., a dislocated disc without reduction) usually occurs at 25 to 30 mm.

At that point the mouth cannot be opened wider, even with mild passive force. This hard "end feel" is commonly associated with a dislocated disc blocking translation of the condyle.

Restricted mouth opening as a result of muscle disorders can occur anywhere during the opening movement. For example, a restricted opening of 8 to 10 mm is most certainly of muscle origin. When the mouth opening is restricted by muscles, mild passive force will usually lengthen the muscles slightly and result in a small increase in opening. This represents a soft "end feel" and is typical of muscle restrictions. Combining these clinical findings with the onset of the limited mouth opening obtained in the history is helpful in understanding the reason for the restriction. Mandibular restriction should also be evaluated by observing the patient move in left and right eccentric positions. In patients with an intracapsular restriction (i.e., disc dislocation without reduction), a contralateral eccentric movement will be limited but an ipsilateral movement will be normal.

However, with muscle disorders the elevators (temporalis, masseter, medial Pterygoid) are responsible for the limited mouth opening and, because eccentric movements do not generally lengthen these muscles, a normal range of eccentric movement exists.

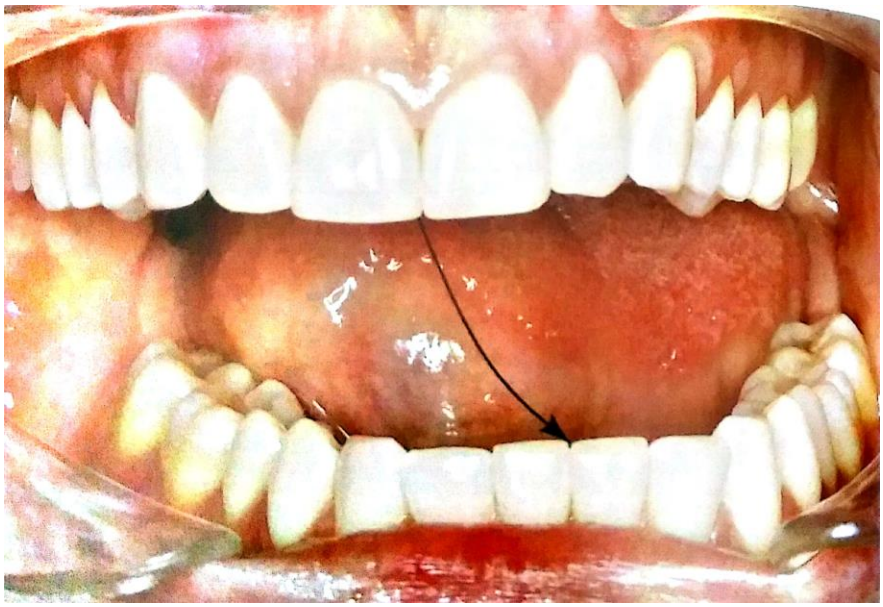
3. Mandibular interference

When the mouth is opened, the pathway of the mandible is observed for any deviations or deflections. If the deviation occurs during opening and the jaw then returns to midline before 30 to 35 mm of total opening, it is likely to be associated with a disc derangement disorder.

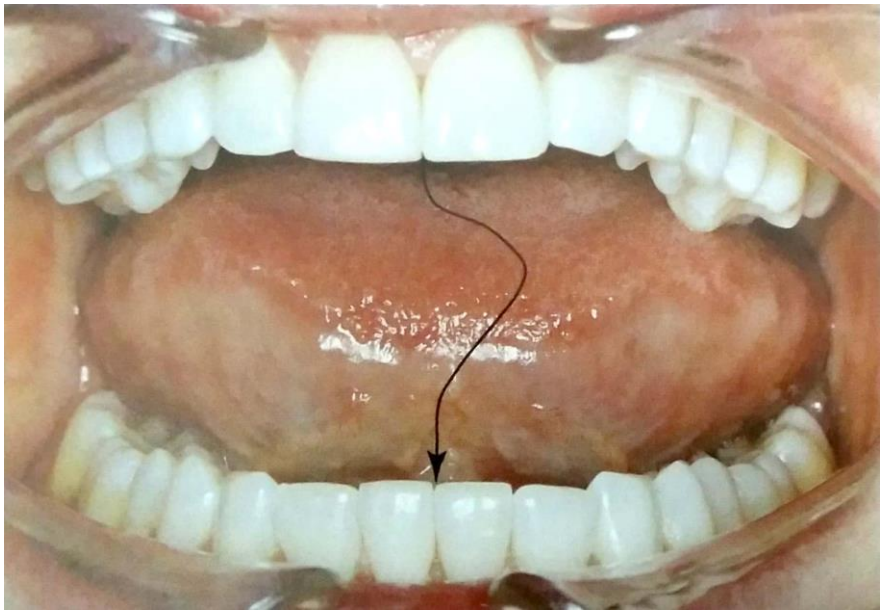
If the speed of opening alters the location of the deviation, it is likely to be discal movement such as disc displacement with reduction. If the speed of opening does not alter the interincisal distance of the deviation and if the location of the deviation is the same for opening and closing, then a structural incompatibility is a likely diagnosis. Muscle disorders that cause deviation of mandibular opening pathways are commonly large, inconsistent, sweeping movements not associated with joint sounds. These deviations are a result of muscle engrams. Deviation can also occur because of subluxation at the wide-open position. This is an intracapsular disorder, but not necessarily a pathologic condition

Deflection of the mandibular opening pathway results when one condyle does not translate. This may be caused by an intracapsular problem such as a disc dislocation without reduction or an adhesion problem. With these problems the mandible will deflect to the ipsilateral side during the late stages of opening. Deflection during opening can also result if a unilateral elevator muscle, such as the masseter, becomes shortened (myospasm). This condition can be separated from intracapsular disorders by observing the protrusive and lateral eccentric movements. If the problem is intracapsular, the mandible will deflect to the side of the involved joint during protrusion and be restricted during a contralateral movement (normal movement to the ipsilateral side). If the problem is extracapsular (i.e., muscle), there will be no deflection during the protrusive movement and no restrictions in lateral movements.

When deflection of the mandible is caused by an intracapsular source, the mandible will always move toward the involved joint. If the deflection is the result of a shortened muscle, the direction in which the mandible moves will depend on the position of the involved muscle with respect to the joint. If the muscle is lateral to the joint (i.e., masseter or temporalis), the deflection will be toward the involved muscle. If it is medial to the joint (i.e., medialpterygoid), deflection will be away from the involved muscle (in a contralateral direction).



Deflection



Deviation

4. Acute malocclusion

An acute malocclusion is a sudden alteration of the occlusion condition secondary to a disorder. An acute malocclusion caused by a muscle disorder will vary according to the muscles involved. If the inferior lateral pterygoid is in spasm and shortens, the condyle will be brought slightly forward in the fossa on the involved side. This will result in a disocclusion of the ipsilateral posterior teeth and heavy contact on the contralateral canines. If the spasms are in the elevator muscles, the patient is likely to report a feeling that the teeth "suddenly don't fit right," yet clinically it may be difficult to visualize any change. An acute malocclusion resulting from an intracapsular disorder is usually closely related to the event that changed the joint function.

If the disc is suddenly displaced, the thicker posterior border may be superimposed between the condyle and fossa and cause a sudden increase in the discal space. This appears clinically as a loss of ipsilateral posterior tooth contact.

5. Loading the joint

Positioning the condyles to their musculoskeletally stable position and loading the structures with manipulative force does not produce pain in a healthy joint. When pain is produced, one should be suspicious of an intracapsular source of pain

6. Functional manipulation

Functional manipulation can be helpful in identifying the location of pain. Functional manipulation procedures that do not produce pain tend to rule out muscle disorders as the source of the problem.

7. Diagnostic anesthetic blockade

For patients in whom the preceding six procedures have not convincingly assisted in making a differential diagnosis between joint and muscle disorders, anesthetic blockade is indicated. Anesthetic blocking of the auriculotemporal nerve can quickly rule in or out an intracapsular disorder.

MASTICATORY MUSCLE DISORDERS

All masticatory muscle disorders are not clinically the same. At least five different types are known, and being able to distinguish among them is important because the treatment of each is quite different.

The five types are [105] (1) Protective co-contraction (muscle splinting), (2) Local muscle soreness, (3) Myospasm, (4) Myofascial (trigger point) pain, and (5) Centrally mediated myalgia.

(1)Protective Co-Contraction (Muscle Splinting)

Protective co-contraction is a central nervous system (CNS) response to injury or threat of injury. In the past this response was referred to as muscle splinting. In the presence of an event, the activity of appropriate muscles seems to be altered so as to protect the injured part from further injury. All muscles are maintained in a mildly contracted state known as tonus. Tonus persists without fatigue by virtue of the alternating contractions and relaxations of the muscle fibers, which keep the overall muscle length unchanged and resist any sudden elongation.

Causes

Three conditions can lead to protective co-contraction:

1. Altered sensory or proprioceptive input. Protective co-contraction may be initiated by any change in the occlusal condition that significantly alters sensory input, such as the introduction of a poorly fitting crown. If a crown is placed with a high occlusal contact, it tends to alter the sensory and proprioceptive input to the CNS. Consequently, the elevator muscles (temporalis, masseter, medial pterygoid) may become protectively co-contracted in an attempt to prevent the crown from contacting the opposing tooth.

2. Constant deep pain input. As already discussed, the presence of deep pain input felt in local structures can produce protective co-contraction of associated muscle.

It is important to note that the source of the deep pain need not be muscle tissue itself but can be any associated structure such as tendons, ligaments, joints, or even the teeth.

3. Increased emotional stress. Clinical observations strongly demonstrate that emotional stress can greatly influence masticatory muscle activity. When an individual experiences increased levels of emotional stress, a common response is for the gamma efferent system to alter the sensitivity of the muscle spindle. This increases the sensitivity of the muscle to lengthening, resulting in an increased tonicity of the muscle. The clinical response of the muscle is seen as protective co-contraction. Increased emotional stress also has the ability to initiate parafunctional activities such as nocturnal bruxism and clenching.

(2)Local Muscle Soreness (Noninflammatory Myalgia)

Local muscle soreness is a primary, noninflammatory, myogenous pain disorder and is often the first response of the muscle tissue to continued protective co-contraction. Whereas co-contraction represents a CNS-induced muscle response, local muscle soreness represents a change in the local environment of the muscle tissues. This change may be the result of prolonged co-contraction or excessive use of the muscle producing fatigue. When

unaccustomed use is the cause, the symptoms may be delayed (delayed-onset muscle soreness).

Local muscle soreness may also result from direct tissue damage (trauma).

Causes

Four principal conditions lead to local muscle soreness:

1. Protracted co-contraction. Continued co-contraction will lead to local muscle soreness. Because this muscle soreness itself is a source of deep pain, an important clinical event can occur. Deep pain produced by local muscle soreness can in fact produce protective co-contraction.

2. Trauma. A muscle can sustain at least two types of trauma:

    a. Local tissue injury: local injury of tissue can occur through events such as local anesthetic injections or tissue strains.

    b. Unaccustomed use: Trauma to muscle tissue can be created by abusive or unaccustomed use of muscle tissues. This may result from bruxing or clenching the teeth or even from unaccustomed chewing of gum.

3. Increased emotional stress. Continued increased levels of emotional stress can lead to prolonged co-contraction and muscle pain.

4. Idiopathic myogenous pain.

Clinical features:

1. Structural dysfunction. When masticatory muscles experience local muscle soreness, there is a decrease in the velocity and range of mandibular movement. This alteration is secondary to the inhibitory effect of pain (protective co-contraction). Unlike co-

contraction, however, slow and careful mouth opening still reveals limited range of movement. Passive stretching by the examiner can often achieve a more normal range.

2. Minimum pain at rest. Local muscle soreness does not generally produce pain when the muscle is at rest.

3. Increased pain to function. Individuals experiencing local muscle soreness report an increase in pain when the involved muscle functions.

4. Actual muscle weakness. Local muscle soreness results in an overall reduction in the strength of the affected muscles.

5. Local muscle tenderness Muscles experiencing local muscle soreness reveal increased tenderness and pain to palpation. Generally the entire body of the involved muscle is tender to palpation.

(3)Myospasm (Tonic Contraction Myalgia)

Myospasm is an involuntary, CNS-induced tonic muscle contraction

Clinical Characteristics

Individuals experiencing myospasms present with the following clinical characteristics:

1. Structural dysfunction. Two clinical findings noted regarding structural dysfunction are as follows:

a. There is marked restriction in the range of movement determined by the muscle or muscles in spasm. For example, if an elevator muscle such as the masseter were in spasm, there would be marked restriction in mouth opening.

b. Structural dysfunction may also present as an acute malocclusion. An acute malocclusion is a sudden change in the occlusal contact pattern of the teeth secondary to a disorder. This may occur as a result of a myospasm in the inferior lateral pterygoid muscle. A spasm and subsequent shortening of the left lateral pterygoid muscle will produce a shifting of the mandible into a right lateral eccentric position. This will result in heavy occlusal contact of the right anterior teeth and loss of occlusal contact between the left posterior teeth.

2. Pain at rest. Myospasms usually produce significant pain when the mandible is at rest.

3. Increased pain with function. When a patient attempts to function with a muscle in spasm, the pain will be increased.

4. Local muscle tenderness. Palpation of the muscle or muscles experiencing myospasm reveals significant tenderness.

5. Muscle tightness. The patient reports a sudden tightening or knotting up of the entire muscle. Palpation of the muscle or muscles experiencing myospasm reveals them to be firm and hard.

(4)Myofascial Pain (Trigger Point Myalgia)

Myofascial pain is a regional myogenous pain condition characterized by local areas of firm, hypersensitive bands of muscle tissue known as trigger points. This condition is sometimes referred to as myofascial trigger point pain.

Myofascial pain may occur periodically for some patients and therefore represent an acute myalgic disorder. However, myofascial pain may also be associated with otherongoing pain disorders, thereby becoming a chronic pain condition demanding more therapeutic efforts for resolution. The clinician needs to learn by the history whether the condition is acute or chronic so that proper management will be instituted.

Travell and Rinzler [106] first described myofascial trigger point pain in 1952.

In 1969 Laskin [107] published an important paper expressing to the dental community that there are many patients with muscle pain complaints in which the cause is not the occlusal condition. He emphasized the importance of emotional stress and other factors. From this article the dental profession began using the term myofascial pain dysfunction (MPD) syndrome.

Clinical characteristics

An individual suffering with myofascial pain will commonly reveal the following clinical characteristics:

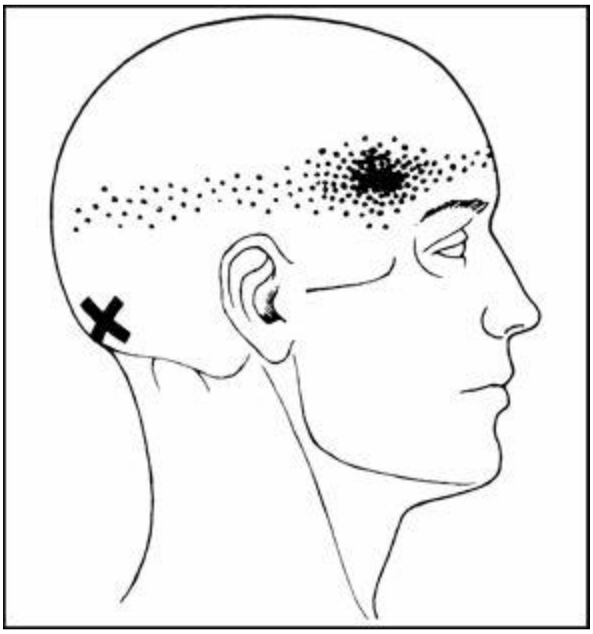
1. Structural dysfunction. Muscles experiencing myofascial pain reveal a decrease in the velocity and range of movement secondary to the inhibitory effect of pain (protective co-contraction). This decreased range of movement is often less than that observed with local muscle soreness.

2. Pain at rest. Patients experiencing myofascial pain report pain even when the muscles are at rest. The pain, however, is not commonly related to the location of the trigger points, but instead represents referred pain.

3. Increased pain with function. Although pain is increased with function of the involved muscles, the amount of pain reported is usually less than with local muscle soreness. The pain is only increased when the trigger point area is provoked by function.

4. Presence of trigger points. Palpation of the muscle reveals local areas of firm, hypersensitive bands of muscle tissue called trigger points. Although palpation of trigger points produces pain, local muscle sensitivity is not the most common complaint of patients suffering from myofascial trigger point pain. The most common complaints center around the central excitatory effects created by the trigger points. In many instances patients may be aware only of the referred pain and not even acknowledge the trigger points. A perfect example is the patient suffering from trigger point pain in the semispinalis capitis in the posterior occipital region of the neck. Trigger points in this region commonly refer pain to

the anterior temple region just above the eye. The patient's chief complaint is temporal headache, with little acknowledgment of the trigger point in the posterior cervical region.



A trigger point (X) in the semispinalis capitis muscle will refer pain to the anterior temporal region slightly above the eye.

(5)Centrally Mediated Myalgia (Chronic Myositis)

Centrally mediated myalgia is a chronic, continuous muscle pain disorder originating predominantly from CNS effects that are felt peripherally in the muscle tissues. This disorder clinically presents with symptoms similar to an inflammatory condition of the muscle tissue and therefore is sometimes referred to as myositis. This condition, however, is not characterized by the classic clinical signs associated of inflammation (e.g., reddening, swelling). Chronic centrally mediated myalgia results from a source of

nociception found in the muscle tissue that has its origin in the CNS (neurogenic inflammation).

Causes

The pain associated with centrally mediated myalgia has its cause more in the CNS than in the muscle tissue itself. As the CNS becomes more involved, antidromic neural impulses are sent out to the muscular and vascular tissues, producing local neurogenic inflammation.

This neurogenic inflammation produces pain in these tissues even though the main cause is the CNS, hence the term centrally mediated myalgia.

History

Two significant features present in the history of a patient with centrally mediated myalgia. The first is the duration of the pain problem. The patient will report a long history of myogenous pain. Typically, the pain will have been present for at least 4 weeks and often several months.

The second feature of centrally mediated myalgia is the constancy of the pain. Pains that last for months or even years but come and go with periods of total remission are not characteristic of centrally mediated myalgia. Patients will commonly report that even if the jaw is at rest, the pain is present. This reflects an inflammatory condition of the tissue.

Clinical characteristics

The following six clinical characteristics are common with centrally mediated myalgia:

1. Structural dysfunction. Patients experiencing centrally mediated myalgia present with a significant decrease in the velocity and range of mandibular movement. This decreased range is secondary to the inhibitory effect of pain (normal range cannot be achieved).

The neurogenic inflammation associated with centrally mediated myalgia may lead to a "sterile" inflammatory response of the muscle tissue, which will further reduce range of mandibular movement.

2. Pain at rest. Pain during rest is a key clinical characteristic of centrally mediated myalgia and is likely caused by the sensitization of muscle nociceptors

3. Increased pain with function. Function of the affected muscles greatly increases the patient's pain.

4. Local muscle tenderness. Muscle tissues are painful when palpated.

5. Feeling of muscle tightness. Patients suffering with centrally mediated myalgia will commonly complain of a feeling of muscle tightness.

6. Muscle contracture. Prolonged centrally mediated myalgia can lead to a muscle condition known as contracture. Contracture is common with centrally mediated myalgia because in order for patients to reduce their pain, they will limit their mouth opening.

TEMPOROMANDIBULAR JOINT DISORDERS

TMJ disorders can be subdivided into three major categories [108]: derangements of the condyle-disc complex, structural incompatibilities of the articular surfaces, and inflammatory disorders of the joint

DERANGEMENTS OF THE CONDYLE-DISC COMPLEX

Causes

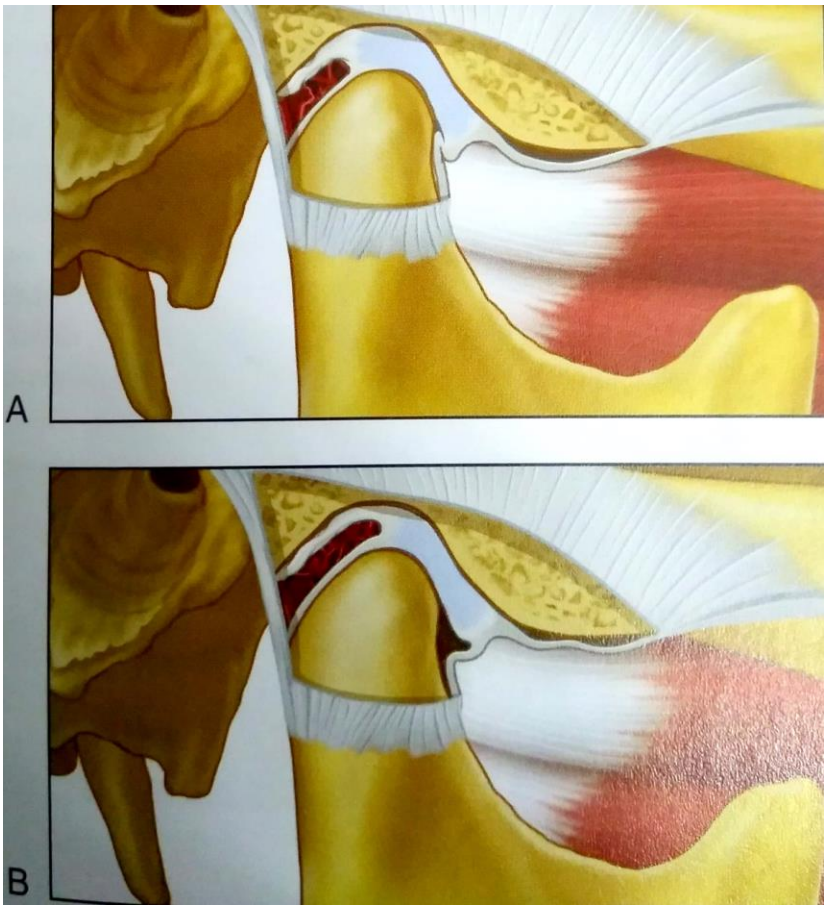
Derangements of the condyle-disc complex arise from breakdown of the normal rotational function of the disc on the condyle. The most common etiologic factor associated with breakdown of the condyle-disc complex is trauma. This may be macrotrauma such as a blow to the jaw (open-mouth macrotrauma is usually seen with elongation of the ligaments) or microtrauma as associated with chronic muscle hyperactivity and orthopedic instability.

The three types of derangements of the condyle-disc complex are **disc displacement, disc dislocation with reduction,** and **disc dislocation without reduction.**

(1)Disc Displacement

If the inferior retrodiscal lamina and the discal collateral ligament become elongated, the disc can be positioned more anteriorly by the superior lateral pterygoid muscle. When this anterior pull is constant, a thinning of the posterior border of the disc may allow the disc to be displaced in a more anterior position. With the condyle resting on a more posterior portion of the disc, an abnormal translatory shift of the condyle over the disc can occur during opening.

Associated with the abnormal condyle-disc movement is a click, which may be felt just during opening (single click) or during both opening and closing (reciprocal clicking).



A, Normal condyle-disc relationship in the resting closed joint.

B, Anterior functional displacement of the disc. The posterior discal border has been thinned, and the discal and inferior retrodiscal lamina are sufficiently elongated to allow the disc to be anteromedially displaced.

History

A history of trauma is commonly associated with the onset of joint sounds. Accompanying pain may or may not exist. If pain is present, it is intracapsular and a concomitant of the dysfunction (the click).

Clinical characteristics

Examination reveals joint sounds during opening and closing. Disc displacement is characterized by a normal range of jaw movement during both opening and eccentric movements. When reciprocal clicking is present, the two clicks normally occur at different degrees of opening, with the closing click occurring near the intercuspal position.

(2)Disc Dislocation with Reduction

If the inferior retrodiscal lamina and discal collateral ligaments become further elongated and the posterior border of the disc sufficiently thinned, the disc can slip or be forced completely through the discal space. Because the disc and condyle no longer articulate, this condition is referred to as a disc dislocation. If the patient can so manipulate the jaw as to reposition the condyle onto the posterior border of the disc, the disc is said to be reduced.

History

Normally there is a long history of clicking in the joint and more recently some catching sensation. The patient reports that when it catches and gets stuck, he or she can move the jaw around a little and get it back to functioning normally. The catching may or may not be painful, but if pain is present it is directly associated with the dysfunctional symptoms.

Clinical characteristics

Unless the jaw is shifted to the point of reducing the disc, the patient presents with a limited range of opening. When opening reduces the disc, there is a noticeable deviation in the opening pathway. After the disc is reduced, a normal range of mandibular movement is present. The interincisal distance at which the disc is reduced during opening is usually greater than when the disc is redislocated during closing.

(3)Disc Dislocation without Reduction

As the ligament becomes more elongated and the elasticity of the superior retrodiscal lamina is lost, recapturing of the disc becomes more difficult. When the disc is not reduced, the forward translation of the condyle merely forces the disc in front of the condyle.

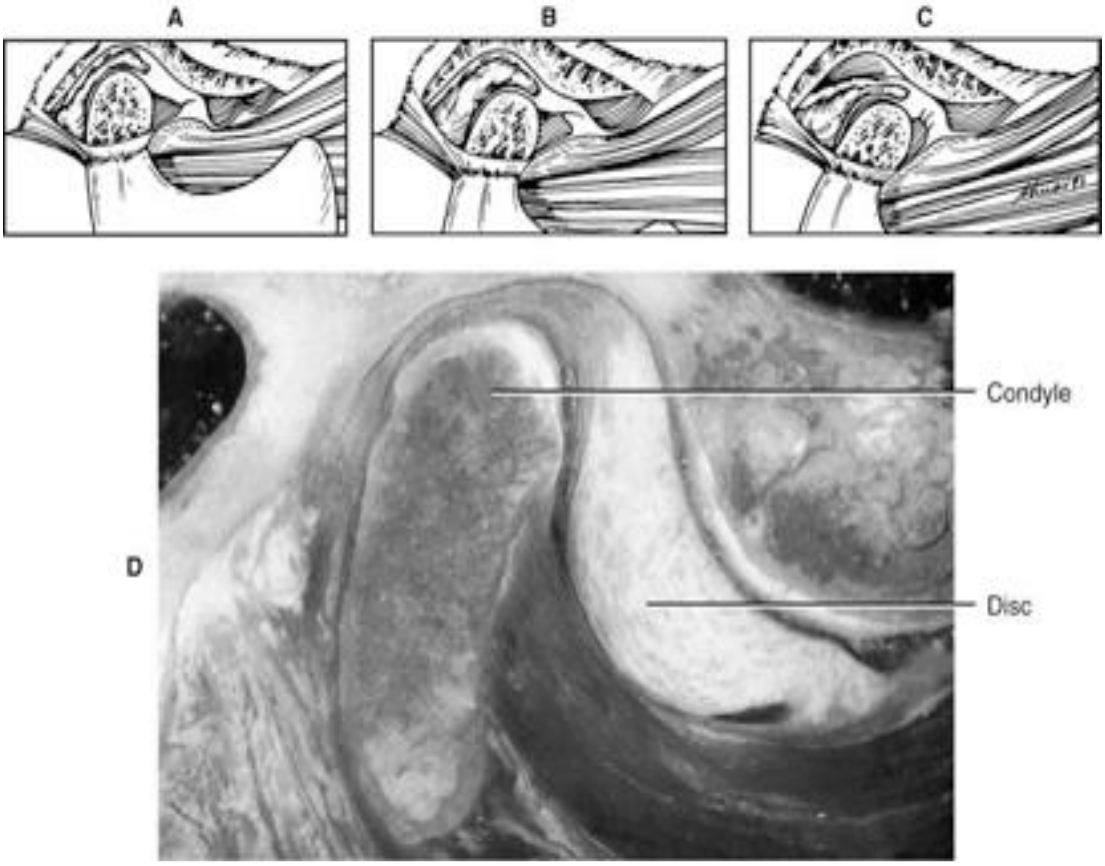
History

Most patients with a history of disc dislocation without reduction know precisely when the dislocation occurred. They can readily relate it to an event (biting on a hard piece of meat or waking up with the condition). They report that the jaw is locked closed, so normal opening cannot be achieved. When pain is present, it usually accompanies attempts to open beyond the joint restriction. The history also reveals that the clicking occurred before the locking but not since the disc dislocation has occurred.

Clinical characteristics

The range of mandibular opening is 25 to 30 mm, and the mandible deflects to the involved joint. The maximum point of opening reveals a hard end feel. In other words, if mild, steady, downward force is applied to the lower incisors, there is little increase in

mouth opening. Eccentric movements are relatively normal on the ipsilateral side but restricted on the contralateral side. Loading the joint with bilateral manual manipulation is often painful to the affected joint because the condyle is seated on the retrodiscal tissues. In some patients the only definitive way to be certain that the disc is permanently dislocated is by soft tissue imaging (i.e., magnetic resonance imaging).



A, Resting closed joint position.

B, During the early stages of translation, the condyle does not move onto the disc but instead pushes the disc forward.

C, The disc becomes jammed forward in the joint, preventing the normal range of condylar translator movement. This condition is referred to clinically as a closed lock.

D, In this specimen the disc **(D)** is dislocated anterior to the condyle **(C)**.

STRUCTURAL INCOMPATIBILITIES OF THE ARTICULAR SURFACES

The four types of structural incompatibilities of the articular surfaces are [109] (1) deviation in form, (2) adhesions, (3) subluxation, and (4) spontaneous dislocation

(1)Deviation in Form

Causes

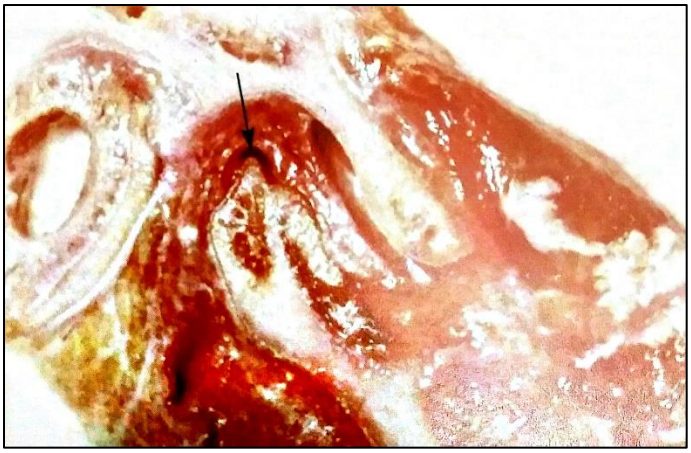
Deviations in form are caused by actual changes in the shape of the articular surfaces. They can occur to the condyle, the fossa, and/or the disc. Alterations in form of the bony surfaces may be a flattening of the condyle or fossa or even a bony protuberance on the condyle. Changes in the form of the disc include both thinning of the borders and perforations.

History

The history associated with alterations in form is usually a long-term dysfunction that may not present as a painful condition. Often the patient has learned a pattern of mandibular movement (altered muscle engrams) that avoids the deviation in form and therefore avoids painful symptoms.

Clinical characteristics

Most deviations in form cause dysfunction at a particular point of movement. Therefore the dysfunction becomes a repeatable observation at the same point of opening. During opening the dysfunction is observed at the same degree of mandibular separation as during closing. This is a significant finding because disc displacements and dislocations do not present in this manner. Also with deviation in form, the speed and force of opening do not alter the point of dysfunction. With a displaced disc, changing the speed and force of opening can alter the interincisal distance of the click.



The bony spur in the posterior superior aspect of the condyle (arrow) is demonstrated. This significant alteration in form appears to impinge on the retrodiscal tissues likely to lead to pain.

(2)Adherences/Adhesions

Causes

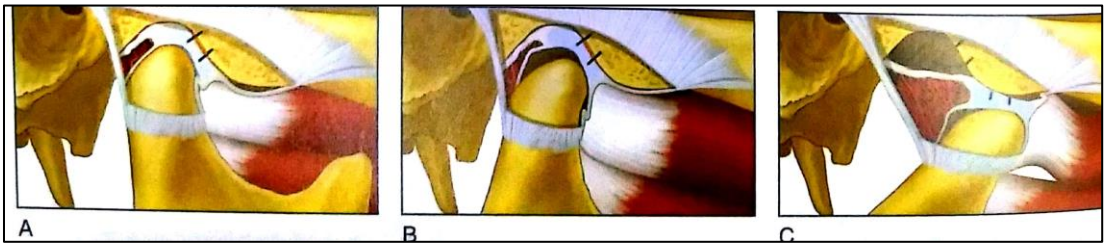
An adherence represents a temporary sticking of the articular surfaces and may occur between the condyle and the disc (inferior joint space) or between the disc and the fossa (superior joint space). Adherences commonly result from prolonged static loading of the joint structures. Adhesions may also arise from a loss of effective lubrication secondary to a hypoxia/reperfusion injury.

Although adherences are normally temporary, if they remain they may lead to the more permanent condition of adhesion. Adhesions are produced by the development of fibrosis connective tissue between the articular surfaces of the fossae or condyle and the disc or its surrounding tissues. Adhesions may develop secondary to hemarthrosis or inflammation caused by macrotrauma or surgery.

History

Adherences that develop occasionally but are broken or released during function can be diagnosed only through the history. Usually the patient will report a long period when the jaw was statically loaded (such as clenching during sleep). This period was followed by a sensation of limited mouth opening.

As the patient tried to open, a single click was felt and normal range of movement was immediately returned. The click or catching sensation does not return during opening and closing unless the joint is again statically loaded for a prolonged time. The adherence occurs because static loading of the joint exhausts weeping lubrication. As soon as enough energy is exerted through joint movement to break the adherence, boundary lubrication takes over and sticking does not recur unless the static loading is repeated. These patients typically report that in the morning the jaw appears "stiff" until they pop it once and normal movement is restored.



A, Adherence in the superior joint space.
B, The presence of the adherence limits the joint to only rotation.
C, If the adherence is freed, normal translation can occur.

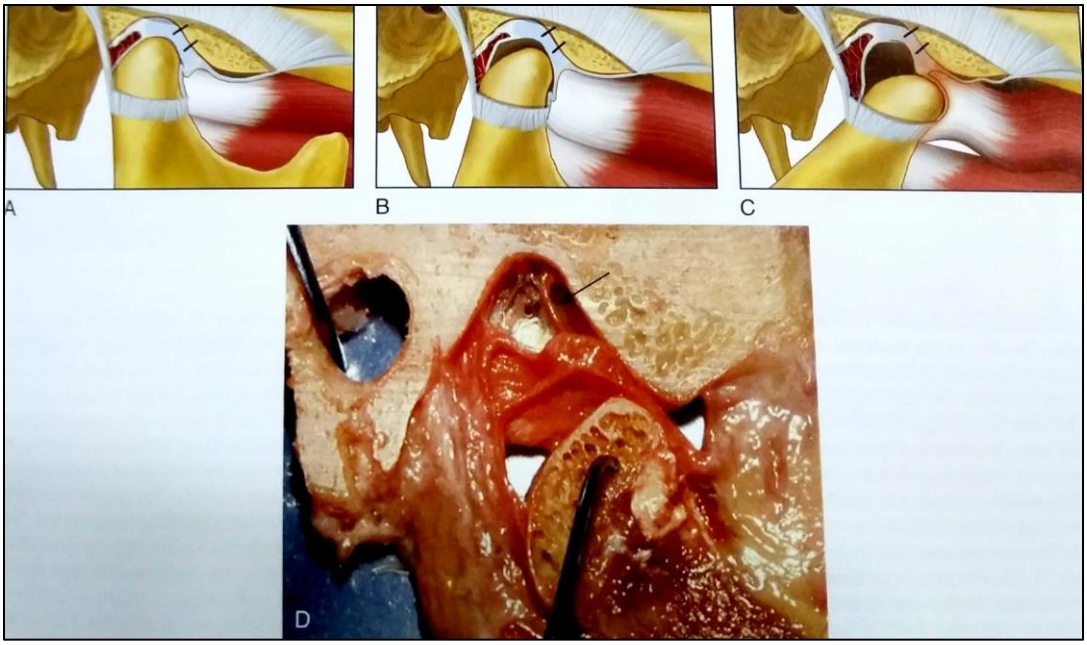
When adhesions permanently fix the articular surfaces, the patient complains of reduced function usually associated with limited opening. The symptoms are constant and repeatable. Pain may or may not be present. If pain is a symptom, it is normally associated with attempts to increase opening that elongates ligaments.

Clinical characteristics

When adherences or adhesions occurs between the disc and fossa (superior joint space), normal translation of the condyle-disc complex is inhibited. Therefore movement of the condyle is limited only to rotation. The patient presents with a mandibular opening of only 25 to 30 mm. This is similar to the finding of a disc dislocation without reduction. The major difference is that when the joint is loaded through bilateral manipulation, the intracapsular pain is not provoked. No pain is noted because the manual loading is applied to a disc that is still in proper position for loading.

With a disc dislocation without reduction, loading occurs on the retrodiscal tissues, which will likely produce pain.

If long-standing superior joint cavity adhesions are present, the discal collateral and anterior capsular ligaments can become elongated. With this the condyle begins to translate forward, leaving the disc behind. When the condyle is forward, it would appear as if the disc is posteriorly dislocated.

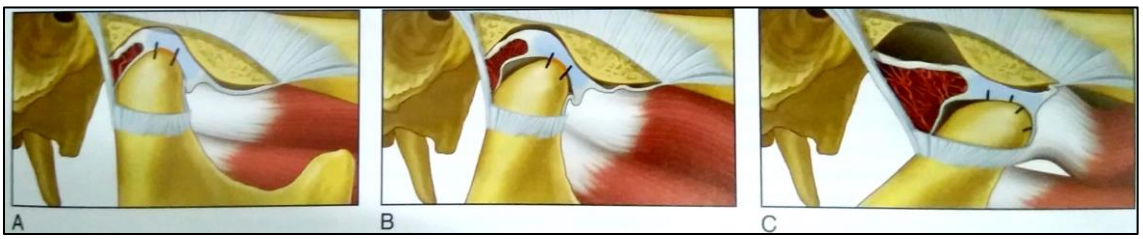


A, Permanent adhesion between the disc and fossa.

B, Continued movement of condyle causes elongation of the discal and anterior capsular ligaments, permitting the condyle to move onto the anterior border of the disc.

C, Eventually the condyle passes over the anterior border of the disc, causing a posterior dislocation of the disc.

D, In this specimen there appears to be a fibrous attachment from the disc to the superior aspect of the fossa (arrow).



A Adherence in the inferior joint space.

B, As the mouth opens, translation between the disc and fossa can occur, but rotation between the disc and condyle is inhibited. This can lead to a sensation of tightness and irregular movement.

C, If the adherence is freed, normal disc movement returns.

(3)Subluxation(Hypermobility)

Subluxation of the TMJ represents a sudden forward movement of the condyle during the latter phase of mouth opening. As the condyle moves beyond the crest of the eminence, it appears to jump forward to the wide-open position.

Causes

Subluxation occurs in the absence of any pathologic condition. It represents normal joint movement as a result of certain anatomic features. A TMJ whose articular eminence has a steep, short, posterior slope followed by a longer anterior slope that is often more superior than the crest tends to subluxate. This occurs because the steep eminence requires a great deal of rotational movement of the disc on the condyle as the condyle translates out of the fossa.

Often the amount of rotational movement of the disc permitted by the anterior capsular ligament is fully utilized before complete translation of the condyle is reached.

Because the disc cannot rotate any farther posteriorly, the remaining condylar translation occurs in the form of an anterior movement of the condyle and disc as a unit. This represents a sudden forward jump of the condyle and disc to the maximum translated position.

History

The patient who subluxates will often report that the jaw “goes out” any time he or she opens wide. Some patients report jaw clicking, but when observed clinically the click is not similar to a disc displacement. The joint sound is best described as a “thud.”

Clinical characteristics

Subluxation can be observed clinically merely by requesting the patient to open wide. At the latter stage of opening the condyle will jump forward, leaving a small depression in the face behind it. The lateral pole can be felt or observed during this movement. The midline pathway of mandibular opening will be seen to deviate and return

as the condyle moves over the eminence. The deviation is much greater and much closer to the maximally open position than that seen with a disc derangement disorder. Usually no pain is associated with the movement unless it is repeated often (abuse). Subluxation is a repeatable clinical phenomenon that does not vary with changes in speed or force of opening.

(4)Spontaneous Dislocation (Open Lock)

Causes

Spontaneous Dislocation represents a hyperextension of the TMJ, resulting in a condition that fixes the joint in the open position and prevents any translation. This condition is clinically referred to as an open lock because the patient cannot close the mouth. Like subluxation, it can occur in any joint that is forced open beyond the normal restrictions provided by the ligaments.

When the condyle is in the full forward translatory position, the disc is rotated to its fullest posterior extent on the condyle and firm contact exists among the disc, condyle, and articular eminence. In this position the strong retracting force of the superior retrodiscal lamina, along with the lack of activity of the superior lateral pterygoid, prevents the disc from being anteriorly displaced. The superior lateral pterygoid normally does not become active until the turnaround phase of the closing cycle. If for some reason it becomes active early (during the most forward translatory position), its forward pull may overcome the superior retrodiscal lamina and the disc will be pulled through the anterior disc space, resulting in a spontaneous anterior dislocation. This premature activity of the muscle can occur during a yawn or when the muscles are fatigued from maintaining the mouth open for a long time

Spontaneous dislocation (with disc anteriorly dislocated).

A, Normal condyle-disc relationship in the resting closed joint position.

B, Maximum translated position. The disc has rotated posteriorly on the condyle as far as permitted by the anterior capsular ligament.

C, If the mouth is forced open wider, the disc is pulled forward by the anterior capsular ligament through the disc space. As the condyle moves superiorly, the disc space collapses, trapping the disc forward.

Spontaneous dislocation (with disc posteriorly dislocated).

A, Normal condyle-disc relationship in the resting closed joint position.

B, In the maximum translated position the disc has rotated posteriorly on the condyle as far as permitted by the anterior capsular ligament.

C, If the mouth is forced open wider, the condyle is forced over the disc, dislocating it posterior to the condyle. As the condyle moves superiorly, the disc space collapses, trapping the disc posteriorly

History

Spontaneous Dislocation is often associated with wide-open mouth procedures, such as a long dental appointment, but it may also follow an extended yawn. The patient reports that he or she cannot close the mouth. Pain is associated with the dislocation, and this usually causes great distress

Clinical characteristics

Spontaneous dislocation is easy to diagnosis because it is sudden and the patient is locked in the wide-open mouth position. Clinically the anterior teeth are usually separated, with the posterior teeth closed. The patient cannot verbalize the problem because his or her jaw is locked open but needs to make known the distress and pain felt.

INFLAMMATORY JOINT DISORDERS

Inflammatory disorders of the TMJ are characterized by continuous deep pain, usually accentuated by function. Because the pain is continuous, it can produce secondary central excitatory effects. These usually appear as referred pain, excessive sensitivity to touch (hyperalgesia), and/or increased protective co contraction.

Inflammatory joint disorders are classified according to the structures involved [109]: **synovitis, capsulitis, retrodiscitis and the arthritides.**

(1)Synovitis or Capsulitis

Inflammation of the synovial tissues (synovitis) and of the capsular ligament (capsulitis) presents clinically as one disorder; thus a differential diagnosis is difficult. The only way they can be differentiated is by using arthroscopy.

Causes

Synovitis and capsulitis usually follow trauma to the tissue, such as macrotrauma (e.g., a blow to the chin) or microtrauma (e.g., a slow impingement on these tissues by an anterior displacement of the disc). Trauma may also arise from wide-open mouth procedures or abusive movements. Sometimes inflammation may spread from adjacent structures.

History

The history often includes an incident of trauma or abuse. The continuous pain usually originates in the joint area, and any movement that elongates the capsular ligament increases it. Because it is a deep, constant pain, secondary central excitatory effects can be created.

Clinical characteristics

The capsular ligament can be palpated by finger pressure over the lateral pole of the condyle. Pain caused by this indicates a capsulitis. Limited mandibular opening secondary to pain is common, and therefore a soft end feel is noted. If edema from the inflammation is present, the condyle may be displaced inferiorly, which will create a disocclusion of the ipsilateral posterior teeth.

(2)Retrodiscitis

Causes

Inflammation of the retrodiscal tissues (retrodiscitis) can result from macrotrauma such as a blow to the chin. This trauma can suddenly force the condyle posteriorly into the retrodiscal tissues. When trauma injures these tissues, a secondary inflammatory reaction may result.

Microtrauma may also cause retrodiscitis such as in the progressive phases of disc displacement and dislocation. During these conditions the condyle gradually encroaches on the inferior retrodiscal lamina and retrodiscal tissues. This gradually insults these tissues, leading to retrodiscitis.

History

An incident of trauma to the jaw or a progressive disc derangement disorder is the usual finding. The pain is constant, originating in the joint area, and jaw movement accentuates it. Clenching of the teeth increases pain, but clenching on an ipsilateral separator often reduces the pain. Because of the constant deep pain, secondary central excitatory effects are common.

Clinical characteristics

Limited jaw movement is caused by arthralgia. A soft end feel is present unless the inflammation is associated with a disc dislocation. If the retrodiscal tissues swell because of inflammation, the condyle can be forced slightly forward and down the eminence. This creates an acute malocclusion that is observed clinically as disocclusion of the ipsilateral posterior teeth and heavy contact of the contralateral anterior teeth.

(3)Arthritides

Arthritis means inflammation of the articular surfaces of the joint. Several types of arthritides can affect the TMJ. The following types are common: **osteoarthritis, osteoarthrosis, and polyarthritides.**

Osteoarthritis and osteoarthrosis

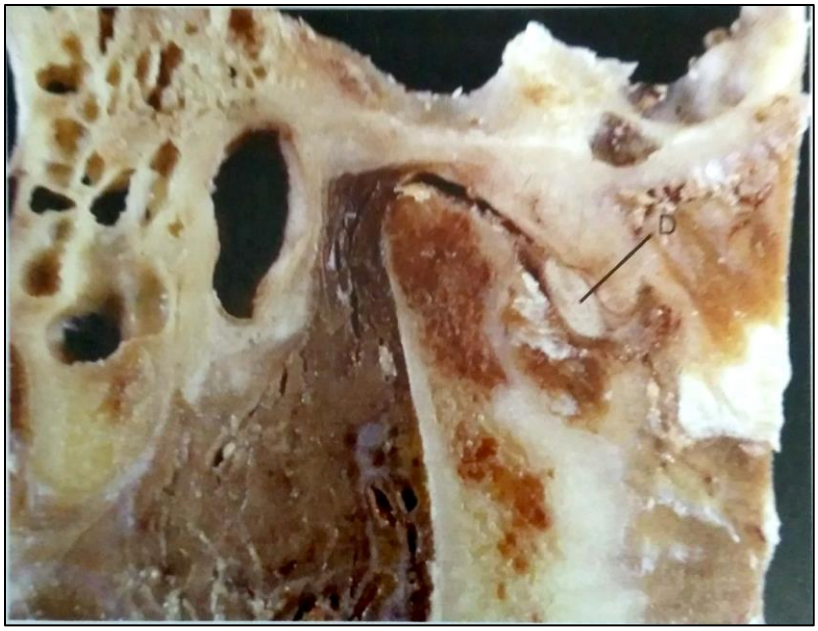
Causes

Osteoarthritisre presents a destructive process by which the bony articular surfaces of the condyle and fossa become altered. It is generally considered to be the body's response to increased loading of a joint. As loading forces continue and the articular surface becomes softened (chondromalacia), the subarticular bone begins to resorb. Progressive degeneration eventually results in loss of the subchondral cortical layer, bone erosion, and subsequent radiographic evidence of osteoarthritis. Importantly, radiographic changes are only seen in later stages of osteoarthritis and may not reflect the disease accurately.

Osteoarthritisis often painful, and symptoms are accentuated by jaw movement. Crepitation (grating joint sounds) is a common finding with this disorder. Osteoarthritis can occur any time the joint is overloaded but is most commonly associated with disc dislocation or perforation. Once the disc is dislocated and the retrodiscal tissues break down, the condyle begins to articulate directly with the fossa, accelerating the destructive process. In time the dense fibrous articular surfaces are destroyed and bony changes occur.

Radiographically, the surfaces seem to be eroded and flattened. Any movement of these surfaces creates pain, so jaw function usually becomes restricted.

Although osteoarthritis is in the category of inflammatory disorders, it is not a true inflammatory condition. Often once loading is decreased, the arthritic condition can become adaptive yet the bony morphology remains altered. The adaptive stage has been referred to as osteoarthrosis. Overloading of the articular surfaces may be caused by high levels of parafunctional activity.



Flattening of the articular surface of the condyle and the osteophyte is demonstrated. The disc is anteriorly dislocated

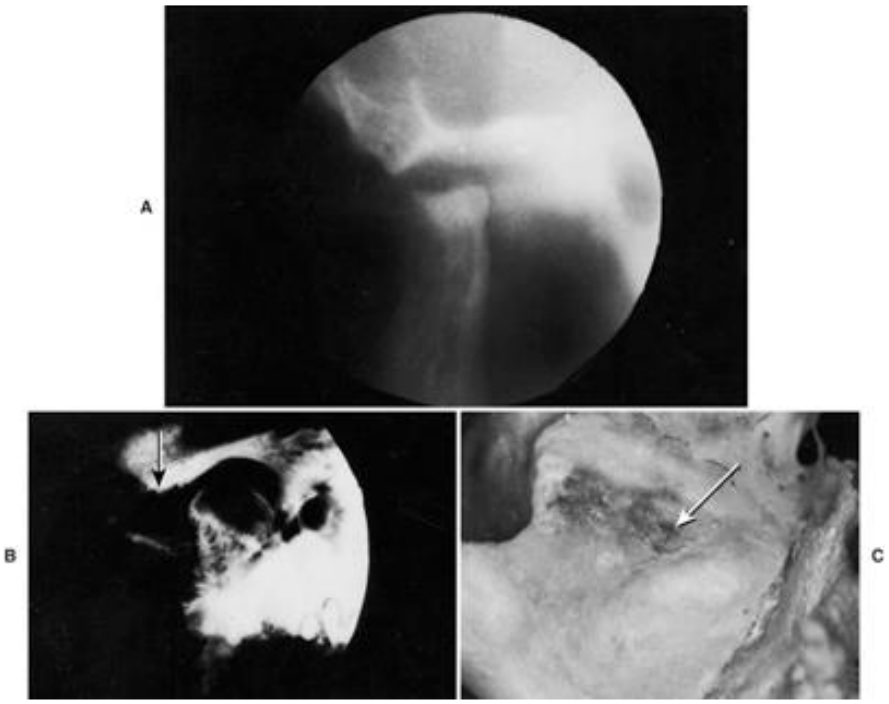
History

The patient with osteoarthritis usually reports unilateral joint pain that is aggravated by mandibular movement. The pain is usually constant but may worsen in the late afternoon or evening.

Secondary central excitatory effects are often present. Because osteoarthrosis represents a stable adaptive phase, the patient does not report symptoms.

Clinical characteristics

Limited mandibular opening is characteristic because of the joint pain. A soft end feel is common unless the osteoarthritis is associated with an anteriorly dislocated disc. Crepitation can typically be felt or reported by the patient. The diagnosis is usually confirmed by TMJ radiographs, which will reveal evidence of structural changes in the subarticular bone of the condyle or fossa (i.e., flattening, osteophytes, erosions). Osteoarthrosis is confirmed when structural changes in the subarticular bone are seen on radiographs but the patient reports no clinical symptoms of pain.



A, Severely deformed condyle resulting from osteoarthritis (lateral tomogram).

B,Condyle and fossa (transcranial projection). Irregular surfaces of the subarticular bone near the crest of the articular eminence (arrow) are shown.

C, Mandibular fossa in the previous radiograph (inferior view). Degenerative changes in the articular eminence (arrow) are shown.

Polyarthritides

Polyarthritides represent a group of disorders in which the articular surfaces of the joint become inflamed. Each is identified according to its causative factors.

Traumatic arthritis

Macrotrauma to the jaw can cause articular surface changes that are great enough to produce inflammation. A positive history of macrotrauma is usually found and can be closely related to the onset of symptoms.

The patient reports constant arthralgia accentuated with movement. There is limited mandibular opening secondary to pain. A soft end feel is common. Acute mal occlusion may exist if swelling is present.

Infectious arthritis

A sterile inflammatory reaction of the articular surfaces can be associated with a systemic disease or immunologic response. A nonsterile, inflammatory arthritis may result from a bacterial invasion caused by a penetrating wound, spreading infection from adjacent structures, or even bacteremia following a systemic infection. The history reveals local infection of adjacent tissues or a penetrating wound to the joint. Constant pain is accentuated with movement. Joint swelling and elevated tissue temperature are present clinically. Blood studies and fluid aspirated from the joint cavity may assist in diagnosis.

Rheumatoid arthritis

The precise cause of this systemic disorder affecting multiple joints in the body is unknown. It is an inflammation of the synovial membranes that extends into the surrounding connective tissues and articular surfaces, which then become thickened and

tender. As force is placed on these surfaces, the synovial cells release enzymes that damage the joint tissues, especially the cartilage. In severe cases even the osseous tissues can be resorbed, with significant loss of condylar support.

In severe cases, when condylar support has been lost, an acute mal occlusion results, characterized by heavy posterior contacts and an anterior open bite. The diagnosis is confirmed by blood studies.

Inflammatory Disorders of Associated Structures

Although not directly related to joint disorders, a few associated structures can also become inflamed [111] Two structures that need to be considered are (1) temporalis tendonitis and (2) stylomandibular ligament inflammation.

(1)Temporalis tendonitis

Causes

The temporal muscle is attached to the coronoid process by a relatively large tendon. This tendon is susceptible to inflammation, as are other tendons (i.e., elbow). Constant and prolonged activity of the temporalis muscle can result in a temporal tendonitis. This muscle hyperactivity may be secondary to bruxism, increased emotional stress, or a constant source of deep pain such as intracapsular pain.

History

Patients with temporal tendonitis will often report a constant pain felt in the temple region and/or behind the eye. It is usually a unilateral complaint that is aggravated by jaw function.

Clinical characteristics

Temporal tendonitis will commonly produce pain whenever the temporalis muscle is activated (mandibular elevation). A restricted jaw opening is noted with a soft end feel. Intraoral palpation of the temporal tendon will produce extreme pain. This is accomplished by placing a finger on the ascending ramus and moving it up as high as possible to the most superior portion of the coronoid process.

(2)Stylomandibular ligament inflammation

Stylomandibular ligament can become inflamed, producing pain at the angle of the mandible and even radiating superiorly to the eye and temple. This condition can be identified by placing the finger at the angle of the mandible and attempting to move inward onto the medial aspect of the mandible where the stylomandibular ligament is attached.

CHRONIC MANDIBULAR HYPOMOBILITY

Chronic mandibular hypomobility is a long-term, painless restriction of the mandible [112]. Pain is elicited only when force is used to attempt opening beyond the limitations. The condition can be classified according to the cause as **ankylosis, muscle contracture, and coronoid process impedance.**

Ankylosis

Sometimes the intracapsular surfaces of the joint develop adhesions that prohibit normal movements. This is called ankylosis. When ankylosis is present, the mandible cannot translate from the fossa, resulting in a restricted range of movement. Ankylosis can result from fibrous adhesions in the joint or fibrotic changes in the capsular ligament. On occasion a bony ankyloses can develop in which the condyle actually joins with the fossa.

Causes

The most common source of ankylosis is macrotrauma. This trauma causes tissue damage resulting in secondary inflammation. Trauma may also cause hemarthrosis or bleeding within the joint that can set up a matrix for the development of fibrosis. Another common source of trauma is TMJ surgery. Surgery often produces fibrotic changes in the capsular ligament, restricting mandibular movement. Osseous ankylosis is more commonly associated with a previous infection.

History

Patients often report a previous injury or capsulitis along with an obvious limitation in mandibular movement. The limited opening has been present for a considerable period of time.

Clinical Characteristics

Movement is restricted in all positions (open, lateral, protrusive), and if the ankylosis is unilateral, midline pathway deflection will be to that side during opening. TMJ radiographs can be used to confirm this. The condyle will not move significantly in protrusion or laterotrusion to the contralateral side, and therefore no significant difference is apparent in these two films. Bony ankylosis can also be confirmed with radiographs.

Muscle Contracture

In this discussion, muscle contracture refers to the clinical shortening of the resting length of a muscle without interfering in its ability to contract further.

Bell has described two types of muscle contracture: (1) myostatic and (2) myofibrotic. It may be difficult to differentiate between these clinically, but differentiation is important because they respond differently to therapy.

Myostatic Contracture

Causes

Myostatic contracture results when a muscle is kept from fully relaxing (stretching) for a prolonged time. The restriction may be caused by the fact that full relaxation causes pain in an associated structure. For example, if the mouth can open only 25 mm without pain in the TMJ, the elevator muscles will protectively restrict movement to within this range. If this situation continues, myostatic contraction will result.

History

The patient reports a long history of restricted jaw movement. It may have begun secondary to a pain condition that has now resolved.

Clinical characteristics

Myostatic contracture is characterized by painless limitation of mouth opening.

Myofibrotic Contracture

Causes

Myofibrotic contracture occurs as a result of tissue adhesions within the muscle or its sheath. It commonly follows a myositic condition or trauma to the muscle.

History

The history for myofibrotic contracture reveals a previous muscle injury or a long-term restriction in the range of movement. The patient has no pain complaints. Sometimes the patient will not even be aware of the limited range of opening because it has been present for so long.

Clinical characteristics

Myofibrotic contracture is characterized by painless limitation of mouth opening. Lateral condylar movement is unaffected. Thus if the diagnosis is difficult, radiographs showing limited condylar movement during opening but normal movement during lateral excursions may help. No acute mal occlusion occurs.

Coronoid Impedance

Causes

During opening the coronoid process passes anteroinferiorly between the zygomatic process and the posterior lateral surface of the maxilla.

If the coronoid process is extremely long or if fibrosis has developed in this area, its movement will be inhibited and chronic hypomobility of the mandible may result. Trauma to or an infection in the area just anterior to the coronoid process can lead to fibrotic adhesions or union of these tissues.

Surgical intervention in the area can also cause coronoid impedance. Possibly in certain conditions the coronoid process becomes elongated, which would prevent its movement through this soft tissue area.

History

Painless restriction of opening in many cases followed trauma to the area or an infection. A long-standing anterior disc dislocation may also have occurred.

Clinical Characteristics

Limitation is evident in all movements, but especially in protrusion. A straight midline opening path is commonly observed, unless one coronoid process is more free than the other. If the problem is unilateral, opening will deflect the mandible to the same side.

GROWTH DISORDERS

TMDs resulting from growth disturbances may be caused by a variety of etiologies. The growth disturbance may be in the bones or the muscles. Common growth disturbances of the bones are agenesis (no growth), hypoplasia (insufficient growth), hyperplasia (too much growth), or neoplasia (uncontrolled, destructive growth).

Common growth disturbances of the muscles are hypotrophy (weakened muscle), hypertrophy (increased size and strength of the muscle), and neoplasia (uncontrolled, destructive growth).

Causes

Deficiency of or alterations in growth typically result from trauma and can induce major mal occlusions. Neoplastic activity involving the TMJ is rare but, if left undiagnosed, can become aggressive.

Clinical Characteristics

Any alteration of function or the presence of pain is secondary to structural changes. Clinical asymmetry may be noticed that is associated with and indicative of a growth or developmental interruption. Radiographs of the TMJ are extremely important in identifying structural (bony) changes that have taken place.

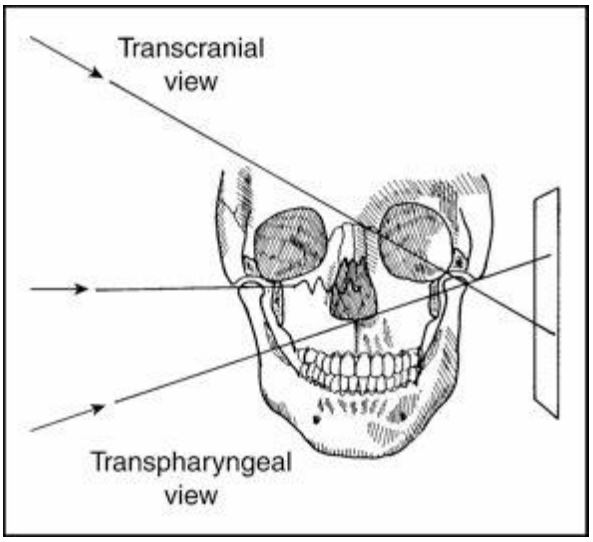
DIAGNOSTIC TOOLS

Imaging of the Temporomandibular Joint

Various types of imaging techniques can be used to gain additional insight regarding the health and function of the TMJs. When painful symptoms arise from the joints and there is reason to believe that a pathologic condition exists, TMJ radiographs should be obtained. These will provide information regarding (1) the morphologic characteristics of the bony components of the joint and (2) certain functional relationships between the condyle and the fossa.

Radiographic Techniques

Radiographs of the TMJs are complicated by several anatomic and technical circumstances that hinder clear and unobstructed visualization of the joints. A pure lateral view of the condyle is impossible with conventional x-ray equipment because of superimposition of the bony structures of the midface. Therefore to achieve a successful projection of the TMJs, the x-rays must be directed across the head either from below the midface in a superior direction (infracranial or transpharyngeal view) or through the skull directed inferiorly above the midface to the condyle (transcranial). Only through a specialized tomographic projection can the pure lateral view of the condyle be obtained.



Four basic radiographic techniques [113] can be used in most dental offices for evaluating the

TMJs: (1) panoramic, (2) lateral transcranial, (3) transpharyngeal, and (4) transmaxillary (anteroposterior [AP]) views.

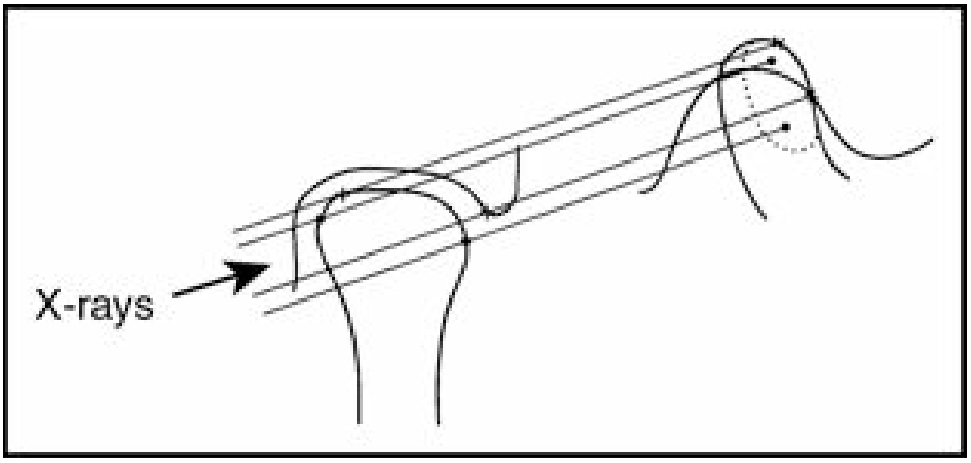
Panoramic view

The panoramic radiograph has become widely used in dental offices. With slight variations in the standard technique, it can provide screening of the condyles. It is a good screening tool because its use results in minimum superimposition of structures over the condyles.

Although the bony structures of the condyle can be evaluated well, the panoramic view has some limitations. To view the condyle best, it is often necessary for the patient to open maximally so that the structures of the articular fossae will not be superimposed on the condyle.

With this technique the condyles are the only structures that are visualized well. The articular fossae are often partially, if not totally, obscured.

Because the panoramic radiograph is an infracranial view, the lateral pole of the condyle becomes superimposed over the condylar head. Therefore the area that appears to represent the superior sub articular surface of the condyle is actually only the subarticular surface of the medial pole.



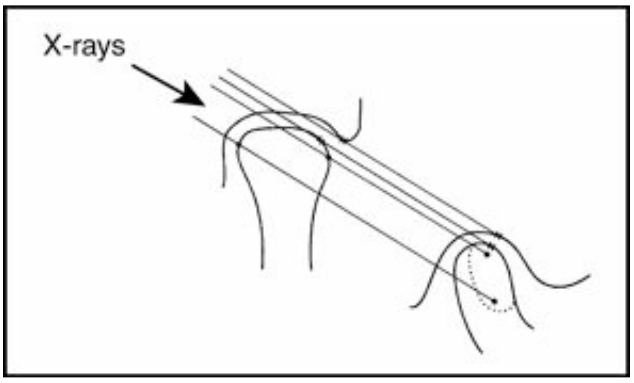
The area that appears to be the superior subarticular surface of the condyle is actually the medial pole. The lateral pole is superimposed inferiorly over the body of the condyle. The fossa is also superimposed over the condyle, which complicates interpretation of the radiograph.

Lateral transcranial view

The lateral transcranial view can provide good visualization of both the condyle and the fossa. In past years this technique was popular because with minimal expense it can be adapted to most general dental radiographic techniques.

The patient is placed in a head positioner, and the x-rays are directed inferiorly across the skull (above the midface) to the contralateral TMJ and recorded. Usually several projections of each joint are taken so that the function can be evaluated. For example, one projection is obtained with the teeth together in maximum intercuspation and another with

the mouth maximally opened. Interpretation of the transcranial view begins with an understanding of the angle by which the projection is made.



The area that appears to be the superior subarticular surface of the condyle is actually the lateral pole. The medial pole is superimposed inferiorly over the body of the condyle. In this projection the fossa is not superimposed over the condyle; thus a clearer view of the condyle is usually obtained..

Transpharyngeal projection

This view is similar to the panoramic view. However, because the x-rays are directed either from below the angle of the mandible or through the sigmoid notch, the angle at which they project the condyle is not as great as in the panoramic view. This means that the projection is closer to a true lateral view. Although the technique demonstrates the condyle satisfactorily, the mandibular fossa is not usually visualized as well as the transcranial view.

Anteroposterior transmaxillary projection

This view can also be helpful. It is obtained from anterior to posterior with the mouth wide open and the condyles translated out of the fossae. If the condyle cannot be translated to the crest of the eminence, superimposition of the subarticular bone results and

much of the usefulness of this radiograph is lost. When this projection can be correctly taken, it offers a good view of the superior subarticular bone of the condyle, as well as the medial and lateral poles. The AP projection also affords an excellent view for evaluating a fracture in the neck of the condyle.

A, Positioning for the left temporomandibular joint.
B, Typical view of a condyle. In this projection the medial (MP) and lateral (LP) poles can be easily visualized along with the superior subarticular surfaces (SSS) of the condyle.

Tomography

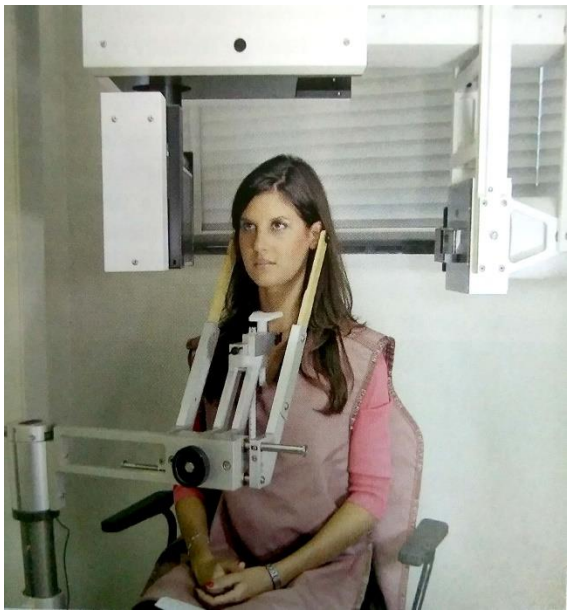
The lateral tomographic view provides a more accurate view of the TMJs. It uses controlled movement of the head of the x-ray tube and the film to obtain a radiograph of the desired structures that deliberately blurs out other structures. These radiographs are not infracranial or transcranial projections but true lateral projections.

AP views can also be taken with tomography, revealing the lateral and medial poles without any superimposition. This view may also be helpful in evaluating the articular surface of the condyle.

The tomogram can be obtained at precise sagittal intervals, so true sections of the joint are seen (lateral, middle, and medial poles). Bony changes and functional relationships of the joint can also be easily visualized.

The advantage of tomography is that it is generally more accurate than panoramic or transcranial radiographs for identifying bony abnormalities or changes. Because it is a true sagittal view, one can evaluate condylar position in the fossae more accurately than with the transcranial view.

The disadvantages of tomography are cost and inconvenience. Another disadvantage is that the patient is exposed to higher levels of radiation than with other techniques.



Patient seating in a tomography unit in the proper position to take a lateral temporomandibular joint tomogram.

Arthrography

Arthrography was a radiographic technique in which contrast medium was injected into the joint spaces to outline important soft tissue structures. This technique became popular in the mid-1980s when the importance of the soft tissues was appreciated (especially disc position). Routine radiographic techniques and tomography only depict bony structures and their interrelationships, with no regard for the soft tissues.

Through careful analysis of the joint spaces outlined by the contrast medium, the position and sometimes the condition of the articular disc could be ascertained. Because arthrography used a fluoroscope, one could visualize the dynamic movements of the disc and condyle, which was helpful in identifying condyle-disc dysfunction and perforations of the disc.

Unfortunately, this procedure was technique sensitive and, with the onset of better soft tissue imaging techniques (magnetic resonance imaging [MRI]), arthrograms became obsolete.

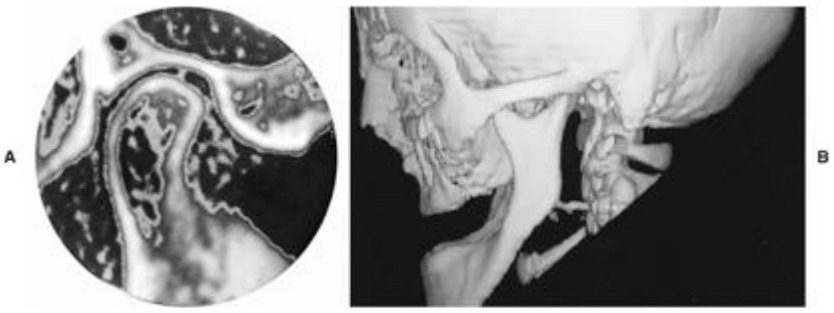
Computed tomography

Another technique that has been developed in the past decade is computed tomographic (CT) scanning. CT scanners produce digital transmission through various tissues. These data may be transformed into a density scale and used to generate or re construct a visual image. This technology can reconstruct the TMJ in a three-dimensional image that can offer even more diagnostic information to the clinician. The latest advancement in this technology is called cone beam tomography. Cone beam tomography allows for viewing the condyle in multiple planes so that all surfaces can be visualized.

This technology is also capable of reconstructing three-dimensional images of the patient that can be rotated on the computer screen for more complete viewing.

Cone beam tomography can image both hard and soft tissues; therefore the disc condyle relationship can be observed and evaluated without disturbing the existing anatomic relationships.

However, CT scans have some disadvantages. The equipment is relatively expensive and therefore not always accessible. Often CT scans expose the patient to more radiation than simpler films, but the new cone beam technology has offered better images with far less radiation.



A, Typical CT projection of the temporomandibular joint. Hard tissue (bone) is visualized better than the soft tissues with this technique.
B, A three-dimensional CT reconstruction of an edentulous patient.

Magnetic Resonance Imaging

MRI has become the gold standard for evaluating the soft tissue of the TMJ, especially disc position. It uses a strong magnetic field to create changes in the energy level of the soft tissue molecules (principally hydrogen ions). These changes in energy levels create an image in a computer similar to a CT scan.

MRI of the TMJs has demonstrated better visualization of the soft tissues than CT scans and has the major advantage of not introducing radiation that might produce tissue damage to the patient. Thus far it has shown no harmful effects.

The disadvantages of MRI are similar to those of CT scanning. MRI units are usually quite expensive and not available in a traditional dental setting. The technology may also vary from site to site, and thus the quality of images may differ greatly. Another disadvantage of the MRI is that it is normally a static image, although more recently cine MRI has begun providing information on disc and joint movement. This technology is becoming more refined and is replacing many of the existing imaging modalities.

Bone Scanning

In certain clinical conditions it is helpful to know whether there is an active inflammatory process in the TMJs. Standard radiographs may reveal that the morphology of a condyle has changed, but they are not helpful in determining whether the process is active (osteoarthritis) or dormant (osteoarthrosis). When this information is important for treatment, a bone scan can be helpful. A bone scan is obtained by injecting a radiolabeled material into the blood stream that concentrates in areas of rapid bone turnover. Once the material has had an opportunity to move to the areas of increased bone activity, an emission image is taken. A similar technique uses single photon emission computed tomography (SPECT) to identify increased areas of activity in bone. Importantly, these techniques cannot discriminate between bone re modeling and degeneration. Therefore the information must be combined with clinical findings to have meaning.

Mounted Cast

If during the examination the clinician identifies significant orthopedic instability, accurately mounted study casts may be helpful to further assess the occlusal condition. Mounted casts are not indicated for all patients being examined for TMDs. Mounted casts may be necessary when future dental treatment will be provided (e.g., prosthodontics, orthodontics). Dental study casts can be of value, not only as a baseline record for tooth and jaw relations but also for evaluating the effects of bruxism over time. The clinician should keep in mind that acute muscle and joint pain and joint edema can decrease the

accuracy of the mountings. Therefore occlusal analysis is most reliable after acute disease processes are resolved.

Electromyography

In recent years much attention has been drawn to the use of electromyograhic (EMG) recordings in the diagnosis and treatment of TMDs. Experts originally believed that if a painful muscle was in spasm, increased EMG activity would be recorded from the involved muscle. Although this is likely true for myospasms, studies now demonstrate that muscle pain is often not associated with any significant increase in EMG activity. Most muscle pain seems to be a result of local muscle soreness, myofascial pain, or centrally mediated myalgia. These conditions are not directly associated with muscle contraction (and muscle contraction is necessary to produce an increase in EMG activity).

It has also been demonstrated that relatively small variations in electrode placement can significantly change EMG recordings.

This means that recordings taken during multiple visits cannot be compared unless extreme care is taken to place the electrode in the exact same location for each recording. With such light differences and such great variations, EMG recordings should not be used to diagnose or monitor treatment of TMDs.

Mandibular Tracking Devices

Certain TMDs can produce alterations in the normal movements of the mandible. One such disorder is a disc displacement with reduction. During opening, the condyle and disc move together until the disc is reduced. During this reduction a click is often felt, and the mandibular opening pathway will deviate. If a jaw-tracking device is used, the exact movement of the mandible can be recorded.

Sonography

Sonography is the technique of recording and graphically demonstrating joint sounds. Some techniques use audio amplifying devices, whereas others rely on ultrasound echo recordings (Doppler ultrasonography). Although these devices may accurately record joint sounds, the significance of these sounds has not been well established.

Vibration Analysis

Vibration analysis has been suggested to help in diagnosing intracapsular TMD, and internal derangements in particular. This technique measures the minute vibrations made by the condyle as it translates and has been shown to be reliable.

Some specific parameters of vibration analysis appear to be sensitive and specific for identifying disc displacement patients compared with other TMD patients.

Thermography

Thermography is a technique that records and graphically illustrates surface skin temperatures. Various temperatures are recorded by different colors, producing a map that depicts the surface being studied. It has been suggested that normal subjects have bilaterally symmetric thermograms. From this concept some have suggested that thermograms that are not symmetric reveal a problem such as a TMD.

The sensitivity and specificity of identifying myofascial trigger points with thermography has not been demonstrated to be reliable. The great variation among sides, patients, and reports suggests that, at this time, thermography is not a useful technique for the diagnosis and management of TMDs.

SUMMARY

The masticatory system is extremely complex. It is made up primarily of bones, muscles, ligaments, and teeth. Movement is regulated by an intricate neurologic control system composed of the brain, brainstem, and peripheral nervous system. Each movement is coordinated to maximize function while minimizing damage to any structure.

Precise movement of the mandible by the musculature is required to move the teeth efficiently across each other during function. The mechanics and physiology of this movement are basic to the study of masticatory function

The TMJ is a compound joint. Its structure and function can be divided into two distinct systems:

1. One joint system is the tissues that surround the inferior synovial cavity (i.e., the condyle and the articular disc). Because the disc is tightly bound to the condyle by the lateral and medial discal ligaments, the only physiologic movement that can occur between these surfaces is rotation of the disc on the articular surface of the condyle. The disc and its attachment to the condyle are called the condyle-disc complex; this joint system is responsible for rotational movement in the TMJ.

2. The second system is made up of the condyle-disc complex functioning against the surface of the mandibular fossa. Because the disc is not tightly attached to the articular fossa, free sliding movement is possible between these surfaces in the superior cavity. This movement occurs when the mandible is moved forward (referred to as translation). Translation occurs in this superior joint cavity between the superior surface of the articular disc and the mandibular fossa

Thus the articular disc acts as a nonossified bone contributing to both joint systems, and hence the function of the disc justifies classifying the TMJ as a true compound joint.

From another point of view, McKay et al. regarded the TMJ as a double joint because the articular disc between the mandibular fossa of the temporal bone and the condyle, dividing the joint cavity into superior and inferior compartments is not a real bone. The articular part of the healthy disc is composed of dense, fibrous connective tissue empty of any nerves or vessels; contrarily, the posterior attachment of the disc is richly vascularized and innervated.

Rotational movement of TMJ occurs between the condyle and the inferior surface of the disc during early opening (the inferior joint space), and translation takes place in the space between the superior surface of the disc and the fossa (the superior joint space) during later opening.

Distinct from most synovial joints, the articulating surfaces of the TMJs are lined with dense fibrocartilage instead of hyaline cartilage. It is generally less susceptible to the effects of aging, less likely to break down over time, and possessed with much better ability to repair than hyaline cartilage.

Functional disorders of the TMJs are perhaps the most common findings due to the high prevalence of signs.

James Costen presented a group of symptoms that centered on the ear and TMJ in 1934, and the term “Costen syndrome” developed; later “temporomandibular joint disturbances” became popular. Shore introduced the term “temporomandibular joint dysfunction syndrome” in 1959, and afterwards “functional temporomandibular joint disturbances” was coined by Ash and Ramfjord. Bell suggested problems not merely to the joints but all disturbances associated with the function of the masticatory system should be included, and the term “temporomandibular disorders” (TMDs) has gained wide acceptance and popularity.

Understanding these basic concepts in TMJ function is essential to understanding joint dysfunction. Normal biomechanical function of the TMJ must follow the orthopedic principles just presented.

Clinicians should remember the following:

1. Ligaments do not actively participate in normal function of the TMJ. They act as guide wires, restricting certain joint movements while permitting others. They restrict joint movements both mechanically and through neuromuscular reflex activity.

2. Ligaments do not stretch. If traction force is applied, they can become elongated (i.e., increase in length). (Stretch implies the ability to return to the original length.) Once ligaments have been elongated, normal joint function is often compromised.

3. The articular surfaces of the TMJs must be maintained in constant contact. This contact is produced by the muscles that pull across the joints (the elevators: temporal, masseter, and medial pterygoid).

The function of the masticatory system is complex. Discriminatory contraction of the various head and neck muscles is necessary to move the mandible precisely and allow effective functioning.

A highly refined neurologic control system regulates and coordinates the activities of the entire masticatory system.

It consists primarily of nerves and muscles; hence the term neuromuscular system. A basic understanding of the anatomy and function of the neuromuscular system is essential to understanding the influence that tooth contacts, as well as other conditions, have on mandibular movement.

The alignment and occlusion of the dentition are extremely important in masticatory function. The basic activities of chewing, swallowing, and speaking depend greatly not only on the position of teeth in the dental arches but also on the relationship of opposing teeth as they are brought into occlusion.

Tooth positions are determined not by chance, but by numerous controlling factors such as arch width and tooth size. They are also determined by various controlling forces, such as those provided by the surrounding soft tissues.

Even though signs and symptoms of disturbances in the masticatory system are common, understanding etiology can be complex. No single cause accounts for all signs and symptoms.

Two explanations for these findings exist: (1) Either the disorder has multiple etiologies and no single treatment can affect all the etiologies, or (2) the disorder is not a single problem but represents an umbrella term under which there are multiple disorders. Regarding TMDs, both explanations are true. Certainly a multitude of conditions can affect masticatory function. Also, according to the structures involved, a variety of disorders can result.

To simplify how TMD symptoms develop, the following formula is suggested: Normal function + Event > Physiologic tolerance ⇒TMD symptoms.

Occasionally, some type of an event interrupts normal function of the masticatory system. Many events are tolerated by the system with no consequence; therefore no clinical effect is noticed. However, if the event is significant, it can exceed the physiologic tolerance of the individual, creating a response by the system. The response of the system can be seen as a variety of clinical symptoms associated with TMDs.

The signs and symptoms of TMD are common in the general population and are not always severe or debilitating. In fact, only a small percentage of the general population will seek advice for these complaints, with even fewer requiring treatment. Nevertheless, individuals seeking care need to be managed effectively and, when possible, conservatively.

To manage TMD effectively, the clinician must be able to recognize and understand its etiology. Although for years the occlusal condition has been thought to be a major cause of TMD, this is not always the case. Certainly occlusion can be a factor, and when it is the clinician must address this effectively. However, occlusion represents only one of five

etiologic considerations. Before the clinician can begin treatment, a sound understanding of the precise cause of the TMD must be attained. This begins with a complete understanding of the different types of TMD patients.

It is important to remember that "the clinician who only evaluates the occlusion is likely missing as much as the clinician who never evaluates the occlusion".

De Boever [114] sums up the current status of occlusion, saying, ''It is not primordial, but it is not a nullity.''

Etiologic factors such as trauma, emotional stress, orthopedic instability, and sources of deep pain and muscle hyperactivity were implicated as significant components.

The clinical signs and symptoms of masticatory dysfunction can be grouped into categories according to structures that are affected: (1) the muscles, (2) the temporomandibular joints (TMJs), and (3) the dentition. Muscle and TMJ disorders make up the group of conditions known as temporomandibular disorders (TMDs).

When evaluating a patient, it is important to identify both signs and symptoms clearly. A sign is an objective clinical finding that the clinician uncovers during a clinical examination. A symptom is a description or complaint reported by the patient. Patients are acutely aware of their symptoms, yet they may not be aware of their clinical signs. The signs and symptoms of temporomandibular disorders (TMDs) are extremely common. The epidemiologic studies suggest that 50% to 60% of the general population has a sign of some functional disturbance of the masticatory system. Some of these appear as significant symptoms that motivate the patient to seek treatment. Many, however, are subtle and not even at a level of clinical awareness by the patient.

The significance of the sign and the etiology, as well as the prognosis of the disorder, are factors that determine the need for treatment. The effectiveness and success of treatment lie in the ability of the clinician to establish the proper diagnosis. This can be established only after a thorough examination of the patient for the signs and symptoms of functional disturbances.

The purpose of a history and examination is to identify any area or structure of the masticatory system that shows breakdown or pathologic change. To be effective, the examiner must have a sound understanding of the clinical appearance and function of the healthy masticatory system

A classification to aid in the identification and diagnosis of TMDs has been presented. It does not include all disorders that cause pain and dysfunction of the head and neck. This classification is useful, however, in identifying the common functional disturbances of the masticatory system.

Clinicians should be vigilant in diagnosing TMD in patients who present with pain in the TMJ area. Conditions that sometimes mimic TMD include dental caries or abscess, oral lesions (e.g., herpes zoster, herpes simplex, oral ulcerations, lichen planus), conditions resulting from muscle overuse (e.g., clenching, bruxism, excessive chewing, spasm), trauma or dislocation, maxillary sinusitis, salivary gland disorders, trigeminal neuralgia, postherpetic neuralgia, glossopharyngeal neuralgia, giant cell arteritis, primary headache syndrome, and pain associated with cancer.

CONCLUSION

Temporomandibular joint disorders do not constitute one particular or single abnormal condition; rather they are multifactorial, and include stressful activities, emotional diseases, structural mal-relationships, trauma, malocclusion and various types of arthritis or viral diseases.

The onset of temporomandibular joint disorders cannot be predicted. No method of prevention of these disorders has been demonstrated. Once it occurs, cure cannot be assured.

Perfect harmony between the teeth, muscles, nerves, supporting tissues and temporomandibular joints must be established to provide health, functional efficiency, esthetics and stability to the entire stomatognathic system.

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