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Temporomandibular Disorders- A Brief Review

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Review Article

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ABSTRACT

Temporomandibular disorders (TMDs) are a collective term that embraces a number of clinical conditions that involve the masticatory musculature and/or temporomandibular (TM) joints and associated structures. Temporomandibular disorders (TMD) are examined from a biopsychosocial or illness perspective. It is considered that TMD share features with many common chronic pain conditions. Functional disturbances of the masticatory system can be as complicated as the system itself. Although numerous treatments have been advocated, the complex nature of TMD requires a multidisciplinary team.

Keywords: TMD, TMJ, Joint, Pain.

Introduction

The term "temporomandibular disorders" (TMD), is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ) and associated structures, or both. These disorders are characterized by facial pain in the region of the TMJ and/or the muscles of mastication, limitation or deviation in the mandibular range of motion, and TMJ sounds during jaw movement and function. The cause of most TMD remains unknown although numerous hypotheses have been proposed. The relationship of occlusal disharmony and TMD became a focus after Costen reported that a group of patients with multiple complaints around the jaws and ears improved after their occlusal-vertical dimension was altered. The occlusal hypothesis was then expanded to include other occlusal discrepancies in addition to the loss of vertical dimension. During the 1950s and 1960s, a muscular cause not directly related to occlusion was proposed. In the late 1970s, advances in diagnostic imaging resulted in a better understanding of the intracapsular problems associated with TMD. The lack of a clear understanding with regard to a cause, the existence of multiple hypotheses, and strongly held beliefs by some clinicians have resulted in a wide spectrum of treatments being offered.[1]

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Temporomandibular Disorders: In the past, disorders of the masticatory system were generally treated as one condition or syndrome, with no attempt to differentiate subtypes of muscle and joint disorders.[2] With increasing understanding, the ability to identify different muscle and joint disorders has become possible; this should lead to a better understanding of the natural course, more accurate predictions of prognosis, and more effective treatments. The term temporomandibular disorders (TMD) is a collective term embracing a number of clinical problems that involve the masticatory muscles, the temporomandibular joints and associated structures, or both.[3]

The term temporomandibular dysfunction has been used to define conditions involving changes to the structure and/or function of the masticatory system (TMJ and associated muscle-skeletal structures).[4]

These disorders are characterized by

- Facial pain in the region of the TMJs and/or muscles of mastication
- Limitation or deviation in mandibular movements and
- TMJ sounds during jaw movement and function

Etiology: The contributing factors and actual underlying etiology for temporomandibular joint disorders and myofascial pain (MFP) has been the subject of debate. The etiology of temporomandibular disorders (TMDs) is multidimensional. Biomechanical, neuromuscular, biopsychosocial, and neurobiological factors may contribute to the disorder.[5] These factors are classified as predisposing (structural, metabolic and/or psychologic conditions), initiating (e.g. trauma or repetitive adverse loading of the masticatory system) and aggravating (parafunction, hormonal, or psychosocial factors) to emphasize their role in the progression of TMD.[6]

Possible risk factors contributing to TMJ pathologies:
Oral K et al[7] enumerated the following as the possible risk factors for TM disorders:

- Occlusal factors
- Parafunction (bruxism)
- Trauma
- Hypermobility
- Stress
- Personality
- Age
- Gender
- Heredity
- Systemic diseases

Occlusal Factors: Occlusal factors and their association and contribution to TMD have been and continue to be the subject of an intense discussion within the dental community. For example, reverse articulation is thought to lead to an asymmetric muscular function, but whether or not it is directly related to TMD has not yet been established. The presence of a large horizontal or vertical overlap is a source of contrasting opinions.[8,9] A large slide between centric relation and maximum intercuspation seems to be weakly associated with some forms of TMD. The presence of mediotrusion interferences is considered a predisposing factor by some authors[11,12] while others have suggested that such interferences may act as a protective factor.[13] Some authors state that the anterior open occlusal relationship may be a consequence of articular remodeling rather than a predisposing factor for TMD; others emphasize open bite as the predisposing factor. The controversies existing in the literature represent a serious limitation for the clinicians treating these disorders. In recent

years, the acceptance of theories about the multifactorial etiology of TMD has resulted in less emphasis being placed on occlusion as a TMD-related factor.[10] Bruxism: Bruxism has also been suggested as an initiating or perpetuating factor for TMD. It may constitute a risk or an etiologic factor for myofascial tenderness, the pain of the masticatory muscles, and pain from the TMJ. In a study evaluating the effect of bruxism, taking into account the effect of clenching only, grinding only, and clenching combined with grinding, the authors concluded that there was no association between chronic MFP and grinding only, but rather with clenching-grinding.[14] The association between bruxism and TMD signs supports the theory that repetitive adverse loading of the masticatory system may cause functional disturbances.[15] Because abnormal joint loading is a major causative factor in cartilage degradation, biochemical and biomechanical abnormalities, and intraarticular temporomandibular pathology, clinicians must identify and address parafunctional masticatory activity during nonsurgical, surgical, and post-surgical treatment regimens.[16] Trauma: The role of trauma in the etiology of TMDs is also controversial. Whiplash injury to the head or neck is often considered a significant risk factor for the development of TMDs, and has been proposed to produce internal derangements of the TMJ.[17] Endotracheal intubation has also been proposed as a risk factor for TMJ dysfunction. Stress and personality: The role of stress and personality in the etiology of temporomandibular pain dysfunction syndrome has undergone extensive scrutiny. Psychological studies have shown that patients with functional disorders of the temporomandibular region have similar psychological profiles and psychological dysfunction as other chronic musculoskeletal pain disorders, such as tension-type headache and back or arthritic pain.[18] Even though studies have indicated the role of stress in the etiology of TMD, the issue of whether psychological factors cause TMD or reflect the impact of TMD on the person remains unknown. The relationships between psychological aspects and parafunction have been emphasized in many studies. Primarily, psychological factors affect TMD symptoms more indirectly than directly. The overall level of anxiety and/or depression could modify the clenching and grinding habits.[14] Gender: Females present a greater risk of chronic MFP and may present with characteristics (e.g. hormonal, constitutional factors, behavioral or psychosocial differences) that contribute to chronic TMD. Even though the lower prevalence of pain conditions in TMJ dysfunction in men has not yet been clarified, the reduction in TMJ pain with testosterone at supraphysiological serum levels can be helpful in explaining this gender difference.[20] mobility: The relationship between hypermobility and TMD has also been examined. An association was determined between loose joint syndrome and TMJ symptoms. Kavuncu et al.[21] found that both local and general hypermobility are more frequently detected in patients with TMD than in the controls and that the risk of TMJ dysfunction is greater if the patient presents both alterations simultaneously; they concluded that both situations may play a role in the etiology of TMD. Heredity and age: Michalowicz et al.[22] evaluated the hypothesis that signs and symptoms of TMD may be hereditary, but in a recent study the authors concluded that genetic factors and the family environment exert no relevant effect upon the presence of symptoms and signs of the TMJ. Age is also not associated with the risk of MFP.

TMD is accepted to be a multifactorial problem that requires a comprehensive examination. One has to be cautious while examining patients and acquiring medical history in order to clarify the picture and clearly address the problem.

Examination Temporomandibular Joint

Clinical Evaluation History: The main complaint may include orofacial pain, joint noises, restricted mouth opening, or a combination of these in addition to other less specific problems such as headache and tinnitus. Pain should be evaluated carefully in terms of onset, nature, intensity, size, duration, aggravating and relieving factors, and especially how it relates to other features such as joint noise and restricted mandibular movements. More specifically, pain is centred immediately in front of the tragus of the ear and projects to the

ear, temple cheek and along the mandible is highly diagnostic for Temporomandibular disorders. The pain may be accompanied by a click or grating sound in the preauricular region during mandibular functions such as chewing or yawning. A history of limited mouth opening, which may be intermittent or progressive, is also a key feature for TMD.[23]

Chronic head, neck, and back pain; irritable bowel syndrome; and idiopathic pruritis are sometimes found in patients with TMD and should be sought by the doctor to help establish the possibility of psychogenic cause. The doctor should ask the patient also about underlying influences such as stress, anxiety, depression, or important life events so that he or she can get a clearer picture of any psychogenic basis for the disorders. In general, the longer the duration of symptoms and the greater the number of treatments, the smaller the likelihood that the patient will respond well to further treatment.[23]

Clinical Examination: Begin by inspecting the preauricular area for swelling or erythema. Palpate directly over the joints when the patient opens and closes the mandible, and the extent of mandibular condylar movements can be assessed. Normally condylar movement is easily felt. Have the patient close slowly and you will feel the condyle move posteriorly against your finger. Tenderness elicited by this maneuver is invariably associated with auricular inflammation. Palpate the superficial temporal artery for nodularity and tenderness.[24]

Palpate the masticatory and cervical muscles and search for areas of tenderness or sustained contraction. Begin with the Sternocleidomastoid, Trapezius, and posterior cervical muscles. Palpate the masseter at is an attachment with the zygomatic arch and angle of the mandible, the temporalis both in the temporal fossa and intraorally along the ascending ramus of the mandible, and the medial pterygoid bimanually, placing one finger externally at the medial aspect of the angle of the mandible and the other finger orally in the lingual vestibule in the retromolar region. The lateral pterygoid is accessible to the examining finger intraorally posterior to the maxillary tuberosity. Areas of identified muscle spasm or tenderness can be injected with local anaesthesia, 2% lidocaine or 0.5% bupivacaine without adrenaline, or can be sprayed with fluoromethane or ethyl chloride to determine whether these are the cause of the patient's symptoms.[24]

Examine the external auditory canal and tympanic membrane with an otoscope and do a tuning fork test to rule out ear pathology, particularly important in patients who complaint of hearing changes in association with TMJ symptoms. An audiogram may be indicated if hearing loss is suspected.[24]

The joint is auscultated during mandibular movements. The normal joint functions relatively quietly. Listen for crepitus or grinding or clicking or popping sound.[24]

The degree of mandibular opening is measured using the distance between the incisal edges of upper and lower anterior teeth. Opening of less than 35 mm is considered abnormal in an adult. There is no upper limit of normal, but a few patients can exceed 60 mm comfortably. Observe the opening pattern for deviation. The mandible often deviates to the affected side during opening because of muscle spasm or mechanical locking by a displaced meniscus.[24]

Examine the hands for signs of systemic diseases (eg. Heberden's nodes for osteoarthritis, the ulnar deviation for rheumatoid arthritis), which may also involve the TMJ. Laboratory tests (complete blood count, erythrocyte sedimentation rate, rheumatoid factor, antinuclear antibody, serum uric acid) are helpful when a systemic

cause for TMJ disorder is suspected. Radiographs provide useful information about the bony architecture of the joint and movement of the condyles during the mandibular opening.[24]

TMJ Imaging: TMJ dysfunction is the most common jaw disorder with 28-86% of adults and adolescents showing one or more clinical signs and symptoms. In most cases, clinical signs and symptoms are transient and treatment is not indicated.

However, a small (5%) suffer from severe dysfunction which requires thorough diagnostic workup including imaging. Purposes of TMJ imaging include:

- Evaluation of the integrity and relationship of TMJ hard and soft tissues
- Confirmation of the extent or stage of disease progression
- Evaluation of treatment effects[25, 26]

A clinician must correlate imaging information with patient history and clinical findings to arrive at the final diagnosis.

Historical Perspectives: Surgeons were the pioneers of TMJ roentgenography in the early 1900s. Until TMJ tomography was introduced in 1939, TMJ imaging mainly involved searching for the most effective transcranial projection.

In 1940s, arthrography was shown to be effective for soft tissue disorders.
The first TMJ study using CT was published in 1980 and later the emergence of MRI paralleled the decline of CT.

Direct arthroscopic examination of TMJ was developed in Japan in the late 1970s
Classification: For years, classifying TMDs has been a confusing issue.

Syndrome Concept: A notion has persisted for half a century that dysfunctions of the masticatory apparatus comprise a 'syndrome' – initially referred to as Costen's syndrome or the Temporomandibular syndrome. In 1956 Schwartz introduced the TM pain dysfunction syndrome.[27] Then came to the myofascial pain dysfunction syndrome in 1969.[28] The term craniomandibular syndrome is now used quite frequently.

Developing a Useful Classification: The first serious attempt to classify TM disorders in the dental literature was in 1970.[29] This divided the disorders into six groups, as follows:

1. Spontaneous dislocation
2. Traumatic joint
3. Masticatory pain-dysfunction syndrome
4. Temporomandibular arthritis
5. Chronic mandibular hypomobilities
6. Developmental anomalies and neoplasms

This classification entailed implications of aetiology which, of course, required diagnostic differentiation. Being dependent on diagnosis, it could not become a very useful diagnostic tool.

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In 1980, the American Academy of Craniomandibular disorders proposed an elaborate classification of masticatory disorders that has been well received.[30,31] It, however, is complex and is not based on symptomatology.

Classification system for diagnosing Temporomandibular disorders by Clark GT et al[32:]

Diagnostic Criteria	Diagnoses
Muscle and Facial Disorders	Myalgia; muscle contracture; splinting; hypertrophy; spasm; dyskinesia; forceful jaw closure habit; myositis (bruxism)
TMJ disorders	Disk condyle incoordination; osteoarthritis; disk condyle restriction; inflammatory polyarthritis; open dislocation; traumatic articular disease; arthralgia
Disorder of Mandibular Mobility	Ankylosis; adhesions (intracapsular); fibrosis of muscular tissue; coronoid elongation –hypermobility of TMJ
Disorders of Maxillomandibular Growth	Masticatory muscle hypertrophy/atrophy; neoplasia (muscle, maxillomandibular or condylar); maxillomandibular or condylar hypoplasia/hyperplasia

Table 1

Classifying by Clinical Symptoms: Weldon Bell[28] presented a classification that logically categorized these disorders, and the American Dental Association[33] adopted it with few changes. It has, in fact become a road map helping clinicians toward a precise and well-defined diagnosis.

The symptoms (including both subjective and objective evidence) that designate a masticatory disorder are:

1. Masticatory pain
2. Restricted range of motion
3. Interference during mandibular movements
4. Acute malocclusion

By utilizing these four cardinal symptoms, TM disorders can be classified as one or more of five categories by Bell.[33]

I. Masticatory muscle disorders

A. Protective muscle splinting
B. Masticatory myospasm
   1. Elevator muscle spasm
   2. Lateral pterygoid muscle spasm
C. Masticatory myositis

II. Disc-interference disorders

A. Class I interference
B. Class II interference
C. Class III interference

1. Excessive passive interarticular pressure
2. Structural irregularity
3. Non inflammatory degenerative joint disease
4. Internal derangement
   a. Disc-condyle adhesions
   b. Damaged articular disc
   c. Displaced articular disc
   d. Detached superior retrodiscal lamina

D. Class IV interference (hypermobile subluxation)
E. Class V interference (spontaneous anterior dislocation)

III. Inflammatory disorders

A. Synovitis and capsulitis
B. Retrodiscitis
C. Inflammatory arthritis
1. Degenerative arthritis
2. Traumatic arthritis
3. Infectious arthritis
4. Rheumatoid arthritis
5. Hyperuricemia

IV. Chronic mandibular hypomobilities

A. Psuedoankylosis
B. Contractured elevator muscle
1. Myostatic contracture
2. Myofibrotic contracture

C. Ankylosis
1. Fibrous ankylosis
2. Osseous ankylosis
3. Total fixation of the jaw

V. Growth disorders

A. Aberration of development
B. Acquired change in structure
C. Neoplasia
1. Benign tumour
2. Malignant tumour

Okeson presented a basic classification of TMD developed by Bell but incorporated some additional modifications. It begins by separating all TMDs into four broad categories as follows:

The Classification system used for diagnosing Temporomandibular disorders by Okeson[21]:

I. Masticatory Muscle Disorders

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

II. Temporomandibular Joint Disorders

1. Derrangement of condyle-disc complex

a. Disc displacement

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. Osteoarthrititis

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 impedence

VI. 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

Conclusion: Over 70 years since the ENT physician James B. Costen presented a syndrome of impaired hearing, sensations to the ears, dull pain within and about the ear, and other symptoms to a meeting of the Ophthalmological and Otolyaryngological Society in Dallas, the etiology, the diagnosis, and the therapy of temporomandibular disorders is still debated. Especially the etiology of the temporomandibular disorder is not well known and several widely diverging concepts exist. The situation affects substantially the way we diagnose and treat temporomandibular disorder patients. Ideally, treatment strategies would be applied to etiologic disease entities to target a homogeneous patient population. In the absence of such information, as it is currently the situation, treatment must be symptom-oriented and as conservative as possible.

References

1. Sharawy M. Developmental and clinical anatomy and physiology of the temporomandibular joint. In: Bays RA, Quinn PD, editors. Fonseca Oral and maxillofacial surgery-Temporomandibular disorders Volume 4. 1st ed. Philadelphia: W.B. Saunders company; 2000.
2. Blasberg B, Greenberg MS. Temporomandibular disorders. In: Greenberg MS, Glick M, Ship JA, editors. Burket's Oral Medicine. 11th ed. Hamilton: BC Decker Inc; 2008.
3. McNeill C. History and evolution of TMD concepts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997 Jan; 83(1):51-60. doi: https://doi.org/10.1016/S1079-2104(97)90091-3
4. Bevilaqua-Grossi D, Chaves TC, de Oliveira AS, Monteiro-Pedro V. Anamnestic index severity and signs and symptoms of TMD. Cranio. 2006 Apr; 24(2):112-8. doi: https://doi.org/10.1179/crn.2006.018
5. Suvinen TI, Reade PC, Hanes KR, Könönen M, Kemppainen P. Temporomandibular disorder subtypes according to self-reported physical and psychosocial variables in female patients: a re-evaluation. J Oral Rehabil. 2005 Mar; 32(3):166-73. doi: https://doi.org/10.1111/j.1365-2842.2004.01432.x
6. McNeill C. Evidence-based TMD guidelines. J Orofac Pain. 1997 Spring; 11(2):93. https://pubmed.ncbi.nlm.nih.gov/10332314/
7. Oral K, Bal Küçük B, Ebeoğlu B, Dinçer S. Etiology of temporomandibular disorder pain. Agri. 2009 Jul; 21(3):89-94. https://pubmed.ncbi.nlm.nih.gov/19779999/
8. John MT, Hirsch C, Drangsholt MT, Mancl LA, Setz JM. Overbite and overjet are not related to self-report of temporomandibular disorder symptoms. J Dent Res 2002; 81:164-9. https://pubmed.ncbi.nlm.nih.gov/11876269/
9. Seligman DA, Pullinger AG. The degree to which dental attrition in modern society is a function of age and of canine contact. J Orofac Pain 1995; 9:266-75. https://pubmed.ncbi.nlm.nih.gov/8995926/
10. McNamara JA Jr, Seligman DA, Okeson JP. Occlusion, Orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain 1995; 9:73-90. https://pubmed.ncbi.nlm.nih.gov/7581209/
11. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. J Prosthet Dent. 1992 May; 67(5):692-6. doi: https://www.thejpd.org/article/0022-3913(92)90173-8/pdf
12. Shiau YY, Chang C. An epidemiological study of temporomandibular disorders in university students of Taiwan. Community Dent Oral Epidemiol. 1992 Feb; 20(1):43-7. doi: https://doi.org/10.1111/j.1600-0528.1992.tb00672.x

13. Minagi S, Ohtsuki H, Sato T, Ishii A. Effect of balancing-side occlusion on the ipsilateral TMJ dynamics under clenching. J Oral Rehabil 1997; 24:57-62. https://doi.org/10.1046/j.1365-2842.1997.00431.x
14. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: a case-control study. Pain. 2003 Aug; 104(3):491-9. doi: https://doi.org/10.1016/s0304-3959(03)00074-5
15. Schierz O, John MT, Schroeder E, Lobbezoo F. Association between anterior tooth wear and temporomandibular disorder pain in a German population. J Prosthet Dent. 2007 May; 97(5):305-9. doi: https://doi.org/10.1016/j.prosdent.2007.03.006
16. Israel HA, Scrivani SJ. The interdisciplinary approach to oral, facial and head pain. J Am Dent Assoc 2000; 131:919-26. https://doi.org/10.14219/jada.archive.2000.0310
17. Schiffman EL, Fricton JR, Haley D. The relationship of occlusion, parafunctional habits and recent life events to mandibular dysfunction in a non-patient population. J Oral Rehabil. 1992 May; 19(3):201-23. doi: https://doi.org/10.1111/j.1365-2842.1992.tb01095.x
18. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? J Prosthet Dent. 1994 Jul; 72(1):29-38. doi: https://doi.org/10.1016/0022-3913(94)90213-5
19. Johansson A, Unell L, Carlsson GE, Söderfeldt B, Halling A. Gender difference in symptoms related to temporomandibular disorders in a population of 50-year-old subjects. J Orofac Pain 2003; 17:29-35. PMID: https://pubmed.ncbi.nlm.nih.gov/12756928/
20. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007 May; 8(5):437-42. doi: https://doi.org/10.1016/j.jpain.2006.12.007
21. Kavuncu V, Sahin S, Kamanli A, Karan A, Aksoy C. The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome. Rheumatol Int. 2006 Jan; 26(3):257-60. doi: https://doi.org/10.1007/s00296-005-0620-z
22. Michalowicz BS, Pihlstrom BL, Hodges JS, Bouchard TJ Jr. No heritability of temporomandibular joint signs and symptoms. J Dent Res 2000; 79:1573-8. https://doi.org/10.1177/00220345000790080801
23. Dimitroulis G. Temporomandibular disorders: a clinical update. BMJ. 1998 Jul 18; 317(7152):190-4. doi: https://doi.org/10.1136/bmj.317.7152.190
24. Meyer RA. The Temporomandibular Joint Examination. In: Walker HK, Hall WD, Hurst JW, editors. Clinical Methods: The History, Physical, and Laboratory Examinations. 3rd edition. Boston: Butterworths; 1990. Chapter 163. https://pubmed.ncbi.nlm.nih.gov/21250114/
25. Lewis EL, Dolwick MF, Abramowicz S, Reeder SL. Contemporary imaging of the temporomandibular joint. Dent Clin North Am. 2008 Oct; 52(4):875-90, https://doi.org/10.1016/j.cden.2008.06.001
26. Brooks SL, Brand JW, Gibbs SJ, Hollender L, Lurie AG, Omnell KA, Westesson PL, White SC. Imaging of the temporomandibular joint: a position paper of the American Academy of Oral and Maxillofacial Radiology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997 May; 83(5):609-18. https://doi.org/10.1016/s1079-2104(97)90128-1
27. SCHWARTZ LL. A temporomandibular joint pain-dysfunction syndrome. J Chronic Dis. 1956 Mar; 3(3):284-93. doi: https://doi.org/10.1016/0021-9681(56)90123-0
28. Bell WE. Temporomandibular disorders. 3rd ed. Chicago: Year Book Medical Publishers, Inc; 1990.

29. Bell WE. Management of temporomandibular joint problems. In: Goldman HM, Gilmore HW, Royer RQ et al, editors. Current therapy in dentistry. Vol 4. St Louis: CV Mosby Co; 1970, 398-415.
30. McNeill C, Danzig WM, Farrar WB, et al. Craniomandibular (TMJ) disorders: The state of art. J Prosthet Dent. 1980; 44: 434.
31. McNeill C. Craniomandibular (TMJ) disorders: The state of art. Part II: Accepted diagnostic and treatment modalities. J Prosthet Dent. 1983; 49: 393-397. https://doi.org/10.1016/0022-3913(83)90284-6
32. Clark GT, Seligman DA, Solberg WK, Pullinger AG. Guidelines for the examination and diagnosis of temporomandibular disorders. J Craniomandib Disord 1989; 3:7-14. https://pubmed.ncbi.nlm.nih.gov/2606995/
33. Report of the president's conference on the examination, diagnosis, and management of temporomandibular disorders. J Am Dent Assoc. 1983 Jan; 106(1):75-7. doi: https://doi.org/10.14219/jada.archive.1983.0020

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