

Etiological factors of temporomandibular joint disorders

Department of Oral Surgery,
TMDCRC, Moradabad,
UP, India

Shalender Sharma, D. S. Gupta, U. S. Pal, Sunit Kumar Jurel

ABSTRACT

The temporomandibular joint receives its name from the two bones that enter into its formation, namely the temporal bone and the mandible. This complex synovial system is composed of two temporomandibular joints together with their articulating ligaments and masticatory muscles. This articulation affects other synovial joints that relate specifically to masticatory function. The causes of temporomandibular disorders are complex and multifactorial. There are numerous factors that can contribute to temporomandibular disorders. In some instances a single factor may serve one or all of these roles. Iatrogenic injuries can act as both initiating as well as predisposing factors. The term craniomandibular disorder is used synonymously with the term temporomandibular disorders and is considered a major cause of nondental pain in the orofacial pain region. The successful management of temporomandibular disorders is dependent on identifying and controlling the contributing factors. The temporomandibular disorders are more common in females, the reason is not clearly known. The following article provides detailed information regarding temporomandibular joint disorders.

Address for correspondence:

Dr. Shalender Sharma,
Department of Oral Surgery,
TMDCRC, Moradabad,
UP-133 203, India.
E-mail: sharma.shalender@
rediffmail.com

Key words: Articulation, pain, temporomandibular joint

INTRODUCTION

Temporomandibular joint and muscle disorders are a group of conditions that cause pain and dysfunction in the jaw joint and the muscles that control jaw movement. The movement of the mandible needs coordination between them to maximize function and minimize the damage to surrounding structures.^[1] A rather unique feature of temporomandibular joint articulation is that it has two joints. The articular disc between the condyle and the temporal bone serves to separate the structures into two separate joint cavities. In the inferior joint between the head of the mandibular condyle and the articular disc the movement is almost totally of a rotary or hinge type whereas in the superior

joint between the temporal bone and the articular disc the movement is gliding or translatory.^[2]

The origin and homologies of the jaws in all vertebrates were clearly outlined in the last century by Gegenbauer,^[3] later a definitive statement of this was made by Reichert in 1837 known as the Reichert Theory.^[4] Fawcett^[5] 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. 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.

It is evident from the numerous epidemiologic studies on the occurrence of temporomandibular disorders in the general population that there are a number of consistent findings. Firstly, signs of temporomandibular disorders appear in about 60–70% of the general population and yet only about one in four people with signs are actually aware of or report any symptoms.^[6]

Access this article online	
Quick Response Code: 	Website: www.njms.in
	DOI: 10.4103/0975-5950.94463

The frequency of severe disorders that are accompanied by headache and facial pain and that are characterized by urgent need of treatment is 1–2% in children, about 5% in adolescents and 5–12% in adults.^[7]

Another consistent finding is that among those who seek treatment for temporomandibular disorders, by far the greatest majority are females, outnumbering males by at least four to one. It is not clear why this is so since it is suspected that temporomandibular disorders affect both males and females in almost equal numbers in the general population. Severe problems are much more common among women in clinical populations, and the ratio between women and men who seeks treatment for TMJ disorder is 8:1.^[8]

During the first half of the 20th century, displacement of the temporomandibular joint meniscus, with attending clinical signs and symptoms including pain and popping, were described. In 1934, James Costen^[9] described a group of symptoms that were centered on the ear and temporomandibular joint. Because of his work the term “Costen’s syndrome” was developed.

In 1947, Nogaard^[10] used arthrographic techniques to demonstrate anterior displacement of the articular disk in clicking or popping temporomandibular joint. During the 1950s, Schwartz^[11] coined the term “Temporomandibular joint pain dysfunction syndrome”. Later came the term the “Functional temporomandibular joint disturbances” coined by Ash and Ramford.^[12]

Some terms described the suggested etiologic factors, such as occlusomandibular disturbance and myoarthropathy of the temporomandibular joint, others stressed pain dysfunction syndrome, myofascial pain dysfunction syndrome and temporomandibular pain dysfunction syndrome. Subsequently, as a result of clinical observation and a variety of research studies, Laskin^[13] proposed the term “Myofascial Pain Dysfunction Syndrome (MPDS)”. He attributed the features of pain, joint sounds and limited mandibular movement to multiple causes and provided experimental evidence for the concept of a psychophysiological disorder.

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^[14] suggested the term temporomandibular disorders. 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. Fascialarthromyalgia, mandibular dysfunction, myofascial pain, masticatory myalgia syndrome and primary myalgia affecting the masticatory musculature are also used synonymously.

ETIOLOGICAL FACTORS

The causes of temporomandibular disorders^[7] are complex and multifactorial. There are numerous factors that can contribute to temporomandibular disorders. Factors that increase the risk of temporomandibular disorders are called “*Predisposing factors*” and those causing the onset of temporomandibular disorders are called “*Initiating factors*” and factors that interfere with healing or enhance the progression of temporomandibular disorder are called “*Perpetuating factors.*”

In some instances a single factor may serve one or all of these roles. The successful management of temporomandibular disorders is dependent on identifying and controlling the contributing factors which include occlusal abnormalities, orthodontic treatment, bruxism and orthopedic instability, macrotrauma and microtrauma, factors like poor health and nutrition, joint laxity and exogenous estrogen.^[15] Psychosocial factors like stress, tension, anxiety and depression may lead to temporomandibular joint disorders.^[16] A case controlled study conducted in a dental school used clinical and neurophysiologic evaluations to examine the role of sleep dysfunction and depression alone or in combination with temporomandibular joint disorders. The resulting analysis demonstrated that depression was significantly more common in the temporomandibular joint disorder group than in the control group.^[17]

Occlusion is the first and probably the most discussed etiologic factor of temporomandibular disorders. Costen^[9] concluded that overclosure was the cause of symptoms in temporomandibular disorders. Because of this reason he and other contemporary dentists adopted bite raising dental procedures as the treatment for temporomandibular disorder, which however failed to give expected relief to the patients. The role of occlusion in the development of temporomandibular joint disorders is controversial. Today its role is widely considered as contributing by initiating, perpetuating or predisposing of temporomandibular joint disorders.^[18] Initiating factors lead to the onset of the symptoms and are related primarily to trauma or adverse loading of the masticatory system. In the perpetuating factors the following may be included:

- a. Behavioral factors (grinding, clenching and abnormal head posture)

- b. Social factors (could effect perception and influence of learned response to pain)
- c. Emotional factors (depression and anxiety)
- d. Cognitive factors (negative thoughts and attitudes which can make resolution of the illness more difficult).

Predisposing factors are pathophysiologic, psychological or structural processes that alter the masticatory system sufficiently to increase the risk of development of temporomandibular disorders. Pullinger, Seligman and Gornbein^[19] applied multiple factor analysis, which indicated the low correlation of occlusion to temporomandibular disorders. However, the following occlusal factors had a slight relation:

- a. Open bite
- b. Overjet greater than 6-7 mm
- c. Retruded contact position/intercuspal position with sliding greater than 4 mm
- d. Unilateral lingual cross-bite
- e. Five or more missing posterior teeth
- f. Faulty restorations and ill-fitting prosthesis.

With regards to the distribution of occlusal contacts, the symmetry of their intensity rather than the symmetry of their number in the posterior occlusion seemed to be more important in relation to temporomandibular function.^[20] Increased number and more frequent headaches in individuals with few occlusal contacts have also been noted.^[21]

Pullinger and Seligman^[22] further estimated that occlusal factors contribute about 10–20% to the total spectrum of etiological factors which differentiates between healthy persons and patients with temporomandibular joint disorders.

Decreased disk: eminence ratio (antero-posterior length of the disk in relation to the length of the articular eminence) is associated with advanced stages of temporomandibular joint internal derangement. In a patient with a flat eminence there is a minimum amount of posterior rotation of the disk on the condyle during opening. As the steepness increases, more rotational movement is required between the disk and condyle during translation of the condyle. Therefore patients with steep articular eminences are more likely to demonstrate greater condyle-disk movement during function. This exaggerated condyle-disk movement may increase the risk of ligament elongation that leads to disk derangement disorders. Perhaps this predisposing factor is only significant when combined with other factors that relate to the amount of joint function and loading.^[23]

Iatrogenic injuries can act as both initiating as well as predisposing factors. This can occur during

Table 1: Examination of temporomandibular joint

Examination protocol

Is it difficult or painful to open the mouth (e.g., yawning)?
Does the jaw get stuck, locked, or go out?
Is it difficult or painful to chew and talk?
Do the jaw joints make sounds?
Do the jaws often feel stiffness and muscular tiredness?
Are headaches, neck aches or toothaches frequent?
Have there been any recent changes in bite? (orthodontic or prosthodontic treatment)
Has there been previous treatment for any unexplained facial pain or a jaw joint Problem?
Is there any history of trauma?

Table 2: Examination of temporomandibular joint

Clinical findings

The following things are evaluated during examination of the temporomandibular joint:
Functional movement of mandible and temporomandibular joint with the jaws separated (range of motion).
The anteroposterior relationship of the mandible and the maxilla.
The symmetry of the jaws.
The presence of clicking and snapping of the joints (auscultation).
The presence of swelling and tenderness of the jaws (tenderness to palpation).

any dental procedure in which there is prolonged opening like orthodontic treatment, single-sitting root canal treatment or because of factors like relapse which causes a functional imbalance between the temporomandibular joints, muscles and occlusion. To avoid this type of damage to the temporomandibular joint, we should always perform an examination of the joint [Tables 1 and 2].^[7] All the principles of physical examination are used. Inspection begins with the operator standing directly in front of the patient. Palpation of the muscles and joints are best accomplished with the operator standing behind the patient. Bilateral palpation is the method of choice, since movements of the joints demand contralateral action of the joints and muscles. The examiner feels for smoothness in the function of the joints. Auscultation of the joints can be accomplished simply by listening for any abnormal sounds of snapping, grating or clicking that may occur during the movements. A stethoscope may be of value to one trained in its use for temporomandibular joint sounds. Percussion of the jaws may be of value when cavities, fractures or reflexive movements of the mandible are to be evaluated. Percussion should be indirect when testing for cavities and fractures of the bone and direct when testing for reflex action of the mandible.

SUMMARY

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.

REFERENCES

- Okeson JP. Treatment of temporomandibular joint disorders. In: Management of temporomandibular disorders and occlusion. 5th ed. St. Louis: Mosby; 2003. p. 413-35.
- Okeson JP. Orthodontic therapy and patient with temporomandibular disorder. In: Graber, Vanasdall, Vig, editors. Orthodontics current principles and techniques. 4th ed. St. Louis; Mosby; 2009. p. 331-44.
- Gegenbauer, Müller, Gerd B, Newman SA. Homology: Origination of Organismal Form. Beyond the Gene in Developmental and Evolutionary Biology. A Bradford Book. Cambridge, Massachusetts, London, England: The MIT Press; 2003. p. 51-6.
- Reichert Theory: Available from: http://en.wikipedia.org/wiki/Evolution_of_mammalian_auditory_ossicles#Reichert_Gaupp_theory. [cited in 2010].
- Fawcett E. The development of the bones around the Mouth. Great Britain Dental Board 1924;1:23.
- Graber, Rakosi, Petrovic. Functional analysis- examination of temporomandibular joint and condylar movement. In: Dentofacial Orthopedics with Functional Appliances. 2nd ed. St. Louis: Mosby; 2009. p. 135-40.
- Athanasίου AE. Orthodontics and craniomandibular disorders. In: Samire, Bishara. Textbook of orthodontics. 2nd ed. Philadelphia: Saunders; 2003. p. 478-93.
- Rugh JD, Solberg WK. Oral health status in the United states: temporomandibular disorders. J Dent Educ 1985;49:398-405.
- Costen JB. Syndrome of ear and sinus symptoms dependent upon functions of the temporomandibular joint. Ann Otol Rhino Laryngol 1934;3:1-4.
- Katzberg R, Dolwick M, Bales D, and Helms C. Arthrotomography of the temporomandibular Joint: New technique and preliminary observations. MR 1979;132:949-55.
- Schwartz RA, Greene CS, Laskin DM. Personality characteristics of patients with Myofascial Pain-Dysfunction (MPD) Syndrome unresponsive to conventional therapy. J Dent Res 1979;58:1435-9.
- Ash MM, Ram fjord SP. Occlusion. 3rd ed. Philadelphia: WB Saunders; 1995. p. 181-92.
- Sarnat BG, Laskin DM. The Temporomandibular Joint: A biological basis for clinical practice. 4th ed. Philadelphia: WB Saunders; 1992. p. 163-78.
- Okeson JP, Bell WE. Bell's Orofacial Pains. 5th ed by ISBN: 0867152931; Chicago: Quintessence Publishing (IL), 1995. p. 348-55.
- Gage JP. Collagen biosynthesis related to temporomandibular joint clicking in childhood. J Prosthet Dent 1985;53:714-7.
- Irby WB, Baldwin KH. Emergencies and urgent complications in Dentistry. St. Louis: The C. V. Mosby Company; 1965. p. 167-73.
- Donovan TE, Becker W, Brodine AH, Burgess JO, Cronin RJ, Summitt JB. Annual review of selected dental literature: Report of the Committee on Scientific Investigation of the American Academy of Restorative Dentistry: J Prosthet Dent 2007;98:36-67.
- McNeill C. Craniomandibular disorders: Guidelines for evaluation, diagnosis and management, Chicago: Quintessence; 1990. p. 25-39.
- Pullinger AG, Seligman DA, Gornbein JA. A multiple regression analysis of risk and relative odds of temporomandibular disorders as a function of common occlusal features. J Dent Res 1993;72:968-79.
- Gianniri AI, Melsen B, Nielsen L, Athanasίου AE. Occlusal contacts in maximum intercuspation and craniomandibular dysfunction in 16 to 17 year old adolescents. J Oral Rehabil 1991;18:49-59.
- Wanmam A, Agerberg G. Etiology of craniomandibular disorders: Evaluation of some occlusal and psychological factors in 19 year olds. J CraniomandibDisord 1991;5:35-44.
- Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. J Prosthet Dent 2000;83:66-75.
- Parker MW. A dynamic model of etiology in temporomandibular disorders. J Am Dent Assoc 1990;120:283-90.

How to cite this article: Sharma S, Gupta DS, Pal US, Jurel SK. Etiological factors of temporomandibular joint disorders. Natl J Maxillofac Surg 2011;2:116-9.

Source of Support: Nil. **Conflict of Interest:** None declared.