Clyde D. Hillier

Temporomandibular Joint Dysfunction: A Dental Overview

SUMMARY

Temporomandibular joint dysfunction is common and often acutely painful. Because of the large and diverse symptom complex created by this disorder, patients frequently first seek relief from their physician rather than their dentist. In this article temporomandibular joint (TMJ) dysfunction is defined and the presenting signs and symptoms are discussed. Their etiology is described in relation to the anatomy of the temporomandibular joint. Examination techniques can help in the differential diagnosis. Current treatment ranges from heat, local anesthesia and ultrasound to anxiolytics, transcutaneous nerve stimulation and nutritional supplementation. (Can Fam Physician 1985; 31:549-555)

SOMMAIRE

La dysfonction de l’articulation temporo-mandibulaire est fréquente et souvent très douloureuse. À cause de la complexité et de la diversité des symptômes engendrés par ce désordre, les patients vont souvent, dans un premier temps, consulter leur médecin de famille plutôt que leur dentiste. Dans cet article, on définit la dysfonction de l’articulation temporo-mandibulaire et on discute les signes et symptômes de présentation. On décrit leur étiologie en fonction de l’anatomie de l’articulation temporo-mandibulaire. Les techniques de l’examen peuvent aider à établir le diagnostic différentiel. Les traitements actuels vont de la chaleur, l’anesthésie locale et les ultrasons aux anxiolytiques, la stimulation nerveuse par voie transcutanée et l’administration d’un supplément nutritif.

Key words: temporomandibular joint, pain, headache

Dr. Hillier practices general dentistry in London, Ontario, and has a special interest in TMJ dysfunction and holistic approaches to dental health care. Reprint requests to: 120 Wellington St., London, ON. N6B 2K6.

Temporomandibular joint dysfunction (TMJ syndrome) may be defined as a loss of function in the masticatory system as a result of deleterious changes in one or both temporomandibular joints and/or in the muscles of mastication. The changes frequently seen in the muscles of mastication involve muscle spasm and pain which is often labelled myofascial pain dysfunction. The cardinal changes in the temporomandibular joints are clicking, crepitus, pain and limitation of mandibular movement.

Solberg found that at least 70% of 739 university students had signs of mandibular dysfunction and that 5% required immediate treatment.1 Thiel and Posselt,2,3 reporting on non-patient groups in a randomly selected population, found temporomandibular joint sounds in 52% and 41% of the samples respectively.

Clinical studies usually report a greater incidence of TMJ dysfunction in females.4-6 Solberg et al.1 found an increased incidence in women of 10-15%. Tyldesley7 suggests that 80% of chronic TMJ dysfunction is found in females.

Whatever the actual numbers, TMJ dysfunction is clearly an important cause of severe, chronic pain for many people.

Recurrent headaches were reported in 78% of female and 59% of male TMJ patients, and judged to be more severe in TMJ patients than in a control group.8 Pain, particularly headache, is what motivates patients to seek the help of their physician.

A patient may present other signs and symptoms, including ear, neck and facial pain; vertigo without nystagmus; tinnitus, clicking and crepitation within one or both temporomandibular joints, during mandibular movement; burning and prickling sensations of the side of the tongue and roof of the mouth; fatigue; difficulty in swallowing; spontaneous subluxation of the mandible; chronic sore throat; forgetfulness; changes in hearing ability and ‘clogged ears’, and diverse muscle spasm throughout the body.

TMJ dysfunction syndrome is clearly a great imitator. Therefore, while definitive diagnosis and treatment most often requires a dentist, it is entirely appropriate that a patient first see a physician so that numerous medical conditions can be ruled out.

The Masticatory System

Head posture is largely determined by a balance in muscle tonus of the cervical/spinal musculature and the primary and secondary muscles of

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mastication. Changes in one anatomical component, such as spasm of a cervical muscle, may easily cause changes in head posture and jaw position. Conversely, a change in mandibular position may change head posture, leading to changes in cervical and spinal muscles. Lieb cites numerous examples of dramatic improvements in spinal posture after mandibular repositioning by dental treatment.

Figure 1 illustrates the essential structures of the temporomandibular joint. Each TMJ is capable of two distinct movements—rotation and sliding. The TMJ functions as a compound joint; the articular disc is a "third" bone. The mandibular condyle articulates with the disc to form a disc-condyle complex. Posterior rotation of the disc around the condyle is limited by the superior head of the lateral pterygoid muscle. Anterior rotation of the disc is limited by the elastic tension of the retrodiscal tissue. Medial and lateral movement of the disc is limited by the collateral capsular ligaments and by the lateral temporomandibular ligaments. As the mandible is opened and closed, ligamentous support and muscle tonus should ensure that the inferior surface of the disc remains in contact with the head of the mandibular condyle. The superior surface of the disc slides forward in contact with the temporal bone. Thus, the TMJ may be considered a double joint; one joint functions on top of another.

Functionally, the teeth may also be considered a part of the TMJ. Once the upper and lower teeth are in contact, the position of the mandibular condyle is largely determined by tooth form and the presence or absence of teeth.

**Mandibular Closing Problems**

Normal joint function is illustrated in Figure 2. When the mandibular position dictated by tooth contact is not congruent with the mandibular position dictated by the temporomandibular joint (or joints) and the tonus of the masticatory muscles, problems can begin.

Inappropriate tooth contacts which deflect the mandible to the side, forward, or backward upon closing, result in dyspnetic signals to the CNS from periodontal, joint capsule and muscle proprioceptors.

Jankelson\(^\text{10}\) states that occlusion of the teeth is a physiological act which occurs every 60-70 seconds, before deglutition. If the mandible cannot complete that act because of the restraint of wedging tooth surfaces, struggling movements are initiated.

Also, a lack of dental support is believed to allow the mandibular condyles to be retruded in the TMJ during periods of tooth contact, resulting in

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**Fig. 1.** Drawing illustrates essential structures that constitute the functioning temporomandibular joint. Note that the inclination of the articular eminence is about 52 degrees from the horizontal supra-articular crest. a. supra-articular crest b. temporal articular surface, composed of nonvascular fibrous tissue c. articular disc d. condylar articular surface, composed of nonvascular fibrous tissue e. superior retrodiscl lamina (elastic) f. inferior retrodiscl lamina (collagenous) g. retrodiscl loose connective tissue h. posterior capsular ligament (collagenous) i. condylar axis of rotation j. discal collateral ligament (collagenous) k. superior lateral pterygoid muscle l. inferior lateral pterygoid muscle m. anterior capsular ligament (collagenous) x. posterior margin of condylar articular facet

**Fig. 2.** Normal temporomandibular joint function during opening movement, as seen by arthrography. The disc is the stippled structure between the condyle below, and the temporal bone above. A. mandible in the closed position. B-D, progressive stages of opening. The disc slides forward with the condyle as it translates to, and sometimes over, the articular eminence. The superior stratum of the bilaminar zone becomes stretched; the inferior stratum does not.

**Figs. 2-8** reproduced with permission from Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins, 1983.

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anterior displacement of the articular disc. Farrar and McCarty have estimated that 71% of patients with TMJ symptoms have some degree of anterior disc displacement. Mandibular closing deflections need not be extensive to cause TMJ problems.

The loss of vertical dimension resulting in condylar retraction or bite interferences may not be directly related to the number of lost posterior teeth. For instance, the loss of four teeth will not necessarily create a greater problem than the loss of two teeth. Franks has observed that tooth loss increases the likelihood of TMJ dysfunction; the maximum incidence occurs with the loss of three to five teeth.

If the articular disc is displaced forward during intercusption, as the jaw is opened again, the mandible translates forward and the condyle is believed to bump up over the posterior lip of the disc, producing an opening click. The disc-condyle complex will then function relatively normally until the condyle moves back off the disc on mandibular closing, creating a 'reciprocal click' (see Fig. 3).

If condylar retraction becomes chronic, the retrodiscal tissue and collateral ligaments of the joint may be stretched. The articular disc may then be displaced further forward. As this occurs, the reciprocal clicks may be heard progressively later on opening and earlier on closing.

If the disc is displaced so far anteriorly that the condyle cannot override the posterior border of the disc, mandibular opening may be prevented (see Fig. 4).

Condylar impingement on the retrodiscal tissue may result in ligamentous damage, joint inflammation with significant effusion into the joint and pain, which may be localized or referred to surrounding musculature.

**Origin of Pain**

Conversely, many authors believe that much of the pain and dysfunction of TMJ problems is of myogenic origin. Pain and dysfunction have been shown to result from myofascial trigger points in shortened muscles. These authors have explicitly identified muscles that refer to the temporomandibular joint and to other parts of the head and face.

Arlen has described the otomandibular syndrome, in which the patient complains of pain in and around the ear, fullness in the ear, hearing loss, tinnitus and loss of equilibrium. These symptoms are explained by disturbances in neuromuscular function of the tensor tympani muscle, which inserts a tendinous attachment onto the neck of the malleus; and the tensor palati muscle which functions to open the eustachian tube. Both these muscles, like the muscles of mastication, are innervated by the trigeminal nerve.

Some of the symptoms seen in otomandibular syndrome can also be explained by Pinto's discovery that a fibro-elastic ligamentous structure (the mandibular-maleolar ligament) originates in the neck of the malleus and spreads in a cone shape forward and downward to insert into the medioposterior part of the TMJ capsule and meniscus. Therefore movement of the TMJ capsule and meniscus can result in movement of the tympanic membrane.

**Psychophysiological Theories**

Clinicians treating TMJ disorders cannot but be impressed with their patients' psychological distress. Several investigators believe that psychological factors are more important than occlusal factors. Two psychophysiological theories of TMJ dysfunction have developed.

One theory holds that there is a pre-existing personality which is prone to TMJ dysfunction and pain. Proponents of this theory may ignore anatomical/dental considerations, empha-

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**Fig. 3.** Mechanism of early click due to slight anterior displacement of the articular disc. A, rest position. B, as the condyle begins to translate forward, it must override a thickness of posterior disc material, causing a click. This seats the condyle in the central, thin part of the disc. C and D, after the click, mandibular opening and translation of the condyle proceed with apparently normal disc mechanics.

**Fig. 4.** Mechanism of blocking mandibular depression at one point due to marked anterior displacement of the articular disc. A, rest position. B, as the condyle translates forward, it impinges on the disc, but is unable to ride over it. C and D, this blocks full forward translation, and thereby, full jaw opening.
sizing treatment such as muscle relaxants, psychotherapy, hypnosis, biofeedback and behavior modification.

The second psychophysiological theory proposes that the psychological phenomena seen in TMJ syndrome are the result of the syndrome. Grieser et al. state that long-term sufferers of TMJ dysfunction usually develop anxiety neurosis and severe depression. De Steno suggests that for these chronic sufferers a cyclic effect may develop in which the psychological component can dominate.

In looking for a “TMJ personality”, Mosak found that TMJ distress syndrome is primarily somatogenic. The physical factor predominated in 80% of the subjects studied. Mosak concluded: “Psychological symptoms associated with the TMJ syndrome appear to be the patient’s response not only to the physical pain but also to his loss of the sense of wellbeing, to his deteriorating social situation, and to the run-around I get from dentists and doctors.”

Travell and Simons warn the physician against assuming that psychological factors are primary. They point out that it is all too easy for the physician to blame the patient’s psyche for the physician’s inability to recognize all of the medical and neurophysiological factors that are contributing to the patient’s myofascial pain; this “wrong assumption can be—and often is—frightfully devastating to the patient”. 

In summary, TMJ dysfunction syndrome has been etiologically related to occlusal (bite) interferences, retruded condyles and psychological distress. However, the extensive dental literature dealing with the relative importance of these causes is often contradictory. Greene comments: “Rarely in the history of dentistry have so many labored for so long, only to end with such extreme disagreement. After more than half a century, the myofascial pain dysfunction syndrome continues to be one of the most controversial areas in dentistry”.

Differentiation

Different clinicians frequently focus on their pet causes and treatment. Adding to the confusion is the fact patients frequently have retruded condyles, bite interferences and great psychological stress, but no TMJ discomfort.

De Steno describes a “TMJ triad” of components which are necessary for the development of TMJ pain and dysfunction:

1. Tissue alterations include dental attrition, loss of posterior occlusion, iatrogenic changes, local pathology, arthritis and other systemic diseases.
2. Predisposing factors include genetic (intrinsic) and acquired (extrinsic) elements such as trauma, deleterious habits and nutritional changes.
3. Psychological factors may prime the patient for TMJ syndrome.

Establishing a tentative diagnosis of TMJ dysfunction first necessitates ruling out systemic diseases such as cardiovascular, renal, and arthritic disease. Sonkin asserts that hypothyroidism is frequently overlooked in the diagnosis of patients with generalized muscle symptoms.

Local pathology, as in the ears, nose, sinuses, throat or cervical spine must next be investigated. Specific abnormalities to consider include trigeminal and glossopharyngeal neuralgia, vascular headaches, temporal arteritis, fracture of the styloid process, ossifi-
cation of the styloid ligament and injury of the auriculotemporal nerve.

It may be helpful to keep the following points in mind in assessing the TMJ patient’s history:
1. TMJ pain is often deep and more difficult to locate than that of most ear diseases.
2. A high frequency hissing type of tinnitus is frequently found.19
3. Patients frequently report an abnormal sound perception which Arlen describes as slight, episodic “waxing and waning” of sound.19
4. When moving their heads, some patients feel off balance but do not show the vertigo typical of Meniere’s disease or labyrinthitis.
5. Movement of the mandible may act as a triggering device.
6. A history of episodic limitation of movement or locking of the jaw is common.
7. Patients often discover that they can get relief by separating their upper and lower teeth, as in lip or cheek biting.

Alder28 has proposed that the clinician ask the patient the following question: “If I had a magic wand or magic pill which would eliminate your symptoms immediately and irrevocably, what would be different in your life?” The patient’s answer may be useful in assessing the psychological component of the problem. For instance, if the patient replied that his/her marriage problems would be over, that he/she would return to school and then travel extensively, one might suspect a predominantly psychological component.

**Examination**

Clinical examination to confirm TMJ dysfunction should begin with a visual analysis of posture. A higher eye in conjunction with a lower shoulder and hip on the ipsilateral side will often coincide with greater condylar retrusion on that side.29

The muscles of the head, neck, back, chest and legs should be examined for the presence of tenderness, spasm and trigger points. TMJ patients frequently present marked tenderness of the muscles of their back and the calves of their legs as well as the muscles more proximal to their jaw.

Fig. 7. Referred pain pattern (solid) and location of the responsible trigger point (x) in the left medial pterygoid muscle. A, external areas of pain to which the patient can point. B, anatomical cut-away to show the location of the trigger point area in the muscle, which lies on the inner side of the mandible. C, coronal section of the head through the temporomandibular joint, looking forward, showing internal areas of pain.

Fig. 8. The referred pain pattern (solid) of trigger points (x’s) in the left lateral pterygoid muscle.

Particular attention should be then paid to the muscles of mastication. As muscles are being examined, it is useful to visualize the areas to which trigger points may refer pain. For instance, the lateral pterygoid, masseter (deep), medial pterygoid and sternocleidomastoid (clavicular) muscles all may refer pain directly to the ear and temporomandibular joint.25

Suspected referred pain may be verified by injection of the affected muscle with local anesthetic. A 0.5% solution of procaine without vasoconstrictor may be used for this.

The referral patterns for the major muscles of mastication are illustrated in Figures 5-8.

Both temporomandibular joints should be palpated while the mandible is at rest. The amount of pain elicited will indicate the amount of inflammation within the joint. While the clinician’s fingers are still over the joints, the mandible should be opened and closed, and moved from side to side. Again the location and amount of pain should be noted, together with any roughness in movement.
The patient should repeat these movements while the examiner listens to each joint with a stethoscope. The examiner should note crepitus and clicking.

Next the patient should be asked to open and close his/her jaw and the maximum interincisal opening should be checked. Normally, the fully opened jaw should admit the patient’s first three knuckles (non-dominant hand) in a tier between the upper and lower incisors. The interincisal distance should also be measured (average = 42 mm).

A deviation of the mandible during opening and closing movements may occur with myospasm, fibrosis or limitations of condylar movement within the joint.

As the patient closes, a retruded condyle can often be palpated with a finger placed against the anterior wall of the external auditory meatus. This palpation may be very painful as the teeth come together.

A comparison of lateral jaw movements with maximum interincisal opening may provide valuable clues to the reason for limited movement. Patients should be asked to open their mouths slightly and then slide their jaw as far to each side as they can. The maximum shift from the midline in each direction should be carefully noted.

The normal ratio of lateral excursion to interincisal distance is approximately 10 mm: 40 mm or 1:4. A decreased ratio (for example, 1:3) indicates extracapsular restriction, while an increased ratio (for example, 1:6) suggests restriction of movement within the joint.

The patient should also be asked to close slowly until the teeth ‘just touch’. Then the patient should close completely (maximum intercuspation) and any pain, noise, or shift in the mandible should be noted. The presence of positive signs may indicate a bite interference and an early stage of joint damage.

**Radiology**

Radiographic visualization of the temporomandibular joint has great value, both in ruling out joint pathology such as arthritic change and in confirming malposition and disc derangement.

Transcranial and panoramic projections of the temporomandibular joints are readily available with ordinary dental X-ray equipment. Although they have limitations, if properly taken and interpreted these techniques provide a great deal of information.

A transcranial series should include at least three positions of the mandible:
1. Mandibular rest position
2. Closed, clenched position
3. Maximum open position

Because mandibular position is critical to proper interpretation, this series should be taken by someone with dental training.

Radiological findings will be most dramatic in patients with late stage TMJ dysfunction. Berrett notes the following findings:

—The condyle is consistently decreased in size (flattened).
—Hypertrophic sclerosis and lipping (osteophyte formation) occur.
—Deforming degenerative arthritis is common.

More critical evaluation of the articular disc space is afforded by tomography. Arthrography applied to cinefluoroscopic techniques has recently become popular in assessing disc derangements and perforations of the articular disc. However, these techniques are not suggested for initial screening and treatment.

A confirmed working diagnosis of temporomandibular joint dysfunction requires the collaboration of a physician and a dentist—and often numerous medical and dental specialists.

**Treatment**

Treatment is aimed at reducing one or more of the etiological components: extrinsic predisposition, tissue alteration or psychological factors. Procedures should be kept simple and should be reversible until positive confirmation of the diagnosis is made.

Palliative therapy is designed to alleviate pain and muscle spasm and to limit functional demands on the TMJ, while the physician establishes the diagnosis and arranges the medical, dental and other consultations needed. The success of some palliative therapies is in itself highly diagnostic.

**Palliative Therapies**

Limitation of movement should be recommended to patients if not already dictated by pain. Patients should stick to soft but nutritious foods during acute periods.

*Heat* should be applied to sore muscles and joints for 15-20 minutes four times a day. This will increase local circulation and reduce muscle spasm, besides providing a sedative effect.

**Ultrasound** increases heat to deep structures, particularly bone, menisci and joint capsules.

**Cryotherapy.** Cold is believed to be effective by reducing the concentration of histamincs in the skin, by raising the cutaneous skin threshold and by offering controlled tactile/thermal stimuli, thereby modifying pain intensity.

**Spray and stretch.** A vapocoolant spray of Fluori-Methane directed to the skin over a muscle trigger point is effective in inhibiting pain and spinal stretch reflexes, therefore permitting stretching of the muscle and inactivation of its trigger points.

The jet stream of spray is directed at an acute angle (30°) and is swept over the skin parallel to the muscle covering the entire muscle length in one direction (toward referred pain). Two or three sweeps is usually maximum. The skin should then be warmed.

Local anesthetic can be injected into active trigger points. A 0.5% solution of procaine without vasoconstrictor is recommended. Immediately after injection, the muscle is passively stretched.

**Analgesics and anxiolytics.** In addition to the wide variety of analgesics available, an antihistamine such as promethazine may help the TMJ patient who is having difficulty sleeping. Corticosteroids may be useful if joint inflammation is present. Markovich reports that 10-25 mg amitriptylene acts as a good anxiolytic-antidepressant and as a potent analgesic.

**Tetanizing and sinusoidal currents** have been used to stop muscle spasm and recover gradual rhythmic movement.

**Electrogalvanic stimulation** is well tolerated even by patients in great pain. Muscle relaxation is obtained quickly with much relief.

**Transcutaneous nerve stimulation** has also proven effective in pain reduction. Optimal treatment is nine to 12 minutes several times per day.

**Nutritional supplementation.** As the prevalence of unrecognized hypovitaminosis is very high, it is prudent to ensure that TMJ patients receive a good quality vitamin supplement as well as dietary supervision. Hypovitaminosis A may also cause bone or joint pain and severe throbbing headache.
which could be confused with TMJ symptoms. 25

Bite plane (mandibular orthopedic repositioner). The placement of a dental appliance to disclude the teeth often produces a dramatic reduction of TMJ pain and dysfunction. Such appliances are designed to eliminate bite interferences and/or prevent the mandibular condyle from moving into a retruded position, thereby preventing joint stress and allowing normal resting length of the muscles of mastication.

Biofeedback training may help some patients become aware of unnecessarily sustained muscle contraction which affects the TMJ and related musculature.

Psychotherapy is often very valuable in helping the patient learn new perspective and skills, with concomitant reduction in anxiety and frustration. Many of my patients have done well when seeing a family counselor to whom I frequently refer. They seem very comfortable with the counselor’s growth oriented model which contrasts with the doctor/patient, illness/treatment models of traditional medicine and dentistry.

Once normal condylar position has been restored and muscle spasms have been eliminated, a dentist can begin definitive therapy specific to the problem.

Specific Treatment

Occlusal correction (coronal reshaping) can eliminate bite interferences and prevent further symptoms or joint damage.

Condylar repositioning can be achieved by several techniques including dentures, occlusal bonding, the fabrication of crowns and bridges, overdentures and orthodontic therapy.

Surgical correction of intercapsular derangements of the disc and condyle have been performed with increasing success recently. Farrar and McCarty report a 94% success rate after 377 such procedures over six years. 34

Conclusion

As the first line health care provider, the family physician who is aware of temporomandibular joint dysfunction syndrome can be invaluable to the patient by recognizing this painful syndrome, providing palliative treatment, taking essential steps in the differential diagnosis and referring the patient to a dentist and other therapists competent to carry out definitive treatment.

References

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