Review Article

Temporomandibular joint disorders in children and adolescent: A review

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ABSTRACT

Temporomandibular disorders (TMD) are a group of joint and muscular dysfunctions affecting the Cranio-orofacial area. Historically, TMDs were once believed to solely affect adults; however, recent studies have discovered an increased prevalence of TMD signs and symptoms in children and adolescents. The reported prevalence of these disorders in pediatric patients varies significantly due to methodological variations among studies; as a result, standardized diagnostic criteria for children are required. In this age group, TMDs can cause pain in the masticatory muscles and TMJs, as well as limited or asymmetric mouth opening and TMJ sounds. Rather than a single causative element, TMDs are produced by a combination of factors such as trauma, occlusion, systemic and developmental problems, and psychological disorders. A complete history and examination, which may include imaging, are essential to obtain a precise diagnosis and assure successful treatment of many disorders. For these issues, there is a range of therapeutic interventions available, although reversible treatments should be favored.

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1. Introduction

Temporomandibular joint (TMJ) is a type of synovial joint with an articulating surface, that allows significant movement by two bones, covered by hyaline cartilage.1 Temporomandibular disorders (TMD) result in pain and disability and it is the second most common musculoskeletal condition (after chronic low back pain).2 Several epidemiological studies of signs and symptoms were conducted by the end of the 1970s, in children and adolescents. Even if the reported prevalence figures of subjective symptoms and clinical signs dissents between studies, it’s obvious that TMD has begun to be seen by preschool. Because of the large variation within the reported frequencies of TMD signs and symptoms, it is troublesome to get a comprehensive image of the ‘real’ prevalence of TMD in children and adolescents and to estimate the requirement and demand for treatment.3 This review article aims to provide a comprehensive compendium of temporomandibular disorders among children and adolescence and their management.

1.1. Development of the TMJ

The mandibular condyle, mandibular fossa, and related connective tissue (including the articular disk)7 are the three main components of the TMJ. The first evidence of TMJ growth in humans appears 8 weeks after conception.8 The mandibular condyle becomes less vascularized over the first decade of life, and the majority of the significant morphological changes are finished. There is a continued yet gradual slowdown of growth in the second decade of life. The shape of the mandibular condyle can change dramatically during development, with about 5% of
condyles experiencing radiographic changes between the ages of 12 and 16.9

2. Definition of TMD

TMD has been defined as “functional disturbances of the masticatory system”, others include masticatory muscle disorders degenerative and inflammatory TMJ disorders, and TMJ disk displacements under the umbrella of TMD.10

2.1. Terminology

Various terminologies have been used to describe functional disruptions of the masticatory system over time. This has undoubtedly added to some of the ambiguity in this field.

1. In 1934 James Costen identified a cluster of symptoms centered on the ear and temporomandibular joint (TMJ). As a result of his work, the syndrome was coined as costen syndrome.
2. In 1959, Shore coined the term TMJ dysfunction syndrome.
3. Soon after Ramfjord and Ash coined the term functional TMJ disturbances.
4. Some authors suggest that the above terms are too narrow and that a broader term, such as craniomandibular disorders, should be used rather.
5. The term TM disorder was coined by Bell and has gained attention. This term encompasses all issues relating to the masticatory system’s not just those involving the TMJs.
6. As a result, the American Dental Association coined the term Temporomandibular disorders, or tm disorders, to better coordinate efforts.11

3. History of Temporomandibular Dysfunction

The dentist was typically the first person drawn into the field of TMDs with an article written by Dr. James Costen in 1934. Dr. Costen was an otolaryngologist who, based on eleven cases, first urged into the profession that changes within the dental condition were responsible for the number of ear symptoms. Only a few dentists became involved in treating TMD pain issues in the late 1930s and early 1940s.

The foremost common therapies provided at that time were bite-raising appliances. The dental profession began to question bite-raising equipment as the treatment of choice for mandibular dysfunction in the late 1940s and early 1950s. At this time, the dental profession began to investigate occlusal interferences as a potential cause of TM disorder complaints. The first textbook on the masticatory system was published in the late 1950s. Masticatory muscle pain disorder was the most commonly reported condition at that time.

Oclusion and emotional stress were widely recognized as key etiologic factors of functional masticatory system
abnormalities in the 1960s and 1970s. Then, in the late 1970s, there was a surge in interest in TM disorders. At the same time, awareness about pain conditions caused by intracapsular sources entered the profession. It wasn’t until the 1980s that the profession started to understand and consider the complexities of TMDs. Because of this complication, the discipline is now attempting to define its proper role in the treatment of TMDs and orofacial pains.

During the 1990s and 2000s, the profession began to adopt the idea of evidence-based medicine, which necessitated the creation of educational programs to better prepare physicians to manage TMD patients. Several universities have started postgraduate training programs to formalize this education process. The Commission on Dental Accreditation, which is responsible for accrediting all dental specialties in the US, recognized the need to accept and standardize these programs in 2010.\(^\text{11}\)

4. **Taxonomic Classification for Temporomandibular Disorders\(^\text{12}\)**

4.1. **Temporomandibular joint disorders**

1. Joint pain
   (a) Arthralgia
   (b) Arthritis

2. Joint disorders
   (a) Disc disorders
      i. Disc displacement with reduction
      ii. Disc displacement with reduction with intermittent locking
      iii. Disc displacement without reduction with limited opening
      iv. Disc displacement without reduction without limited opening
   (b) Other hypomobility disorders
      i. Adhesions/adherence
      ii. Ankylosis
         A. Fibrous
         B. Osseous
   (c) Hypermobility disorders
      i. Dislocations
         A. Subluxation
         B. Luxation

3. Joint diseases
   (a) Degenerative joint disease
      i. Osteoarthrosis
      ii. Osteoarthritis
   (b) Systemic arthritides
   (c) Condylysis/idiopathic condylar resorption
   (d) Osteochondritis dissecans
   (e) Osteonecrosis
   (f) Neoplasm
   (g) Synovial chondromatosis

4. Fractures
5. Congenital/developmental disorders
   (a) Aplasia
   (b) Hypoplasia
   (c) Hyperplasia

4.2. **Masticatory muscle disorders**

1. Muscle pain
   (a) Myalgia
      i. Local myalgia
      ii. Myofascial pain
      iii. Myofacial pain with referral
   (b) Tendonitis
   (c) Myositis
   (d) Spasm

2. Contracture
3. Hypertrophy
4. Neoplasm
5. Movement disorders
   (a) Orofacial dyskinesia
   (b) Oromandibular dystonia

6. Masticatory muscle pain related to systemic/central pain disorders
   (a) Fibromyalgia/widespread pain

4.3. **Headache**

1. Headache associated with TMD

4.4. **Associated structures**

1. Coronid hyperplasia

5. **Prevalence of TMD in Children and Adolescents**

TMDs have been highlighted as an important source of orofacial non-odontogenic pain. In the literature, the prevalence of TMD in children and adolescents varies greatly. This variance may be due to differences in the populations surveyed, diagnostic criteria, examination methods, and/or examining practitioner inter-and intra-rater variations.\(^\text{10}\) In research settings, the Diagnostic Criteria (DC) TMD test protocol is used to reduce diagnostic variability. TMD was found to be prevalent in 11.9 percent of adolescents in one study using DC/TMD criteria. The majority of evidence shows that the prevalence of TMD signs and symptoms increases with age. TMD pain was recorded by 4.2 percent of adolescents aged 12 to 19 years.
in another study. According to one study of primary dentition, 34% of patients had signs and/or symptoms of TMD. A survey of 4724 children aged 5 to 17 years reported that 25% of them had symptoms. Clicking was seen in 2.7 percent of children in primary dentition, 10.1 percent of children in the late mixed dentition, and 16.6 percent of permanent dentition patients.  

In a systematic review and metaanalysis of intra-articular TMD in children and adolescents, clinical signs were found to be 16 percent common and TMJ sounds were found to be 14 percent common. While both boys and girls experience TMD pain as they grow older, recent surveys show that girls have a substantially higher prevalence of symptoms and a greater need for care in girls than boys. The onset of puberty in girls has been correlated to the development of symptomatic TMD. School absences and analgesic intake due to TMD-related pain were recorded by 32.5 percent of girls and 9.7 percent of boys between the ages of 16 and 19. In teenagers, headaches tend to be independently and highly correlated to TMD, with the majority occurring before the onset of jaw pain.  

5.1. Etiology of TMD

There are a variety of causes for temporomandibular disorders. There is insufficient evidence to determine which patients will develop TMD and which will not. TMDs are influenced by numerous factors: predisposing (risk) factors, precipitating (initiating) factors and perpetuating (or sustaining) factors. The evidence indicates that there is a weak association between any particular etiological factor and the subsequent signs (i.e., findings identified by the dentist during the examination) and symptoms (i.e., findings reported by the child or parent).  

Evidence-based literature reveals five major factors that are contributing to TMD. These factors are:

1. Occlusal condition,
2. Trauma
3. Emotional stress,
4. Deep pain input,
5. Parafunctional activities
6. Other factors

5.2. Occlusal factor

The relation between skeletal and occlusal factors and the onset of TMD is tenuous at best. Furthermore, regardless of whether premolars were removed, the available evidence does not support that orthodontic treatment causes or improves the development of TMD. Occlusal changes, disease, muscle spasms, nervous tension, and/or restorative prosthetics can all affect the freeway dimension of the rest position (normally two to four millimeters). Although most children and adolescents can compensate without issue, failure of the masticatory system to adapt in others can lead to an increased risk of dysfunction. Despite the lack of evidence linking skeletal or occlusal factors to TMD, the following have some correlation:

1. Skeletal anterior open bite
2. Steep articular eminence of the temporal bone
3. Overjet greater than six to seven millimeters
4. Skeletal class II profile
5. Class III malocclusion
6. Unilateral posterior crossbite
7. Posterior crossbite

5.3. Trauma

Masticatory system function can be disrupted as a result of trauma to the facial structures. Intracapsular disorders seem to be more affected by trauma than muscular disorders. Trauma can be classified into two categories:

5.3.1. Macro trauma

Any unexpected force that can cause structural changes, such as a direct blow to the face, is known as macro trauma.

5.3.2. Microtrauma

Any small force that is continuously applied to the structures over a long period is referred to as microtrauma. Microtrauma may also be affected by activities like bruxism or clenching, which cause microtrauma to the tissues that are being loaded (i.e., teeth, joints, or muscles).

5.4. Emotional stress

A raise in emotional stress is a common factor that can affect masticatory activity. Muscle function is influenced by the emotional centers of the brain. The hypothalamic-pituitary-adrenal (HPA) axis is activated by stress, which in turn prepares the body to respond (through the autonomic nervous system).

The sympathetic activity or tone of a person may be influenced by emotional stress. Numerous subconscious mechanisms that maintain homeostasis are continuously monitored and controlled by the autonomic nervous system. The autonomic nervous system regulates blood flow within the body as one of its functions. The sympathetic nervous system is linked to the stress-induced fight-or-flight reflex. Capillary blood flow in the outer tissues is constricted in the presence of stress, allowing increased blood flow to the more critical musculoskeletal structures and internal organs. The skin, especially the hands, cools as a result. The sympathetic nervous system’s prolonged operation may affect some tissues, such as muscles. Sympathetic behavior has been linked to an increase in muscle tone, resulting in a painful muscle condition. As a result, increased sympathetic activity or tone can be an etiologic factor that influences TMD symptoms. Moreover, in those with pre-existing TMD, symptoms may be exacerbated during...
Table 1: There are various commercial instruments available in diagnosing the various temporomandibular disorder. A partial list of the specific masticatory signs and associated measurement devices are presented

<table>
<thead>
<tr>
<th>Sign</th>
<th>Type of device</th>
<th>Specific device</th>
<th>Manufacturer</th>
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</thead>
<tbody>
<tr>
<td>Masticatory muscle pain</td>
<td>Pressure algometers</td>
<td>Pain Threshold Meter Model PTH-AF2</td>
<td>Pain Diagnostics and Thermography Co, Great Neck, N.Y</td>
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<tr>
<td></td>
<td>Surface EMG devices</td>
<td>Electronic Pressure Algometer Type BioEMG*</td>
<td>Somedic Sales AB, Farsta, Sweden</td>
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<td></td>
<td></td>
<td>K6i/EMG*</td>
<td>BioResearch Inc, Milwaukee, Wis.</td>
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<td></td>
<td></td>
<td>Grass Model 7B/7P3</td>
<td>Electromyograph, Myotronics Inc, Seattle, Wash.</td>
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<td></td>
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<td>Coulbourn bioamplifier</td>
<td>Grass Inc, Quincy, Mass</td>
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<td></td>
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<td>EMG Monitor</td>
<td>Coulbourn Instruments, Inc, Lehigh Valley, Pa.</td>
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<tr>
<td>Joint noises (clicking and</td>
<td>Sound/vibration detection devices</td>
<td>SonoPAK system*</td>
<td>BioResearch Inc, Milwaukee, Wis.</td>
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<td>crepitation</td>
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<td>TMJ Doppler II</td>
<td>Three Brothers Enterprises, Tonawanda, N.Y.</td>
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<td></td>
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<td>Electret type MCE-2000</td>
<td>Volkner Elektronik, Brounschweig, Germany</td>
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<tr>
<td>(trismus, clicking, locking,</td>
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<td>Sirognathograph</td>
<td>Siemens, Munich, Germany</td>
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<td>and dyskinesia)</td>
<td></td>
<td>Visi-Trainer C-II†</td>
<td>US Shizai, Santa Monica, Calif.</td>
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<td></td>
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<td>BioEGN*</td>
<td>BioResearch Inc, Milwaukee, Wis.</td>
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*Has the seal of approval from the American Dental Association.
†Not currently available in the commercial market.

Table 2: Are the Differential diagnoses of orofacial pain that may mimic TMD

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<thead>
<tr>
<th>Neuropathic Pain</th>
<th>Odontogenic Pain</th>
<th>Intracranial Pain</th>
<th>Pain from Other Adjacent Structures</th>
<th>Headaches not Attributed to TMD</th>
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<tr>
<td>Trigeminal neuralgia</td>
<td>Dental sensitivity</td>
<td>Ear, Nose, Throat, Eyes, Sinus, Salivary glands, Lymph nodes, Vasculature, Cervical region.</td>
<td>Cluster headache</td>
<td>Tension-type headache</td>
<td>Temporal arthritis</td>
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<td>Glossopharyngeal neuralgia</td>
<td>Cracked tooth syndrome</td>
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<td>Postherpetic neuralgia</td>
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<td>Traumatic neuralgia</td>
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<td>Burning mouth syndrome</td>
<td>Aneurysms</td>
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<td>Atypical odontalgia</td>
<td>Bleeding</td>
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<td>Dental caries</td>
<td>Infection</td>
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<td>Dental abscess</td>
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times of stressful events. According to recent studies, there was an impact on the prevalence of depressive symptoms, stress, and TMD pain during times of lockdown and social isolation brought on by the ongoing COVID-19 pandemic.14

5.5. Deep pain input

Deep pain input will centrally excite the brainstem, producing a muscle response known as protective co-contraction in the muscles. This is how the body reacts to injury or the threat of injury in a normal, healthy way. As a result, it is fair to expect a patient with discomfort, such as a toothache (i.e., necrotic pulp), to have minimal mouth opening. Any source of persistent deep pain input can be an etiologic factor for limited mouth opening, which can manifest clinically as TMD. The restricted mouth opening is merely a secondary response to the intense pain. However, if the clinician is unaware of this phenomenon, he or she can mistakenly believe that the restricted mouth opening is a primary TMD issue, and treatment will be ineffective. Any source of persistent deep pain input can be an etiologic factor for limited mouth opening, which can manifest clinically as TMD.11

5.6. Parafunctional activity

TMD is believed to be caused by joint overloading, which contributes to cartilage breakdown, synovial fluid changes, and other changes within the joint. TMD is hypothesized to be caused by bruxism, clenching, hyperextension, and other practices that are repeated over time. When children grind their teeth, they experience more pain and muscle tenderness when eating.10

5.7. Systemic and pathologic factors

Connective tissue diseases such as rheumatoid arthritis, systemic lupus erythematosus, juvenile idiopathic arthritis, and psoriatic arthritis are examples of systemic factors.5,15,16 An imbalance of pro-inflammatory cytokines causes these systemic disorders, which leads to oxidative stress, the production of free radicals, and eventually joint degeneration.16 Systemic contributions include joint hypermobility, hereditary susceptibility, and hormonal changes. TMDs with pathologic hyperplasia and condylar tumors are a distinct category.15

5.8. Genetic and hormonal factors

Hormone’s role in the etiology of TMD is debatable. Although randomized controlled trials reveal that estrogen does not have a role in the etiology of TMD, cohort and case-control studies show the opposite.17 There has been limited research on genetic predisposition to TMD development. Research of catechol-O-methyltransferase haplotypes recently discovered that having one low pain sensitivity haplotype reduced the risk of TMD.18 Additional studies have shown TMJ pain and other symptoms differ according to the stages of the menstrual cycle.19

6. Diagnostic Approach

The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) is now the most widely accepted and standardized method for assessing and classifying TMD, with high sensitivity and specificity. It is a multifaceted diagnostic research tool that is accepted all over the world.

The DC/TMD examination covers two-axis, recognizing that TMD has both a structural and a biopsychosocial component. Axis-I has a checklist for a specific clinical examination to arrive at a specific physical diagnosis of TMD in terms of joint and musculature, whereas Axis-II contains several instruments to assess the patient’s psychological status.14

6.1. Imaging and other investigations

Imaging is considered to be a valuable tool in the diagnosis of TMD. While plain radiographs like orthopantomogram provide limited diagnostic information, they are convenient, quick, and serve to rule out some of the most common differential diagnoses of the bony TMJ, such as fractures, ankylosis, growth abnormalities, and neoplasms. This is owing to cost and availability, as well as the fact that additional imaging may not alter the initial management strategy. When further information is needed, magnetic resonance imaging (MRI) is the gold standard for TMJ imaging. It can be used to check the condition of both the osseous and non-osseous components of the TMJ, such as the masticatory muscles, ligaments, and cartilaginous disc.20

The osseous structure of the TMJ would be further assessed using cone-beam computed tomography (CBCT).21 This may be useful in situations of TMJ ankylosis, benign bone neoplasms, as well as for osseous surgery planning, such as eminectomy for recurrent TMJ dislocation. However, for another diagnosis of TMJ in terms of soft tissue the CBCT is not well recognized.22 Furthermore, ultrasonography has been proposed as a diagnostic technique for TMD.23 Ultrasound has the advantages of being non-invasive, inexpensive, and widely available in many medical facilities, but its efficacy as a diagnostic tool has yet to be proven. Bone scintigraphy may be useful as a diagnostic tool for some TMJ inflammatory disorders, such as osteoarthritis and joint inflammation. Furthermore, bone scintigraphy has been proposed as a technique for evaluating active TMJ condylar development, however, the sensitivity and specificity for this indication have been proven below.14
Nuclear imaging is beneficial in assessing bone metabolism in temporomandibular joint bony components, determining facial skeletal growth, detecting synovitis, and quantifying arthritis in individuals with rheumatoid arthritis or osteoarthritis.

There are three categories of imaging devices in use today: those used for planar nuclear imaging, those for single photon emission computed tomography (SPECT), and those for Positron Emission Tomography (PET). Radionuclide mTc is used for the examination. The temporomandibular joint is ideal for what is called SPECT (single photon emission computed tomography) because it is a quite small joint situated close to the skull base and paranasal sinuses. As a result, unlike double-dimensional imaging, SPECT can distinguish TMJ from high-density bone areas. The sensitivity of the radionuclide examination is excellent, but the specificity is low. Because local isotope concentrations are increased by inflammation, trauma, and tumors, several studies conclude that radionuclide testing is only useful as a screening procedure.

However, recently, the problem of the worldwide shortage of molybdenum-99 has arisen, and fluoride-18 positron emission tomography (F-PET) has attracted attention as an alternative modality that has both greater sensitivity and better image quality than those of a conventional bone scan. F-PET/CT showed high TMJ uptake with osteoarthritis and demonstrated higher sensitivity and accuracy than those of a conventional bone scan for detecting TMD with osteoarthritis.24

6.2. Thin section MR imaging25

Each time MR scanners are upgraded, the signal to noise ratio, soft tissue and, spatial resolution improve. This indicates that the image quality will continue to increase. Because of the reduced slice thickness and smaller field of view, it has a dramatically better spatial and soft tissue resolution. In this image, the trabecular pattern of condyle can be identified, and the perforation of the disk and the osteophytes of the condyle, protruding into the perforation, can be seen with exquisite detail.

6.3. Dynamic MR imaging26

This will allow joint dynamics to be studied without the need for contrast media or a local anesthetic. Multiple images are collected with the mouth opened to different widths, and the images are rapidly viewed on a cine loop. This creates the illusion of a real time examination even though the images were acquired at separate points in time. However, the disk’s position may be followed, and the condyle’s snapping across the back of the disc can be appreciated.

6.3.1. Diagnosis of TMD

Recognizing the causes of TMD-related pain and dysfunction is critical for making treatment decisions. For example, while treating myogenous vs arthrogenous TMD, different therapeutic methods are frequently used. The most crucial aspect of TMD diagnosis is distinguishing between common disorders and clinically significant but atypical conditions, as well as conditions that are more dangerous and require immediate attention.

The following clinical and physical assessments may be performed on the TMJ:

1. Palpation of the muscles of mastication, lateral capsule of TMJ and cervical muscles for tenderness, pain, or pain referral patterns;
2. Mandibular function and provocation tests;
3. Palpation and auscultation for TMJ sounds;
4. Mandibular range of motion

The use of a millimeter ruler to measure the mandibular range of motion (i.e., maximum unassisted opening, maximum assisted opening, maximum lateral excursion, maximum protrusive excursion) and mandibular opening pattern (symmetrical vs. asymmetrical) can aid in the diagnosis of TMD.6,17

7. Treatment of TMD

Only a few studies have looked at the long-term success or failure of various TMD treatments in newborns, children, and adolescents. These findings imply that treatment that is basic, conservative, and reversible is successful in lowering most TMD symptoms in children.26 Finding a balance between active and passive therapy techniques should be the goal of treatment.

Active treatment includes the participation of the patient whereas passive treatment may include wearing a stabilization splint. Information combined with occlusal appliance therapy was the most prevalent treatment for TMD in children.27 Combination techniques have been demonstrated to be more effective in treating TMD than single treatment modalities.

There are two types of TMD treatment: reversible and irreversible.

Reversible therapies may include:

1. Patient education (e.g., relaxation training, developing behavior coping strategies, modifying inadequate perceptions about TMD, patient awareness of clenching and bruxing habits, if present).28
2. Physical therapy [e.g, physiotherapy or transcutaneous electrical nerve stimulation (TENS), ultrasound, iontophoresis, thermotherapy, coolant therapy].10
3. Behavioral therapy (e.g, avoiding excessive chewing of hard foods or gum, voluntary avoidance of
stressors, habit reversal, decreasing stress, anxiety, and/or depression). 29

4. Prescription medication (eg, nonsteroidal antinflammatory drugs, muscle relaxers). While antidepressants have proved to be beneficial, they should be prescribed by a physician. 30

5. Occlusal splints. The purpose of an occlusal appliance is to provide orthopedic stability to the TMJ. These temporarily modify the patient’s occlusion and may be used to reduce parafunctional activity. 31

Irreversible therapies can include:

1. Occlusal adaptation (ie, permanently modifying the occlusion by selective grinding or full mouth restorative dentistry);

2. Mandibular repositioning (eg, headgear, functional appliances) is a technique for altering the growth pattern or permanently changing the position of the mandible.

3. Orthodontics. When the diagnostic and/or treatment needs are beyond the treating dentist’s scope of practice, referrals to other health care providers, such as those with experience in TMD, oral surgery, or pain management, should be made. 10

8. Recommendations

A comprehensive dental history and examination should include a TMJ history and assessment. Questions on the existence of head and neck discomfort, mandibular dysfunction, past orofacial injuries, and a history of current illness with an account of current symptoms should all be included in the history. Additional information is suggested in the case of positive history and/or indications and symptoms of TMD, and a referral may be considered. Other experts may offer joint imaging to evaluate joint sounds if there are no other TMD indications or symptoms. For example, the presence of crepitus may suggest a degenerative change that is not yet painful. Therapeutic strategies for preventing TMD in children have yet to be proven in controlled investigations. Reversible therapy should be investigated for children and adolescents with TMD indications and symptoms. Irreversible therapy should be avoided due to a lack of data on its effectiveness. 32 When otitis media, allergies, aberrant posture, airway congestion, rheumatoid arthritis, or other medical issues are detected, a referral to a medical professional may be required. 24

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10. Conflict of Interest

The authors declare no conflict of interest.

References


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