Temporomandibular Disorders: Etiology and Classification

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2.1 Introduction

Temporomandibular disorders (TMDs) are a group of disorders that have their origin in the musculoskeletal structures of the masticatory system [1]. Therefore, symptoms of TMDs are associated with either the muscles of mastication or the temporomandibular joints (TMJs), or both. Pain can be a common symptom associated with TMDs. These disorders are quite common in the general population. In fact, after dental pains, TMDs are the next most common pain complaint reported by patients in the dental office. Depending on which epidemiologic studies are reviewed the numbers of signs and symptoms associated with TMDs range from 40 % and 60 % of the general population [2, p. 102–28]. However, the numbers of patients requiring professional TMD treatment are reported to be only in the range of 10–15 % [3, 4]. Because TMDs are so common, every dentist needs to have a basic understanding of the etiology, diagnosis, and management of these conditions. This chapter will focus on the etiology and diagnosis of the most common TMD conditions seen in the dental office. Other texts should be reviewed for a more complete overview [2].

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It needs to be appreciated that all TMDs are not the same. There are a great variety of musculoskeletal conditions associated with disorders of the masticatory system. The two broad conditions are muscle pain disorders and intracapsular disorders. These conditions are quite different in their etiology, pathology, and clinical presentations. Because of these differences, they demand different treatment strategies. The most common types of TMDs are muscle pain disorders, which are found to be almost twice as common as intracapsular pain disorders in a chronic pain clinic. It would therefore be very inappropriate to label these patients as "TMJ patients" when the majority of cases may have nothing to do with their temporomandibular joints. Failing to distinguish between these myogenous and arthrogenous conditions will likely lead to the selection of an ineffective treatment that will ultimately fail.

This chapter will highlight the etiologic factors associated with TMDs and describe a classification for the most common conditions seen in the orthodontic practice. It should be noted that although management is not meant to be a part of this chapter, most of these TMDs can be successfully managed with conservative therapies (see Chap. 8). Orthodontic therapy may be considered as a treatment option in a few of these patients according to their specific diagnosis and etiologic factors, but only after clinical symptoms have been successfully managed.

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2.2 Etiologic Considerations of Temporomandibular Disorders

Over the years there has been significant controversy regarding the etiology of TMDs. Early on dentists were very convinced that temporomandibular disorders were primarily caused by occlusal factors. Many dentists directed their therapies toward changing the patient's occlusion; and if that failed, the operator was thought to be incompetent or the patient was considered to have major psychological problems. By the mid-1980s and 1990s, however, the profession demanded more research evidence, which provided a much broader look at TMDs. Over the last 20–30 years we have learned that there are at least five known TMD etiologic factors that need to be considered. Occlusal factors remain as one of these factors, thereby maintaining TMDs as conditions that need a dental evaluation, but the manner by which the occlusion can affect the onset of a TMD must be revisited. Epidemiologic studies do not reveal a strong association between the static relationship of the teeth, such as Angle Class II or III, and the presence of a TMD [2, p. 102–28]. Therefore, in this chapter a new concept regarding the association between occlusion and TMDs will be presented.

The five etiologic factors that have gained significant research support are the occlusal condition, trauma, emotional stress, deep pain input, and parafunctional activity such as bruxism and clenching. Each will be briefly discussed in this section.

2.2.1 The Occlusal Condition

As mentioned earlier, occlusal factors have been thought to be associated with TMDs for many years. Even today this relationship is continuously debated, with proponents remaining on both sides of the discussion. Recent data do not support the traditional belief that the static relationship of the teeth is strongly associated with TMD (e.g., deep bites, class II, cross bites, eccentric contacts) [5]. Yet to believe that the occlusal condition could not influence masticatory system function and dysfunction seems rather naive. Perhaps instead of studying static occlusion relationships, one needs to investigate some of the dynamic functions of the masticatory system. There appears to be two ways the occlusal relationship of the teeth may be associated with TMD symptoms. The first is related to an acute change in the occlusal condition, and the second is related to loading of the masticatory structures in the absence of TM joint stability. Each will be further explained here.

2.2.1.1 An Acute Change in the Occlusal Condition

Every dentist has observed a situation when a crown or filling is placed and it is left a little high; afterward, the patient will often report back to the office complaining of discomfort. Frequently this discomfort is not only around the sore tooth, but there also is muscle tightness and pain. This occurs because in the presence of injury or even a threat of injury, the muscles protectively co-contract to minimize any damage. This muscle response can lead to pain, especially if the condition is prolonged. Once the offending restoration is corrected, the condition resolves. If it is not corrected in a reasonable amount of time, either the individual will adapt to the change (i.e., by tooth movement, altered biting, or avoidance), or a significant muscle TMD may develop.

2.2.1.2 Orthopedic Instability Coupled with Loading

There is a second mechanism by which the occlusal condition can contribute to a TMD. This relates to the degree of orthopedic stability in the masticatory system. Every mobile joint is designed to be loaded, and this loading comes from the muscles that pull across the joint. Therefore every joint has a musculoskeletally stable position, and in the TMJ this is defined as the condyles resting on the articular eminences with the disks correctly positioned between those articulating surfaces. Orthopedic stability in the masticatory system is present when the teeth are in their stable biting position at the same time the joints are in their stable position. When this is

present, joints and teeth can be loaded without injury or consequence.

However, when the stable joint position is not in harmony with the stable occlusal position, the condition is considered orthopedically unstable. If this were the critical factor leading to TMD, epidemiologic studies should reveal this relationship, but clearly this is not the case. Perhaps the missing element from those studies is the dynamics of loading. When the teeth are loaded by activities such as heavy biting, chewing, or bruxism, the joints need to be in a stable position. When this does not exist, continued loading can result in changes in the joint structures. Common types of changes are fibrous connective tissue breakdown, bony degeneration, clicking, locking, and pain. It is important to appreciate that a lack of orthopedic stability between the stable joint position and the stable occlusal position does not by itself lead to TMD; this only represents a risk factor. However, once this relationship is coupled with excessive loading in a susceptible patient, there is an increased risk of developing intracapsular disorders [2, p. 102–28].

Thus, it is interesting to note that occlusion can affect both muscle disorders and intracapsular disorders, but it does so through different mechanisms. The manner by which occlusion affects TMDs can be summarized by the following two statements: Problems that occur while bringing the teeth into occlusion, such as high restorations, are answered by the muscles. However, once the teeth have reached intercuspation, problems with loading are answered by the joints.

2.2.2 Trauma

Certainly trauma is a known etiology of certain TMDs. A single blow to the face can immediately change the structures of the joint, resulting in an intracapsular issue. Trauma seems to be more related to intracapsular disorders than muscle disorders. It is common to hear a patient report that "ever since I received the blow to my face, my TMJ has been clicking." Once joint pain begins, muscles protectively respond and then it may be difficult to separate the painful conditions. A sudden blow to the face represents macrotrauma. However, microtrauma can also be an issue whereby small but repeated traumas can occur to the joints. The orthopedic instability coupled with loading previously mentioned is an example of microtrauma.

2.2.3 Emotional Stress

There is ample evidence that increased levels of emotional stress can be an etiologic factor associated with TMDs. It has been demonstrated that individuals placed under acute emotional stress show slight increase in EMG activities of their masseter muscles [6]. This is normal, but if the stress is prolonged the muscle may show signs of fatigue, tightness, and pain. Prolonged stressors can result in an increase or upregulation of the autonomic nervous system [7]. When this occurs, the central nervous system can play an active role in maintaining the pain condition, making management more difficult.

2.2.4 Deep Pain Input

Deep pain input refers to any source of neural impulses that originate in the deep structures and lead to a pain experience. This excludes the skin and oral mucosa. Common sources of deep pain input are muscle and joint structures. Pain experienced in the deep structures has the unique characteristic of eliciting a muscle response, which is the same protective co-contraction response already discussed in the section on occlusion. The clinician must appreciate that deep pain input can have its origin in many structures [8]. A common example is cervical pain that elicits a masticatory muscle response. A patient who experiences a whiplash injury initially experiences only cervical pain. However, after a few days the pain will often radiate to the face, eliciting a muscle response that limits mouth opening. The clinical examination will reveal limited mouth opening and pain upon palpation of the muscles of mastication, which in fact is a TMD. However, this

TMD is secondary to another pain disorder and will continue until the primary source of pain is resolved. Clinicians often overlook this relationship and question why their therapies that have been directed to the masticatory structures (such as an occlusal appliance) do not resolve the pain.

2.2.5 Parafunctional Activities

For many years dentists have focused on bruxing and clenching as a significant etiologic factor associated with TMDs. Although this activity can certainly be related, it is not as strongly linked as once believed. We know that bruxing and clenching of the teeth can produce pain [9]. However, sleep studies reveal that most individuals put their teeth together during sleep, often with no pain associated. We have also learned that the patients' occlusal relationships are not strongly related to these parafunctional activities. Instead, they are more correlated with sleep stages and other aspects of the sleep cycle. We have also learned that many individuals clench their teeth during the day with very little awareness. Patients who report that they wake up in the morning with painful muscle are certainly likely to be experiencing sleep related bruxism, and in those cases that can be considered as an etiologic relationship. However, there are other patients who report no pain upon awakening but instead their pain is in the late afternoons or evening. These individuals may be experiencing daytime clenching, or they may have a completely different etiologic basis for their myogenous pain. It is important to appreciate that these activities are different and likely to respond to different treatment strategies [2, p. 291–316].

The five etiologic factors that have been reviewed above reveal that TMDs are a complex group of conditions that are influenced by multiple factors. Making it even more complicated is the fact that more than one of these factors may be involved at any given time, which is often the case. This becomes a real challenge for the clinician attempting to initiate treatment strategies. Furthermore, we need to recognize the fact that all individuals are different in their capacity to adapt to less than ideal circumstances. Most people have less than perfect occlusion, have received some trauma, have some emotional stress, have experienced deep pain, and have some parafunction, and yet they do not develop TMD symptoms. This is likely due to their capacity for adaptability, which is an important clinical consideration since it helps us understand the great variability of patient responses. Clinicians need to appreciate patient adaptability, since it is probably the major reason for our clinical success. Yet, we actually know little about this important issue.

A better understanding of human adaptability would likely lead to better selection of treatment and prediction of outcomes. A more complete understanding of adaptability would be helpful, but investigating this concept is certainly not an easy task. There are likely many variables that contribute to adaptability. A few of the factors may include the individual's biology, learned experiences, psychological conditions (e.g., obsessive compulsive disorders), and genetics.

Some recent genetic studies are offering interesting insights about adaptability, especially with respect to pain. It has been demonstrated that variations in genetic makeup may have significant impact on pain perception [10, 11]. The gene that encodes for catechol-O-methyltransferase (COMT), an enzyme associated with pain responsiveness, varies in patients. It has been shown that for this gene, there are three clusters of individuals who respond differently to painful stimuli. Some individuals are more pain sensitive, while others less pain sensitive. In an interesting prospective cohort study of 186 orthodontically treated females, patients who were genetically in the pain sensitive cluster developed more TMD symptoms than the pain insensitive cluster group [12]. This suggests that the actual orthodontic therapy was not the significant factor in developing TMD; instead, it was performing orthodontic therapy in the patient with a genetically determined pain sensitive haplotype. Perhaps future research will help us recognize the patients who are more vulnerable to develop pain disorders, which may affect our choices of treatment options.

When considering all these issues, assuming that orthodontic therapy is completely unrelated to TMD is a relatively naïve thought. The question that really needs to be asked is how can orthodontic therapy be used to minimize any risk factors that may relate to TMD? In reviewing the known etiologies of TMD, orthodontic therapy routinely affects only one of those factors: occlusion. However, it is clear that occlusion factors are not always related to TMD [2, p. 102–28, 13]. So, where does orthodontic therapy fit in the big picture of TMD? Since occlusal factors may be a potential source of TMD in some patients, it would seem logical that the orthodontist should develop an occlusion condition that will minimize any risk factors that might be associated with TMD. However, developing a sound occlusal relationship does not mean the patient will not develop TMD, because there are at least four other etiologies that are outside the control of the orthodontist. Developing an orthopedically stable occlusal condition should be thought of as minimizing a dental risk factor. It seems logical that since orthodontic therapy will change the patient's occlusal relationships, emphasis should be placed on creating an occlusion condition that will provide the best opportunity for successful masticatory function for the lifetime of the patient.

2.3 Classification of Temporomandibular Disorders

Most temporomandibular disorders fall into one of two broad categories: muscle pain disorders or intracapsular disorders. Muscle pain disorders are by far the more common of these two problems [4, 14]. This is not surprising, since all humans experience some type of muscle pain periodically throughout their lives.

Masticatory muscle pain complaints are very common in patients seeking treatment in the dental office. With regard to orofacial pain, they are second only to odontalgia (i.e., tooth or periodontal pain) in terms of frequency. They are generally grouped in a large category known as masticatory muscle disorders [2, p. 291–316]. The most common symptoms reported by patients with these muscle disorders are pain associated with functional activities (i.e., chewing) and dysfunction (limitation in mouth opening).

2.3.1 Masticatory Muscle Disorders

Patients who experience masticatory muscle pain will describe the pain in terms of ranging from slight tenderness to extreme discomfort. Although muscle pain is common, dentists have generally not been taught well regarding its etiology. In fact, most dentists have been taught that muscle pain is a reflection of a structural problem, such as a poor occlusion or an incorrect joint position. They also associate muscle pain with bruxing and clenching activities. Although some of these thoughts may be true for some patients, they are not the etiologic basis for myogenous TMDs in a large number of patients.

Some muscle pain may arise from increased levels of muscular use. The symptoms are often associated with a feeling of muscle fatigue and tightness. Although the exact origin of this type of muscle pain is debated, some researchers have reported that it is related to vasoconstriction of the relevant nutrient arteries and the accumulation of metabolic waste products in the muscle tissues. Within the ischemic area of the muscle certain algogenic substances (e.g., bradykinins, prostaglandins) are released, causing muscle pain [15–20].

Muscle pain, however, is far more complex than simple overuse and fatigue. In fact, muscle pain associated with most TMD does not seem to be strongly correlated with increased activity such as spasm, or even with the burden of daily activities like hard chewing, cheerleading, singing, etc. [6, 21–24]. It is now appreciated that muscle pain can be greatly influenced by central nervous system mechanisms [16, 25, 26], especially if it has been present for a prolonged period of time.

One of the significant clinical findings with muscle pain disorders is that the pain is increased with function. Therefore, patients often report

that the pain affects their ability to chew and even talk. However, these functional activities are not usually the cause of the disorder, but instead they heighten the patient's awareness of it. More likely some other type of activity or a central nervous system effect has led to the muscle pain [27]. Therefore, directing treatment toward the functional activity itself will not be appropriate or successful. Instead, treatment needs to be directed towards diminishing the CNS effects and/or possibly muscle hyperactivity.

Many clinicians consider all masticatory muscle disorders to be the same. If this were the case it would certainly make treatment considerations quite simple. However, experienced clinicians realize that this is not the case, since all muscle pain disorders do not successfully respond to the same treatment. There are at least five different clinical presentations of muscle pain, and being able to distinguish among them is important because the treatment of each is quite different. The five types are protective co-contraction (muscle splinting), local muscle soreness, myofascial (trigger point) pain, myospasm, and chronic centrally mediated myalgia [2, 27, p. 129–69]. The first three conditions (protective co-contraction, local muscle soreness, and myofascial pain) are commonly seen in the dental office while myospasm and chronic centrally mediated myalgia are less frequently seen. Many of these muscle disorders appear and resolve in a relatively short period of time, sometimes without any type of professional treatment. For most cases a conservative regimen of treatment will be successful. However, when these conditions do not resolve, more chronic pain disorders may result. Chronic masticatory muscle disorders become more complicated and treatment is generally oriented differently than for acute problems. It therefore becomes important that the clinician be able to separate acute muscle disorders from chronic disorders so that proper therapy can be applied.

Since the intent of this chapter is not to review all muscle conditions, only the most common disorders orthodontists will encounter in their practices will be discussed. These are local muscle soreness and myofascial pain.

2.3.1.1 Local Muscle Soreness

Local muscle soreness is the most common type of acute muscle pain seen in the dental practice. It represents a condition that is characterized by changes in the local environment of the muscle tissues. These changes arise from the release of certain algogenic substances (i.e., bradykinin, substance P, histamine [28]) that produce pain. These initial changes may represent nothing more than fatigue. The most likely causes of local muscle soreness are overuse of the muscle or trauma. Overuse may be associated with a protective co-contraction of the muscle secondary to an acute change in sensory input or emotional stress. Trauma may be caused by a direct blow to the muscle, but a more likely reason is simply unaccustomed use of the muscle. When excessive use is the etiology a delay in the onset of muscle soreness can occur [29]. This type of local muscle soreness is often referred to as delayed onset muscle soreness or post exercise muscle soreness [30–34].

Clinically, local muscle soreness TMD patients present with muscles that are tender to palpation and they report increased pain with function. When the elevator muscles are involved, the patient will report limited mouth opening which is secondary to the pain. This means the patient can open wider but is not willing to do this because it increases the pain. The patient may also report muscle weakness [35–37] which usually will be returned to normal strength once the muscle soreness has been resolved [36–38].

2.3.1.2 Myofascial Pain

Myofascial pain is a regional myogenous pain condition characterized by local areas of firm, hypersensitive bands of muscle tissue known as "trigger points." Myofascial pain is common yet not widely appreciated or completely understood. In one study [39] more than 50 % of the patients reporting to a university pain center were diagnosed as having this type of pain.

The trigger points are often felt as taut bands when palpated, which elicit pain. The exact nature of a trigger point is not known. It has been suggested [40–42] that certain nerve endings in the muscle tissues may become sensitized by algogenic substances that create a localized zone of

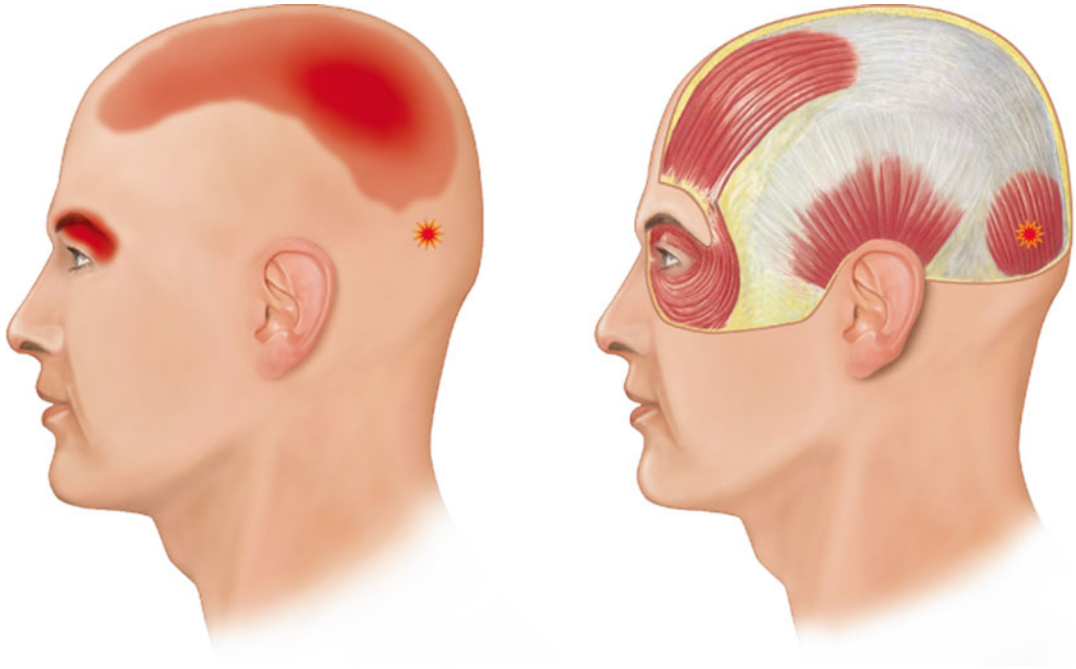

Fig. 2.1 Note how a trigger point (marked with X) in the occipital belly of the occipitofrontalis muscle produces referred headache pain behind the eye (in red) (From Okeson [2], p. 133)

hypersensitivity [43]. There may be a local temperature rise at the site of the trigger point, suggesting an increase in metabolic demand and/or reduction of blood flow to these tissues [44, 45]. A trigger point is a very circumscribed region in which just a relatively few motor units seem to be contracting [46].

The unique characteristic of trigger points is that they are a source of constant deep pain and therefore can produce central excitatory effects. If a trigger point centrally excites a group of converging afferent interneurons, referred pain will often result, generally in a predictable pattern according to the location of the involved trigger point (Figs. 2.1 and 2.2) [2, 47, p. 21–45]. The pain is often reported by the patient as headache pain. In many instances patients may be aware only of the referred pain and not even acknowledge the trigger points. A perfect example is the patient suffering from myofascial trigger point pain in the trapezius muscle that creates referred pain to the temple region (Fig. 2.3) [47–49]. The chief complaint is temporal headache, with very little acknowledgment of the trigger point in the shoulder. This clinical presentation can easily distract the clinician from the source of the problem. The patient will draw the clinician's attention to the site of the pain (the temporal headache) and not the origin of the pain.

2.3.2 Temporomandibular Joint Disorders

Functional abnormalities of the temporomandibular joints are probably the most common findings one observes when examining a patient for masticatory dysfunction. The reason for this is due to the high prevalence of signs, and not necessarily symptoms. (See Chap. 3 for discussion of the significance of signs and symptoms discovered during TMD screening exams). Many of the signs such as joint sounds or deviated opening are not painful, and therefore the patient may not seek treatment. These TM joint disorders generally fall into two broad categories: Internal derangements and inflammatory joint disorders. These conditions will be described separately.

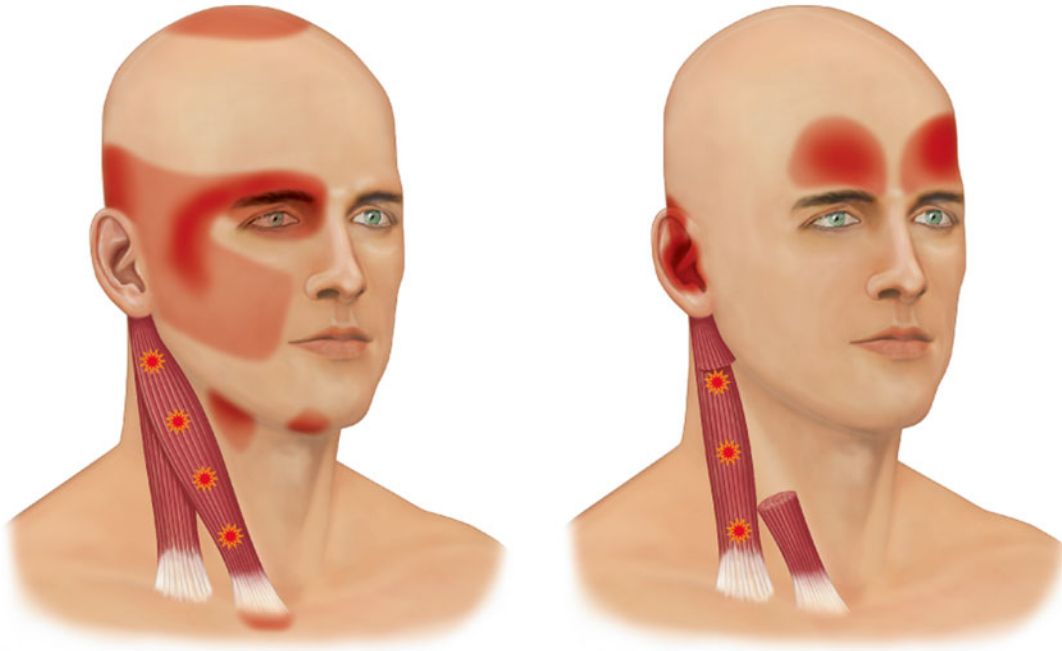

Fig. 2.2 Note how trigger points located in the sternocleidomastoideus refer pain to the preauricular (TMJ) area, the eye, the forehead, and the ear (From Okeson [2], p. 135)

2.3.2.1 Internal Derangements

Internal derangements represent a group of functional disorders that arise from abnormalities in the anatomy and/or positional relationships of the TM joint structures. A review of TMJ anatomy demonstrates that the disc is attached to the poles of the condyle by the medial and lateral collateral ligaments. Many internal derangement disorders arise from alterations of the integrity or lengths of these ligamentous attachments. Once these ligaments become elongated the disc is allowed more freedom to move within the joint. The disc will often begin to assume an anteromedial position in relationship with the condyle (Fig. 2.4). When the disc is in this more forward and medial position, function of the joint can be somewhat altered. As the mouth opens and the condyle moves forward, a short distance of translatory movement can occur between the condyle and the disc until the condyle once again assumes its normal position on the thinnest area of the disc (intermediate zone). Once it has translated over the posterior surface of the disc to the intermediate zone, inter-articular pressure due to joint loading maintains this relationship and the disc is again carried forward with the condyle through the remaining portion of the translatory movement. Upon closing, the disc reassumes its abnormal position on the condyle in the closed joint position. Once in the closed joint position, the disc is again free to move according to the demands of its functional attachments. In this condition, the disc will assume the most anteromedial position allowed by the discal attachments and its own morphology.

As the disc is more chronically repositioned forward and medially by action of the superior lateral pterygoid muscle, the discal ligaments are further elongated. With continuous thinning of the posterior border of the disc and further elongation of the disc ligaments, the disc can move through the discal space and be trapped anterior to the condyle. (Fig. 2.4). When this occurs, the condyle can now function or load the retrodiscal tissues which may be associated with pain.

The important feature of this functional relationship is that the condyle translates across the disc to some degree when movement begins. This type of movement does not occur in the normal joint. During such movement the increased

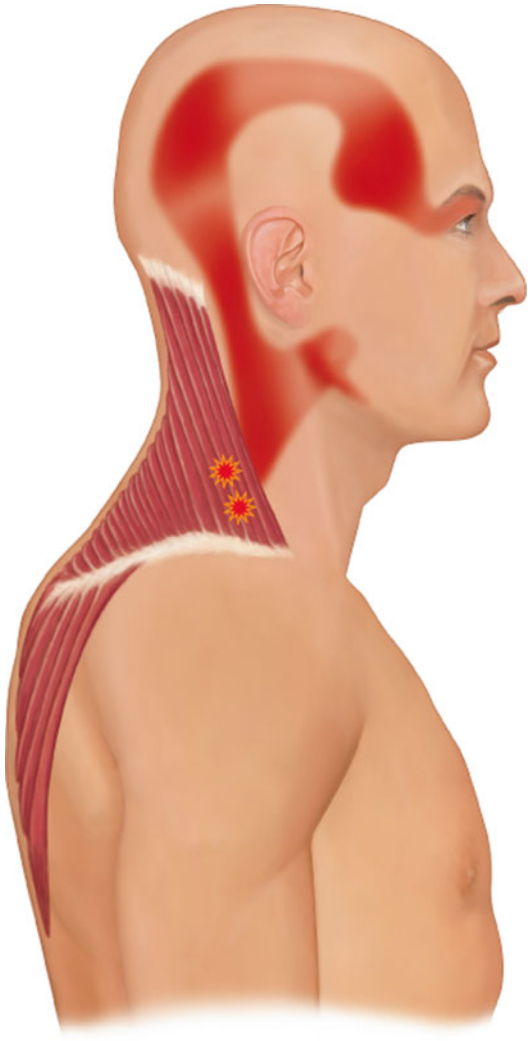

Fig. 2.3 Note how trigger points located in the trapezius muscle (marked with X) refer pain to behind the ear, the temple, and the angle of the jaw (From Okeson [2], p. 134)

interarticular pressure may prevent the articular surfaces from sliding across each other smoothly. The disc can stick or be bunched slightly, causing an abrupt movement of the condyle over it into the normal condyle-disc relationship. A clicking sound often accompanies this abrupt movement. Once the joint has clicked, the normal relationship of the disc and condyle is reestablished and this relationship is maintained during the rest of the opening movement. A second click can occur as the disc is re-displaced during the later stages of closing the mouth. This is called "reciprocal clicking" [50]. As the disc displacement progresses, the condyle actually begins to function behind the disc with loading occurring on the retrodiscal tissues (Fig. 2.4).

As the disc is more chronically repositioned forward and medially, the discal ligaments are further elongated. With continuous thinning of the posterior border of the disc and further elongation of the disc ligaments, the disc can move further forward and be trapped anterior to the condyle. This often is accompanied by folding of the disc into a ball-like shape. When this occurs it will initially lead to a decrease in how far the condyle can move forward. Therefore the patient will have the sensation that he or she cannot open the mouth completely. This has been called a "closed lock" (Fig. 2.5) [50] since the patient feels he or she is locked near the closed mouth position. Patients may report pain when the mandible is moved to the point of limitation, but pain does not always accompany this condition [51–54].

If a closed lock continues, the condyle will be constantly positioned on the retrodiscal tissues. These tissues are not anatomically structured to accept forces, but they often remodel to form a functional pseudodisc. However, as force is applied, some likelihood arises that these tissues may break down in some patients [55–57]. With this breakdown comes tissue inflammation and pain (retrodiscitis).

It is important to appreciate that pain is not always a factor with these conditions. Pain is not generated by the disc, since it is aneural. The structures that are able to produce pain are the connective tissues such as the ligaments and the very highly innervated retrodiscal tissues. If these structures are loaded quickly due to a sudden disc shifting, pain is likely. However, if the changes occur slowly over time, these tissues are often able to adapt and pain may not be associated.

Etiology of Internal Derangements

Any condition or event that leads to elongation of the discal ligaments or thinning of the disc can cause these derangements of the condyle-disc complex disorders. Certainly one of the most common factors is trauma. Two general types of trauma need to be considered: macrotrauma and

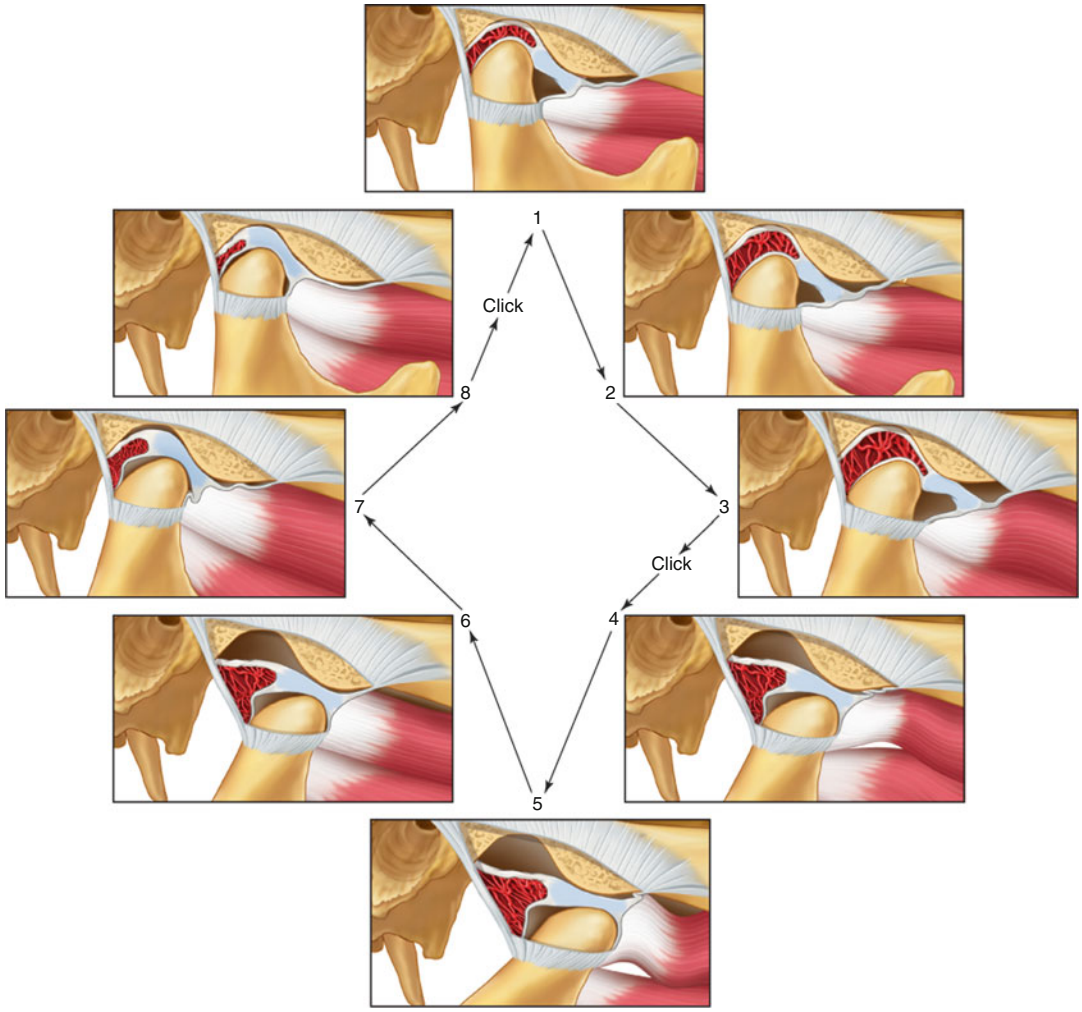

Fig. 2.4 Functional displacement of the disc with reduction. Note that while the mouth is closed (stage 1) the disc is displaced anterior to the condyle. During opening the condyle passes over the posterior border of the disc onto the intermediate area of the disc, thus reducing the displaced disc (stage 4). At this stage a click is felt. During the rest of the opening movement the condyle and disc function normally. During closing the disc is re-displaced (stage 8–1) and a second click is felt (*reciprocal click*) (From Okeson [2], p. 145)

microtrauma. Macrotrauma relates to a sudden blow to the face that can result in a quick elongation of ligaments. This is well documented in the literature [58–71].

Microtrauma represents lower levels of force but repeated over longer periods of time. It can result from joint loading associated with muscle hyperactivity such as bruxism or clenching [72, 73]. This may be especially true if the bruxing activity is intermittent and the tissues have not had an opportunity to adapt. It is likely that if the bruxing is long standing, the articular tissues have adapted to the loading forces and changes will not be seen. In fact, in most patients gradual loading of the articular surfaces leads to an adaptive, more tolerant articular tissue [74–76].

Microtrauma may be the result of mandibular loading in the presence of orthopedic instability, as stated in an earlier section. When orthopedic instability exists, repeated loading, such as clenching the teeth, can lead to slight movements of the condyle resulting in microtrauma to the ligaments. The results can be elongation of these ligaments and eventually

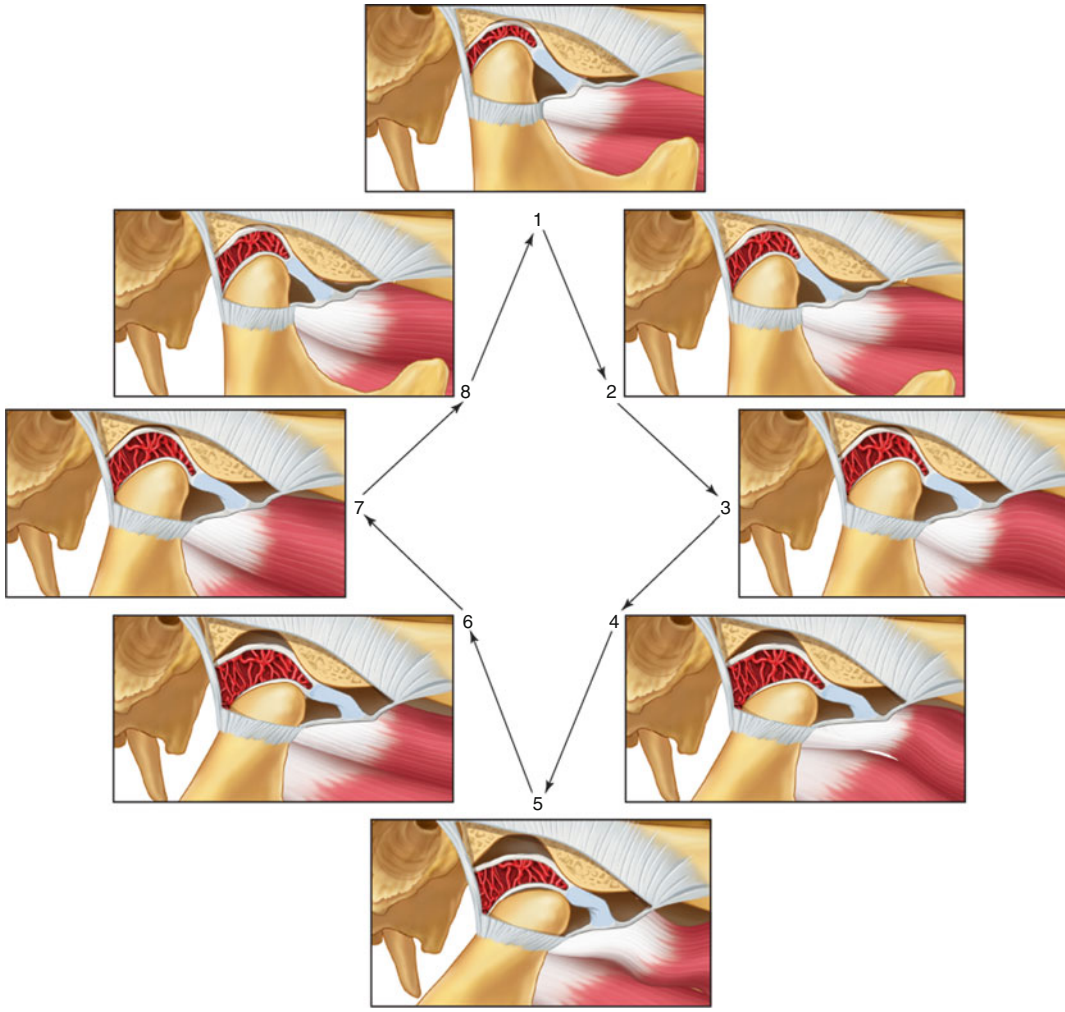

Fig. 2.5 Functional displacement of the disc without reduction Note that during opening the condyle never assumes a normal relationship on the disc but instead causes the disc to move forward ahead of it. This condition limits the distance it can translate forward (*closed lock*). Clicking often resolves when this occurs (From Okeson [2], p. 146)

disc movements. Remember that the amount and intensity of the loading greatly influence whether the orthopedic instability will lead to a disc derangement disorder. Bruxing patients with orthopedic instability, therefore, are more likely to develop problems than non-bruxers with the same occlusion.

An important question that arises in dentistry is "What occlusal conditions are commonly associated with internal derangements?" The most orthopedically stable position of the condyle is in the superior anterior position resting against the posterior slope of the articular eminence. This is referred to as the musculoskeletally stable position [2, p. 291–316] and it is determined by the loading forces of the elevator muscles. It has been demonstrated that when an occlusal condition causes a condyle to be positioned posterior to the musculoskeletally stable position the posterior border of the disc can be thinned [77]. A common occlusal condition that has been suggested by some orthodontists to produce this problem is the skeletal Class II deep-bite malocclusion; advocates of this concept also

believe that this situation may be further aggravated when a Division 2 anterior relationship also exists [78–82]. However, most studies show no relationship between Class II malocclusion and these disorders [13, 83–89]. Other studies show no association between the horizontal and vertical relationship of the anterior teeth and disc derangement disorders [90–94]. While occlusal conditions are not the main etiologic factors for internal derangements, the important feature of an occlusal condition that leads to disc derangement disorders is the lack of joint stability when the teeth are tightly occluded. Therefore, it is likely that some Class II malocclusions provide joint stability (a stable malocclusion) while others do not; but the same can be said for every type of static malocclusion category.

It is obvious that no simple relationship exists between orthopedic instability and intracapsular disorders. It is very important, however, that when orthopedic instability exists, it be identified as a potential etiologic factor. It should be noted that orthodontic therapy can be a viable treatment for orthopedic instability and may need to be considered when this instability has been determined to be a contributing factor to a TMD.

2.3.2.2 Osteoarthritis

When internal derangements of the TMJ occur, the structures affected by these changes often respond. The most common tissues affected are the retrodiscal tissues and the articular surfaces of the condyle and articular eminence. These changes can result in adaptation or destruction, depending upon many factors. Some of these factors are the acuteness of the changes as well as the intensity and duration of the loading. There are also important biologic and genetic factors that may regulate the patient's ability to repair tissues.

If the articular surfaces of the condyle become affected, the subarticular bone receives additional loading and changes can occur. Similar changes also can occur in the absence of internal derangements. These changes represent a group of disorders that are considered joint arthritides. The most common type of TMJ arthritis is osteoarthritis (sometimes called degenerative joint disease). Osteoarthritis represents a destructive process by which the bony articular surfaces of the condyle and fossa become altered. It is generally considered to be the body's response to increased loading of a joint [95]. As loading forces continue and the articular surface becomes softened (chondromalacia), the subarticular bone begins to resorb. Progressive degeneration eventually results in loss of the subchondral cortical layer, bone erosion, and subsequent radiographic evidence of osteoarthritis [96]. It is important to note that radiographic changes are only seen in later stages of osteoarthritis and may not reflect the clinical symptoms accurately.

Osteoarthritis is often painful, and jaw movement accentuates the symptoms. Crepitation (multiple grating joint sounds) is a common finding with this disorder. Osteoarthritis can occur any time the joint is overloaded, but is most commonly associated with disc displacements [97, 98] or perforation [99]. Once the disc is displaced and the retrodiscal tissues break down, the condyle begins to articulate directly with the fossa accelerating the destructive process. In time, the dense fibrous articular surfaces are destroyed and bony changes occur. Radiographically, the surfaces seem to be eroded and flattened. Any movement of these surfaces creates pain, so jaw function usually becomes very restricted. Although osteoarthritis is in the category of inflammatory disorders, it is not a true inflammatory condition. With appropriate treatment and reduction of joint loading, the arthritic condition can become adaptive. The adaptive stage has been referred to as osteoarthrosis [95, 100].

Other types of arthritides can certainly affect the temporomandibular joint. The most common after osteoarthritis is rheumatoid arthritis. This is thought to be an autoimmune disorder and therefore has its origin in systemic factors. The juvenile form of this problem can produce significant joint changes as well as serious occlusal problems. Other common causes of TMJ arthritis are traumatic arthritis, infectious arthritis, psoriatic arthritis, and hyperuricemia (gout). These conditions are not reviewed in this chapter. Other texts can be reviewed for a more thorough review of TMJ arthritides [2, p. 317–61].

2.4 Summary of the Continuum of TMJ Intracapsular Conditions

Disorders of the temporomandibular joints may follow a path of progressive events, i.e., a continuum, from the initial signs of dysfunction to osteoarthritis. They are summarized in Fig. 2.6.

Although this continuum is logical, the question must be asked whether these stages are always progressive for every patient. It is a question of great significance, because if all patients continue to progress in this manner, then there would be a professional obligation to resolve any joint symptoms as soon as they first appear. The sequence of breakdown as summarized in Fig. 2.6 is logical and has clinical support [101–103]. However, clinical longitudinal studies have clearly shown that some intracapsular TMD patients will present in one stage but may not necessarily progress to the next. At any given stage of disc derangement the patient may reach a level of adaptability and no further progression or breakdown will occur [104, 105]. This can be supported by histories of asymptomatic single and reciprocal clicks over many years [106]. Perhaps the key to determining who needs treatment lies in the obvious progression from one stage to the next. Also the presence of pain is important, since it implies continuous breakdown; in any case, the pain should be treated for its own sake. Therefore, it is this

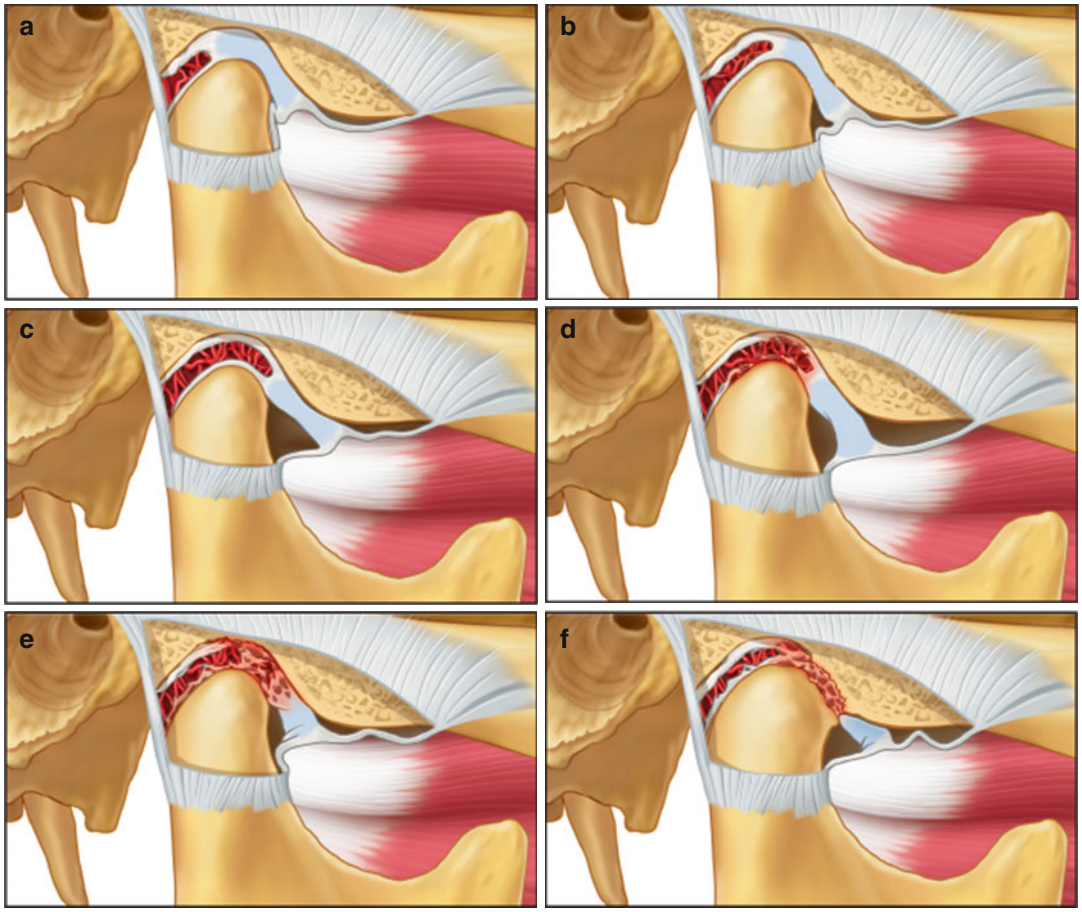

Fig. 2.6 Various states of internal derangement of the TMJ. (**a**) normal joint, (**b**) partial displacement of the disc, (**c**) complete displacement of the disc, (**d**) impingement of retrodiscal tissues, (**e**) retrodiscitis and tissue breakdown, (**f**) osteoarthritis (From Okeson [2], p. 156)

author's opinion that treatment for such patients needs to be instituted when pain is associated with the condition. The treatment should be directed toward controlling pain and changing loading, thereby allowing a better opportunity for the tissues to repair and eventually adapt. Treatment of these conditions is beyond the objectives of this chapter and therefore other sources should be pursued.

Conclusion

Temporomandibular disorders are common conditions that may be encountered by orthodontists: during pre-treatment screening, during their treatment procedures, or during orthodontic retention. Every orthodontist needs to have a basic understanding of these musculoskeletal disorders so that he or she can respond to their patients' needs. The purpose of this chapter was to present the pathophysiology, etiology, and clinical characteristics of the most common temporomandibular disorders. The goal was not to provide detailed therapeutic considerations for TMDs. However, it should be mentioned that most TMDs can be successfully managed by very conservative, reversible treatments. This should always be the initial approach before any irreversible treatments are considered.

Although some patients with TMDs may respond to orthodontic therapy, most will not because their occlusal conditions are not the cause of their symptomatology. Also, the positive responses seen may simply be due to placebo effects, spontaneous improvements, or the passage of time. Therefore, the orthodontist needs to understand which TMD patients will benefit before any orthodontic therapy is begun, and that treatment should not be initiated until acute symptoms of pain and dysfunction have been addressed. A review of the five etiologic factors presented in this chapter reveals that orthodontic therapy potentially affects only one of them, i.e., the occlusal condition. The manner by which orthodontic therapy affects this factor is by providing orthopedic stability, because proper orthodontic therapy can provide a stable occlusal position in the most stable joint position. Accomplishing this provides orthopedic stability in the masticatory structures, which minimizes risk factors associated with TMDs. Since orthodontic treatment will always disrupt the existing occlusal and TMJ relationships, establishing this orthopedic stability at the end of treatment should be the goal for every patient who receives orthodontic therapy.

When a patient presents to the orthodontist with a TMD, the etiology of the TMD needs to be determined before any treatment is begun. Assuming that the patient's malocclusion is the major etiologic factor causing the TMD is a very naïve assumption. Nothing is more discouraging to the patient (and doctor) than to provide excellent orthodontic treatment for 2 years and then hear the patient report that the pain is still present. Although the orthodontic therapy may have been successful, the orthodontist has failed to successfully treat the patient. Therefore, before beginning orthodontic therapy on any symptomatic patient, the clinician needs to confirm that orthopedic instability is an etiology for that patient, because this is the only scenario in which orthodontics can be considered as an appropriate treatment for TMD.

Take Home Messages

- TMD signs and symptoms are common in the general population, but only a small percentage of those require treatment.
- Orthodontists need to be aware how their treatments can affect masticatory function.
- There are five recognized etiologic factors associated with TMD.
- Muscle pain is the most common painful TMD encountered in the orthodontic practice.

- Internal derangements are associated with TMJ conditions and joint sounds are the most common sign.
- Most TMD symptoms can be managed by conservative approaches.
- Treatment goals for all orthodontists should include developing or maintaining orthopedic stability in the masticatory system.

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