

# 10



## TEMPOROMANDIBULAR DISORDERS

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This chapter focuses on the assessment and management of disorders of the masticatory system. The masticatory apparatus is a specialized unit that performs multiple functions, including those of suckling, speaking, cutting and grinding food, and swallowing. The loss of these functions in association with pain is characteristic of masticatory system disorders and causes significant distress that can be severely disabling.

In the past, disorders of the masticatory system were generally treated as one condition or syndrome, with no attempt to differentiate subtypes of muscle and joint disorders. With increased understanding, the ability to identify different muscle or joint disorders has become possible; this should lead to a better understanding of the natural course, more accurate predictions on prognosis, and more effective treatments of temporomandibular disorders (TMD). The term “temporomandibular disorders” (TMD), used in this chapter, is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ) and associated structures, or both.<sup>1</sup> These disorders are characterized by (1) facial pain in the region of the TMJ and/or the muscles of mastication, (2) limitation or deviation in the mandibular range of motion, and (3) TMJ sounds during jaw movement and function.<sup>2</sup>

The cause of most TMD remains unknown although numerous hypotheses have been proposed. The relationship of occlusal disharmony and TMD became a focus after Costen reported that a group of patients with multiple complaints around the jaws and ears improved after their occlusal-vertical dimension was altered.<sup>3</sup> The occlusal hypothesis was then expanded to include other occlusal discrepancies in addition to loss of vertical dimension.<sup>4</sup> During the 1950s and 1960s, a muscular cause not directly related to occlusion was proposed.<sup>5-7</sup> In the late 1970s, advances in diagnostic imaging resulted in a better understanding of the intracapsular problems associated with TMD.<sup>8,9</sup> The lack of a clear understanding with regard to cause, the existence of multiple hypotheses,

and strongly held beliefs by some clinicians have resulted in a wide spectrum of treatments being offered. Standardized methods for assessment, classification, and treatment do not exist, and this has impeded the ability to interpret the existing research and make comparisons between studies. This chapter presents a general approach to the diagnostic assessment and nonsurgical management of the most common TMD.

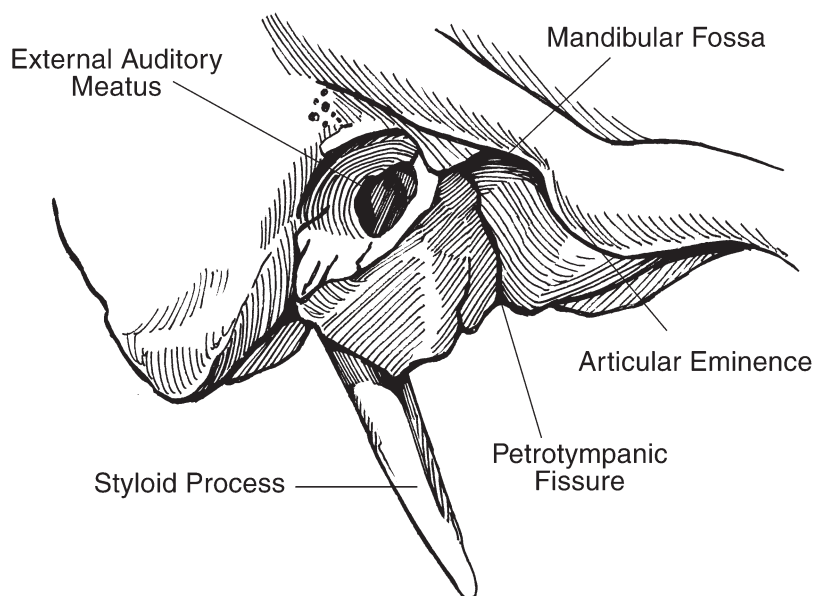
## ▼ FUNCTIONAL ANATOMY

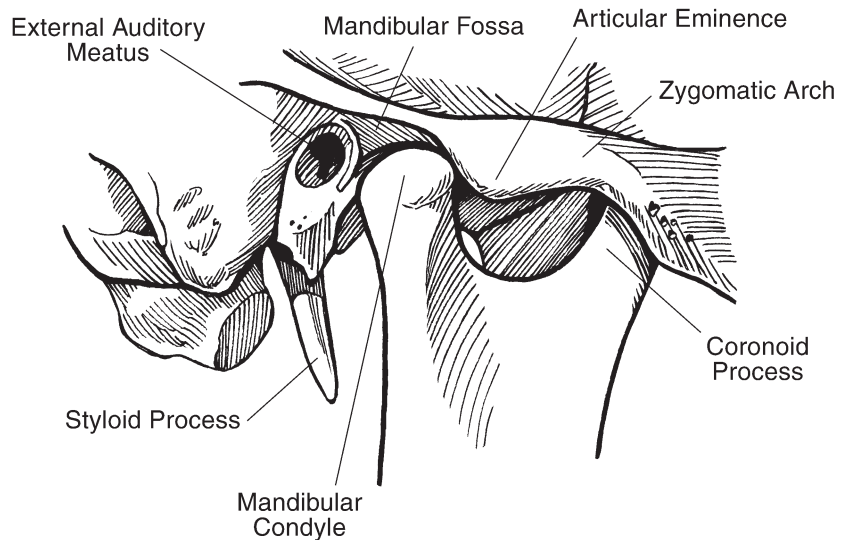
### Temporomandibular Joint

The TMJ articulation is classified as a ginglymodiarthrodial joint, namely, a joint that is capable of hinge-type movements (ginglymos) and gliding movements, with the bony components enclosed and connected by a fibrous capsule. The mandibular condyle forms the lower part of the bony joint and is generally elliptical although variations in shape are common.<sup>10</sup> The articulation is formed by the mandibular condyle occupying a hollow in the temporal bone (the mandibular or glenoid fossa) (Figures 10-1 and 10-2). The S-shaped form of the fossa and eminence develops at about 6 years of age and continues into the second decade.<sup>11</sup> During wide mouth opening, the condyle rotates around a hinge axis and glides, causing it to move beyond the anterior border of the fossa, the articular eminence.<sup>12</sup> The TMJ has a rigid end point when the teeth contact.

The capsule is lined with synovium and the joint cavity is filled with synovial fluid. Synovial tissue is a vascular connective tissue lining the fibrous joint capsule and extending to the boundaries of the articulating surfaces. Both upper and lower joint cavities are lined with synovium. Synovial fluid is a filtrate of plasma with added mucins and proteins. Its main constituent is hyaluronic acid. Fluid forms on the articulating surfaces, decreasing friction during joint compression and motion.<sup>13</sup> Joint lubrication is achieved by mechanisms described as weeping lubrication and boundary lubrication. Weeping lubrication

**FIGURE 10-1** The S-shaped form of the fossa and eminence develops at about 6 years and continues into the second decade. The mandibular condyle occupies the space of the fossa, with enough room to both rotate and translate during mandibular movements.





**FIGURE 10-2** The articulation is formed by the mandibular condyle occupying a hollow in the temporal bone (the mandibular or glenoid fossa) during wide mouth opening, the condyle rotates around an axis and glides, causing it to move beyond the anterior border of the fossa, the articular eminence.

tion occurs as fluid is forced laterally during compression and expressed through the unloaded fibrocartilage. As the adjacent areas become loaded, this weeping lubrication aids in reducing friction. Boundary lubrication is a function of water physically bound to the cartilaginous surface by a glycoprotein.

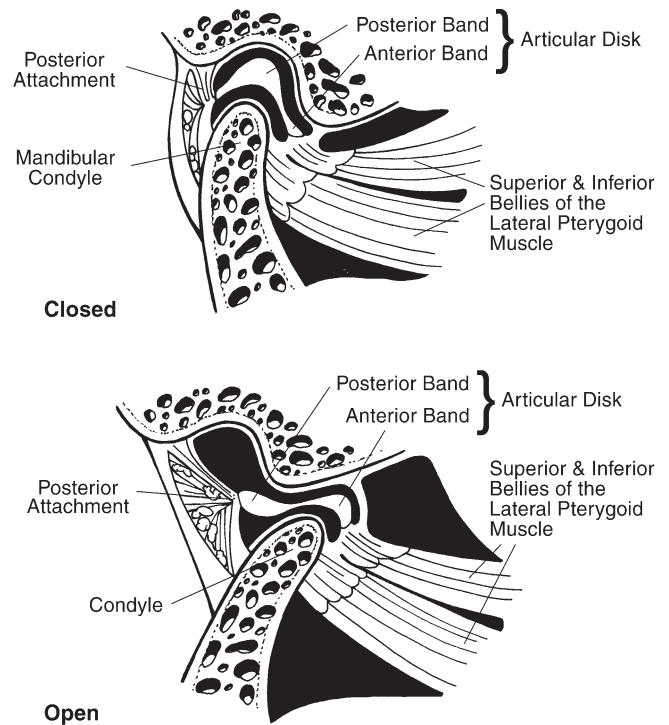
The distinguishing features of this joint include a covering of fibrocartilage over the articulating surfaces, rather than hyaline cartilage and its bilaterality. Fibrocartilage is less distensible than hyaline cartilage due to a greater number of collagen fibers. The matrix and chondrocytes are decreased because of the larger irregular bundles of collagen fibers. Fibrocartilage derives its nutrition from the diffusion of nutrients into the synovial fluid; these then diffuse through the dense matrix to the chondrocytes.

**ARTICULAR DISK**

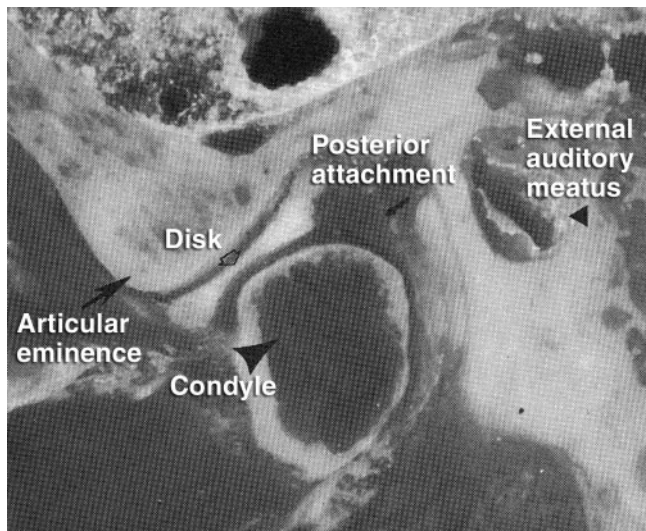
The space between the condyle and mandibular fossa is occupied by collagenous fibrous tissue of variable thickness, called the articular disk (Figures 10-3 and 10-4). The disk consists of collagen fibers, cartilage-like proteoglycans<sup>14</sup> and elastic fibers.<sup>15</sup> The disk contains variable numbers of cartilage cells and is referred to as a fibrocartilage. The collagen fibers in the center of the disk are oriented perpendicular to its transverse axis. The collagen fibers become interlaced as they approach the anterior and posterior bands, and many fibers are oriented parallel to the mediolateral aspect of the disk. The cartilage-like proteoglycans contribute to the compressive stiffness of articular cartilage.<sup>16</sup> The disk is attached to the lateral and medial poles of the condyle by ligaments consisting of collagen and elastic fibers.<sup>17</sup> These ligaments permit rotational movement of the disk on the condyle during the opening and closing of the jaw. The disk is thinnest in its center and thickens to form anterior and posterior bands. This arrangement is considered to help stabilize the condyle in the glenoid fossa. The disk is primarily avascular and has little sensory nerve penetration.

The disk provides an interface for the condyle as it glides across the temporal bone. The disk and its attachments divide

the joint into upper and a lower compartments that normally do not communicate. The passive volume of the upper compartment is estimated to be 1.2 mL, and that of the lower compartment is estimated to be 0.9 mL.<sup>17</sup> The roof of the superior compartment is the mandibular fossa whereas the floor is the superior surface of the disk. The roof of the inferior compartment is the inferior surface of the disk, and the floor is the artic-



**FIGURE 10-3** The temporomandibular joint is a ginglymoarthrodial joint that is capable of hinge-type movements and gliding movements. The articular disk has ligamentous attachments to the mandibular fossa and condyle. The disk's attachments create separate superior and inferior joint compartments.



**FIGURE 10-4** A cadaver section through the temporomandibular joint shows the relationship of the condyle, fossa, and articular disk.

ulating surface of the mandibular condyle.<sup>17</sup> At its margins, the disk blends with the fibrous capsule. Muscle attachments inserting into the anterior aspect of the disk have been observed.<sup>18</sup> Fibers of the posterior one-third of the temporalis muscle and deep masseter muscle may attach on the anterolateral aspect.<sup>18</sup> Fibers of the superior head of the lateral pterygoid have been observed to insert into the anteromedial two-thirds of the disk.<sup>18</sup>

#### RETRODISCAL TISSUE

A mass of soft tissue occupies the space behind the disk and condyle and is often referred to as the posterior attachment.

**FIGURE 10-5** The capsular ligament is a thin inelastic fibrous connective-tissue envelope, oriented vertically, that attaches to the margins of the articular surfaces. The capsular ligament does not restrain condylar movements. The temporomandibular ligament is lateral to the capsule but is not easily separated from it by dissection. Its fibers pass obliquely from bone lateral to the articular tubercle in a posterior and inferior direction to insert in a narrower area below and behind the lateral pole of the condyle.

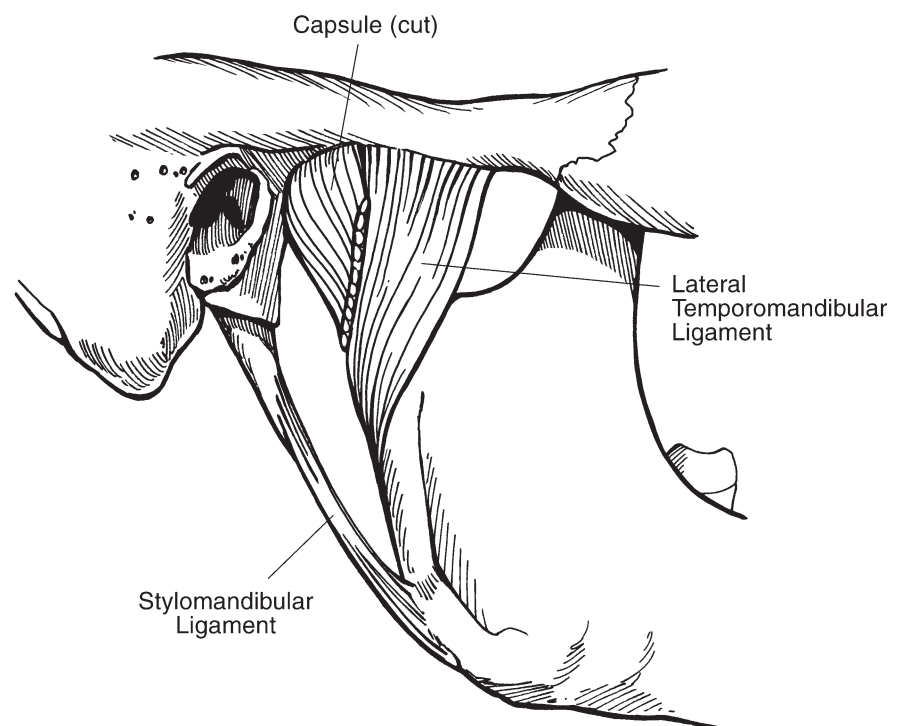
The posterior attachment is a loosely organized system of collagen fibers, branching elastic fibers, fat, blood and lymph vessels, and nerves. Synovium covers the superior and inferior surfaces. The attachment has been described as being arranged in two laminae of dense connective tissue,<sup>19</sup> but this has been challenged.<sup>20</sup> Between the laminae, a loose areolar, highly vascular, and well-innervated tissue has been described. The superior lamina arises from the posterior band of the disk and attaches to the squamotympanic fissure and consists primarily of elastin.<sup>19</sup> The inferior lamina arises from the posterior band of the disk and inserts into the inferior margin of the posterior articular slope of the condyle and is composed mostly of collagen fibers.<sup>19</sup>

#### CAPSULAR LIGAMENT

The capsular ligament is a thin inelastic fibrous connective-tissue envelope that attaches to the margins of the articular surfaces (Figure 10-5). The fibers are mainly oriented vertically and do not restrain joint movements.

#### TEMPOROMANDIBULAR LIGAMENT

The temporomandibular ligament is the main ligament of the joint, lateral to the capsule but not easily separated from it by dissection. Its fibers pass obliquely from bone lateral to the articular tubercle in a posterior and inferior direction and insert in a narrower area below and behind the lateral pole of the condyle (see Figure 10-5). In earlier literature, this ligament was identified as an oblique band from the condylar neck to the anterosuperior region on the eminence and as a horizontal band from the lateral condylar pole to an anterior attachment of the eminence.<sup>17</sup> A recent study was unable to confirm two distinct structures separate from the capsule.<sup>21</sup>



## Muscles Associated with Mandibular Movement and Function

### MUSCLES OF MASTICATION

The muscles of mastication are the paired masseter, medial and lateral pterygoid, and temporalis muscles (Figures 10-6, 10-7, and 10-8). Mandibular movements toward the tooth contact position are performed by contraction of the masseter, temporalis, and medial pterygoid muscles. Masseter contraction also contributes to moving the condylar head toward the anterior slope of the mandibular fossa. The posterior part of the temporalis contributes to mandibular retrusion, and unilateral contraction of the medial pterygoid contributes to a contralateral movement of the mandible. The masseter and medial pterygoid muscles have their insertions at the inferior border of the mandibular angle. They join together to form a sling that cradles the mandible and produces the powerful forces required for chewing. The masseter is divided into a deep portion and a superficial portion.

The temporalis muscle is broadly attached to the lateral skull and has been divided into anterior, middle, and posterior parts. The muscle fibers converge into a tendon that inserts on the coronoid process and anterior aspect of the mandibular ramus. The anterior and middle fibers are generally oriented in a straight line from their origin on the skull to their insertion on the mandible. The posterior part traverses anteriorly

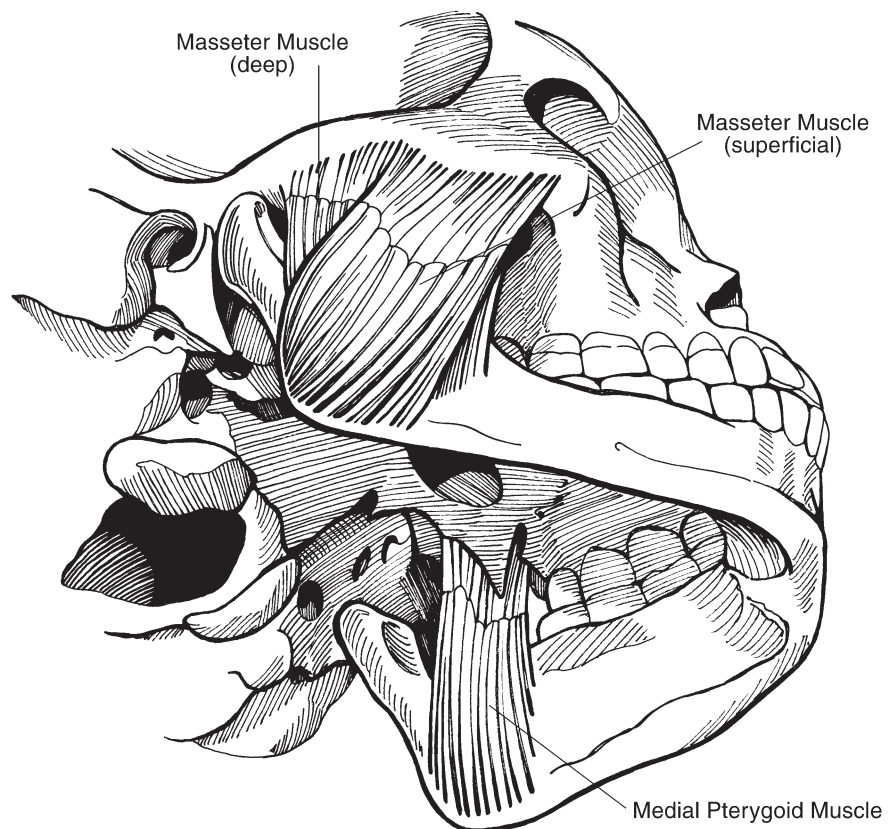
then curves around the anterior root of the zygomatic process before insertion.

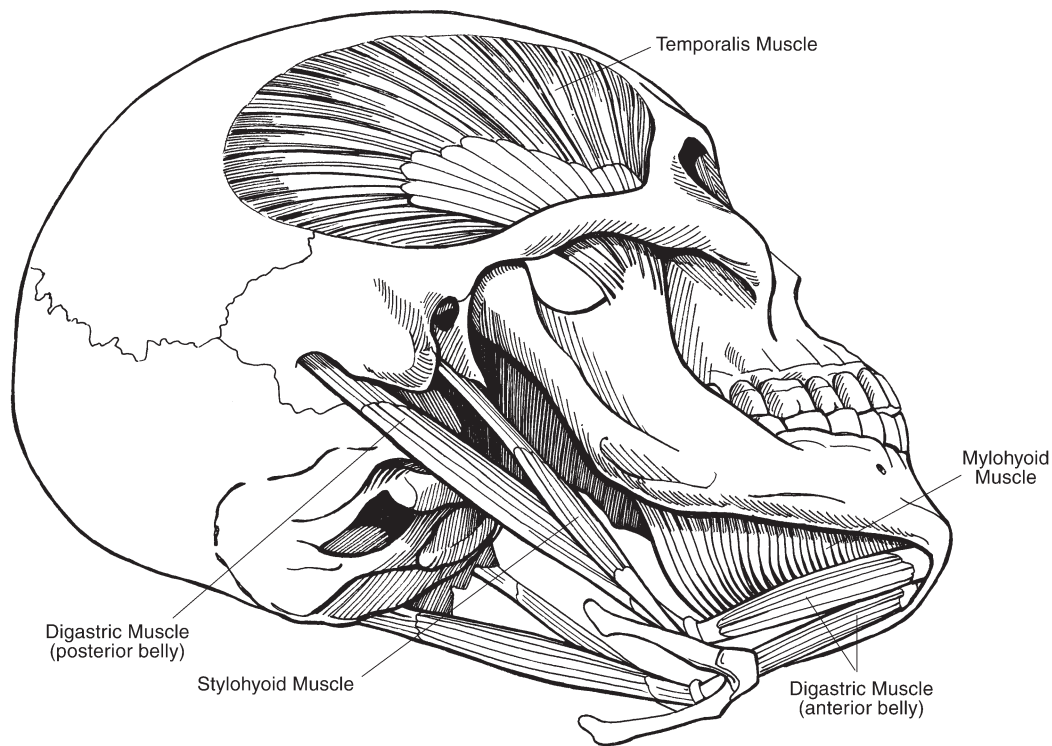
The lateral pterygoid is the main protrusive and opening muscle of the mandible. It is arranged in parallel-fibered units whereas the other muscles are multipennated. This arrangement allows greater displacement and velocity in the lateral pterygoid and greater force generation in the jaw-closing muscles.<sup>22</sup> The lateral pterygoid muscle is divided into two parts. The inferior part arises from the outer surface of the lateral pterygoid plate of the sphenoid and the pyramidal process of the palatine bone. The superior part originates from the greater wing of the sphenoid and the pterygoid ridge. The fibers of the upper and lower heads course posteriorly and laterally, fusing in front of the condyle.<sup>23</sup> They insert into the anteromedial aspect of the condylar neck. Some of the fibers insert into the most anterior medial portion of the disk, but most of the lateral pterygoid fibers insert into the condyle.<sup>23</sup> Debate continues about the functional anatomy of the lateral pterygoid. The superior head is thought to be active during closing movements, and the inferior head is thought to be active during opening and protrusive movements.<sup>24-26</sup> Translation of the condylar head onto the articular eminence is produced by contraction of the lateral pterygoid.

### ACCESSORY TO MASTICATORY MUSCLES

The digastric muscle is a paired muscle with two bellies. The anterior belly attaches to the lingual aspect of the mandible at the parasymphysis and courses backward to insert into a

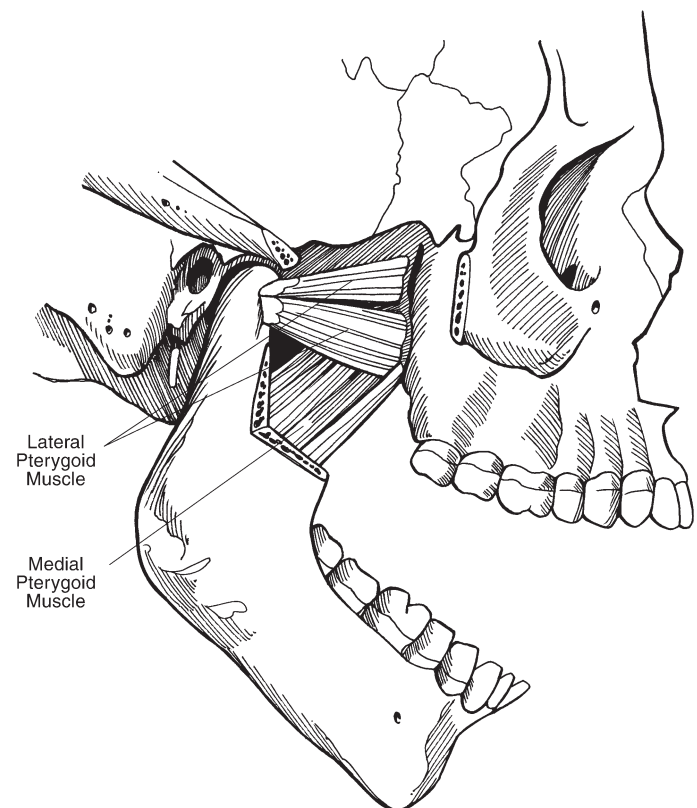
**FIGURE 10-6** The masseter and medial pterygoid muscles have their insertions at the inferior border of the mandibular angle. They join together to form a sling that cradles the ramus of the mandible and produces the powerful forces required for chewing. The masseter muscle has been divided into a deep portion and a superficial portion.





**FIGURE 10-7** The digastric muscle is a paired muscle with two bellies. The anterior belly attaches to the lingual aspect of the mandible at the parasymphysis and courses backward to insert into a round tendon attached to the hyoid bone. Contraction produces a depression and repositioning of the mandible. The mylohyoid and geniohyoid muscles contribute to depressing the mandible when the infrahyoid muscles stabilize the hyoid bone during mandibular movement. These muscles may also contribute to retrusion of the mandible. The temporalis muscle is broadly attached to the lateral skull. The muscle fibers converge to insert on the coronoid process and anterior aspect of the mandibular ramus. The posterior part traverses anteriorly then curves around the anterior root of the zygomatic process before insertion. The posterior part of the temporalis contributes to mandibular retrusion.

**FIGURE 10-8** The lateral pterygoid muscle is the main protrusive and opening muscle of the mandible. It is arranged in parallel-fibered units that allow for greater displacement and velocity than that of the multipennated closing muscles. The lateral pterygoid muscle is divided into two parts. The inferior part arises from the outer surface of the lateral pterygoid plate of the sphenoid and the pyramidal process of the palatine bone. The superior part originates from the greater wing of the sphenoid and the pterygoid ridge. The fibers of the upper and lower heads course posteriorly and laterally, fusing in front of the temporomandibular joint. They insert into the anteromedial aspect of the condylar neck. Some of the fibers insert into the most anterior medial portion of the disk, but most of the lateral pterygoid fibers insert into the condyle. Translation of the condylar head onto the articular eminence is produced by contraction of the lateral pterygoid.



round tendon attached to the hyoid bone. Contraction produces a depression and repositioning of the mandible. The mylohyoid and geniohyoid muscles contribute to depressing the mandible when the infrahyoid muscles stabilize the hyoid bone during mandibular movement. These muscles may also contribute to retrusion of the mandible. The buccinator attaches inferiorly along the facial surface of the mandible, just behind the mental foramen and superiorly high on the alveolar surface behind the zygomatic process. The fibers are arranged horizontally. Anteriorly, fibers insert into mucosa, skin, and lip. The buccinator helps position the cheek during chewing movements of the mandible.

### Vascular Supply of Temporomandibular Structures

The external carotid artery is the main blood supply for the temporomandibular structures. The artery leaves the neck and courses superiorly and posteriorly, embedded in the substance of the parotid gland. The artery sends two important branches, the lingual and facial arteries, to supply the region. At the level of the condylar neck, the external carotid bifurcates into the superficial temporal artery and the internal maxillary artery. These two arteries supply the muscles of mastication and the TMJ. Arteries within the temporal bone or mandible may also send branches to the capsule.

### Nerve Supply of Temporomandibular Structures

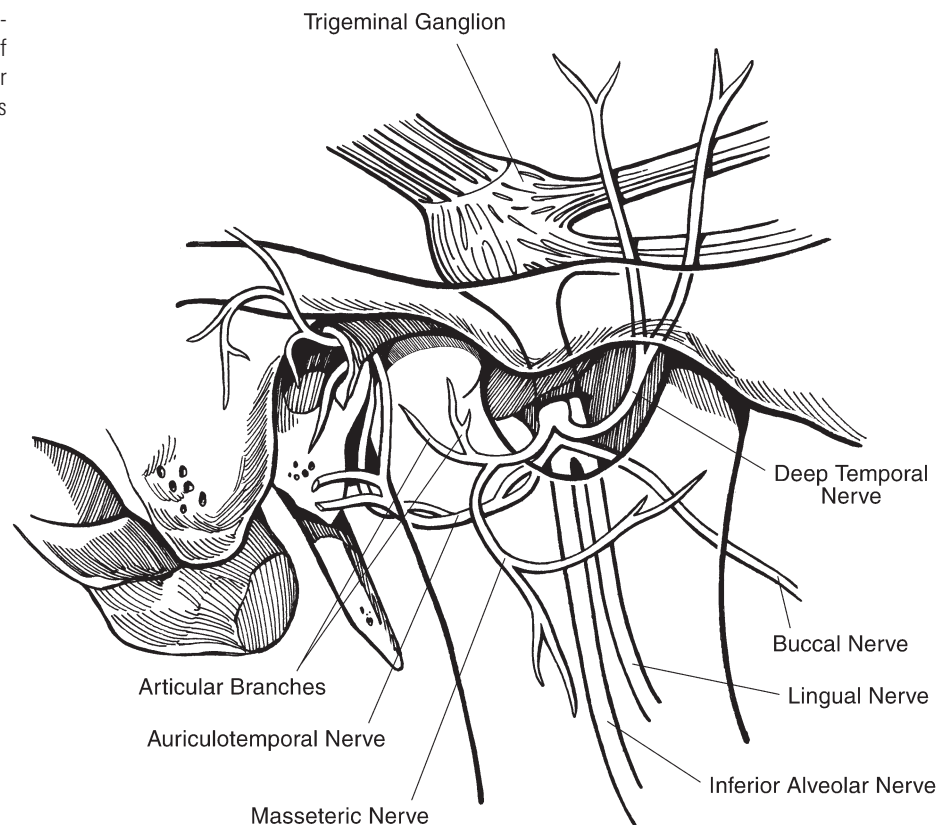
The masticatory structures are innervated primarily by the trigeminal nerve, but cranial nerves VII, IX, X, and XI and

cervical nerves 2 and 3 also contribute. The peripheral nerves synapse with nuclei in the brainstem that are associated with touch, proprioception, and motor function. The large spinal trigeminal nucleus occupies a major part of the brainstem and extends to the spinal cord. The spinal trigeminal nucleus is thought to be the main site for the reception of impulses from the periphery involved in pain sensation. The mandibular division of the trigeminal supplies motor innervation to the muscles of mastication and the anterior belly of the digastric muscle. Branches of the auriculotemporal nerve supply the sensory innervation of the TMJ; this nerve arises from the mandibular division in the infratemporal fossa and sends branches to the capsule of the joint (Figure 10-9). The deep temporal and masseteric nerves supply the anterior portion of the joint. These nerves are primarily motor nerves, but they contain sensory fibers distributed to the anterior part of the TMJ capsule. The autonomic nerve supply is carried to the joint by the auriculotemporal nerve and by nerves traveling along the superficial temporal artery.

### JAW JERK REFLEX

The jaw jerk reflex is analogous to the knee jerk reflex. It is a stretch reflex whereby stretching the jaw-closing muscles (by applying a downward tap on the chin) produces a reflex contraction of these muscles. It demonstrates the existence of a feedback loop from the jaw-closing muscles to their own motor neurons in the central nervous system. This reflex is thought to relate to the fine control of jaw movements to take into account different consistencies of food.<sup>27</sup>

**FIGURE 10-9** Branches of the auriculotemporal nerve supply sensory innervation of the TMJ. This nerve arises from the mandibular division in the infratemporal fossa and sends branches to the capsule of the joint.



## JAW-OPENING REFLEX

Stimulating mechanoreceptors within the mouth or nociceptors from the mouth or face triggers the jaw-opening reflex. The pathway is polysynaptic; the first synapse is in either the trigeminal sensory nuclei or the adjacent reticular formation, and the final synapse is in the trigeminal motor nucleus. The reflex results in an inhibition of the activity of the jaw-closing muscles. This reflex is thought to help prevent injury when biting or chewing objects that may cause damage.<sup>27</sup>

## Anatomy of Clinical Interest

### REST POSITION

When the mandible is not functionally active, it adopts a rest position in which the condyle occupies a relatively central position in the glenoid fossa and in which the teeth are separated. This position varies for a number of reasons (including head posture and levels of muscle activity) and is not an exact position.

### RANGE OF MANDIBULAR MOVEMENT

The position of widest mouth opening is associated with the condyle moving to the crest of the articular eminence or beyond. A wide variation in mandibular movement exists. Incisor displacement remains the most common diagnostic indicator.<sup>28</sup> The temporomandibular, sphenomandibular, and stylomandibular ligaments, together with the articular eminence, have been suggested as the main constraints of jaw opening.<sup>29</sup> Muscular constraint of jaw opening has also been proposed as a significant contributing factor.<sup>30</sup>

### ARTICULAR COVERING

The fibrocartilage found on the articulating surfaces of the TMJ is thought to provide more surface strength against forces in many directions while allowing more freedom of movement than would be possible with hyaline cartilage. This is the tissue that also forms the articular disk. This covering is thickest on the posterior slope of the articular eminence and on the anterior slope of the condylar head; these are the areas that are thought to receive the greatest functional load. The thinnest area of fibrocartilage is the roof of the mandibular fossa. Fibrocartilage has a greater repairing ability than hyaline cartilage. This may be a factor in how the TMJ responds to degenerative changes and may also be a factor in the treatment of degenerative joint disease.<sup>31</sup>

### DISK DISPLACEMENTS

The angle or steepness of the mandibular fossa has been considered to be a contributing factor in intra-articular disorders. The steep and more vertical form of the fossa has been associated with articular disk displacements in some published reports but has not been substantiated in others. Chronic recurring condyle subluxation or dislocation has also been related to the form and steepness of the fossa and articular

eminence. Surgical treatments that increase the steepness or flatten the eminence have been proposed.

Demonstration of the lateral pterygoid's attachment to the anterior articular disk has led to the theory that at least some anterior disk displacements may be related to lateral pterygoid-muscle dysfunction. The theory suggests that hyperactivity of the superior head of the lateral pterygoid may be capable of pulling the disk forward, displacing it from its normal position over the mandibular condyle.<sup>32</sup> Research on cadaver specimens has indicated that muscle fibers inserting into the disk or the condyle are not differentiated into inferior and superior heads.<sup>23,33</sup> The fibers that do insert into the disk are located primarily at the medial portion. Carpentier postulated that the two heads did not have distinct independent actions and that the lateral pterygoid was not a significant cause of disk displacement.<sup>23</sup> While "clicking" has been described as the most common irregularity detected during clinical examination, disk displacement may occur in the absence of clinical findings.<sup>34</sup>

### OCCLUSION

The intercuspal position is a position of the mandible in which maximum intercuspatation of opposing teeth occurs.<sup>35</sup> Occlusal stability has been defined as "the equalization of contacts that prevents tooth movement after closure."<sup>36</sup> A physiologic occlusion has been defined as "an occlusion in which a functional equilibrium or state of homeostasis exists between all tissues of the masticatory system."<sup>37</sup> There is general agreement that occlusal forces at the intercuspal position are best directed toward the long axes of teeth.<sup>38,39</sup> A reduced number of contacting teeth in the intercuspal position and loss of posterior teeth have been reported as risk factors for the development of TMD,<sup>40</sup> but they are not likely to be major causes in the majority of patients with TMD.

### EAR SYMPTOMS ASSOCIATED WITH TMD

A ligament between the disk and the malleolus of the middle ear has been observed in some anatomic specimens. The superior retrodiscal lamina has been considered to be a remnant of the discomalleolar ligament of the fetus, connecting the lateral pterygoid tendon to the malleus through the squamotympanic fissure.<sup>41</sup> This finding has been used to speculate about the prevalence of ear or hearing symptoms in TMD, but research has not established that this is a functioning ligament between the TMJ and the middle ear.<sup>42</sup>

Close proximity of the auriculotemporal nerve to the medial aspect of the condyle has been described. Extension of the medial wall of the fossa, exposing the auriculotemporal nerve to possible mechanical irritation in circumstances in which the articular disk becomes displaced medially, has also been described.<sup>43</sup> Nerve entrapment or compression was originally proposed by Costen as the explanation for pain and ear symptoms but was discounted by subsequent investigators. While nerve entrapment or compression is probably not the cause of most TMD, it reflects the need to review existing hypotheses in light of new information.



## INJECTION SITES

TMJ injections may be part of diagnostic assessment or therapy. The site of injection should be anterior to the tragus to minimize the risk of intravascular injection of the external carotid artery or the accompanying vein. Because the auriculotemporal nerve enters the capsule from the medial aspect, injections (normally given from the lateral aspect) may not completely anesthetize the joint.<sup>44</sup>

## PALPATION EXAMINATION

Examination of the lateral pterygoid muscle by intraoral palpation has been challenged because of the inaccessibility of the muscle (Figure 10-10).<sup>45</sup> The technique is likely to cause discomfort in individuals without a TMD, diminishing its value as a diagnostic test.<sup>46</sup>

The fibers of the deep masseter muscle are intimately related to the lateral wall of the joint capsule. This makes differentiating pain to palpation of this area difficult.<sup>47,48</sup>

## JAW JERK REFLEX AND THE SILENT PERIOD

A prolonged period of electrical inactivity on electromyography recordings has been observed in TMD patients.<sup>49</sup> This “silent period” never evolved into a clinically useful test.

## ▼ ETIOLOGY, EPIDEMIOLOGY, AND CLASSIFICATION

### Etiology

The etiology of the most common TMD is unknown. Two hypotheses, occlusal disharmony and psychological distress, have dominated the literature, but neither has been supported by the literature.<sup>50</sup> Research studying discrepancies between centric relation and centric occlusion, nonworking side interferences, and Angle’s classification has not shown a strong association in myofascial-pain patients when compared to controls.<sup>51–53</sup> Studies of patients with myofascial pain and control subjects have failed to demonstrate significant differences in occlusion although there may be some cases in which occlusal problems are an initiating factor.<sup>54</sup> A relationship between tooth loss and osteoarthritis has been found in patient studies<sup>55</sup> but has not been observed in nonpatient studies.<sup>56</sup> No difference between a symptomatic and control population was found when attempting to correlate incisal relationships, condylar position, and joint sounds.<sup>57</sup> An observed relationship between severe overbite and TMD symptoms has been reported but has not been consistently observed.<sup>58,59</sup> Alternatively, there is some experimental evidence to suggest that some observed occlusal changes could be produced by masticatory-muscle pain.<sup>60</sup> Anterior open-bite malocclusion may result from severe TMJ involvement in patients with rheumatoid arthritis.<sup>61</sup>

Masticatory-muscle hyperactivity progressing to a “vicious cycle” has been proposed as the cause of myofascial pain. The diagnostic terms of “myospasm,” “muscle spasm,” and “reflex

splinting” have been used to describe these conditions. A link between muscle hyperactivity and the pain disorder has not been demonstrated.<sup>62–66</sup> Differences between resting electromyographic activity in painful jaw-closing muscles and nonpainful muscles have not been found.<sup>67</sup> Tooth attrition signaling dental wear due to bruxing has not been associated with TMJ clicking or tenderness or with masticatory-muscle tenderness.<sup>68</sup>

The results of a number of experimental studies of myofascial pain are consistent with the hypothesis of pain caused by altered central nervous system processing,<sup>69–73</sup> but these findings could also be interpreted as a consequence of the pain rather than the cause of the pain.

The psychological hypothesis proposes that the disorder evolves as a consequence of psychological distress that is usually due to the individual’s stressful environment; the psychological distress leads to parafunctional habits (tooth clenching and grinding) that result in muscle pain.<sup>74–76</sup> A challenge that is continually faced in chronic pain disorders is determining how much of the psychological distress is a cause or is a consequence of the chronic pain.<sup>77</sup> The weight of the evidence suggests that in most cases, the emotional distress is more a consequence than a cause of pain.<sup>78</sup>

The lack of a clear single cause has resulted in the proposal of a multifactorial etiology.<sup>75</sup> These factors may contribute to the initiation, aggravation, and/or perpetuation of the disorder. Some of the factors proposed are the following:

1. Parafunctional habits (eg, nocturnal bruxing, tooth clenching, lip or cheek biting)<sup>79,80</sup>
2. Emotional distress<sup>81,82</sup>
3. Acute trauma from blows or impacts<sup>83</sup>
4. Trauma from hyperextension (eg, dental procedures, oral intubation for general anesthesia, yawning, hyperextension associated with cervical trauma)<sup>84</sup>
5. Instability of maxillomandibular relationships<sup>85</sup>
6. Laxity of the joint<sup>86</sup>
7. Comorbidity of other rheumatic or musculoskeletal disorders<sup>87</sup>
8. Poor general health and an unhealthy lifestyle<sup>88</sup>

The frequency and the importance of these factors as causes are unknown.

### Epidemiology

Between 65 and 85% of people in the United States experience some symptoms of TMD during their lives, and approximately 12% experience prolonged pain or disability that results in chronic symptoms.<sup>89</sup> Although the prevalence of one or more signs of mandibular pain and dysfunction is high in the population, only about 5 to 7% have symptoms severe enough to need treatment.<sup>89,51,90</sup> TMD patients are similar to headache and back pain patients with respect to disability, psychosocial profile, and pain intensity, chronicity, and frequency.<sup>91,92</sup> The lower prevalence of TMD signs and symptoms in older age groups supports the probability that most TMD are self-limiting.



**FIGURE 10-10** The clinical examination. **A**, measuring maximum interincisal opening. **B**, Palpation of the preauricular area; the lateral aspect of the TMJ. **C**, Intra-auricular palpation; the posterior aspect of the TMJ. **D**, Palpation of the masseter muscles. **E**, Bi-manual palpation of the masseter muscle. **F**, Palpation of the lateral pterygoid muscle. **G**, Palpation of the medial pterygoid muscle. **H**, Palpation of the temporalis muscle. **I**, Palpation of the sternocleidomastoid muscle. **J**, Palpation of the trapezius muscle. Note that the lateral and medial pterygoid muscle palpations are from an intra-oral approach. Access to these muscles is limited and the procedure may produce an unacceptable rate of false positives (pain on palpation). The results of lateral and medial pterygoid palpation should be interpreted with caution.

Available evidence indicates that TMD are most prevalent between the ages of 20 and 40 years and predominantly affect women. The reason why women make up the majority of patients presenting for treatment is still unclear. In a community-based study, a greater likelihood of developing TMD was found if oral contraceptives were used and, in women over 40 years of age, if estrogen replacement was used.<sup>93</sup>

While the prevalence of TMD is highest in the 20- to 40-year age range, signs and symptoms of masticatory-muscle and joint dysfunction are commonly observed in children.<sup>94-96</sup> Belfer reported on a group of 40 children, aged 10 to 16 years, presenting with signs and symptoms of TMD; 14 (35%) of the 40 were diagnosed as having acute reactive depression.<sup>97</sup> In another study, arthrography and computed tomography were performed on 31 pediatric patients complaining of TMJ pain and dysfunction.<sup>98</sup> Twelve were diagnosed with disk displacement with reduction, and 17 were found to have disk displacement without reduction. In 12 of the 29 patients with internal derangement, the problem was thought to be due to a previous injury. In a survey of 1,000 12-year-old children, 1% had a maximum mouth opening of less than 40 mm, and few children presented with clinical findings severe enough to warrant treatment.<sup>99</sup>

A number of studies have been performed to investigate a possible relationship between orthodontic treatment and the development of TMD, but the results do not support a causal relationship between orthodontic treatment and the subsequent development of TMD.<sup>100,101</sup>

## Classification

Due to the uncertainty about etiology, current diagnostic classifications of TMD are based on signs and symptoms. Earlier classifications characterized disorders as intracapsular (TMJ) or extracapsular (muscle) disorders and were often not versatile enough to allow for multiple diagnoses of masticatory muscle and TMJ abnormalities. More recent classifications do allow for more than one diagnosis, and this better reflects the clinical reality.<sup>102,103</sup> Arthrographic techniques have provided evidence resulting in more-accurate descriptions of intracapsular disorders in relation to presenting clinical features.<sup>63,104</sup> Disk disorders are now differentiated on the basis of arthrographic or magnetic resonance imaging findings.

In 1989, Clark and colleagues published a classification system that was useful to the practicing clinician (Table 10-1).<sup>105</sup>

**TABLE 10-2 Diagnostic Classification of Temporomandibular Disorders**

Diagnostic Category	Diagnoses
Cranial bones (including the mandible)	Congenital and developmental disorders: aplasia, hypoplasia, hyperplasia, dysplasia (eg, 1st and 2nd branchial arch anomalies, hemifacial microsomia, Pierre Robin syndrome, Treacher Collins syndrome, condylar hyperplasia, prognathism, fibrous dysplasia) Acquired disorders (neoplasia, fracture)
Temporomandibular joint disorders	Deviation in form Disk displacement (with reduction; without reduction) Dislocation Inflammatory conditions (synovitis, capsulitis) Arthritides (osteoarthritis, osteoarthritis polyarthritides) Ankylosis (fibrous, bony) Neoplasia
Masticatory-muscle disorders	Myofascial pain Myositis Spasm Protective splinting Contracture

Adapted from McNeill C.<sup>87</sup>

The American Academy of Orofacial Pain (AAOP) has published a general classification of disorders that affect the cranial bones, temporomandibular joints, and masticatory muscles<sup>87</sup> (Table 10-2). This classification system is useful because it attempts to define the diagnostic terms and provide diagnostic criteria. This classification has not been subjected to testing for validity. It represents an attempt by experts to apply available knowledge to the development of a more acceptable and useful system for clinical practice. Table 10-3 provides a partial list of the diagnostic terms and their diagnostic criteria. This classification does provide the clinician with an aid in clinical decision making. A number of other clinical findings, described as possibly accompanying the diagnosis, are listed in the latest AAOP publication on orofacial pain guidelines.<sup>1</sup> The supporting signs in disk disorders may have additional clinical value and are listed in Table 10-3 as “clinical findings that may support the diagnosis.”

**TABLE 10-1 Classification for Diagnosing Temporomandibular Disorders**

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

Adapted from Clark GT et al.<sup>105</sup>

TMJ = temporomandibular joint.

**TABLE 10-3 Diagnostic Terms and Clinical Criteria for Temporomandibular Disorders**

Diagnostic Terms	Clinical Criteria
Deviation in form (painless mechanical dysfunction or altered function due to irregularities or aberrations in form of the intracapsular soft and hard articular tissues)	Complaint of faulty or compromised joint mechanics Reproducible joint noise, usually at the same position during opening and closing Radiographic evidence of structural bony abnormality or loss of normal shape
Disk displacement with reduction (abrupt alteration or interference of the disk-condyle structural relation during mandibular translation with mouth opening and closing; from a closed-mouth position, the “temporarily” misaligned disk reduces or improves its structural relation with the condyle when mandibular translation occurs with mouth opening, which produces joint noise described as clicking or popping)	Pain (when present) is precipitated by joint movement. Reproducible joint noise, usually at variable positions during opening and closing mandibular movements Soft-tissue imaging reveals displaced disk that improves its position during jaw opening. Clinical findings that may support the diagnosis: pain (when present) precipitated by joint movement; deviation during movement coinciding with a click; no restriction in mandibular movement (episodic and momentary catching of smooth jaw movements during mouth opening [ $< 35$ mm] that self-reduces with voluntary mandibular repositioning)
Disk displacement without reduction (altered or misaligned disk-condyle structural relation that is maintained during mandibular translation)	Pain precipitated by function Marked limited mandibular opening Straight-line deviation to the affected side on opening Marked limited laterotrusion to the contralateral side Soft-tissue imaging reveals displaced disk without reduction. Clinical findings that may support the diagnosis: pain precipitated by forced mouth opening; history of clicking that ceases with the locking; pain with palpation of the affected joint; ipsilateral hyperocclusion
Synovitis or capsulitis (inflammation of the synovial lining or capsular lining)	Localized pain at rest exacerbated by function, especially with superior and posterior joint loading Limited range of motion secondary to pain T2-weighted MRI may show joint fluid
Osteoarthritis (degenerative noninflammatory condition of the joint, characterized by structural changes of joint surfaces secondary to excessive straining of the remodeling mechanism)	Crepitus Limited range of motion causes deviation to the affected side on opening. Radiographic evidence of structural bony change (subchondral sclerosis, osteophyte formation) and joint-space narrowing
Osteoarthritis (degenerative condition accompanied by secondary inflammation [synovitis] of the TMJ)	Same as for osteoarthritis, plus crepitus or multiple joint noises, pain with function due to inflammation, and point tenderness on palpation
Myofascial pain (regional dull aching pain and presence of localized tender spots [trigger points] in muscle, tendon, or fascia that reproduce pain when palpated and may produce a characteristic pattern of regional referred pain and/or autonomic symptoms on provocation)	Regional pain, usually dull Localized tenderness in firm bands of muscle and/or fascia Reduction in pain with local muscle anesthetic injection or vapocoolant spray and stretch of muscle trigger points
Myositis, delayed onset (painful condition due to intermittent overuse that results in interstitial inflammation)	Increased pain with mandibular movement Onset following prolonged or unaccustomed use (up to 48 hours afterward)
Myositis, generalized (constant, acutely painful, and generalized inflammation and swelling, usually of the entire muscle)	Pain usually acute in localized area Localized tenderness over entire region of the muscle Increased pain with mandibular movement Moderately to severely limited range of motion, due to pain and swelling Onset following injury or infection
Protective muscle splinting (restricted or guarded mandibular movement due to cocontraction of muscles as a means of avoiding pain caused by movement of the parts)	Severe pain with function but not at rest Marked limited range of motion without significant increase on passive stretch
Contracture (chronic resistance of a muscle to passive stretch, as a result of fibrosis of the supporting tendon, ligaments, or muscle fibers themselves)	Limited range of motion Unyielding firmness on passive stretch History of trauma or infection

Adapted from McNeill C.<sup>87</sup>

MRI = magnetic resonance imaging; TMJ = temporomandibular joint.

The interpretation of the TMD literature and advances in knowledge have been impeded by the lack of widely accepted methods or standards for selecting or describing patients who are part of clinical research projects. Dworkin and colleagues developed a classification for the most common TMD, to pro-

vide a system that could be used in clinical research. The Research Diagnostic Criteria for TMD (RDC/TMD) were published as a system “offered to allow standardization and replication of research into the most common forms of muscle- and joint-related TMD.”<sup>28</sup> The classification scheme is

intended to provide a means for standardizing data collection and for comparing findings among clinical investigators. Because of the chronicity of TMD, a classification system that reflects psychological, behavioral, and social issues is as important as an accurate description of the physical pathology. The RDC/TMD classification has a separate axis that assesses psychosocial status to create profiles of disability, depression, anxiety, and preoccupation with other physical symptoms. The RDC/TMD classification has not yet been subjected to the research required for validation.

The classifications published by Clark and the AAOP were designed for clinical practice and are more comprehensive. The RDC/TMD classification was developed for research purposes but is useful in clinical practice for the types of TMD most likely to present to a dentist. The classification does not include the conditions that are less common but still likely to present to clinicians. The RDC/TMD system allows for multiple diagnoses for each individual but only one muscle diagnosis and allows for each joint only one disk disorder diagnosis and one articular bone diagnosis. The terms used are clearly defined, and the criteria required to meet the diagnosis are detailed although the validation of these criteria and the classification system will have to await further research. To allow greater use in the research environment, the criteria do not include diagnostic imaging. While the clinician will likely not adhere to all the guidelines or diagnoses, the assessment and classification system serves as a useful method of organizing clinical information for the most commonly presenting TMD<sup>28</sup> (Tables 10-4 and 10-5). The reliance on clinical findings for diagnosis is consistent with the research purpose, but diagnostic imaging would likely be required to establish a disk disorder diagnosis such as “disk displacement without reduction, without limited opening.” The clinician should add diagnostic imaging as part of the assessment whenever the diagnosis, treatment choice, or outcome may benefit. If the RDC/TMD classification system is used more frequently in research, clinicians who are familiar with it will be in a better position to evaluate the published research. (The reader is referred to the publication by Dworkin and colleagues.)<sup>28</sup> Muscle conditions (such as myositis, contracture, and myospasm) and joint conditions associated with systemic arthritis, acute trauma, hyperplasia, and neoplasia are not part of the defined conditions.

Schiffman and colleagues compared clinical findings and tomographic findings to define criteria for intra-articular disorders and presented criteria for the classification of articular disorders.<sup>106</sup> No single sign or symptom was consistently accurate for establishing a diagnosis, but the patterns listed in Table 10-6 show 75% agreement when compared to findings by arthrotomography.<sup>106</sup> The diagnosis of articular disk displacement without reduction, chronic (similar to RDC/TMD articular disk displacement without reduction and without limited opening), may be the most problematic diagnosis without imaging. Other clinical findings may support the diagnosis and include pain, markedly reduced from the acute stage and usually presenting as a

**TABLE 10-4 Research Diagnostic Criteria for Clinical Temporomandibular Disorder Conditions, Axis 1<sup>28</sup>**

Clinical Location	RDC/TMD Diagnoses
Muscle	Myofascial pain Myofascial pain with limited opening
Disk displacement	Disk displacement with reduction Disk displacement without reduction, with limited opening Disk displacement without reduction, without limited opening
Articular bone	Arthralgia Osteoarthritis of the TMJ Osteoarthrosis of the TMJ

RDC/TMD = Research Diagnostic Criteria for temporomandibular disorders;  
TMJ = temporomandibular joint.

feeling of stiffness; a history of clicking that resolves with the sudden onset of the locking; and gradual resolution of limited mouth opening.

## ▼ ASSESSMENT

Present examination methods have not yet demonstrated the ability to differentiate accurately persons with a TMD from those without a TMD.<sup>107</sup> The most valuable aspects of the diagnostic assessment are a thorough history and physical examination.<sup>108</sup> Most of the tests used to assess TMD patients have not been validated and are not standardized, and an ideal method for classification has not been established.<sup>109</sup> Diagnostic tests such as ultrasonographic analysis of joint sounds, thermography, jaw-tracking devices, and electromyography do not offer the assurance of a more accurate diagnosis.<sup>110</sup> These tests require sophisticated instrumentation that increases health care costs to the patient. In most cases, the correct diagnostic classification can be reached by using the self-reporting of the patient and the findings on clinical examination.<sup>106,111</sup> Diagnostic imaging is of value in selected conditions but not as part of a standard assessment. Diagnostic imaging can increase accuracy in the detection of internal derangements and abnormalities of articular bone.

While pain is a characteristic feature of TMD, it may also be associated with serious undetected disease. Muscle or joint pain may be a secondary feature of other disease or may mimic a TMD, and a diagnosis may be missed or delayed.<sup>112,113</sup> Severe throbbing temporal pain associated with a palpable nodular temporal artery, increasingly severe headache associated with nausea and vomiting, and documented altered sensation or hearing loss are all indications of serious disease requiring timely diagnosis and management.

Assessment should result in a diagnosis of a TMD and an estimation of psychological distress and pain-related disability. The lack of a direct relationship between (a) physical pathology and intensity of pain and (b) subsequent disability emphasizes the need to assess the psychological and behavioral effects of the disorder. Friction recommends developing

**TABLE 10-5 Definitions and Clinical Criteria for Temporomandibular Disorders**

Definitions*	Clinical Criteria*
Myofascial pain (pain of muscle origin, including complaint of pain associated with localized areas of tenderness to palpation in muscle)	Report of pain or ache in jaw, temples, face, preauricular area, or inside ear at rest or during function, and pain on palpation in three or more muscle sites
Myofascial pain with limited opening	Myofascial pain, pain-free unassisted mandibular opening of < 40 mm, and a maximum assisted opening of $\geq 5$ mm greater than the pain-free unassisted opening
Disk displacement with reduction (disk is displaced from its position between the condyle and eminence to an anterior and medial or lateral position but is reduced in full opening, usually resulting in a noise)	Click on both vertical opening and closing that occurs at a point at least 5 mm (interincisal opening) greater than on closing, is eliminated on protrusive opening, and is reproducible in two of three consecutive trials <i>or</i> click on opening or closing and click on lateral excursion or protrusion, reproducible in two of three consecutive trials
Disk displacement without reduction, with limited opening (disk is displaced from normal position between condyle and fossa to an anterior and medial or lateral position, associated with limited opening)	History of significant limitation of opening Maximum unassisted opening $\leq 35$ mm, passive stretch increases opening by $\leq 4$ mm, and contralateral excursion < 7 mm and/or uncorrected deviation to the ipsilateral side on opening Absence of joint sounds or sounds that do not meet criteria for disk displacement with reduction
Disk displacement without reduction without limited opening (disk is displaced from its position between condyle and eminence to an anterior and medial or lateral position, not associated with limited opening)	History of significant limitation of mandibular opening Maximum unassisted opening > 35 mm, passive stretch increases opening by $\geq 5$ mm over maximum unassisted opening, contralateral excursion $\geq 7$ mm, and presence of joint sounds not meeting criteria for disk displacement with reduction
Arthralgia (pain and tenderness in joint capsule and/or synovial lining of the TMJ)	Pain in one or both joint sites and self-report of pain in region of joint Pain in joint during maximum opening (assisted or unassisted) Pain in joint during lateral excursion
Osteoarthritis of the TMJ (inflammatory condition within the joint, resulting from a degenerative condition of joint structures)	Arthralgia and coarse crepitus or imaging showing one or more of the following: erosion of normal cortical outlines, sclerosis of parts or all of condyle and articular eminence, flattening of joint surfaces, osteophyte formation
Osteoarthrosis of the TMJ (degenerative joint disorder in which joint form and structure are abnormal)	Absence of arthralgia Coarse crepitus or imaging showing one or more of the following: erosion of normal cortical outlines, sclerosis of parts or all of condyle and articular eminence, flattening of joint surfaces, osteophyte formation

TMJ = temporomandibular joint.

\* For the complete description, refer to Dworkin S, LeResche L.<sup>28</sup>

a problem list of the contributing factors associated with TMD.<sup>114</sup> Contributing factors may affect the symptom control and the long-term success of any treatment program. No one individual can be expected to manage or address the var-

ious lifestyle emotional, cognitive, and social issues that may have an impact on the chronic pain. The importance of these factors to TMD pain and to the patient's general health needs to be assessed; an appropriate plan to address them can then

**TABLE 10-6 Clinical Findings of Disk Disorders, Correlating with Arthrotomography**

Assessment Procedure	Clinical Findings			
	Normal	ADD with Reduction	Acute ADD without Reduction	Chronic ADD without Reduction
History	None	None	Positive for mandibular limitation	Positive for TMJ noise
Physical examination	Reciprocal click No coarse crepitus Passive stretch $\geq 40$ mm Lateral movements $\geq 7$ mm If S-curve present, joint is silent	Reciprocal click or popping No coarse crepitus Passive stretch $\geq 35$ mm	No reciprocal click No coarse crepitus Maximum opening $\leq 35$ mm Passive stretch < 40 mm Contralateral movement < 7 mm No S-curve deviation	No reciprocal click Coarse crepitus or joint sounds other than reciprocal clicking
Tomography	No decreased translation in ipsilateral condyle No osseous change	None	Decreased translation of ipsilateral condyle	Decreased translation of ipsilateral condyle <i>or</i> positive osseous changes

Adapted from Schiffman et al.<sup>106</sup>

ADD = articular disk displacement; TMJ = temporomandibular joint.

**TABLE 10-7 Problem List of Contributing Factors Associated with Temporomandibular Disorders**

Lifestyle	Emotional Factors	Cognitive Factors	Biologic Factors	Social Factors
Diet	Prolonged anger	Negative self-image	Other illnesses	Work stresses
Sleep	Anxiety	Unrealistic expectations	Past trauma	Unemployment
Alcohol	Excessive worry	Inadequate coping	Past jaw surgery	Family stresses
Smoking	Depression			Litigation
Overwork				Financial difficulty

Adapted from Fricton.<sup>114</sup>

be developed. Table 10-7 lists some of the contributing factors discussed by Fricton.<sup>114</sup>

A validated and empirically derived classification of TMD patients, based on psychosocial and behavioral parameters, has identified three unique subgroups: dysfunctional patients, interpersonally distressed patients, and adaptive copers.<sup>115</sup> TMD is not unique in the psychosocial and behavioral parameters; TMD patients and back pain patients have demonstrated similar profiles.<sup>91,116</sup> Interventions targeting pain and psychological distress are of equal importance to the pathophysiology of temporomandibular structures in managing a chronic TMD. Psychosocial assessment should provide the clinician with an appreciation of the extent to which pain and dysfunction interfere with or diminish the patient's quality of life. The assessment should identify patients with psychological distress that warrants further investigation and possible treatment by a psychologist or psychiatrist. In addition to assessments of pain intensity and emotional state, an assessment of limitation in activity will provide a reflection of the magnitude of the condition.<sup>117,118</sup> In one report, approximately 16% of TMD patients experienced significant activity limitation, compared to approximately 3% of controls.<sup>119</sup> A systematic method of screening is necessary because dentists have been found to be inaccurate in identifying psychological problems in TMD patients.<sup>120</sup> The RDC/TMD uses a questionnaire partly developed for this classification system and from previously used scales to assess pain intensity and disability, depression, and nonspecific physical symptoms. (The reader is referred to the publication by Dworkin and colleagues.)<sup>28</sup>

## History

The most common symptom related to TMD is pain. This pain usually shows some relation to mandibular function, or an alternative diagnosis should be suspected. A "pain diary" can be a useful tool for identifying events or times of increased and decreased pain; it may also serve to identify behaviors or situations that are contributing to the persistence of symptoms. A pain diagram of the head and neck is helpful in defining the extent of pain and may also be used to assess treatment progress. A diagram that includes the whole body may help identify patients who have multiple sites of pain, which suggests a more systemic disorder. Table 10-8 lists questions that are useful (as part of the history) for assessing mandibular function.

## Physical Examination

No one physical finding can be relied on to establish a diagnosis, but a pattern of abnormalities may suggest the source of the problem and a possible diagnosis. Masticatory-muscle tenderness on palpation (see Figure 10-10) is the most consistent examination feature present in cases of TMD.<sup>102</sup> The clinical features that distinguish patients from controls are

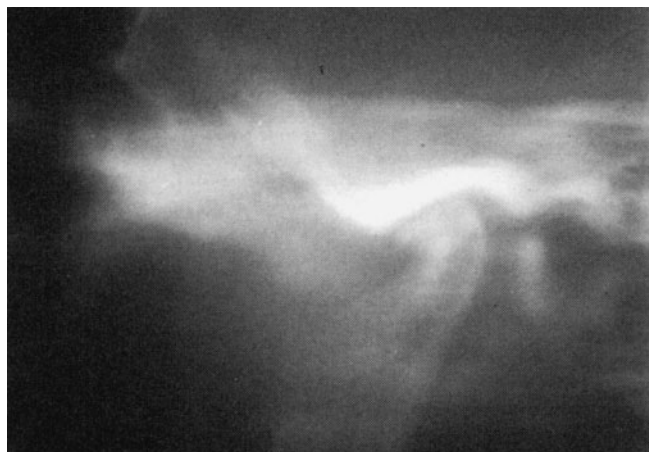
1. passive mouth opening,<sup>121</sup>
2. masticatory-muscle tenderness on palpation and maximal mouth opening,<sup>122</sup> and
3. an uncorrected deviation on maximum mouth opening and tenderness on palpation.<sup>89</sup>

Components of the physical examination that are discussed in this section are summarized in Table 10-9.

**TABLE 10-8 History: Questions to Ask when Evaluating a Patient for Mandibular Dysfunction\***

Do you have pain in the face, in front of the ear and temple areas?
Do you get headaches, earaches, neckache, or cheek pain?
When is pain at its worst (morning [on awakening] or as day progresses [toward evening])?
Do you experience pain when using the jaw (opening wide, yawning, chewing, speaking, or swallowing)?
Do you experience pain in the teeth?
Do you experience joint noises when moving your jaw or when chewing (clicking, popping, or crepitus)?
Does your jaw ever lock or get stuck (locking in the open position or locking in the closed position)?
Does your jaw motion feel restricted?
Have you had an abrupt change in the way your teeth meet together?
Does your bite feel "off" or uncomfortable?
Have you had any jaw injuries?
Have you had treatment for the jaw symptoms? If so, what was the effect?
Do you have any other muscle, bone, or joint problem such as arthritis or fibromyalgia?
Do you have pain in any other body sites?

\*Miscellaneous symptoms are sometimes reported in association with temporomandibular dysfunction and may include dizziness; nausea; fullness or ringing in the ears; diminished hearing; facial swelling; redness of the eyes; nasal congestion; altered sensation such as numbness, tingling, or burning; altered vision; and muscle twitching.



**FIGURE 10-11** Temporomandibular joint tomogram displaying flattening of the condylar head in degenerative joint disease.

### RANGE OF MANDIBULAR MOVEMENT

Interincisor separation plus or minus the incisor overlap in centric occlusion provides the measure of mandibular movement. Maximum opening should be measured without pain, as wide as possible even with pain, and after opening with clinician assistance. Assisting the jaw during mouth opening is accomplished by applying mild to moderate pressure against the upper and lower incisors with the thumb and index finger. Assisted opening should be measured. Passive stretching is a technique for assessing and differentiating restrictions due to muscle or joint. It results in a measure to be compared with active opening and an experience of the quality of resistance at the end of the movement. Muscle restrictions are associated with a soft-end feel, and a mouth opening increase of  $> 5$  mm can often be achieved. Joint disorders such as acute nonreducing disk displacements are described as having a hard-end feel and characteristically restrict assisted opening to  $< 5$  mm. The normal maximum mouth opening is  $\geq 40$  mm. In a study of 1,160 adults, the mean maximum mouth opening was 52.8 mm (with a range of 38.7 to 67.2 mm) for men and 48.3 mm (with a range of 36.7 to 60.4 mm) for women.<sup>123</sup> Measures of lateral movement are made with the teeth slightly separated, measuring the displacement of the lower midline from the maxillary midline and adding or subtracting the lower-midline displacement at the start of movement. Protrusive movement is measured by adding the horizontal distance between the upper and lower central incisors and adding the distance the lower incisors travel beyond the upper incisors. Normal lateral and protrusive movements are  $\geq 7$  mm.<sup>62,124–126</sup> Measures of the mandibular range of movement are similarly performed in children. The mean maximum mouth opening recorded in 75 boys and 75 girls aged 6 years was 44.8 mm.<sup>127</sup> A study of 189 individuals with a mean age of 10 years reported similar values (a mean maximum opening of 43.9 mm, with a range of 32 to 64 mm).<sup>128</sup> The means of left,

right, and protrusive maximal movements were each approximately 8 mm.

### PALPATION OF MASTICATORY MUSCLES

The primary finding related to masticatory-muscle palpation is pain. The methods for palpation are not standardized in clinical practice. The amount of pressure to apply and the exact sites that are most likely associated with TMD are unknown. Some clinicians have recommended attempting to establish a baseline (to serve as a general guide or reference) by squeezing the muscle between the index finger and thumb or by applying pressure in the center of the forehead or thumbnail to gauge what pressure becomes uncomfortable. The RDC/TMD guidelines recommend 1 lb of pressure for the joint and 2 lb of pressure for the muscles.<sup>28</sup> The RDC/TMD pressures have been established for research purposes. In the clinical setting, a greater range of pressures is probably required. All of the examination procedures should be accompanied by questioning the patient about the production of pain and the site of pain during the particular examination procedure. Reproducing the site and the character of the pain during the examination procedure helps identify the possible anatomic site of the pain. This may also provide insight into the possibility of referred pain as a factor. Palpation of the joint and the muscles for pain should be done with the muscles in a resting state. There are no standardized methods of assessing the severity of palpable pain, and the patient should be asked to rate the severity by using a scale (eg, a numerical scale from 1 to 10, a visual analogue

**TABLE 10-9** Physical Examination Directed Toward Mandibular Dysfunction

Examination Component	Observations
Inspection	Facial asymmetry, swelling, and masseter- and temporal-muscle hypertrophy Opening pattern (corrected and uncorrected deviations, uncoordinated movements, limitations)
Assessment of range of mandibular movement	Maximum opening with comfort, with pain, and with clinician assistance Maximum lateral and protrusive movements
Palpation examination	Masticatory muscles Temporomandibular joints Neck muscles and accessory muscles of the jaw Parotid and submandibular areas Lymph nodes
Provocation tests	Static pain test (mandibular resistance against pressure) Pain in the joints or muscles with tooth clenching Reproduction of symptoms with chewing (wax, sugarless gum)
Intraoral examination	Signs of parafunction (cheek or lip biting, accentuated linea alba, scalloped tongue borders, occlusal wear, tooth mobility, generalized sensitivity to percussion, thermal testing, multiple fractures of enamel and restorations)



scale, or a ranking such as none, mild, moderate, or severe). The RDC/TMD recommend using the categories of pressure only, mild pain, moderate pain, and severe pain. These ratings may be useful as part of the process of assessing treatment progress. Palpation may reproduce the patient's pain symptoms or may produce pain referred to a distant site such as the molar teeth, which may help in differential diagnosis. Abnormalities such as trigger points and taut bands in muscle have not been sufficiently characterized in the masticatory muscles to always enable the clinician to distinguish these sites anatomically from normal muscle.

### **PALPATION OF CERVICAL MUSCLES**

Patients with TMD/MPD often have musculoskeletal problems in other regions that are particularly associated with the neck.<sup>129,130</sup> The sternocleidomastoid and trapezius muscles are often part of a neck muscle disorder and may refer pain to the face and head. Other muscle groups to palpate include the paravertebral (scalene) and suboccipital muscles.

### **PALPATION OF THE TMJ**

Palpation of the TMJ will reveal pain and irregularities during condylar movement, described as clicking or crepitus. The lateral pole of the condyle is most accessible for palpation during mandibular movements. Palpating just anterior and posterior to the lateral pole should detect pain associated with the TMJ capsular ligaments. In addition to joint noises and pain, there may be palpable differences in the form of the condyle when comparing right and left. The condyle that does not translate may not be palpable during mouth opening and closing. The click that occurs on opening and closing and that is eliminated by bringing the mandible into a protrusive position before opening is most often associated with articular disk displacement with reduction.

### **PROVOCATION TESTS**

Provocation tests are designed to elicit the described pain. Since pain is often aggravated by jaw use, a positive response adds support for a diagnosis of TMD. The static pain test involves having the mandible slightly open and remaining in one position while the patient resists the slowly increasing manual force applied by the examiner in a lateral, upward, and downward direction. If the mandible remains in a static position during the test, it is the muscles that will be subjected to activation. However, the ability of this test to discriminate between muscle and joint pain is not known. Clenching the teeth or chewing wax or gum is expected to load the joints and muscles. According to one report, approximately 50% of TMD patients who chewed one-half of a leaf of gauge-28 green casting wax for 3 minutes reported an increase in pain, but 30% reported decreased pain, and 20% reported no change.<sup>131</sup>

### **Assessment of Parafunctional Habits**

It is difficult to determine the presence of active severe oral habits, and only indirect means are generally available. The patient is often unaware of tooth clenching or other behaviors contributing to jaw hyperactivity while awake. Self-reports, instructions for

checking jaw activity during the day, and reports by sleeping partners of tooth-grinding noises are helpful. Examination for tooth wear, soft-tissue changes (lip or cheek chewing, a hyperplastic occlusal line, and scalloped tongue borders), and hypertrophic jaw-closing muscles may suggest hyperactivity.

### **Diagnostic Imaging**

When the clinical presentation suggests a progressive pathologic condition of the TMJ, imaging should be part of the assessment. Recent injury, sensory or motor abnormality, severe restriction in mandibular motion, and acute alterations of the occlusion are clinical findings for which imaging is indicated. The most frequent abnormalities that are imaged in TMD patients are degenerative changes of bone and disk displacement. TMJs can be examined by using plain-film radiography, tomography, arthrography, computerized tomography (CT), magnetic resonance imaging (MRI), single-photon emission computed tomography, and radioisotope scanning. MRI has become the imaging method of choice to assess disk position. For the majority of TMDs, diagnostic imaging has not proven to be a valuable test for directing treatment or for predicting outcome and long-term course. No differences were found in joint-space narrowing in the centric occlusion position in symptomatic and asymptomatic patients by transcranial plain-film radiography and tomography.<sup>132,133</sup> A large variation exists in condylar position in plain-film radiographic and tomographic studies, making the condyle-fossa relationship of little value in the diagnosis or treatment of TMD.<sup>134</sup> Using plain films (such as in transcranial radiography) to determine condylar position or using the condylar position on these films to assess disk position is not recommended.<sup>132,133,135</sup> Imaging such as tomography and CT is relied on to document osteodegenerative joint disease. CT provides detail for bony abnormalities and is an appropriate study when considering ankylosis, fractures, tumors of bone, and osteodegenerative joint disease. MRI is the method of choice for establishing alterations in articular disk position in the open- and closed-mouth positions. MRI studies in asymptomatic volunteers have shown disk abnormalities in approximately one-third of subjects.<sup>34,136</sup> With the use of T2-weighted MRI, a correlation between joint pain and joint effusion has been suggested, but the results are conflicting.<sup>137,138</sup> Radioisotope scanning for detecting increases in metabolic activity has been used to detect condylar hyperplasia. Bone scanning is a sensitive indicator of metabolic bone activity and may therefore show a positive result in a joint that is undergoing physiologic remodeling as well. Radioisotope scanning in combination with other imaging and clinical findings (including findings on periodic examinations) is usually effective in diagnosing continued condylar change due to hyperplasia.

### **Diagnostic Local Anesthetic Nerve Blocks**

Injections of anesthetics into the TMJ or selected masticatory muscles may help to confirm a diagnosis. A positive test may result in the elimination of pain and improved jaw motion.

Diagnostic injections may also be helpful in differentiating pain arising from joints or muscle. In situations in which a joint procedure is being considered, local anesthetic injection of the joint may confirm the joint as the source of pain. Lidocaine (2%) without a vasoconstrictor can be used. Injecting trigger points or tender areas in the muscle should eliminate pain from that site and should also eliminate referred pain associated with the injected tender or trigger point.<sup>139</sup> These tests, like all others, require interpretation in the context of all the diagnostic information since a positive result does not insure a specific diagnosis.

### Prediction of Chronicity

While most TMD patients respond to nonsurgical management that can be provided or coordinated by a dentist, some individuals develop chronic pain and disability. This group experiences great psychological distress and disruption of their normal daily activities, as well as the need to access ongoing health care resources. Predicting the cases that are likely to become chronic is important to provide alternative or additional interventions.

Psychological factors seem to be more important than peripheral injury or physical disease of the masticatory system in predicting chronicity.<sup>140–144</sup> Epker and colleagues found that the combination of high pain intensity (as measured by RDC/TMD scales) and a myofascial pain diagnosis (reported pain on palpation of muscles) was predictive of persisting TMD symptoms in their population.<sup>145</sup>

Comorbidity with widespread musculoskeletal pain is likely to contribute to the persistence of TMD symptoms. The prevalence of fibromyalgia in masticatory myofascial pain patients is higher than in the general population.<sup>146,147</sup> The presence of pain in other body sites in myofascial pain dysfunction (MPD) patients is high<sup>129</sup> and may indicate that the musculoskeletal problem affecting the jaws is part of a more generalized problem. In a follow-up study on MPD patients, the group that self-reported the coexistence of fibromyalgia had a higher frequency of chronic TMD symptoms.<sup>148</sup>

Trauma associated with the onset of TMD has always been considered to be a factor likely to increase severity and extend the course of the disorder. In a comparison of treatment between groups with TMD associated with trauma and without trauma, there was no difference to the treatment outcome, suggesting that trauma may not be an important factor; but more research is needed to draw conclusions.<sup>149</sup>

### Referral to a Pain Specialist

While the majority of patients with TMD are responsive to treatment and are appropriately managed by dental professionals in association with psychologists, physiotherapists, and other health care professionals, a small group of patients may be more appropriately managed by a pain specialist. This may be indicated when (1) the disability greatly exceeds what is expected on the basis of physical findings, (2) the patient makes excessive demands for tests and treatments that are not indicated, (3) the patient displays significant psychological distress (eg, depression), or (4) the patient displays aberrant behavior, such as continual nonadherence to treatment.<sup>150</sup>

## ▼ GENERAL CLINICAL CHARACTERISTICS

The most important feature of TMD is pain. Pain may be present at rest, may be continuous or intermittent, and characteristically increases with jaw functions such as chewing or opening wide. Other common findings include pain reported during palpation of the TMJ and/or muscles of mastication; a restricted range of mandibular movement or uncoordinated movements; and irregularities in the joints during movement, characterized by clicking or grating sounds. Myofascial pain is the most common TMD<sup>102</sup> and may present with or without restricted mouth opening. Pain causes the jaw-closing muscles to co-contract, so that the pain itself may influence the degree of mandibular restriction in cases of MPD.<sup>151,152</sup> Chronic TMD pain (like all chronic pain) results in psychological, behavioral, and social disturbances. The assessment and the treatment of these problems are equally important to the physical pathology.

Treatment goals for TMD are to control pain and to return mandibular motion and function to normal or as close to normal as possible. Clinical case studies suggest that the majority of individuals with TMD respond to conservative noninvasive therapy, making the use of invasive procedures unwarranted as initial therapy. No one treatment has emerged as superior although many of the treatments studied have shown some beneficial effect.<sup>153</sup> The symptoms of TMD tend to be intermittent, fluctuate over time, and are often self-limiting.<sup>154–156</sup> The process of deciding whether to treat and how aggressively to treat should include an assessment of the course of symptoms. Patients who are improving at the time of assessment may require a minimum of care and monitoring, compared to the individual whose symptoms are becoming progressively more severe and disabling.

The variations in treatment recommended by dentists have been explained by the gap between published information in the medical and dental literature and the individual beliefs and attitudes of practicing dentists.<sup>157,158</sup> These observations suggest that the treatment effect may be nonspecific and related more to the therapeutic relationship established between therapist and patient than to the specific treatment.<sup>159</sup>

Patients with irreversible anatomic abnormalities such as disk disorders are still able to regain painless jaw function.<sup>160,161</sup> Decreasing pain and improved physical findings are not directly related.<sup>162</sup> The presence of joint noises and deviations from the ideal in occlusion, in maxillomandibular relationships, and in the morphology of bony structures such as the condyle are relatively common in the general population. Evidence indicating prophylactic treatment of these anatomic abnormalities when no pain, impairment of function, or disability exists is lacking. Rather, treatment should be based on the severity of pain and disability and should be directed toward those factors that are considered to be important in initiating, aggravating, or perpetuating the disorder.

Episodes of pain and dysfunction may recur even after successful symptom control. Re-injury or factors that contributed to earlier episodes of symptoms may be responsible. Recurrence

should not be considered a treatment failure, and initiating previous treatment that was successful should be considered first. In one study, myogenous disorders required recurrent treatment more frequently than did articular disorders.<sup>163</sup>

For the smaller group of patients in whom TMD progresses to a chronic pain disorder, treatment becomes more complex. These patients may still benefit from local therapies but will also require more comprehensive management to address the emotional and behavioral disabilities that result from chronic pain. The drug therapy may also be complex, requiring knowledge and experience that are not common in general dental practice. These patients are often at risk for unnecessary investigations or treatments that may be harmful and that may further complicate their problems.<sup>164,165</sup>

At a recent National Institutes of Health conference on TMD therapy, the following conclusions were published:<sup>166</sup>

1. Significant problems exist with present diagnostic classifications because these classifications appear to be based on signs and symptoms rather than on etiology.
2. No consensus has been developed regarding which TMD problems should be treated and when and how they should be treated.
3. The preponderance of the data does not support the superiority of any method for initial management, and the superiority of such methods to placebo controls or to no treatment controls remains undetermined.
4. Because most individuals will experience the improvement or relief of symptoms with conservative treatment, the vast majority of TMD patients should be initially managed with noninvasive and reversible therapies.
5. The efficacy of most treatment approaches for TMD is unknown because most have not been adequately evaluated in long-term studies and because virtually none have been studied in randomized controlled group trials.
6. Therapies that permanently alter the patient's occlusion cannot be recommended on the basis of current data.
7. Surgical intervention should be considered for the small percentage of patients with persistent and significant pain and dysfunction who show evidence of pathology or evidence that an internal derangement of the TMJ is the source of their pain and dysfunction and for whom more conservative treatment has failed.
8. Relaxation and cognitive-behavioral therapies are effective approaches to managing chronic pain.

## ▼ SPECIFIC DISORDERS AND THEIR MANAGEMENT

### Myofascial Pain of the Masticatory Muscles

The term most commonly used for muscle pain that occurs with palpation is "myofascial pain." The ability to diagnose and explain the pathology associated with muscle pain is still a challenge for further research. Since treatment cannot be

designed to address a particular cause, multiple therapies for controlling symptoms and restoring range of movement and jaw function are usually combined in a management plan. These therapies are more effective when used together than when used alone.<sup>103,167-171</sup>

Most of the research on the natural course of this disorder suggests that for most individuals, symptoms are intermittent and usually do not progress to chronic pain and disability. The dentist is the appropriate clinician to manage these patients, using conservative methods. The principles of treating this disorder are based on a generally favorable prognosis and an appreciation of the lack of clinically controlled trials indicating the superiority, predictability, and safety of the treatments that are presently being recommended. The literature suggests that most treatments can be expected to have some beneficial effect although this effect may be nonspecific and not directly related to the particular treatment. Treatments that are relatively accessible, not prohibitive due to expense, safe, and reversible should be given priority. Treatments with these characteristics include education, self-care, physical therapy, intraoral appliance therapy, short-term pharmacotherapy, behavioral therapy, and relaxation techniques (Table 10-10). There is evidence to suggest that combining treatments produces a better outcome.<sup>172</sup> Occlusal therapy continues to be recommended by some clinicians as an initial treatment or as a requirement to prevent recurrent symptoms. Research does not support occlusal abnormalities as a significant etiologic factor. The evaluation of occlusion and the correction of occlusal abnormalities are an important part of dental practice but should not be a standard treatment of TMD.

### EDUCATION AND INFORMATION

A source of great anxiety for the patient is the possibility that the problem is a progressive and degenerative one that will lead to much greater pain and loss of function in the future. Patients may have sought prior consultations from other physicians and dentists who were not able to establish a diagnosis or explain the nature of the problem. This often leads to fears of a more catastrophic problem such as a brain tumor or other life-threatening disease. Explaining where the pain comes from and the varied nature of the symptoms that may occur is effective in reducing the patient's anxiety. Education is the basis for the self-care activities that patients can perform to aid in symptom control. This requires enough time in an unhurried environment for health care workers to provide information and to allow the patient to express his or her concerns and also to ask questions. This interaction is the basis for the therapeutic relationship and provides the patient with the understanding and ability to perform daily activities and to make choices about using the jaw. The patient has to participate in developing strategies to avoid stresses that aggravate symptoms or interfere with the ability to manage therapy.

### SELF-CARE AND HABIT REVERSAL

Attention to jaw activities that are unrelated to function (such as tooth clenching, jaw posturing habits, jaw-muscle tensing,

**TABLE 10-10 Initial Treatment of Myofascial Pain**

Treatment Component	Description
Education	Explanation of the diagnosis and treatment Reassurance about the generally good prognosis for recovery and natural course Explanation of patient's and doctor's roles in therapy Information to enable patient to perform self-care
Self-care	Eliminate oral habits (eg, tooth clenching, chewing gum) Provide information on jaw care associated with daily activities
Physical therapy	Education regarding biomechanics of jaw, neck, and head posture Passive modalities (heat and cold therapy, ultrasound, laser, and TENS). Range-of-motion exercises (active and passive) Posture therapy Passive stretching, general exercise, and conditioning program
Intraoral appliance therapy	Cover all the teeth on the arch the appliance is seated on Adjust to achieve simultaneous contact against opposing teeth Adjust to a stable comfortable mandibular posture Avoid changing mandibular position Avoid long-term continuous use
Pharmacotherapy	NSAIDs, acetaminophen, muscle relaxants, antianxiety agents, tricyclic antidepressants, clonazepam
Behavioral/relaxation techniques	Relaxation therapy Hypnosis Biofeedback Cognitive-behavioral therapy

NSAIDs = nonsteroidal anti-inflammatory drugs; TENS = transcutaneous electrical nerve stimulation.

and leaning on the jaw) is a critical beginning. Those behaviors associated with hyperactivity need to be replaced with restful jaw postures, and this should be part of any initial therapy. Habit control was found to be helpful in reducing pain in myofascial pain patients.<sup>173</sup> Dispensing a set of instructions to patients can help focus their attention on habits that are contributing to the aggravation or persistence of symptoms (Table 10-11). Instructions for habit reversal and for resting the jaw should also be accompanied by physical therapy that can be performed at home without specialized equipment (eg, the application of moist heat to the affected areas for 15 to 20 minutes twice daily, range-of-motion exercises that stay within the comfort zone, and the occasional use of ice for pain control or for relief of an acute injury superimposed over a chronic TMD [ice or a cold compress 10 minutes every 2 hours during an acute episode may be helpful]).

### PHYSIOTHERAPY

While clinical trials necessary to confirm the effectiveness of physiotherapy are lacking, the clinical literature suggests that

physiotherapy is a reasonable part of initial therapy.<sup>174</sup> Physiotherapy has been shown to be better than placebo, but no differences between various physical therapies have been shown.<sup>175</sup> Both passive and active treatments are commonly included as part of therapy. Posture therapy has been recommended to avoid forward head positions that are thought to adversely affect mandibular posture and masticatory-muscle activity.

Passive modalities such as ultrasound, laser, and transcutaneous electrical nerve stimulation (TENS) are often used to start physical therapy, reduce pain, and allow the patient to perform jaw exercises that promote recovery. TENS uses a low-voltage biphasic current of varied frequency and is designed for sensory counterstimulation for the control of pain. It is thought to increase the action of the modulation that occurs in pain processing at the dorsal horn of the spinal cord and (in the case of the face) the trigeminal nucleus of the brainstem. Ultrasound therapy relies on high-frequency oscillations that are produced and converted to heat as they are transmitted through tissue; it is a method of producing deep heat more effectively than the patient could achieve by using surface warming.

**TABLE 10-11 Instructions to Patients for Self-Care As Part of Initial Therapy**

Be aware of habits or patterns of jaw use. Avoid tooth contact except during chewing and swallowing. Notice any contact the teeth make. Notice any clenching, grinding, gritting, or tapping of teeth or any tensing or rigid holding of the jaw muscles. Check for tooth clenching while driving, studying, doing computer work, reading, or engaging in athletic activities and also when at work or in social situations and when experiencing overwork, fatigue, or stress.
Position the jaw to avoid tooth contacts. Place the tip of the tongue behind the top teeth, and keep the teeth slightly apart; maintain this position when the jaw is not being used for functions such as speaking and chewing.
Modify your diet. Choose softer foods and only those foods that can be chewed without pain. Cut foods into smaller pieces; avoid foods that require wide mouth opening and biting off with the front teeth or foods that are chewy and sticky and that require excessive mouth movements. Do not chew gum.
Do not test the jaw. Do not open wide or move the jaw around excessively to assess pain or motion. Avoid habitually maneuvering the jaw into positions to assess its comfort or range. Avoid habitually clicking the jaw if a click is present.
Avoid certain postures. Do not lean on or cup the chin when performing desk work or at the dining table. Do not sleep on the stomach or in postures that place stress on the jaw. Avoid elective dental treatment while symptoms of pain and limited opening are present.
Adopt other helping practices. During yawning, support the jaw by providing mild pressure underneath the chin with the thumb and index finger or with the back of the hand. Apply moist hot compresses to the sides of the face and to the temple areas for 10 to 20 min twice daily.

Exercises include exercises for increasing the range of motion in the comfort zone, passive stretching to increase mandibular motion, and isotonic and isometric exercises. Mouth-opening and mouth-closing exercises in a straight line in front of a mirror and/or with the tongue in contact with the palate are common controlled mouth-opening exercises.

Some physiotherapists apply mobilization techniques to increase mandibular motion. These are done passively under the control of the physiotherapist and will usually include distraction and some combination of lateral and protrusive gliding movements. The choice of treatment and timing is an individual consideration since the literature is not developed enough to provide specific guidelines.

### **INTRAORAL APPLIANCES**

Intraoral appliances (splints, orthotics, orthopedic appliances, bite guards, nightguards, or bruxing guards) are used in TMD treatment, and their use is considered to be a reversible part of initial therapy. A number of studies on splint therapy have demonstrated a treatment effect although researchers disagree as to the reason for the effect.<sup>156,172,176,177</sup> In a review of the literature on splint therapy, Clark found a reported improvement of 70 to 90%.<sup>178</sup> A decrease in masticatory-muscle activity has been associated with splint therapy.<sup>179,180</sup> Other studies suggest that the treatment effect cannot be specifically attributed to appliance therapy.<sup>181</sup> Theories that explain the effects include occlusal disengagement, altered vertical dimension, re-aligned maxillomandibular relationship, mandibular condyle repositioning, and cognitive awareness of mandibular posturing and habits.<sup>182</sup> This question will require further research; for the present, however, intraoral appliance therapy is considered to be a reversible treatment and is often included in initial treatment. The choice of material for the construction of an appliance remains one of individual preference. A study comparing a hard and soft material during a 3-month trial found no difference in outcome when either the hard or the soft appliance was used.<sup>183</sup> Long-term continuous wearing of an occlusal appliance is a risk for a permanent change in the occlusion.<sup>184</sup> This is a greater concern with appliances that provide only partial coverage or that occlude only on selected opposing teeth.

General dentists and dental specialists commonly use appliance therapy. Many designs and materials are used, but in a survey, a flat-plane splint made of hard acrylic was used more frequently than any other design or material.<sup>185</sup> The most common purposes advocated for appliance therapy are to provide joint stabilization, protect the teeth, redistribute forces, relax elevator muscles, and decrease bruxism.<sup>182</sup> The appliance most commonly used is described as a stabilization appliance or muscle relaxation splint. Such appliances are designed to cover a full arch and are adjusted to avoid altering jaw position or placing orthodontic forces on the teeth. The appliance should be adjusted to provide bilateral even contact with the opposing posterior teeth on closure and in a comfortable mandibular posture. Anterior guidance in the canine or incisor area is preferred and can usually be achieved with an appro-

priate acrylic contour. During the period of treatment as symptoms improve, the appliance should be re-examined periodically and re-adjusted as necessary to accommodate changes in mandibular posture or muscle function that may affect the opposing tooth contacts on the appliance. At the beginning of appliance therapy, a combination of appliance use during sleep and for periods during the waking hours is appropriate. This should be monitored to determine the most effective schedule for appliance use. Factors such as tooth-clenching when driving or exercising or pain symptoms that tend to increase as the day progresses may be better managed by increasing splint use during these times. To avoid the possibility of occlusal change, the appliance should not be worn continuously (ie, 24 hours per day) over prolonged periods. Many patients continue to wear stabilization splints during sleep with periodic monitoring. Full-coverage appliance therapy during sleep is a common practice to reduce the effects of bruxing and is not usually associated with occlusal change.

Splints that reposition the mandible anteriorly have been used effectively in treating disk displacements,<sup>186</sup> but they increase the risk of permanently altering the occlusion and should be used with caution. These splints have been made for the upper or lower arch although the maxillary appliance is better able to maintain a forward mandibular posture by using a ramp extending from the anterior segment that guides the mandible forward during closure. These appliances were used with greater frequency in the past to correct disk position as a step toward more permanently altering mandibular position through permanent changes in the occlusion. This approach was associated with great technical difficulties, and the treatment failed to correct disk displacement in a significant percentage of patients. Repositioning appliances used for short periods intermittently can be useful in controlling symptoms arising from the mechanical instability of the disk-condyle relationship. Short-term intermittent repositioning therapy may be helpful when transient episodes of jaw locking occur due to disk displacement and are accompanied by pain and dysfunction.

### **PHARMACOTHERAPY**

Mild analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), antianxiety agents, tricyclic antidepressants, and muscle relaxants are medications used as part of initial treatment. The long-acting benzodiazepine clonazepam was effective in a pilot study of TMD treatment.<sup>187</sup> Opioids are generally reserved for complex chronic pain disorders or (briefly) for acute injuries to the TMJ or muscles. Drug therapy as part of TMD management should follow the general principles of analgesic therapy and be used on a fixed dose schedule rather than as needed for pain. Drug therapy requires a thoughtful assessment of the potential risks relative to the benefits, including the clinician's own professional ability and confidence in using the particular drug or drugs.

NSAIDs are commonly prescribed for pain control in TMD therapy. There are a number of classes of NSAIDs, and the selective cyclo-oxygenase (cox-2) inhibitors celecoxib and rofecoxib offer the same analgesic effect, with a reduced risk

of gastrointestinal injury. These drugs should be used for a period of 2 weeks on a fixed dose schedule to assess their effectiveness. Other NSAIDs in common use include ibuprofen (400 mg four times daily, obtainable without a prescription), naproxen, diclofenac, and nabumetone. Diclofenac has been incorporated into a gel (pluronic lecithin organogel) and is applied externally on the skin over the painful muscle or joint. Capsaicin cream (0.025% or 0.075%) has also been used as an analgesic and can be applied to the skin in the painful area four times daily. Capsaicin has a burning quality on application that sometimes limits its usefulness.

Antianxiety agents are useful, especially during acute exacerbations of muscle pain. They are best used at night to avoid daytime sedation, and their potential for dependence is another limiting factor in their usefulness.

Muscle relaxants are a class of drugs that act in the central nervous system, inhibiting interneurons and depressing motor activity. They also have sedative effects that may contribute to their affect on symptoms. These drugs include carisoprodol, methocarbamol, chlorzoxazone, orphenadrine, and the tricyclic derivative cyclobenzaprine. Because their sedative effects will interfere with daily activities, these medications are best taken at night, before sleep.

Tricyclic antidepressants, particularly amitriptyline, have proven to be effective in managing chronic orofacial pain. Amitriptyline is analgesic at low doses, has sedative effects, and promotes restful sleep; all of these effects can be helpful in treatment. It is the anticholinergic effects of the drug (dry mouth, weight gain, sedation, and dysphoria) that often make it intolerable. An effective dose can be as low as 10 mg at night but can be increased gradually to 75 to 100 mg, depending on the patient's tolerance of the side effects.

Drug therapy with an NSAID and a benzodiazepine or cyclobenzaprine, along with the other components of initial therapy, may contribute to symptom control.<sup>188</sup> NSAIDs and benzodiazepines have adverse effects that require caution and monitoring of the drug therapy. A 2-week course with re-evaluation as initial therapy is a reasonable trial. TMD symptoms that are more chronic may require long-term medication use. The choice of drugs and their management as a part of a complex chronic pain disorder is different and is not covered in this chapter.

### **BEHAVIORAL THERAPY AND RELAXATION TECHNIQUES**

Integrating behavioral therapy and relaxation techniques in chronic pain management is effective.<sup>189</sup> In some cases, self-care and awareness of habits may not be sufficient to change behaviors that are contributing to symptoms. A more structured program supervised by a clinician who is competent in behavioral therapy offers a greater chance of addressing issues that are contributing factors. There is general agreement in the literature that behavioral and educational therapies are effective in the management of chronic pain disorders although the existing research is not sufficient to conclude that any one technique is superior. Relaxation techniques, biofeedback, hypnosis, and cognitive-behavioral therapy have all been used

to reduce symptoms in patients with TMD.<sup>190</sup> The mechanism of action with these techniques is unclear.

Relaxation techniques generally decrease sympathetic activity and (possibly) arousal. Deep methods include autogenic training, meditation, and progressive muscle relaxation.<sup>189</sup> These techniques are aimed at producing comforting body sensations, calming the mind, and reducing muscle tone. Brief methods for relaxation use self-controlled relaxation, paced breathing, and deep breathing. Hypnosis produces a state of selective or diffuse focus in order to induce relaxation. The technique includes pre- and postsuggestion and is used to introduce specific goals. Individuals vary greatly in their susceptibility to hypnosis and suggestion. Hypnosis does not effect endorphin production, and its effect on catecholamine production is not known.

Cognitive-behavioral therapy, which often includes relaxation techniques, changes patterns of negative thoughts. Hypnosis and cognitive-behavioral therapy have been hypothesized to block pain from entering consciousness, by activating the frontal limbic attention system to inhibit pain impulse transmission from the thalamic to the cortical structures.<sup>189</sup> Biofeedback is a treatment method that provides continuous feedback, usually by monitoring the electrical activity of muscle with surface electrodes or by monitoring peripheral temperature. The monitoring instruments provide patients with physiologic information that allows them to reliably change physiologic functions to produce a response similar to that produced by relaxation therapies. The patient performs relaxation exercises that are aimed at either lowering the electrical activity of the muscle or raising peripheral temperature. Repetitive practice using the biofeedback instrumentation provides the training for the patient to achieve a more relaxed state and also a greater sensitivity to the activities that have adverse effects.

Barriers to integrating behavioral and relaxation therapy exist in standard medical and dental care. The biomedical model of disease is emphasized in medical and dental education. The biomedical model explains disease in anatomic and pathophysiologic terms and does not stress psychosocial issues or the importance of the patient's experience of disease. Behavioral therapies can be time-intensive and may also not be supported by insurance companies.

For the patient who does not respond to initial treatment and who continues to have significant pain and disability, additional therapies beyond those described above are usually required. These patients are characterized more as having a chronic pain disorder than as having an anatomic abnormality that is unique to the masticatory system. Treatments used in the management of chronic pain are indicated for this group. Multidisciplinary pain clinic management may be required. The use of chronic pain medications, including opioids and the drugs described as adjuvant analgesics (tricyclic antidepressants, anticonvulsants, membrane stabilizers, and sympatholytics), may be part of a long-term management plan. Chronic pain disorders cause psychosocial changes that require management to reduce the associated disability.

A greater focus on behavioral therapies and coping strategies may provide additional benefits. Sleep disorders may require the use of hypnotics or other drug combinations to increase restorative sleep. Depression commonly accompanies chronic pain. Surgery for a chronic muscle pain disorder has no value.

### TRIGGER POINT THERAPY

Trigger point therapy has used two modalities: the cooling of skin over the involved muscle and stretching and the direct injection of local anesthetic into the muscle.

Spray and stretch therapy is performed by cooling the skin with fluoromethane (a refrigerant spray) and then gently stretching the involved muscle. The cooling is done to allow the stretching to take place without the pain leading to a reactive contraction or strain. This technique is described in detail by Travell,<sup>139</sup> who introduced the method as a treatment of MPD. Patients who respond to this therapy can use a variation at home by first warming the muscle, then briefly icing it, and then gently stretching the jaw passively.

Intramuscular trigger point injections have been performed by injecting local anesthetic, saline, or sterile water or by dry needling without depositing a drug or solution. The choice of solution for injection exists because of the lack of established benefits of any one method.<sup>191</sup> Injection of sterile water was associated with greater injection pain than was injection of saline<sup>192</sup> and should thus probably be avoided. In a study in which MPD patients were treated with injection of lidocaine or with dry needling, both groups reported decreased pain immediately after injection, but the group that received dry needling experienced greater soreness 48 hours after the procedure.<sup>193</sup> Procaine diluted to 0.5% with saline has been recommended because of its low toxicity to the muscle,<sup>194</sup> but lidocaine (2% without vasoconstrictor) is also used, with a standard dental syringe. There are no tested protocols for trigger point injection therapy, but the injections are often given to a muscle group in a series of weekly treatments for 3 to 5 weeks; this may be continued with modification of the intervals between injections, depending on the response.<sup>195</sup> If there is no response to the initial series of injections, this treatment should be abandoned. Hopwood and Abram analyzed treatment outcomes for 197 patients who received trigger point injection therapy for myofascial pain.<sup>196</sup> They found that (1) unemployment due to pain increased the odds of treatment failure threefold, (2) a longer duration of pain and greater change in social activity increased the risk of failure twofold, and (3) constant pain (versus intermittent pain) increased the likelihood of treatment failure by 80%. These results emphasize that chronic pain is a multidimensional and complex problem and that a variety of factors not directly related to the specific treatment effect will influence the outcome. Botulinum toxin has also been injected in trigger points for myofascial pain, but clinical trials to assess its effectiveness have not been performed.

### Oral Health Care Delivery in TMD Patients

Patients who require elective dental treatment should defer such procedures until the MPD symptoms have resolved or are

under reasonable control. Patients who develop active dental disease requiring treatment while they are suffering from myofascial pain are likely to have increased myofascial pain after dental procedures. The dentist should attempt to minimize the effect of a procedure on myofascial pain by using a variety of measures, as outlined in Table 10-12.

### Other TMD Treatments

This section has highlighted only the most common treatment methods and has not addressed many treatments that have been applied in the management of TMD. Acupuncture has received attention in chronic pain management. There are few clinical trials using acupuncture to treat TMD. Acupressure, different forms of injection therapy using natural substances, massage therapy, naturopathic and homeopathic remedies, and herbal remedies are just a few of the treatments that patients may pursue. There are also treatment systems for which there are several Web sites that provide patients with information for determining whether the system or product may be of value to them. There is a present need (which will increase in the future) for dentists to help patients interpret the treatments and products that are promoted, to avoid harm to patients and unnecessary expense in pursuing treatment to control a distressing disorder. Most of these treatments lack a significant literature that is even descriptive in relation to TMD treatment. This fact, coupled with the present lack of clarity in the scientific research about causes and about the effect of treatment, makes the need to establish a trusting doctor-patient relationship critical.

### Bruxism

Nocturnal bruxing is thought to aggravate or contribute to the persistence of pain symptoms associated with TMD. The etiology is not understood, but the evidence suggests that occlusal

**TABLE 10-12 Managing Temporomandibular Disorder Patients Requiring Dental Procedures**

Prior to the procedure	
	Use hot compresses to masseter and temporalis areas 10 to 20 minutes two to three times daily for 2 days
	Use a minor tranquilizer or skeletal-muscle relaxant (eg, lorazepam, 1 mg; cyclobenzaprine, 10 mg) on the night and day of the procedure.
	Start a nonsteroidal anti-inflammatory analgesic the day of the procedure, before the procedure.
During the procedure	
	Use a child-sized surgical rubber mouth prop to support the patient's comfortable opening; remove periodically to reduce joint stiffness.
	Consider intravenous sedation and/or inhalation analgesia.
	Provide frequent rest periods to avoid prolonged opening.
	Apply moist heat to masticatory muscles during rest breaks.
	Gently massage masticatory muscles during rest breaks.
	Perform the procedure in the morning, when reserve is likely to be greatest.
After the procedure	
	Extend the use of muscle relaxant and NSAID medication as necessary.
	Apply cold compresses to the TMJ and muscle areas during the 24 hours after the procedure.

NSAID = nonsteroidal anti-inflammatory drug; TMJ = temporomandibular joint.

abnormalities are not the cause.<sup>197,198</sup> Occlusal appliances may protect the teeth from the effects of bruxism but cannot be expected to prevent or decrease the bruxing activity.<sup>199</sup> When bruxing is considered to be the cause or a factor of TMD symptoms, oral appliance therapy is effective, but symptoms are likely to return when appliance therapy is withdrawn.<sup>200</sup> In one report, nocturnal aversive biofeedback and splint therapy caused a decrease in the frequency and duration of bruxing, but bruxing activity returned after treatment was withdrawn.<sup>201</sup> Occlusal splints worn during sleep have not been found to stop bruxing but do reduce the signs of bruxing.<sup>202</sup>

Recently, case reports of bruxism and symptoms of facial pain, earache, and headache associated with the onset of therapy with selective serotonin reuptake inhibitors (SSRIs) for depression have been published.<sup>203</sup> Symptoms of bruxing resolved when the dosage was decreased or when buspirone was added.<sup>204</sup> Buspirone has a postsynaptic dopaminergic effect and may act to partially restore suppressed dopamine levels associated with the use of SSRIs.

Tan injected severe bruxers in the masseter muscles with botulinum toxin in an open-label prospective trial and reported significant improvement in symptoms and minimal adverse effects.<sup>205</sup> The treatment effect lasted approximately 5 months and had to be repeated. Botulinum toxin exerts a paralytic effect on the muscle by inhibiting the release of acetylcholine at the neuromuscular junction.

### **Intracapsular Disorders of the TMJ: Articular Disk Disorders**

Intracapsular disorders affecting the TMJ are divided into two broad categories: arthritis and articular disk disorders. Either of these disorders may be present with or without symptoms. It is important for the clinician treating patients with TMD to distinguish between clinically significant intracapsular disorders that require therapy from those that are an incidental finding in a patient with facial pain from other causes.

Articular disk displacement (ADD) is an abnormal relationship between the disk, the mandibular condyle, and the articular eminence, resulting from the stretching or tearing of the attachment of the disk to the condyle and glenoid fossa. ADD may result in abnormal joint sounds, limitation in mandibular range of motion, and pain during mandibular movement, but the majority of cases of ADD occur without significant pain or joint dysfunction. MRI studies have demonstrated that ADD is present in 25 to 35% of the normal asymptomatic adult population.<sup>206,207</sup> This is similar to the finding of asymptomatic clinically insignificant disk displacement that is well documented in the knee and spine.<sup>208,209</sup> ADD of the TMJ does not appear to effect children below the age of 5 years.<sup>210</sup>

Loosened disks become displaced anterior to the mandibular condyle in a vast majority of cases. It is theorized that ADD occurs more frequently when the superior head of the lateral pterygoid muscle attaches to the disk. This attachment would pull a loosened disk anterior and medial to the condyle. Posterior disk displacement (when a portion of the disk is found posterior to the top of the condyle) does occur occasionally.<sup>211</sup>

The specific etiology of the majority of cases of disk displacement is poorly understood. Some cases result from direct trauma to the joint from a blow to the mandible. It is also generally believed that chronic low-grade microtrauma resulting from long-term bruxism or clenching of the teeth is a major cause of ADD, and studies using arthroscopic examination of the TMJ have demonstrated a relationship between intracapsular disorders and bruxism.<sup>212</sup> There is also evidence that ADD may be associated with a generalized laxity of joints, and studies have demonstrated a significantly higher incidence of generalized joint laxity in ADD patients than in normal controls.<sup>213</sup> Craniofacial morphology may also play a role in ADD.<sup>214</sup>

Clinicians have also theorized that indirect trauma from cervical flexion/extension injuries or certain types of malocclusion may also predispose an individual to disk displacement. These theories are not proven, and the specific series of events that commonly result in ADD is unknown. It is likely that a combination of mechanisms related to the anatomy of the joint and the facial skeleton, connective-tissue chemistry, and chronic loading of the joint increases the susceptibility of certain individuals to a disturbance of the restraining ligaments and displacement of the disk. ADD results in significant pain or dysfunction when accompanied by capsulitis, synovitis, and joint effusions.

### **CLINICAL MANIFESTATIONS**

Disk displacement is divided into stages based on signs and symptoms combined with the results of imaging studies. A simple classification system divides ADD into (1) anterior disk displacement with reduction (clicking joint), (2) anterior disk displacement with intermittent locking, and (3) anterior disk displacement without reduction (closed lock).

**Anterior Disk Displacement with Reduction.** This condition is caused by an articular disk that has been loosened because of elongation or tearing of restraining ligaments and has moved from its normal position on the top of the condyle. Alteration in the form of the disk may also cause movement from the normal position. A reducing disk displacement is common in the general population, and a clicking or popping joint is of little clinical significance unless the clicking is accompanied by pain or unless the patient experiences dysfunction due to intermittent locking. There are occasional patients who seek professional advice regarding treatment of an audible click that is not accompanied by pain but that may be socially embarrassing.

The clinician who is treating disk displacement in patients with jaw pain must distinguish the patient with myofascial pain and a co-incidental clicking joint from the patient whose pain is related directly to disk displacement. Clinicians should also be aware that symptoms of pain and dysfunction associated with anterior disk displacement with reduction resolve over time with minimal noninvasive therapy in the majority of cases.<sup>215</sup>

Patients with clinically significant anterior disk displacement with reduction will complain of pain during mandibu-



lar movement; the pain is most noticeable at the time of the click. Palpation and auscultation of the TMJ will reveal a clicking or popping sound during both opening and closing mandibular movements (the so-called reciprocal click). The clicking or popping sound due to anterior disk displacement with reduction is characterized by a click that occurs at a different point during opening and closing. For example, the opening click may be present at 25 mm of opening, and the closing click may be present at 10 mm. This is due to movement of the disk as the condyle moves it forward during mandibular opening. Clinicians examining patients with ADD may observe a deflection of the mandible early in the opening cycle, with correction towards the midline after the click. Tenderness will be present when ADD is accompanied by capsulitis or synovitis. TMJ effusion may be noted on a T2-weighted MRI scan.<sup>216</sup>

**Anterior Disk Displacement without Reduction (Closed Lock).** Closed lock may be the first sign of TMD occurring after trauma or severe long-term nocturnal bruxism. It is detected more frequently in patients with clicking joints that progress to intermittent brief locking and then permanent locking. A patient with an acute closed lock will often have a history of a long-standing TMJ click that suddenly disappears with a sudden restriction in mandibular opening. This limited mandibular opening occurs when the disk interferes with the normal translation of the condyle along the glenoid fossa. Other findings include pain directly over the joint during mandibular opening (especially at maximum opening) and limited lateral movement to the side away from the ADD since disks are most frequently displaced medially as well as anteriorly. During maximum mandibular opening, the mandible will deviate towards the side of the displacement. Palpation of the joints will reveal decreased translation of the condyle on the side of the disk displacement.

**Posterior Disk Displacement.** Posterior disk displacement has been described as the condyle slipping over the anterior rim of the disk during opening, with the disk being caught and brought backward in an abnormal relationship to the condyle when the mouth is closed. The disk is folded in the dorsal part of the joint space, preventing full mouth closure.<sup>217</sup> The clinical features are (1) a sudden inability to bring the upper and lower teeth together in maximal occlusion, (2) pain in the affected joint when trying to bring the teeth firmly together, (3) displacement forward of the mandible on the affected side, (4) restricted lateral movement to the affected side, and (5) no restriction of mouth opening.<sup>217</sup>

## MANAGEMENT

Longitudinal studies demonstrate that most symptoms associated with ADD resolve over time either with no treatment or with minimal conservative therapy.<sup>218</sup> For example, one study of patients with symptomatic anterior disk displacement without reduction showed that symptoms had resolved without treatment in 75% of cases after 2.5 years.<sup>219</sup> A long-term study

reported 87% of patients to be without pain or restricted movement 30 years after ADD was initially documented. Since symptoms associated with anterior disk displacement with and without reduction tend to decrease with time, the clinician should not treat patients on the assumption that asymptomatic clicking will inevitably progress to painful clicking or locking. Painful clicking or locking should initially be treated with conservative therapy

Recommended treatments for symptomatic ADD include splint therapy, manual manipulation and other forms of physical therapy, anti-inflammatory drugs, arthrocentesis, arthroscopic lysis and lavage, arthroplasty, and vertical ramus osteotomy. Many of these nonsurgical and surgical techniques are effective in decreasing pain and in increasing the range of mandibular motion although the abnormal position of the disk is not corrected.<sup>220</sup>

**Anterior Disk Displacement with Reduction.** Patients with TMJ clicking or popping that is not accompanied by pain do not require therapy. Flat-plane stabilization splints that do not change mandibular position and anterior repositioning splints have both been used to treat painful clicking. Anterior repositioning splints maintain the mandible in an anterior position, preventing the condyle from closing posterior to the disk. One meta-analysis that summarized results of previous studies concluded that repositioning splints were more effective than stabilization splints in eliminating both clicking and pain in patients with ADD.<sup>221</sup> Clinicians must weigh the potential benefits of using repositioning splints against the potential side effects of these appliances, which include tooth movement and open bite. Clinicians have advocated techniques that are designed to “recapture” the disk to its normal position, but studies have indicated that splint therapy, arthrocentesis, or arthroscopy rarely replaces the disk in a normal position. The painful symptoms resolve although the disk remains displaced.

**Anterior Disk Displacement without Reduction.** Some patients with closed lock may present with little or no pain whereas others have severe pain during mandibular movement. Treatment options should depend on the degree of pain associated with the ADD. Management of a locked TMJ may be nonsurgical or surgical. The goals of successful therapy are to eliminate pain and to restore function by increasing the range of mandibular motion. Replacing the disk in a normal position is not necessary to achieve these goals.

Patients who present with restricted movement but minimal pain frequently benefit from manual manipulation of the mandible and from an exercise program that is designed to increase mandibular range of motion by using manual methods or commercially available mandibular range-of-motion devices. Many practitioners will also use a flat-plane occlusal stabilization appliance to decrease the adverse effects of bruxism. Sato and colleagues reported that a combination of a flat-plane stabilization splint and anti-inflammatory drugs was successful in reducing pain and increas-

ing the range of motion in over 75% of patients.<sup>222</sup> The success of this therapy was attributed to decreased inflammation and to the gradual elongation of the posterior attachment, permitting increased movement of the condyle in the fossa. This therapy was also helpful in reducing secondary muscle pain. Patients with severe pain on mandibular movement may benefit from either arthrocentesis or arthroscopy. Flushing the joint with intra-articular corticosteroids to decrease inflammation or with sodium hyaluronate to increase joint lubrication and decrease adhesions has also been reported to help in decreasing the pain associated with nonreducing disk displacement.<sup>216</sup>

## ▼ ARTHRITIS OF THE TEMPOROMANDIBULAR JOINT

### Degenerative Joint Disease (Osteoarthritis)

Degenerative joint disease (DJD), also referred to as osteoarthritis, osteoarthritis, and degenerative arthritis, is primarily a disorder of articular cartilage and subchondral bone, with secondary inflammation of the synovial membrane. It is a localized joint disease without systemic manifestations. The process begins in loaded articular cartilage, which thins and clefs (fibrillation) and then breaks away during joint activity. This leads to sclerosis of underlying bone, subcondylar cysts, and osteophyte formation.<sup>223</sup> It is essentially a response of the joint to chronic microtrauma or pressure. The microtrauma may be in the form of continuous abrasion of the articular surfaces as in natural wear associated with age or as increased loading forces possibly related to chronic parafunctional activity. The fibrous tissue covering in patients with degenerative disease is preserved.<sup>224</sup> This may be a factor in remodeling and the recovery that is usually expected in osteoarthritis and osteoarthritis. The relationship between internal derangements and DJD is unclear, but a higher frequency of radiographic signs of DJD was observed in subjects with disk displacement without reduction.<sup>225</sup>

Degenerative joint disease may be categorized as primary or secondary although both are similar on histopathologic examination. Primary degenerative joint disease is of unknown origin, but genetic factors play an important role. It is often asymptomatic and is most commonly seen in patients above the age of 50 years, although early arthritic changes can be observed in younger individuals. Secondary degenerative joint disease results from a known underlying cause, such as trauma, congenital dysplasia, or metabolic disease.

### CLINICAL MANIFESTATIONS

DJD of the TMJ begins early and has been observed in over 20% of joints in individuals over the age of 20 years.<sup>226</sup> A study of patients below the age of 30 years presenting to a TMD clinic demonstrated that two-thirds of the patients had degenerative changes detected on tomograms.<sup>227</sup> The inci-

dence of degenerative changes increases with age, and such changes are found in over 40% of patients over 40 years of age. Richards and Brown observed a direct relationship, irrespective of age, between the rate and extent of dental attrition and degenerative disease of the TMJs in cadavers of aboriginal humans.<sup>228</sup> They also noted that the temporal bone exhibited more changes than did the condyle. Many patients with mild to moderate DJD of the TMJ have no symptoms although arthritic changes are observed on radiographs. The presence of pain in patients with DJD is associated with inflammation and joint effusions.

Degenerative changes of the TMJ detected on radiographic examination may be incidental and may not be responsible for facial pain symptoms or TMJ dysfunction; however, some degenerative changes may be underdiagnosed by conventional radiography because the defects are confined to the articular soft tissue. These soft-tissues changes are better visualized with MRI.<sup>229</sup>

Patients with symptomatic DJD of the TMJ experience unilateral pain directly over the condyle, limitation of mandibular opening, crepitus, and a feeling of stiffness after a period of inactivity. Examination reveals tenderness and crepitus on intra-auricular and pretragus palpation with deviation of the mandible to the painful side. Radiographic findings in degenerative joint disease may include narrowing of the joint space, irregular joint space, flattening of the articular surfaces, osteophytic formation, anterior lipping of the condyle, and the presence of Ely's cysts. These changes may be seen best on tomograms or CT scans (Figure 10-11). The presence of joint effusion is most accurately detected in T2-weighted MRI images.

**Treatment.** Degenerative joint disease of the TMJ can usually be managed by conservative treatment. Significant improvement is noted in many patients after 9 months, and a "burning out" of many cases occurs after 1 year.<sup>230</sup> It seems prudent to manage a patient with conservative treatment for 6 months to 1 year before considering surgery unless severe pain or dysfunction persists after an adequate trial of non-surgical therapy.

Conservative therapy includes nonsteroidal anti-inflammatory medications; heat; soft diet; rest; and occlusal splints that allow free movement of the mandible. It also may be necessary to concomitantly treat myofascial pain or meniscal defects. Intra-articular steroids can be used during acute episodes, but there is concern that repeated injections may cause degenerative bony changes.<sup>231</sup> Preliminary reports suggest that the anti-inflammatory effects of doxycycline therapy may be helpful in reducing pain associated with TMJ DJD.<sup>232</sup> When TMJ pain or significant loss of function persists and when distinct radiographic evidence of degenerative joint changes exists, surgery is indicated.<sup>233</sup> An arthroplasty, which limits surgery to the removal of osteophytes and erosive areas, is commonly performed. Artificial TMJs have been developed to treat patients with advanced degenerative changes of the TMJ.

## Synovial Chondromatosis

### CHONDROMETAPLASIA

Synovial chondromatosis (SC) is an uncommon benign disorder characterized by the presence of multiple cartilagenous nodules of the synovial membrane that break off resulting in clusters of free-floating loose calcified bodies in the joint. It is theorized that SC originates from embryonic mesenchymal remnants of the subintimal layer of the synovium that become metaplastic, calcify, and break off into the joint space.<sup>234,235</sup> SC most commonly involves one joint, but cases of multiarticular SC have been reported.<sup>236</sup> Some cases appear to be triggered by trauma whereas others are of unknown etiology. The knee and elbow are most commonly involved, and less than 100 cases of SC of the TMJ have been reported in the world medical literature.

More sophisticated imaging techniques, such as CT, and arthroscopy have revealed cases of SC that previously would have received other diagnoses, causing authors of recent publications to suspect that SC is more common than previously believed.<sup>237–240</sup> Extension of SC from the TMJ joint to surrounding tissues (including the parotid gland, middle ear, or middle cranial fossa) may occur.<sup>241</sup>

### CLINICAL MANIFESTATIONS

Slow progressive swelling in the pretragus region, pain, and limitation of mandibular movement is the most common presenting clinical picture. TMJ clicking, locking, crepitus, and occlusal changes may also be present.<sup>235</sup> The extension of the lesion from the joint capsule and involvement of surrounding tissues may make diagnosis difficult, causing SC to be confused with parotid, middle-ear, or intracranial tumors. Cases of SC which were mistaken for a chondrosarcoma have been reported. Intracranial extension may lead to neurologic deficits such as facial nerve paralysis. Conventional radiography may not lead to the diagnosis, due to superimposition of cranial bones that may obscure the calcified loose bodies.<sup>242</sup> A CT scan should be obtained if SC is suspected after clinical evaluation. The lesion may appear as a single mass or as many small loose bodies.<sup>237</sup> Arthroscopy may be necessary for accurate diagnosis, particularly when the loose bodies are not calcified and cannot be visualized by conventional radiology or CT.<sup>241</sup>

### TREATMENT

Treatment should be conservative and consist of removal of the mass of loose bodies. This may be done arthroscopically when only a small lesion is present, but arthrotomy is required for larger lesions. The synovium and articular disk should be removed when they are involved. Lesions that extend beyond the joint space may require extensive resection.

## Rheumatoid Arthritis

The percentage of rheumatoid arthritis (RA) patients with TMJ involvement ranges from 40 to 80%, depending on the group studied and the imaging technique used.<sup>229, 243–245</sup>

Studies using conventional radiography and tomography find fewer abnormalities than CT finds.<sup>229</sup> For example, Goupille, using CT, found TMJ changes in 88% of RA patients, but changes were also detected in more than 50% of controls.<sup>245</sup> CT changes did not correlate with clinical complaints. Avrahami detected condylar changes in approximately 80% of RA patients, using high-resolution CT.<sup>229</sup> Ackerman and colleagues, using tomography, detected erosive condylar changes in two-thirds of RA patients and stated that symptoms were related to the severity of radiographic changes.<sup>246</sup> The disease process starts as a vasculitis of the synovial membrane. It progresses to chronic inflammation marked by an intense round cell infiltrate and subsequent formation of granulation tissue. The cellular infiltrate spreads from the articular surfaces eventually to cause an erosion of the underlying bone.

### CLINICAL MANIFESTATIONS

The TMJs are usually bilaterally involved in RA. The most common symptoms include limitation of mandibular opening and joint pain. Pain is usually associated with the early acute phases of the disease but is not a common complaint in later stages. Other symptoms often noted include morning stiffness, joint sounds, and tenderness and swelling over the joint area.<sup>247</sup> The symptoms are usually transient in nature, and only a small percentage of patients with RA of the TMJs will experience permanent clinically significant disability.

The most consistent clinical findings include pain on palpation of the joints and limitation of opening. Crepitus also may be evident. Micrognathia and an anterior open bite are commonly seen in patients with juvenile RA. Larheim attributes the micrognathia to a combination of direct injury to the condylar head and altered orofacial muscular activity.<sup>248</sup> Ankylosis of the TMJ related to RA is rare. Radiographic changes in the TMJ associated with RA may include a narrow joint space, destructive lesions of the condyle, and limited condylar movement. There is little evidence of marginal proliferation or other reparative activity in RA in contrast to the radiographic changes often observed in degenerative joint disease. High-resolution CT of RA patients' TMJs will show erosions of the condyle and glenoid fossae that cannot be seen by conventional radiography.<sup>229</sup>

### TREATMENT

Involvement of the TMJ by RA is usually treated by anti-inflammatory drugs in conjunction with the therapy for other affected joints.<sup>249</sup> The patient should be placed on a soft diet during acute exacerbation of the disease process, but intermaxillary fixation is to be avoided because of the risk of fibrous ankylosis. Use of a flat plane occlusal appliance may be helpful, particularly if parafunctional habits are exacerbating the symptoms. An exercise program to increase mandibular movement should be instituted as soon as possible after the acute symptoms subside. When patients have severe symptoms, the use of intra-articular steroids should be considered. Prostheses appear to decrease symptoms in fully or partially edentulous patients.<sup>250</sup>

Surgical treatment of the joints including placement of prosthetic joints, is indicated in patients who have severe functional impairment or intractable pain not successfully managed by other means.

### Psoriatic Arthritis

Psoriatic arthritis (PA) is an erosive polyarthritis occurring in patients with a negative rheumatoid factor who have psoriatic skin lesions.<sup>251</sup> The skin lesions precede the joint involvement by several years. PA affects 5 to 7% of patients with psoriasis. Investigators suspect that the cutaneous and joint manifestations of the disease may be traced to the same immunologic abnormality.<sup>252</sup> PA commonly involves the fingers and spine. Pitting of the nails is observed in 85% of patients.

TMJ involvement was once considered rare in PA, with only 28 cases having been reported in the world literature, but recent studies by Könönen and Kilpinen suggest that TMJ involvement is more common than previously believed.<sup>253</sup>

### CLINICAL MANIFESTATIONS

The symptoms of PA of the TMJ are similar to those noted in RA, except that the signs and symptoms are likely to be unilateral.<sup>254</sup> Limitation of mandibular movement, deviation to the side of the pain, and tenderness directly over the joint may be observed on examination. Radiographic findings show erosion of the condyle and glenoid fossae rather than proliferation. Coronal CT is particularly useful in showing TMJ changes of PA.<sup>256</sup>

### TREATMENT

The management of PA is similar to the treatment of RA, with an emphasis on physical therapy and NSAIDs that control both pain and inflammation in many cases. Antimalarial drugs should not be used because they may cause severe skin reactions in patients with psoriasis. Immunosuppressive drugs, particularly methotrexate, are used for patients with severe disease that does not respond to conservative treatment. Only when there is intractable TMJ pain or disabling limitation of mandibular movement is surgery indicated. Arthroplasty or condylectomy with placement of costochondral grafts has been performed successfully.<sup>251</sup> Surgery may be complicated by psoriasis forming in the surgical scar (Koebner effect).<sup>255</sup>

### Septic Arthritis

Septic arthritis of the TMJ most commonly occurs in patients with previously existing joint disease such as rheumatoid arthritis, or underlying medical disorders (particularly diabetes). Patients receiving immunosuppressive drugs or long-term corticosteroids also have an increased incidence of septic arthritis. The infection of the TMJ may result from blood-borne bacterial infection or by extension of infection from adjacent sites such as the middle ear, maxillary molars, and parotid gland.<sup>256</sup> Gonococci are the primary bloodborne agents causing septic arthritis in a previously normal TMJ

while *Staphylococcus aureus* is the most common organism involved in previously arthritic joints.<sup>257</sup>

### CLINICAL SYMPTOMS

Symptoms of septic arthritis of the TMJ include trismus, deviation of the mandible to the affected side, severe pain on movement, and an inability to occlude the teeth, owing to the presence of inflammation in the joint space. Examination reveals redness and swelling in the region of the involved joint. In some cases, the swelling may be fluctuant and extend beyond the region of the joint.<sup>258</sup> Large tender cervical lymph nodes are frequently observed on the side of the infection; this helps to distinguish septic arthritis from more common types of TMJ disorders. Diagnosis is made by detection of bacteria on Grams stain and culture of aspirated joint fluid.

Serious sequelae include osteomyelitis of the temporal bone, brain abscess, and ankylosis. Facial asymmetry may accompany septic arthritis of the TMJ, especially in children. Of the 44 cases of ankylosis of the TMJ reviewed by Topazian, 17 resulted from infection.<sup>259</sup> The primary sources of these infections were the middle ear, teeth, and the hematologic spread of gonorrhea.

Evaluation of patients with suspected septic arthritis must include a review of signs and symptoms of gonorrhea, such as purulent urethral discharge or dysuria. The affected TMJ should be aspirated and the fluid obtained tested by Grams stain and specially cultured for *Neisseria gonorrhoeae*.

### TREATMENT

Treatment of septic arthritis of the TMJ consists of surgical drainage, joint irrigation, and 4 to 6 weeks of antibiotics.

### Gout and Pseudogout

Gouty arthritis is caused by long-term elevation of serum urate levels, which results in the deposition of crystals in a joint, triggering an acute inflammatory response. Acute pain in a single joint (monoarticular arthritis) is the characteristic clinical manifestation of gouty arthritis. Gouty arthritis of the TMJ appears to be very rare although crystal deposition may be apparent in tissues adjacent to the joint. An attack of gouty arthritis is most accurately diagnosed by examination of aspirated synovial fluid from the involved joint by polarized light microscopy. The detection of monosodium urate crystals confirms the diagnosis of gout.

An acute attack of gout may be successfully treated with colchicine, NSAIDs, or the intra-articular injection of corticosteroids.

The deposition of other crystals, such as calcium pyrophosphate dihydrate (CPPD) or calcium hydroxyapatite, may cause a syndrome that resembles gout and that has been referred to as pseudogout.<sup>260</sup> This disorder most frequently affects elderly individuals, and involvement of the TMJ has been reported in cases documented by the demonstration of characteristic CPPD crystals in synovial fluid.<sup>261</sup> Pseudogout of the TMJ has been successfully treated with colchicine or arthrocentesis.

## ▼ DEVELOPMENTAL DEFECTS AND TRAUMA

### Developmental Defects

Developmental disturbances involving the TMJ may result in anomalies in the size and shape of the condyle. Hyperplasia, hypoplasia, agenesis, and the formation of a bifid condyle may be evident on radiographic examination of the joint. Local factors, such as trauma or infection, can initiate condylar growth disturbances.

True condylar hyperplasia usually occurs after puberty and is completed by 18 to 25 years of age. Limitation of opening, deviation of the mandible to the side of the enlarged condyle, and facial asymmetry may be observed. Pain is occasionally associated with the hyperplastic condyle on opening.

Facial asymmetry often results from disturbances in condylar growth because the condyle is considered to be a site for compensatory growth and adaptive remodeling. The facial deformities associated with condylar hyperplasia involve the formation of a convex ramus on the affected side and a concave shape on the normal side. If the condylar hyperplasia is detected and surgically corrected at an early stage, the facial deformities may be prevented.

Deviation of the mandible to the affected side and facial deformities also are associated with unilateral agenesis and hypoplasia of the condyle. Rib grafts have been used to replace the missing condyle to minimize the facial asymmetry in agenesis. In cases of hypoplasia, there is a short wide ramus, shortening of the body of the mandible, and antegonial notching on the affected side, with elongation of the mandibular body and flatness of the face on the opposite side. Early surgical intervention is again emphasized to limit facial deformity.

### Trauma

#### FRACTURES

Fractures of the condylar head and neck often result from a blow to the chin (Figure 10-12). The patient with a condylar fracture usually presents with pain and edema over the joint area and limitation and deviation of the mandible to the injured side on opening. Bilateral condylar fractures may result in an anterior open bite. The diagnosis of a condylar fracture is confirmed by radiographic examination. Intracapsular nondisplaced fractures of the condylar head are usually not treated surgically. Early mobilization of the mandible is emphasized to prevent bony or fibrous ankylosis.

#### DISLOCATION

In dislocation of the mandible, the condyle is positioned anterior to the articular eminence and cannot return to its normal position without assistance. This disorder contrasts with subluxation, in which the condyle moves anterior to the eminence during wide opening but is able to return to the resting position without manipulation. It has been demonstrated that subluxation is a variation of normal function and



**FIGURE 10-12** Fractured and medially displaced condyle.

that the normal range of motion of the condyle is not limited to the fossa.

Dislocations of the mandible usually result from muscular incoordination in wide opening during eating or yawning and less commonly from trauma; they may be unilateral or bilateral. The typical complaints of the patient with dislocation are an inability to close the jaws and pain related to muscle spasm. On clinical examination, a deep depression may be observed in the pretragus region corresponding to the condyle being positioned anterior to the eminence.

The condyle can usually be repositioned without the use of muscle relaxants or general anesthetics. If muscle spasms are severe and reduction is difficult, the use of intravenous diazepam (approximately 10 mg) can be beneficial. The practitioner who is repositioning the mandible should stand in front of the seated patient and place his or her thumbs lateral to the mandibular molars on the buccal shelf of bone; the remaining fingers of each hand should be placed under the chin. The condyle is repositioned by a downward and backward movement. This is achieved by simultaneously pressing down on the posterior part of the mandible while raising the chin. As the condyle reaches the height of the eminence, it can usually be guided posteriorly to its normal position.

Postreduction recommendations consist of a decrease in mandibular movement and the use of aspirin or nonsteroidal anti-inflammatory medications to lessen inflammation. The patient should be cautioned not to open wide when eating or yawning because recurrence is common, especially during the period initially after repositioning. Long periods of immobilization are not advised due to the risk of fibrous ankylosis.

Chronic recurring dislocations have been treated with surgical and nonsurgical approaches. Injections of sclerosing solutions are not used as often now because of difficulty in controlling the extent of fibrosis and condylar limitation. Various surgical procedures have been advocated for treating recurrent

dislocations of the mandible; these include bone grafting to the eminence, lateral pterygoid myotomy, eminence reduction, eminence augmentation with implants, shortening the temporalis tendon by intraoral scarification, plication of the joint capsule, and repositioning of the zygomatic arch.

## Ankylosis

True bony ankylosis of the TMJ involves fusion of the head of the condyle to the temporal bone. Trauma to the chin is the most common cause of TMJ ankylosis although infections also may be involved.<sup>258</sup> Children are more prone to ankylosis because of greater osteogenic potential and an incompletely formed disk. Ankylosis frequently results from prolonged immobilization following condylar fracture. Limited mandibular movement, deviation of the mandible to the affected side on opening, and facial asymmetry may be observed in TMJ ankylosis. Osseous deposition may be seen on radiographs. Ankylosis has been treated by several surgical procedures. Gap arthroplasty using interpositional materials between the cut segments is the technique most commonly performed.

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