TEMPOROMANDIBULAR DISORDERS

by Steven L. Kraus, PT OCS MTC

This publication is a reprint of Chapter 8, Temporomandibular Disorders

from: Evaluation, Treatment and Prevention of Musculoskeletal Disorders Volume One—Spine 4th Edition

Original Work's Library of Congress Control Number 2004091469

Original Work's ISBN Number 1-879190-12-5

Reproduced with permission from:
The Saunders Group, Inc.
4250 Norex Drive
Chaska, MN 55318
www.thesaundersgroup.com

Author contact:
Steven L. Kraus PT OCS MPT
Physiotherapy Associates
2770 Lenox Road, Suite 102
Atlanta, GA 30324
stevekraus@mindspring.com

8
Temporomandibular Disorders

Steven L. Kraus PT OCS MTC

Section One—Introduction, Anatomy and Kinematics

Clinicians are faced with challenges in the management of patients with head and neck pain. Head and neck pain can originate from dental, neurologic, otolaryngologic, vascular, metaplastic, infectious disease or musculoskeletal conditions.[117] *Temporomandibular disorder* (TMD) is a collective term embracing a number of clinical problems that involve the temporomandibular joints (TMJ), muscles of mastication and associated structures.[132] Epidemiological studies indicate 3% to 6% of the population would benefit from treatment of TMD. This figure represents up to 17,000,000 patients.[47] Since physical therapists are skilled in treating non-disease musculoskeletal structures, they have a significant role to play in the management of TMD. The physical therapist's primary role in TMD management involves the evaluation and treatment of the temporomandibular joints, muscles of mastication and cervical spine tissues. Not all patients with signs of TMD will require modalities, exercises or manual procedures. However, all patients will require education to decrease fear and anxiety and correct misperceptions about their TMD conditions.

Voluminous textbooks, journal articles and continuing education courses address TMD. Unfortunately, discussion about the etiology, terminology, evaluation and management of TMD is often clouded by confusion and controversy. Despite all the scientific evidence supporting a particular evaluative and treatment procedure, an equal volume of contradictory material exists. This leaves both novice and seasoned clinicians in the physical therapy, dental and medical professions confused about TMD management.

This chapter focuses on the subclassifications of TMD that fall within the domain of physical therapists. Essential background such as anatomy, kinematics and terminology is presented, and the reader is exposed to a comprehensive classification, evaluation and treatment scheme.

Ideal management of patients with head and neck pain involves a team approach. For maximum patient benefit, each member of the dental, medical and physical therapy teams needs to understand what the other can offer in the management of TMD.

Osseous Structures

Temporal Bone

The temporal bone forms the roof of the TMJ. Pertinent bony landmarks are the postglenoid spine, mandibular fossa, articular eminence, articular crest, and articular tubercle (Fig 8-1).

The *postglenoid spine* or process is a downward extension of the squamosal portion of the temporal bone.[53] The postglenoid spine forms the posterior aspect of the *mandibular fossa* and is positioned anterior to the external auditory meatus. The postglenoid spine does not extend all the way laterally. Inserting a finger partially into the external auditory meatus provides reasonable access to tissues located posterior and lateral to the head of the condyle. This is one of several examination procedures used to identify possible inflammation of the TMJ.

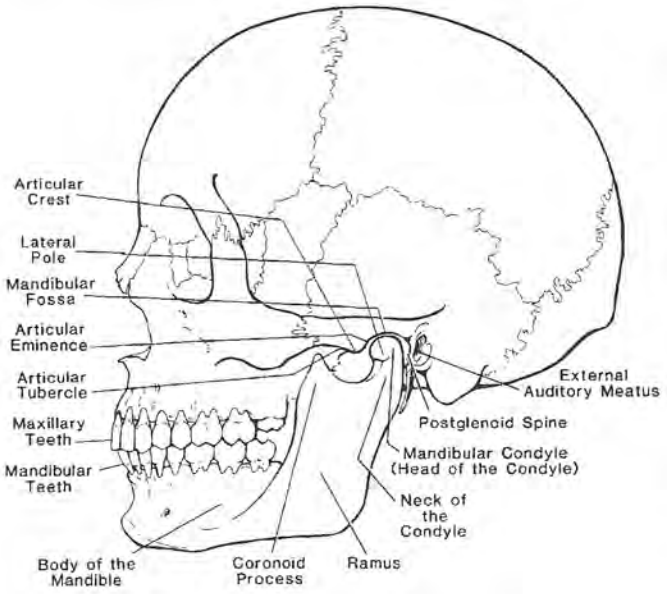

Figure 8-1: Skeletal Anatomy

The postglenoid spine offers attachments for the capsule and posterior attachment.[118] The concave mandibular fossa is occupied by the posterior band of the disc. The mandibular fossa is a non-articular portion of the TMJ.

The *articular eminence* is convex in the anteroposterior direction and concave mediolaterally. The articular eminence has a slope ranging from 40° to 60°.[10] Anteriorly, the articular eminence is separated from the *articular tubercle* by a bony landmark referred to as the *articular crest*. The articular tubercle is the most anterior portion of the roof of the TMJ. The articular tubercle area is concave in the mediolateral direction. During mandibular opening, the condyle translates along the articular eminence. With full mouth opening, if the head of the condyle translates onto the articular tubercle, TMJ hypermobility is present.[44]

Condyle

The condyle forms the floor of the TMJ (Fig 8-1). The condyle has an elliptical shape measuring approximately 20 mm mediolaterally and 10 mm anteroposteriorly.[190] The condyle is convex both anteroposteriorly and mediolaterally. Variation in the size and shape of the condyle is common, both from person to person and from one side to the other (Fig 8-2).[190]

The lateral pole of the condyle lies anterior to the transverse axis of the condyle and the medial pole lies posterior to the transverse axis of the condyle (Fig 8-3). A line running between the medial and lateral poles of each condyle

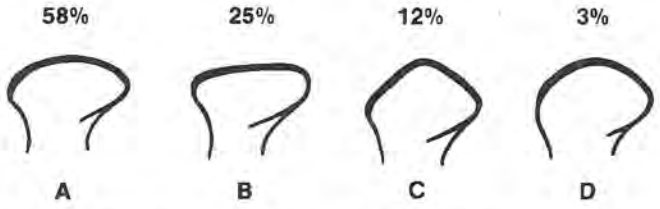

Figure 8-2: Various shapes of the mandibular condyle viewed in the frontal plane: A) Convex; B) Flat; C) Angular; D) Rounded

is referred to as the *long axis of the condyle*. Extending the long axis of each condyle medially forms an obtuse angle varying from 145° to 160° (Fig 8-3).[10]

Only the lateral pole can be palpated. The lateral pole is located directly in front of the tragus of the ear (Fig 8-1). Inflammation of the tissues that attach to or extend over the lateral pole can occur with TMD. Inferior to the head of the condyle is the neck of the condyle. Between the neck and the ramus (angle of the mandible) is the projection of the coronoid process for the attachment of the temporalis muscle (Fig 8-1). The ramus continues anterior to become the body of the mandible, which contains the lower arch of teeth.

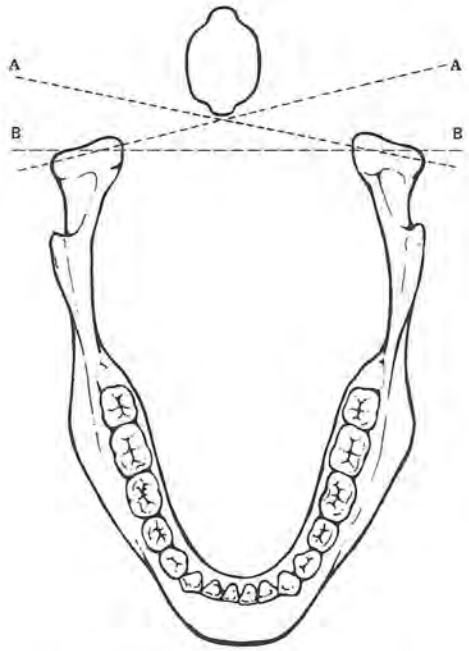

Figure 8-3: A) The long axis of the condyle is represented by a line drawn from the lateral to the medial pole. If the long axes of each condyle are extended, they intersect anterior to the foramen magnum. B) The lateral pole of the condyle lies anterior to the transverse axis of the condyle and the medial pole lies posterior to the transverse axis of the condyle.

The Occlusion

Unlike any other joint, the TMJ has teeth at one end of its lever arm (the mandible). The maxillary teeth are contained in the maxillary bone (Fig 8-1). Dawson defines centric occlusion or maximum intercuspation as the "relationship of the mandible to the maxilla when the teeth are in maximum occlusal contact, irrespective of the position or alignment of the condyle-disc assemblies".[36] Centric occlusion is the rigid end point of mandibular closure.

Malocclusion is any deviation or irregularity in the position, form or relation of teeth.[2] There are varying opinions as to the contribution of a malocclusion in the development and treatment of TMD. Clinicians who believe that malocclusion contributes to TMD would treat TMD by any one or a combination of occlusal adjustments, prosthetic rehabilitation, orthodontics and orthognathic surgery.[106] However, the literature suggests that malocclusion does not significantly contribute to TMD.[30,92,108,155,168] No reliable criteria exists to identify patients whose malocclusion is contributing to their TMD. McNamara and colleagues suggests that "the dental profession should be encouraged to manage TMD symptoms with reversible therapies, only considering permanent alterations of the occlusion in patients with very unique circumstances."[108] Nonetheless, clinicians should have a basic understanding of occlusal factors that have a potential association with TMD. A thorough discussion of occlusal factors is beyond the scope of this text, but patients with TMD who have malocclusion and have not responded to physical therapy should be referred to a dentist knowledgeable in TMD management.

TMJ—A Load Bearing Joint

The TMJ is a load bearing joint.[17,112] Joint loading occurs at the articulating surfaces on the temporal bone and head of the condyle.[10] The articulating surface on the temporal bone is located on the articular eminence, articular crest and articular tubercle areas. On the head of the condyle, the articulating surface is located on the anterior/anterosuperior portion (Fig 8-1).[118]

The articulating surfaces of the TMJ are covered by fibrocartilage that is avascular and aneural. Fibrocartilage has the same general properties found in hyaline cartilage but tends to be less dense,[81] has greater potential to remodel and is less likely to breakdown over time.[133]

In the TMJ, degenerative joint disease occurs first laterally, because TMJ loading occurs more laterally than medially. Lateral loading also might explain why the lateral collateral ligament of the TMJ loses its integrity with a subsequent affect on disc position.

The articular surfaces of the TMJ have been shown to be remarkably adaptable.[34] However, the adaptive capacity of the TMJ is not infinite, and some joints adapt better than others. Degenerative changes in the TMJ are the result of maladaptation to increased joint loading.[111] The articular disc affords some protection against excessive loading. Disc displacements may contribute to degenerative joint disease by increasing functional demands on the articular surfaces of the TMJ. The notion that disc displacement leads to degenerative joint disease has encouraged both surgical and nonsurgical approaches to "reposition" the displaced disc. Successful long-term repositioning of the disc is believed to stop the progression of advanced degenerative joint disease.[111] On the other hand, successful long term repositioning of the disc is sometimes problematic (see *Treatment for Disc Displacements – All Stages* on page 199). Some histological models suggest that degenerative joint disease may actually precede disc displacements. Therefore, disc displacement may be a sign of degenerative joint disease and not its cause.[41]

Intracapsular Structure

Articular Disc

The TMJ disc is a biconcave fibrocartilage structure lying between the head of the condyle and the temporal bone (Fig 8-4).[118] The disc consists of dense bundles of collagen fibers.[112,114] A firm yet flexible structure, the disc accommodates to the incongruities in the shape of the articulating surfaces. Knowledge of the disc's anatomy and attachments is essential to understand disc displacements and gain a realistic perspective on treatment.

The disc divides the joint into superior and inferior compartments or joint spaces. The upper joint space is larger than the lower joint space, extending further anterior

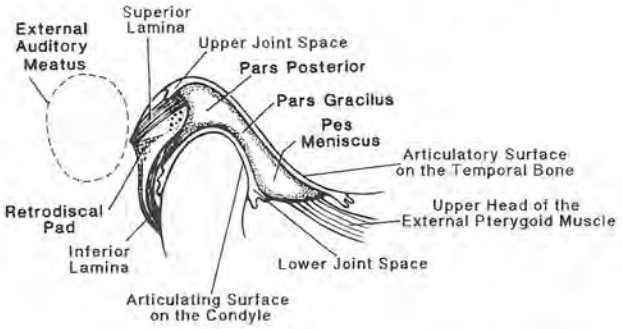

Figure 8-4: A sagittal view of the intracapsular structures of the right temporomandibular joint.

in the sagittal plane and overlapping the lower joint space in the coronal plane (Fig 8-5). The volumes of the upper and lower joint space are 1.2 ml and 0.9 ml respectively.[13]

Rees divides the disc into three bands according to thickness: anterior (pes meniscus), intermediate (pars gracilis) and posterior (pars posterior) (Fig 8-4).[141] The posterior band is thicker than the anterior band and the intermediate band is the thinnest. In centric occlusion, the posterior band of the disc is positioned superior on the condyle and in the mandibular fossae (Fig 8-4).[174] The intermediate band is positioned over the anterosuperior part of the condyle and along the articular eminence. The anterior band lies anterior to the condyle and anterior on the articular eminence.

The intermediate band of the disc is avascular and aneural.[189] In contrast, the disc's peripheral non-load bearing areas are vascularized and innervated. During mandibular function, the intermediate band maintains its position between the temporal bone and head of the condyle where load-bearing occurs.[174]

The thin intermediate band connecting the thicker posterior and anterior bands creates a biconcave shape. This anatomical feature creates a "self-seating" relationship of the disc to the condyle.[103,133] The self-seating feature, along with tight medial and lateral collateral ligaments, allows the disc to rotate anterior and posterior on the condyle[10,133] without displacing anterior to the condyle.[103]

Disc Attachments

Anterior Attachment. The disc attaches anteriorly to the capsule and to the upper head of the lateral pterygoid muscle (Fig 8-4).[12] The upper head of the lateral pterygoid muscle influences disc movement.

Medial and Lateral Attachments. The medial and lateral collateral ligaments attach the disc firmly to the medial and lateral poles of the condyle.[174] The lateral collateral ligament is relatively thin compared to the medial collateral ligament.

Posterior Attachment. The disc is contiguous with the posterior attachment (Fig 8-4).[133] The posterior attachment consists of the superior lamina or stratum and the inferior lamina or stratum with the retrodiscal pad lying between the two laminae.[151,152]

The posterosuperior disc attaches to the superior lamina. The superior lamina travels posteriorly to attach in the area of the postglenoid spine. The superior lamina contains a branching meshwork of elastic fibers.[151] The extensibility of the superior lamina ranges from 7 to 10 mm in the fresh cadaver specimen.[141]

The posteroinferior disc attaches to the inferior lamina. The inferior lamina courses posteriorly around the back of the condyle to attach to the posterior aspect of the neck of the condyle.[151] The inferior lamina is composed mainly of collagenous fibers with little elastic tissue.

The retrodiscal pad is a part of the posterior attachment.[151,152] The retrodiscal pad contains small-caliber, loosely associated collagen fibers, a branching system of elastic fibers, fat deposits, a specialized arterial supply, a large venous plexus, lymphatics, a profuse nerve supply and many large blood-filled endothelium-lined spaces.[151,152] When the condyle translates forward, the volume of the retrodiscal tissue expands due to venous distention, filling the mandibular fossa.[152,174] During closure, the retrodiscal pad returns to its smaller size and shape.

Intracapsular inflammation of the TMJ can result from inflammation of the posterior attachment. Inflammation of the posterior attachment should be suspected if the patient complains of pain during jaw movement and the patient's symptoms are reproduced or increased with palpation via the external auditory meatus.

Periarticular Tissues

Capsule

The capsule is composed of fibrous connective tissue.[74] Superiorly, the capsule is attached to the temporal bone. Inferiorly, the capsule tapers to attach to the neck of the condyle. The capsule blends medially and laterally with the medial and lateral collateral ligaments only at the medial and lateral poles in the lower joint space (Fig 8-5).[74]

In the superior joint space, the capsule has no medial and lateral attachments to the disc.[74] Further laterally, the capsule thickens to become the TMJ ligament. The close anatomical relationship between the capsule and TMJ ligament makes it difficult to distinguish between the two.[53] Anteriorly, the capsule blends with the upper and lower head of the external pterygoid muscle and anterior disc.[12] Posteriorly, the capsule attaches to the postglenoid spine.[74]

The capsule is lined by a highly vascular, synovial fluid-producing membrane that supplies nutrients to the avascular intracapsular tissues. The capsule is richly innervated with sensory receptors and nociceptors.[125,171]

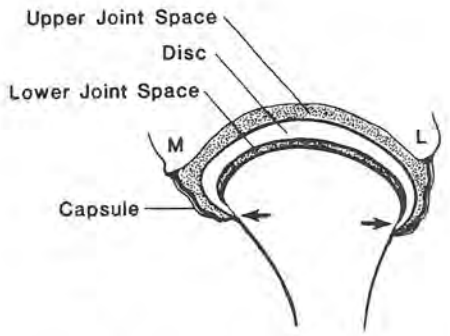

Figure 8-5: A frontal view of the left TMJ. Arrows depict attachments of the collateral ligaments and capsule to the medial and lateral poles.

The capsule contains articular mechanoreceptors that initiate reflexes possibly involved in jaw control and kinesthetic and perceptional awareness of the mandible.[125] The capsule contains four types of receptor nerve endings that are differentiated based on morphological and functional characteristics.[33,171] TMJ receptors are located in the fibrous joint capsule, the TMJ and lateral collateral ligaments and the posterior attachment, but are absent from the central disc and synovial tissues. Terminating on the receptors are the deep temporal, masseteric and auriculotemporal nerves which originate from the mandibular division of Cranial V.[86]

Injection of a local anesthetic into the capsule of healthy subjects causes decreased jaw control and significant deterioration in perception of mandibular position.[85] Fibrous adhesions of the capsule or joint inflammation causing joint effusion may contribute to a patient's lack of awareness of jaw movement and position.

Temporomandibular (TMJ) Ligament

The temporomandibular (TMJ) ligament is often referred to as the lateral ligament because it is continuous with the capsule and reinforces the capsule laterally (Fig 8-6).[21] The TMJ ligament is composed of two parts: a superficial oblique portion running laterally from the zygomatic portion of the temporal bone to the neck of the condyle, and a deeper more horizontal part from the same origin to the lateral pole.[149]

The inner (horizontal) portion of this ligament limits posterior movement of the condyle, protecting the posterior attachment from trauma.[120] Some authors state that the arrangement of the outer (oblique) fibers prevents separation of the condyle, disc and temporal fossa and restrains condylar movement on maximum mandibular opening,

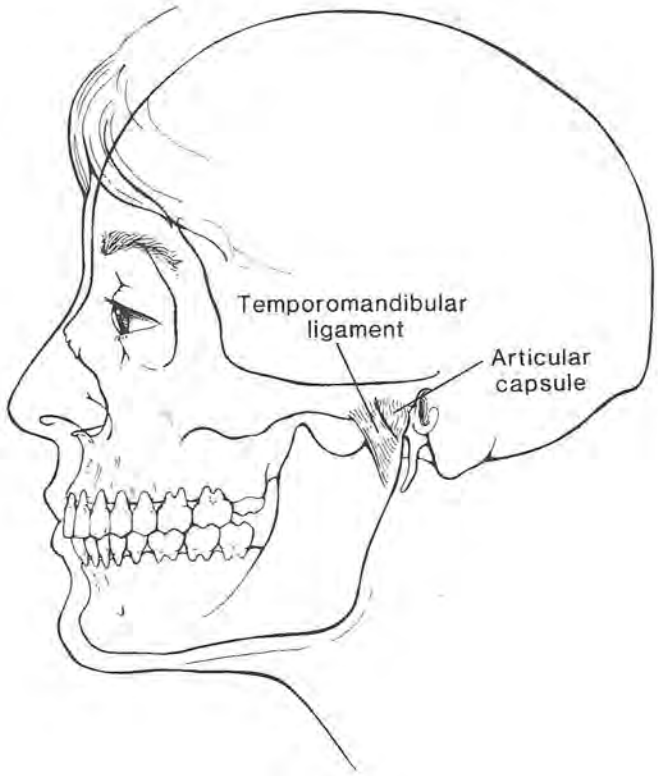

Figure 8-6: A lateral view of the temporomandibular joint showing the outer oblique TMJ ligament and capsule.

protrusion and lateral excursion.[69,120,133] The TMJ ligament is also believed to assist in the transition from condylar rotation to condylar translation.[69,70]

Stylomandibular and Sphenomandibular Ligaments

The stylomandibular and sphenomandibular ligaments are extracapsular ligaments (Fig 8-7).[21] The stylomandibular ligament extends from the styloid process of the temporal bone to the angle of the mandible. The sphenomandibular ligament has partial attachment superiorly to the sphenoid spine of the sphenoid bone; the remaining attachment is continuous with the medial capsule. In the medial capsule, a portion of the sphenomandibular ligament enters the petrotympanic fissure merging with the anterior malleolar ligament. Inferiorly, the sphenomandibular ligament attaches medially to the mandible.[149]

The role of the stylomandibular and sphenomandibular ligaments during mandibular dynamics is uncertain. Their role may be to protect the joint during wide excursive movements.

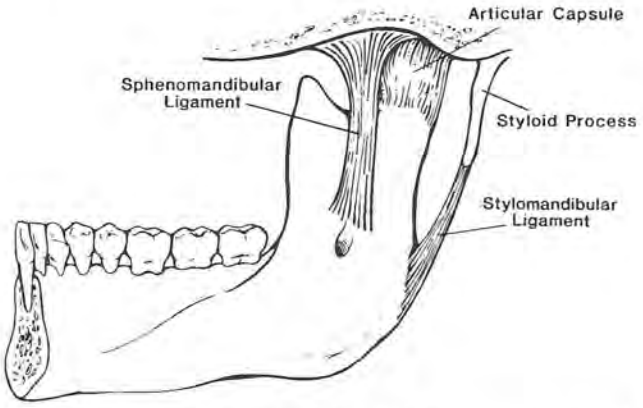

Figure 8-7: A medial view of the right temporomandibular joint showing the spenomandibular and stylomandibular ligaments and capsule.

Anterior Malleolar Ligament

The anterior malleolar ligament is a connection of fibrous tissue between the TMJ and the middle ear.[5,139] The anterior malleolar ligament, along with the sphenomandibular ligament, originates from the sphenoid bone and the medial capsule. It passes through the petrotympanic fissure to insert on the malleus. When tension is applied to the sphenomandibular ligament or the medial capsule of cadavers, movement of the chain of ossicles and the tympanic membrane is observed.[139] The functional importance of the anterior malleolar ligament has been debated.[5,149] It is speculated that ear symptoms may result from tension of the anterior malleolar ligament. Anterior malleolar ligament tension may be caused by tension of the sphenomandibular ligament occurring at end range jaw movements. Anterior malleolar ligament tension may also occur from disc displacement placing tension on the medial capsule.

Innervation

The TMJ is primarily innervated by the auriculotemporal nerve.[170] The auriculotemporal nerve is a branch of the posterior trunk of the mandibular nerve, a division of the trigeminal nerve. The auriculotemporal nerve innervates the posterior attachment and the posterior and lateral joint capsule. The anterior and medial joint is innervated by the masseteric and posterior deep temporal nerves, which come from the anterior trunk of the mandibular nerve.

Anesthetic block of the auriculotemporal nerve can help determine whether the patient's pain is arthrogeneous in origin.[10] If the patient's symptoms are myogenous or referred from other adjacent areas (e.g., cervical spine), symptoms are not affected by an anesthetic block. Clinicians should be aware of the placebo effect, which may result in a false positive response to this procedure.

Muscles Associated with the TMJ

The many skeletal muscles involved with mandibular movement are called the muscles of mastication. Muscles that attach directly to the mandible have the greatest influence on jaw function. Muscles in the neck provide secondary support during jaw function and should not be ignored. Eggleton and Langton provide a more detailed description of the origin and insertion of each muscle.[53]

Controversy exists over the number of heads of the lateral pterygoid muscle, but most sources agree there are two heads—a superior and an inferior.[12] Therefore, the following discussion assumes two heads.

Primary Muscles for Mandibular Closure

Temporalis. The temporalis is a large fan-shaped muscle originating from the temporal fossa. The temporalis inserts primarily along the coronoid process and extends to the anterior border of the ramus just posterior to the third molar.

Masseter. The masseter is a quadrilateral muscle originating from the anterior two thirds of the lower border of the zygomatic arch. The fibers run down and back to insert on the lateral aspect of the ramus.

Medial (Internal) Pterygoid. The medial pterygoid is a quadrilateral muscle originating from the pterygoid plate and palatine bone. The insertion is along the medial angle of the mandible.

Primary Muscles for Mandibular Opening

Inferior Head of the Lateral (External) Pterygoid. The lateral pterygoid muscle originates from the lateral surface of the lateral pterygoid plate. The insertion is into the medial half of the neck of the condyle. This muscle opens the mouth and protrudes the mandible. It cannot be palpated extraorally or intraorally.

Masticatory muscle involvement often causes a limitation in mandibular opening. However, if the inferior head of the lateral pterygoid is in spasm, patients are unable to bring their back teeth together on the side of the lateral pterygoid spasm. In theory, spasm of the lateral pterygoid muscle is caused by a quick stretch. Examples include a blow to the chin or an abrupt change in the patient's centric occlusion.

Other Muscles of Mastication

Superior Head of the Lateral Pterygoid. The origin of the superior head is on the greater wing of the sphenoid bone. Approximately $^1/_3$ of the superior head inserts on the anterior and medial disc and capsule.[12] The remaining portion of the superior head attaches to the medial $^1/_3$ of the neck of the condyle.[12,133] The superior head stabilizes the disc during function.

Hyoid Muscles. The hyoid muscles facilitate mandibular opening. The infrahyoid muscles stabilize the hyoid bone while the suprahyoid muscles open the mouth. The origin and insertions of the suprahyoid and infrahyoid muscles are not detailed here.[53]

Osteokinematics

The TMJ is a synovial ginglymoarthrodial joint. The TMJ is ginglymoid in that it provides a hinging movement and arthrodial in that it provides for a freely movable gliding motion.[118] The disc moves independently of both the condyle and temporal bone during active movements of the mandible.

Osteokinematics pertains to the overall movement of bones with little reference to their related joints.[100,101] Osteokinematics of the mandible are depression, protrusion and lateral excursion. These movements are often measured in millimeters during the physical examination.

Reported ranges for normal mandibular movements are rather arbitrary.[167] Clinically, it is more important to base treatment decisions on the patient's perception of what is functional. If measurements are used, age and gender should be taken into account, because wide variation exists. Baseline measurements can be useful to assess the patient's response to treatment. "Normal" ranges for osteokinematic movements of the mandible should be used as guidelines only and not as rigid goals in treatment.

Deviation and deflection are also assessed during mandibular opening and protrusion. *Deviation* is movement of the mandible away from midline and back to midline. Deviation is a "S" curve movement of the mandible. *Deflection* is movement of the mandible away from midline that does not return to midline. Deflection is a "C" curve movement of the mandible.

Clinicians like to see symmetry with jaw dynamics. However, when deviations and deflections are observed, it is important to remember that midline opening can be affected by deviations in anatomy caused by adaptive remolding of the joint surfaces and normal anatomical variations in the size and shapes of the condyle heads, the slope of the articular eminence, the long axes of the condyle heads and their relationship to the necks of the condyles, and differences in the distance between the condyles and rami.[157,176] Asymmetry is the rule rather than the exception. Treatments goals should be directed toward pain reduction and functional improvement, and not necessarily toward symmetrical mandibular dynamics. As a general rule, deviations and deflections associated with functional opening and protrusion are not significant. Deflection associated with limited mandibular movement is significant, and may indicate limited condylar translation on the side the deflection occurred toward. Deviation is rarely associated with limited mandibular function.

In addition to providing a baseline, measuring osteokinematic movements may help to differentially diagnosis TMD arthrogenous involvement from TMD myogeneous involvement. A patient who has limited opening but has normal protrusion and lateral excursion likely has myogeneous dysfunction. On the other hand, if the patient has limited opening, limited protrusion and limited lateral excursion, the dysfunction is likely arthrogenous.

Depression

Mandibular depression is mouth opening. Maximum opening of normal subjects ranges from 33 to 72 mm, depending on gender and age.[71,167] *Functional mandibular depression* is roughly 40 mm.[167] Mandibular depression is measured with a millimeter ruler between the tip of the right or left maxillary and mandibular central incisors (Fig 8-8).

Figure 8-8: Mandibular depression measured with a millimeter ruler. Functional mandibular depression is roughly 40 millimeters.

Figure 8-9: Functional protrusion with the bottom central incisors moving past the tip of the upper central incisors.

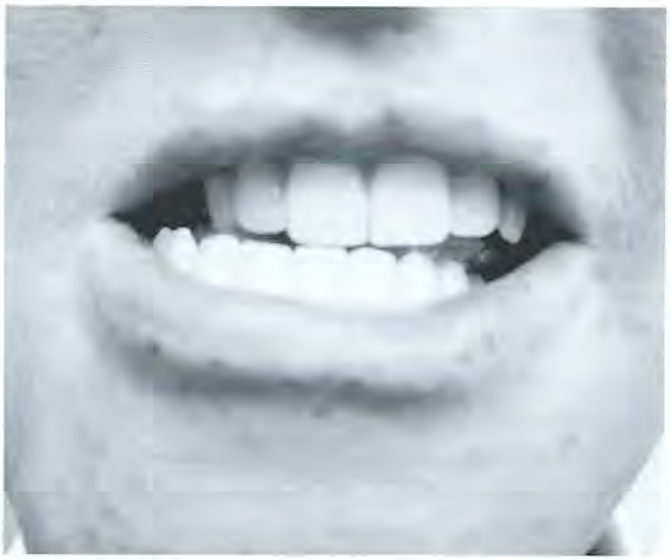

Figure 8-10: Functional lateral excursion to the right with the bottom canine moving past the tip of the upper canine.

Protrusion

Protrusion is anterior movement of the mandible in the horizontal plane. Normal protrusion ranges from 5 to 7 mm.[167] Though a ruler can be used to measure protrusion, it is easier to assess by visually inspecting the relationship between the patient's central incisors.

Functional mandibular protrusion is the ability to actively protrude the mandible to at least an end to end position between the maxillary and mandibular central incisors. Ideally, the mandibular central incisors should move past the maxillary central incisors by 1 to 2 mm (Fig 8-9). This amount of protrusion will allow patients to pronounce words such as "hiss", "house", "church", "judge", and "steeples", and to bite into an apple.

Lateral Excursion

Lateral excursion involves moving the mandible laterally in the horizontal plane. Normal lateral excursion is 5 to 7 mm to each side.[167] Though a ruler can be used to measure lateral excursion, it is easier to assess by visually inspecting the relationship between the patient's canines.

Functional lateral excursion is the ability to actively move the mandible laterally to at least an end to end position between the mandibular and maxillary canines. Ideally, the mandibular canine should move past the maxillary canine by 1 to 2 mm bilaterally (Fig 8-10). This range of lateral excursion will allow patients to chew food, since chewing involves lateral movements.

Arthrokinematics

Arthrokinematics pertains to active and passive accessory movements between two joint surfaces. Arthrokinematic movements permit full, pain-free movements in diarthrodial joints.[101]

Active accessory movements occur in response to muscle contraction. The active accessory movements of the TMJ are compression, rotation, translation, and spin. Compression occurs during all mandibular movements since the TMJ is a load bearing joint.

Passive accessory movements of the condyle are distraction and lateral glide. Passive accessory movements are the result of an external force, not muscular contraction. Passive accessory movement is also referred to as *joint play*. Joint play is the inherent quality of the joint to "give" and is minute.[110] An example of joint play is rotation of the metacarpophalangeal (MCP) joint. Although one cannot actively rotate the MCP joint, MCP rotation occurs when grasping a baseball. The external force causing MCP rotation is the baseball. Similarly, chewing food causes ipsilateral condylar distraction and lateral glide.[70,73,78,140] The bolus of food is the outside force causing these joint play movements.

Accessory Movement During Mandibular Depression

Condylar rotation and translation occur during mandibular opening and closing.[59] Contraction of the supra and infrahyoid muscles initiates mandibular opening.

Condylar rotation occurs during the first 10 mm of opening. After 10 mm, contraction of the lower head of the lateral pterygoid translates the head of the condyle anteriorly. After 10 mm, rotation and translation occur together until functional opening has been achieved.

During opening, the TMJ ligament is thought to assist the muscles in the transition from condylar rotation to translation. As the condyle rotates, the neck of the condyle moves posteriorly. This tightens the oblique portion of the TMJ ligament (Fig 8-11). TMJ ligament tightening occurs at approximately 10 mm of opening. Additional opening occurs only if the condyle translates anteriorly, thereby decreasing tension in the TMJ ligament.[69,70]

Clinically, tightness of the TMJ ligament may cause limited translation of the condyle. A tight TMJ ligament can occur from trauma or joint immobility, the same events that cause fibrous adhesions (See *Fibrous Adhesions* on page 203.)

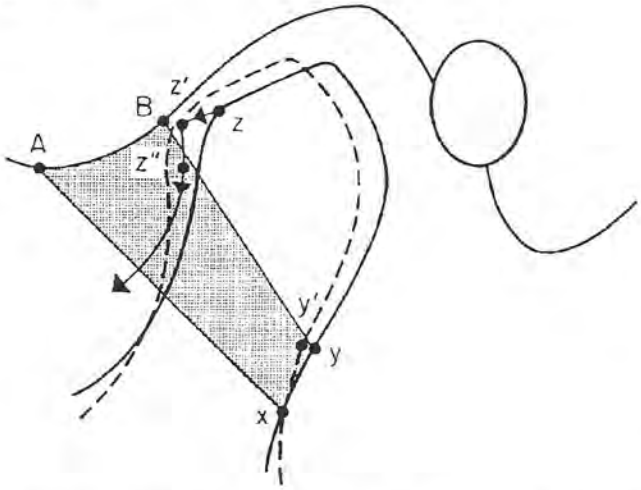

Figure 8-11: Upper (A and B) and lower (x and y) attachments of the temporomandibular ligament. During opening, rotation of the condyle moves points x and y posteriorly, thereby tightening the ligament. Functional opening occurs when the condyle translates forward.

Accessory Movement During Mandibular Protrusion

Condylar translation occurs during mandibular protrusion, and is a result of contraction of the lower head of the lateral pterygoid muscle.

Accessory Movement During Mandibular Lateral Excursion

Mandibular lateral excursion is accompanied by condylar translation contralaterally and condylar spin ipsilaterally.

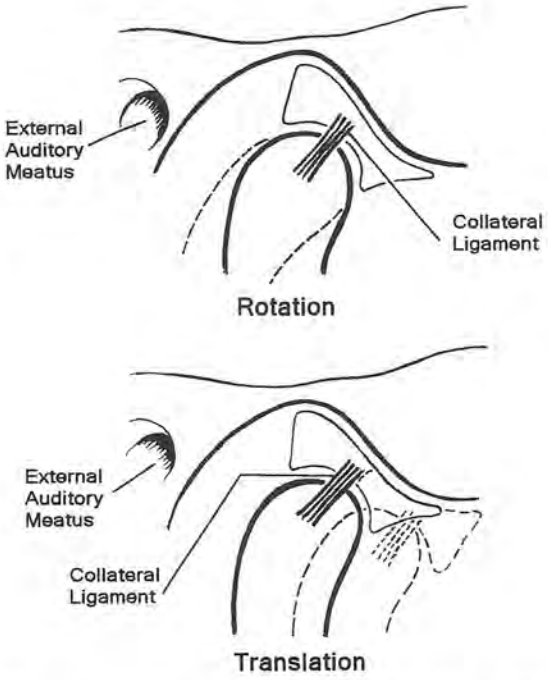

Figure 8-12: Rotation occurs in the lower joint space between the condyle and the inferior surface of the disc. Translation occurs in the upper joint space between the superior surface of the disc and temporal bone.

Condylar translation is a result of ipsilateral contraction of the lower head of the lateral pterygoid.

Disc Movement with Mandibular Opening

Though the disc is firmly attached to the head of the condyle by collateral ligaments, the disc and condyle can still rotate independently from one another. During active mandibular opening, rotation occurs in the lower joint space between the condyle and the inferior disc surface.[10,133] Translation occurs in the upper joint space between the superior disc and the temporal bone (Fig 8-12).

For the first 10 mm of mandibular opening, the condyle rotates below a relatively stationary disc. After 10 mm of opening the condyle translates anteriorly. The disc also translates anteriorly with the condyle because it is firmly attached to the condyle by the collateral ligaments.[133] As the disc and condyle both translate anteriorly, the disc rotates posteriorly in relationship to the condyle. Posterior rotation of the disc on the condyle occurs because of the "self-seating" disc to condyle relationship and from tension developing in the posterior attachment (superior lamina).[10,103,137] At the end of mandibular opening, both the disc and condyle have translated anterior in relationship to the temporal bone as the disc rotated posteriorly on the condyle (Fig 8-13).

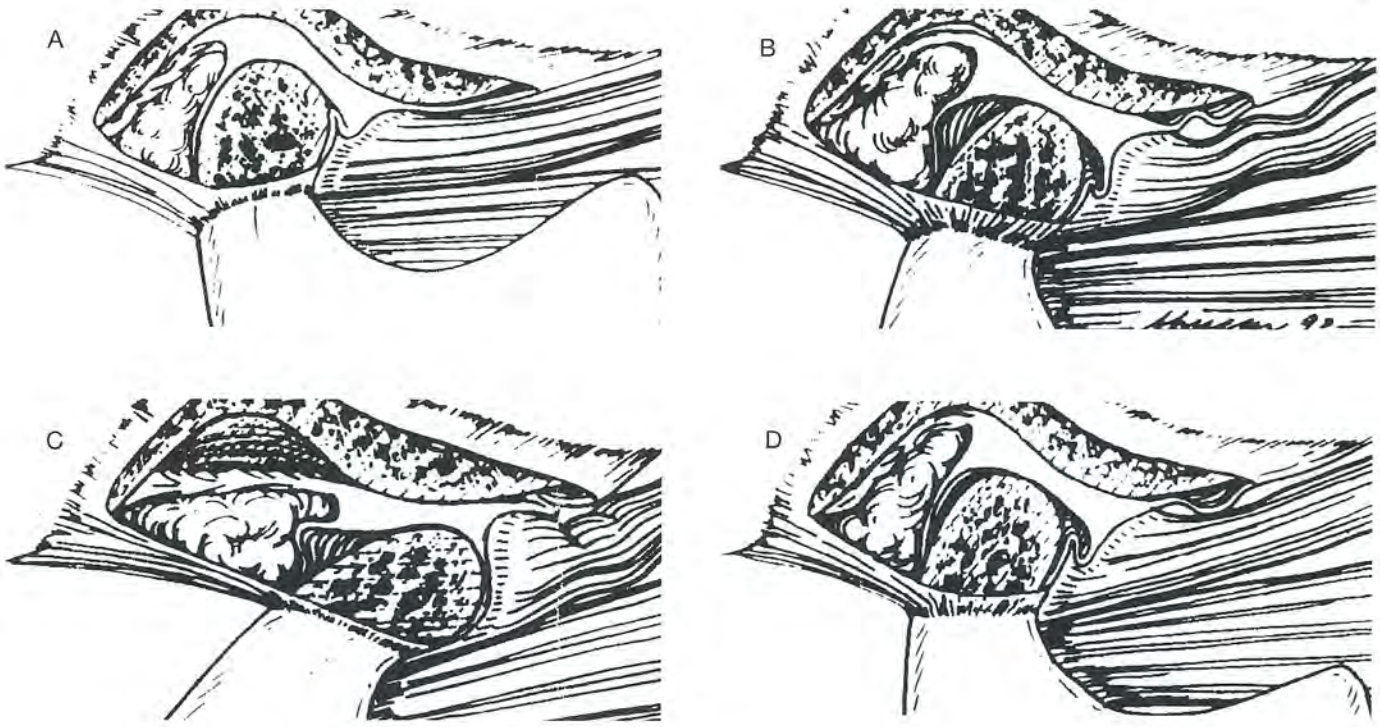

Figure 8-13: Normal disc movement during mandibular opening and closing. A) Position of disc with back teeth together; B) After 10 mm of mouth opening, condylar rotation and anterior translation occur as the disc rotates posterior in relationship to the condyle; C) At full opening, the disc is rotated posterior in relationship to the condyle and translated anterior in relationship to the temporal bone; D) During closing, condylar rotation and posterior translation occur in relationship to the temporal bone as the disc rotates anterior in relationship to the condyle (adapted from Okeson).[133]

Disc Movement with Mandibular Closing

During closing, both the disc and condyle translate posteriorly in relationship to the temporal bone. However, the disc rotates anteriorly on the condyle (Fig 8-13). Anterior rotation of the disc occurs because of the "self-seating" disc to condyle relationship and from tension developing in the superior head of the lateral pterygoid muscle.[102,118,137]

Electromyographic (EMG) studies have demonstrated that the superior head of the lateral pterygoid muscle is active on mouth closure.[102,107] The superior head of the lateral pterygoid "pulls" or rotates the disc in an anteromedial direction in relation to the condyle.The disc's behavior during opening and closing allows its thin avascular and aneural intermediate portion to stay between the condyle and temporal bone. MRI, CT, arthrotomy and arthroscopy inspections confirm this behavior.[174]

Comments on Arthrokinematic Movements

Fibrous adhesions of the capsule, TMJ ligament or disc can limit both active and passive accessory movements, including translation, distraction and lateral glide. A restriction in translation appears to be the most significant factor limiting functional mandibular dynamics. Three classic osteokinematic restrictions are observed when translation is limited (Fig 8-14). Condylar rotation and spin are rarely limited with arthrogeneous conditions except with the uncommon condition of bony ankylosis.

Even with restricted condylar translation, mandibular opening of 20 to 25 mm can be achieved.[133] Patients with less than 20 mm opening may have muscle pain or joint inflammation limiting their opening. Knowing this helps direct treatment toward decreasing pain and inflammation. Conversely, patients with more than 20 mm but less than functional opening may require passive, dynamic or static stretching techniques and exercises.

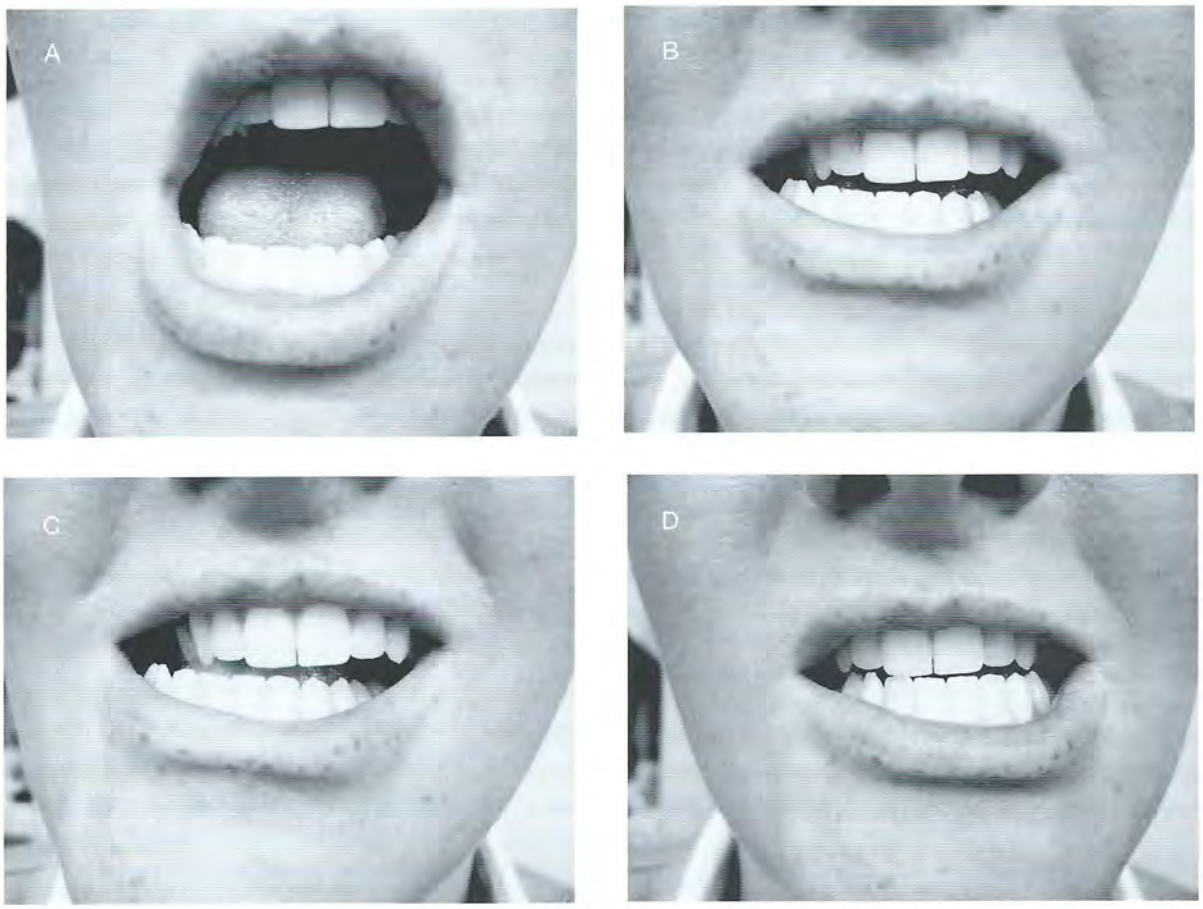

Figure 8-14: Osteokinematic movements observed when limited right TMJ translation is present secondary to fibrous adhesions or Stage II disc displacement: A) Limited opening with right deflection; B) Limited protrusion with right deflection; C) Normal right lateral excursion; D) Limited left lateral excursion.

Section Two—Evaluation and Classification of TMD

The evaluation is intended to identify the *source* of the patient's pain (in other words, which anatomic structure is producing the pain). The evaluation is not intended to identify the etiology.[66] Green states, "...what we have at the individual TMD patient level is nearly always an idiopathic situation—we simply cannot know enough, or cannot measure enough, or cannot precisely determine why each patient has TMD. Even in the absence of a perfect understanding of etiology, we still can provide good conservative care, and we should avoid aggressive and irreversible treatments, especially when they are based on flawed concepts of etiology."[66]

The physical therapy evaluation consists of a history and physical examination. The history and physical examination will uncover most TMD disorders.[29] Adjunctive tests such as imaging studies usually are not needed.[65] Adjunctive tests become necessary if the evaluation findings are questionable or vague, if a fracture or disease is suspected, or if invasive treatment options are being considered.[29]

Following the evaluation, the clinician should be able to determine whether or not TMD is present and should be able to classify it. It is unwise to assume that every patient

with TMD will fit neatly into a specific subclassification with a predictable outcome to treatment. Management of TMD can include a myriad of treatments, including anti-inflammatory, analgesic, and antidepressant medications; occlusal appliances, occlusal equilibration and reconstruction; biobehavioral treatments such as biofeedback, hypnosis, cognitive-behavioral therapy and education; arthrocentesis, arthroscopy and arthrotomy surgery; and physical therapy.[50] No evidence-based rationale for selecting among TMD treatments has emerged. Rather, experts agree that treatments for TMD should be reversible and noninvasive.[119,129,132]

History

The history helps the clinician determine whether the TMJ(s) and/or muscles of mastication are the source of the patient's head, face and jaw symptoms. Greene states, "Now you can call me old-fashioned, but I happen to believe that people who are supposed to have jaw problems ought to have pain and dysfunction clearly associated with mandibular function."[65] Though colloquial, Dr. Green's statement appropriately warns clinicians to avoid overdiagnosing TMD.

The main symptoms of TMD are:[39,50,63,109,119,128,129,132]

1. Pain or discomfort in the TMJ and masticatory muscles that is influenced by jaw movement
2. Joint noises during jaw movement
3. Limitation or difficulty in jaw movement

Additional information to obtain during the history includes:

- Whether the onset of symptoms was related to a specific event or was insidious
- Intensity, frequency and duration of symptoms
- Change in symptom behavior over time
- Functional limitations
- Past and/or current treatment results
- Pertinent medical history (medication, surgeries, allergies, etc...)
- Patient's understanding of the condition and what the symptoms mean
- Patient's expectations and goals of treatment

Physical Examination

The physical examination is performed to reproduce or change the patient's symptoms through active movements and provocative tests, and to establish a baseline from which to assess a treatment's effectiveness. To achieve these two objectives, the examination must have an acceptable level of reliability. *Reliability* is the consistency of a measurement or observation.[145] The clinician's training and experience affects the reliability of measurements and observations.

To identify tissue involvement, tests should be valid. *Validity* means that a test measures what it is supposed to measure.[145] Clinicians need to be responsible with respect to the objectivity of examination methods used, and their appropriate interpretation. Tests or measurements that do not have a high degree of reliability or validity are not necessarily useless. However, acknowledgement of their poor or unknown reliability and validity will keep the clinician honest. Clinicians should avoid using the results of such tests to exaggerate findings to the patient, other health professionals and insurance companies.

The following tests have above average reliability and validity: [39,49,63,65,93]

- Measurement of active jaw movements
- Pain response to muscle palpation
- Pain response to joint palpation
- Assessment of joint sounds

The following tests can be used, but are not as reliable or valid as those listed above. Of the following tests, only compression and force biting are discussed in this text.[39]

- Intraoral evaluation of joint play
- Passive end-feel assessment
- Static resistance tests
- Compression tests
- Force biting tests

Adjunctive Tests

Imaging Studies

The most common TMJ imaging studies are plain radiography, tomography, panoramic radiography, computed tomography (CT), arthrography, and magnetic resonance imaging (MRI). Bone can be visualized with all the techniques, but visualizing soft tissue structures such as the disc requires arthrography or MRI.[49,138] MRI and arthrography tend to overdiagnose TMD disc displacements.[180]

Plain films or conventional radiographs are readily produced in most dental or medical offices. Projections include transcranial, transpharyngeal and transorbital.[138] Each provides limited information of the bony anatomy and no information on soft tissue conditions of the TMJ.[68]

Tomograms are views of a preselected plane of joint anatomy, from .5 to 10 mm thickness. An advantage over

plain films is that clear images of the selected joint anatomy can be seen, and abnormalities not seen on plain films can be detected.[11,68]

Panoramic radiography is a modified tomogram that provides an image of the maxilla, mandible and condyle. The mandibular fossa and articular eminence are not well visualized. Common in dental offices, panoramic radiography provides a convenient, relatively low-radiation method of screening the TMJs for bony and dental abnormalities.[11,68]

Computed tomography produces images of both hard and soft tissues, but since TMJ soft tissue resolution is generally poor, CT is mainly used for bony analysis.[11,68]

Arthrography is an invasive procedure that involves the injection of radiopaque contrast medium usually into the lower joint space.[173] Arthrography can be combined with any imaging technique. It is frequently used with lateral transcranial or lateral tomographic views to indirectly evaluate the disc's integrity and position. Contrast medium that flows from the injected lower joint space to the uninjected upper joint space indicates a perforation, usually in the posterior attachment. Arthrography combined with fluoroscopy is useful for dynamic analysis of the disc-condyle complex.[173] Though once considered the gold standard, the arthrographic procedure may distort the joint so that an actual disc displacement looks normal.

Magnetic resonance imaging (MRI) is inferior to CT for bone studies but superior for evaluating TMJ soft tissues, including disc position. MRI is generally accepted as the current gold standard for evaluating TMJ disc position. The disadvantage of MRI is that it is not dynamic.[20] However, echo planar imaging (EPI) is a recent ultrafast MRI technique that can scan a single frame in less than a second.[23] Though EPI is dynamic, the patient must be able to perform slow mandibular movements and some technological issues can lead to unpredictable results.[23]

Electronic Devices

Electronic devices used to evaluate the TMJ include surface electromyography, neuromuscular stimulation, jaw tracking, and sonography. However, results from electronic devices have low specificity, resulting in false positive diagnoses. Evidence does not support the routine use of electronic devices to diagnose TMD or to monitor the patient's response to treatment.[65,185,186]

Surface electromyography is thought to distinguish between normal and abnormal masticatory muscle activity during rest and function. However, it does not discriminate patients from non-patients and should not be used to justify treatment.

Table 8-1: The American Academy of Orofacial Pain's classification system for TMD with ICD-9 CM codes.

Articular Disorders
Congenital or Developmental Disorders
Aplasia (754.0)
Hypoplasia (526.89)
Hyperplasia (526.89)
Neoplasia [benign] (213.1)
Neoplasia [malignant] (170.1)
Disc Derangement Disorders (524.63)
Disc Displacement With Reduction
Disc Displacement Without Reduction
Acute Disc Displacement Without Reduction
Chronic Disc Displacement Without Reduction
TMJ Dislocation [Open] (830.1)
Inflammatory Disorders
Synovitis and Capsulitis (727.09)
Polyarthritides (714.9)
Osteoarthritis (Non-Inflammatory Disorders) (715.18)
Osteoarthritis Primary
Osteoarthritis Secondary
Ankylosis (524.61)
Fibrous Ankylosis
Bony Ankylosis
Fracture (802.21)
Masticatory Muscle Disorders
Myofascial Pain (729.1)
Myositis (728.81)
Myospasm (728.85)
Local Myalgia Unclassified
Myofibrotic Contracture (728.9)
Neoplasia (171.0)

Neuromuscular stimulation is applied to the masseter muscle and may relax the muscles. Proponents of neuromuscular stimulation use it along with jaw tracking analysis.

Jaw tracking devices (mandibular kinesiographs) are used to evaluate jaw mobility or position. A magnet is attached to the anterior mandibular teeth and the patient performs various mandibular movements. The jaw tracking device is said to assess mandibular range of motion, speed and regularity of movement, chewing movements, and other parameters.

Sonography devices are used to enhance the TMJ sounds and monitor the timing of the sounds during mandibular movement. The intent is to record joint sounds from one TMJ. However, the microphone or transducer also records arterial blood flow, ambient room noise, and skin/hair noise.

Physical Therapy Classification of TMD

TMD is a term that was adopted at *The President's Conference on the Diagnosis and Management of Temporomandibular Disorders* in 1982.[9] The American Academy of Orofacial Pain (AAOP), formerly the American Academy of Craniomandibular Disorders, formed a committee of experts to develop a classification system for TMDs.

The AAOP classification system was influenced by and follows closely the *International Headache Society's Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain*.[67] The International Headache Society lists *Disorders of the Temporomandibular Joint* as one of eight subcategories of the 11 major classifications of pain titled, *Headache or facial pain associated with disorders of the cranium, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures*.[67] The 1982 TMD classification system was expanded by AAOP's 1993 TMD guidelines, which were updated in 1996[132] and 2004[144] (Table 8-1).

Temporomandibular disorders are classified as TMD arthrogenous disorders (TMD-A), TMD myogeneous disorders (TMD-M), or both.[132] The arthrogenous and myogeneous groupings are subclassified into additional categories. In addition to the AAOP guidelines on TMD, there are eight other published TMD guidelines.[49] However, none of them accurately and comprehensively address physical therapy management issues. Identifying the TMD diagnoses that can be helped by physical therapy will facilitate the appropriate utilization of physical therapists by the dental and medical professions.

The physical therapy TMD classification I propose is similar to the AAOP classification, but specifically identifies TMD conditions that a physical therapist treats (Table 8-2). Modification of this classification system will surely occur over time as research enables better understanding of the physical therapist's role in head and neck pain management.

Table 8-2: Physical Therapy Classification for TMD with ICD-9 CM codes.

Arthrogenous
Inflammation (727.09)
Hypermobility (830.1)
Hypomobility
Disc Displacement without Reduction (524.63)
Fibrous Adhesions (524.61)
Myogenous
Masticatory Muscle Pain (728.5)

Section Three—
Evaluation and Treatment of Myogeneous Involvement (TMD-M)

Muscles of mastication are a primary source of pain related to TMD. Single muscles or groups of muscles may be involved. According to the AAOP guidelines, muscle pain falls under the broad category of masticatory muscle disorders.

The AAOP guidelines list six subclassifications for TMD masticatory muscle disorders (Table 8-1). Establishing reliable and valid criteria to honestly identify each of the muscle conditions listed is difficult.[51,158] Simply understanding the physiology and neurophysiology of muscle pain and its clinical presentation is a challenge.[166]

An increase in muscle activity is postulated to lead to increased muscle tone or spasm and subsequent muscle pain.[89,172] However, there is no experimental proof that increased tone leads to pain.[93,158] Patients with masticatory muscle pain do not always have masticatory muscle hyperactivity as identified by an increase in EMG activity.[22,158,163] Likewise, patients who brux regularly (clench, tap and/or grind the teeth while either asleep or awake) do not always have masticatory muscle pain.[22,40,93,163] Masticatory muscle pain does not always cause a limitation in mandibular dynamics. One study found no differences in mandibular mobility between people who were functionally healthy and those with muscle pain.[167] Clinically, patients with 40 mm of opening may complain of restricted opening, while some patients with less than 40 mm do not have any complaints.

These examples are included to show that there is not necessarily a correlation between mandibular dynamics and masticulatory muscle involvement. However, pain in masticatory muscles is clearly a common finding with TMD.[158,164] The physical therapy diagnosis for pain originating from the muscles of mastication is *masticatory muscle pain (MMP)*. In addition to being a primary problem, MMP may play a role as a predisposing, precipitating and perpetuating factor for TMD arthrogeneous conditions.[127,153]

History

The patient complains of facial or jaw pain and may or may not be aware of bruxism.[29] Patients should be asked if they make contact with their teeth since some patients may not know what bruxism means. The patient may wake at night or in the morning with pain, soreness or tension in the TMJ or jaw muscles.

Physical Examination

Palpation of the muscles of mastication is the primary method of identifying MMP. Palpation is performed to reproduce or increase the patient's symptoms. Depending upon severity or frequency of symptoms, a symptomatic response may not occur at the time of the examination, but the clinician may be able to identify increased tone or tension in the muscles being palpated.

Muscles that elevate the mandible are often the symptomatic muscles and will be the primary muscles to palpate. Of the three muscles that elevate the mandible, the temporalis and masseter muscles can be palpated directly, thereby providing a high level of reliability and validity. Both the temporalis and masseter are palpated extraorally (Fig 8-15).[49] Other sites can be palpated, but with less reliability and validity (Table 8-3).

Table 8-3: Palpation technique for muscles related to the TMJ.[49]

Temporalis	Palpate anterior, middle and posterior fibers located in the temporal fossa
Masseter	Palpate from the zygomatic process to the angle of the mandible
Posterior Digastric	Palpate extraorally, posterior to the angle of the mandible
Anterior Digastric	Palpate extraorally, inferior to the body of the mandible
Medial Pterygoid	Palpate intraorally, along the medial rim of the mandible
Lateral Pterygoid	Palpate intraorally, placing the finger posterior to the third maxillary molar, and palpating in a superior, posterior and medial direction
Temporalis Tendon	With the mouth wide open, palpate intraorally following the ramus of the mandible in a posterior direction until the tip of the coronoid is felt

Treatment for Masticatory Muscle Pain

The challenge is to control the habits that perpetuate MMP. Jaw habits include gum chewing, object, nail, lip or cheek biting, protrusive jaw positioning and diurnal or nocturnal bruxism.[121] Diurnal oral habits can be controlled once the patient is made aware of them. Bruxism, especially noc-

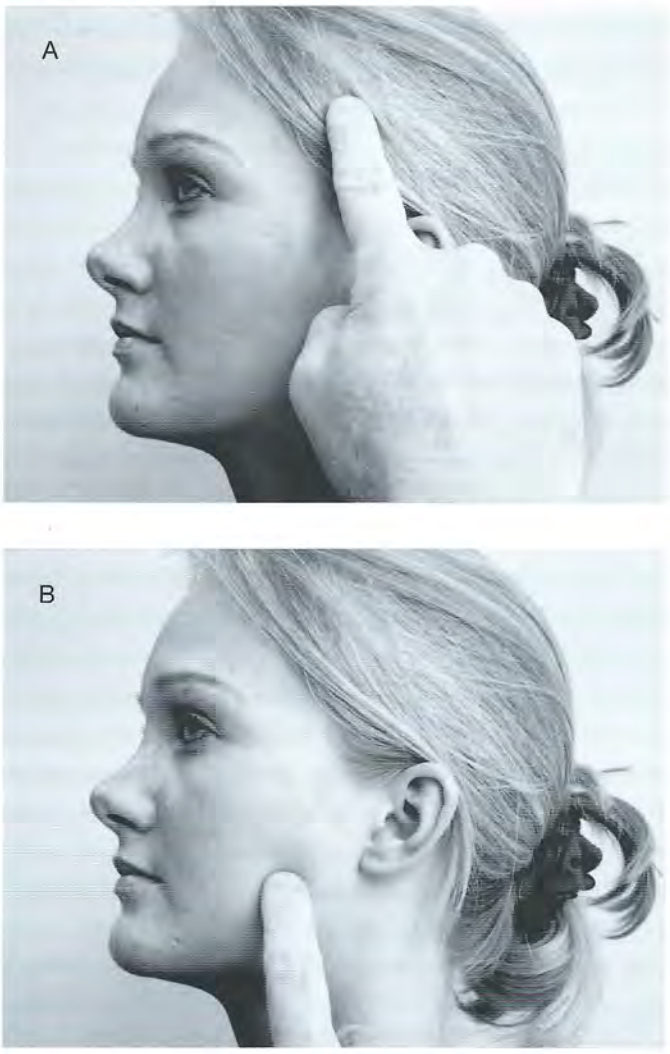

Figure 8-15: Palpation of A) the anterior fibers of the temporalis; and B) the masseter muscle.

turnal bruxism, is one of the more difficult oral habits to manage. Bruxism is more prevalent in TMD patients than in the general population.[89] The etiology of bruxism is multifactorial.[93] The relationship between bruxism and TMD is still unclear.[93,97,166]

Oral Modification and Awareness Training

Clinicians should not assume patients will know that certain oral activities are harmful. Patients should be encouraged to stop biting fingernails, pencils and lips, leaning on their chins, chewing ice and gum, and biting on a smoker's pipe.

Tongue Up—Teeth Apart—Breathe. Teaching the patient the "tongue up, teeth apart and breathe (TTB)" awareness exercise will provide a means of controlling diurnal bruxism that may occur in response to stressful events.

- *Tongue up*—The tongue is composed of various intrinsic and extrinsic muscles. The genioglossus is the main muscle responsible for maintaining the tongue against the palate.[96] The muscles that elevate the mandible (temporalis, masseter, medial pterygoid) have the least amount of activity when the tongue is against the palpate (jaw-tongue reflex).

  The rest position of the tongue, also referred to as the postural position of the tongue, is described in the following way: The anterosuperior tip of the tongue lies just behind the upper central incisors but does not press against them. The rest of the tongue, at least the first half, touches the palate. The patient must be told to not "push" or "poke" the tongue against the palate. It helps to visualize the tongue as floating in this position (Fig 8-16).

  Tongue up may be difficult to achieve in patients who have upper airway disturbances such as colds, allergies, or nasal septum deviations.
- *Teeth Apart* – Teeth apart should naturally follow once the patient's tongue is in the correct rest position.
- *Breathe* – Patients should be taught to breathe through the nose, using the diaphragm. Breathing through the nose makes better use of the diaphragm, the principle driver of respiration. Diaphragmatic breathing promotes general relaxation of the body. Mouth breathing decreases diaphragmatic breathing, increases use of accessory muscles (the scalenes and sternocleidomastoid), and should generally be avoided.

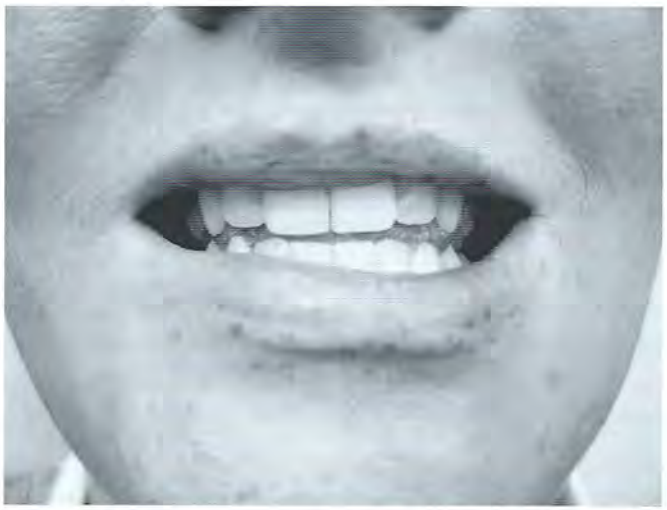

Figure 8-16: The resting position of the tongue for awareness training to control diurnal bruxism.

Tongue Up and Wiggle. Some patients with MMP may not brux during their waking hours. Instead, they brace their mandible with the teeth apart. Patients who are suspected of "bracing" their mandible with their teeth apart should be taught the *tongue up and wiggle* exercise. With the tongue in its rest position, the patient is instructed to oscillate or wiggle the jaw from side to side. The amplitude of side to side movement is kept low—the patient should exert minimal effort. The exercise should be repeated frequently throughout the day. The more the patient wiggles, the less likely he will be bracing. If joint noises occur, the amplitude of the wiggle should be reduced.

Modalities

Modalities are used to decrease pain and to improve healing.[129] Modalities frequently used to treat MMP include:

- Hot or cold packs
- Ultrasound
- Iontophoresis
- Electric Stimulation

Electrical stimulation over the bilateral masseter muscles works particularly well for decreasing muscle pain and increasing mouth opening. The current can be constant but tends to be better tolerated if intermittent (e.g., 12 seconds on and 12 seconds off). The intensity should be high enough to get a contraction of the masseter muscles yet comfortable for the patient. The patient can alternate between actively and passively opening the mouth three or four times when the current is on. The patient should rest with the tongue up when the current is off (Fig 8-17). Active and passive mouth opening during electrical stimulation (stretching with contraction) often achieves decreased pain and increased mouth opening.

Massage to the Muscles of Mastication

Extra-oral or intra-oral massage to tight and painful muscles may provide short or long-term relief of MMP. Detailing specific techniques is beyond the scope of this text.

Therapeutic Exercises

Therapeutic exercises are done to relax or strengthen the muscles of mastication. Relaxing the masticatory muscles is clinically more important than strengthening them.

Muscles that elevate the mandible often need relaxation exercises. Relaxing the elevator muscles can involve contracting the muscles directly (contract-relax) or contracting the antagonist muscle (depressor muscles of the

Figure 8-17: Electric stimulation on surge mode for MMP and/or limited opening due to MMP or Stage II disc displacement. A) Current off; B) Patient opens mouth 3-4 times with current on; C) Patient passively stretches mouth 3-4 times with current on.

mandible). A strong contraction is not recommended. Contract-relax technique provides the patient with feedback. The goal is to have the patient identify more with the relaxation portion of the exercise.

Strengthening the muscles of mastication is rarely needed. Furthermore, it is difficult to identify a truly weak masticatory muscle because pain or reflex inhibition may cause a pseudo-weakness, and the masticatory muscles are difficult to isolate. Jaw muscle weakness tends to be the exception rather than the rule. After immobilization following facial trauma, orthognathic surgery or surgery to repair fractures, a return to normal diet is usually adequate to restore strength.

In the rare case where the mandibular elevator muscles need to be strengthened, the patient can close the mouth against resistance by placing the index finger over the lower central incisors and contracting the elevator muscles. A 5-10 second contraction, repeated 10 times, 3-5 sessions per day is usually satisfactory to normalize strength.

Oral Appliance

An oral appliance is a very common treatment for TMD offered by dentists.[31,159] Oral appliances are removable devices usually fabricated in hard acrylic plastic resin, but occasionally made with a soft resilient material. The oral appliance fits on either the upper or lower arch of teeth. There is a wide variety of opinions about the mechanism of effectiveness and the optimum design of the appliance. Five major theories covering an oral appliance's mechanism of action have been described.[31,32]

A detailed discussion of each theory is beyond the scope of this text; they are listed here to illustrate the diversity of opinion about the use of oral appliances:

1. Occlusal disengagement theory
2. Restored vertical dimension theory
3. Maxillomandibular realignment theory
4. TMJ repositioning theory
5. Cognitive awareness theory

Clinicians treating TMD will hear various names for oral appliances, such as stabilization splint, flat plane appliance, Tanner mandibular appliance, Gelb splint, centric related splint, occlusal splint, night guard, and bite guard.[31,159] Some appliances are given names based upon special features of the appliances such as the modified hawley or anterior bite splint,[181] Nociceptive Trigeminal Inhibition (NTI),[16] repositioning splint,[31] pivot appliance,[95] hydrostatic appliance,[91] mandibular orthopaedic repositioning appliance (MORA),[60] and myo-monitor appliance.[80] Physical therapists need not be concerned about the name of the oral appliance, but should be aware of its design, purpose and goals from the dentist's point of view. At a minimum, oral appliances must be comfortable, aesthetic, retentive, functional, and, most importantly, reversible and non-invasive to the occlusion, muscles of mastication and cervical spine.[31,159] A stabilization splint has these features and has the most evidence of effectiveness in the literature.[159] Clinicians treating TMD should become familiar with oral appliance design and theory, and should refer to the reference list for more resources.[104,159]

Why oral appliances work is still unknown.[132,159] All oral appliances have the potential for a positive effect via increased cognitive awareness and/or positive patient expectations.[31,132,159] Though oral appliances are sometimes effective in controlling MMP, symptoms sometimes worsen or do not respond with their use.[130] In such situations, the dentist should investigate whether the patient has cervical involvement that can contribute to MMP and intolerance to the appliance.

Cervical Spine Considerations

The cervical spine is often overlooked in the management of head and jaw pains. The vast majority of patients experiencing neck pain can be classified as having nonspecific neck pain, also termed mechanical neck pain.[87,144] In the rest of this chapter, the term *neck pain* will be used when referring to patients with cervical spine involvement.

Neck pain can originate from various tissues associated with the cervical spine, and has an influence on TMD management for the following reasons:

Neck pain can mimic TMD pain. Pain originating in the cervical spine can be perceived in the head, face, and jaw areas via an area in the brain termed the *trigeminocervical nucleus.*[15] Bogduk describes the trigeminocervical nucleus as the area in the brain where the trigeminal and cervical afferents converge. He states that the trigeminocervical nucleus is the nociceptive nucleus for the entire head and upper neck. Nociceptive information from cervical spine tissues can be transmitted to the trigeminocervical nucleus, giving the patient the perception of symptoms in the head, face and jaw areas.[15,82]

Neck pain often coexists with TMD. The coexistence of neck symptoms with TMD is more prevalent than one might expect and must be recognized if patients with head and neck pain are to be treated successfully.[1,18,25,27,37,38,43,76,84,94,135,178]

Clinical research supports the following statements:

- Neck pain is associated with TMD 70% of the time.[135]
- Bruxism is more common in patients with pain in both the masticatory and cervical spine musculature.[76]
- Patients with TMD complain about neck pain more frequently than patients without TMD.[40]
- Patients with neck pain report more signs and symptoms of TMD than healthy controls.[40]
- Neck pain is more prevalent with myogenous than arthrogenous TMD.[94,43]

Neck pain can cause MMP. It is clear that neck symptoms and TMD coexist. This suggests that treatment will be required for both the neck and the masticatory muscles. The cause of MMP is multifactorial and is not well understood. It is possible that neck pain may be a cause of MMP. Therefore, effective treatment for MMP may, in fact, focus on treatment of the neck. Investigating a cause and effect relationship is one of the most difficult challenges in clinical research, and no one can say for certain the exact relationship between neck problems and MMP. However, two theories are proposed that shed light on how neck problems may precipitate, perpetuate, or predispose one to MMP.

The first theory is that cervical spine neurophysiological influences cause masticatory muscle pain via the tonic neck reflex and/or by the agonist/antagonist relationship of the anterior cervical muscles (including the muscles of mastication) to the posterior cervical muscles.[148]

The second theory postulates that MMP actually occurs in response to neck pain. For example, patients may respond to neck pain by bruxing, which may lead to MMP. A detailed explanation of these theories is beyond the scope of this text, but clinicians are encouraged to seek more information and certainly consider the cervical spine when patients present with head and jaw pain.

Neck Pain and Oral Appliances

Neck pain and an oral appliance both have an influence on the resting position of the mandible and the subsequent trajectory of jaw closure. If the neck and oral appliance affect the resting position of the mandible in different ways, an increase in MMP and a decrease in tolerance to the oral appliance may result.

The rest position of the mandible is defined by Atwood as "the habitual postural position of the mandible when the patient is at ease in an upright position."[7] Mohl states, "we must logically conclude that, if rest position is altered by a change in head position, the habitual path of closure of the mandible must also be altered by such a change."[116] Studies of normal subjects have demonstrated that the resting position of the mandible and the trajectory of mandibular closure into occlusion is affected by the relationship of the head to the cervical spine and gravity.[19,52,62,98,105]

Head and neck mobility and position affect the tension and tone of connective tissues and muscles that traverse from the cervical spine and cranium to the mandible (Fig 8-18). Neck pain with associated altered mobility and positioning of the head and neck is believed to influence mandibular position and the trajectory of jaw closure into centric occlusion or into the oral appliance.

All oral appliances influence the rest position of the mandible. Therefore all oral appliances have an influence on the trajectory of mandibular closure into the oral appliance.[90] Depending on the design of the oral appliance, some can have a significant effect on jaw dynamics. The thickness of an oral appliance may influence head and neck posture. The head may extend on the cervical spine to compensate to the increase in vertical dimension of a thick appliance.[35,143] An oral appliance should be kept as thin as the possible unless an increase in vertical dimension can be justified.

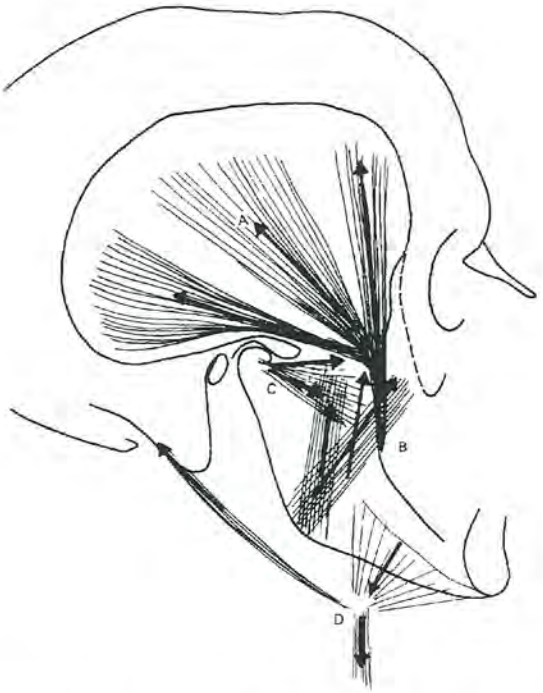

Figure 8-18: The connective tissues and muscles that traverse from the cervical spine and cranium to the mandible. Connective tissue tension and muscle tone can be affected by neck pain, thereby influencing mandibular rest position and the trajectory of jaw closure.

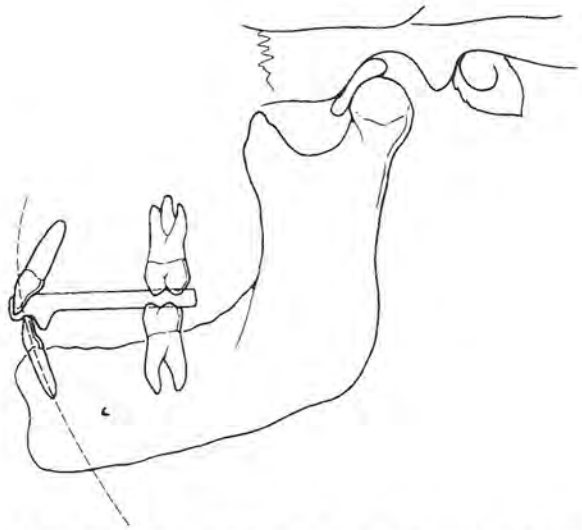

Figure 8-19: An ARA may be used by some dentists to treat a disk displacement with reduction. It positions the mandible forward and down to prevent the disc from displacing anteriorly on mandibular closing.

An example of an oral appliance that influences jaw and neck equilibrium is the anterior repositioning appliance (ARA). An ARA may be used by some dentists to treat disc displacements. The ARA is thicker than other oral appliances, and repositions the mandible in a forward and down position, significantly changing the rest position of the mandible and the trajectory of jaw closure (Fig 8-19).

The patient with neck pain who is wearing an ARA may not respond to its use or may have an increase in neck pain. This results because both the ARA and the cervical spine are competing over influencing mandibular rest position and the trajectory of jaw closure. Other oral appliance designs have more or less similar influences on mandibular rest position, the trajectory of jaw closure, and head and neck position.

In summary, the cervical spine should be considered when the patient's MMP does not improve or worsens with the use of an oral appliance. If the patient perceives that the "bite" is off, but the dentist cannot identify any occlusal factors to account for the perception, the cervical spine should be suspected as influencing the trajectory of jaw closure. If the patient is treated initially with an ARA and does not achieve a favorable response, referral to a physical therapist for evaluation of the cervical spine is appropriate, rather than more prolonged treatment with an ARA or referral to an oral surgeon.

Not all patients with neck pain respond poorly to an oral appliance. Variables to consider are the structural design of the appliance, the technique used by the dentist to balance/adjust the appliance, the degree of acute or chronic neck pain, and the adaptive response of the individual.[144] Reliable criteria do not exist to identify those patients who may adversely respond to an oral appliance secondary to cervical spine influences. Therefore, treatment of neck pain should be done before or at the time of delivery of an oral appliance. The stabilization splint, with proper features, currently represents the best design for oral appliance treatment of TMD with or without neck pain.

Screening for NNP by the Dentist

Dentists should screen for neck problems during the history. Symptoms suggesting neck involvement are grouped by most commonly to least commonly seen (Table 8-4). Although the list in Table 8-4 was compiled from a reference on whiplash injury, it is also useful for neck pain that did not result from a whiplash injury. Patients who have any of the symptoms listed should consult with a physical therapist for a comprehensive evaluation of the cervical spine.

Table 8-4: Symptoms associated with non-specific neck pain (NNP).[160]

GROUP ONE – Most Common
Neck and/or shoulder pain
Reduced and/or painful neck movements
Headaches
Numbness, tingling or pain in arm or hand
Dizziness or unsteadiness
Nausea or vomiting
GROUP TWO
Difficulty swallowing
Ringing in the ears
Memory problems
Problems concentrating
Vision problems
Reduced and/or painful jaw movement
GROUP THREE – Least Common
Numbness, tingling, or pain in leg or foot
Lower back pain

Section Four—
Evaluation and Treatment of Arthrogeneous Involvement (TMD-A)

TMJ Inflammation

TMJ inflammation can involve the capsule, TMJ ligament, lateral and medial collateral ligaments, anterior and posterior bands of the disc and posterior attachment. Tests are not sensitive enough to differentiate between these tissues. Even if such tests were available, the results of the tests would not affect treatment planning.

History—TMJ Inflammation

Symptoms are located in the preauricular area with or without referral into the temporal and mandibular areas. Pain is typically reproduced with chewing, talking and/or yawning. Mandibular dynamics may or may not be affected. Patients with significant joint effusion may be unable to bring their back teeth together on the ipsilateral side.

Physical Examination—TMJ Inflammation

The physical examination includes the provocation tests of palpation and TMJ loading. Whether or not the patient's symptoms are affected depends on the degree of inflammation. With the exception of limited mandibular dynamics and rarely seen joint swelling, provocative tests require the patient's verbal response. The clinician should carefully correlate all elements of the evaluation to properly interpret the patient's verbal response, and avoid over or under-diagnosing TMJ inflammation.

TMJ Palpation

Palpation over the lateral pole. Facing the patient, the clinician uses the index or middle fingers to palpate over and then slightly posterior to the lateral pole with the patient's back teeth together (Fig 8-20). Palpation continues as the patient opens the mouth. A positive test is an increase or reproduction of the symptoms on the ipsilateral side. Targeted tissues are the lateral collateral ligament, capsule, and TMJ ligament.

Palpation via the external auditory meatus. Facing the patient, the clinician inserts his little finger (pad of the finger facing towards the condyle) in the patient's external auditory meatus (EAM) and asks the patient to open the mouth (Fig 8-21). The clinician applies slight pressure forward with his finger as the patient then closes the mouth, bringing the back teeth together. A positive test is an increase or reproduction of the symptoms on the side being palpated. The target tissue is the posterior attachment of the disc.

Figure 8-20: Palpation over the lateral pole to provoke the lateral collateral ligament, capsule and TMJ ligament. It is performed with the patient's back teeth together and then with the mouth opened.

Figure 8-21: Palpation via the external auditory meatus to provoke the posterior attachment of the disc. The clinician inserts his finger with the mouth opened, then asks the patient to close the mouth, bringing the back teeth together.

The clinician should not be concerned about joint noises during this test. If excessive pressure is applied by the finger, the disc position may be influenced enough to produce a joint noise, but it is not significant.

TMJ Loading

Dynamic Loading. Biting with the molars against resistance causes joint loading (compression) on the contralateral side with less compression and possible distraction on the ipsilateral side.[70,73,78,140] Dynamic loading is accomplished by asking the patient to bite hard on a cotton roll placed between the molars on one side. A rolled up 4x4 gauze cut in half can also be used (Fig 8-22). A positive test is an increase or reproduction of the symptoms on the contralateral side.

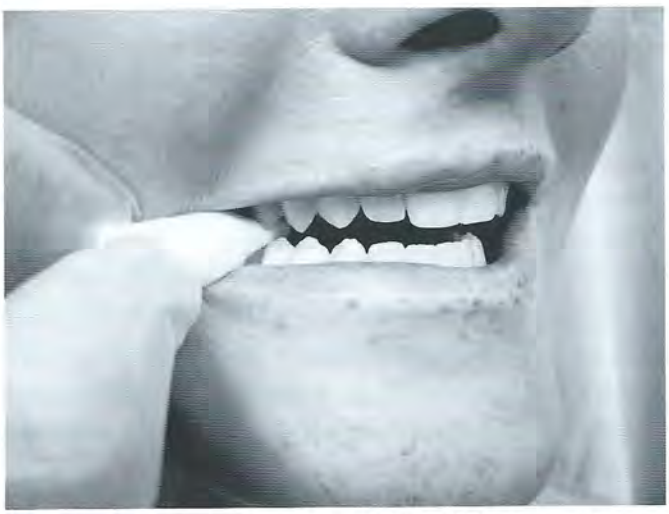

Figure 8-22: Dynamic loading to provoke inflamed intracapsular tissues. The patient bites on a cotton roll placed between the molars to load the contralateral side.

Dynamic loading requires contraction of the elevator muscles of the mandible. Patients can have a false positive response to TMJ loading if MMP is present. Patients can also have both TMJ inflammation and MMP. Asking the patient to point to the location of the pain during dynamic loading can help differentiate. Patients with joint pain will point to the temporomandibular joint; patients with MMP will point to the temporalis, masseter, or both.

Passive Loading. Passive loading of the TMJ involves applying a posterosuperior force to the mandible. The clinician grasps the patient's chin with the index finger and thumb. With the patient's back teeth slightly apart, the clinician applies pressure on the chin in a posterosuperior direction and then in a posterosuperior and slightly lateral direction to the left and right. The opposite hand gives appropriate counterforce on the back of the patient's head (Fig 8-23). This test is not selective for either the right or left TMJ. A positive test is a reproduction or increase of the patient's TMJ symptoms. A false negative response is possible for patients who are unable to relax the jaw.

Treatment for TMJ Inflammation

The treatment goal is to decrease inflammation in and around the TMJ. Modalities frequently used to treat TMJ inflammation include:

- Hot or cold packs
- Ultrasound
- Iontophoresis
- Electric Stimulation

TMJ inflammation should respond within a relatively short period of time to modality treatment. Patients have

Figure 8-23: Passive loading to provoke tissues posterior and posterolateral to the head of the condyle. A) Posterosuperior loading; B) Posterosuperior and lateral loading—repeat to right and left.

varying responses to modalities, and a variety of modalities can be tried.[129,179] If inflammation persists, MMP or a disc displacement may be present.

TMJ Hypermobility

Hypermobility occurs when the condyle translates excessively beyond the articular eminence onto the articular tubercle.[44] Hypermobility has been thought to cause osteoarthritis and disc displacement. However, studies have not confirmed this relationship.[44,45,46] TMJ hypermobility does not impair function and is often a benign condition.

History—TMJ Hypermobility

The patient may state, "My jaw feels like it goes out of place." The patient may also point to the condyle with mouth opening, demonstrating the excessive movement. The appearance of excessive condyle movement may be magnified in patients with narrow faces. Clinicians must avoid placing undue emphasis on the appearance or sensation of excessive condyle movement, because these perceptions do not always correlate with actual pathology.[161]

The patient may report joint noises. Joint noises do not always occur with hypermobility. The noise typically occurs either at the end of opening (caused by the condyle moving abruptly past the articular crest onto the articular tubercle) or at the beginning of closing (caused by the condyle moving abruptly past the articular crest onto the articular eminence). The patient may incorrectly believe that the clicking or popping is related to a disc displacement.

The patient may relate short-term episodes of jaw "catching" with mouth closure. This symptom may point to an intermittent dislocation of the condyle. A predisposing factor for dislocation appears to be an articular eminence with a short, steep slope.[10,133]

In the event a patient presents with a dislocated condyle, the diagnosis is straightforward. The patient is unable to close the mouth from a fully opened position. With a unilateral dislocation, the jaw deflects contralaterally (Fig 8-24). The physical therapist rarely sees these patients, because they typically seek emergency treatment. Once the condyle has been reduced, the patient may be referred to physical therapy for treatment of inflammation and control of mandibular opening.

Physical Examination—TMJ Hypermobility

Facing the patient, the clinician uses the index or middle fingers to palpate over the lateral pole as the patient opens and closes the mouth.

The clinician may be able to detect excessive translation of the condyle or feel a "jutter" at the end of mouth opening and beginning of mouth closing (Fig 8-25). The "jutter" is the condyle moving across the articular crest onto the articular tubercle. No consistent deviation or deflection is observed.

Figure 8-24: Dislocated right condyle. The jaw is deflected to the left and the patient is unable to close the mouth from a fully opened position.

Figure 8-25: Condylar hypermobility is identified by palpating excessive translation of the condyle and/or a "jutter" that occurs at the end of mouth opening and the beginning of mouth closing.

Diagnosing hypermobility via palpation is not reliable or valid. However, since the condition and its treatment are benign, false positives and negatives can be tolerated. The alternative is to request an x-ray, which is not justified.

Treatment for TMJ Hypermobility

Treatment of TMJ hypermobility is important when:

- The patient makes it an issue. Some patients are anxious about the possibility that their jaw will displace or believe that their popping is related to a disc displacement.
- The patient has TMJ inflammation. If hypermobility is not controlled, the joint can be aggravated every time the patient opens wide while eating or yawning.
- The patient has a history of his jaw catching intermittently when closing from an open mouth position that is confirmed on examination.

Treatment focuses on patient education to reassure the patient that there is no severe pathology. Patients should be taught to eat smaller portions of food (avoid large sandwiches) and to place the tongue against the roof of the mouth when yawning to avoid excessive opening (Fig 8-26). Patients should inform their dentists that they have hypermobility so the amount and duration of opening during dental treatment can be minimized.

Treatment for hypermobility very rarely involves surgery. Surgical approaches could include eminectomy, condylotomy, sectioning of the lateral pterygoid muscle, intracapsular injection of sclerosing solutions, and increasing the height of the articular eminence to block anterior movement of the condyle.[56]

Figure 8-26: The tongue up position to control translation of the condyle during yawning.

Hypomobility, Including Disc Displacements

Hypomobility can be a result of TMD-A or TMD-M involvement. Examples of arthrogeneous hypomobility include disc displacement without reduction (Stage II) and articular adhesions discussed later in this chapter.

There is a high prevalence (34%) of disc displacements in asymptomatic children and young adults.[142] Why some disc displacements are painful and others are not, or why some non-painful disc displacements progress to painful disc displacements is not understood.

Disc displacement or internal derangement has been defined as, "a disturbance in the normal anatomic relationship between the disc and condyle that interferes with smooth movement of the joint and causes momentary catching, clicking, popping or locking."[88] The disc is considered to be displaced when the patient has the back teeth together and the posterior band of the disc is anterior or anteromedial to the head of the condyle (Fig 8-27).[10]

A disc displacement is believed to be caused by laxity of the medial or more likely the thin lateral collateral ligament.[10,133,137,156] Ligamentous laxity allows excessive movement between the disc and condyle, with subsequent loss of the self-seating relationship.[183]

Collateral ligament laxity can result from trauma. It can also result from microtrauma in the form of continuous or repetitive loading from daily functional and parafunctional activities such as bruxism and other oral habits. The health of the disc and articulating surfaces is dependent upon the frequency, duration, magnitude, direction and location of the microtrauma and variations in bony and

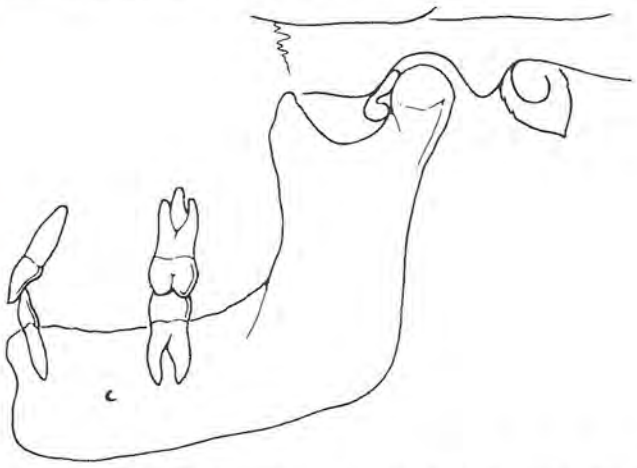

Figure 8-27: Disc displacement. With the back teeth together, the posterior band of the disc is displaced anterior to the condyle.

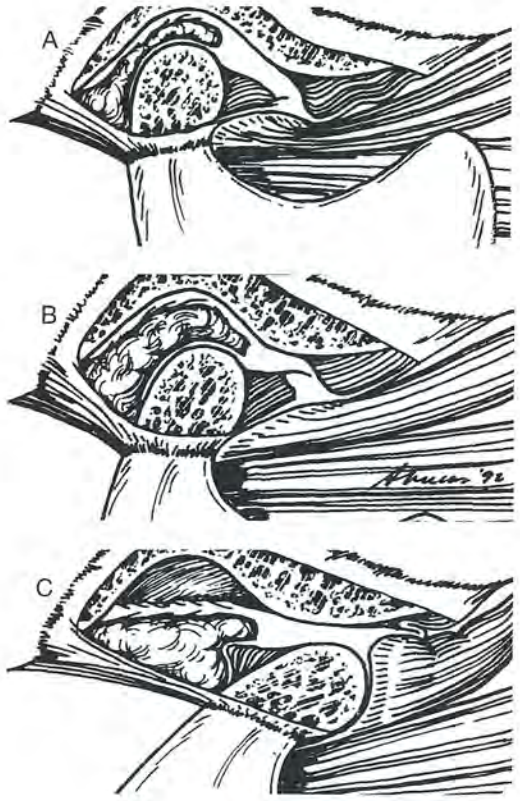

Figure 8-28: Disc displacement with reduction—Stage I. A) With the back teeth together, the disc is displaced anterior to the condyle; B) With opening, translation of the condyle tenses the superior lamina; C) Tension in the superior lamina "snaps" the disc posterior, causing the classic opening "click" (adapted from Okeson).[133]

muscular anatomy.[118] Understanding the etiological events and anatomical changes that contribute to a disc displacement enables the clinician to formulate realistic goals for treatment.

Several classifications of disc displacements have been recorded in the literature.[115,187] I prefer Moffett's classification, which is also used by the AAOP.[115] This classification does not require imaging studies and relies only on the history and physical examination. Moffett's three stages of a disc displacement are:

Stage I—Disc Displacement with Reduction

Stage II—Disc Displacement without Reduction

Stage III—Disc Displacement with Osteoarthritis

A study of the natural history of disc displacements suggests that patients start with a Stage I and progress to a Stage II and III.[42,150] However, many variations from this progression are possible and are not understood.[77] In all stages of disc displacement, the disc is displaced when the patient's back teeth are together.

The history and physical examination help the clinician determine the stage. Except in unusual circumstances (e.g., conservative care has failed and surgery is being considered), MRI, arthrography or other sophisticated assessments are unnecessary to diagnose disc displacement.[169]

Disc Displacement with Reduction—Stage I

When the patient's back teeth are together, the disc is already displaced. As the patient opens the mouth, the condyle rotates on the posterior attachment. Approximately 10 mm into opening, the condyle begins to translate but is initially limited because the disc displacement blocks translation. As translation continues, the condyle pushes against the posterior band of the disc. Additional tension develops in an already stretched superior lamina, which causes the superior lamina to "snap" the disc posterior, causing an opening "click" (Fig 8-28).

A *click* is a distinct sound, of brief and very limited duration, with a clear beginning and end.[49] The disc reduction can occur at various points of mandibular opening, depending upon the amount of tension in the superior lamina.

On closing, the disc displaces anterior to the condyle and is identified by a closing "click". The closing click is softer than the opening click, and occurs at the end of closing. The disc displaces anterior because of laxity in the collateral ligaments, loss of the self-seating relationship of the disc to condyle and by activity of the upper head of the lateral pterygoid muscle. A Stage I is therefore identified by the classic *reciprocal click* (Fig 8-29).[57]

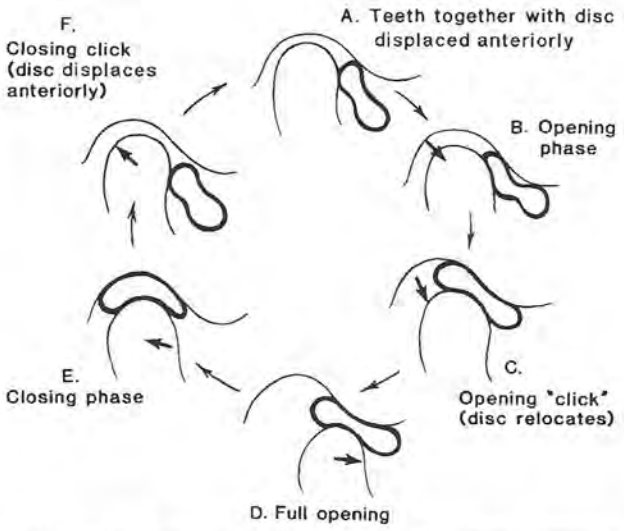

Figure 8-29: Disc displacement with reduction—Stage I is identified by the classic reciprocal click.

Figure 8-30: Elimination of the reciprocal click from a protruded position of the mandible. Starting with the back teeth in contact, the patient first opens wide enough to cause the opening click (A), then closes with the central incisors at an end to end position (B). From this position, the patient open and closes several times to confirm that the reciprocal click is gone.

History—Disc Displacement Stage I

The patient reports hearing an opening and a closing click during mandibular movements. If noises are not heard by the patient, then a Stage I is not present.

Physical Examination—Disc Displacement Stage I

The physical examination consists of two parts: First, the clinician identifies the reciprocal click by palpating over the lateral poles during mandibular opening and closing. The clinician may not actually hear the noises, but should feel a palpable irregularity. Therefore, in the rest of this text, a

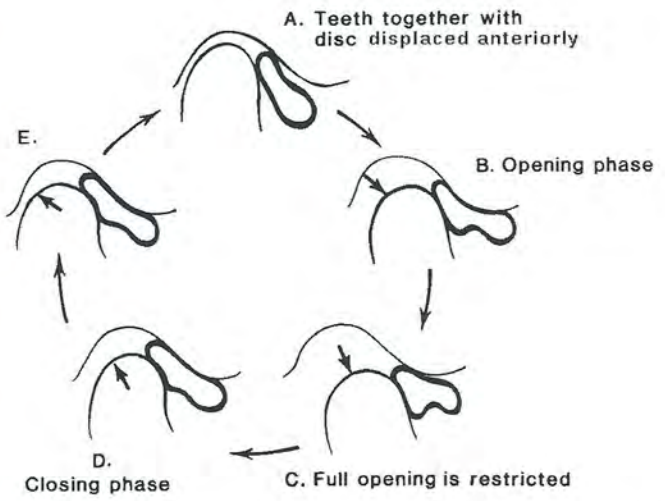

Figure 8-31: Disc displacement without reduction—Stage II. The disc remains displaced through mandibular opening and closing and there is no reciprocal click.

TMJ joint noise is defined as a noise that is heard by the patient and is *always felt* by the clinician. Noises that are heard by the patient but not felt by the clinician may not be significant.

Second, the clinician attempts the elimination of the reciprocal click with the mandible protruded. Starting with the patient's back teeth together, the clinician palpates the patient's lateral poles. The patient is asked to open wide enough to get the opening click and then close forward, bringing the central incisors to an end to end position. Then, the patient is asked to open several times while maintaining this protruded position of the mandible (Fig 8-30). More often than not, the reciprocal click is eliminated because the condyle is not allowed to go back to its original position where the disc displaced anterior to the condyle.[49]

Disc Displacement without Reduction—Stage II

With a Stage II, the disc remains anterior to the condyle throughout the entire phase of opening and closing (Fig 8-31).[169] The disc obstructs condylar translation. The disc is unable to reduce because excessive elongation of the superior lamina has occurred and/or increased activity of the elevator muscles compress the joint space, preventing disc reduction (Fig 8-32).[169]

History—Disc Displacement Stage II

The patient often reports previous joint noises (Stage I), with or without previous episodes of intermittent "locking". There are no current joint noises, but the patient is unable to open the mouth wide and has difficulty performing movements such as chewing and yawning.

Physical Examination—Disc Displacement Stage II

No joint noises are present. Mandibular dynamics are limited (Fig 8-14).

Chronic Disc Displacement without Reduction—Stage III

Stage III involves the perforation of the posterior attachment, resulting in bone-on-bone contact with associated degenerative changes. The posterior disc may eventually become detached from the postglenoid spine.[48] As the disc becomes more deformed and the posterior attachment degenerates, mandibular dynamics are near normal.[54]

History—Disc Displacement Stage III

The patient may report a history of a Stage I and/or Stage II disc displacement. The patient complains of multiple joint noises (crepitus) with mandibular opening and closing. Crepitus often identifies a Stage III even with no prior history of a Stage I or II. Crepitus is distinguished from the short "click". It is a continuous sound over a longer range of jaw movement.[49] Crepitus is the sound of bone grinding against bone, and is highly indicative of Stage III disc displacement. Osteoarthrosis (OA) and degenerative joint disease (DJD) can also accompany disc displacement.[24] These conditions are likely to be related to each other. Stage III disc displacement, OA and DJD will show radiographic evidence of structural bony change.[75,182] OA and DJD are treated using the same methods as treatment for Stage III disc displacement.

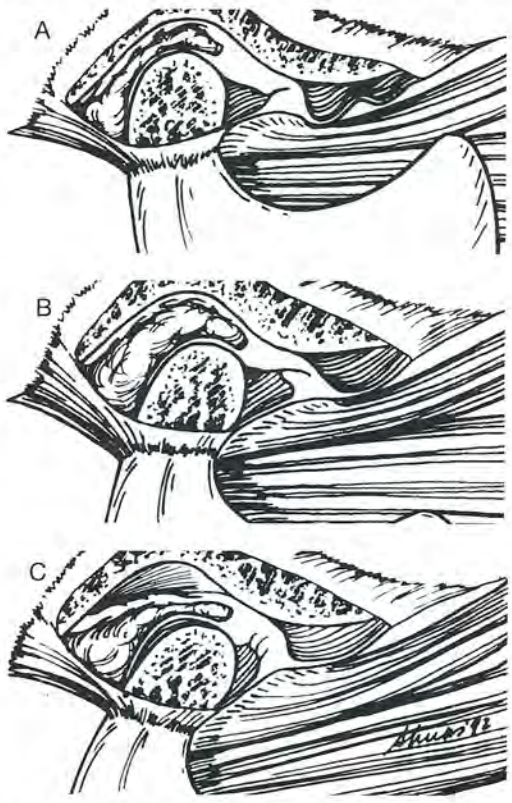

Figure 8-32: Disc displacement without reduction—Stage II. A) With the back teeth together, the disc is displaced anterior to the condyle; B) With opening, translation of the condyle is blocked by the displaced disc; C) The disc acts as a mechanical obstruction causing limited mouth opening (adapted from Okeson).[133]

Physical Examination—Disc Displacement Stage III

Mandibular dynamics are normal or near normal. With palpation over the lateral pole during opening and closing, palpable irregularities of crepitus are identified.

Comments on Joint Noises

Three types of joint noises have been discussed:

- A reciprocal click that may occur with hypermobility
- A reciprocal click that occurs with a Stage I disc displacement
- Crepitus that is present with a Stage III disc displacement

Yet another type of joint noise can occur with *deviation in form* (DIF) (ICD 719.68).[132,162] A DIF involves irregular surfaces on the articulating surfaces of the condyle or temporal bone that form obstacles for rotation of the disc against the condyle or translation of the disc against the articular eminence.[132,162] DIF joint noises occur at the exact same condylar position during mandibular opening, closing or both. Palpation reveals a repetitive, non-variable palpable irregularity that cannot be explained by hypermobility or Stage I disc displacement. Noises that are a result of DIF require no treatment other than patient education.

Treatment for Disc Displacements—All Stages

Treatments for disc displacements have historically involved either the use of oral appliances or invasive procedures such as arthrocentesis, arthroscopy or arthrotomy.[8,72,134,191] However, depending on the treatment goal, physical therapy for disc displacements may result in equally good outcomes.

Oral Appliance

Anterior repositioning appliances (ARAs) are used by some dentists to treat Stage I disk displacements (Fig 8-19). As the name implies, an ARA repositions the mandible forward. Repositioning the mandible forward keeps the disc from displacing. The recapture of the disc is confirmed by the elimination of the reciprocal click. The objective of an ARA is to maintain the proper relationship between condyle and disc until sufficient healing of injured tissues

(lateral collateral ligament and posterior attachments) has occurred. Often this means the ARA must be worn 24 hours a day for three to six months.

Disadvantages of treatment with ARA include a change in the original bite and jaw position (Fig 8-33), which sometimes requires additional treatment with orthodontics, prosthodontics, equilibration or orthognathic surgery.

Furthermore, treatment with an ARA or other appliance does not result in a permanent corrected position of the disc to condyle, except in a very a small percentage of patients.[99,122,188,165] Proper patient selection with thorough patient education is essential before treating Stage I disc displacement with an ARA. When patients are informed of what may have to occur with their occlusion following treatment with an ARA, they may elect other treatment options such as physical therapy.

For Stage II disc displacements, dentists may use a stabilization appliance and/or intraoral techniques, with the goal of achieving the reciprocal click, indicative of a Stage I disc displacement with functional opening. Then, the dentist may elect to treat the Stage I as previously described or leave it alone if the patient is asymptomatic. For a Stage III disc displacement, the dentist may use a stabilization splint to minimize adverse loading secondary to bruxism.

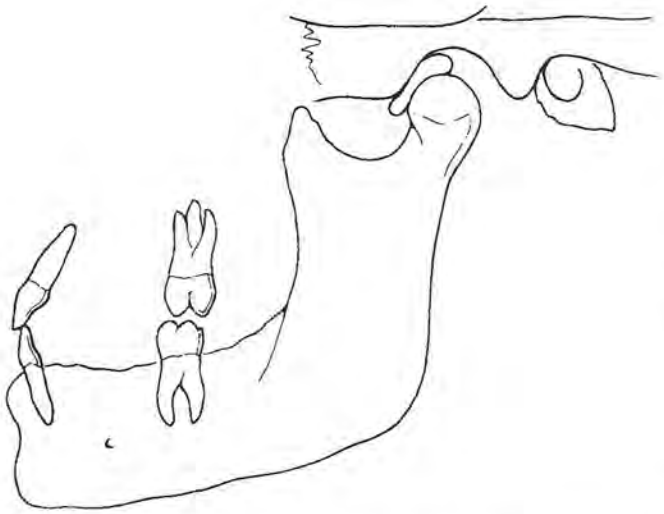

Figure 8-33: Following ARA treatment of a Stage I disc displacement, the patient's mandible is repositioned anteriorly. To bring the molars into contact, a combination of orthodontics, prosthodontics, equilibration and/or orthognathic surgery is often required.

Surgery

Arthrocentesis is lavage and manipulation. This procedure can be done for a Stage II to restore mandibular function or for any stage of disc displacement that involves inflammation.

The joint is anesthetized and the patient is under conscious sedation. In its simplest form, a 1.2 mm needle is used for outflow and is positioned in the anterosuperior joint space.[72] The irrigation is performed with a syringe and a 0.6 mm needle placed in the posterosuperior joint space. One hundred milliliters of isotonic saline solution are slowly injected. The patient is asked to move the jaw during the lavage, with the goal of increasing mouth opening.[72] Though mandibular movement improves, disc displacement to the condyle often remains unchanged.[72] Improvement in mandibular function may have occurred due to elongation of the posterior attachment with further anterior displacement and deformation of the disc.

Arthroscopy is the placement of an arthroscope with camera into the superior joint space. Lavage and lysis of adhesions are performed, and corticosteroid or sodium hyaluronate is injected. This procedure can be done for all stages of disc displacements but is most frequently used for Stage II.[72]

Improvement of mandibular opening and decrease in pain frequently occurs following arthroscopy for a Stage II. However, the improvement is not necessarily a result of restoring the normal disc/condyle relationship.[28,72,124,131,136] Instead, improvement in mandibular function may be due to elongation of the posterior attachment with further anterior displacement and deformation of the disc.

Arthrotomy—Patients who do not respond to conservative care or who have not responded to arthrocentesis and arthroscopy may be appropriate for arthrotomy.

Some authors feel that arthrotomy should be performed only when arthroscopy or arthrocentesis has been tried and has failed, but this point is debated.[6] Arthrotomy can be done for any of the disc displacement conditions. Arthrotomy is also the treatment of choice for ankylosis.

There are many methods of arthrotomy, and none have been shown to have a distinct advantage. Surgical method choices are influenced by the evolving philosophy of the surgeon and long-term results reported in the literature.[8] The results of arthrotomy may be similar to those of arthrocentesis and arthroscopic surgery—disc displacement may still be present despite improved mandibular dynamics and symptoms.[123,147] Physical therapy post-sur-

gery for disc displacements focuses on maintaining the opening achieved from the procedure (see *Physical Therapy Treatment for Fibrous Adhesions* on page 204) and should address any concurrent inflammation or MMP.

Physical Therapy Treatment for Disc Displacements—General Comments

Disc displacements have not been convincingly demonstrated to be entirely pathological.[26,64] Imaging studies have documented the presence of disc displacements in both patient and non-patient populations who have no pain or functional limitations.[55,83] So common are asymptomatic disc displacements that they may be considered a normal anatomic variability.[83,175,184] The long term success of maintaining a "normal" disc to condyle position with oral appliances or surgery is mixed.[6,123]

Given the less than satisfactory long term results with appliance therapy and surgery, it seems unlikely that physical therapy treatment (exercise and manual therapy) can restore disc position. Nonetheless, physical therapists play an important role in the treatment of disc displacements. Physical therapy can facilitate the normal progression of disc displacements. Since the articular tissues of the human TMJ often adapt or remodel in response to disc displacement, the success of physical therapy is not dependent upon the positioning of the disc or the absence of joint noises. Appropriate goals for physical therapy treatment of disc displacements are freedom from pain and improved function. Patients must clearly understand these goals prior to treatment so their expectations are accurate.

Physical Therapy Treatment for a Stage I and a Stage III Disc Displacement

To review, Stage I and Stage III disc displacements present with functional mandibular dynamics and the joint itself may not be painful. Why, then, would a patient with good mandibular function and no TMJ pain seek help? Patients may present with head and neck pain that is originating from other tissues and not from the disc displacement. The patient may mistakenly believe that the clicking or crepitus is the source of the pain. In such cases, the patient's attention should be directed toward the treatment of pain originating from TMJ inflammation, MMP and/or the cervical spine.

TMJ joint pain that accompanies disc displacement is often due to joint inflammation. In fact, the physical therapist should assume that inflammation and disc displacement simply co-exist. Treating the inflammation frequently resolves the pain, even though the Stage I or III disc displacement persists. Sometimes, TMJ inflammation cannot be easily controlled because it is being aggravated by the disc displacement.[47,126] These are the cases that should be referred to a dentist or oral surgeon.

Physical therapy treatment for Stage I and III should focus on patient education about the meaning of joint noises.[47] Patients should be made aware of other treatment options such as oral appliances and surgery and more importantly, have realistic expectations about the outcome of such treatments (i.e., the unlikelihood that "normal" disc position will be achieved long-term). The physical therapist and dentist who are co-treating a patient must be in philosophical agreement and have good communication to avoid sending the patient mixed messages. Joint noises associated with Stage I are often louder and more disconcerting to patients than joint noises associated with Stage III. Patients with a Stage I may experience occasional catching during opening or closing of the mouth. Therefore, one treatment option for a Stage I is to prevent the disc from relocating on opening by stretching out the already stretched posterior attachment. The electrical stimulation stretching technique described below for Stage II disc displacement can be used. The goal is to allow the condyle to translate with the disc permanently displaced on opening and closing. Essentially, the disc behaves as a Stage III but without crepitus. Crepitus may occur in later years.

Physical Therapy Treatment for a Stage II Disc Displacement

Unlike Stages I and III, patients with Stage II disc displacements with or without pain have functional limitations. Physical therapy objectives for a Stage II are to restore pain free functional mandibular dynamics regardless of the disc position. It is often necessary to treat concurrent TMJ inflammation, MMP and neck pain. Several treatment options are helpful:

Electric stimulation. The electric stimulation is applied over the masseter muscles. The current is intermittent (12 seconds on and 12 seconds off). The intensity should be high enough to get a contraction of the masseter muscle as long as the patient can tolerate the current. The contracting masseter loads the condyle on the posterior attachment. The posterior attachment (which is already stretched) can be further stretched by having the patient actively open or passively stretch the mouth three to four times while the current is on (Fig 8-17). Patients with joint pain seem to tolerate the active and passive stretching better during electric stimulation. The result is often an increase in mandibular opening.

Manual Intraoral Techniques. Intraoral mobilization techniques are used to successfully restore functional mandibular dynamics (Fig 8-34).[58,79,113,154,177] Distraction

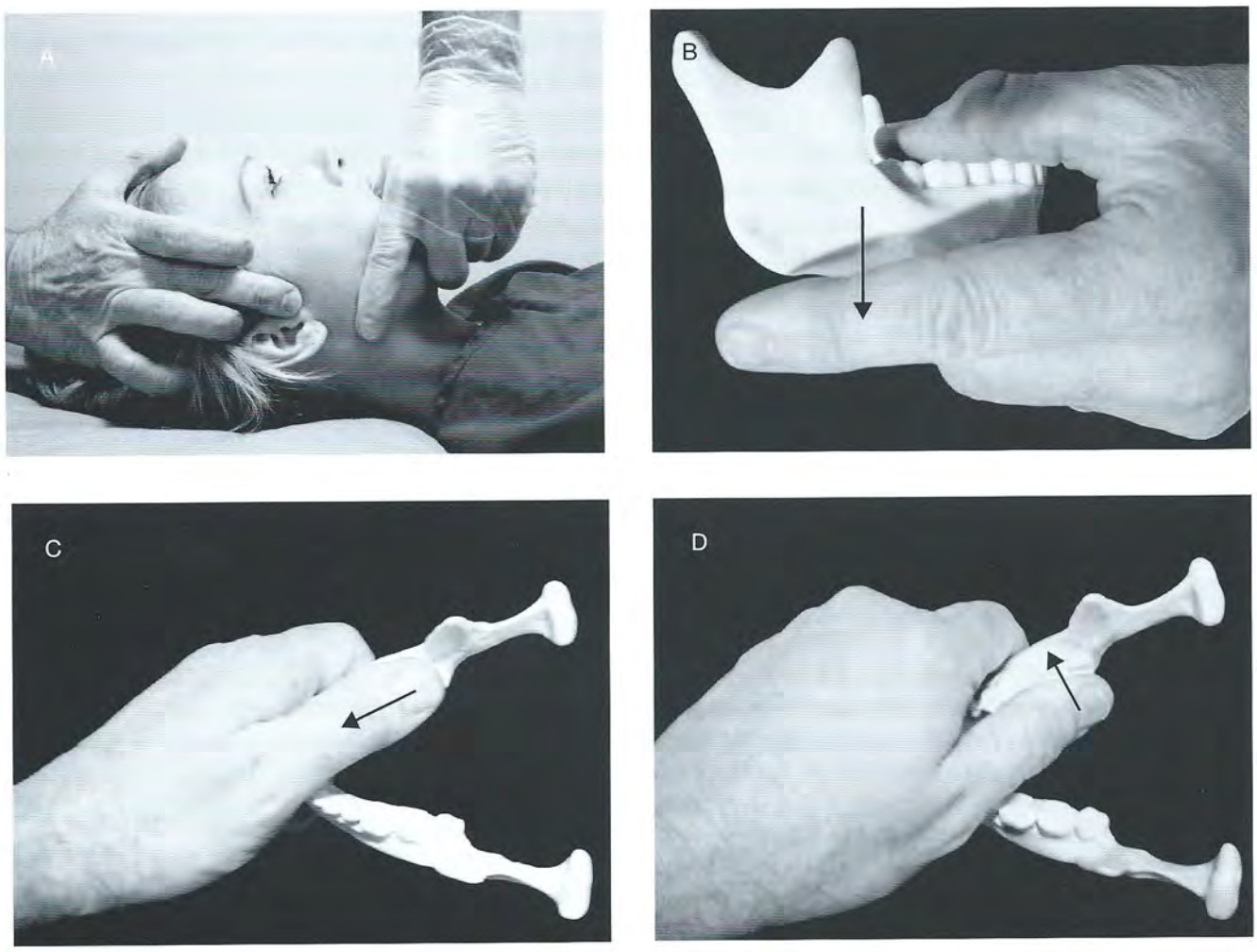

Figure 8-34: Intraoral mobilization techniques for TMJ hypomobility. A) Hand placement with the right hand stabilizing and palpating the condyle, and the left hand mobilizing; B) Distraction technique; C) Translation technique; D) Lateral glide technique.

and translation are the techniques typically used. The lateral glide mobilization also can be used but is usually more effective in the treatment of fibrous adhesions.

Home Exercise Program. Home exercises can be very effective alone or with the other treatments described, and consist of both dynamic and static stretches.

Some recommended home exercises (Fig 8-35):

A. Finger Spread Stretch—The patient uses the thumb and finger to apply a stretch as shown. Five to ten repetitions are performed 6-8 times/day.

B. Repetitive Protrusion—The patient holds seven tongue depressors horizontally between the upper and lower central incisors. Seven tongue depressors are approximately 10 mm, which is where translation usually begins. This position improves the patient's tolerance to repetitive protrusion. The patient performs 20-30 repetitions, 2-3 times/day.

C. Touch and Bite—Some patients lose proprioceptive awareness of mandibular lateral excursion and protrusion. The touch and bite exercise helps restore awareness and range of motion. To gain lateral excursion, the patient places a fingertip on the contralateral maxillary canine tooth. The patient is then asked to attempt to bite the index finger. The patient has to move the jaw laterally to accomplish this movement. The finger provides proprioceptive feedback of the direction the jaw is to move. To gain protrusion, the patient touches the maxillary incisors with the index finger and is instructed to attempt to bite the index

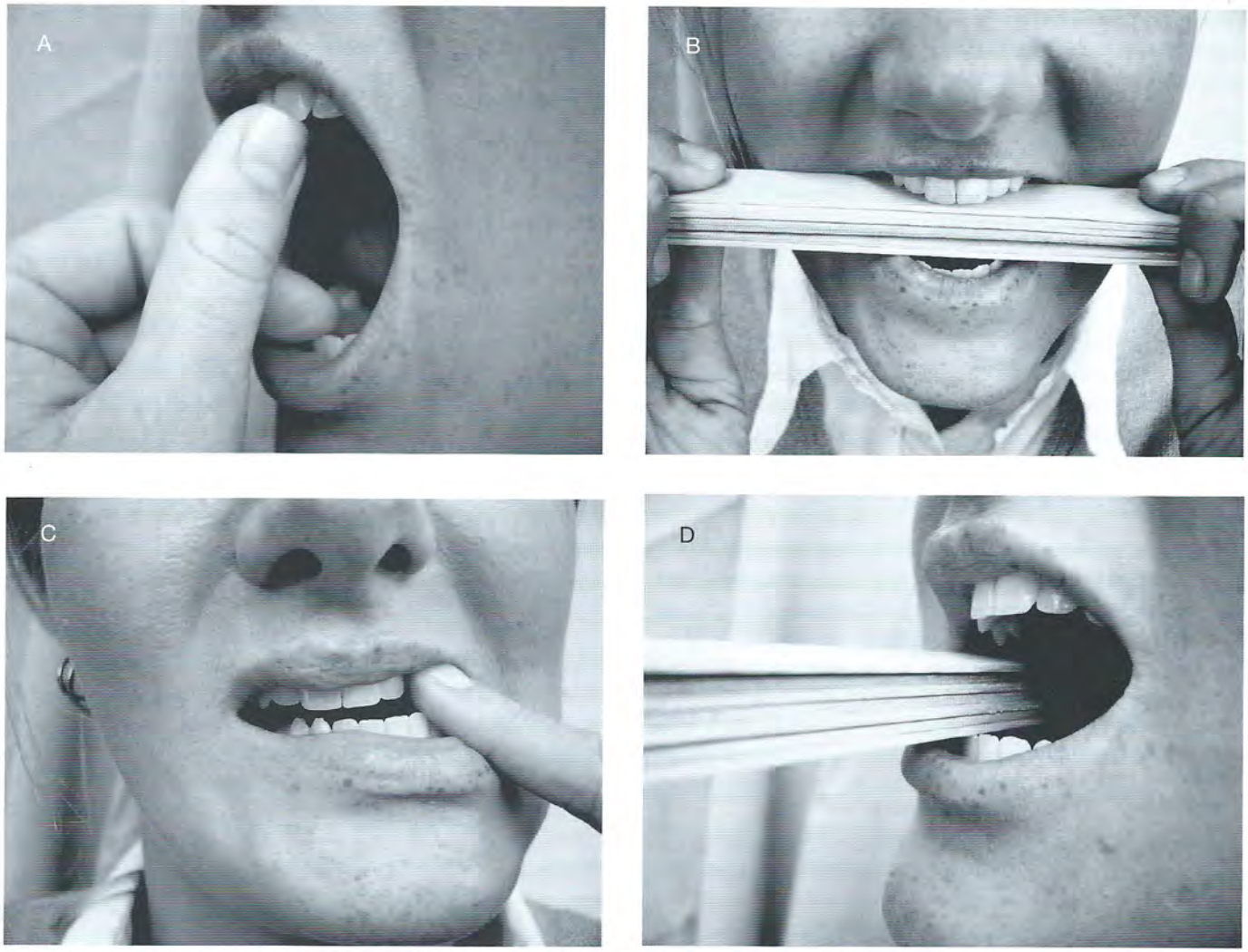

Figure 8-35: Home exercises for TMJ hypomobility. A) Passive finger spread stretch; B) Repetitive protrusion with 7-10 tongue depressors to keep the mouth opened to 10 mm; C) Touch and bite exercise to improve proprioceptive awareness for lateral excursion; D) Static stretch with tongue depressors.

finger. Five to ten repetitions are performed 6-8-times/day.

D. Static Stretch—Stacked tongue depressors are inserted between the patient's ipsilateral molars. The clinician should choose enough tongue depressors to take up the joint slack and provide a slight stretch. A static stretch should be held up to 5 minutes, 2-3 times/day.

It should be noted that increased opening achieved from physical therapy techniques may be due to elongation of the posterior attachment, which causes further anterior displacement and advanced deformation of the disc. When the disc is displaced and the condyle functions on the posterior attachment, the posterior attachment is capable of remolding and fibrosing. In effect, a pseudo-disc develops.[14] Remolding of the posterior attachment is not always successful, or may be inadequate or temporary. If physical therapy, a trial of a stabilization appliance and medication have all failed to restore function and eliminate pain, a surgical consultation is in order.

Fibrous Adhesions

Fibrous adhesions involve tightness of the TMJ capsule and/or TMJ ligament. Fibrous adhesions result from trauma or from a period of joint immobility. Immobility has been well documented to cause physiological changes in the joint capsule,[4,146] with adhesions occurring after as little as two weeks of immobility.[3]

Immobility can be caused by patient apprehension of pain, a Stage II disc displacement, intermaxillary fixation post-orthognathic surgery, trauma or arthrotomy involving

a surgical incision into the joint capsule. Limited joint mobility can lead to additional problems. Adhesions can develop in the superior joint space producing decreased movement between the disc and temporal bone.[132] Limited joint mobility can contribute to an accumulation of waste products on the surfaces of the cartilage, leading to cartilage cell dystrophy and potential arthritis.[61] Finally, limited joint mobility can affect mechanoreceptor activity.

Polyarthritic disease is an uncommon cause of fibrous adhesions.[132] Polyarthritides are caused by generalized systemic polyarthritic conditions such as rheumatoid arthritis, juvenile rheumatoid arthritis, spondyloarthropathies, crystal-induced diseases and Reiter's syndrome. Each of the polyarthritides is best diagnosed via serologic tests and managed by a rheumatologist. Physical therapy management may include addressing pain and functional limitations.

Other uncommon causes of adhesions are fibrous ankylosis or bony ankylosis of the TMJ. Fibrous ankylosis is the result of significant trauma or surgery causing hemarthrosis and eventual fibrous adhesions.[10] Bony ankylosis or ossification is even less common than fibrous ankylosis. Ossification is more likely to occur when an infection has been present.[10] Bony ankylosis is often bilateral due to the etiology of infection.

History—Fibrous Adhesions

The history is often the key to identifying fibrous adhesions of the capsule. The clinician should pay particular attention to situations that can cause immobility, which include chronic pain, Stage II disc displacement, intermaxillary fixation, trauma and arthrotomy. An open incision into the joint capsule is a leading cause of fibrous adhesions.

Physical Examination—Fibrous Adhesions

Altered mandibular dynamics are seen with fibrous adhesions, and are the same as for a Stage II disc displacement (Fig 8-14). The key to differential diagnosis is the history.

Physical Therapy Treatment for Fibrous Adhesions

Treatment for fibrous adhesions is essentially the same as treatment for a Stage II disc displacement. The most important difference is how aggressive the treatments are applied and how soon translation is engaged. For example, the physical therapist should be fairly conservative in the early days post-arthrotomy that involved repairing or replacing the disc with autogeneous tissue or alloplastic material.

The physical therapist should tailor the exercises, modalities and manual procedures to a level that is acceptable for healing tissues. If there are any questions as to the arthrotomy procedure done, the physical therapist should consult with the oral surgeon to find out if there are any special precautions required. Exercises, modalities and manual procedures can be applied more aggressively post-arthrotomy for discectomy, bony ankylosis, intermaxillary fixation and total joint prosthesis.

REFERENCES

1. A De Wijer, de Leeuw JR, Michel H. Steenks, et al: Temporomandibular and Cervical Spine Disorders: Self-Reported Signs and Symptoms. Spine 21:1638-1646, 1996.
2. Abdel-Fattah RA: An Introduction to Occlusal Biomechanics in Temporomandibular Disorders. Cranio 15(4):349-350, 1997.
3. Akeson WH, Amiel D, and Woo S: Immobility Effects of Synovial Joints: The Pathomechanics of Joint Contracture. Biorheology 17:95-110, 1980.
4. Akeson WH, Amiel D, LaViolette D, et al: The Connective Tissue Response to Immobility: An Accelerated Aging Response. Exp Gerontol 3:289-301, 1968.
5. Alkofide EA, Clark E, El-Bermani W, et al: The Incidence and Nature of Fibrous Continuity Between the Sphenomandibular Ligament and the Anterior Malleolar Ligament of the Middle Ear. J Orofac Pain 11(1):7-14, 1995.
6. Assael LA: Arthrotomy for Internal Derangements. In: Temporomandibular Disorders, Diagnosis and Treatment. AS Kaplan, LA Assael eds. WB Saunders, Philadelphia 1991.
7. Atwood DA: A Review of the Fundamentals on Rest Position and Vertical Dimension. Int Dent J 9:6-19, 1959.
8. Bays RA. Surgery for Internal Derangement. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 275-300. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
9. Bell WE: Classification of TM Disorders. In: The President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders, pp 24-29. DM Laskin, W Greenfield, E Gale, et al eds. American Dental Association, Chicago 1982.
10. Bell WE: Temporomandibular Disorders; Classification, Diagnosis and Management, 3rd ed. Year Book Medical Publishers, Inc, Chicago 1990.
11. Benoit PW and Razook SJ: TMJ Imaging. In: Clinics in Physical Therapy; Temporomandibular Disorders, 2nd ed, pp 115-124. SL Kraus, ed. Churchill Livingstone, New York

1994.
12. Bertilsson O and Strom D: A Literature Survey of a Hundred Years of Anatomic and Functional Lateral Pterygoid Muscle Research. J Orofac Pain 9(1):17-23, 1995.
13. Blaustein DI and Heffez LB: Arthroscopic Atlas of the Temporomandibular Joint. Lea and Febiger, Philadelphia 1990.
14. Blaustein DI, and Scapino RP: Remolding of the Temporomandibular Joint Disc and Posterior Attachment in Disc Displacement Specimens in Relation to Glycosaminoglycan Content. Plast Reconstr Surg 79:756-764, 1986.
15. Bogduk N: Cervical Causes of Headache and Dizziness. In: Modern Manual Therapy, pp 289-302. G Grieve, ed. Churchill Livingstone, Edinburgh 1986.
16. Boyd JP, Shankland W, Brown C, et al: Taming the Muscular Forces That Threaten Everyday Dentistry. Postgraduate Dentistry, Nov 2000.
17. Boyd RL, Gibbs CH, Mahan PE, et al: Temporomandibular Joint Forces Measured at the Condyle of Macaca Arctoides. Am J Orthod Dentofac Orthop 97:472-479, 1990.
18. Braun BL, DiGiovanna A, Schiffman E, et al: A Cross-Sectional Study of Temporomandibular Joint Dysfunction in Post-Cervical Trauma Patients. J Orofac Pain 6(1):24-31, 1992.
19. Brenman HS, and Amsterdam, M: Postural Effects on Occlusion. Dental Progress 4:43–47, 1963.
20. Brooks SL, Brand JW and Gibbs SJ, et al: Imaging of the Temporomandibular Joint: A Position Paper of the American Academy of Oral and Maxillofacial Radiology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 83:609-618, 1997.
21. Burch JG: Activity of the Accessory Ligaments of the Temporomandibular Joint. J Prosthet Dent 24:621-628, 1970.
22. Carlson, CR, Okeson JP, Falace DA et al: Comparison of Psychologic and Physiologic Functioning Between Patients With Masticatory Muscle Pain and Matched Controls. J Orofac Pain 7(1):15-22, 1993.
23. Chen Y-S, Gaallo LM and Meier D, et al: Dynamic Magnetic Resonance Imaging Technique For the Study of the Temporomandibular Joint. J Orofac Pain 14(1):65-73, 2000.
24. Cholitgul W, et al. Clinical and Radiological Findings in Temporomandibular Joints With Disc Perforations. Int J Oral Maxillofac Surg 19:220-225, 1990.
25. Ciancaglini R, Testa M and Radaelli G: Association of Neck Pain With Symptoms of Temporomandibular Dysfunction in the General Adult Population. Scand J Rehab Med 31: 17-22, 1999.
26. Clark GT, and Mulligan RA. A Review of the Prevalance of Temporomandibular Dysfunction. J Gerodontaol 3:231, 1986.
27. Clark GT, Green EM, Doman MR, et al: Craniocervical Dysfunction Levels in a Patient Sample From a Temporomandibular Joint Clinic. J Am Dent Assoc 115: 251-256, 1987.
28. Clark GT, Moody DG, and Saunders B: Arthroscopic Treatment of Temporomandibular Joint Locking Resulting From Disc Derangement: Two-year Results. J Oral Maxillofac Surg 49:157-164, 1991.
29. Clark GT, Seligman DA, Solberg WK, et al: Guidelines for the Examination and Diagnosis of Temporomandibular Disorders. J Orofac Pain 3(1):7-14, 1989.
30. Clark GT, Seligman DA, Solberg WK, et al: Guidelines for the Treatment of Temporomandibular Disorders. J Orofac Pain 4(2):80-88, 1990.
31. Clark GT: A Critical Evaluation of Orthopedic Interocclusal Appliance Therapy: Design, Theory, and Overall Effectiveness. J Am Dent Assoc 108:359-368, 1984.
32. Clark GTL Occlusal Therapy: Occlusal Appliances. In: The President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders, pp 137-146. DM Laskin, W Greenfield, E Gale, et al eds. American Dental Association, Chicago 1982.
33. Clark RF and Wyke BD: Contributions of Temporomandibular Articular Mechanoreceptors to the Control of Mandibular Posture: An Experimental Study. J Dent 2:121-129, 1974.
34. Copray JC, Dibbets JM and Kantomaa T: The Role of Condylar Cartilage in the Development of the Temporomandibular Joint. Angle Orthod 58: 369, 1988.
35. Daly P: Postural Response of the Head to Bite Opening in Adult Male. Am J Orthod 82:157-160, 1982.
36. Dawson P: Evaluation, Diagnosis, and Treatment of Occlusal Problems, 2nd ed. Mosby, St Louis 1989.
37. Kronn S: The Incidence of TMJ Dysfunction in Patients Who Have Suffered a Cervical Whiplash Injury Following a Traffic Accident. J Orofac Pain 7(2):209-213, 1993.
38. De Laat, Meuleman H, Stevens A, et al: Correlation Between Cervical Spine and Temporomandibular Disorders. Clinical Oral Invest 2:54-57, 1998.
39. de Wijer A, Lobbezoo-Scholte AM, Steenks MH, et al: Reliability of Clinical Findings in Temporomandibular Disorders. J Orofac Pain 9(2):181-190, 1995.
40. De Wijer A: Temporomandibular and Cervical Spine Disorders. (Thesis) Utrecht University (Dissertation). Utrecht, The Netherlands, Elinkwijk BV 1995.
41. DeBont LGB, Boering G, Liem RSB, et al: Osteoarthritis and Internal Derangement of the Temporomandibular Joint: A Light Microscopic Study. J Oral Maxillofac Surg 44:634-643, 1986.
42. deBont LGM, Dijkeraaf LC, and Stegenga B: Epidemiology and Natural Progression of Articular Temporomandibular Disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 83: 72-76, 1997.
43. DeLeeuw JRJ, Steenks MH, Ros WJG, et al: Psychosocial Aspects and Symptom Characteristics of Craniomandibular Dysfunction. The Assessment of Clinical and Community Findings. J Oral Rehabil 21:127-143, 1994.
44. Dijkstra PU, de Bont LGM, Leeuw R, et al. Temporomandibular Joint Osteoarthrosis and Temporomandibular Joint Hypermobility. J Craniomandibular Pract 11(4):268-275, 1993.
45. Dijkstra PU, de Bont LGM, Stegenga B, et al: Temporomandibular Joint Osteoarthrosis and Generalized Joint Hypermobility. J Craniomandibular Pract 10(3):221-227, 1992.
46. Dijkstra PU, de Bont LGM, van der Weele LT, et al: The Relationship Between Temporomandibular Joint Mobility and Peripheral Joint Mobility Reconsidered. J Craniomandibular Pract 12:149-154, 1994.
47. Dodson TB: Epidemiology of Temporomandibular Disorders. In: Oral and Maxillofacial Surgery.

Temporomandibular Disorders Vol 4, pp 93-107. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
48. Dolwick MF: Diagnosis and Etiology of Internal Derangements of the Temporomandibular Joint. In: The President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders, pp 112-117. DM Laskin, W Greenfield, E Gale, et al eds. American Dental Association, Chicago 1982.
49. Dworkin S and LeResche L, eds: Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examination and Specifications, Critique. J Orofac Pain 6(4):301-355, 1992.
50. Dworkin SF, Huggins KH, Wilson L, et al: A Randomized Clinical Trial Using Research Diagnostic Criteria for Temporomandibular Disorders—Axis II to Target Clinic Cases for a Tailored Self-Care TMD Treatment Program. J Orofac Pain 16(1):48-63, 2002.
51. Dworkin SF, LeResche L, DeRouen T, and Korff M: Assessing Clinical Signs of Temporomandibular Disorders: Reliability of Clinical Examiners. J Prosthet Dent 63: 574-580, 1990.
52. Eberle WR: A Study of Centric Relation as Recorded in a Supine Position. J Am Dent Assoc 42:15, 1951.
53. Eggleton TM and Langton DP: Clinical Anatomy of the TMJ Complex. In: Clinics in Physical Therapy; Temporomandibular Disorders, 2nd ed, pp 1-40. SL Kraus, ed. Churchill Livingstone, New York 1994.
54. Eriksson L and Westesson PL: Clinical and Radiological Study of Patients With Anterior Disc Displacement of the Temporomandibular Joint. Swed Dent J 7:55, 1983.
55. Eriksson L and Westesson PL: Clinical and Radiological Study of Patients With Anterior Disc Displacement of the Temporomandibular Joint. Swed Dent J 7:55-64, 1983.
56. Esposito C, Clear M, and Veal S: Arthroscopic Surgical Treatment of Temporomandibular Joint Hypermobility With Recurrent Anterior Dislocation: An Alternative to Open Surgery. J Craniomandib Pract 9(3):286-292, 1991.
57. Farrar WB and McCarty WL: Inferior Joint Space Arthrography and Characteristics of the Condylar Path in Internal Derangements of the TMJ. J Prosthet Dent 41:548-555, 1979.
58. Friedman MH: Closed Lock. A Survey of 400 Cases. Oral Surg Oral Med Oral Pathol 75:422-427, 1993.
59. Fukushima S: Function of Temporomandibular Joint During Habitual Opening and Closing Movements. J Jpn Prosthodont Soc 15: 267-290, 1971.
60. Gelb H and Tarte J: A Two-year Clinical Evaluation of 200 Cases of Chronic Headache: The Craniocervical-Mandibular Syndrome. J Am Dent Assoc 91:1230-1236, 1975.
61. Glineburg RW, Laskin DM, and Blaustein DI. The Effects of Immobilization on the Primate Temporomandibular Joint: A Histologic and Histochemical Study. J Oral Maxillofacial Surgery 40:3-8, 1982.
62. Goldstein DF, Kraus SL, Williams WB, et al: Influence of Cervical Posture on Mandibular Movement. J Prosthet Dent 52:421-426, 1984.
63. Goulet JP and Clark GT: Clinical TMJ Examination Methods. CDA Journal 18(3):25-33, 1990.
64. Greene CS, Turner C, and Laskin DM: Long-term Outcome of TMJ Clicking in 100 MPD Patients. J Dent Res 61:218, 1982.
65. Greene CS: Can Technology Enhance TM Disorder Diagnosis? CDA Journal 18(3):21-24, 1990.
66. Greene CS: The Etiology of Temporomandibular Disorders: Implications for Treatment. J Orofac Pain 15(2): 93-105, 2001.
67. Headache Classification Committee of the International Headache Society. Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain. Cephalalgia: An International Journal of Headache 8(Suppl 7):9-93, 1988.
68. Helms CA and Kaplan P: Diagnostic Imaging of the Temporomandibular Joint: Recommendations for Use of the Various Techniques. AM J Roentgenol 154:319-326, 1990.
69. Hesse JR and Hansson T: Factors Influencing Joint Mobility in General and in Particular Respect of the Craniomandibular Articulation: A Literature Review.J Orofac Pain 2(1): 19-28, 1988.
70. Hesse JR, Naejie M: Biomechanics of the TMJ. In: Clinics in Physical Therapy; Temporomandibular Disorders, 2nd ed, pp 41-69. SL Kraus ed. Churchill Livingstone, New York 1944.
71. Hochstedler JL, Allen BA and Follmar MA: Temporomandibular Joint Range of Motion: A Ratio of Interincisal Opening to Excursive Movement in a Healthy Population. J Craniomandibular Pract 14(4):296-300, 1996.
72. Holmlund AB: Arthroscopy.In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 255-274. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
73. Hylander WL: An Experimental Analysis of Temporomandibular Joint Reaction Force in Macaques. Am J Phys Anthropol 51:433-456, 1979.
74. Ide Y and Nakazawa K: Anatomical Atlas of the Temporomandibular Joint. Quintessence Publishing Co, Tokyo 1991.
75. Irby WB and Zetz MR: Osteoarthritis and Rheumatoid Arthritis Affecting the Temporomandibular Joint. In: The President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders, pp 106-111. DM Laskin, W Greenfield, E Gale, et al eds. American Dental Association, Chicago 1982.
76. Isaccsson G, Linde C and Isberg A: Subjective Symptoms in Patients With Temporomandibular Joint Disc Displacement Versus Patients With Myogenic Craniomandibular Disorders. J Prosthet Dent 61:70-77, 1989.
77. Isberg A, Stenstrom B and Isacsson G: Frequency of Joint Bilateral Joint Disc Displacement in Patients With Unilateral Symptoms: A 5-year Follow-Up Of the Asymptomatic Joint. Dentomaxillofac Radiol 20:73-76, 1991.
78. Ito T, Gibbs CH, Marguelles-Bonnet R, et al: Loading on the Temporomandibular Joint With Five Occlusal Conditions. J Prosthet Dent 56(4):478-484, 1986.
79. Jagger RG: Mandibular Manipulation of Anterior Disc Displacement Without Reduction. J Oral Rehab 18:497-500, 1991.
80. Jankelson B: The Myo-Monitor: Its Use and Abuse. Quint Int 2:47, 1978.
81. Jee W: The Skeletal Tissues. In: Histology, Cell and Tissue Biology. L Weiss ed. Elsevier Biomedical Publishers, New York 1988.
82. Kerr FW: Mechanism, Diagnosis and Management of Some

Cranial and Facial Pain Syndromes. Surg Clin N Am 43:951-961, 1963.
83. Kircos LT, Ortendahl DA, Mark AS, et al: Magnetic Resonance Imaging of the TMJ Disc in Asymptomatic Volunteers. J Oral Maxillofac Surg 45:852-854, 1987.
84. Kirveskari P, Alanen P, Karskela V, et al: Association of Functional State of Stomatognathic System With Mobility of Cervical Spine and Neck Muscle Tenderness. Acta Odont Scand 46:281-286, 1988.
85. Klineberg I: Influences of Temporomandibular Articular Mechanoreceptors on Functional Jaw Movements. J Oral Rehabil 7:307-317, 1980.
86. Klineberg IJ, Greenfield BE and Wyke BD: Contributions to the Reflex Control of Mastication From Mechanoreceptors in the Temporomandibular Joint Capsule. Dent Pract 21:73-83, 1970.
87. Kraus SL: Physical Therapy Management of Temporomandibular Disorders. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 161-193. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
88. Laskin DM: Etiology and Pathogenesis of Internal Derangements of the Temporomandibular Joint. In: Oral and Maxillofacial Surgery Clinics of North America: Current Controversies in Surgery for Internal Derangements of the Temporomandibular Joint. pp 217-222. DM Laskin, ed. WB Saunders, Philadelphia 1994.
89. Laskin DM: Etiology of the Pain-Dysfunction Syndrome. J Am Dent Assoc 79:147-153, 1969.
90. Lawrence ES, and Razook SJ: Nonsurgical Management of Mandibular Disorders. In: Clinics in Physical Therapy, Temporomandibular Disorders, 2nd Ed, p 161. SL Kraus, ed. Churchill Livingstone, New York 1994.
91. Lerman M: The Hydrostatic Appliance: A New Approach to Treatment of the TMJ Pain Dysfunction Syndrome. J Am Dent Assoc 89:1343-1350, 1974.
92. Lipp MJ: Temporomandibular Symptoms and Occlusion: A Review of the Literature and the Concept. NY State J Dent 56(9):58-64, 1990.
93. Lobbezoo F and Lavigne GJ: Do Bruxism and Temporomandibular Disorders Have a Cause-Effect Relationship? J Orofac Pain 11(1):15-23, 1997.
94. Lobbezoo-Scholte AM, Leeuw De JRJ, Steenks MH, et al: Diagnostic Subgroups of Craniomandibular Disorders. Part 1: Self-Report Data and Clinical Findings. J Orofac Pain 9(1):24-36, 1995.
95. Lous J: Treatment of TMJ Syndrome by Pivots. J Prosth Dent 40:(2)179-182, 1978.
96. Lowe AA: Tongue Movements—Brainstem Mechanics and Clinical Postulates. Brain Behavior 25:128-137, 1984.
97. Lund JP: Pain and the Control of Muscles. Adv Pain Res Therapy 21:103-115, 1995.
98. Lund P, Nishiyama T and Moller E: Postural Activity in the Muscles of Mastication With the Subject Upright, Inclined, and Supine. Scand J of Dent Res 78:417-424, 1970.
99. Lundh H, Westesson PL, Kopp S, et al: Anterior Repositioning Splint in the Treatment of Temporomandibular Joints With Reciprocal Clicking: Comparison With a Flat Occlusal Splint and an Untreated Control Group. Oral Surg Oral Med Oral Pathol 60:131-136, 1985.
100. MacConaill MA: Studies in the Mechanics Of Synovial Joints. Ir J Med Sc 6:223, 1946.
101. MacConaill MA: The Movements of Bones and Joints. JBJS [Br] 35:290, 1953.
102. Mahan PA, Wilkinson TM, Gibbs CH, et al: Superior and Inferior Bellies of the Lateral Pterygoid Muscle EMG Activity at Basic Jaw Positions. J Prosthet Dent 50:710-718, 1983.
103. Mahan PE: The Temporomandibular Joint in Function and Pathofunction. In: Temporomandibular Joint Problems. WK Solberg and GT Clark eds. Quintessence Publishing Co, Lombard, IL 1980.
104. Major PW and Nebbe B: Use and Effectiveness of Splint Appliance Therapy: Review of Literature. J Craniomandib Pract 15(2):159-166, 1997.
105. Mclean LW, Brenman HS, Friedman MGF: Effects of Changing Body Position on Dental Occlusion. J Dent Res 52:1041-1045, 1973.
106. McNamara JA Jr, Seligman DA and Okeson JP: The Relationship of Occlusal Factors and Orthodontic Treatment To Temporomandibular Disorders. In: Temporomandibular Disorders and Related Pain Conditions, pp 399-427. BJ Sessle, PS Bryant, and RA Dionne eds. IASP Press, Seattle 1995.
107. McNamara JA, Jr: The Independent Functions of the Two Heads of the Lateral Pterygoid Muscle. Am J Anat 138:197-206, 1973.
108. McNamara JA, Seligman DA and Okeson JP: Occlusion, Orthodontic Treatment, and Temporomandibular Disorders: A Review. J Orofac Pain 9(1):73-115, 1995.
109. McNeill C, Danzig WM, Farrar WB, et al. Craniomandibular (TMJ) Disorders—The State of Art. Position Paper of the American Academy of Craniomandibular Disorders. J Prosthet Dent 44: 434-437, 1980.
110. Mennell J: Joint Pain. Little Brown and Company, Boston 1964.
111. Milam SB: Pathophysiology of Articular Disk Displacement of the Temporomandibular Joint. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 46-72. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
112. Mills D, Fiandaca D, and Scapino R: Morphologic, Microscopic, and Immunohistochemical Investigations Into the Function of the Primate TMJ Disc. J Orofac Pain 8(2):136-154, 1994.
113. Minagi S, Nozaki S, Sato T, et al: Manipulation Techniques for Treatment of Anterior Disk Displacement Without Reduction. J Prosthet Dent 65:686-691, 1991.
114. Minarelli A, Del Santo M, and Liberti E: The Structure of the Human Temporomandibular Joint Disc: A Scanning Electron Microscopy Study. J Orofac Pain 11(2):95-100, 1997.
115. Moffett BC. Definitions of Temporomandibular Joint Derangements. In: Diagnosis of Internal Derangements of the Temporomandibular Joint, Vol 1. Double-Contrast Arthrography and Clinical Considerations. BC Moffett and PL Westesson eds. Proceedings of a Continuing Dental Education Symposium, Seattle 1984.
116. Mohl N: Head Posture and Its Role in Occlusion. NY State Dent J 42:17-23, 1976.
117. Mohl ND and Dixon DC: Current Status of Diagnostic Procedures for Temporomandibular Disorders. J Am Dent

Assoc, 125:56-64, 1994.
118. Mohl ND: Functional Anatomy of the Temporomandibular Joint. In: The President's Conference on the Examination, Diagnosis and Management of Temporomandibular Disorders, pp 3-12. DM Laskin, W Greenfield, E Gale, et al, eds. American Dental Association, Chicago 1982.
119. Mohl ND: The Anecdotal Tradition and the Need for Evidence-Based Care for Temporomandibular Disorders. J Orofac Pain 13(4)227-231, 1999.
120. Mohl ND: The Temporomandibular Joint. In: Textbook of Occlusion. ND Mohl, GA Zarb, GE Carlsson, et al eds. Pp 81-96, Quintessence Publishing Company, Chicago 1988.
121. Molina OF, Santos JD, Mazzetto M, et al: Oral Jaw Behavior in TMD and Bruxism: A Comparison Study by Severity of Bruxism. J Craniomandib Pract 19(2):114-122, 2001.
122. Moloney F and Howard JA: Internal Derangements of the Temporomandibular Joint III. Anterior Repositioning Splint Therapy. Aust Dent J 31:30-39, 1986.
123. Montgomery MT, Gordon SM, Van Sickels JE, et al: Changes in Signs and Symptoms Following Temporomandibular Joint Disc Repositioning Surgery. J Oral Maxillofac Surg 50:320-328, 1992.
124. Montgomery MT, Van Sickels JE, Harms SE. Success of Temporomandibular Joint Arthroscopy in Disk Displacement With and Without Reduction. Oral Surg Oral Med Oral Pathol 71:651-659, 1991.
125. Morani V, Previgliano V and Schierano G: Innervation of the Human Temporomandibular Joint Capsule and Disc as Revealed by Immunohistochemistry for Neurospecific Markers. J Orofac Pain 8(1):36-41, 1994.
126. Murakami KI, Segami N, Fujimura K, et al: Correlation Between Pain and Synovitis in Patients With Internal Derangement of the Temporomandibular Joint. J Oral Maxillofac Surg 49:1159-1161, 1991.
127. Naeije M, and Hansson TL: Electromyographic Screening of Myogenous and Arthrogenous TMJ Dysfunction Patients. J Oral Rehabil 13:433-441, 1986.
128. Nassif NJ and Talic YF: Classic Symptoms in Temporomandibular Disorder Patients: A Comparative Study. J Craniomandibular Pract 19(1):33-41, 2001.
129. National Institutes of Health Technology Assessment Conference On Management of Temporomandibular Disorders. pp 15-120. Judith Albino, Chairperson. Bethesda, Maryland, April 29 - May 1, 1996.
130. Nicolakis P, Nicolakis M, Piehslinger E, et al: Relationship Between Craniomandibular Disorders and Poor Posture. J Craniomandibular Pract 18(2):106-112, 2000.
131. Nitzan DW, Dolwick MF and Heft MW: Arthroscopic Lavage and Lysis of the Temporomandibular Joint: A Change In Perspective. J Oral Maxillofac Surg 48:798-801, 1990.
132. Okeson JP ed. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. Quintessence Publishing Co, Chicago 1996.
133. Okeson JP: The Management of Temporomandibular Disorders and Occlusion. 3rd ed. Mosby, St. Louis 1993.
134. Orenstein ES: Anterior Repositioning Appliances When Used for Anterior Disk Displacement With Reduction—A Critical Review. J Craniomandibular Pract 11(2):141-145, 1993.
135. Padamsee M, Mehtan N, Forgione A, et al: Incidence of Cervical Disorders in a TMD Population (IADR; Abstract No. 680). J Dent Res 73 1994.
136. Perrott DH, Alborzi A, Kaban LB, et al: A Prospective Evaluation of the Effectiveness of Temporomandibular Joint Arthroscopy. J Oral Maxillofac Surg 48:1029-1032, 1990.
137. Pertes R and Attanasio R: Internal Derangements. In: Temporomandibular Disorders, Diagnosis and Treatment, pp 142-164. AS Kaplan and LA Assael eds. WB Saunders Company, Philadelphia 1991.
138. Pharoah M: The Prescription of Diagnostic Images for Temporomandibular Joint Disorders. J Orofac Pain 13(4): 251-254, 1999.
139. Pinto OF: A New Structure and Function of the Mandibular Joint. J Prosthet Dent 12:95-103, 1962.
140. Pruim GJ, de Jongh HJ and Ten Bosch JJ: Forces Acting on the Mandible During Bilateral Static Bite at Different Bite Force Levels. J Biomech 13:755, 1980.
141. Rees LA: The Structure and Function of the Mandibular Joint. Br Dent J 96:125, 1954.
142. Ribeiro RF, Tallents RH, Katzberg RW, et al: The Prevalence of Disc Displacement in Symptomatic and Asymptomatic Volunteers Aged 6-25 Years. J Orofac Pain 11(1):37-47, 1997.
143. Root GR, Kraus SL, Razook SJ, et al: Effect of an Intraoral Appliance on Head and Neck Posture. J Prosthet Dent 58:90-95, 1987.
144. Rosenbaum RS, ed. Orofacial Pain Guidelines for Assessment, Diagnosis, and Management, 4th ed. Quintessence Publishing. Chicago 2004.
145. Rothstein JM: Measurement and Clinical Practice: Theory and Application. In: Measurements in Physical Therapy, pp 1-46. J Rothstein ed. Churchill Livingstone, New York 1985.
146. Salter RB, Hamilton WH, Wedge JH, et al: Clinical Applications of Basic Research on Continuous Passive Motion for Disorders and Injuries of Synovial Joints, a Preliminary Report of a Feasibility Study. J Orthop Res 3:325-342, 1983.
147. Sanders B and Buonocristiani R. Temporomandibular Joint Arthrotomy, Management of Failed Cases. Oral and Maxillofac Surgery Clinics of North America 1:443, 1989.
148. Santander H, Miralles R, Perex J, et al: Effects of Head and Neck Inclination on Bilateral Sternocleidomastoid EMG Activity in Healthy Subjects and in Patients With Myogenic Cranio-Cervical- Mandibular Dysfunction. J Craniomandibular Pract 18(3):181-191, 2000.
149. Sato H, Strom D and Carlsson G: Controversies on Anatomy and Function of the Ligaments Associated with the Temporomandibular Joint: A Literature Survey. J Orofac Pain 9(4):308-316, 1995.
150. Sato S, Goto S, Kawamura H, et al: The Natural Course of Nonreducing Disc Displacements of the TMJ: Relationship of Clinical Findings at Initial Visit to Outcome After 12 Months Without Treatment. J Orofac Pain 11(4):315-320, 1997.
151. Scapino R: The Posterior Attachment: Its Structure, Function and Appearance in TMJ Imaging Studies. Part 1: J Orofac Pain 5(2):83-94, 1991.
152. Scapino R: The Posterior Attachment: Its Structure, Function and Appearance in TMJ Imaging Studies. Part 2: J Orofac Pain 5(3):155-166, 1991.
153. Schiffman EL, Fricton JR, Haley DP, et al: The Prevalence and Treatment Needs of Subjects With Temporomandibular

Disorders. J Am Dent Assoc 120:295-303, 1999.
154. Segami N, Murakami KI, and Iizuka T: Arthrographic Evaluation of Disk Position Following Mandibular Manipulation Technique for Internal Derangement With Closed Lock of the Temporomandibular Joint. J Orofac Pain 4(2):99-108, 1990.
155. Seligman DA and Pullinger AG: The Role of Intercuspal Occlusal Relationships in Temporomandibular Disorders: A Review. J Orofac Pain 5(2):96-106, 1991.
156. Sharawy M: Developmental and Clinical Anatomy and Physiology of the Temporomandibular Joint. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 1-19. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
157. Sheppard SM: Asymptomatic Morphologic Variations in the Mandibular Condyle-Ramus Region. J Prosthet Dent 46:539-544, 1982.
158. Simons DG, and Mense S: Understanding and Measurement of Muscle Tone as Related to Clinical Muscle Pain. Pain 75:1-17, 1998.
159. Sollecito T: Role of Splint Therapy in Treatment of Temporomandibular Disorders. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 145-160. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
160. Spitzer WO, Skovron ML, Salmi LR, et al. Scientific Monograph of the Quebec Task Force on Whiplash-Associated Disorders: Redefining "Whiplash" and its Management. Spine 20(8 Suppl) p1S-73S, 1995.
161. Spruijt RJ and Hoogstraten J: Symptom Reporting in Temporomandibular Joint Clicking: Some Theoretical Considerations. J Orofac Pain 6(3):213-218, 1992.
162. Spruijt RJ and Hoogstraten J: The Research on Temporomandibular Joint Clicking: A Methodological Review. J Orofac Pain 5(1) 45-50, 1991.
163. Stohler CS, Zhang X and Lund JP: The Effect of Experimental Jaw Muscle Pain on Postural Muscle Activity. Pain 66:215-221, 1996.
164. Stohler CS: Muscle-Related Temporomandibular Disorders. J Orofac Pain 13(4):273-284, 1999.
165. Summer JD, Westesson PL: Mandibular Repositioning Can Be Effective in Treatment of Reducing TMJ Disk Displacement. A Long Term Clinical and MR Imaging Follow-up. J Craniomandibular Pract 15(2):107-120, 1997.
166. Svensson P and Graven-Nielson T: Craniofacial Muscle Pain: Review of Mechanisms and Clinical Manifestations. J Orofac Pain 15(2):117-145, 2001.
167. Szentpetery A: Clinical Uility of Mandibular Movement Ranges. J Orofac Pain 7(2):163-168, 1993.
168. Tallents RH, Stein SI and Moss ME: The Role of Occlusion in Temporomandibular Disorders. In: Oral and Maxillofacial Surgery. Temporomandibular Disorders Vol 4, pp 194-297. RJ Fonseca ed.; RA Bays and PD Quinn vol eds. WB Saunders Company, Philadelphia 2000.
169. Tennenbaum HC, Freeman BV, Psutka DJ, et al: Temporomandibular Disorders: Disc Displacements. J Orofac Pain 13(4):285-289, 1999.
170. Thilander B: Innervation of the Temporomandibular Disc in Man. Acta Odontol Scand 22:151, 1964.
171. Thilander B: Innervation of the Temporomandibular Joint Capsule in Man. No 7, Transactions of the Royal Schools of Dentistry. Stockholm-Umea, 1961.
172. Travell J, Rinzler S and Herman M: Pain and Disability of the Shoulder and Arm.Treatment by Intramuscular Infiltration With Procaine Hydrochloride. JAMA 120:417-422, 1942.
173. Trumpy I, Erickson J and Lyberg T: Internal Derangement of the Temporomandibular Joint: Correlation of Arthrographic Imaging With Surgical Findings. Int J Oral Maxillofac Surg 26:327-330, 1997.
174. Turell J, and Ruiz G: Normal and Abnormal Findings in Temporomandibular Joints in Autopsy Specimens. J Orofacial Pain 1(4):257-275, 1987.
175. Turell J, and Ruiz HG: Normal and Abnormal Findings in Temporomandibular Joints in Autopsy Specimens. J Orofac Pain 1(4):257-275, 1987.
176. Turp JC, Alt KW, Vach WV, et al: Mandibular Condyles and Rami are Asymmetric Structures. J Craniomandibular Pract 16(1):51-56, 1998.
177. Van Dyke AR, and Goldman SM: Manual Reduction of Displaced Disk. J Craniomandibular Pract 8(4):350-352, 1990.
178. Visscher CM, et al: Cervical Spinal Pain in Chronic Craniomandibular Pain Patients. Thesis prepared at the Department of Oral Function, Section CMD of the Academic Center for Dentistry Amsterdam (ACTA), the combined Faculty of Dentistry of the University of Amsterdam and the Vriji Universiteit, the Netherlands, 2000.
179. Watson T: The Role of Electrotherapy in Contemporary Physiotherapy Practice. Manual Therapy 5(3):132-141, 2000.
180. Watt-Smith S, Sadler A, Baddeley H, et al: Comparison of Arthrotomographic and Magnetic Resonance Images of 50 Temporomandibular Joints With Operative Findings. Br J Oral Maxillofac Surg 31:139-143, 1993.
181. Weinberg L: Treatment Prosthesis in TMJ Dysfunction-Pain Syndrome. J Prosth Dent 39(6):654-669, 1978.
182. Westesson PL, and Rohlin M: Internal Derangement Related to Osteoarthritis in Temporomandibular Joint Autopsy Specimens. Oral Surg Oral Med Oral Pathol 57:17-22, 1984.
183. Westesson PL, Bronstein SL and Liedberg J. Internal Derangement of the Temporomandibular Joint: Morphologic Description With Correlation to Joint Function. Oral Surg Oral Med Oral Pathol 59:323, 1985.
184. Westesson PL, Eriksson L, and Kurita K: Reliability of a Negative Clinical Temporomandibular Joint Examination: Prevalence of Disk Displacement in Asymptomatic Temporomandibular Joints. Oral Surg Oral Med Oral Pathol 68:551-554, 1989.
185. Widmer C, Lund J and Feine J: Evaluation of Diagnostic Tests for TMD. CDA Journal 18(3):53-60, 1990.
186. Widmer C: Evaluation of Diagnostic Test for TMD. In: Clinics in Physical Therapy; Temporomandibular Disorders, 2nd ed, pp 99-114, SL Kraus, ed. Churchill Livingstone, New York 1994.
187. Wilkes C: Internal Derangements of the Temporomandibular Joint: Pathological Variations. Arch Otolaryngol Head Neck Surg 115:469-477, 1989.
188. Williamson EH, and Rosenzweig. The Treatment of Temporomandibular Disorders Through Repositioning Splint Therapy: A Follow-up Study. J Craniomandibular Pract 16(4):222-225, 1998.
189. Wong GV, Weinberg S and Symingen JM: Morphology of

the Developing Articular Disc of the Human Temporomandibular Joint. J Oral Maxillofac Surg 43:565-569, 1985.
190. Yale SH, Allison BD and Hauptfuehrer JD: An Epidemiological Assessment of Mandibular Condyle Morphology. Oral Surg, 21:169, 1966.
191. Zamburlini I, and Austin D: Long-term Results of Appliance Therapies in Anterior Disk Displacement With Reduction: A Review of the Literature. J Craniomandibular Pract 9(4):361-367, 1991.

ACKNOWLEDGEMENTS

To my wife, Pattie, for her love, understanding and patient support; to my daughter Emily Jane, who gives me such joy and happiness; and to my parents, Dottie and Kenneth L. Kraus, my inspiration.