

Guidelines for Diagnosis and Management of Disorders Involving the Temporomandibular Joint and Related Musculoskeletal Structures^I

approved by
American Society of
Temporomandibular Joint Surgeons^{II}

I. PREAMBLE

The following document was developed as a consensus statement by members of a multidisciplinary organization of clinicians who have extensive experience in the diagnosis and treatment of patients with temporomandibular joint (TMJ) arthropathy and associated musculoskeletal disorders. These guidelines represent current accepted theory and practice among the membership.

II. BACKGROUND

Temporomandibular Joint

The temporomandibular joint differs from other joints of the body primarily by its sliding function and having joint surfaces and a disc of fibrocartilage. The disc is attached to the condyle by ligaments that permit rotation on the condyle during translational jaw movements. A highly vascular attachment with elastic fibers secures the posterior border of the disc and a muscle (lateral pterygoid) the anterior border. The sides of the disc are attached to the medial and lateral poles of the condyle.

During jaw movements the condyle and disc slide in the temporal fossa. Without this sliding movement, sideways movement of the jaw during chewing, and especially wide opening, would not be possible. The disc is believed to have several roles, such as, cushioning and distributing joint loads, promoting joint stability during chewing, facilitating lubrication and nourishment of the joint surfaces, preventing gross degenerative changes in the condyle and fossa, and promoting normal growth of the mandible (1). Although some properties of this joint are unique, the disc and joint basically behave similarly to other load-bearing appendicular joints with discs or menisci.

Classification

Temporomandibular disorders (TMD) is a collective term embracing all the problems relating to the TMJ and related musculoskeletal structures. One classification of TMD disorders is listed in Table I. These guidelines, however, will focus on the clinical and pathological condition of disc displacement, which is an internal derangement (ID), and the usual coexisting osteoarthritis (OA), also known as osteoarthrosis or degenerative joint disease (DJD) (2). The reason for this focus is that ID/OA is considered to be the most common cause of serious TMJ pain and dysfunction and therefore the most likely to be treated surgically.

Prevalence

Disc displacement was not found in 30 infants or young children imaged by magnetic resonance (3). However, asymptomatic disc displacement was documented in 8% of juveniles with a mean age of 11 years (4) and about 30% of adult volunteers (5-9). In symptomatic patients, ID is present 80-90% of the time (5-11). Moreover, patients referred for surgical consultation because of symptoms, seem to have an even higher incidence of ID. Only a small and yet-to-be determined fraction of persons with ID and OA become sufficiently symptomatic to seek treatment. The reason for this observation has not been explained, but is possibly related to differences in pathology. Patients have more advanced pathology and more disc interference on opening than asymptomatic volunteers (9).

The prevalence of ID/OA in males and females is not clearly defined, but occurs most often in females (9). Women also comprise about 80% of patients seeking treatment for joint pain (7, 8, 12-15). The average age of patients seeking surgical care is near 30 years (13-16). Those with late stage ID/OA are on average, slightly older (13, 15) than patients with early-stage disease (14).

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Etiology

Failure to find disc displacement or OA in infants and very young children (3) strongly suggests that the condition is not congenital, but is acquired. Joint overloads play a central role in the development of OA in other joints and likely do so in the TMJ as well. Joint overloads may also have a role in the development of disc displacement (17). In addition to overloads, blunt force to the face (18) or flexion-extension (whiplash) neck injuries (19, 20) may cause or aggravate ID/OA. The repetitive loading of clenching and bruxism possibly are etiologic factors, but a clear relationship has not been established. Genetic and metabolic factors may contribute by lowering the threshold for tissue damage from overloads (relative) or trauma and therefore may also be an important factor in the development of ID/OA.

The relationship of the time of onset between ID and OA is poorly understood. It seems more plausible that ID precedes OA than vice versa. A third possibility is that the causative event simultaneously initiates both ID and OA. Once disc displacement is present, however, the ID almost certainly facilitates the progression of pathology, especially the development of the marked bone changes seen in the condyle and temporal fossa (21) in Wilkes stages IV and V.

Pathology

OA is a degenerative disease of movable joints (22). In the TMJ, the disease is characterized by deterioration of the articular cartilage, disc, synovium, and subchondral bone, and with rare exception, disc displacement. A small proportion of joints imaged by magnetic resonance show bone marrow changes in the condyle (23, 24). Histopathological evaluation of these marrow changes suggest they represent osteonecrosis (25). The gross pathological changes, including those of the disc, are well described by Wilkes (12) and illustrate the progressive nature of the disease.

There are also secondary inflammatory changes that result from tissue damage in the joint. Several inflammatory mediators (neuropeptides, cytokines, serotonin, free radicals) have been isolated from fluid in symptomatic joints with ID and OA and may play a role in producing pain (26-29).

Clinical Presentation

The typical signs and symptoms of ID and OA are pain in the joint (preauricular region), headaches behind and around the eyes, and pain radiating from the joint to the temple, ears, side of neck and upper shoulder. The pain is typically aggravated by wide opening, chewing or other joint activities, such as clenching and bruxism. There often is clicking, popping or "locking" because of

disc interference, which results in reflex masticatory muscle spasm. Incisal opening, protrusion, and contralateral movements are decreased and may interfere with mastication. The joint, muscles of mastication, sternocleidomastoid muscle and trapezius muscle are often tender to palpation. A history of changing occlusion and the acquired facial skeletal deformity of mandibular deficiency, open-bite and/or asymmetry may also be signs of ID/OA (1, 30-32).

Natural History

The natural course of ID/OA differs depending on whether one examines pathology or signs and symptoms. Both signs and symptoms improve with time in most persons. Toller reported the rate at which patients improve with non-interventional treatment (33). His findings that about 50% improved by one year, 75% by 2 years and 85% by 3 years, are in general agreement with findings of other studies (21, 30, 34-39). Most or all of Toller's patients had late-stage ID/OA, and except for Boering's patients (30), so did those in the other studies (21, 34-39). Thus, the rate and extent of improvement with early-stage ID/OA is not clear, but may be faster and/or more complete in view of data showing that Wilkes stage IV & V joints are less likely than stage II & III joints to improve with either non-surgical (37) or surgical (13, 15, 40) care.

Pathological changes in the TMJ, on the other hand, progress with time, but at a rate that varies among patients (11, 12, 16, 30, 34). Even though the pathology may progress with changes in occlusion and development of facial deformity, the joint may be asymptomatic. Wilkes staging (Table II), which is based on the progression of gross pathology of ID and OA in the joint, is useful for predicting treatment outcomes (13, 15, 37, 40), as well as for diagnosis.

III. EVALUATION AND DIAGNOSIS

History and Physical Examination:

A detailed history, head and neck evaluation, and general physical examination when indicated, are essential (41-43).

Laboratory Studies:

Laboratory studies are rarely indicated for ID/OA. In other suspected conditions of the TMJ, for example rheumatoid arthritis, the appropriate tests should be ordered.

Imaging

Imaging of the temporomandibular joints and associated structures is necessary to establish the presence or absence of pathology and stage of disease in

order to select the appropriate treatment, assist in prognosis, and assess patient response to therapy. Imaging results will influence treatment strategy. In general, it is recommended that imaging studies be bilateral because of the high incidence of bilateral joint disease. Basic screening radiographs should be used to demonstrate temporal bone and condylar morphology. Radiographs which can provide this information include plain films, panoramic films, and tomograms (frontal and lateral) (44). The disc and associated soft tissue structures should also be imaged. Magnetic resonance imaging (MRI) (45, 46) or arthrography (47, 48) can provide this information. Other radiological studies may also be indicated.

Computed Tomography (CT)

Computed tomography (CT) is very useful to assess bone abnormalities such as ankylosis, dysplasias, growth abnormalities, fractures, and osseous tumors (49). 3-D CT is a valuable diagnostic advancement for complex cases needing major reconstructive surgery. Moreover, a stereolithography model of a patient's maxillofacial skeleton can be fabricated utilizing 3-D CT technology (50).

Magnetic Resonance Imaging

MRI is used to assess soft tissues, bone marrow changes, disc position, morphology, mobility, and joint effusion (45, 46, 51-53). Cine MRI has been used to study the "adherent disc" (also known as: "static disc", "stuck disc", "anchored disc", "suction cup effect") (54).

Arthrography

MRI has largely replaced arthrography (47, 48) as the primary imaging study for disc pathology. However, in selected cases, arthrography still has a role.

Isotope Bone Scan

Radioactive isotope bone scans have a high sensitivity for detecting metabolic activity and inflammation (55). Increased vascularity on the scan appears as increased isotope activity. Single Photon Emission Computed Tomography (SPECT) is a form of isotope imaging utilizing computer techniques to improve visualization of the plane of interest.

Other Studies

To evaluate other sources of pathology adjacent to or related to the TMJ, other head and neck imaging studies may be necessary. A panoramic radiograph is a valuable screening test for assessing sources of facial pain that are often confused with TMJ pain, such as dental infections, neoplasms, sinus pathology and Eagle's syndrome. Plain radiographs, such as transcranial views, are useful for general determinations of condylar morphology and position in the fossa. In instances where more information is required, tomography is recommended.

Diagnostic arthroscopy permits obtaining synovial fluid for analysis and specimens for biopsy.

IV. TREATMENT

General

As in any disease, an understanding of the natural course of ID/OA is necessary to guide rational treatment. Most TMJ symptoms resolve over time, but a significant percentage requires a year or more to do so. The seriousness of the symptoms also varies greatly. On the other hand, the pathology tends to be progressive and can result in loss of condylar bone and development of facial deformity. Thus, when reviewing treatment options, the time course for resolution of symptoms, their seriousness, and the progressive nature of the disease should all be considered. Treatment efforts are directed toward: 1) reduction of pain 2) improvement of dysfunction 3) slowing the progression of ID/OA. Although few current treatment options appear to affect progression of ID/OA favorably (13, 14, 56, 57), this goal gains importance with accumulating evidence suggesting that progression to late-stage disease has a deleterious effect on pain resolution (13, 15, 37, 40) and plays a role in the development of facial deformity (1, 30, 32). A secondary goal of any therapy, should be reduction of disc interference, because of its adverse effect on pain (15, 58), dysfunction (58, 59), and its possible role in development of the deformed condyle of late-stage ID/OA (21).

Non-surgical Treatment

Non-surgical treatment should be considered for all symptomatic patients with ID/OA. For mild or moderate pain and dysfunction, this treatment alone often suffices. Patients with severe pain and dysfunction may also be treated non-surgically, but if adequate reduction of symptoms does not occur within 2-3 weeks, surgical consultation is indicated. In instances of closed lock, regardless of the degree of pain, early surgical consultation is indicated. One or more of the following non-surgical treatment modalities may be utilized (43,60).

1. Diet

Load reduction in the TMJ is achieved by modifying the patient's diet to reduce joint loading from forces of mastication. This is achieved primarily by a non-chewing diet such as liquid or pureed food. As the joint pain improves, the diet may be advanced.

1. Pharmacologic Agents

The nonsteroidal anti-inflammatory drugs (NSAID) are the mainstays in the pharmacological treatment of musculoskeletal disorders where pain and

inflammation are prominent features (61). Low dose tricyclics are effective in controlling pain from nighttime bruxism, when doses are adjusted to provide improved sleep. After psychiatric consultation, if it is determined that clinical depression is an aggravating factor, antidepressant medication can be helpful as part of the treatment. Prolonged use of other medications such as tranquilizers, muscle relaxants, sedatives, and narcotic pain medications are seldom indicated. Narcotic pain medications are commonly used for a short period after surgery. If needed for extended periods of time, it is recommended that a pain-management specialist be consulted.

2. Maxillomandibular Appliances

Maxillomandibular appliances (occlusal splints, orthotics, night guards, bite guards) are widely used for bruxism control. Prolonged use of repositioning appliances for ID/OA can cause undesirable and irreversible changes in dental occlusion, skeletal structure, and muscle dynamics. Other dental treatments, such as occlusal equilibrations, extensive dental restoration, or orthodontic treatment are not indicated as the primary treatment for ID/OA.

3. Physical Therapy (PT)

PT in conjunction with other methods of treatment is used to relieve musculoskeletal pain and improve range of motion (62). Range of motion exercises, whether guided by a physical therapist or the surgeon, is a valuable adjunct after joint surgery.

4. Injections

Injections of tender muscles, trigger areas, and/or joint spaces with local anesthetic solution is used for diagnosis and relief of symptoms. Corticosteroid injection can be effective in reducing capsulitis (63). The use of Botox to eliminate muscle spasm and reduce strength of contraction, while retaining voluntary control, has allowed this drug to be used in a variety of clinical conditions involving muscle hyperactivity. Although its therapeutic use was first reported 20 years ago (64), its application for clenching and bruxism is recent. It appears to be an effective method for treating severe bruxism when traditional methods fail (65, 66). It also appears to be an effective method for treatment of masseteric hypertrophy (67, 68).

5. Behavior Modification

Behavior modification is intended to help patients understand and avoid stress-related lifestyle habits, such as clenching, bruxism, and excessive gum chewing (69). Psychological consultation may be indicated for stress management.

Surgical Treatment

Surgery for treatment ID/OA has the twin advantages of effectiveness and a rapid response. Surgical consultation should be offered within 2-3 weeks to patients with documented ID/OA and in whom severe pain and dysfunction persists after a trial of non-surgical therapy. Early surgical consultation is especially important in cases of closed lock where delay in treatment can accelerate the progression of ID/OA.

Patients contemplating surgery should be given the information necessary for informed consent. This information includes the nature of their condition, its natural history, potential surgical complications, side-effects, prognosis, and reasonable alternative treatment options. A signed consent form should be a part of the medical record.

The following surgical procedures are accepted and effective methods for treatment of joints with ID/OA:

1. Arthrocentesis (70-76)
2. Arthroscopy (26, 27, 77-92)
3. Condylotomy (indirect arthroplasty) (14, 15, 17, 56, 93-113)
4. Arthrotomy (13, 40, 50, 114-153)
Alloplastic implants are not generally indicated for initial surgical treatment of joints with ID/OA. Prosthetic joint replacement may be indicated in selected patients with severe joint degeneration, destruction, or ankylosis. These devices should be considered for use only when their safety and efficacy has been recognized by the FDA.
5. Other Procedures
 - a. Coronoidotomy/coronoidectomy (154-157)
 - b. Styloidectomy (Eagle's Syndrome) (158-160)
 - c. Procedures for Recurrent Dislocation (123, 161-167)

Postoperative Care

Good care after an operation is essential for obtaining an optimal outcome. Patient instructions immediately after surgery should include the following: wound care, thermal applications (ice, heat), non-chew diet regimen, medications, occlusal management, bruxism control as needed, joint motion plans, and any special instructions related to the specific operation. Active or passive joint exercise to increase range of motion is a key component of management after surgery (168). Long-term follow-up is recommended.

TABLE I

Temporomandibular Joint & Related Musculoskeletal Disorders

- I. Intra-articular (Intracapsular) Pathology
 - A. Articular Disc
 - 1. Displacement
 - 2. Deformity
 - 3. Adhesions
 - 4. Degeneration
 - 5. Injury
 - 6. Perforation
 - 7. Anomalous development
 - B. Disc Attachments
 - 1. Inflammation
 - 2. Injury (laceration, hematoma, contusion)
 - 3. Perforation
 - 4. Fibrosis
 - 5. Adhesions
 - C. Synovium
 - 1. Inflammation/effusion
 - 2. Injury
 - 3. Adhesions
 - 4. Synovial hypertrophy/hyperplasia
 - 5. Granulomatous inflammation
 - 6. Infection
 - 7. Arthritides (rheumatoid, degenerative)
 - 8. Synovial chondromatosis
 - 9. Neoplasia
 - D. Articular Fibrocartilage
 - 1. Hypertrophy/hyperplasia
 - 2. Degeneration (chondromalacia)
 - a. Fissuring
 - b. Fibrillation
 - c. Blistering
 - d. Erosion
- E. Mandibular condyle and glenoid fossa (see also Musculoskeletal)
 - 1. Osteoarthritis (osteoarthrosis, degenerative joint disease)
 - 1. Avascular necrosis (osteonecrosis)
 - 2. Resorption
 - 3. Hypertrophy
 - 4. Fibrous and bony ankylosis
 - 5. Implant arthropathy
 - 6. Fracture/dislocations
- II. Extra-articular (Extracapsular Pathology)
 - A. Musculoskeletal
 - 1. Bone (temporal, mandible, styloid)
 - a. Anomalous development (hypoplasia, hypertrophy, malformation, ankylosis)
 - b. Fracture
 - c. Metabolic disease
 - d. Systemic inflammatory disease (connective tissue/arthritides)
 - e. Infection
 - f. Dysplasias
 - g. Neoplasia
 - 2. Masticatory muscles and tendons
 - a. Anomalous development
 - b. Injury
 - c. Inflammation
 - d. Hypertrophy
 - e. Atrophy
 - f. Fibrosis, contracture
 - g. Metabolic disease
 - h. Infection
 - i. Dysplasias
 - j. Neoplasia
 - k. Fibromyalgia
 - B. Central nervous system/peripheral nervous system
 - 1. Reflex sympathetic dystrophy

TABLE II

Staging of Internal Derangement of TMJ (12, 13)

STAGE	CLINICAL	IMAGING	SURGICAL
I. EARLY	Painless clicking No restricted motion	Slightly forward disc, reducing* Normal osseous contours	Normal disc form Slight anterior displacement Passive incoordination (clicking)
II. EARLY/ INTERMEDIATE	Occasional painful clicking Intermittent locking Headaches	Slightly forward disc, reducing Early disc deformity Normal osseous contours	Anterior disc displacement Thickened disc
III. INTERMEDIATE	Frequent pain Joint tenderness, headaches Locking Restricted motion Painful chewing	Anterior disc displacement, reducing early progressing to non-reducing* late Moderate to marked disc thickening Normal osseous contours	Disc deformed & displaced Variable adhesions No bone changes
IV. INTERMEDIATE/ LATE	Chronic pain, headache Restricted motion	Anterior disc displacement, non-reducing Marked disc thickening Abnormal bone contours	Degenerative remodeling of bony surfaces Osteophytes Adhesions, deformed disc without perforation
V. LATE	Variable pain Joint crepitus Painful function	Anterior disc displacement, non-reducing with perforation and gross disc deformity Degenerative osseous changes	Gross degenerative changes of disc and hard tissues; Perforation Multiple adhesions

* refers to disc position in relation to the condyle when the mouth is open

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