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Internal Derangement of the Temporomandibular Joint
New Perspectives on an Old Problem

Howard A. Israel, DDS*

KEYWORDS
- Temporomandibular joint • Internal derangement • Classification system • Cause • Synovitis • Osteoarthritis • Arthroscopy

KEY POINTS
- Internal derangement of the temporomandibular joint is not a disease, but a nonspecific sign of tissue failure leading to biomechanical dysfunction of the joint.
- Establishing the cause of the internal derangement is essential, because successful management must be based on the underlying cause of the pathologic process.
- Major categories of disease that cause temporomandibular joint internal derangement include inflammatory/degenerative arthropathy caused by joint overload, systemic arthropathy making the joint susceptible to tissue failure, atypical localized arthropathy (disorder localized to 1 temporomandibular joint), and false arthropathy (signs and symptoms that simulate internal derangement but are caused by extra-articular disorders).
- Minimally invasive operative arthroscopy is indicated when signs and symptoms persist, and often provides essential information on the cause of disease.
- Arthroscopic temporomandibular joint surgery permits biopsy of intra-articular disorders and is successful in reducing pain, increasing range of motion, and improving mandibular function, particularly in patients with inflammatory/degenerative arthropathies.

INTRODUCTION
Internal derangement of a synovial joint is not a disease. The biomechanical joint dysfunction that is associated with internal derangement represents a failure of the intra-articular tissues caused by the loss of the structure and function. Identifying the cause of the breakdown of the tissues within a synovial joint that leads to internal derangement is an important component of successful treatment. Clinicians must ask what disease process is causing the tissue breakdown. Is there a history of acute or chronic trauma to the joint? Is there a systemic disorder that is contributing to the breakdown of connective tissues? Is there an infection or a tumor present that is causing the nonspecific symptoms of internal derangement? A clear understanding of this concept by clinicians is essential and has significant implications on patient management and the outcome of therapy. On review of the literature on temporomandibular joint disorders over the past 35 years, the problem of internal derangement of the temporomandibular

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joint is often the central focus of the diagnosis and management of patients with orofacial pain caused by temporomandibular disorders (TMDs). Clear guidelines for diagnosis and management of internal derangement of the temporomandibular joint are often elusive, although there has been much excellent research on the validation of classification systems, such as the Research Diagnostic Criteria for TMDs [DC] for TMDs and the Wilkes Staging System for temporomandibular joint disorders. For any given diagnosis, there are multiple management options that have been recommended, including no treatment, nonsurgical therapies, minimally invasive surgical procedures (arthrocentesis, arthroscopy), arthroplasty (repair of intra-articular tissues), discectomy, and total joint replacement. The main focus of this article the concept of internal derangement and temporomandibular joint disorders from a new perspective, based on clinical research, basic science research on synovial joint pathophysiology, and the principles of diagnosis and management from the perspective of the specialties of rheumatology and orthopaedics. This information ultimately leads to new concepts in the classification of internal derangement based on cause and pathophysiology, and leads to new perspectives on the management and treatment of internal derangement of the temporomandibular joint.

CURRENT CLASSIFICATION SYSTEMS FOR TEMPOROMANDIBULAR JOINT DISORDERS

The Research Diagnostic Criteria (RDC) for TMDs, published in 1992, was an excellent first step in helping to standardize diagnostic categories of TMDs. The RDC have undergone extensive testing and much research has led toward validating this classification system for TMDs to enable clinical research investigators to use the same system. This progress has improved the overall ability to develop further insights into epidemiology, diagnostic categories, causes, and ultimately treatment/management of these disorders. The original investigators recognized that many of these patients had high levels of psychosocial stress along with the physical aspects of their disease, and so this diagnostic system included AXIS I, a classification system of the physical categories of TMDs, and AXIS II, a classification system of the psychosocial behavioral aspects of patients who develop these disorders. Following years of research involving validity testing of the RDC, more recently it became apparent that updates were necessary in this system to include a larger variety of disorders of the temporomandibular joint and surrounding structures. Thus recent changes in this classification system have been made, ultimately combining the RDC (recently changed to DC for TMD) with the American Association of Orofacial Pain (AAOP) Taxonomic Classification, which encompasses a larger and more accurate description of the variety of diseases affecting the temporomandibular joint and surrounding structures. Although it is beyond the scope of this article to review the details of the most current DC/TMD and AAOP taxonomic classification systems (Fig. 1), many of the classification categories describe nonspecific signs and symptoms, and not a disease process.

The Wilkes Staging System for internal derangement is frequently used by oral and maxillofacial surgeons and helps to provide a guide for treatment based on the severity of the damage to the joint. This system includes 5 stages with stage I being a painless disc displacement with reduction and stage V being an advanced disc displacement with severe degenerative changes, adhesions, subchondral bone changes, and disc perforation (Box 1). Because the main focus of the Wilkes

Fig. 1. AAOP taxonomic classification and DC for TMDs.
system involves categorizing the extent of damage there is to joint tissues, it is useful to oral and maxillofacial surgeons in planning the operative procedure that they perceive will best treat the patient. However, in spite of the precise description of the various stages described for internal derangement, there is no corresponding information on the causal diagnosis associated with these stages.

From the standpoint of treating clinicians, there are flaws in these current classification systems that may further confuse diagnosis, cause, and ultimately treatment and management of these patients. For the most part, both of these systems are descriptive of a compilation of signs and symptoms and there is no useful categorization of cause and pathogenesis. For example, merely categorizing a patient with a disc displacement with or without reduction describes a sign of a disease process without any information as to the cause of the condition. The importance of this cannot be overstated, because many treatments over the past 35 years have focused on trying to recapture the disc. Oral repositioning appliances, mandibular manipulation, disc repositioning surgery, and disc replacement surgery have been the focus of most treatments for internal derangement. Without knowledge of the underlying cause, these treatments often fail, because causative factors persist. For example, if a patient has internal derangement associated with a systemic arthropathy, failure to treat and manage the systemic disorder is likely to result in persistent symptoms and treatment failure. Patients with excessive joint overload from mandibular parafunction ultimately fail disc repositioning treatment because of the physiologic effects of joint overload on the intra-articular tissues. Further detailed discussion on the importance of identification and categorization of causal factors is provided later in this article.

Another intriguing factor in the development of the RDC for TMD is AXIS II, which is an essential component of the diagnosis. Clinicians must ask whether there is something unique about the temporomandibular joint that makes a psychosocial categorization model a component of the diagnosis. Do diagnostic classification systems for other synovial joints and musculoskeletal conditions include a psychosocial component as an integral part of the diagnosis?

A literature search of the orthopaedic and rheumatologic literature on diagnostic systems failed to find that AXIS II psychosocial classification is an integral part of disorders of the knee, hip, shoulder, patella-femoral pain and dysfunction, cervical spine, and lower back. The American College of Rheumatology has classification systems for osteoarthritis, systemic sclerosis, and other rheumatologic diseases and a psychosocial component is not part of the diagnostic categorization of these conditions.

This is not to suggest that the psychosocial aspects of these diseases are not important. All chronic pain conditions have accompanying AXIS II diagnoses because chronic pain and loss of function affect quality of life. The impact of the disease and associated pain with loss of normal

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**Box 1**

Wilkes staging of internal derangements

| Stage I: early | Painless clicking, anterior disc displacement with reduction |
| Stage II: early-intermediate | Clicking with intermittent pain and locking, anterior disc displacement with reduction |
| Stage III: intermediate | Pain, joint tenderness, frequent and prolonged locking, restricted motion, anterior disc displacement with or without reduction, no degenerative changes |
| Stage IV: intermediate/late | Chronic pain, restricted motion, no clicking, anterior disc displacement without reduction, degenerative bony changes, adhesions |
| Stage V: late | Variable pain, painful/reduced function, crepitus, anterior disc displacement without reduction, advanced degenerative bony changes, gross disc deformity and/or perforation, advanced adhesions |

Adapted from American Society of Temporomandibular Joint Surgeons. Guidelines for diagnosis and management of disorders involving the temporomandibular joint and related musculoskeletal structures. 2001.
function must be an essential factor for clinicians to consider in the overall management of the patient. Perhaps the failures in appropriately and successfully treating many TMDs has led to patients with chronic pain, loss of function, and frustration because failed treatments have a profound effect on quality of life and result in psychological conditions such as depression and anxiety, which are common in those patients who seek treatment of TMDs. Regardless of whether preexisting psychosocial factors play an important role in causing the symptoms associated with TMDs or whether they are the result of the disease process itself, psychosocial factors must be addressed when considering the overall management of the patient. However, because psychosocial issues play an important role in most chronic disease entities, there is the need for primary treating clinicians to assess whether appropriate referral to specialists in psychology, psychiatry, and stress management is indicated.

INTERNAL DERANGEMENT OF THE TEMPOROMANDIBULAR JOINT: DEFINITION FROM AN ORTHOPAEDIC PERSPECTIVE

The most popular definition of internal derangement of the temporomandibular joint has generally alluded to joint dysfunction associated with an abnormal disc position. The Merck Manual describes internal derangement of the temporomandibular joint as a condition with damage to the internal structures of the joint and “the most common form of internal temporomandibular joint derangement is anterior misalignment or displacement of the articular disc above the condyle.” Moli

nari and colleagues defined internal derangement as follows: “the term derangement refers to an alteration in the normal pathways of motion of the TMJ [temporomandibular joint] that largely involves the function of the articular disc.” A definition of internal derangement of the temporomandibular joint that has been widely used by the dental profession for the past 4 decades describes a disruption of the internal aspects of the joint involving displacement of the disc from a normal functional relationship between the condyle of the mandible and the articular eminence of the temporal bone. This definition has been widely accepted in the dental profession, but perhaps would not be accepted if temporomandibular joint disorders were treated by specialists in orthopaedics or rheumatology.

The specialties of orthopaedics and rheumatology have a much broader definition for internal derangement of a synovial joint. For example, these specialties describe knee internal derangement as an intra-articular disorder caused by damage to internal structures within the joint. These conditions are usually caused by trauma and result in ongoing signs and symptoms of pain, instability, or abnormal mobility. Internal derangement is an old term that is nonspecific and requires a detailed history, physical examination, and diagnostic images to more clearly diagnose the condition. The classic textbook Campbell’s Operative Orthopaedics, 12th Edition, defines internal derangement of the knee as follows:

The term internal derangement...loosely applied to a variety of intra-articular and extra-articular disturbances, usually of traumatic origin, that interfere with the function of the joint.

I choose to use a more orthopaedic and rheumatologic approach to the term, and thus all references to internal derangement of the temporomandibular joint in this article use the following definition.

A condition in which there are damaged intra-articular tissues leading to disturbances in the biomechanical functioning of the temporomandibular joint.

Based on this broader definition, anterior disc displacement is considered one type of internal derangement without exclusivity. Thus, a patient who has severely limited opening with decreased translation of the temporomandibular joint caused by adhesions, osteoarthritis, synovial inflammation, disc displacement, or other intra-articular disorder is also considered to have the clinical signs and symptoms of internal derangement.

IS INTERNAL DERANGEMENT A DISEASE?

Mosby’s Dictionary of Medicine, Nursing & Health Professions, Ninth Edition, defines the term disease as follows: “a specific illness or disorder characterized by a recognizable set of signs and symptoms attributable to heredity, infection, diet or environment.” A key aspect of this definition of disease is that there is an abnormal function or process involving an organ and/or system with characteristic symptoms that have a specific cause. Internal derangement of the temporomandibular joint represents signs and symptoms of altered biomechanical function (failure of translation, locking, intermittent locking, clicking) with damaged intra-articular tissues without alluding to a specific cause. The signs and symptoms associated with internal derangement are nonspecific and can be caused by a multitude of disease conditions. Therefore, internal derangement should not, by itself, be considered a disease, but should be considered a manifestation of a process in which there is damage to intra-articular
tissues from a specific cause that must be identified by the clinician. The variety of disease categories that commonly cause internal derangement is further described later in this article.

Further complicating the understanding of internal derangement are the current diagnostic classification systems that are used in the staging of temporomandibular joint disorders. The DC/TMD classification system uses physical diagnosis to further subclassify temporomandibular disease into muscle disorders, disc disorders, arthritic disorders, hypermobility disorders, and tension headaches. However, these disorders mostly represent signs and symptoms that are nonspecific and are not mutually exclusive.

The Wilkes classification system also focuses on the progressive stages of internal derangement, ultimately leading to failure of normal joint function. These progressive changes leading to loss of the structure and function of the cartilage, synovium, and subchondral bone represent the end result of a disease process. Both of these classification systems are not based on the causal conditions that lead to failure of the joint tissues and loss of normal joint function.

Understanding the true cause of failure of the joint tissues is essential for proper treatment, prevention, and/or delay in the progression of joint disease. Stegenga recognized this deficiency in the current classification of temporomandibular joint disorders and proposed a system based on the pathologic process that is causing the structural failure of the joint tissues. This proposed change in nomenclature emphasizes the importance of the diagnosis in providing the basis for treatment. Therefore, the author proposes control of risk factors that lead to pathologic intra-articular structural changes, reducing pain and improving function rather than attempting to control the position of the disc, as essential components of patient management. For clinicians to truly understand and manage the variety of disease conditions that affect the temporomandibular joint, it is important to understand that our current classification systems describe nonspecific stages of a disease process, without being specific for the true diagnosis that is responsible for the failure of the joint tissues.

The detailed review of the problem of internal derangement of the temporomandibular joint later in this article clearly shows that internal derangement is not a disease, but represents a variety of stages of biomechanical failure of the joint tissues, which can be caused by several specific disease entities.

**DOES INTERNAL DERANGEMENT REQUIRE TREATMENT?**

*Historical Perspectives*

A review of the history of treatment of internal derangement of the temporomandibular joint reveals changing perspectives on management. In the 1970s and 1980s, internal derangement was viewed as a mechanical problem, resulting in mechanical attempts at repositioning or replacing the disc. In 1979, McCarty and Farrar published an article in the *Journal of Prosthodontics* that emphasized the importance of disc displacement as a major disorder of the temporomandibular joint. It was thought that the failure to have the disc in the proper position between the condyle and the articular eminence inevitably leads to severe degenerative joint disease. Thus, repositioning the disc became a central focus for many clinicians in the dental profession. Oral appliances designed to reposition the disc, as well as mandibular manipulations, were considered mainstays of conservative therapy.

Patients who had persistent symptoms and internal derangement were often referred to oral and maxillofacial surgeons and there were a variety of surgeries designed to solve the problem of internal derangement of the temporomandibular joint. Discoplasty, involving disc repositioning surgery, was a common surgical procedure. Discectomy was often performed if there was a perforation in the disc. A variety of tissues were used for disc replacement, including ear cartilage, temporalis muscle, temporalis fascia, Silastic, and Proplast-Teflon. However, the use of Proplast-Teflon led to foreign body reaction and destruction of articular tissues.

In the 1990s, the realization that arthroplasty with disc repositioning or disc replacement often resulted in degenerative changes and fibrosis and did not reliably maintain a repositioned disc resulted in a significant change in the surgical management of patients with severe symptoms and internal derangement. Arthroscopic temporomandibular joint surgery was shown to be a safe and effective alternative to arthroplasty, which reliably reduced pain and improved maximum interincisal opening distance.

Excellent results were achieved with arthroscopic surgery without changing disc position. MRI studies have shown a high percentage of disc displacement in asymptomatic patients (32%–38%) and this has raised further questions about the importance of internal derangement in patients with symptomatic temporomandibular joint disease.
Arthrocentesis\textsuperscript{32–35} was introduced as another minimally invasive treatment of internal derangement and has been shown to be safe and effective. A major advance in the understanding of the pathogenesis of temporomandibular joint disease occurred as a result of arthroscopy and arthrocentesis and resulted in research on biochemical mediators in the synovial fluid. Mediators of inflammation, cartilage degradation, and adhesion formation have been identified, which represent the biochemical basis for destruction of joint tissues leading to internal derangement.\textsuperscript{36–49} Synovial fluid research has continued since the 1990s and has offered promising strategies for the identification of biochemical markers of disease and the potential for new therapies designed to alter or block pathogenic mechanisms.

**Internal Derangement Treatment: Clinical Research Results**

Clinical research on the natural course of internal derangement without treatment\textsuperscript{50–52} has shown the following:

- Most patients improve without any treatment
- The length of time for symptoms to resolve is variable, but generally a minimum of 1 year
- A percentage (25\%–33\%) of patients do not improve
- Older patients and those with MRI evidence of more advanced disease (osteoarthritis and advanced internal derangement) are at higher risk for not improving spontaneously

There have been a variety of good evidence-based literature reviews and studies\textsuperscript{53–58} that compared the results of nonsurgical treatments of internal derangement. Nonsurgical therapies that have been studied include patient education, nonsteroidal antiinflammatory drugs (NSAIDs), muscle relaxants, hot/cold packs, mouth opening exercises, softer diet, and occlusal appliances.

Because appliance therapy is often the initial treatment intervention for patients who develop temporomandibular joint symptoms, knowledge of the evidence-based literature on occlusal appliances is necessary for clinicians. Lundh and colleagues\textsuperscript{55} compared treatment outcomes on patients with anterior disc displacement without reduction who were placed into one of 2 groups. The first group was treated with an occlusal splint and the second group had no treatment. The results of treatment after 12 months were as follows:

- Pain disappeared in approximately 33\% of patients in both groups
- Increased joint pain was experienced by 40\% of the occlusal splint group, and 16\% of the no-treatment group after 12 months

The investigators concluded that there was no significant benefit in occlusal splint therapy compared with no treatment.

Truelove and colleagues\textsuperscript{56} evaluated treatment outcomes in 200 patients with anterior disc displacement with reduction, arthralgia, and myalgia. The patients were randomly assigned to one of 3 treatment groups:

- Usual treatment, which included self-care, education, NSAIDs, hot/cold packs, and passive stretching
- Hard flat plane splint and usual treatment (as described earlier)
- Soft splint and usual treatment

Treatment outcomes were evaluated at 3 months and 12 months, which revealed no significant difference in all 3 groups. The investigators concluded that self-care, low-cost therapy is as effective as occlusal splint therapy.

Clark and Minakuchi\textsuperscript{57} provided an excellent review of the evidence-based literature on appliance therapy and their conclusions on occlusal stabilization appliances were:

- Occlusal stabilization appliances decrease symptoms of myalgia and arthralgia
- They protect the dentition from wear caused by parafunctional habits
- They are low risk as long as they are not worn 24 hours a day
- They do not change disc position

Clark and Minakuchi\textsuperscript{57} also concluded that appliances that are designed to reposition the mandible do not change disc position.

Based on current research, clinicians should follow a common-sense approach concerning appliance therapy. Appliances should be designed to provide a buffer between the maxilla and the mandible, in theory to offset the forces of mandibular parafunction and to reduce the load on the temporomandibular joints. Therefore, occlusal stabilization appliances that equally distribute these forces throughout the arch should be considered as an appropriate initial treatment of myalgia and arthralgia. However, clinicians must continuously evaluate the patient’s response to treatment. Some patients with a significant parafunctional habit develop increased parafunction with an appliance. Patients often tell clinicians that they find themselves clenching on the appliance and that their jaw muscles are more sore in
the morning, following the nighttime use of an occlusal stabilization appliance. For patients with daytime clenching, an appliance can be used for 1 minute, to assist in self-awareness with the goal of breaking the habit. Continuous and/or excessive use of an appliance can contribute to the development of a malocclusion and must be avoided, particularly for appliances that provide partial coverage of the dentition, and thus these appliances are to be avoided. In addition, appliances that reposition the mandible in an attempt to recapture the disc in patients with arthralgia and/or internal derangement should be avoided, because the scientific literature does not support this. Most importantly, patients treated with appliance therapy must continuously be evaluated to determine whether the appliance is effective in reducing myalgia and arthralgia. If appliance therapy is not effective in reducing symptoms, the clinician must reevaluate the diagnosis and alter the therapeutic regimen.

Further complicating the understanding of response to treatment is the placebo effect of all therapeutic interventions. Greene and colleagues studied placebo responses to orofacial pain and reported that “present knowledge suggests that every treatment for pain contains a placebo component, which sometimes is as powerful as the so-called active counterpart.” A summary of the results of evidence-based studies on nonsurgical therapy for internal derangement is listed here:

- Most patients have improvement in signs and symptoms with time
- No significant differences between treatment and nontreatment groups
- Palliative care (NSAIDs, education, diet modification, exercises) seem to be as effective as more costly appliance therapy
- Occlusal appliances do not change disc position
- Occlusal stabilization appliances may reduce myalgia and arthralgia
- Although patients with internal derangement improve with time, the length of time for symptoms to improve is not clearly identified
- All treatments have a powerful placebo effect

Patients with internal derangement with severe symptoms of pain and dysfunction who have failed nonsurgical therapy are often candidates for surgical treatment. Laskin provided an excellent review of evidence-based research on the surgical management of internal derangement. Clinical research on the results of surgical treatment of internal derangement is summarized here:

- There are no prospective, randomized controlled, double-blinded trials; only case series, and comparison of preoperative and postoperative signs and symptoms
- Arthroscopy, arthrocentesis, discoplasty, and discectomy have all been reported to have reasonably good success with reduction in signs and symptoms in the range of 80% to 90%
- Surgical success is highest with the first surgery, and each surgical procedure reduces the success rate
- Surgical failure is often caused by lack of control of causal factors such as joint overload
- When surgery is indicated, the least invasive approach is recommended

Because of the lack of randomized controlled studies on surgical management of internal derangement, Reston and Turkelson performed a meta-analysis of the results of surgical treatment of disc displacement without reduction. This biostatistical approach studied many reported surgical trials to help compensate for the lack of parallel control groups. The investigators concluded that only arthroscopic surgery and arthrocentesis showed effectiveness significantly greater than all assumed control group improvement rates. Al-Moraissi performed a systematic review and meta-analysis comparing arthroscopy and arthrocentesis for management of internal derangement. The results suggested that arthroscopy yielded superior efficacy to arthrocentesis in increasing joint movement and decreasing pain.

**Does Any Surgical Procedure Reposition and Maintain a Normal Disc Position?**

The literature on surgical outcomes assess pain relief, improved function, and increased interincisal opening distance, but do not show the maintenance of normal disc position. Zhang and colleagues assessed the postoperative disc position following discoplasty and disc stabilization with bone anchors. The investigators reported 96% successful disc repositioning based on MRI scans taken 7 days postoperatively. However, conclusions based on a early postoperative MRI do not provide information about patients who function and load their temporomandibular joints. Therefore, based on a review of the literature, there does not seem to be any evidence that surgically repositioning a disc maintains the disc in a normal position; there does not seem to be any evidence that any procedure, treatment, or appliance repositioning and maintains the disc in a normal position.
Thus the major goals of treatment of internal derangement should not be to reposition the disc, but should be to:

- Establish the diagnosis and the cause of the internal derangement
- Reduce inflammation
- Reduce pain
- Reduce joint overload
- Improve range of motion
- Restore mandibular function
- Identify and control causal factors

MANAGEMENT OF TEMPOROMANDIBULAR JOINT DISORDER BASED ON DISEASE CAUSE: NEW PERSPECTIVES ON INTERNAL DERANGEMENT AS A SIGN OF DISORDER

The DC for TMDs and the Wilkes staging of internal derangement are helpful for clinicians in assessing the extent to which a pathologic process has caused damage to the intra-articular tissues, resulting in biomechanical failure and/or compromise in joint function. However, these classification systems do not provide information on factors that cause damage and dysfunction of joint tissues leading to internal derangement. Thus, merely repositioning a disc ultimately leads to joint failure if the causal factors are not recognized and managed. The classification of intra-articular temporomandibular joint disease based on causal factors is discussed here, and is intended to provide clinicians with a different perspective in the management of temporomandibular joint internal derangements. Internal derangement should be viewed by clinicians as a sign of a disease process leading to biomechanical compromise or failure of the temporomandibular joint. The challenge for clinicians is to diagnose the condition that is causing the internal derangement. Once identified, the basis for treatment is to reduce the patient’s symptoms while simultaneously identifying and managing the disease process. The following major categories of disease can cause internal derangement of the temporomandibular joint (Fig. 2):

1. Inflammatory/degenerative arthropathy: joint overload (acute and/or chronic) leading to inflammation and degeneration of intra-articular tissues
2. Systemic arthropathy: systemic disorder causing temporomandibular joint disease
3. Localized atypical arthropathy: intra-articular temporomandibular joint disorder that is atypical and not caused by joint overload
4. False arthropathy: extra-articular disorder simulating and/or causing temporomandibular joint symptoms

Inflammatory/Degenerative Arthropathy: Pathogenesis

Chronic joint overload is the most common cause of internal derangement of the temporomandibular joint. There is a significant body of research on temporomandibular joint synovial fluids and arthroscopic tissue morphology, which has shown that synovitis, osteoarthritis, and adhesions are the major tissue changes that occur in symptomatic patients requiring arthroscopic surgery. Chronic joint overload, often caused by mandibular parafunction, results in a change in articular cartilage metabolism, with degradation of the cartilaginous matrix exceeding production. This overload of the cartilage upsets the balance between the buildup and degradation of the cartilage matrix, ultimately resulting in a breakdown of

Fig. 2. Major categories of temporomandibular joint (TMJ) disease based on cause.
the cartilaginous surfaces. In the earliest stages of this pathologic process, fibrillation of articular cartilage is seen arthroscopically (Fig. 3). This fibrillation ultimately results in biomechanical failure impairing the sliding of articular surfaces. The clinical correlation with this early failure of articular cartilage is joint noise (clicking and/or crepitus). Tissue changes that occur in the cartilage impair the sliding ability of the joint, often resulting in a change in disc position (see Fig. 3). These early degenerative changes do not necessarily cause pain. If there is no associated inflammation, the patient may function with a clicking joint and no major functional disability. This possibility may explain why a significant percentage of the population (32%–38%) who are without complaints and totally functional have disc displacement, which can be seen on MRI.28–31

Individuals who have severe and persistent mandibular parafunction continue to load the intra-articular tissues beyond their adaptive capacity, leading to further changes in the structure and function of these tissues. Continued cartilage degradation results in significant osteoarthritis and can ultimately lead to a disc perforation (Fig. 4). The alteration in joint biomechanics often leads to loading of the synovial tissues, which is not what the synovium normally experiences. The synovium is a connective tissue that is very vascular and is well innervated, unlike articular cartilage. The major function of synovium is the production of synovial fluid, which is necessary for joint lubrication and also for nutrition of chondrocytes in the articular cartilage, which does not have a blood supply. Thus, loading of the synovial tissues results in a significant escalation in symptoms because:

1. The synovial membrane becomes inflamed, erythematous, and edematous, which results in the clinical appearance of synovitis (Fig. 5)
2. The abnormal loading of the synovial tissues causes pain, because this tissue has a nerve supply and does not normally undergo loading
3. Inflamed synovium impairs production of synovial fluid, impairing lubrication of the joint, further altering the biomechanics of the joint and reducing the ability of the temporomandibular joint to slide
4. Once synovitis develops in the temporomandibular joint, it is difficult to resolve, because this joint is constantly being used, resulting in further loading and further synovial inflammation

With the onset of an acute synovitis, patients have a noticeable increase in temporomandibular joint symptoms:
1. Acute pain localized to the temporomandibular joint
2. Reduced translation of the affected joint with limited maximum interincisal opening distance, deviation of the mandible to the affected side with opening, and reduced lateral excursion to the contralateral side
3. If there is significant intra-articular swelling associated with the synovitis, there may be an alteration in the occlusion with an ipsilateral posterior open bite and deviation of the mandibular midline to the contralateral side at rest
4. Myospasm of the surrounding muscles of mastication (masseter and temporalis become significantly tender to palpation) as the body attempts to splint the injured joint
5. MRI shows a synovial effusion best seen on the T2 images and anterior disc position (Fig. 6)

Patients who develop acute synovitis of the temporomandibular joint that does not resolve with nonsurgical therapies such as joint unloading (oral appliances, diet modification), anti-inflammatory medications, and muscle

![Fig. 3. Chronic joint overload: degradation exceeds repair.](image-url)
relaxant medications often transition to a chronic synovitis of the temporomandibular joint. The continued loading of inflamed synovial tissues combined with reduced mobilization often results in adhesions, which also affects the ability of the mandible to translate. Patients with chronic synovitis of the temporomandibular joint often present with a history of acute locking and pain, followed by a period of gradual reduction in pain and a slight increase in the maximum interincisal opening distance over several months. However, these chronic changes occur at the expense of greatly reduced masticatory function. Patients complain that they cannot open their mouths widely and they can only eat soft foods. If these patients do not pursue surgical treatment designed to reduce inflammation, remove adhesions, and increase mandibular mobility, they may eventually develop less pain, with reduced synovial inflammation, with persistent reduction in mandibular range of motion because of adhesions (Fig. 7).

The simultaneous occurrence of synovitis and reduced mobility leads to the development of adhesions in the synovial joint. If the pathologic process continues with persistence of synovitis, cartilage degradation, reduced mobility, and adhesion formation, ultimately this may lead to a fibrous and/or bony ankylosis.

There are some patients with synovitis and internal derangement of the temporomandibular joint who do not progress to further joint disease and eventually return to normal masticatory function. However, clinicians do not have the capacity to predict which patients are likely to have a return to asymptomatic function and it is not clear why some patients have this healing capacity. Clinicians can speculate on those factors that may lead to a resolution of symptoms. Theoretically, if the clinician is successful in reducing joint overload, maximizing joint mobility, and reducing inflammation, this may create an intra-articular environment that is more likely to heal, given

**Fig. 4.** Arthroscopic view of left TMJ in patient with severe masticatory parafunction. Osteoarthritis with disc perforation and exposed condyle. *Black arrow* indicates disc perforation.

**Fig. 5.** The synovial membrane becomes inflamed, erythematous, and edematous, which results in the clinical appearance of synovitis.
enough time. Clinical arthroscopic observations have shown that some joints with anterior disc displacement have remodeled retrodiscal tissue that has the white appearance of cartilage but clearly is not disc, because of the presence of blood vessels that can be seen in the tissue (Fig. 8). Recent research showed that increased mechanical loading on the disc increased protein levels and proteoglycan messenger RNA expression in temporomandibular joint discs in a rat model. It is likely that functional loading of the retrodiscal synovial tissues has the capacity to result in stimulation of the production of proteoglycans, leading to the formation of tissue that has the appearance and function of cartilage.

In patients with internal derangement caused by overload associated with a chronic inflammatory/degenerative arthropathy, it is also important to realize that the maintenance of the structure and function of synovial and cartilaginous tissues is interdependent. Inflamed synovial tissues and adhesions lead to reduced joint motion, decreased pumping action of the synovial fluid, and a decrease in the nutrition of chondrocytes. Failure to maintain the viability and function of chondrocytes results in the loss of the matrix of the cartilage (collagen and proteoglycans), resulting in further cartilage degradation. This degradation leads to further progression of degenerative joint disease (osteoarthritis) within the joint. Joints with persistent degradation of articular cartilage have increased levels of degradation products (glycosaminoglycans) in the synovial fluid, which can overwhelm the phagocytic function of the synovial membrane and lead to further synovial dysfunction and synovitis. It has been shown that osteoarthritis and synovitis occur simultaneously in symptomatic temporomandibular joints that have undergone arthroscopic surgery. Thus the inflammatory and degenerative processes that occur in temporomandibular joints that are chronically overloaded occur simultaneously, resulting in the alteration of the intra-articular tissues, leading to biomechanical failure and internal derangement (Fig. 9).

**Treatment of Inflammatory/Degenerative Arthropathy**

A key principle in the management of internal derangement caused by an inflammatory/degenerative arthropathy is for clinicians to realize that the internal derangement is the end result of damaged intra-articular tissues usually caused by chronic overload. Therefore, identification and management of factors that contribute to the failure of the synovium and articular cartilage are essential for a favorable outcome. Once the diagnosis of inflammatory/degenerative arthropathy...
with concurrent joint overload is established, essential principles of management include:

1. Reduction of joint loading
2. Maximizing joint mobility
3. Reduction of inflammation
4. Control of pain

Clinicians who treat these patients are well versed in nonsurgical therapies to accomplish these goals, including:

1. Diet modification
2. Awareness and control of mandibular parafunction
3. Passive-motion exercises
4. Physical therapy
5. Masticatory muscle massage and heat
6. Occlusal stabilization oral appliances (appliances that provide equal distribution of forces throughout the arch and do not attempt to reposition the mandible)
7. Antiinflammatory medications
8. Muscle relaxant medications
9. Pain management modalities
10. Improving sleep

Patient education is an essential part of management and it is important that these patients become partners in their care, focused on control of causal factors that are contributing to the disease process.

Patients often present to the oral and maxillofacial surgeon with symptoms associated with internal derangement. Clinicians must first approach these patients with an open mind with the realization that internal derangement is a nonspecific sign of intra-articular disorder that can have a variety of diagnoses. Thus, establishment of the diagnosis and the cause of the joint disorder is

![Remodeled Retrodiscal Tissue](image1)

**Fig. 8.** Retrodiscal and other joint tissues have the capacity to adapt to functional loads.

![Retrodiscal and Other Joint Tissues](image2)

**Fig. 9.** The response (or lack thereof) to nonsurgical management determines whether the joint has adaptive capacity and dictates future treatment.
Internal Derangement of the Temporomandibular Joint

essential. If the patient has signs and symptoms caused by joint overload (chronic or acute) and if all other causal diagnoses are ruled out, an inflammatory/degenerative arthropathy is likely. These patients are placed on an intense 2-week to 3-week regimen of nonsurgical therapy as described earlier. If the symptoms begin to resolve, then nonsurgical management is continued (see Fig. 9). However, if the symptoms persist, minimally invasive surgical intervention is often required.

The least invasive procedure that will resolve the symptoms is the treatment of choice; therefore, arthroscopic surgery (or arthrocentesis) is performed.25,27 A major advantage of arthroscopic surgery compared with arthrocentesis is the ability to directly see the disorder and to obtain specimens for histopathologic examination to further confirm the diagnosis. Furthermore, arthroscopic surgery involves the removal of pathologic tissue, lysis of adhesions, direct injection of medications into the synovium, and debridement of degenerative articular cartilage. Arthrocentesis requires less surgical skill and is performed at a lower cost but does not permit direct visualization and removal of pathologic tissue. Patients with symptoms of limited opening for greater than 3 months often have intra-articular adhesions, and arthrocentesis is not as effective as arthroscopy, which permits removal of these adhesions.

A major challenge to the specialty of oral and maxillofacial surgery is to properly train interested clinicians in the required skills necessary to perform arthroscopic surgery. Failure to train this specialty will ultimately prevent these patients from having access to minimally invasive temporomandibular joint surgery. Furthermore, clinicians in other fields, without advanced training in oral and maxillofacial surgery, will attempt to develop the skills for arthroscopic surgery, to the detriment of oral and maxillofacial surgery and of patients. The skills required for arthroscopic surgery are unique and thus it is necessary for experienced surgeons to train the next generation of oral and maxillofacial surgeons. Although many advanced education programs in oral and maxillofacial surgery do not provide training in arthroscopy, this should not be a justification for performing more invasive surgery when arthroscopy is indicated. Excellent hands-on training courses do exist for those oral and maxillofacial surgeons who want to provide arthroscopic temporomandibular joint surgery as an effective minimally invasive option for their patients.68–70

Regardless of which minimally invasive modality is performed, a postoperative rehabilitation regimen with control of causal factors is essential. This regimen is the same as the nonsurgical regimen that was prescribed before the decision to perform minimally invasive surgery. Aside from the reduction of joint loading, it is essential for patients to perform passive-motion exercises several times daily for a minimum of 2 months postoperatively. Mobilization of the mandible with gentle stretching prevents the formation of adhesions, which are removed during arthroscopic surgery, and helps to stimulate the synovial fluid to provide nutrition to articular cartilage chondrocytes. Passive mobilization exercises should be started within 24 hours of surgery and are repeated 3 to 4 times daily, with each session lasting 15 minutes. Physical therapy can be added to the postoperative regimen, but this must not be a replacement for the daily passive-motion exercises that the patient performs at home.

The treatment of an inflammatory/degenerative temporomandibular joint arthropathy varies based on the stage of the internal derangement and the disease process. The early stages of internal derangement (Wilkes I and II) can often be managed with nonsurgical therapy. More advanced stages of internal derangement with persistence of significant symptoms, in spite of appropriate nonsurgical therapy, are treated with minimally invasive surgery (arthroscopy or arthrocentesis) if diagnostic imaging confirms the presence of a joint space. Advanced stages of internal derangement leading to fibrosis and/or ankylosis with loss of joint space require arthrotomy. Fig. 10 shows the treatment algorithm used for the various stages of internal derangement caused by inflammatory/degenerative arthropathies.

**Systemic Arthropathy: Systemic Disorders Causing Temporomandibular Joint Disease**

Systemic diseases can often contribute to temporomandibular joint disorders. In the case of an inflammatory/degenerative temporomandibular joint arthropathy, excessive joint loads exceed the adaptive capacity of the intra-articular tissues, resulting in cartilage degradation and synovial inflammation, which then cause internal derangement. Patients with a systemic arthropathy are those with joint tissues that will fail with normal joint loads, because the systemic disorder affects the structure and function of the intra-articular connective tissues. Therefore, internal derangement of the temporomandibular joint can be caused by a systemic disorder and it is essential for oral and maxillofacial surgeons to be aware of this when considering patient management. Examples of systemic disorders that can cause internal derangement include rheumatoid arthritis, psoriatic arthritis, juvenile idiopathic arthritis, ankylosing spondylitis, systemic lupus erythematosus, osteoarthritis, rheumatic arthritis, osteoarthropathy, and gout.
arthritides, Lyme disease, polymyalgia rheumatica, chondrocalcinosis, Ehlers-Danlos syndrome, lupus, and other connective tissue disorders.

Arthroscopic surgery is often performed for obtaining pathologic tissue for diagnostic purposes, and is also helpful in managing symptoms. However, management of the systemic disorder is essential for prolonged control of the patient’s symptoms and this frequently requires coordination of treatment with a rheumatologist (Figs. 11–13).

Localized Atypical Arthropathy: Intra-Articular Temporomandibular Joint Disorder that Is Atypical and Not Caused by Joint Overload or Systemic Disease

Because the temporomandibular joint is a synovial joint, it is subject to the same local disorders as other synovial joints. A localized atypical arthropathy is uncommon for the temporomandibular joint; however, these conditions do occur in clinical practice. These patients have intra-articular temporomandibular joint disorder that is localized to 1 joint, and is not caused by systemic disease or joint overload. The clinical presentation of this group of disorders is nonspecific, but ultimately the signs and symptoms (which occur with any internal derangement) include any combination of the following:

1. Joint pain
2. Joint noise: clicking and/or crepitus
3. Altered mandibular range of motion
4. Gross changes in the occlusion

**Fig. 10.** Management of internal derangement caused by inflammatory/degenerative joint disease. a Arthroscopy is the minimally invasive treatment of choice.

**Fig. 11.** A 46-year-old woman with systemic lupus erythematosus and synovitis, disc perforation, and severe degenerative changes involving the left temporomandibular joint. (A) Synovitis posterior recess. (B) Osteoarthritis and disc perforation. (C) MRI shows degenerative changes and anterior disc position.
Clinical findings that are suggestive of a localized atypical arthropathy include a gradual change in the occlusion, with the development of an ipsilateral posterior open bite, and shifting of the mandibular midline to the contralateral side. An osteochondroma of the condyle is the most common neoplasm affecting the temporomandibular joint and is in the category of an atypical (neoplastic) localized arthropathy (Fig. 14). Diagnostic images that show unusual findings, such as multiple loose dense bodies, calcifications, or very large synovial effusions are suggestive of atypical localized arthropathies such as synovial chondromatosis, crystal deposition joint disease, and a synovial cyst (Fig. 15). Some patients present with routine signs and symptoms and the diagnosis is confirmed with arthroscopic examination and biopsy of pathologic tissue (Fig. 16). The algorithm for the diagnosis and management of patients with atypical localized arthropathies can be seen in Fig. 17. The importance of establishing the correct diagnosis cannot be overemphasized.

**False Arthropathy**

Some patients present with the signs and symptoms of temporomandibular joint internal derangement but the cause is not an intra-articular disorder. The most common example of this is the patients who develop trismus following an inferior alveolar nerve local anesthetic block. The

![Fig. 12. A 41-year-old with psoriatic arthritis and severe synovitis left temporomandibular joint. (A) Severe inflammation of the synovial membrane. (B) MRI showing anterior disc position and small synovial effusion anterior recess (arrow).](image)

![Fig. 13. A 61-year-old man with painful swelling of the right temporomandibular joint. Arthroscopic biopsy led to the diagnosis of chondrocalcinosis (pseudogout). The patient was referred to a rheumatologist for further work-up and management. (A) MRI images showing dense body (Yellow arrow) (above) and effusion (Yellow arrow) (below) in joint space. (B). Arthroscopic surgery identified the calcifications in the synovial membrane and biopsy of the tissue established the diagnosis of chondrocalcinosis.](image)
trauma to the muscle and associated hematoma formation is the cause of the limitation of mandibular opening. Infection of the deep spaces of the head and neck, as well as radiation fibrosis, are also examples of a false arthropathy which are common and easy to diagnose based on the history and clinical presentation.

False Arthropathy Caused by Neoplasia

There are some patients who present with signs and symptoms of pain and limitation of mandibular range of motion, with a history of being treated as a routine temporomandibular joint internal derangement, who ultimately are diagnosed with a neoplastic process. It is essential for oral and maxillofacial surgeons to establish the diagnosis of false arthropathy caused by a neoplastic process as early as possible. Clinician should be suspicious of a neoplastic process in patients who

Fig. 14. A 45-year-old man with osteochondroma of the left mandibular condyle. (A). Cone beam scan three-dimensional reconstruction showing osteochondroma of the left condyle. (B) Surgical specimen following condylectomy.

Fig. 15. Examples of atypical localized arthropathies affecting the temporomandibular joint. (A) Synovial chondromatosis. (B) Crystal deposition disease. (C) Synovial cyst.

Fig. 16. Arthroscopic biopsy of synovial disorder is extremely valuable in establishing the correct diagnosis. Histopathology in this case was consistent with synovial chondromatosis.
present with temporomandibular joint symptoms with the following:

1. Recent rapid weight loss
2. Cranial nerve deficit
3. Prolonged nonsurgical therapy without a typical response to treatment in patients who have not had diagnostic imaging
4. Orofacial pain that is atypical and not well localized to the temporomandibular joint

Although each case presents differently, the essential component in making the diagnosis is for clinicians to be diligent in continually reevaluating the diagnosis and response to treatment. Diagnostic imaging of the head and neck (computed tomography [CT] and MRI) is critical in making the diagnosis (Fig. 18) and it is the responsibility of the treating clinician to review the images and not depend solely on the report by the radiologist. The algorithm for management of patients who are suspected of having a false arthropathy of the temporomandibular joint is shown in Fig. 17.

**How Common Is Each Etiologic Category in the Classification of Arthropathies of the Temporomandibular Joint?**

Because an etiologic classification of temporomandibular joint disorder has not been routinely used, there is a paucity of information on the prevalence of each category of arthropathy. Preliminary research on this classification based on cause has yielded surprising results. An unpublished review of 104 consecutive patients with signs and symptoms of internal derangement and failure of response to nonsurgical treatment, who underwent arthroscopic biopsies, has been conducted. The etiologic classification was based on the history,
diagnostic imaging, arthroscopic morphology, and histopathologic findings. The results revealed the following (Fig. 19):

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory/ degenerative arthritis caused by joint overload</td>
<td>77%</td>
</tr>
<tr>
<td>Systemic arthropathy</td>
<td>13%</td>
</tr>
<tr>
<td>Localized atypical arthropathy</td>
<td>9%</td>
</tr>
<tr>
<td>False arthropathy</td>
<td>1%</td>
</tr>
</tbody>
</table>

Further investigation using an etiologic classification system is required, but even in this small sample the results are surprising. Systemic arthropathies and localized atypical arthropathies affecting the temporomandibular joint were common. A total of 23% of the cases did not have the more common inflammatory/degenerative arthropathy, and thus treatment of these patient groups may be different from treatment of those with the more common inflammatory/degenerative arthropathy (Israel H. Etiologic classification of temporomandibular joint disease in 104 consecutive patients undergoing arthroscopic surgery. Manuscript in preparation, 2015).

**SUMMARY: ETIOLOGIC CLASSIFICATION OF TEMPOROMANDIBULAR JOINT DISORDERS**

A review of the current state of knowledge of internal derangement of the temporomandibular joint and synovial joint pathophysiology leads to the conclusion that internal derangement is not a disease. The signs and symptoms associated with internal derangement are caused by loss of the structure and function of the intra-articular tissues, ultimately leading to a failure in the biomechanics of the temporomandibular joint. The cause of this tissue failure is most often joint overload, leading to tissue failure and an inflammatory/degenerative arthropathy of the temporomandibular joint. However, the intra-articular changes associated with internal derangement of the temporomandibular joint can also be caused by a systemic arthropathy or a localized atypical arthropathy involving the temporomandibular joint. In addition, there is a group of disorders that simulates the signs and symptoms of internal derangement, but are caused by extra-articular disease, and thus are classified as a false arthropathy of the temporomandibular joint.Clinicians must be diligent in establishing the correct diagnosis and cause of the internal derangement, which ultimately leads to the appropriate management of patients with these disorders.

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