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Position Paper

American Association of Oral and Maxillofacial Surgeons

The Contemporary Management of Temporomandibular Joint Intra-Articular Pain and Dysfunction

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The understanding of the causes of temporomandibular joint pain and dysfunction has evolved over 50 years. Historically, the term internal derangement has been used to describe the abnormal relationship between the articular disc, condyle and glenoid fossa, which was thought to correlate with patient symptoms. It is now known that the pathophysiology of intra-articular pain and dysfunction (IPD) involves synovitis, capsular impingement, symptomatic disc displacement or a combination of these. Symptomatic disc displacement should only be considered to be a potential source of IPD after synovitis and capsular impingement have been treated. This philosophy provides the opportunity for most patients with IPD to be initially treated nonsurgically or with minimally invasive procedures such as arthrocentesis or arthroscopy.

The paradigm for the management of temporomandibular joint (TMJ) intra-articular pain and dysfunction (IPD) has undergone a significant transformation over the last three decades as sufficient data have been reported to allow the development of evidence-based guidelines that enable both the provider and the patient to identify best practices. Recognizing the opportunity for advances in patient care, the National Academies of Science, Engineering, and Medicine (NASEM) in 2019 convened representatives from the FDA, Medical Device Epidemiology Network, patient advocacy groups, oral and maxillofacial surgeons, and orofacial pain experts to identify specific steps to improve care for patients with TMJ pain and dysfunction. The final recommendations from NASEM were released and published in 2022.[1,2] In response, the American Association of Oral and Maxillofacial Surgeons (AAOMS) created the Special Committee on Temporomandibular Joint Care whose assignment was to develop contemporary evidence-based guidelines for the management of patients with TMJ disorders, including IPD. This position paper presents the findings and recommendations of that committee.

Throughout this paper, the term IPD will be used for the variety of intra-articular conditions that result in joint pain and/or dysfunction with mandibular motion. It is recommended that this term be widely adopted as the preferred replacement for the older terms internal derangement and anterior disc displacement, which specify a mechanical finding that alone may not be causative or associated with signs or symptoms. The latter, when determined to cause pain or dysfunction, may be more accurately described as symptomatic disc displacement (SDD), which is discussed more fully within this paper.

Background

The historical approach to diagnosing the etiology of IPD was based on assessing the anatomical relationship between the mandibular condyle, the articular disc and the glenoid fossa. It was thought that disc position was critical to the development and perpetuation of IPD, ultimately leading to osteoarthritis (OA). This led to the development of the Wilkes staging system for classifying disc position, disc shape, condyle morphology and the dynamic relationship between the condyle, disc and glenoid fossa.[3,4] The Wilkes classification relies on a history and physical examination, radiographic imaging (arthrography or magnetic resonance imaging) and histological features to enable patients to be staged appropriately. The perceived mechanical etiology of IPD and OA led to the development of several surgical procedures whose purpose was to reposition, reshape or remove the disc. Arthroplasty with disc plication, discectomy and discectomy with replacement are all based on the assumption that IPD is the result of abnormal disc position.[3-11]

Understanding the cause of IPD has evolved to the point that it is now known that excessive mechanical loading can trigger a cascade of molecular events involving the TMJ synovial membrane, leading to symptoms in susceptible individuals. These events involve the production and release of free radicals, cytokines, fatty acid catabolites, neuropeptides and matrix-degrading enzymes.[12] Although the reason for excessive mechanical loading may vary among patients, there is significant evidence it results in an initial tissue hypoxia with subsequent reperfusion leading to inflammatory cell migration and a sustained inflammatory response.[13-17]

The initial inflammatory process – which develops as a result of synovitis – leads to an increase in pain, edema, disruption of mandibular function and OA.[18] The features of synovitis include hyperemia and an increased ratio of both fibroblast-like synoviocytes and macrophage-like synoviocytes cells. Quantitative and qualitative changes in the synovial fluid composition also include upregulated cytokines.[19-22] The synovial membrane and the synovial fluid it produces are critical to maintaining the health and function of the intra-articular tissues.[23,24] Therefore, if left untreated, continued progression of the inflammatory response will lead to cartilage and bone damage. Persistent inflammation within the TMJ also is known to result in both neo-angiogenesis and neural sprouting. This can involve the soft tissues of the capsule, including the retrodiscal tissue and articular disc, as well as the glenoid fossa and mandibular condyle. Macroscopically, synovitis also can result in synovial hyperplasia, creeping synovitis, synovial plica, fibrous adhesions and capsular impingement.[12] Disc displacement may be present concomitantly, though this should not be taken to mean the patient's signs and symptoms are the result of the disc position.

The recognition that most IPD is secondary to synovitis and its consequences has led to the development and successful utilization of minimally invasive surgical procedures such as arthrocentesis and arthroscopy. Arthrocentesis has been shown to reduce pain and improve range of motion in 70 to 95 percent of patients.[25-30] Arthroscopy has shown similar outcomes with reported pain reduction and improvement in range of motion in 80 to 90 percent of patients.[25,31-36] Arthrocentesis and arthroscopy allow the removal of inflammatory mediators and degraded proteins from within the joint, which otherwise would perpetuate inflammation. Arthroscopy has the additional advantage of being able to directly address the physical manifestations of synovitis, including synovial plica, capsular impingement, fibrous adhesions and chondromalacia. Comparing outcomes following arthrocentesis and arthroscopy suggests that arthroscopy is superior.[37]

Recommendations

To optimize patient care, the AAOMS Special Committee on Temporomandibular Joint Care has identified four key pillars of patient care:

1. A thorough history, screening and physical examination.
2. Appropriate diagnostic imaging.
3. Make the correct diagnosis.
4. Initiate the least invasive evidence-based nonsurgical or surgical treatment.

Thorough History, Screening and Physical Examination

The art and science of history-taking and examination are well-delineated in the diagnostic criteria/ temporomandibular disorders (DC/TMD) guidelines, which enable the recognition of arthralgia with a sensitivity and specificity of 89 percent and 98 percent, respectively.[38] Unfortunately, the proposed etiology of the arthralgia within the DC/TMD is limited to disc position, OA or subluxation, and it fails to appreciate the importance of the synovial molecular events involved in IPD. As a result, although the DC/TMD can identify IPD, it is unable to discern whether synovitis, capsular impingement, fibrous adhesions or SDD is the etiology. This remains a limitation of the DC/TMD.

The potential etiologies of jaw and facial pain are numerous. The complexity of the trigeminal neuroanatomic connections within the central nervous system and the convergence of afferent fibers from the trigeminal and upper four cervical nerves within the trigeminal spinal nucleus ensures pain referral patterns may result in perceived TMJ pain that is in fact unrelated to the TMJ. Screening for the presence of TMD (arthrogenous and myogenous) and cervical pain remains critical. The sensitivity and specificity of the DC/TMD screening instrument for identifying TMD is 99 percent and 97 percent, respectively[39] (Table 1).

Although diagnosing TMD is important, the term TMD remains problematic as it includes both arthrogenous and myogenous sources of pain. Distinguishing between the two can be facilitated by additional questions (Table 2). Screening for cervical pain and cervicogenic headache also is important[40-43] (Table 3).

The physical examination should be performed in a consistent and standardized manner. It is prudent to evaluate the external auditory canal and tympanic membrane to identify any otological sources of pain. This should be followed by manual palpation of the masticatory and cervical muscles and active range of motion exercises of the cervical spine. If radiculopathy is suspected, it is important to assess upper extremity strength and sensation. This should be followed by palpation of the TMJ capsule both laterally and endaurally. Joint sounds should be appreciated through both palpation and use of a stethoscope. The intraoral examination should assess the occlusion, maximum pain-free opening, maximum assisted opening with pain, lateral excursions and protrusion. In addition, it is helpful to perform that Mahan test bilaterally to help identify intra-articular pain sources. When the history and examination is performed correctly, it is more likely that the correct Axis I diagnosis will be obtained[38] (Figure 1).

Appropriate Diagnostic Imaging

It is recommended that any patient presenting with symptoms of a TMJ disorder have imaging of the TMJs. Initial orthopantogram (Panorex) imaging is considered appropriate. More advanced imaging – such as cone-beam computer tomography (CBCT), medical-grade CT and magnetic resonance imaging (MRI) – should be considered based on the history, clinical examination and differential diagnosis.

Make the Correct Diagnosis

When developing a differential diagnosis, it is important that it be comprehensive so as to ensure other sources of pain and dysfunction are considered.[44] It is well-known that psychosocial factors and comorbid medical conditions play a significant role in patients who present with temporomandibular disorders, while also having important prognostic implications.[38,45-47] Comorbid conditions such as anxiety, depression, catastrophizing, interstitial cystitis, fibromyalgia and lower back pain are but a few. The ability to identify and recognize psychosocial factors and comorbid medical conditions is critical to assuring patient and surgeon expectations for the outcome of any surgical procedure are concordant and realistic. The DC/TMD provides several validated questionnaires and instruments that allow the surgeon to readily identify

psychosocial factors and medical comorbid conditions. These instruments are simple to administer and score and can effectively screen for the presence of important psychosocial factors (Figure 2).

The presence of comorbid medical conditions and psychosocial factors has been qualified further using the Brief Symptom Inventory-18 and pressure pain threshold. This has allowed all pain patients to be classified into one of three groups: adaptive, pain sensitive or global symptoms. There is robust evidence that pain patients who are classified as global symptoms do poorly.[45] It may be prudent to avoid invasive surgical procedures such as arthroplasty and total joint replacement (TJR) in patients with comorbid medical conditions and problematic psychosocial factors. Although the prognosis following minimally invasive procedures such as arthrocentesis or arthroscopy also is likely to be guarded in this patient population, they are rarely associated with worsening symptoms or complications and should be considered as an alternative to more invasive procedures.

Initiate the Least Invasive Evidence-based Nonsurgical or Surgical Treatment

Nonsurgical treatment for the management of IPD may include rest, heat, ice, nonsteroidal anti-inflammatory drugs, muscle relaxants, physical therapy and orthotics, which may improve both pain and function. SDD should only be considered as the cause of the patient's IPD once all other potential causes have been treated.[44] It is possible, if not probable, that some patients with IPD have a combination of contributing factors including synovitis, capsular impingement and SDD (Figure 3).

Synovitis

The potential causes of synovitis include macrotrauma, microtrauma, autoimmune conditions, reactive arthritis and metabolic arthritis. The nonsurgical treatment of synovitis will depend on the cause of the synovitis (Figure 4).

Synovitis is a key feature of IPD and is associated with the presence of synovial plica, fibrous adhesions, chondromalacia, impingement and SDD. The surgical management of patients with IPD is identical regardless. Nonsurgical treatment with rest, heat, ice, nonsteroidal anti-inflammatory drugs, physical therapy and orthotics should be considered. There is good evidence that nonsurgical treatment will improve pain and function in most patients. There also is evidence that initiating minimally invasive procedures earlier in the process will result in improved surgical outcomes. This likely is to be the result of limiting the stage and progression of disease within the TMJ as well as reducing the potential for the development of peripheral and central sensitization. If minimally invasive procedures are unsuccessful in reducing pain and improving function at one month postprocedure, consideration for arthroplasty or TJR is reasonable providing the diagnosis of IPD remains correct and patient expectations realistic.

The presence of TMJ OA may be associated with the presence of synovitis, chondromalacia, capsular impingement/fibrous adhesions and disc perforation. It also is evident that OA is associated with subchondral bone loss as well as vascular and neural ingrowth into the subchondral bone. The presence of OA should therefore be reason to consider TJR (as opposed to arthroplasty) as all the pathologically involved tissues can be removed (Figure 5). The presence of OA, diagnosed through a medical-grade CT scan, should influence the decision as to whether arthroplasty or TJR is indicated, as the latter is more likely to be successful. Patients who previously have undergone a single arthroplasty but present with recurrent or persistent IPD also may be candidates for arthrocentesis or arthroscopy. Factors that influence the surgeon's ability to perform these procedures include the specific type of prior arthroplasty and whether the articular disc was maintained or removed. Prior arthroplasty with disc plication may maintain the superior joint space to enable arthrocentesis or arthroscopy. If the prior procedure was arthroplasty with discectomy, with or without replacement, the ability to perform either procedure is limited. If arthrocentesis is attempted, but irrigation unsuccessful, consideration for intra-articular injection of corticosteroid should be given. If a decision to proceed with another open procedure is made, it is recommended that the open procedure be TJR. A second arthroplasty is not recommended given that the likelihood of a good outcome is much less when compared to proceeding with a TJR.[48]

Patients having previously undergone TJR, but presenting with pain and limited function, present a unique challenge. The etiology of continued or increasing post-TJR pain can be difficult to discern. It is suggested that there are two broad categories of continued pain that should be considered[49] (Table 4).

The intrinsic sources of pain are more challenging to identify. Heterotopic bone, dislocation and component or screw fracture can be appreciated with imaging such as non-contrast CT scans. Periarticular joint infection (PJI), neuroma, synovial entrapment and material hypersensitivity remain the most challenging to diagnose. PJI is the most common complication occurring after any joint replacement. Definitive diagnosis can be difficult since obtaining synovial fluid for analysis from a TJR is difficult. To date, C-reactive protein and erythrocyte sedimentation rate remain the most ideal test for the diagnosis of PJIs.[49-51] This should be supplemented with a CT scan with contrast and, on occasion, an hexamethylpropylene amine oxime technetium 99-labeled white blood cell scan. Early-stage PJI (<3 weeks after TJR) can be treated with debridement, antibiotics and implant retention.[52] Late-stage PJI (>3 weeks after TJR) requires two-stage removal and replacement with long-term antibiotic therapy. An intra-articular neuroma can form following TJR.[53] Synovial entrapment, which is thought to occur from the proliferation of the neosynovium, also can develop around a TJR.[54] A local anesthetic block of the auriculotemporal nerve can help make these diagnoses. Careful arthrotomy to remove this tissue from the articulating surfaces with fat grafting can resolve both of these issues.

Material hypersensitivity response to TJR materials is often a diagnosis of exclusion. The two most commonly used tests are the in vivo skin patch test and in vitro lymphocyte transformation test.[55] The presence of a positive skin patch test or elevated lymphocyte transformation test may not correlate with device failure or patient symptoms.[49] Device removal should be considered a last resort as the relationship between documented metal hypersensitivity and patient-reported outcomes has not been established. Routine testing for metal hypersensitivity is not currently recommended.

Summary

Significant advances in the understanding of the pathophysiology of TMJ pain and inflammation, along with the application of established principles underlying joint disease, have enabled IPD to be better diagnosed and treated. The well-documented success of nonsurgical treatment as well as minimally invasive surgical options provides the patient and surgeon with sound treatment choices. Arthroplasty and TJR are surgical options that should be reserved for patients in whom minimally invasive procedures have not been successful. It is incumbent on the oral and maxillofacial surgery community, which is uniquely positioned to offer relief to patients suffering from TMJ disease, to treat patients according to the best available level of evidence and, where possible, continue to add new knowledge to support ongoing improvements in care.

Supplementary Data

Supplementary data associated with this position paper can be found in the online version, at 10.1016/j.joms.2024.01.003.

Table 1

Screening Questions to Identify Temporomandibular Disorders

Temporomandibular pain disorder screening instrument
1. In the last 30 days, on average, how long did any pain in your jaw or temple area on either side last? a. No pain b. From very brief to more than a week, but it does stop c. Continuous
2. In the last 30 days, have you had pain or stiffness in your jaw on awakening? a. No b. Yes
3. In the last 30 days, did the following activities change any pain (that is make it better or worse) in your jaw or temple on either side? A. Chewing hard or tough food a. No b. Yes B. Opening your mouth or moving your jaw forward or to the side a. No b. Yes C. Jaw habits such as holding the teeth together, clenching, grinding or chewing gum a. No b. Yes D. Other jaw activities such as kissing, yawning or talking a. No b. Yes

Responses: a = 1 point, b = 2 points, c = 3 points

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(continued on following page)

Table 2

Questions that Distinguish Arthrogenous and Myogenous Sources of Pain

Question	Favors arthrogenous	Favors myogenous
When eating something hard (like a bagel) do you have pain with the first bite?	Yes	
When eating something hard (like a bagel) do you have pain that worsens with prolonged chewing?		Yes
Do you have pain with opening your jaw wide (yawning)?	Yes	
Does your jaw ever lock or catch where you cannot close or open it?	Yes	
Does your jaw make any sounds?	Yes	
Is there any pain with those sounds?	Yes	
Is your pain worse in the morning when you wake up?		Yes

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Table 3

Questions to Identify Cervical Sources of Pain

Cervical pain screening
1. Do you have neck pain? a. No b. Yes
2. Does neck, shoulder or arm movement aggravate your neck pain? a. No b. Yes

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Table 4

PostTJR Pain

Extrinsic Sources of Pain	Intrinsic Sources of Pain
• Prior misdiagnosis • Chronic centrally mediated pain • Persistent myofascial/muscular pain • Complex regional pain syndrome 1 • Complex regional pain syndrome 2 • Temporalis tendonitis • Coronoid impingement • Frey syndrome • First bite syndrome	• Heterotopic bone formation • Infection • Dislocation • Synovial entrapment syndrome • Component or screw fracture • Neuroma formation • Material hypersensitivity

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Figure 1

Validated Axis I Pain-related TMD* Diagnoses

Disorder	History	Examination Findings
Myalgia† (Sensitivity, 90%; Specificity, 99%)	Pain in a masticatory structure modified by jaw movement, function or parafunction	Report of familiar pain‡ in temporalis or masseter muscles with: • palpation of these muscles • maximum unassisted or assisted opening movements Note: Assessment of other masticatory muscles may be indicated in some clinical situations.
Myofascial Pain With Referral (Sensitivity, 86%; Specificity, 98%)	Same as for myalgia	Report of familiar pain‡ with palpation of the temporalis or masseter muscles. Report of pain at a site beyond the boundary of the muscle being palpated (for example, referral to a tooth).
Arthralgia (Sensitivity, 89%; Specificity, 98%)	Same as for myalgia	Report of familiar pain‡ in TMJ§ with: • palpation of the TMJ • maximum unassisted or assisted opening, right or left lateral, or protrusive movements
Headache Attributed to TMD (Sensitivity, 89%; Specificity, 87%)	Headache in temporal area modified by jaw movement, function, or parafunction	Report of familiar headache¶ in temple area with: • palpation of temporalis muscles • maximum unassisted or assisted opening, right or left lateral, or protrusive movements Note: A diagnosis of pain-related TMD also must be present (for example, myalgia, arthralgia).

* TMD: Temporomandibular disorder.

† Myalgia can be subclassified into three disorders: local myalgia, myofascial pain and myofascial pain with referral; only myofascial pain with referral has been validated. See Schiffman and colleagues[9] for diagnostic criteria for local myalgia and myofascial pain.

‡ Familiar pain is similar to or like the pain the patient has been experiencing. The intent is to replicate the patient's pain complaint.

§ TMJ: Temporomandibular joint.

¶ Familiar headache is similar to or like the headache the patient has been experiencing. The intent is to replicate the patient's headache complaint.

Figure 2

Axis II Assessment Protocol

Questionnaire*	No. of items	Usefulness
Graded Chronic Pain Scale†,‡	7	Pain intensity component: pain amplification and central sensitization Pain-related disability component: decreased functioning because of pain
Pain Drawing†,‡	1	Distinguishes among local, regional and widespread pain; assesses for other comorbid pain condition; and may indicate pain amplification, sensitization and central dysregulation
Jaw Functional Limitation Scale†,‡	8 or 20	Quantifies effect on jaw mobility, mastication and verbal and emotional expression
Patient Health Questionnaire-4†	4	Identifies psychological distress (depression and anxiety)
Patient Health Questionnaire-9‡	9	Identifies depression; contributes to chronicity
Generalized Anxiety Disorder-7‡	7	Identifies anxiety; contributes to stress reactivity and to parafunction
Patient Health Questionnaire-15‡	15	Measures physical symptoms; assesses for specific comorbid functional disorders
Oral Behaviors Checklist†,‡	21	Measures parafunction: contributes to onset and perpetuation of pain prognosis

* Questionnaires to assist in the identification of patients with a range of simple to complex presentations that affect treatment and prognosis.

† Questionnaire included in screening protocol.

‡ Questionnaire included in comprehensive protocol.

Figure 3

Potential Sources of Intra-articular Pain and Dysfunction

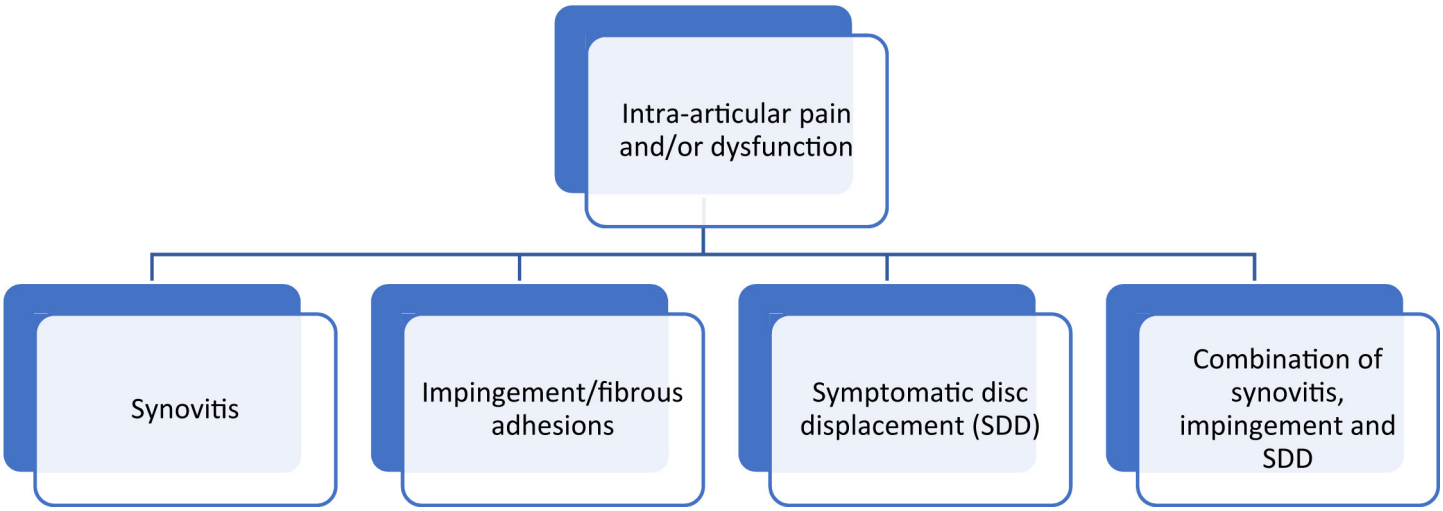


Figure 4

Potential Causes of TMJ Synovitis

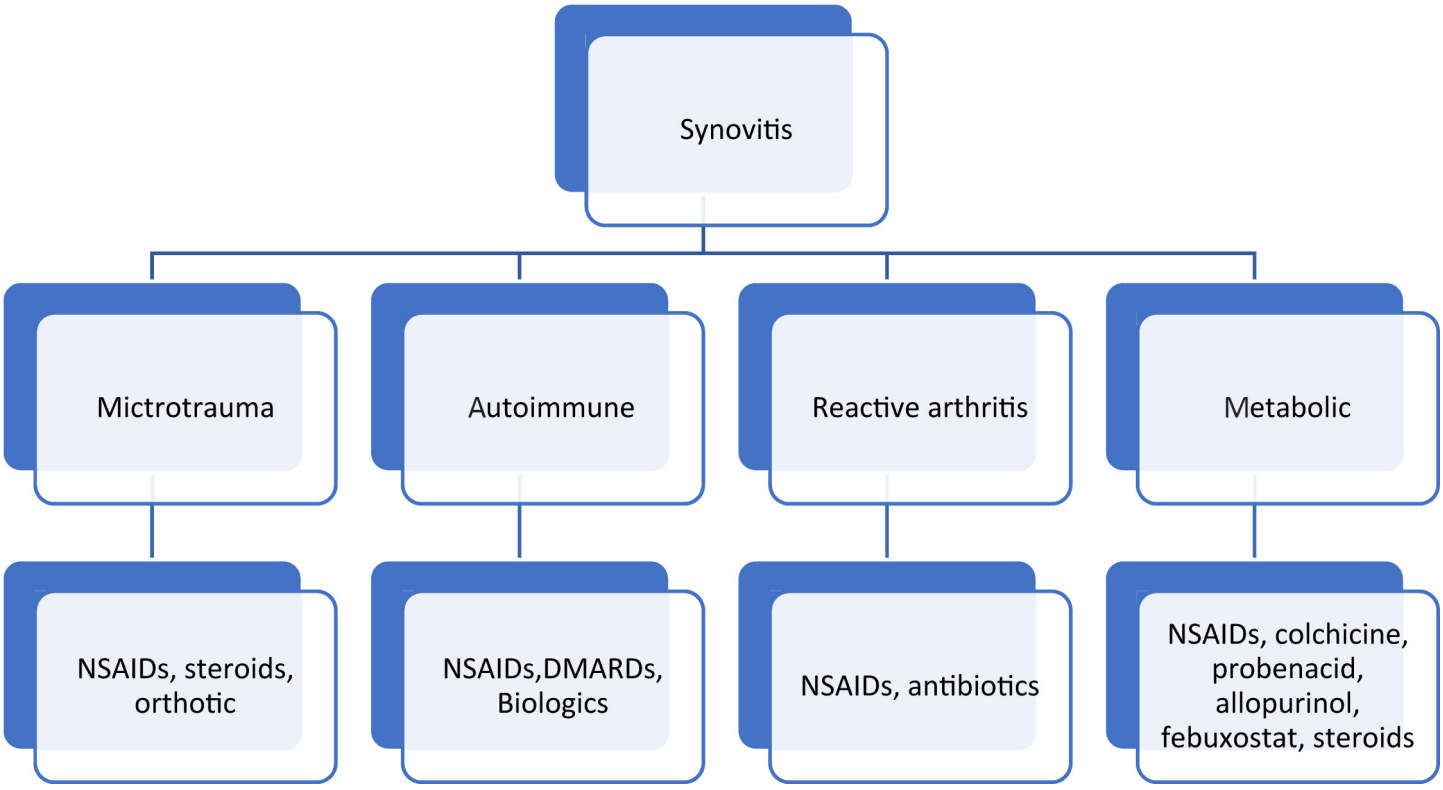
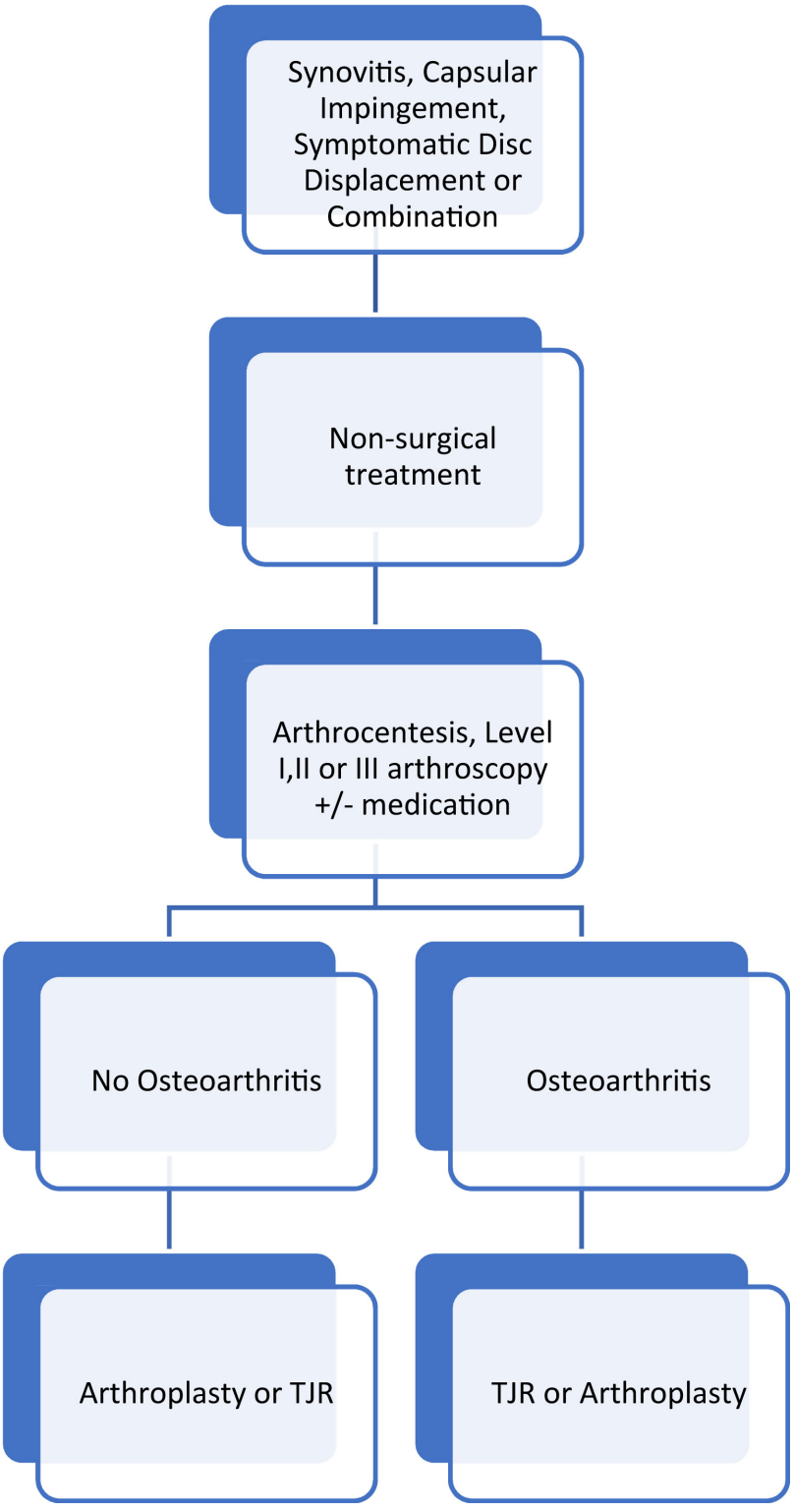


Figure 5

Algorithm for the Management of Intra-articular Pain and Dysfunction



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