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Temporomandibular joint pain and dysfunction

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Abstract
Pain caused by temporomandibular disorders originates from either muscular or articular conditions, or both. Distinguishing the precise source of the pain is a significant diagnostic challenge to clinicians, and effective management hinges on establishing a correct diagnosis. This paper examines terminology and regional anatomy as it pertains to functional and dysfunctional states of the temporomandibular joint and muscles of mastication. A review of the path physiology of the most common disorders is provided. Trends in evaluation, diagnosis, treatment, and research are presented.

Keywords: Temporomandibular joint, Dysfunction

Introduction
Signs and symptoms of temporomandibular disorders (TMDs) may include pain, impaired jaw function, malocclusion, deviation or deflection, limited range of motion, joint noise, and locking. Headache, tinnitus, visual changes, and other neurologic complaints may also accompany TMDs. Because of many etiologic factors, the diagnosis and treatment of patients with TMDs is complex. TMDs can be subdivided into muscular and articular categories. Differentiation between the two is sometimes difficult because muscle disorders may mimic articular disorders, and they may coexist like in disc displacement disorders. Myogenic disorders include myalgia (myofascial pain, fibromyalgia), myospasm, splinting, and fibrosis/contracture. Articular disorders include sinusitis/capsulitis, joint effusion, trauma/fracture, internal derangement, arthritis, and neoplasm. Accurate diagnosis allows for appropriate therapy whether it is nonsurgical or surgical. Current trends favour conservative (nonsurgical) therapy, and the surgical interventions have become less aggressive, moving away from open arthroplasty and toward arthroscopic procedures. Research continues to look toward biochemical markers of disease. The interrelationship between the various disorders continues to be explored. The temporomandibular joint (TMJ) is a compound articulation formed from the articular surfaces of the temporal bone and the mandibular condyle. Both surfaces are covered by dense articular fibrocartilage. Each condyle articulates with a large surface area of temporal bone consisting of the articular fossa, articular eminence, and preglenoid plane. The TMJ functions uniquely in that the condyle both rotates within the fossa and translates anteriorly along the articular eminence. Because of the condyle's ability to translate, the mandible can have a much higher maximal incisal opening than would be possible with rotation alone. The joint is thus referred to as "gynglimo diarthrodial": a combination of the terms ginglymoid (rotation) and arthroidial (translation) [1]

The joint is stabilized by three ligaments: collateral (discal), capsular, and temporomandibular. These attach to the disc at the medial and lateral poles of the mandibular condyle, as well as to the temporal fossa. These ligaments limit extreme condylar movement. The capsular ligament surrounds the joint space and disc and acts to contain the synovial fluid within the joint space. The capsule is lined by a synovial membrane. Synovial tissue covers all intra-articular surfaces except for the pressure- bearing fibrocartilage (ie, disc, condyle, eminence). The synovial tissue is highly innervated and vascularised and has regulatory, phagocytic, and secretory functions [2].

The masseter, medial pterygoid, lateral pterygoid, and temporalis muscles are the muscles of mastication. The masseter, medial pterygoid, and temporalis are primarily responsible for mandibular closure and bite force, whereas the lateral pterygoid and infrahyoid muscles are responsible for mandibular opening. Mandibular movement is also influenced by the digastric, geniohyoid, mylohyoid, stylohyoid, sternohyoid, omohyoid, sternothyroid, and thyrohyoid

muscles, which as a group coordinate complex mandibular movements including opening, protrusion, retrusion, lateral excursion, and closure [3].

Temporomandibular Disorders

The term "TMJ pain" varies greatly in meaning among clinicians, patients, and the general population. Historically, symptom-based classification of the disorder has been problematic. As stated by Laskin [4], the difficulty began with the introduction of a "TMJ syndrome."

Then clinicians erroneously grouped a "variety of etiologically unrelated conditions into one diagnostic category based on the fact that they produced similar signs and symptoms," and this led to "one diagnosis equals one treatment." Only later was it recognized that many of these patients suffered from muscle-related conditions. The terms myofascial pain (MFP) and myofascial pain and dysfunction (MPD) evolved [5] and "TMJ disorders" became "TMDs."

Myogenic Disorders

Within this category, MFP and MPD syndrome are encountered frequently. Other muscular disorders include myositis, fibrosis, tendonitis, whiplash injury, and fibromyalgia. Patients suffering from MFP will have tenderness to palpation of two or more muscle sites. Myalgias involving the muscles of mastication predominate[6] Muscles were thought to be under an increased burden in the presence of these skeletal and/or dental misalignments. As such, a "vicious cycle" model was proposed: Structural → abnormality → muscle hyperactivity ↔ pain ↔ mandibular [7, 8] dysfunction where pain and muscle hyperactivity potentiate each other and emotional stress is thought to have an additive effect. Over time, there has been a lack of scientific evidence to support this theory. Others have put forth a pain-adaptation model in which motor behaviour is altered or limited as a response to pain, thus serving a protective purpose. One must differentiate muscular from joint conditions in order to appropriately treat the patient. At the same time, the clinician must understand the role of MPD within the spectrum of TMDs. It has been reported that approximately 50% of all TMDs are masticatory myalgias or painful masticatory muscle disorders. Treatment of masticatory MFP may include pharmacologic therapy (nonsteroidal anti-inflammatory drugs, muscle relaxants, tricyclic antidepressants, anxiolytics), occlusal appliance/splint therapy, trigger point therapy (spray and stretch, injections), and physical therapy (mandibular exercises). Splint therapy is considered an adjunct to pharmacologic therapy and most appropriate when nocturnal parafunctional activities can be identified. Typically, a flat-plane maxillary occlusal splint designed for bilateral contact of all teeth is fabricated [8]. Such splints are thought to unload the joint by disarticulating the dentition and increasing the vertical dimension of occlusion. By unloading the joint, there will be a reduction in both synovitis and masticatory muscle activity.

Articular Disorders

Disc displacement (also known as internal derangement) is defined as "a disturbance in the normal anatomic relationship between the disc and condyle that interferes with smooth movement of the joint and causes momentary catching, clicking, popping, or locking" [9, 10] The etiology of articular disorders may be degenerative, traumatic, infectious, immunologic, metabolic, neoplastic, congenital, or developmental. Articular disc displacement (internal derangement) anterior disc displacement (ADD) is the most frequently encountered articular disorder. Therapy is indicated if pain and significant limitation in range of motion are present. The incidence of ADD is unknown. Numerous radiographic, clinical, and cadaveric studies of asymptomatic subjects have shown rates up to 30%. The clinical significance of this finding remains uncertain. When the articular disc becomes displaced anteriorly [11], there is excessive stretching of the retrodiscal tissue, which then bears repeated loading force from the mandibular condyle. This tissue has been shown to have some capacity to adapt to these forces and may transform into a "pseudodisc." In many patients the disc is recaptured and is known as "disc displacement with reduction," resulting in TMJ noise (clicking or popping) and full translational movement of the condyle. With mandibular closure, a reciprocal (closing) click represents the condyle returning to the retrodiscal tissue and the disc returning to an anterior position. ADD without reduction, also known as closed lock, will have a much different clinical presentation because the condyle's forward translation is limited by the disc's anterior position and is unable to reduce onto the disc, allowing only for rotational and not translational movement. Patients with acute or subacute closed lock typically report a sudden onset of pain and inability to open more than 20 to 30 mm. The patient may give a history of joint noise that suddenly ceased with the onset of signs and symptoms. Clinically, the mandible deviates on opening to the affected side due to the ability of the unaffected joint to translate. Additionally, excursive mandibular movements to the contralateral side are limited.

This diagnosis of TMDs continues to present the clinician with a significant challenge. Establishment of an accurate diagnosis is necessary for effective management. The difficulty lies not in creating a distinction between articular and muscular disorders, but in the interrelation of the two entities. Although patients may have isolated joint or muscular disorders, many have a component of each. Simply stated, joint disorders may lead to muscle dysfunction, and muscle disorders may lead to joint dysfunction. This may not be possible to elicit on examination because the patient will tend to guard against pain.

In chronic disc displacement without reduction, the patient can usually recount a history consistent with acute closed lock that resolved over time. Recovery of function is due to stretching the retro discal tissue over weeks to months, restoring translational movement.

MRI allows for evaluation of soft tissue abnormalities of the TMJ. MRI is non-invasive and avoids radiation exposure. The disc can be visualized making diagnosis possible. T1 images show a hypodense biconcave disk between the condyle and eminence. Effusion, bone marrow edema, and soft tissue pathology are well visualized with T2 imaging. Multiplane views of the TMJ are available; with high-speed MRI, dynamic studies are also available. The ability of the joint to adapt to biomechanical stress and disc derangement has been a subject of debate. In his classification system, Wilkes [12] promotes the theory that internal derangement logically progresses to degenerative joint disease (DJD). Historically, surgical and nonsurgical approaches have been used to reposition the displaced disc, with the goal of arresting this progression [13] In an opposing view, Milam [9] states that "the adaptive capacity of the TMJ is not infinite…some individuals are… capable of mounting an adaptive response to an articular disc displacement; other individuals may not adapt to these structural derangements, and a progressive DJD may result." Factors considered to compromise the adaptive response include age, sex, stress, and illness [14] He concludes that disc

derangement may exist variably as cause or effect, but does not always progress to disease. Although patients without internal derangement may develop osteoarthritis (OA) [15] a complex two way relationship exists.

Capsulitis and synovitis

Inflammation of the capsular ligament may manifest with swelling and continuous pain localized to the joint. Movements that stretch the capsular ligament cause pain with resultant limitation of such movement. Significant inflammation may increase joint fluid volume. When this occurs, one may see an ipsilateral posterior open bite (lack of contact between maxillary and mandibular teeth) secondary to inferior displacement of the condyle. Similarly, inflammation due to trauma or abnormal function may affect the retrodiscal tissue. Edema in this area may cause anterior displacement of the condyle and an acute malocclusion with painful limitation of mandibular movements [20]. Inflammatory and pain mediators have been identified in TMJ synovial fluid [21, 22] Chemical breakdown of degenerative by products is thought to stimulate the production of inflammatory and pain mediators (prostaglandin E2 and leukotriene B4, among others) through the arachidonic acid cascade. Prostaglandin E2 is a powerful vasodilator and leukotriene B4 attracts inflammatory cells. Their presence creates acute synovitis pain and stimulates further damage from cytokines and proteases. For this reason arthrocentesis and arthroscopy for joint lavage and lysis of adhesions are believed to have a therapeutic effect. These procedures remove particulate debris and pain mediators, aiding reduction of joint inflammation and pain [23, 24, 25]. Results are similar with and without disc repositioning. Lysis of adhesions may improve range of motion. Steroid injections are also used to reduce synovial inflammation and pain. Recent investigations have looked at intra-articular morphine for sustained pain relief in patients. Research is now focusing on the role of biochemical mediators in the development and progression of TMJ pain and dysfunction and the identification of biochemical "markers" of TMJ disease [26]

The Arthritides

Arthritis of the TMJ has many etiologies: frequently OA and rheumatoid arthritis (RA) and less often infectious, metabolic (gout), or immunologic (ankylosing spondylitis, lupus). DJD, also known as OA, has a multifactorial pathogenesis including biomechanical, biochemical, inflammatory, and immunologic insults. Excessive and repetitive mechanical stress has been implicated. Inflammatory mediators and waste products may play a role in DJD Inflammatory states cause changes in the viscosity of synovial fluid, which changes its ability to nourish the articular cartilage, thus changing cartilage metabolism. OA is classified as primary (no known predisposing factors) or secondary (associated with known abnormalities or injuries). Primary OA symptoms begin in the fifth to sixth decade. Secondary OA produces symptoms at an earlier age [27].

In contrast to the other arthritides, OA symptoms will not necessarily be present in other joints. Patients suffering from OA complain of increasing pain during increased function and load bearing throughout the day. Joints are tender and will exhibit decreased range of motion. Crepitus may indicate loss of articular cartilage. Patients may have referred pain to head and neck regions. Radiography may reveal joint space narrowing, osteophyte formation, condylar head flattening, and subchondral bone cysts. In the osteoarthritic joint, there is progressive softening and loss of cartilage, which Quinn [28] calls chondromalacia (softening of the articular cartilage) of the TMJ. It is thought that repeated stress-related microtrauma (ie, bruxism) eventually overloads the joint's articular cartilages leading to compression and shearing of cartilage. Chondrocyte injury stimulates release of proteolytic enzymes and other collagenases. Eventually, there is loss of water and loss of cartilage resilience [29, 30, 31]

Four stages of TMJ OA are based on the amount of cartilage degeneration and the grade of synovitis. In stage 2, the early stage, patients may report pain and limited range of motion. Joint noise may occur due to disc displacement or perforation. This continues into the later stages, and patients may develop crepitation secondary to bone exposure. Pain and adhesion formation result in limitation of joint movement. Dijkgraaf *et al.* [32] found that "in many patients, the signs and symptoms of TMD are attributable to osteoarthritis." The authors place less emphasis on the stage of internal derangement and more emphasis on both the stage of cartilage degradation and grade of synovitis.

Panoramic radiography is an excellent screening tool for the presence of bony degenerative changes. In addition to identifying disc displacement, MRI is useful in the diagnosis of joint effusion, osteoarthritic changes, bone marrow abnormalities of the mandibular condyle, retrodiscal tissue changes, and neoplasms [33]. TMJ arthroscopy now allows clinicians to visualize degenerative changes of both the articular cartilage and disc at early stages Arthroscopy is considered to be the "gold standard" in the diagnosis of OA because degenerative changes are visualized earlier than with radiographic techniques.

The age of onset is younger (fourth to sixth decade) than that seen with OA. In contrast to OA, patients with RA typically have morning stiffness that lasts for more than an hour, but report improvement of mobility with function throughout the day. They complain of deep, dull pre-auricular pain that worsens with function. Patients may also report fever, malaise, and fatigue. They will eventually experience decrease in jaw mobility, joint destruction, and fibrous ankylosis. Patients may progress to loss of mandibular ramus height, retrognathia, and open bite. Patients will have symptoms long before there is radiographic evidence of disease. Early imaging with MRI may be beneficial to evaluate disc morphology and pathologic changes.

Neoplasms

Pain and/or changes in occlusion may be presenting signs and symptoms of a pathologic joint lesion. Neoplasms of the condyle and joint space may be benign (osteoma, chondroma, synovial chondromatosis, giant cell lesions) or malignant (chondrosarcoma, osteosarcoma, synovial sarcoma, multiple myeloma). The most common TMJ neoplasms are the osteoma and osteochondroma. These can be distinguished from condylar hyperplasia by the presence of a normal condylar neck length. Pathologic lesions may be first noted on screening panoramic radiographs. Further evaluation of bony tumors is best performed with CT.

Conclusions

The subject of TMJ pain and dysfunction is complex. Signs and symptoms may be specific or nonspecific. The strong relationship between articular and muscular disorders makes accurate diagnosis difficult. A thorough knowledge of joint anatomy and function serves as a basis for understanding the effect of dysfunction on the joint's component parts. Myogenic causes of pain are the majority and may coexist with articular disorders. Internal derangement and DJD are the most frequently encountered articular disorders. The etiology of each is multifactorial, and the cause-effect relationship

between the two remains a controversial subject. Imaging techniques have greatly advanced in the evaluation and diagnosis of TMDs. Treatment trends now involves a comprehensive conservative plan along with surgical options. Treating these dysfunctions with only surgical techniques lessens the chance for treatment success.

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