Chiropractic Management of Capsulitis and Synovitis of the Temporomandibular Joint

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Localized inflammatory conditions (eg, synovitis and capsulitis) of the temporomandibular joint are commonly seen in clinical practice. Regardless of their frequency of occurrence, these conditions must be differentially diagnosed from conditions that also may cause pain in the temporomandibular joint region. Capsulitis or synovitis should be considered if such pain is present and historical, physical, and laboratory findings do not indicate a referred pain phenomena or systemic, tumorous, or infectious involvement. This article reviews the clinical characteristics, etiology, physical examination methods, treatment, and prognosis for capsulitis and synovitis, and three cases that illustrate these conditions are reported.
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Complaints of dysfunction and pain should be differentially diagnosed to choose a correct and successful method of treatment. Pain, when it occurs in the region of the temporomandibular joint (TMJ), may arise from several causes: inflammation of the pre-auricular lymph node[1]; otitis media or externa[2]; referred pain from a trigger point[3]; and tendonosynovitis of the temporalis tendon as it passes behind the zygomatic arch.[4] Certain facial or dental pains, such as trigeminal neuralgia[5] or dental caries,[6] may initially present as pain over the TMJ. Bony tumors, both benign and malignant (primary and metastatic), that lie in the TMJ region have been reported to initially present as TMJ pain.[7,8] The inflammatory arthritides (ankylosing spondylitis, rheumatoid arthritis, juvenile arthritis, psoriatic arthritis, etc) may initially present as TMJ pain, and the initial presentation is commonly of isolated TMJ involvement.[9-14] In the case of chronic pain, the practitioner must first differentiate between whether the complaints are due to organic changes or psychosocial factors.[15,16]

Then, of course, there are the common organic changes involving the TMJ that may cause local pain. Such changes have been classified within the International Headache Society's Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias, and Facial Pain (IHSC).[17] Inflammatory conditions of the TMJ (IHSC no. 11.7.5), the topic of this paper, are further subdivided to synovitis (IHSC no. 11.7.5.1; ICD no. 727.09) and capsulitis (IHSC no. 11.7.5.2; ICD no. 716.98). For the purposes of this paper, synovitis refers to intracapsular inflammation primarily affecting the posterior attachment, whereas capsulitis refers to inflammation of the structures primarily comprising the joint capsule. The term "posterior attachment" is used as described by Scapino[18] and refers to the vascular and innervated tissue lying behind the articular disc, which has been previously referred to as "bilaminar zone" or "retrodiscal tissue" (Fig 1).
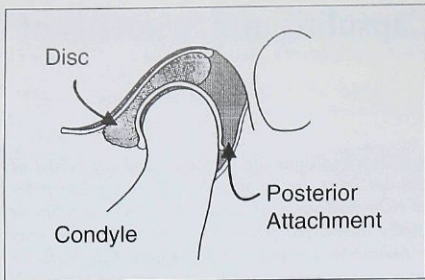
Fig 1 Posterior attachment.

Clinical Characteristics

Synovitis

The term synovitis, as it is currently used, has been preceded by various terms such as prearthritis, traumatic arthritis, intracapsular edema, retrodiscitis, and posterior capsulitis.[19] Of these, retrodiscitis and posterior capsulitis have seen popular use up to the late 1980s, with the latter being used most often. Posterior capsulitis was first recognized and defined as a specific organic condition of the TMJ by Farrar[20] in 1968. In that article and subsequent communications,[21] he states that this disorder follows a typical course of events: (a) the posterior attachment becomes edematous; (b) intracapsular pressure increases; (c) the condyle becomes displaced anteriorly in the rest position, causing a barely perceptible midline shift to the opposite side; (d) ipsilateral disocclusion occurs; and (e) joint pain results and is aggravated when the patient attempts to fully occlude the ipsilateral teeth, thus forcing the condyle backward against the inflamed posterior attachment. This original characterization closely parallels the diagnostic criteria for synovitis as described by the IHSC.

Capsulitis

This lesion was first described by Cooper[22] in 1823. Historically, the term capsulitis has been used interchangeably with synovitis, and both of these terms have been used interchangeably with arthritis, arthralgia, contusion, and arthrosis temporomandibularis. The recognition of capsulitis as a specific disorder was slow to develop (its development coincided with the increased understand-

ing of the anatomy of the TMJ), and it was often intermingled with many other temporomandibular disorders (TMDs)—all of which were cluttered together under one, two, or three diagnostic terms. This trend—to view TMD as an all-inclusive syndrome—would continue until a more detailed understanding of the TMJ developed. For example Shore,[6] in 1976, includes at least three different disorders in the single clinical condition of TMJ arthrosis:

> . . . a noninfectious, trophic, degenerative affliction of the joint tissues . . . Pyrexia does not occur, but swelling caused by joint effusion . . . may be present. [This may include synovitis, capsulitis or one of the inflammatory arthritides.] The trauma causes changes within the tissues of the joint which may result in joint effusion. The effusion is noninfectious swelling of the arthrotic lesion and is usually nonarticular . . . The clinical manifestations of temporomandibular joint arthrosis . . . [include] . . . clicking, crackling noises, crepitation, tenderness, pain in and around the joint.

In the mid 1980s, Friedman et al,[23] Bell,[24] and many others presented capsulitis as a distinct clinical entity. In 1985, Bell[25] described capsular pain as a result of inflammation of the fibrous capsule and its inner synovial lining. He reported that technically this results in both synovitis and capsulitis and presented the argument that "to distinguish between the two is difficult if not impossible clinically." That is to say, one cannot be certain if just the fibrous capsule or just the inner synovial lining of the capsule is inflamed. In any event, both Bell and the IHSC agree that capsular pain is provoked when the inflamed capsule is stretched (eg, by translatory movement of the capsule). The pain, therefore, is exacerbated by protrusion or lateral excursion of the mandible, contralateral chewing, and wide mouth opening. Capsulitis is further characterized by palpable tenderness or pain directly over the condyle, and minor swelling over the joint may be detected (Table 1).

Etiology

It is commonly believed that the various factors that alter joint dynamics contribute to the formation of synovitis or capsulitis. Factors such as changes in occlusion, occlusal interferences, loss of posterior support, iatrogenic malocclusion, abusive oral habits, occupational conditions (eg, holding

Table 1 Clinical Characteristics of Synovitis and Capsulitis

Synovitis	Capsulitis
Pain is increased by clenching the teeth to maximum intercuspation	No increase in pain when clenching
Pain is decreased by biting against a separator that prevents maximum intercuspation of the teeth	No change in pain when biting against a separator
Pain is increased by forced ipsilateral excursive movement of the mandible	Increased pain during translatory movements
No change in pain with resisted protrusion of the mandible	Decreased range of motion
Acute malocclusion = ipsilateral disocclusion	Acute malocclusion = ipsilateral disocclusion
Secondary central effects may be displayed	Secondary central effects may be displayed
	Palpable tenderness directly over condyle
	Joints sounds during first moments following periods of inactivity

the telephone receiver between the shoulder and ear), bruxism, microtrauma, and some of the conditions mentioned at the beginning of this article can cause muscular imbalance and lead to increased loading of the TMJ.[26]

Synovitis

Friedman and Weisberg[27] have reported that one of the most common causes of TMJ synovitis is an "excessively posteriorly positioned condyle," which traumatizes the synovial membrane and encroaches on the highly vascular retrodiscal tissues. Extrinsic trauma, such as an impact to the jaw sustained during a motor vehicle accident or abusive use of the jaw, such as from repetitive behaviors (eg, bruxism, atypical chewing habits, and chronic gum chewing)[28] have also been reported to cause synovitis. Systemic disease, infections, inflammatory arthropathies, and surgical contamination[29] may also include the complaint of synovitis.

Capsulitis

Virtually any activity that produces excessive opening of the mouth may cause injury to the overstretched capsule. This includes prolonged or difficult dental procedures, endotracheal intubation, and wide mouth yawning.[30,31] Intrinsic trauma, such as occlusally caused microtrauma and abusive chewing habits, may also cause injury to the capsule.[32] Lundeen et al[33] have reported that psychological stress is a major etiologic factor in the production of TMJ pain.

Mediators of Inflammation and Pain

Many studies of synovial fluid obtained from the large joints of patients with inflammatory joint disorders have demonstrated the existence of metabolites of the arachidonic acid cascade.[14–38] Similar studies on the TMJ, using patients with acute synovitis, are rare. Up until recently, it was assumed that the inflammation of the TMJ occurs as it does in any synovial joint of the body. The recent work of Quinn and Bazar[39] has shed some light on the pathophysiologic events leading to the painful TMJ. Their work isolated two powerful mediators of pain and inflammation and showed that prostaglandin E_2 (PE_2) and leukotriene B_4 (LB_4) exist in significant concentrations in the synovial fluid of inflamed TMJs. Bradykinins and excessive particulate debris initiate the production of PE_2 and LB_4 from the arachidonic acid cascade (Fig 2). Prostaglandin E_2 is a powerful vasodilator of capillaries, it increases vascular permeability and can cause bone resorption.[40] Leukotriene B_4 triggers vascular permeability and is a powerful chemotactic and activator of inflammatory cells. The discovery of these mediators is consistent with previous reports of increased presence of white blood cells and synovial membrane hypertrophy.[41,42]

Physical Examination Methods

If local pain in the area of the TMJ is present, and historical, physical, and laboratory findings do not indicate systemic, tumorous, or infectious involvement, local causes should be considered.

Local Examination of the TMJ

Local examination begins with a visual inspection of the area. Signs of external trauma such as small cuts, insect bites, furunculosis, abrasion, or evidence of impact trauma should be noted and may
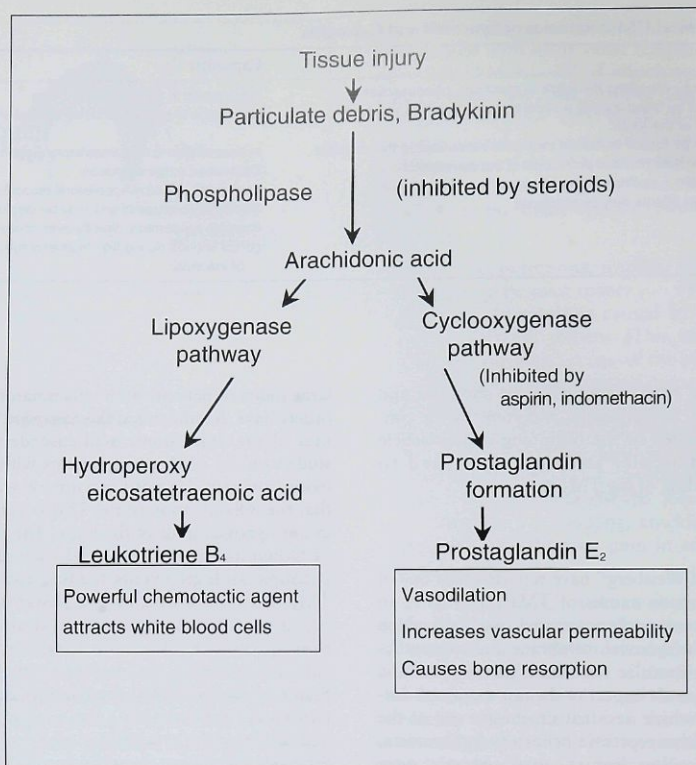
Fig 2   Key arachidonic acid metabolites of pain and inflammation found in the TMJ; also shown are the principle mediators, points of therapeutic intervention, and major actions of leukotriene B_4 and prostaglandin E_2.

explain the presence of pain. The skin surface overlying the TMJ should be inspected for signs of inflammation or infection, eg, redness, warmth, swelling, or exudate. Significant swelling is usually seen with acute infection or systemic inflammatory disease.[43] During the time of the visual inspection, muscle activity should be observed. Evidence of contributing factors, such as signs of abusive oral habits, eg, habitual jaw jutting or bruxing, often may be observed.

Palpation of the Lateral Joint and Surrounding Area

Manual palpation usually follows visual inspec-

tion. When joint injury is suspected, the surrounding musculature should be evaluated, as reflexive muscle splinting activity is usually seen. Wänman and Agerberg[44] have reported on the use of a three-point grading scale when palpating the TMJ and surrounding area (Table 2). Capsulitis or synovitis generally evokes a grade II or III response to palpation and is restricted to the area immediately surrounding the TMJ.

Palpation of the Posterior Joint

Palpation of the posterior aspect of the TMJ is usually performed by placing the distal pad of the smallest finger in the external auditory meatus
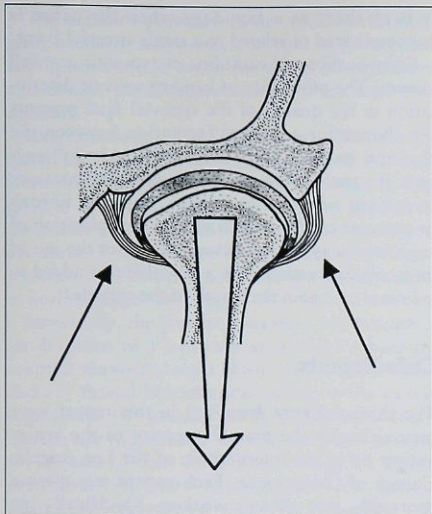
Fig 3 Long-axis distraction (large arrow) of the TMJ stressing the fibrous capsule (small arrows), which will elicit a pain response if the tissue is inflamed.
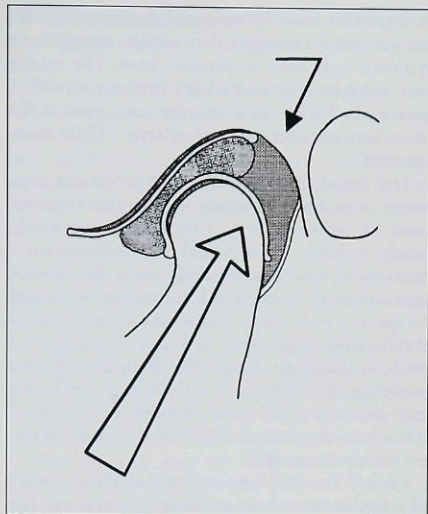

Fig 4   Compression of the condyle (large arrow) into the posterior attachment (small arrow), which will elicit a pain response if the tissue is inflamed.

Table 2   Objective Criteria Applied to Palpation

Grade	Palpation criteria
I	Patient feels slight tenderness, no obvious pain reaction is involved
II	Pain gives rise to a palpebral reflex
III	Pain gives rise to a protective reflex

(EAM). Palpation via the EAM may detect inflammation of the posterior attachment,[45] and it has been empirically used to detect a posteriorly positioned condyle, reciprocal clicking, or crepitus.

Provocative Tests

Passive joint play movements and their clinical applications are well known.[46] Procedures evaluating passive joint motion of the TMJ are valuable to the diagnosis of capsulitis and synovitis. Friedman and Weisberg[47,48] have described joint play assessment techniques. Manually assisted condylar distraction is useful as a provocative test for capsulitis because of its ability to stress the fibrous capsule (Fig 3). Manually assisted condylar compression (in the direction of the posterior

attachment) is useful as a provocative test for synovitis, since the movement forces the condyle backward against the inflamed posterior attachment (Fig 4).

Clenching activity can also serve as a provocative test and can assist the clinician in establishing the diagnosis of synovitis or capsulitis (Table 1). However, there is a small difficulty in relying solely on the findings of clenching activity. Muscular effort is required during clenching, so that this may evoke pain if there exists any pathologic change in the muscles involved (see case 1). This can be confusing because of the heterotropic nature of pain of some type of muscle disorders (eg, trigger points).

Treatment

Management goals for patients with capsulitis or synovitis are similar to those for patients with other joint-related disorders—namely, decrease pain, tenderness, and adverse loading; restore normal function; and allow the resumption of activities of daily living. Factors found to be etiological-

ly important must be managed during treatment, because those etiologies that initiate capsulitis or synovitis may also perpetuate them. The reader may want to refer to Parker's dynamic model of etiology in TMD[49] for a complete discussion of the important considerations of effective TMD management.

The initial management of noninfectious capsulitis or synovitis is similar to that prescribed for most any inflamed joint. If the condition is highly acute, aspirin, nonsteroidal anti-inflammatory medication, mild heat or cold, a soft diet, and/or instructions to limit mandibular movement should be quickly prescribed. In the case of synovitis, a stabilization appliance[50] may prove useful, as it tends to disengage the condyle from the inflamed posterior attachment. A stabilization appliance may also be used for either capsulitis or synovitis if it has been determined that relaxation of the elevator muscles is needed.[51]

Or and Yucetas[52] have reported on the treatment of synovitis by occlusal grinding of premature contacts. Current knowledge strongly disfavors this form of treatment. Sarnat and Laskin[53] state that, "adjustment of the occlusal surfaces of the teeth is absolutely contraindicated for the management of [synovitis]."

Agus et al,[54] Solberg,[55] Kopp and Wenneberg,[56] and others[57,58] have reported on the use of intraarticular injection of corticosteroids. Most supporters of this method agree that multiple injections (more than two or three a year or only one or two total injections in a lifetime—depending upon the dose and specific diagnosis) are to be strongly discouraged because of the likelihood of accelerated joint destruction.

Generally, treatment of capsulitis or synovitis includes mild cryotherapy to the area for 10- to 20-minute periods, followed by ultrasound or other physiotherapy. As the acute stage resolves, 20-minute applications of moist heat can be used to further reduce inflammation and associated muscle complaints. Instructions to the patient routinely include resting the jaw, maintaining a soft diet, and taking a mild analgesic (eg, aspirin). In the final stages of healing, mobilization and manipulation of the joint have been reported to be useful in accelerating tissue repair.[59]

Prognosis

The resolution of synovitis and capsulitis is usually uneventful. The clinical outcome and course of symptoms depends upon the etiology, but general-

ly is as short as a few days when the cause is uncomplicated or related to a single stressful event.

Complications of capsulitis or synovitis are well known. The possibility of hemarthrosis or deterioration in the quality of the synovial fluid presents the chance for adhesion formation between the disc and its bony compliment.[60] Rocabado[61] suggests the use of long-axis distraction as a measure to prevent adhesion formation. Capsular fibrosis or capsular contracture is another complication of capsulitis or synovitis. Kraus[62] proposes the use of long-axis distraction with a lateral vector added to maintain or regain the length of the capsule.[62]

Case Reports

The three patients described in this report were seen either at the private practice of the senior author or at the intern clinic of the Los Angeles College of Chiropractic. Each patient was given a thorough, new-patient workup. Specifically, an exhaustive history was obtained and there was a complete review of systems with a physical examination (including, but not limited to, eyes, ears, nose, throat, and neurologic tests—motor, sensory, reflexes). Relevant or contributory findings are extracted for this article.

Case 1: Capsulitis of Latent Onset

A 21-year-old man presented with mild to moderate right-side preauricular facial pain, joint noise, headache, and acute malocclusion. The patient described the preauricular pain as a focal, dull, and deep ache, approximately 2 to 3 cm anterior to the tragus of the ear, which had been present for almost 4 weeks. Approximately 2 weeks following the development of this pain, the patient noticed a change in his preexisting headache pattern, frequency, and quality. He further reported that at about the same time as the headache pattern changed, he noticed an abrupt change in his dental occlusion. He described the malocclusion as an inability to fully occlude the right posterior teeth. He reported that within the last week any attempt to force the right-side teeth together, or any other clenching activity, elicited a sharp, intense preauricular pain.

There had been no recent trauma. However, a history of nocturnal grinding was made known and the patient attributes this to the stress of rigorous college exams. A history of a painful ectopic eruption of the upper third molar several months before the initiation of his problem was also obtained.

The patient's previous history included cervicogenic headache and myofascial pain of the cervical and upper thoracic areas, for which manipulation and muscle stretching techniques were used successfully.

Examination. The TMJs, mandibular gait, dental occlusion, and muscles of the head and neck were thoroughly examined—in addition to the general assessment of the patient—to determine the nature of this patient's complaint. Listed here are those findings that are contributory to the patient's chief complaint, not the normal or "within normal range" findings.

Intraorally, the patient revealed a left mandibular deviation of 1 mm only at rest and a buccally erupted maxillary right third molar. Mild lock-and-key facetal indexing was noted on the incisal edge of the left anterior teeth (incisor to canine). Mouth opening greater than 31 mm was painful (in the area of chief complaint), as was left lateral excursion. Left lateral excursion was also limited due to the ectopic third molar. An occasional soft or muffled late opening click was observed on jaw opening. His right lateral pterygoid fossa was very tender (grade III) to palpation, and conventional, resisted-muscle testing of this muscle revealed pain and weakness, suggesting a mild intrinsic muscle spasm.

Focal, painful, taut bands with characteristic referral patterns —including referral to the TMJ— were detected in the masseter and the temporalis muscles, suggesting myofascial trigger points. Sustained compression of the trigger points also reproduced the patients headache complaint. Preauricular pain was also elicited when the patient was asked to clench the teeth.

Local pain (grade II) was evoked when palpating the lateral aspect of the TMJ. Manually assisted, condylar-compression provocation testing (into the posterior attachment) of the joint did not evoke pain, whereas the distraction provocation test (stretching of the capsule) reproduced the joint pain.

Diagnosis. The preauricular pain that was evoked with clenching was similar to what would be expected if synovitis were present. However, this differential diagnosis was eliminated when it was recognized that (*a*) this pain could not be reproduced during passive compression of the joint and (*b*) the preauricular pain following clenching was identical to the preauricular pain evoked when palpating the corresponding trigger point.

The findings of local pain upon palpation of the TMJ, with this pain being provoked by capsular distension, and the absence of historical or physical findings suggesting tumorous or infectious involvement indicated a diagnosis of simple capsulitis.

The classical findings on muscle palpation yielded the diagnosis of myofascial pain syndrome involving the right masseter and temporalis muscles, and the intraoral examination revealed a suspected mild intrinsic spasm of the right lateral pterygoid and the presence of an ectopically erupted third molar.

A treatment protocol was devised to address each diagnosis (ie, the muscular and TMJ pain) and to also address both presurgical and postsurgical care of the third molar.

Treatment. The patient was advised to start a soft, noninflammatory diet (eg, no alcohol, low animal fats and meat). Bromelaine, a pineapple extract, was recommended to aid in the reduction of inflammation.[63] Cryotherapy instructions (10 minutes per hour over the TMJ 6 to 8 times per day) were also given to the patient. Travell's stretch and vapocoolant spray technique was applied to the temporalis, masseter, and lateral pterygoid muscles.[64] Mild, rhythmic, distractive mobilization maneuvers were applied to the TMJ capsule. Ultrasound (pulsed at 0.5 W/cm^2) was applied to the masseter and anterior temporalis muscles, after which subsensory electrical stimulation (100 to 300 Hz) was applied, using the Myopulse (Electro-Medical, Fountain Valley, CA) device on all the painful sites.

Outcome. Therapy was followed for the 2 consecutive days preceding the oral surgery, wherein there was nearly a 100% reduction in the patient's extraoral symptoms. On the second day after the uneventful surgery, the patient returned, as expected, with exacerbation of all the complaints. Therapy was applied as described above for 2 consecutive days, and a significant reduction of symptoms was accomplished. The patient was then seen six more times at 2-day intervals, after which complete resolution of the chief complaint was achieved. Follow-up visits 4 and 7 months later confirmed the resolution of the presenting condition, and the patient was dismissed from further care. The patient reported that the nocturnal bruxing habit appeared to cease shortly following the removal of the third molar—an event also coinciding with reduction of his class load.

Case 2: Capsulitis of Traumatic Onset

A 24-year-old hockey player was hit on the right side of the jaw with a hockey stick. The patient described the pain as intense and it had worsened

in the days following the date of injury. The patient indicated that chewing was painful and that sleeping on the injured side provoked the pain. He also stated that his "back teeth do not touch properly." No history of bruxism or previous TMJ complaints was elicited.

Examination. Distension in the lateral joint capsule was observed upon inspection. The patient's pain increased substantially (grade III) with digital palpation over the lateral TMJ. Opening of the mandible past 10 mm gradually increased the pain. Mandibular gait was restricted in all planes of movement, due to the joint pain and local muscle splinting activity.

Intraorally, the patient revealed a left mandibular deviation of 3 mm at rest. His right lateral pterygoid fossa was mildly tender (grade I) to palpation, and the tissue palpated boggy and edematous. Testing of this muscle did not reveal significant pain or weakness.

Manually assisted, condylar-compression provocation testing (into the posterior attachment) of the joint did not evoke pain, whereas the distraction provocation test (stretching of the capsule) significantly reproduced the joint pain. There was no clinical evidence suggesting subcondylar fracture.

Diagnosis. These findings indicated a nearly classic case of traumatic capsulitis, with corresponding local muscle splinting. A treatment protocol was devised to address TMJ pain and decrease the impetus towards muscle splinting activity.

Treatment. The patient was advised to start a soft, noninflammatory diet. Bromelaine, a pineapple extract, was suggested in addition to aspirin (four 250-mg tablets/day) to aid in the reduction of inflammation. Cryotherapy instructions (10 minutes per hour over the TMJ 6 to 8 times per day) were also given to the patient. Mild, rhythmic, distractive mobilization maneuvers were applied to the TMJ. Ultrasound (pulsed at 0.5 W/cm^2) was applied to the surrounding area to stimulate blood flow, after which subsensory electrical stimulation (100 to 300 Hz) was applied, using the Myopulse device on the TMJ and surrounding tissues.

Outcome. A significant reduction in the patient's complaints was achieved within the patient's first two visits (each visit was 1 day apart). However, even with the dissipation of pain and other symptoms, the patient's care was continued twice weekly for 2 weeks due to the risk of intra-articular adhesion formation. Follow-up visits, 2 and 6 months later, confirmed the complete resolution of the condition, with no evidence of

intracapsular adhesions, and the patient was dismissed from further care.

Case 3: Synovitis (Retrodiscitis) Following Bruxism

A 22-year-old man presented with moderate right-side preauricular pain and acute malocclusion. The patient described the preauricular pain as a focal, sharp, and deep pain immediately anterior to the tragus of the ear, which had been present for 72 hours. He stated that his complaints "appeared to come out of nowhere" as he was driving home to San Diego on a Friday afternoon. He further reported that at about the same time as the pain developed, he noticed an abrupt change in his dental occlusion. He described the malocclusion as an inability to fully occlude the right posterior teeth. He reported that within the last 72 hours any attempt to force the right side teeth together, or any other clenching activity, elicited a sharp, intense preauricular pain.

A careful history revealed that this episode was the only occurrence, and there had been no recent trauma. The initial history revealed that the patient denied bruxing activity. However, a subsequent interview confirmed a strong bruxing tendency. The patient also revealed a history of obstructed nasal airway, and he was currently aware of a mouth-breathing habit.

Examination. The TMJs, mandibular gait, dental occlusion, and muscles of the head and neck were thoroughly examined—in addition to the general assessment of the patient—to determine the nature of his complaint.

Distension of the lateral joint capsule was observed upon inspection. The patient's pain increased substantially (grade III) with digital palpation over the lateral TMJ. Maximal opening of the mandible was 50 mm and did not evoke muscular pain. Mandibular gait was normal in all planes of movement, with right lateral excursion provoking mild preauricular pain. Inspection revealed an open mouth habitus, and an examination of the patency of the nasal airway revealed bilateral obstruction.

Intraorally, the patient revealed a left mandibular deviation of 2 mm at rest. His right lateral pterygoid fossa was mildly tender to palpation (grade II), and the tissues palpated boggy and edematous. Testing of this muscle did not reveal significant pain or weakness. Manually assisted, condylar-compression provocation testing (into the posterior attachment) of the joint significantly increased the pain of the chief complaint, whereas

the distraction provocation test (stretching of the capsule) did not alter the joint pain.

Examination of the dentition revealed a class II molar relationship. Palpation of the condyles during full closure subjectively suggested mild posterior condylar encroachment in the right TMJ.

Diagnosis. These findings indicated a working diagnosis of synovitis—primarily of the posterior attachment apparatus—of the TMJ. A treatment protocol was devised to address TMJ pain.

Treatment. The patient was advised to start a soft, noninflammatory diet. Aspirin (1 g per day) was prescribed, and cryotherapy instructions (10 minutes per hour over the TMJ 6 to 8 times a day) were given as an aid in the reduction of inflammation. Mild, rhythmic, distractive mobilization maneuvers were applied to the TMJ to increase intra-articular movement of fluids and prevent adhesion formation. Ultrasound (pulsed at 0.5 W/cm², using a Dexamethasone-coupling agent mixture (4 cm³ per 100 cm³) was applied to the surrounding area on only the first two visits. The following applications of ultrasound included the use of motor-level electrical stimulation (5 to 10 Hz) to assist in stimulating local circulation. The patient was provided with an intraoral device (Aqualizer—a temporary, water-filled appliance) to disengage the occlusion and relieve pressure on the TMJ.

Outcome. A significant reduction in the patient's complaints was achieved within the first three visits. However, even with the dissipation of pain and other symptoms, the patient's care was continued twice weekly for 2 more weeks because of the risk of intra-articular adhesion formation and the possibility of exacerbation due to bruxing.

Three important factors relating to treatment outcome were discussed with the patient. The first involved the dentition (ie, the possibility of insufficient vertical dimension on the right) and the second concerned the obstructed nasal airway (a sleep-lightening factor that is believed to be a necessary condition for bruxing[65]). The patient was advised that these components could be etiologic to this first-time episode of synovitis and, if proven to be so, would require etiologic-specific therapy if repeated or prolonged bouts of synovitis occurred in the future. The third factor was addressed by properly advising the patient that a singular bout of synovitis which responds quickly to therapy is not substantive enough evidence to predict the likelihood of future synovitis attacks, nor does it prove (or disprove) any etiologic relationship to

the dentition or obstructed airway. The patient declined further recommended examinations to pursue the etiology of this synovitis attack.

Follow-up visits, 2 and 6 months later, confirmed the complete resolution of the presenting condition, and the patient was dismissed from further care with instructions to report back to the senior author's office if the condition recurred.

Discussion

The intention of this article was to elucidate those conditions that may produce the complaint of preauricular pain and to focus on two common inflammatory changes involving the TMJ, capsulitis and synovitis. While these two localized inflammatory conditions of the TMJ are commonly seen in clinical practice, they must be differentially diagnosed from rarer and more serious conditions that also may cause pain in the region of the TMJ.

The review portion of this article investigated the clinical characteristics, etiology, physical examination methods, treatment, and prognosis for capsulitis and synovitis. It is acknowledged that any attempt to distinguish capsulitis from synovitis may be confusing to some clinicians. Synovitis may occur in either the inner lining of the capsule and/or the posterior attachment apparatus, since both of these structures contain synovial tissue. Further, in this strict sense, synovitis of the inner capsular lining cannot be distinguished, clinically, from inflammation of the fibrous portion of the capsule.

However, every attempt was made to follow the nomenclature established by the IHSC as it pertains to TMD. The term synovitis, as used throughout this article, followed the diagnostic criteria as described by the IHSC (IHSC no. 11.7.5.1; ICD no. 727.09).

The three case reports illustrated acute presentations of these conditions and demonstrated how these conditions may respond quickly to early intervention using effective conservative measures. Reasonable follow-ups were made on each of these patients to confirm the complete resolution of the presenting condition before the patient was dismissed from further care.

Additionally, the cases illustrate the careful and concise management that can be offered in a chiropractic setting.

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Resumen

Manejo Quiropráctico de la Capsulitis y la Sinovitis de la Articulación Temporomandibular

Las condiciones inflamatorias localizadas (p. ej., sinovitis y capsulitis) de la articulación temporomandibular (ATM) son vistas comunmente en la práctica clínica. Sin considerar la frecuencia de su ocurrencia, estas condiciones deben ser diagnosticadas diferencialmente de las que también pueden causar dolor en la región de la ATM. La capsulitis o la sinovitis deberían ser consideradas si tal dolor está presente, y los hallazgos semiológicos, físicos y de laboratorio no indican fenómenos de dolor referido o sistémico, tumoral o un compromiso infeccioso. Este artículo revisa las características clínicas, la etiología, los métodos de examen físico, tratamiento y pronóstico de la capsulitis y la sinovitis. Se reportan tres casos que ilustran estas condiciones.

Zusammenfassung

Chiropraktische Behandlung bei Kapsulitis und Synovialitis des Kiefergelenkes zur Linderung von Kopf- und Gesichtsschmerzen

Lokal entzündliche Zustände des Kiefergelenkes (z. B. Synovialitis und Kapsulitis) sind in der Praxis keine Seltenheit. Ungeachtet der Häufigkeit ihres Vorkommens müssen diese Zustände differentialdiagnostisch unterschieden werden von andern schmerzhaften Sensationen im Bereich des Kiefergelenkes. Wenn lokaler Schmerz im Kiefergelenk vorliegt und Anamnese, klinischer Befund und allfällige Laborbefunde nicht auf systemische, tumoröse oder infektiöse Erkrankungen hinweisen, müssen lokale Ursachen wie Synovialitis oder Kapsulitis in Betracht gezogen werden. Dieser Artikel zeigt klinische Charakteristika, Aetiologie, Untersuchungsmethoden, Therapie und Prognose für Synovialitis und Kapsulitis anhand dreier Fallpräsentationen auf.