TM Disorders: Diagnostic Classification of Temporomandibular Disorders

(Part 2 of 3)[i]

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The head, face, masticatory sytem, and cervical region are common sites in which pain is experienced. Many conditions present with similar signs and characteristic patterns that may lead to diagnostic confusion and ultimately misdirected care. Defined, validated classification systems relating to the multiplicity of painful entities can simplify and enhance diagnostic outcomes. Due to the rapid advances in our knowledge regarding pain mechanisms and pathways, classification systems must be ever evolving, not rigid. Presently an ideal system related to masticatory system disorders does not exist.

One set of diagnostic criteria will not satisfy all circumstances to which it might be applied. More importantly, many classifications systems were developed for the purpose of enhancing the formation of study populations for clinical research endeavors and are not absolutely applicable to every clinical case presentation.

For example, the inclusion criteria for a clinical trial might require the presence of all criteria for a specific disease, while a clinical diagnosis might require the presence of only a few. These criteria are meant only to provide clinical guidance for diagnosis. Final diagnostic decisions must be based on the clinical judgment of the health care professional. This article will provide the reader with a review of the most accepted diagnostic classification system related to temporomandibular disorder (TMD).

It is generally recognized that two basic categories of TMD exist, extracapsular (myogenous) and intracapsular (arthrogenous). The majority of TMDs are extracapsular in nature; however, it is not uncommon for these two basic categories to co-exist.

Masticatory muscle-related conditions are found to be the most common subgroup of TMD.[1,2]

The current understanding of the complexity and the dynamic relationship between the masticatory and cervical musculature enables the practitioner to better assess the condition(s) possible etiology(ies). The individual variations and demands placed on the system, as well as normal function while awake or sleep, are true considerations in our patient evaluation.

Myofascial pain is a regional pain, usually dull and achy with the presence of localized tenderness in firm bands of muscle, tendons and/or fascia that reproduce pain when palpated and may produce a characteristic pattern of regional referred pain and/or autonomic symptoms on provocation.[3,4] Patients may complain of muscle stiffness, acute malocclusion, ear symptoms, tinnitus, vertigo, toothache, tension-type headache and masticatory muscles involvement. The most common differential diagnoses to consider includes osteoarthritis, myositis, myalgia, neoplasia and fibromyalgia.

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Myositis is inflammation of a muscle due to local causes such as infection or injury. Pain is usually acute and in a localized area with localized tenderness over the entire region of the muscle. The inflammation can occur also in the tendinous attachment of the muscle, "tendonitis or tendomyositis". Increased pain with mandibular activity with alteration in function due to inflammation or pain. Swelling, tissue reddening and an increase in temperature over the entire muscle can be noticed. The most common differential diagnoses to consider includes myositis, local myalgia-unclassified and myofascial pain.

Myospasm is an involuntary, sudden, continuous (fasciculation) tonic contraction of the muscle. Previously used terms are trismus, "cramp". A muscle in spasm is acutely shortened. The patient experiences acute pain, a limited range of motion and often acute malocclusion. EMG studies verify sustained muscle contraction even at rest.[5] The most common differential diagnoses to consider includes myositis, local myalgia-unclassified and neoplasia.

Local Myalgia-Unclassified This category includes muscle pain secondary to ischemia[6], bruxism[7], fatigue, metabolic alterations, delayed onset muscle soreness, autonomic effects and protective splinting (co-contraction).[8] Although there is significant evidence that these conditions exists, there are few reliable clinical characteristics that can be used to distinguish them from each other.

Myofibrotic contracture refers to the painless shortening of a muscle. Previous terms used include chronic trismus, muscle fibrosis and muscle scarring. It is a chronic resistance to a passive stretch as a result of fibrosis of the supporting tendons, ligaments or muscle fibers themselves. The patient usually does not complain of pain unless the muscle is extended beyond its functional length. There are two basic subcategories: myostatic (reversible condition) and myofibrotic (irreversible condition). Clinical characteristics include a limited range of motion, unyielding firmness on passive stretch and a history of trauma or infection is usually reported by the patient. The most common differential diagnoses to consider includes TMJ ankylosis and coronoid hypertrophy.

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Masticatory muscle neoplasia can be benign or malignant and may be associated with pain or not. Neoplasia is defined as a new, abnormal or uncontrolled growth of muscle tissue (eg, myxoma). Confirmation must be obtained by biopsy and imaging.

Congenital or developmental disorders Most congenital or developmental disorders are not associated with orofacial pain. They can be categorized as agenesis, hypoplasia, hyperplasia and neoplasia.

Neoplasia, a new, often uncontrolled growth of abnormal tissue, and in this case arising in or involving the TM joint. Neoplasms can be categorized as benign, malignant or metastatic from a distant site. Approximately 1% of malignant neoplasia metastasize to the jaws.[9,10] Squamous cell carcinomas of the head and neck region, nasopharyngeal tumors, neoplasm arising from the parotid gland (adenoid cystic carcinoma) and mucoepidermoid carcinomas have been reported to extend to the TMJ region resulting in pain and alteration of normal function.[11,12] Dysfunction is not usually caused by neoplasm.[13]

Primary tumors known to have involved the condyle include osteoma, benign osteoblastoma[14], chondroma and chondrosarcoma, benign giant cell tumor, ossifying fibroma, fibrous dysphasia and myxoma.[15] Malignant neoplasm have been reported originating from the temporomandibular joint space (fibrosarcoma, synovial sarcoma).[16,17,18]

Congenital or developmental disorders of the cranial bones and mandible includes aplasia (agenesis), hypoplasia, hyperplasia and neoplasia. Lesions and disorders of the jaws can be either odontogenic or non-odontogenic in origin and generalized or metastatic in nature. Most congenital or developmental disorders primarily cause problems with esthetics or function and are rarely accompanied by orofacial pain unless associated with Neoplasia (eg, osteomyelitis, multiple myeloma, Paget's disease). Complete agenesis is extremely rare.[19]

Aplasia is a faulty or incomplete development of the cranial bones or mandible. Most of the aplasias conditions of the mandible are categorized under hemifacial microsomia syndromes. The auditory system is frequently affected in these syndromes

Hypoplasia is the incomplete development or underdevelopment of cranial bones or the mandible that is congenital or acquired. The growth is considered normal but proportionately reduced. Treacher-Collins Syndrome is an example of incomplete development.[20] Condylar hypoplasia can occur secondary to trauma, resulting from incomplete or underdevelopment of the mandibular condyle.

Hyperplasia is the overdevelopment of the cranial bones or the mandible. This can be developmental or acquired. Hyperplasia can occur as a localized enlargement, such as in condylar hyperplasia or coronoid hyperplasia, or as an overdevelopment of the entire mandible or side of the face. Fibrous dysplasia is a form of hyperplasia due to a benign, slow growing swelling of the mandible and/or maxilla. It is characterized by the presence of fibrous connective tissue.

The disease occurs in children and young adults and becomes inactive when they reach skeletal maturity. Radiographically the lesion may appear from an opaque ground-glass to a lucent appearance, depending on the ratio of fibrous tissue to bone. Clinically, usually there is no displacement of teeth and the cortical bone and occlusion remain intact.

Disc derangement disorders are an abnormal arrangement of intra-capsular joint parts causing interference with the structural relation during mandibular condyle translation with mouth opening and closing. In the TM joint this alteration can relate to the elongation, tear or rupture of the capsule or ligaments causing a disruption in the disc position or morphology. The subclassification of disc displacement represents a disc-condyle misalignment and is subdivided into disc displacement with reduction or disc displacement without reduction.[21–23]

Disc displacement with reduction is characterized by the "temporary" alteration or inter-

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Editorial Note

Some of the editorial corrections intended for Part I of this article in *Dental Tribune Asia Pacific*, No. 9 Vol. 5, September 2007 did not make into the final print. Please e-mail r.goodman@dental-tribune.com for a corrected PDF of this article, which will also be posted to our Web site for those subscribers who have access. The publisher sincerely regrets the errors.

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ference of the disc-condyle structural relationship during mandibular translation resulting in an opening joint sound, for example clicking or popping. A reciprocal closing noise is usually of less magnitude and is thought to be produced by the displacement once again of the disc (to its original position) in usually an anterior or anteromedial position.[24]

It has been theorized that the momentary misalignment of the disc is due to articular surface irregularities, disc-articular surface adherence, synovial fluid degradation and disc/condyle incoordination as a result of abnormal muscle activity or disc deformation. Although the concept of natural progression has been suggested, there is currently no convincing evidence that TMJ clicking typically progresses to locking and degeneration or that arthritic changes must develop[25,26], probably demonstrating a normal physiological response.[27,28,29]

Diagnostic criteria include: reproducible joint noise usually at variable position (opening, closing), soft-tissue imaging confirms a displaced disk that improves its position during jaw opening and hard tissue imaging will demonstrate absence of extensive degenerative bone changes. Pain may be precipitated by joint movement and deviation during movement coinciding with a click.

Disc displacement without reduction, or "closed-lock", is described as an altered or misaligned disc-condyle structural relationship that is maintained during mandibular translation. It is characterized by a lack of joint noise and limited jaw motion (opening <35 mm), mandibular deflection to the affected side (if not bilateral), soft-tissue imaging reveals disc displaced without reduction and hard-tissue imaging reveals no extensive osteoarthritic changes.

Patient may experience pain precipitated by forced mouth opening. A history of clicking that ceased with the occurrence of locking, ipsilateral hyperocclusion (during acute stage) and occasionally hard-tissue imaging can reveal moderate osteoarthritic changes. Studies on the progression of the disease have demonstrated very few reducing displaced disc cases progressing to a non-reducing stage, but almost all the non-reducing displaced disc cases developed structural bone changes.[30]

Joint dislocation, or "open-lock", is characterized by the condyle and usually the disc position anterior to the articular eminence and unable to return to a closed position without a specific manipulation. Elevator muscles activity and/or a true hyperextension of the disc-condyle complex may be responsible for the patient's difficulty in returning to a normal position. A temporary dislocation that can be reduced by the patient is referred to as subluxation. Patient usually reports a history of excessive range of motion (hypermobility) that is not painful, but pain can occur at the time of dislocation with mild residual pain after the episode. Radiographic evidence reveals the condyle well beyond the eminence. The most common differential diagnosis to consider is fracture.

Inflammatory conditions can occur as localized synovitis, capsulitis or retrodiscal tissues of the temporomandibular joint that can be due to infection, an immunologic condition secondary to articular degeneration or trauma. Clinically it is difficult and may be impossible to differentiate between these. Diagnostic criteria must include localized TM joint pain exacerbated by function, especially with superior or posterior joint loading on palpation. No evidence of extensive osteoarthritic changes with hard-tissue imaging. Additional clinical findings may exist, such as localized pain at rest, limited range of motion secondary to pain, fluctuating swelling (due to effusion) causing a decrease in the ability to occlude on ipsilateral posterior side and ear pain.

The most common differential diagnoses include: osteoarthritis, polyarthritis, ear infection, neoplasia, generalized systemic polyarthritic condition, rheumatoid arthritis, juvenile rheumatoid arthritis (Still's disease), spondyloarthropathies (ankylosing spondylitis, psoriatic arthritis, infectious arthritis, Reiter's syndrome), crystal-induced disease (gout, hyperuricemia), and autoimmune disease and other mixed connective tissue diseases (lupus erythematous, sclero-

derma, Sjögren's Syndrome). Polyarthritides are characterized by pain during acute and subacute stages, possible crepitus, limited range of motion secondary to pain and/or degeneration and bilateral radiographic evidence of structural bony changes.[31] The complexity of the disease mandates serology studies and management by a rheumatologist. Bilateral resorption of condylar structures can result in an anterior open bite.

Osteoarthritis is considered a non-inflammatory arthritic condition that is commonly found in synovial joints. Osteoarthritis is classified according to the etiology of the condition. It is divided into a primary and secondary non-inflammatory arthritic condition.[32] The recognition of secondary osteoarthritis is clinically significant because it may represent the first stage of treatment.

Osteoarthritis (primary) is a degenerative non-inflammatory condition of the joint characterized by deterioration and abrasion of the articular tissue and concomitant remodeling of the underlying subchondral bone due to overload on the remodeling mechanism.[33–36] Osteoarthritis is categorized as primary on the absence of identifiable etiologic factors.[37]

Clinical characteristics include: pain with function, point tenderness with palpation, limited range of motion with deviation to the affected side on opening and crepitus or multiple joint noises. Radiographically, evidence of structural bony changes (subchondral sclerosis, osteophytic formation, erosion). Pain and dysfunction can vary depending on the degree of inflammation and morphologic changes. Studies suggest that the course of the disease usually progresses favorably; allowing remodeling and adaptation. Treatment must be rendered on a case specific basis depending upon the degree of pain and dysfunction.[38] The most common differential diagnoses to consider: inflammation, polyarthritis, neoplasia.

Osteoarthritis (secondary) is a degenerative condition of the joint characterized by deterioration and abrasion of the articular tissue and the concomitant remodeling of the underlying subchondral bone due to a prior event or disease that overload the remodeling mechanism. Clinical characteristics include: a clearly documented disease or event associated with osteoarthritis, pain with function, point tenderness on palpation, limited range of mandibular motion with deviation to the affected side on opening and crepitus or multiple joint sounds. Potential etiological factors include direct trauma to the TM joint (traumatic arthritis), local TMJ infection or history of active systemic arthritis (eg, rheumatoid arthritis).

Ankylosis is clinically characterized by the restriction of a mandibular movement with deviation to the affected side on opening and is usually not associated with pain.[40] Fibrous adhesions occur mainly in the superior compartment of the TM joint, affecting the translation movement of the affected condyle. Adhesions can occur secondary to joint inflammation resulting from trauma or systemic conditions such as polyarthrotic disease. Bony ankylosis can lead to a complete immobilization of the TM joint. Clinically, evidence of bone proliferation is appreciated radiographically. Patient demonstrates deviation to the affected side and significant limited movement to the contralateral side.

Fracture is direct trauma to the mandible and may result in fracture to the condylar process. All related components of the masticatory system—soft tissue, disc, capsule, synovium, retrodiscal tissue, ligaments, and/or articular surface—may also be affected. Condylar fractures are usually unilateral and may occur in the condylar neck or in the capsule (intra- or extracapsular) with or without displacement. Location of the fracture and degree of the fracture will determine the direction of displacement. A displacement anterior-medial-inferior usually results due to the action of the lateral pterygoid muscle. Clinical characteristics include: associated trauma, preauricular pain and swelling (synovitis, capsulitis), limited opening, and if the condylar fragment is displaced, occlusal changes and deviation to the affected side. The development of adhesions and osteoarthrosis are common findings implicated in condylar fractures.[39, 40] DT

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