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 include osteoarthritis, myositis, myalgia, neoplasia and fibromyalgia.

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 also can occur in the tendonous attachment of the muscle, “tendonitis or tenomyositis.”

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 include osteoarthritis, myositis, local myalgia-unclassified and fibromyalgia.

Myospasm is an involuntary, sudden, continuous (fasciculation) tonic contraction of the muscle. Previous 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 include myositis, local myalgia-unclassified and neoplasia.

Local myalgia — unclassified

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.

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 (e.g., myxoma). Confirmation must be obtained by biopsy and imaging.

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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. 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, chondroma and chondrosarcoma, benign giant cell tumor, ossifying fibroma, fibrous dysplasia and myxoma.14 Malignant neoplasms have been reported originating from the temporomandibular joint space (fibrosarcoma, synovial sarcoma).14,15,16 Congenital or developmental disorders of the cranial bones or mandible include 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 (e.g., osteomyelitis, multiple myeloma, Paget’s disease). Complete agenesis is extremely rare.17 Aplasia is a faulty or incomplete development of the cranial bones or mandible. Most of the aplasias conditions of the mandible are categorized as bone and occlusion remain intact. Hypoplasia is the incomplete development or unopposed growth 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 capsular ligaments causing a disruption in the disc position or morphology. The sub-classification of disc displacement represents a disc-condyle misalignment and is subdivided into disc displacement with reduction or disc displacement without reduction.21

Although the concept of natural joint derangement has been suggested, there is currently no convincing evidence that TMJ clicking typically progresses to locking and degenerative joint disease.22–24,92–24,92 Joint derangement 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.

Inflammatory conditions can occur as localized synovitis, capsulitis or retrodisccal lesions of the temporomandibular joint that can be due to infection, an immunologic condition secondary to a particular degeneration or trauma. Clinically it is difficult and may be impossible to differentiate between these. Diagnostic criteria for an arthritic 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 articular eminence on ipsilateral posterior side and ear pain. The most common differential diagnoses include: osteoarthritis, polyarthritis, ear infection, neoplasia, general systemic polyarthritis, ear infection, neoplasia, polyarticular 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 erythematosus, scleroderma, Sjögren’s Syndrome).

Osteoarthritis is 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.11 The complexity of the disease mandates serology studies and is managed 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.