

The Temporomandibular Joint and Related Orofacial Disorders

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Etiology

Etiology is the theory or study of the causation of a disease. Because there are many possible etiologies for TMD, a thorough history is crucial for making a decision. After a review of the patient's history, the clinician must decide if one factor or several factors seem plausible. Arriving at this decision is not easy.

Most TM disorders are idiopathic, that is, of unknown origin. Some TMD complaints have been associated with serious organic diseases. Yet the factors that make a person prone to developing TMD have not been carefully identified. Some factors that appear causal occur coincidentally in other individuals. Even in frank cases of pathology, little reliable information exists for differentiating joint changes from muscular changes during the natural course of development of TMD.

Single Versus Multifactorial Etiologies

Historically, the single etiology versus the multifactorial etiology abounds in controversy. This controversy continues even though few studies find a single etiologic factor. Extensive review of the literature suggests that a multifactorial etiology is the most reasonable solution. Speculation is that distinct etiologies may be identified once an empiric classification for diagnosis is found.

Recently, an attempt has been made to clarify the single versus the multifactorial issue. The authors agreed on the multifactorial hypothesis, and factors including external trauma, intubation during general anesthesia, difficult dental extractions, and orthodontic procedures were evaluated in 195 TMD cases and 50 non-TMD controls. Positive associations were found for TMD between the occurrence of factors versus no factors, between two or more factors versus one or no factor, and between multiple factors versus a history of general anesthesia. A trend was found between TMD and trauma with multiple factors versus single factors. No significant associations were found between TMD and the presence of one factor and between multiple factors and extractions.

Potential etiologic factors can be conceptualized as a continuum from the least severe to the most severe changes affecting the masticatory system. The least severe factors might include acute microtraumatic events, whereas the most severe factors would include degeneration of the TMJs.

The kinds of factors contributing to TMD can be divided into predisposing, precipitating, and perpetuating factors (Table 4-1). All must be regarded as potential factors. Predisposing factors are factors occurring naturally during the lifetime of an individual.

Precipitating factors are initial events leads to onset of symptoms. Perpetuating factors are repetitive events that tend to create a continuing cycle of symptoms. Distinguishing one group of factors from another is not always easy. Considerable overlap may occur between factors.

Table 4-1. Factors That Contribute to TMD

Predisposing

Anatomic
Pathophysiologic

Precipitating

Microtrauma
Macrotrauma

Perpetuating

Behavioral
Emotional.

Predisposing Factors

Several predisposing factors have been discussed in the literature. They primarily include anatomic and pathophysiologic conditions in the person's life.

Anatomic Conditions

Review of the etiology of anatomic conditions will find many possible etiologies. Some of the earlier literature viewed structural deformities as potential contributing factors. These factors mainly involved developmental abnormalities of the jaws. Most reports recognized abnormal maxillomandibular relations, dental or skeletal malocclusions, malalignment of the mandible, and improper position of the head and the spinal column.

Attempts to identify anatomic factors in the etiology of TMD are fraught with problems. Confusion arises because of difficulties in classifying different conditions. For example, the same morphologic variants have been classified as tumors, congenital anomalies, and disturbances of postnatal growth. Most conditions are now classified on a morphologic basis because little to no information exists about anomalies.

Nonetheless, a thorough review has been conducted of the relation between facial anomalies and development of the TMJs. Various prenatal and postnatal causes were discussed, but few etiologic factors were mentioned. None was related to TMD.

An impressive study has been conducted on the anatomy of the TMJs of young adults. The study critically evaluated 95 autopsied joints of subjects in an age group (mean age 26.4 years) that comprises the largest segment of the population seeking TM treatment. These individuals might be considered the most susceptible to physical changes within the joint. Joints were examined for deviations in form (DIF), arthrosis, size, shape, and possible disk displacement. DIF was defined as a deviation from the normal rounded contours of the temporal and condylar surfaces and as deformation of the disk.

Most gross changes in DIF were attributable to adaptive phenomena occurring in response to articular fit and function. Most joints (83 of 95) had intracapsular changes, and 39% had mild-to-marked DIF of the three components, with condylar changes the most prevalent. Three percent showed arthritic lesions, and 12% showed disk displacement. Disk displacement was significantly higher in women than in men. It was greater overall when the disk was deformed or folded. The authors did not fully agree that such changes represented precursors to arthropathy.

Morphologic Malocclusions

Certain skeletal and dental malocclusions have been suggested as causing neuromuscular disturbances in the masticatory system, including TMJ disorders. The aberrant functions may be either a direct result of the morphologic malocclusion or an indirect result of functional occlusal interferences. Potential causes have been categorized into vertical, horizontal, and lateral discrepancies. Recognition of these categories is particularly useful if one accepts the theory that malocclusion plays a dominant role in TMD symptomatology (Table 4-2).

Table 4-2. Extra-Articular Skeletal and Dental Causes Leading to TMD

Vertical discrepancy

Lack of posterior tooth support
Molar or bicuspid fulcrum

Horizontal Discrepancy

Anterior slide or anterior posturing
Distal displacement

Lateral Discrepancy

Functional side shift
True vertical asymmetry.

Classically, individuals with Angle class II, especially division 2, have been considered particularly vulnerable to TMD. Because of excessive overjet, it has been suggested that these individuals habitually posture the mandible during protrusion movements. Presumably, this overuse causes chronic strain of the muscles and may be harmful to the TMJs. These individuals have been cited as commonly represented in many populational studies. Other findings tend to support these conclusions. Morphological malocclusion, class II and class III occlusions, frontal open bite, and cross bite, if correlated with functional malocclusion, may predispose to dysfunction. A rather weak relation between dysfunctional symptoms and Angle malocclusions was demonstrated in children by regression analysis.

Skeletal and dental variations and functional changes in the TMJs have been evaluated in patients examined from a private practice population. Clinical examination and cephalometric analysis of 33 variables were conducted on 62 patients with confirmed internal derangement of the TMJs. Data were compared with identical parameters of 102 matched controls from the general population at large. Analysis showed no statistically significant

differences in proportion of subjects in any Angle class, or in dental or occlusal parameters between the groups.

Several variables of morphologic malocclusion and anatomic characteristics have been studied in the TMJs of young adults. Most associations proved age dependent. Autopsy of 96 cadavers showed that certain deviations in form (DIF) of the temporal bone, condyle, and position of the disk correlated significantly with some variables of malocclusion. Angle classes II and III dentitions correlated with DIF, and class II dentitions correlated with histologically evident morphologic changes in the TMJs. Cross bite was related to increasing presence of DIF in the three anatomic areas. Anterior cross bite correlated with DIF of the articular eminence.

Furthermore, abnormal overbite and overjet were associated with condylar DIF. Deep overbite was related to flat condyles and wide fossae. Excessive overjet was associated with DIF of the disk, particularly displacement. The authors argued that TMJ changes become more extensive the longer the exposure to malocclusion. They alluded to an association between malocclusion and TMD symptoms and noted that previous authors concluded these associations were weak.

Significance: The uncertainty of a precise definition for Angle classification, coupled with the findings of other investigations, weakens an argument that Angle malocclusion and a bevy of TMD symptoms are closely related. No definitive correlations have been made between severity of morphologic malocclusion and TMD.

Functional Malocclusion and Occlusal Disharmonies

When clinicians define malocclusion as a change in bite position caused by occlusal disharmonies, the literature on TMD symptoms is replete with conjecture. Different kinds of occlusal interferences have been argued as being significant or insignificant in causing symptoms. The absence of any well-defined relation does not agree with the philosophy of some clinicians who consider that many TMD problems originate from occlusal interferences.

A consensus statement issued by a group of leading health care practitioners confirms a previous statement that an association between occlusal factors and certain symptoms of TMD is weak. Additional evidence refutes this single factor etiology. A study of many occlusal parameters and symptoms concluded that occlusion alone could not be responsible for etiology of TMD. Stepwise logistic regression analysis of specific occlusal factors did not prove valuable in deciding whether specific symptoms would develop. These findings have been supported by other studies. A dysfunctional index system, devised from combinations of structural and neuromuscular factors, showed that structural factors are frequently related to some symptoms but that neuromuscular factors accounted for the head pain.

A comparative study of 260 nursing students showed that differences in occlusion played no major role in TMD symptoms. The students were diagnosed into the following subgroups: normal, joint disorder, muscle disorder, or joint and muscle disorder. Regression analysis revealed positive associations between the degree of dysfunction and parafunctional habits for the normal, muscle disorder, and joint and muscle disorder subgroups. Positive associations were found between dysfunction and emotional stress in the muscle subgroup.

The authors concluded that any relation between potential etiologic factors and dysfunction depended on whether the subjects had muscle or joint disorders.

Significance: Although practitioners may conclude that occlusal disharmonies affect TM symptoms, the variation among studies that no major change can be expected.

Malalignment of the Mandible

Some clinicians consider joint dysfunction as primarily a postural problem. They identify symptoms with malalignment of the condyle in the glenoid fossa. The position of the articular disk is considered deranged if the condyles are positioned improperly. Presumably, the dysfunction derives from a developmental problem concomitant with a dental malocclusion. According to this theoretical cause, an unstable occlusion resulting from either premature tooth contacts or parafunctional oral habits causes malalignment of the mandible. Usually posterior displacement of the mandible results in compression of soft tissues. Undue stress is placed on muscles, ligaments, and bones. Pain, impairment of the blood supply, and degeneration of the joint follow. Based on this malalignment, TMD and malocclusion have been considered synonymous.

This concept has remained popular. As late as 1990, a paper appeared in a leading journal supporting this concept. Condylar displacement originating from occlusal changes was endorsed as the probable cause of TMD, vertigo, tinnitus, and associated auditory symptoms. Several recent papers were listed in the bibliography; none supported a relation between condylar displacement, occlusal prematurities, and TMD. Recognition of these symptoms resurrects Costen's syndrome, a historical relation no longer considered meaningful or valid.

The argument for condylar displacement as an etiology for TMD contrasts sharply with radiologic findings on condylar position in asymptomatic individuals. A critical analysis of 46 tomographs selected from a population of 441 individuals found that a diagnosis of dysfunction could not be based entirely on radiography of nonconcentric condyle-fossa relations. Evaluation of eight additional studies confirmed an unidentifiable relation. Some patients in these studies were symptomatic, and judgments made from transcranial radiographs did not allow for variations in angulations of the condyle.

Another radiographic study of condyle position showed the subjective nature of interpretation. Interexaminer reliability and relation with clinical parameters were studied from transcranial radiographs and tomographs. Observed eccentricity was not related to symptoms, clicking, or crepitation.

Significance: A conclusion that condylar displacement caused by altered occlusion leads to TMD symptomatology must be regarded cautiously. Statements that TMD and malocclusion are synonymous have little merit.

Improper Position of the Head and Spinal Column

The older literature on TMD is filled with descriptions of an orthopedic approach to management. Claims range from a need to clarify joint structure and neuromuscular function to correcting either occluso-muscle imbalance or jaw position. The modern literature has

perpetuated many of these claims. The recognition that faulty curvature of the cervical spine may contribute to TMD has been especially noteworthy. Some clinicians may become confused by symptoms that arise from the neck and not the TMJs.

The position of the head is guided by information received from sensory innervations of the neck. Muscles of the dorsal neck region have rich sensory innervations, contain a high number of muscle spindles and Golgi tendon organs, and have many small afferents that conduct pain. This sensory apparatus provides the central nervous system with vital information about the position of the head.

Because the TMJs are anatomically close to the spine, it is logical to argue that the complex of TMJ symptoms includes head, neck and shoulder problems. There is unsubstantiated opinion that subjects presenting with forward head posture and protracted shoulders are prone to TMD. Although poor mechanics between the head and neck may contribute to TMD-like symptoms, no well-controlled studies have been done.

Postural problems were assessed in 164 patients with myofascial pain of the head and neck. The examinations were conducted under conditions considered standard in physical therapy. Most patients had postural problems (Table 4-3). This study would have been more meaningful if comparable data had been available on subjects without myofascial pain matched for age and gender.

Table 4-3. Percentage of Postural Problems in 164 Patients With Myofascial Pain

Postural Problem	Patients (%)
Poor sitting or standing posture	96.0
Forward head tilt	84.7
Rounded shoulders	82.3
Poor tongue position	67.7
Abnormal lordosis	46.3
Scoliosis	15.9
Leg length discrepancy	14.0

Significance: The relation of these structural deviations to TMD remains controversial. Unfortunately, overlap in symptoms between the jaw and the neck has led to some exaggeration of treatment outcome. A letter to the editor of a leading TMJ journal typifies this kind of thinking. The letter stated that "patients with scoliosis, especially those who are asymptomatic, have a highly complex compensatory mechanism functioning in their bodies. Dental procedures tend to change the proprioceptive balance by either changing TMJ orientation or by totally stabilizing TMJ migration and therefore eliminating further craniomandibular compensation to further bodily change." Although dental or TMJ treatment of some patients with scoliosis may be compromised, such statements are questionable.

Pathophysiologic Conditions

Most etiologic theories concerned with the source of TM symptoms focus either on pathology of the joints or on changes in the masticatory muscles. A complete health history

followed by a screening radiographic examination may lead to the source of potential TM pathology.

TMJ pathology may derive from various sources. Intra-articular disorders such as internal derangement, osteoarthritis, and inflammatory arthritis have been widely discussed in the literature. There is an ample literature on extra-articular pathology, including muscular, vascular, and neurologic disorders. Neoplastic disease has been addressed.

Joint Disorders

Internal Derangement. Internal derangement of the TM joint is defined as an abnormal relation of the articular disk to the mandibular condyle, fossa, and articular eminence. It has been used to describe problems associated with the movement of the disk between the head of the condyle and the eminence. Joint noise or a history of joint sounds is the key symptom of this disorder. The kinds of joint noises are described in another section of the text.

No aspect of TMD inquiry has become more controversial than the etiology of internal derangement. Little is known about predisposing factors, although numerous precipitating factors have been postulated, including the following: macrotrauma to the joint by impact or hyperextension injuries, microtrauma resulting from occlusal changes, chronic muscular hyperactivity, meniscal perforation secondary to muscle spasm, secondary ligamentous laxity with discontinuity, infection, and arthritis.

The symptoms of internal derangement often mimic symptoms present in patients with myofascial pain. Myofascial pain dysfunction (MPD) syndrome is stress related, and some clinicians have not recognized emotional stress as leading to TMD. They argue that most TMD symptoms derive from internal derangements. The clinical features of internal derangement are essentially similar to features of MPD syndrome or TMJ arthropathy. Malposition and altered position of the disk have been proposed as the basic lesions responsible for the clinical manifestations of these conditions. This argument has been based on an almost 100% correlation between disk displacement and the presence of signs and symptoms, on clinical expression in cases proportional to the degree of pathology found, and on findings of inflammation and mechanical signs and symptoms consistent with pathophysiologic mechanisms.

On the other hand, the view that internal derangement is the primary etiology has been criticized as too narrow. According to one noted clinician, "This is not to say that some percentage of patients with derangements will not develop more advanced problems or even have severe problems initially, but no one knows how large the subgroup is. Most clinicians recognize muscular disorders as distinct from intrinsic joint derangement.

Highly sophisticated studies that TMD symptoms derive from internal derangements. Analyses of the TMJs of adult cadavers indicated that there is a progression of events during internal derangement. Changes in both disk position and disk configuration have been found. The most common disk displacement was anteromedial, but posterior displacement also occurred. Oblique position with biconcave disks or disks of even thickness progressed to

complete displacement with disks of biconvex configurations. Perforations were found in advanced stages. Advanced disk displacement characterized cases of osteoarthritis.

Three models have been proposed as causing disk displacement: hyperextension of the mandible, condylar displacement caused by trauma, and chronic muscular hypertonicity. In the first model, the mandible is hyperextended by a sudden event or a series of events with ultimate displacement. Once the displacement occurs, arthralgia ensues and protective muscular splinting develops. The second model presumes that the condyle is displaced superiorly and posteriorly in the fossa and that the disk prolapses anteromedially. Usually a blow to the mandible precipitates the onset. The third model presumes that hypertonicity develops within the elevator and lateral pterygoid muscles. Localized myalgia follows as the disk is pulled anteriorly by contracted lateral pterygoid muscles. The disk is displaced anteriorly, and arthralgia develops.

The rationale for the first model appears more plausible than the rationales for the second and third models. Hyperextension of the mandible may result from minor or major events. Sudden yawning or prolonged mouth opening are potential minor causes. Recurrent subluxation from hypermobility of the joint capsule could precede each. Effusion into the joint spaces may cause edema of soft tissues.

An acceleration-deceleration force comparable to the force of whiplash could hyperextend the mandible and induce displacement. A mechanism has been proposed for disk displacement resulting from whiplash. In a rear-end auto collision, the cranium and neck initially are hyperextended backward. The suprahyoid and infrahyoid muscles are too weak to prevent the backward motion. The mandible moves downward and forward. This hypertranslation occurs because the anterior capsule is weakly attached. The forward motion of the condyles stretches the posterior attachment of the disk. As the brakes are applied or the vehicle decelerates to a stop, the body and head move forward. The forward movement of the head cause hyperflexion of the cervical spine. The condyle moves posteriorly and jams the stretched attachment against the posterior wall of the fossa. In effect, the soft tissues of the joint are subjected to a double whammy - hyperextension followed by hyperflexion.

Permanent disk displacement resulting from condylar luxation may be caused by a blow to the mandible. The force must be sufficient to drive the condyle superiorly and posteriorly. Because of the proximity of the condyle to the fossa, logic would have this to be in a shearing direction to tear the disk. In light of the high frequency of disk dislocations reported in the literature, one hardly imagines that many displacements derive from injuries of this sort.

One study showed that incisal clenching stretches both the lateral diskal ligament and bilaminar zone. Because of this relation, incisal clenching has been suggested as a major etiologic factor in anterior disk displacement. According to the author of this study, the bilaminar zone is not stretched excessively even on wide opening. The lateral diskal ligament would seem to have a greater role in diskal changes than that of the superior head of the lateral pterygoid muscle. Prolapse could occur only by detaching this muscle from the pterygoid fovea. This was not the case in any of the 26 cadaver TMJs dissected.

A view that the disk prolapses from pull of the lateral pterygoid holds little respect if one considers the anatomy of the lateral pterygoid muscle and its attachment to the disk. Dissection of 25 TM joints obtained from 22 cadavers showed that the upper head of the lateral pterygoid inserts at the medial half of the disk in 60% of the joints, whereas 40% had no muscular insertion to the disk. Manual pull on irradiating fibers stretched the disk, but no forward movement occurred. The authors concluded that the function of the lateral pterygoid could be compared with that of an articular muscle. Thus, if pulling on the lateral pterygoid muscle with fingers fails to induce prolapse, any effect of incisal clenching on disk displacement should be considered minimal.

Significance: Although these studies are revealing, the etiology of internal derangement is unclear. Somehow, some way, the disk becomes displaced. There is no evidence to suggest spontaneous displacement or predisposition for displacement.

Osteoarthritis. Osteoarthritis is second to internal derangement as a common pathology of the TMJs. Although osteoarthritis can lead to major TMD complaints, most patients suffer mild symptoms. It has been predicted that, on average, 1 person in 10 with generalized osteoarthritis will experience TM joint symptoms. Masticatory muscle tenderness has been the predominant symptom found in these patients (Table 3-6). The presence of other symptoms varies greatly from patient to patient.

The etiology of TMJ arthritis is unknown. It can be differentiated into primary or secondary types. Reportedly, the etiologies differ between the two. Secondary osteoarthritis occurs in young individuals and derives from a prior incidence of trauma. Primary osteoarthritis may be found in the elderly. Usually this degeneration is an age-related deterioration of the articular surfaces and, sometimes, the disk.

A strong argument has been made that prior internal derangement leads to TMJ osteoarthritis. The argument is that soft tissue derangement leads to features recognized as either osteoarthritis, avascular (aseptic) necrosis of the condyle, or regressive remodeling of the condyle. Accordingly, osteoarthritis represents one of three possible degenerative and adaptive changes that occur after internal derangement.

Facial deformity may be an end product of these changes. Osteoarthritis may create insidious skeletal changes, simultaneous intrusion of the teeth, and realignment on the ipsilateral side. On the contralateral side, realignment or tooth eruption occurs as an adaptive response to the ipsilateral skeletal changes. Over the long term, little occlusal change is evident because of this adaptation. A posterior open bite may occur during an occasional episode of joint inflammation. Generally, most joints remain stable unless interrupted by trauma, systemic disease, or hyperextension of the mandible.

Avascular Necrosis. Avascular necrosis may occur in the mandibular condyle. According to the hypothesis, this may follow systemic disease, trauma, orthognathic surgery, or orthodontics, if the joint is previously deranged. As the surface of the condyle degenerates, mechanical failure follows, leading to a reduction of vertical dimension. Occlusal changes, caused by the decreased vertical dimensions, may include contralateral anterior open bite with ipsilateral premature contact of the molars. Ultimately, these changes produce facial deformity and lateral shift of the mandible towards the affect joint on mouth opening.

This necrosis may develop in an otherwise metabolically inactive osteoarthritic joint. Potential triggers may include trauma, iatrogenic manipulation, systemic inflammatory illness, or administration of exogenous steroids. Secondary osteoarthritis may be a consequence of avascular necrosis.

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Regressive Remodeling. This remodeling is the third presumed sequela of internal derangement. It is most frequently observed on the articular eminence and on the posterior part of the condyle. There is loss of convexity or flattening of the contour of surfaces. It is both a degenerative and an adaptive process. In contrast to osteoarthritis and avascular necrosis, this remodeling is characterized by osseous remodeling and resorption with eventual decrease in condylar size. The process is slow developing, insidious, and without hypertrophic changes. Loss of vertical dimension is probably accomplished by osteoporosis.

Regressive changes start with early derangement of the disk. As the disk thickens, osteocartilaginous remodeling results. The articular surfaces of both the temporal bone and the condyle undergo this regressive-adaptive process. Occlusal changes may occur if condylar regression is rapid.

Tomographic films show stages of regressive remodeling that differ from stages of osteoarthritis. Unlike osteoarthritis, in which the joint space becomes narrow or obliterated, regressive remodeling shows either a widened or normal anterosuperior articular space. Radiologic findings indicate that regressive remodeling may lead to avascular necrosis and structural deterioration.

On the other hand, there is conflicting evidence that internal derangement precedes the degenerative-adaptive processes. Symptomatic internal derangements have not been associated with high levels of prostaglandins and thromboxane, two inflammatory mediators present in rheumatoid synovial fluid. Cases of osteoarthritis have been found without internal derangement. Some authors argue that prior internal derangement may be regarded as an accompanying sign of osteoarthritis rather than as its cause. For this reason, they consider osteoarthritis a primary rather than secondary pathology.

Some evidence supports this assertion. The microscopic anatomy of osteoarthritic joints resembles the histology of joints with internal derangement. The usual sequence of signs and symptoms of osteoarthritic TMJs is similar to the sequence found in other bodily joints undergoing osteoarthritis. In cases of internal derangement caused by laxity of soft tissues or direct trauma to the joint, osteoarthrosis may develop secondarily.

Although the above conclusions merit consideration, other features rule against osteoarthritis as the primary factor in TMD. Osteoarthritis occurs in a much lower frequency than other diagnosed conditions among populations of clinic patients. One example illustrates this point. In 274 patients examined with TMD symptoms, diagnoses were as follows: MPD syndrome (71%), osteoarthritis (9%), symptomless clicking, recurrent subluxation, and rheumatoid arthritis (8%), and conditions unrelated to the TMJs (12%). It is not likely that 7 of 10 patients with MPD syndrome convert to osteoarthritis.

Significance: No one has identified with any degree of certainty a predisposing factor for osteoarthritis. It is not clear if disk displacement precedes osteoarthritic changes, if they

occur simultaneously, or if osteoarthritis initiates diskal derangement. The goal of every clinician will be to discover the sequence.

Rheumatoid Arthritis. Rheumatoid arthritis is an example of an inflammatory synovitis of unknown etiology. Inflammation develops mainly within the synovial membranes of the joints. Involvement of numerous extra-articular organs makes this condition a systemic disorder. Although diverse TMD symptoms have been found in patients with rheumatoid arthritis, some are less pronounced than those found in osteoarthritic patients (Table 3-6). As in osteoarthritis, the major TMD symptoms are masticatory muscle tenderness and joint crepitation. A chief difference is that rheumatoid arthritis is a symmetric synovitis affecting joints bilaterally. Usually joints of the hands and feet are affected first. The cervical spine may become involved late in the course of the disease. Patients may suffer from lateral neck pain as inflammation develops at the C1-C2 articulation. Orofacial pain may be referred from the neck.

Rheumatoid arthritis affects few individuals of the general population (about 3%). Usually, the more the severe the disease, the greater the risk of TMJ involvement. A study of 123 patients with rheumatoid arthritis predicted that 1 in 3 individuals with generalized rheumatoid arthritis will experience TM symptoms.

Significance: Any patient with TMD symptoms and simultaneous symmetric joint symptoms lasting for 3 weeks or longer should be referred to a rheumatologist for evaluation of possible rheumatoid arthritis.

Muscle Disorders

Judged purely from clinical impressions, changes in the masticatory muscles have been considered secondary to mechanical jamming of the disk or trauma-induced internal derangements. Because many patients with internal derangement suffer complaints of the masticatory muscles, alternate opinions favor muscle changes over joint problems. The logic is that the masticatory muscles initiate symptoms and subsequent joint pathology. The literature is replete with information about muscular changes involved in TMD. Some terms include hyperactivity, hypertonicity (splinting), myospasm, myalgia, and trigger points.

Hyperactivity. Hyperactivity of the jaw muscles has long been considered a primary etiologic factor in TMD. Theoretically, excessive emotional stress promotes the hyperactivity. The stress-related hyperactivity forms the basis for the Psychophysiological Model of the Etiology of Myofascial Pain Dysfunction Syndrome. A pain-dysfunction cycle is set off in patients already predisposed psychologically and physiologically to inner stress. Hyperactivity also may develop secondarily if one is subjected to physical stresses.

Some of the problems in accepting hyperactivity as a prime etiologic factor derive from semantics. Traditionally, hyperactivity means abnormally sustained muscle tension, but it also may mean elevated muscle activity detected electromyographically. The reader is not always certain which definition is meant. The first definition is used here.

A thorough review of hyperactivity has been discussed from a theoretical basis. The authors considered local causes and centrally mediated causes for this activity. They

concluded that local factors exclusive of trauma appear unlikely to cause hyperactivity. Under centrally mediated causes, psychological and physical stresses were discussed. The authors summarized studies of subjects with MPD who were exposed to experimentally induced stress. They concluded that causal factors cannot be determined from these correlational studies and that methodologic problems make testing for differences between symptomatic and asymptomatic subjects appear futile.

Since publication of this review, a pathophysiologic model for masticatory muscle hyperactivity in TMD has been proposed. The model proposes a centrally mediated cause, yet it maintains support for present peripheral causes. The authors hypothesize that the hyperactivity represents a mild extrapyramidal disorder distantly related to orofacial dyskinesias (eg, tardive dyskinesia). They consider this relation based on experimental evidence of an underlying neurotransmitter imbalance in the basal ganglia. This imbalance involves dopaminergic preponderance or cholinergic and gamma-aminobutyric acid-nergic hypofunction. Supposedly, during nonstressful periods, muscular activity remains in a subclinical state. During stressful periods, an imbalance results in hyperactivity. This hypothesis requires additional study.

Hyperactivity has been hypothesized as a contributing factor in disk displacement, but recent investigations fail to support this assumption. Based on anatomic studies of the relation of the lateral pterygoid muscle and the disk, hyperactivity can be ruled out as a direct cause of disk displacement. Some disks lack direct connection with the lateral pterygoid muscle, and even pulling on this muscle did not cause significant prolapse.

Although it is logical to expect a progressive increase in symptoms after prolonged hyperactivity, studies show that this does not occur. No progression of symptoms was found in a study of emotional stress among 39 subjects with combined muscle and TMJ pain compared with 24 subjects having only muscle pain or 28 with joint pain. The lowest levels of stress occurred in the combined pain group. This group was rated lower than the muscle pain group by clinicians in terms of psychological factors, stress, and pain chronicity. Combined pain subjects and muscle pain subjects had comparable levels of visual analogue scores (VAS) for pain intensity and activity impairment. Comparable clinician ratings were found for the combined pain and joint pain groups. In summary, no well-defined relation has been established between hyperactivity and TM symptoms at this time.

Myospasm. The definition of muscle spasm does not seem to fit within the etiologic framework of the MPD theory. Muscular responses may include spasm, but to call an elevated tensional period a spasm is a misnomer. Occasional trismus may occur. Under normal adaptive circumstances, however, there is no evidence that the masticatory muscle is predisposed to spasm. Many patients with TMD present with widespread tenderness of the muscles to palpation. Although some subjects may report that their face hurts, many are unaware that the muscles are sore. Few could tolerate the pain of sustained contraction diffusing throughout the orofacial musculature on a long-term basis.

Fatigue. Fatigue may be a significant factor in causing muscle pain. Levels of tension may build in the musculature, leading to chronic fatigue and, potentially, to dysfunction. Subjective reports of fatigue in the jaws have been evaluated in adolescents. A longitudinal study of 285 adolescents found that fatigue in the jaws was prevalent in 18% of 17 year olds.

Results obtained by questionnaire from 264 of these subjects at 19 years old showed higher percentages, with fatigue related to pain of the face or jaws, difficulties in chewing and opening wide, and recurrent headaches. Subjects who desired themselves as being tense reported significantly more fatigue in the jaws. Awareness of clenching was related to fatigue, whereas teeth grinding and frequent chewing of gum were not. Clinical examination revealed that 56% of subjects reporting fatigue in the jaws had four or more sites tender to palpation. The authors concluded that subjective reports of fatigue in the jaw may be symptomatic of dysfunction in adolescents.

Trigger Points. A trigger point is a tender area in a firm band of muscle tissue. These localized hyperirritable foci can be found in any skeletal muscle. They populate the muscles of the head, neck, shoulders, and lower back. Trigger points may refer pain. The referred area is the zone of reference. They can be active causing referred pain on palpation or can be latent (silent) with the potential for action. There is a local twitch response to quick tapping. Generally speaking, the pattern of pain from a trigger point in any specific muscle is consistent from person to person. Details for specific trigger points and zones of referral are presented in the chapter under diagnosis.

Trigger points are thought to be activated by acute or chronic muscular overload, fatigue, or trauma. Speculation is that myofascial pain is the end product of this overload. Two different causes have been proposed for the origin of myofascial pain resulting from trigger points. Histologic examination has confirmed the presence of degenerated areas of muscle termed *fibrous nodules*. Most of the nodule consists of acid mucopolysaccharide. In these areas, the muscle fascicle herniates, releasing histamine and 5-hydroxytryptamine from degrading mast cells. Presumably, hyperactive tonic contraction produces the structural modification. These areas may be active or silent. There is no agreement on how these tiny fibrous nodules elicit pain.

A modification of this hypothesis attributes the myofascial symptoms to release of noxious substances from chronically contracting muscles. Within the entire muscle or localized area, oxidative activities may increase. These activities release inflammatory substances (eg, bradykinin, prostaglandin) and potassium, which could cause excitation of type III and IV muscular nociceptors. If this sustained contraction occurs locally, say at a trigger point, muscular fatigue may result. Steady contraction cause less blood flow, lowers ATP reserves, and slows the calcium pump. Free calcium interacts with ATP to trigger contractile activity, particularly if actin and myosin are overlapping in the shortened muscle. A self-sustaining cycle develops so that noxious substances are released near the trigger point followed by a reduction in the activity of the calcium pump.

Feedback systems link trigger points and zones of referral to the central nervous system. This linkage establishes a reflex. When mechanical stresses, perhaps faulty body posture, are added to this reverberatory neural circuit, myofascial pain results. Trigger point activity can be perpetuated by lesions in the joints or by remove visceral disease.

Tender Points. Like myofascial pain, fibromyalgia is a common cause of musculoskeletal pain and fatigue. This condition is a generalized disease with bilaterally symmetrical tender points. Tender points are the hallmark of fibromyalgia. Like active trigger

points, they are tender when palpated. Unlike trigger points, the pain remains localized. Usually trigger points of MPD are unilateral.

Tender points may occur within the same general area as trigger points. Because of this simultaneous presence, individuals with trigger points of the masticatory muscles may represent only a subgroup of patients with fibromyalgia. These subjects could be thought of as having masticatory fibromyalgia. A discussion of the overlapping characteristics of fibromyalgia and MPD has been published.

An etiologic hypothesis has been suggested for fibromyalgia. Muscle microtrauma at the sarcomere has been proposed as the initiating factor. This injury changes the permeability of the sarcolemmic membrane and permits an influx of calcium ions and an efflux of potassium. Contraction of contiguous sarcomeres results from this calcium influx. Normal mechanisms for decreasing calcium levels are overwhelmed, and the elevated levels of calcium maintain sarcomere contraction. The ATP becomes depleted leading to an uncoupling of actin-myosin cross-bridges. The involved sarcomeres remain contracted, causing a focal rigor mortis. Injured sarcomeres release potassium ions, acting type C unmyelinated nerve fibers causing pain. Eventually, localized response to injury results in the release of inflammatory substances that stimulate type C fibers. Pain continues. This mechanism may also explain the origin of pain at trigger points.

Significance. Muscle disorders account for most complaints reported by patients. Increased tension, rather than spasm or hyperactivity, would seem to fit most muscular complaints of the jaw. Some tension may end in soreness, but not terminate in pain. Evidence suggests fatigue and trigger points relate to the genesis of pain. Some patients develop muscle complaints and joint complaints simultaneously. Few patients develop joint disorders independent of muscle disorders.

Cervical Spinal Disorders

Cervical spinal disorders may lead to complaints often associated with TMD (Table 4-4). Pain may be felt along the side of the head from disorders of the C2 to C3 vertebrae. A possible mechanism whereby pathology of the cervical spine could lead to pain in the trigeminal system would include the input by way of the upper cervical nerve roots. The descending tract of the trigeminal nerve passes ipsilaterally to the midcervical part of the spinal cord. A nociceptive impulse from degenerated cervical facets could refer pain to dermatomal distribution of the trigeminal nerve. Thus, this referred pain would not originate from the TMJs.

Table 4.4. Relation of Cervical Source of Pain and Referred Location of Pain

Source	Referred Area
Occiput-atlas	Forehead, retro-orbital, may be bilateral
Cervical-2nd vertebra	Retro-orbital, temporal
Cervical-3rd vertebra	Occiput, lateral side of head
Cervical-4th-7th vertebra and thoracic-1st vertebra	Shoulder.

Cervical spine disorders may, in fact, result in bizarre symptoms. These disorders may involve pain of the head, sinuses, face, ear, and throat. Sensory disturbances of the pharynx, vertigo, tinnitus, diminished hearing, sweating, flushing, lacrimation, and salivation are possible symptoms. Because of the arrangement of the sympathetic nervous system in the cervical spine, headache, nystagmus, nausea, vomiting, and suboccipital tenderness may occur. Some patients with TMD may experience these symptoms. A thorough cervical neck examination may clarify the issue.

Headache

Because some patients with jaw symptoms have concomitant headache, the question remains whether headache should be considered part of the overall TMD symptomatology. According to the *Guide to Dental Health* published by the American Dental Association in 1985, TMD is often misdiagnosed because of an array of symptoms common to several diseases. Headaches, sometimes of migrainous proportions, are listed as symptoms.

The etiology of most headaches remains complex and uncertain, and it is difficult to clarify any probable relation. This uncertainty, coupled with the multifactorial etiologies of TMD, offers few opportunities for solving this puzzle.

Because the classification of headaches is subjective and many variants exist, this discussion focuses on those painful cranial complaints neurologically diagnoses as vascular (migraine, cluster), tension (muscle contraction), and cervicogenic headaches. Only those publications that suggest an association between headache and TMD are discussed.

Muscle contraction headache predominated in a study of patients who presented with unsatisfactory dentures. Forty percent of the 43 patients reported recurrent headache. No patient was completely free of TMD symptoms. In two different groups of 80 patients, the frequency of recurrent headache correlated with the frequency of masticatory muscle pain and TMJ pain on palpation, but not with several other TMD symptoms.

TMD symptoms were studied in three groups of 38 patients with different kinds of headache and in 25 healthy subjects without headaches. Subgroups of patients were identified with either muscle contraction headache (13), common migraine (17), or a combination of both (18). Tenderness on palpation of the pericranial muscles proved significantly greater in the headache patients than in the healthy subjects. The pain reported was least severe with migraine headaches, increased with muscle contraction headaches, and was most severe with combination headache. On an average, nine tender areas were found per headache patient. Twenty-nine of the 38 headache group had pain referred to other areas on palpation of the tender areas. Tenderness was blamed on abnormal tonic hyperactivity in the masticatory muscles and neck. Bruxism was greater in headache patients than in healthy subjects. No differences were found for malocclusion or loss of molars.

There is considerable support for a relation between common migraine and TMD symptoms. It has been argued that the common migraine is a muscular rather than vascular disorder. Comparison of 100 patients referred to a hospital neurology clinic showed variability of TMD symptoms and the type of headache. Patients were diagnosed as having tension, migraine, or combination headaches. Just three of 19 common signs and symptoms proved

significantly different among the subgroups: tenderness on palpation of the masseter, tenderness of the temporalis muscles, and dynamic pain. This pain was identified as the patient performed mandibular movements with the examiner exerting slight manual resistance against the mandible. Several other differences were found between groups. Unfortunately, the data were marred by unacceptable statistics. The three significant symptoms should have been tested further. Only tenderness on palpation of the masseter proved greater for patients with combination headaches versus migraine patients. An acceptable level of significance should have been $P < 0.01$, not $P < 0.05$. Finally, the authors were unable to prove that the subgroups differed in myogenous or arthrogenous origins of pain. They concluded that patients with TMD pain suffered more from permanently present headache and subjective neck problems than patients without pain.

In contrast to the findings of the above-mentioned studies, no significant differences in TMD symptoms were found between groups with a different neurologic diagnosis of headache.

There is considerable evidence that headache may arise from myofascial trigger points of masticatory, neck, and back muscles. Palpation studies illustrate the complexity of these relations. Both the location of the painful area and the respective trigger points were identified in 164 MPD patients who presented with headache. About one third of these patients complained of postauricular pain traced to trigger points in the sternocleidomastoid, deep masseter, and digastric musculature. Just over one half complained of temporal pain that originated from four trigger point areas, namely, the temporalis, trapezius, and splenius capitis muscles (Table 4-5). Just less than one half had occipital pain emanating from the trapezius, semispinalis capitis, and levator scapulae.

A specific variant, cervicogenic headache, has been defined as a unilateral pain felt in the head but originating from the neck. The anatomic proximity of certain cranial nerves and the trigeminal nerve may account for this pathophysiology. Because neurons of the trigeminal nerve and nerves VII, IX, and X share the same neuron pool with neurons of C_1 to C_3 of the cervical spine, pain emanating from the neck may be referred to the face and mandible. Pain impulses may be felt at the trigeminal sclerotomes and dermatomes (V_1 , V_2) from afferent pain fibers mediated by C_2 rootlets. Additional evidence to support this assertion is that surgical decompression or dissection of the C_2 root eliminated or improved the cervicogenic headaches in 15 of 16 patients.

Table 4-5. Relation of Painful Area with Trigger Point Location in Headache Patients

Pain Area	Trigger Point	Patients (%)
Supraorbital	Anterior temporalis	9.8
	Sternocleidomastoid	
	Trapezius	
Retro-orbital	Anterior temporalis	29.9
	Trapezius	
	Sternocleidomastoid	
Forehead	Sternocleidomastoid	36.0
	Splenius capitis	
Temple	Anterior temporalis	51.8
	Middle temporalis	
	Trapezius	
Postauricular	Splenius capitis	36.6
	Sternocleidomastoid	
	Deep masseter	
	Digastric	
Vertex	Splenius capitis	34.7
	Semispinalis capitis	
Occipital	Trapezius	45.7
	Semispinalis capitis	
	Levator scapulae	

Cervicogenic headache can be precipitated or intensified by head movement or by pressing on trigger points. Palpation of several muscles of the neck, including the splenius capitis, splenius cervicis, and sternocleidomastoid, was conducted on 11 patients with cervicogenic headache. Headache was reproduced in 8 patients by palpation. All had at least three trigger points on the symptomatic side. A comparison of symptomatic and asymptomatic sides found significant differences in the tender areas. Some 70 tender points (35 very tender and 35 tender) and 17 nonfascial tender points occurred on the symptomatic side, whereas there were 22 tender (one very tender and 21 tender) and 19 nonfascial tender points on the asymptomatic side. All patients had cervical dysfunction. Specific segmental dysfunction was found at the occiput on atlas or at the atlas on axis in 10 patients. The author concluded that trigger points may be a significant pain-producing mechanism in cervicogenic headache and segmental cervical dysfunction.

Some migraineurs present with TMD symptoms. Trigger factors, identified by patient self-report, have been studied in 217 migraineurs. These trigger factors included the menstrual cycle (51.5% of the women), alcoholic beverages (51.6%), and psychic stress (48.8%). Alimentary triggers predominated in alcohol-susceptible patients compared with patients lacking these triggers. It was hypothesized that the gut of some migraineurs may be unduly permeable for certain substances. No significant difference was found between menstrual-related migraine and patients lacking triggers. Menstrual migraine attacks were often preceded by depressive symptoms. The authors speculated that migraineurs may share similar precipitating factors as individuals suffering from TMD.

Significance: The link between certain kinds of headaches and TMD symptoms seem plausible. Sufficient evidence exists to tie certain orofacial pains with dysfunctions originating from neck and masticatory musculature. Some trigger factors may be the same for both. Headaches are present in some patients with TMD symptoms and absent in others. Those of cervical origin should be considered distinct from the symptom complex of TMD.

Neoplastic Disease

The literature reveals few cases of malignant disease directly evolving from the TMJs. Therefore, most of these tumors should be classified as precipitating factors rather than as predisposing factors.

Malignant disease may disguise as TMD. A review of the literature found 42 cases of malignancy that mimicked TMD. A summary of the symptoms showed that swelling was the chief problem, occurring in 54% of these cases (Table 4-6). An inability to open the mouth widely and deficits of the acoustic nerve were the next most prevalent symptoms. Pain did not prove to be a specific symptom, but altered motor sensory sensations (paresis or paresthesia) were common in just over one fourth of the cases.

Table 4-6. Signs and Symptoms of 41 Cases of Malignant Disease that Mimicked TMD

Symptom	Patients (%)
Swelling	54
Trismus	34
Auditory changes	34
Paresis or paresthesia	27
Occlusal change	10

These studies indicate that gender differences exist in the prevalence of these malignancies (Table 4-7). Women had more metastasis from the breast and pharynx than men. On the other hand, men were found with more squamous cell carcinomas of the maxillofacial area, sarcomas, and adenocarcinomas than women. These findings indicate that previously asymptomatic individuals 50 years or older should be examined carefully for malignancy if TMD-like symptoms suddenly appear.

Table 4-7. Final Diagnosis of 42 Cases of Malignant Disease That Mimicked TMD

Malignant Disease	No of Cases
Women (21 Patients)	
Metastatic breast	6
Pharyngeal	4
Nasopharyngeal (3)	
Oropharyngeal (1)	
Parotid	3
Mucoepidermoid (2)	
Adenocystic (1)	
Maxillofacial squamous cell	2
Hypophyseal	1
Transitional cell	1
Undifferentiated	1
Schwannoma	1
Fibrous histiocytoma	1
Metastatic adenocarcinoma	1
Men (21 Patients)	
Maxillofacial squamous cell	5
Prostatic adenocarcinoma	3
Adenocarcinoma	3
Sarcoma	3
Synovial cell (1)	
Synovial fibrosarcoma (1)	
Chondrosarcoma of TMJ (1)	
Nasopharyngeal	2
Multiple myeloma	1
Adenoid cystic	1
Metastatic lung	1
Schwannoma	1
Intercranial metastatic melanoma	1

Significance: Subjects should be questioned about the possibility of previous malignancy.

Uncommon Causes

Neuralgias. Neuralgias, including trigeminal, glossopharyngeal, or atypical (vascular) neuralgias, contribute little to TMD symptoms. No mention was made of concomitant TMD symptoms in a review of 689 cases of trigeminal neuralgia or in 6 cases of glossopharyngeal neuralgia. The quality of pain for most neuralgias differs so sharply from the pain of TMD that they are not easily confused. Well-defined jabs of pain characterize these neuralgias, whereas the quality of muscular pain is more constant. The severity of neuralgia may lead

secondarily to increased muscular complaints. Other clinical findings, such as tenderness or limitation on opening, may be present simultaneously. They are not primarily related to the neuralgia.

A case of acute inflammation of the TMJ has been proposed as causing the onset of trigeminal neuralgia. The authors concluded that this distant focus of inflammation represented a peripheral mechanism triggering the onset of neuralgia by increased afferent stimulation.

Significance: Neuralgias should be ruled out during the diagnostic phase. no firm relation has been established between TMD and neuralgias.

Vascular Disorders. Reports of TMD-like pain from cardiac origin are rare. An episode of mandibular pain appeared a few hours preceding an acute myocardial infarction in a white woman with coronary insufficiency. Four other cases of mandibular pain were attributed to myocardial infarction and 1 case to angina pectoris. The 2 male patients with infarcts had right mandibular pain, and the 2 women had bilateral mandibular pain. Another case of left jaw pain or toothache present in a white woman was attributed to left atrial hypertrophy and right bundle branch block. Simultaneous presence of jaw pain and vascular pain was explained by overlap of cervical nerve C₃-spinal nerve T₁ with the trigeminal nerve. These examples of myocardial pain referred to the jaw or teeth were identified by historical relation with vascular disease.

Significance: Vascular disorders, exclusive of headache, generally contribute little to the TMD symptoms.

Respiratory Disorders. Other bizarre causes of orofacial pain have been described. Hyperventilation, or overbreathing, has been documented as a cause of orofacial pain. Light-headedness, dizziness, and shortness of breath were described as classic symptoms of hyperventilation. The authors examined a patient with recurring episodes of sharp, burning pain on the left side of the face, radiating from the top of the scalp to the midline of the midline. The pain was accompanied by dizziness, sweating, and a tingling sensation over the affected area. Deep breathing for 1 minute initiated severe orofacial pain. Blood pressure and heart rate rose dramatically. Psychometric evaluation found elevated levels for hypochondriasis, depression, and hysteria.

Significance: Hysteria leading to hyperventilation may mimic some TMD symptoms. Fortunately, this is not a common occurrence.

Connective Tissue Disorders. Few patients with TMD symptoms have connective tissue disease. In systemic lupus erythematosus, muscle pains occur in up to one third of these patients and appear unrelated to the degree of disease. Some patients may develop a painless form of myositis.

Significance: Individuals with suspicious recurrent skin or circulatory complaints should be referred for laboratory testing.

Precipitating Factors

Trauma

Trauma as a potential precipitating factor in TMD has created much interest and speculation. Aside from the pain and discomfort experienced by the injured person, the potential for litigation may be considerable. A blow to the head or whiplash of the neck, especially during a motor vehicle accident, has been and continues to be a reason for litigation.

Retrospective studies have implicated many kinds of physical trauma in the etiology of TMD symptoms. An analysis of different investigations revealed that the prevalence of trauma ranges from 9% to 43% in large clinical populations with TMD symptoms. A history of trauma was found in 68% of 75 patients. The sample was limited, to patients with secondary osteoarthritis of the TMJ.

Trauma was considered the precipitating factor in 43% of TMD cases in a large private practice. Two percent of the accident victims had no immediate or residual complaints from the head or neck injuries. Attempts have been made to divide cases of precipitating from predisposing trauma. Evaluation of a large TMD clinic population showed that 30% had a history of trauma. Precipitating trauma was identified as the major factor in causing TMD pain. Percentages were not significantly different for precipitating and predisposing trauma.

Trauma to the jaw was considered etiologically important in only 16.8% of 246 patients consecutively admitted to a TMD clinic.

Significantly greater TMD symptoms have been found in patients with a trauma history compared with nontrauma subjects. Examination of patients after road traffic accidents showed significantly higher levels of orofacial pain, abnormal joint sounds, and restricted jaw opening when compared with other TMD cases. Trauma figured in the difference between TM symptomatic patients and asymptomatic normals and between symptomatic women and asymptomatic normal dental students and hygienists.

Trauma may cause specific disturbances of the TMJ area. Direct injury to the joint has been proposed as a cause of TMJ osteoarthritis. Several authors have proposed it as the etiology for anterior dislocation of the disk. Statistical analysis showed a positive relation between trauma and internal derangement.

Extensive damage to the joint has been documented after injury. In a study of 30 patients who sustained TM joint injuries, direct blows to the mandible accounted for 20 of the injuries, and 10 cases were whiplash injuries. Various symptoms were found. Of the 30 patients, 27 denied that they had previously bothersome TMD symptoms. Eight patients had mandibular fractures. Radiologic imaging performed on the joints 2 days to 2 years after the injuries showed evidence of disk derangement, degenerative condylar remodeling, osteoarthritis, osteochondritis dissecans, avascular necrosis, swelling of retrodiskal tissue, joint effusion, musculotendinous injuries, and atrophy of masticatory muscles.

If injury is sufficient to alter normal mechanics of the mandible, then internal derangement might account for the dental occlusal discomfort experienced by traumatized individuals. Sudden changes in the vertical dimension, caused by disk derangement, have been suggested as capable of altering the occlusion. A little trauma may be sufficient to promote the appearance of symptoms. Primary trauma was differentiated from secondary trauma. Secondary trauma was designated when there was an obvious, preexisting derangement.

Significance: The clinician must document any serious trauma experienced by the patient. The chief problem in most cases will be to determine whether the joint was struck directly by a blow or whether the injury occurred indirectly when another part of the head, jaw, face, or neck was injured.

Whiplash

Anecdotal remarks imply a direct relation between whiplash and TMD symptoms. Judged from complaints of subjects injured in vehicular mishaps, whiplash may precipitate TMD symptoms. Because reports of whiplash are obtained retrospectively, pretrauma objective data are usually lacking. Some information may be obtained from preexisting dental or medical records.

The similarity of whiplash symptoms in so many patients under different circumstances points to pathophysiologic changes. A review of the literature indicates that whiplash accounted for 19% of the accident-related TMD symptoms in a large clinic population. Although TMD pain was not related specifically to whiplash, its onset was reported to be related to motor vehicular accidents in 17.7% of 164 cases of myofascial pain.

One study predicted that nearly 60% of 37 whiplash victims or subjects sustaining blows to the face or neck and developing myofascial pain or headache would remain symptomatic for the remainder of their lives.

Whiplash may produce disturbances at specific anatomic sites of the head, TMJs, and cervical musculature. Ten of 14 patients treated for whiplash obtained relief from head pain and 8 from cervical pain. Assessment of 28 patients injured by whiplash showed that 22 of 25 suffered arthrographically evident internal derangement. Arthrograms of the inferior joint space confirmed varying degrees of derangement. On average, the patients were examined 126 days after the accident.

Ten patients symptomatic for TMJ pain were examined for internal derangement 2 days to 2 years after the whiplash event. Imaging studies showed the joints of 3 patients to be normal. Six patients had build-up of intra-articular fluid. Of 3 patients with painful, clicking joints, 2 showed early stages of disk derangement and 1 was normal. These variable findings confirm the possibility of internal derangement in certain patients. A complete radiographic and imaging study of the joints may be necessary to rule out derangement.

The literature on the effects of whiplash on the cervical spine is large. There is popular support for and against the argument that whiplash produces cervical dysfunction and TMD-like symptoms. Whiplash may alter normal craniovertebral posture. Mechanoreceptors present in the upper cervical spine are quite sensitive to slight changes in normal posture of the head,

neck, and mandible. Postconcussional syndrome, including headache, may occur in the absence of cranial injury when the subject has experienced acceleration-deceleration forces. Post-traumatic craniovertebral symptoms mimic many symptoms of TMD. Superficial structures of the head and face posterior to the trigeminal area and extending to the angle of the mandible are innervated by C₁ to C₃ nerves (see Table 4-4). The ear surface and surfaces posteriorly, and the sternocleidomastoid and trapezius muscles, are innervated by these sensory fibers. Irritation, compression, or entrapment of these nerves may lead to localized pains of the orbit, occiput, and angle of the mandible. Posttraumatic headache may occur from occipital and posterior cervical pain.

Research findings differ with respect to the adverse effects of whiplash. A comparison of non-TMD patients with subjects having a history of head or neck trauma showed a statistically significant greater distribution of mild and moderately severe symptoms of headache-cervical pain in the traumatized group than in the nontrauma group.

Many similarities and few differences have been found between subjects who experienced different kinds of overt injury and whiplash. A comparison of patients (31 with overt head trauma, 35 with whiplash without overt head trauma, and 34 with whiplash and over head trauma) found no consistent differences in muscle pressure pain threshold, prior pain duration, time before pain onset, or scores obtained on psychological tests between groups. Whiplash subjects with over trauma reported the greatest pain on presentation. Subjects without overt trauma were more likely to be in litigation and to have more sleep disturbances, painful sites on palpation, and neck pain than subjects of the other groups.

In contrast, another study presents contradictory evidence of adverse effects. Patients with cervical musculoskeletal injury were interviewed, examined, and divided into those with radiologic and those without radiologic evidence of injury. Just 1 patient without radiologic damage reported new TMJ symptoms. Follow-up conducted 1 month later revealed that just 2 patients with radiologic pathology had TMJ pain. No patient without radiologic pathology had new TMJ symptoms. The follow-up interview was made by telephone.

Significance: Whatever role whiplash plays in precipitating TMD symptoms, its effects vary widely between individuals and situations. Differences between studies confirm the need for thorough neuromuscular examination of the cervical spine in subjects with TMD symptoms who have experienced whiplash.

Dental Treatment

Patients often ask if a particular dental treatment causes adverse effects or improves a preexisting TMD condition. Loss of teeth, orthodontics, prosthetic treatment, or root canal therapy are the most frequently asked about procedures.

Loss of Teeth

Many clinicians consider that the loss of teeth and subsequent loss of vertical dimension produces overclosure. The overclosure causes the mandible to be malaligned, resulting in joint disturbances. This malalignment alters the position of the condyle in the

fossa, which tends to "overload" the joint. Although collapse after loss of posterior teeth has been suggested as a contributing factor in TMD, doubt remains about any positive relation.

There is evidence to contradict the argument that joint disturbances occur with tooth loss. No significant difference between tooth loss and joint disturbances was found in one study, but opposite findings were presented in two other studies. Unilateral tooth loss was common on the side opposite the dysfunction but was identified with the same side in another study. The latter study involved 326 patients admitted to a hospital dental clinic. Three fourths of the patients had missing teeth. The author concluded that occlusal support was important in the etiology of TMD, reaching a maximal relation with 3 to 5 teeth missing and declining thereafter. Differences were found between patients when compared with individuals of the general population, but the data were not analyzed statistically. Finally, no statistically significant correlation was found between loss of molar support and crepitation or tenderness to palpation of the TMJ.

Studies on muscle tenderness to palpation showed that equal and unequal tooth loss occurred in 20 of 22 patients with myofascial pain. Tooth loss and dysfunction were not correlated among 406 Swedish patients diagnosed as having various TM disorders. Eighty-two percent of the patients had myofascial complaints. An investigation of 72 Egyptian men found that 50% with premolar and molar support had muscles tender to palpation. No similar occlusal support was found in 33% of the subjects with painful muscles. These findings suggest that complete occlusal support is not a preventive measure for controlling muscle tenderness because the number of subjects with complete and incomplete posterior teeth was almost equal.

One might expect the loss of teeth to have a significant effect on joints undergoing degeneration. According to one author, the status of the dentition appears to have no direct effect on the production of TMJ osteoarthritis.

Extraction of Teeth

Overmanipulation of the mandible during the extraction of molars has been considered a factor in producing TMD problems. Oral surgery and other surgical procedures were associated with TMD pain in 17.7% of 164 patients diagnosed with myofascial pain. The findings in another study muddle the picture because a history of molar extractions in the general population was considered marginal. A history of molar extractions was more common in patients with TMD at 66%, compared with students who were either symptomatic at 53% or asymptomatic at 46%. Women TMD patients had the highest history, but no difference was found between asymptomatic and symptomatic students.

A slight increase in TMD symptoms has been reported after third molar surgery. The effects proved transient. Symptoms diminished within the first week after surgery. They disappeared after 4 to 6 weeks or remained at the level found before surgery.

Significance: Although the loss of tooth support or overmanipulation of the mandible might be detrimental to the health of the TMJs and surrounding soft tissues, evidence is lacking on any long-term adverse effect.

Restoration of the Occlusion

If occlusal support is necessary to avoid TMD, then restoration of the occlusion should reduce or eliminate symptoms. If dysfunction is present, restoration should help lessen the severity. Although this thinking may be correct, information on denture patients fails to support it completely.

Although transient symptoms were present in 2000 male hospital patients, none was classified as having TMD. About 26% had complete dentures, 16% had mutilated dentitions, and most suffered from overclosure. Another study of 93 TMD patients showed mixed patterns of dentition. Nearly 20% had complete dentures, 20% had an upper denture opposing either restored or unrestored dentitions, and 34% had natural dentitions. Just 15% of 160 complete denture patients demonstrated TMJ disturbances. Joint pain was not always present with muscular tenderness, joint sounds, or restricted movements.

A survey of 43 Swedish patients fitted with unsatisfactory complete dentures found that the frequency of dysfunction was 63% for men and 83% for women. Most dysfunction was attributed to myogenic pain. The investigator concluded that tolerance to dysfunction appeared greater in complete wearers than in groups not wearing them.

The restoration of occlusion by fixed partial dentures does not necessarily eliminates symptoms. In 50 subjects, who had fixed partial dentures placed in all posterior quadrants, the frequency of symptoms was 68%. Most dysfunction was classified as slight (38%), but some was moderate (20%) and severe (10%).

Significance: A summary of these results complements the previous findings of no well-defined relation between occlusal support and TMD. Restoration of the occlusion has not been demonstrated to improve a preexisting TMD condition.

Orthodontics

Numerous anecdotal remarks have been made about orthodontics and TMD. Some clinicians suspect that there is a greater prevalence of TMD in people treated by conventional orthodontics. Other believe that orthodontic treatment causes changes in occlusal function that lead to TMD. Still others believe that orthodontic treatment can be used successfully to avoid TMD. Recent literature noted that shortened orthodontic treatment time and an improved occlusion can reduce potential TM problems. The different opinions make a review of the literature worthwhile.

Cross-sectional survey of 347 patients examined for TMJ sounds before, during, and after orthodontic treatment showed significant association between joint sound, age, and treatment. It was unclear whether joints sounds increased from orthodontic treatment, age, or both. The status of sounds was unrelated to functional occlusal factors, dental wear, and chewing of gum. The authors recognized that joint sounds represent one sign of TMD and that other dysfunctions are possible.

No major long-term effects of orthodontic treatment were found after evaluation of dental and dysfunctional parameters in another study. The analyses made between 82

orthodontically treated patients 52 months postretention and 28 untreated controls were of extraction patterns, self-reports of symptoms, presence of a slide between retruded contact and intercuspal positions, occlusal stability, posterior interferences, tenderness on palpation, joint noises, and number of restorations. Among these parameters, soft clicks and number of restorations were slightly higher in the post-treatment group than in the untreated group.

Neither orthodontic nor other dental treatments figured as significant etiologic factors based on self-reported symptoms obtained by questionnaire among 343 women. A total of 451 asymptomatic patients undergoing orthodontic treatment were evaluated for TMD symptoms. No significant relation was found between symptoms and the course of orthodontic treatment 18 months later. Eleven subjects with preexisting symptoms were not worsened by treatment.

Correlational studies showed that significantly more TMD patients had prior orthodontic treatment than non-TMD individuals. In particular, women with TMD symptoms had a greater history of orthodontics than asymptomatic women. According to the authors, the relation of specific symptoms to the orthodontic treatment was very weak.

Other information weights against detrimental effects of orthodontics on factors often associated with TMD. Test of null hypothesis that a slide from retruded contact position to intercuspal position was not different in postretention orthodontic patients than in an untreated population showed that slides of orthodontically treated patients were comparable to slides of untreated controls and measurements of slides of the general population reported in the literature.

Self-reports of the onset of myofascial pain and a history of orthodontic treatment were extremely low among 164 patients admitted to a TMJ-Facial Pain clinic. Just 2.4% of the patients mentioned orthodontics as an event related to the pain. TMD symptoms, excluding joint sounds, were considered rare in 1180 patients seeking orthodontic treatment.

Evaluations of condyle position and vertical overlap of anterior teeth were made in patients who had the premolars extracted for orthodontic purposes and in patients without extractions. No statistically significant differences were found in joint space, condyle position, or vertical overlap of anterior teeth between 30 patients after extraction and 37 patients without extractions.

Significance: Although some authors argue that orthodontic treatment superimposed on preexisting subclinical symptoms may lead to a full blown TMD condition, evidence is lacking to confirm or to deny how much or how little orthodontics produces or improves TMD conditions. Thus, despite the personal impressions that clinicians might form about orthodontically treated cases, the literature refutes hypotheses of cause.

Root Canal Therapy

Tooth pain may be eliminated by root canal therapy. Because these procedures require that the mouth remains open under a rubber dam for long periods, it is generally agreed that prolonged mandibular hyperextension may elicit new or aggravate preexisting TMD symptoms. One can never rule out the possibility that additional suffering may incur if there is preexisting TMD condition.

Significance: Although prolonged hyperextension during root canal therapy may be considered significant as an etiologic factor, evidence is lacking in the literature that shows the genesis of new symptoms. Subjects should be examined for tenderness of muscles before treatment.

Perpetuating Factors

Bruxism

There are many who feel that excessive clenching or grinding of the teeth produces unhealthy symptoms of the masticatory system. Some bruxers may complain of pain or other symptoms of the jaw, head, or neck. The simultaneous presence of these complaints does not necessarily mean that the symptoms resulted from the bruxing. Both may occur at the same time and be independent of one another. Arguments for and against this relation are addressed.

Support for the concept that bruxing contributes to dysfunction appears widespread. Complaints about the masticatory muscles have received the most attention. The words used to express these complaints are similar (Table 4-8). This suggests that different investigators have observed similar complaints or different stages of the same complaint but have used different words to express them. The same conclusion can be drawn from complaints of the jaw, face, head, and neck. Pain was the dominant complaint regardless of the anatomy.

Table 4-8. Location of Symptoms Associated With Bruxism

Location	Symptom
Masticatory muscle	Tenderness Soreness Spontaneous contraction Spasm Trismus Hyperactivity Hypertrophy Pain
Jaw	TM joint pain Limited opening Tiredness Aching
Face	Pain
Head	Headache
Neck	Cervical dysfunction Pain
TMD symptoms	Positive association Children Adults

TMD symptoms have been reported among adult bruxers. Positive associations have been found between bruxing and TMD symptoms in children. Equating adult symptoms with children symptoms may be questionable.

An association between muscle-related complaints and bruxing remains controversial. One study found little association between bruxing and myofascial symptoms. Among 899 dentists who attended a dental convention, 16% bruxed only, 3.7% had muscle symptoms only, and 4.6% bruxed with muscle symptoms.

In a group of college students, nocturnal tooth contacts, tenderness of the superficial masseter muscles, and limited mouth opening were significantly related. Still, the investigators concluded tooth grinding was not a major controlling factor in dysfunction. This conclusion was supported by a study of 410 students that found no association between bruxism and facial pain. The literature reveals an exception to this conclusion. Tenderness of the masticatory muscles was found in 39% of 219 male Siwian desert dwellers. The prevalence of incisal or occlusal abrasion was 39%. Two thirds of the dwellers with incisal abrasion had muscle tenderness. Muscle tenderness was present among 30% with generalized occlusal abrasion. The author concluded that bruxing habits involving protrusive movements were more harmful than posterior occlusal habits.

If bruxism causes muscular changes, the damage should be obvious from biochemical analysis of this tissue. In fact, assay of an enzyme has been used to assess the degree of muscle damage in bruxers. No statistically significant differences were found in the levels of serum creatinine kinase activity between bruxers and individuals with TMD and normal, nonbruxer, non-TMD individuals.

Significance: Research has provided little direct support for a link between most TM symptoms and bruxism. Muscle tenderness may be a prominent symptom in certain bruxers. It does not appear to be reflected in major enzymatic changes in the muscle.

Other Oral Behavioral Patterns

Different kinds of oral behavior patterns have been related to head pain, orofacial pain, or increased masticatory muscle activity. A few incidental remarks have been made about chronic chewing of gum and TMD symptoms. No positive association was found in two studies. Jutting of the mandible forward or prolonged lip biting induced pain in the anterior temporalis muscle. The pain was moderate, lasting from 30 minutes to 2.5 hours. Active motions, such as retruding the mandible or biting the tongue, lips, or cheeks of the oral cavity significantly increased the electromyographic (EMG) level in the anterior temporalis muscle. Facial posturing, such as cupping the chin in the hand or resting the hand on the side of the face, produced similar effects. Most of the symptoms are transient in nature.

Interesting associations have been found between oral habits, muscular changes, facial pain, and different kinds of headache. The study involved three groups. Group 1 included subjects with a history of facial pain, migraine, and combined headache. Group 2 subjects had tensional (muscular) headaches, and Group 3 subjects had no similar pains. Clenching was significantly more frequent among group 1 subjects than subjects of the other groups. Resting the hand under the chin was more frequent for group 1 than for group 3, and thrusting the

mandible forward was more common for group than for groups 2 and 3. Reclassification of groups into subjects with TMJ pain, TMJ and muscle pain, or no facial pain showed that nocturnal bruxism was more frequent in the combined TMJ and muscle pain subjects than in other subjects. Ongoing assessment for 28 days revealed that migraineurs had significantly higher frequencies of clenching and putting their hands on the face than nonheadache subjects. Additional study showed subjects with common migraine reported significantly more oral habits than subjects with tensional headaches. No statistically significant differences were found between a nonheadache group and either of the other groups. The authors concluded that high frequencies of oral habits can be expected in migraineurs but that not all cases of common migraine headaches are related to daily clenching or resting the chin on the hands. The number of subjects studied in these groups was small. Replicate studies are needed to confirm these findings.

Significance: It is unclear whether even the most noxious of these habits leads to chronic, painful syndromes. In some cases, they may simply reflect expressions of behavior symbolic of preexisting painful conditions.

Emotional Factors

Research on psychological and social factors has shown that patients with generalized chronic pain may differ from subjects without chronic pain. Emotional factors have been suggested as being involved in the genesis of TMD pain. Whether any particular social or psychological factors can be found, however, to predict which individuals will become susceptible or prone to TMD is problematic. So many potential trigger factors exist that causal associations in relation to TMD symptoms are likely to be complex and involve multiple competing mechanisms. It is unclear to what degree these relations are mediated by neurotic and hypochondrial tendencies that would influence somatic focus and symptom report in general.

Successful identification of the trigger factors is difficult because of the complex psychological somatic interrelations of any chronic pain syndrome. Recent information on the US population shows the magnitude of this problem. About 15% of the population between the ages of 25 and 74 years suffer with definite chronic pain related to the joints and musculoskeletal system. Another 7.4% have pain of uncertain origin. Eighteen percent of the definite pain group and 8% without pain suffer from depression. Many attempts have been made to determine the causes of depression and the relation with pain. The number of painful conditions reported has proved a better predictor of major depression than other measures of the pain experience, including pain severity and persistence. Studies of different populations have not consistently related TMD syndrome to depression.

The psychophysiological concept of the etiology of myofascial pain syndrome relies on emotional tension or stress as the initiating factor. A stressful event was reported by just one fourth of 164 MPD patients with the onset of pain. Subjective reports, including the general history of 170 clinic patients, showed no significant relation between stressful periods and kind of TMD dysfunction (Table 4-9). Self-reports of stressful working or stressful private lives were similar in patients with either reversible disk displacement, permanent disk displacement, or myogenic disorders. A higher percentage of patients diagnosed with myogenic complaints had histories of somatic or psychiatric disease than patients of the other

groups. The findings associated with psychological and social predictors must be interpreted with caution because of significant methodologic shortcomings in many of these reports.

Table 4-9. Events Associated With Onset of Chief Complaint

Report	Reversible Disk (%)	Permanent Disk (%)	Myogenic Complaints (%)
Hyperextension from chewing or yawning	33	35	5*
Dental treatment	7	12	16
Blow to jaw	4	4	6
Stressful period	4	11	18
Unknown	52	38	55

* Reversible disk and myogenic complaints and permanent disk and myogenic complaints were significantly different, but reversible disk and permanent disk complaints were not.

Evaluation of 356 chronic pain patients found that problems in concentration and memory were related to emotional stress, poor family support, and interference with daily activities. Both concentration and memory were unrelated to pain intensity, pain duration, and demographic factors. Analysis of 93 chronic pain patients showed that most patients tended to overestimate their pain intensity levels. Patients with headache, facial pain, or abdominal pain were less accurate in remembering their pain than patients with cervical and low back pain. Individuals who had histories of emotional stress or home conflicts, who were less active, and who relied on medication were most inaccurate in remembering their pain.

Significance: Many factors besides pain quality and quantity influence the discomfort and suffering associated with recurrent pain. The emotional response to pain is influenced by factors such as ethnic background, socioeconomic status, current environment, and the meaning of pain to the individual. It is unclear whether emotional factors, including individual personality traits such as neuroticism, vulnerability to stress, and anxiety, predispose to TMD.