Temporomandibular Disorders: Osteoarthritis

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The musculoskeletal system may be affected by more than 100 different diseases. Several also involve the temporomandibular joint (TMJ) and masticatory muscles and are associated with symptoms of temporomandibular disorders (TMD). Dentists' knowledge in this area has been limited and regrettably beset with problems of definition and reliability of measures. This has compromised the merits of many past reports, since the use of diagnostic labels has often verged on overdiagnosis.

In recent years, an emerging body of clinical and basic science information, coupled with strong academic resolve, has yielded intellectual rigor in the understanding and management of TMD. It has also permitted the introduction of a limited diagnostic classification, which recognizes 3 major groups of TMD diagnosis.[1] These are: I. muscle diagnoses, II. disc displacements, and III. arthritis. The Dworkin and LeResche classification is a non-hierarchical one, and it conveniently recognizes the most frequently encountered TMD in clinical practice. It was originally proposed for clinical and epidemiologic research purposes, but it has proven to be a very useful clinical teaching and patient management tool. This paper reviews salient aspects of Group III, or TMD that are arthritis-related.

Osteo- or degenerative arthritis (OA) is a non-inflammatory disease of moveable joints. It is generally regarded as the result of a time-dependent pattern of joint reactions to injury, rather than as a single entity. It is mainly a disease of articular cartilage that produces symptoms in single body joints. It also can affect the TMJs, whose articular surfaces are covered with fibrocartilage rather than hyaline cartilage. While the disease can be crippling, leading to a vast range of morphologic and functional deformities, it very rarely affects the TMJs to such a dramatic extent.

The disease process is characterized by deterioration and abrasion of articular cartilage and soft tissue surfaces, the occurrence of thickening and remodeling of the underlying bone, and formation of marginal spurs and subarticular "cysts." Such changes are very common in many joints in older people but are often asymptomatic. These changes are, however, frequently accompanied by the superimposition of secondary inflammatory changes, which can cause symptoms. It should be emphasized that the general physical health of the individual is seldom affected by the disease, in spite of its widespread nature and the risk of multiple joint involvement. Nevertheless, it has been stated that OA may

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account for more disability among the elderly than any other disease, not because individuals with OA are severely disabled, but because the disease is disabling in so many. This statement sounds particularly ominous when it is considered that OA affects 1 in 10 Canadians or Americans and that, by age 70, 85% of the population is likely to be affected.

Osteoarthritis has been generally regarded as inevitably progressive once it has become clinically symptomatic. More recent findings, however, support the concept of reversibility of OA, and the sooner treatment is started, the more effective it will be.[2] The progression of severity of TMJ arthritis is not known, but it is not regarded as being likely to lead to serious disability.

Prevalence

Osteoarthritis is a very common joint disorder that occurs in all populations and is strongly correlated with age. Its prevalence varies in different joints, with autopsy studies revealing that most individuals demonstrate macroscopic changes in their hip and knee joints. On the other hand, frequency of macroscopic OA lesions in TMJ autopsy material has varied from 22 to 38%.[3] Osteoarthritis occurs more often and is more generalized in women than in men, but this difference does not become evident until after age 50. Whereas radiographic and histologic studies report a very high prevalence of osteoarthritic changes, clinical investigations usually find much lower frequencies of signs and symptoms. This lack of correlation between structural OA changes and pain and other clinical symptoms has been emphasized for several joints.

The lack of concordance between structural changes and pain also applies to OA of the TMJ. Therefore, reports on the prevalence of OA in the TMJ tend to be confusing, since patients with OA do not differ from patients with other TMD with respect to subjective symptoms. This is complicated further by a growing recognition of a conceptual framework that regards OA as a late symptomatic stage in a continuum of adaptive joint changes or a process of joint degeneration. Such symptoms tend to subside on a variable time-dependent basis. While epidemiologic data on signs and symptoms of TMD occurrence are readily available, a specific focus on OA is lacking. One study reported a 24% prevalence of OA in a population of shipyard workers in southern Sweden.[4] This study did rely, however, on the assertion that the occurrence of TMJ crepitation was a reproducible clinical sign of OA.

Most reported studies on the prevalence of OA have relied on interpretations of radiographic signs. This method can be inconclusive, since incipient or early OA is unlikely to show up on conventional TMJ radiographs.[5] It appears that a substantial change in mineralized tissues is required for such changes to be recognized radiographically. Nevertheless, Boering[6] observed radiographic changes in 86% of individuals under 20 years of age, but many of these findings disappeared with time and were probably often a result of remodeling associated with growth and recovery or repair. This should have resulted in adaptation and normalized function.[7] Furthermore, the line of demarcation between adaptive joint changes and degenerative ones is not a very clear one, and in the absence of frank clinical symptoms, a diagnosis of OA may not be the correct one.[8,9]

There is a controversy in the literature with respect to the prevalence of osseous changes in asymptomatic TMJs. Almost half of a sample of 80 symptom-free individuals demonstrated radiographic changes, including OA ones in the TMJs.[10] Such observations are commonly encountered (one study even reported osseous changes in 90% of TMJs[5]), and clinical experience also suggests the frequent lack of a significant correlation between the presence of symptoms and the degree of radiographic changes. In a recent study[5] that used cephalometrically corrected tomograms, minimal flattening of the condyle or eminence was seen in 35% of TMJs in asymptomatic persons who had no arthrographic or magnetic resonance imaging (MRI) evidence of internal derangement. More advanced osseous changes were not seen, and the authors concluded that minimal flattening is probably of no clinical significance. It appears that OA prevalence is age- and gender-related and not unlike arthritis in other body joints.

Etiology

The etiology of OA is unclear but appears to include both systemic and local factors. Some authors suggest that OA may be a final common pathway for several joint conditions, including inflammatory, endocrine, metabolic, developmental, and biomechanical disorders. Age is clearly a predisposing factor, since both frequency and severity of the disease appear to increase with age. However, the evidence in favor of functionally

important, age-dependent alterations in joint tissues is not clear-cut. This tends not to support the notion that aging per se plays a primary etiologic role. On the other hand, the aging masticatory system is frequently associated with a high prevalence of complete and partial edentulism. It is therefore tempting to suggest that it is in this context of depleted dentitions and consequent adverse biomechanical loading that aging's role becomes significant. This correlation has, however, not been convincingly validated.

It has been proposed that OA is analogous to the process of heart failure, since it reconciles concepts of absolute and relative overloading. In the former, repetitive or abnormal demands (loading) may exceed functional capacity. In the latter, the functional capacity is intrinsically reduced, although the loads may be within a normal range. In the cardiac situation, heart failure secondary to hypertension results from an increase in the functional demands on essentially healthy tissues. Alternatively, heart failure after myocardial infarction leads to a decrease in the functional capacity of the tissues themselves. Both situations have their counterparts in the masticatory system; in the former, overload of parafunction can elicit gradual adaptive changes in the TMJs, which can spill over or "cross the threshold"[11,12] into OA. In the latter analogy, a macrotraumatic episode of the TMJ or the gross insult of sudden loss of molar support can render it vulnerable to future demands, with OA developing eventually. The overlap of both analogies would, of course, be likely to accelerate or magnify the process, with the earlier development of perhaps more severe signs and symptoms of OA. While this proposed paradigm may appear somewhat simplistic, it continues to offer a useful explanation for the condition as well as a rational basis for its clinical management.

Both physicians and dentists have been inclined to believe that the single most popular etiologic factor is increased mechanical loading, although OA is known to develop in non-heavy load-bearing joints, eg, the sternoclavicular joints. There exists good evidence in favor of the TMJs being exposed to increased load during function. This increased loading may lead to the conversion of shearing stresses into compressive stresses. This occurs in particular during parafunction, when a great deal of force acts across a joint—a force resolution that could very well be influenced by the morphologic state of the dentition. Autopsy evidence suggests a strong correlation between loss of molar support and the occurrence of OA, especially in those individuals over 40 years of age. These studies suffer from the limitations implicit in all correlative studies. However, when the findings are reconciled with the frequent clinical association of compromised dentitions and clinical signs and symptoms of OA, the concept of OA as a process, rather than a disease entity per se, becomes an attractive hypothesis.

While the TMJ appears to demonstrate an impressive adaptive response to morphofunctional needs, it is clearly a vulnerable component of the masticatory system. It is vulnerably located; most, if not all, of mandibular movements terminate with contact between rigid, unyielding enamel surfaces; and the variable areas of contact surfaces on occlusal tooth morphology offer considerable scope for stress concentration and deflection in the TMJ. Clearly both the macro- and micro-traumatic episodes that induce joint tissue changes, which occur at different tissue levels, may be cumulative in nature. They become part of the continuum of an adaptive response, which may eventually be exceeded. Clearly the adaptive capacity of the TMJ is not infinite.[13] Consequently, strong convictions still prevail among dentists, who identify various dentally related factors as acting singly or in combination to predispose the TMJ to OA. Differences in jaw morphology, malocclusion, occlusal discrepancies, chewing habits, partial edentulism, and parafunction are frequently indicted. However, it must be emphasized that strong scientific support for an etiologic role for any dentally related factors is lacking, even though some evidence has been presented.[11,14]

In the absence of conclusive evidence to confirm this, it cannot be stated that biomechanical factors alone are causative. The "wear and tear" explanation associated with such a notion and especially with aging is attractive and popular, and it is supported by several studies, which suggest that there is a link between repeated occupational activities and OA in overused joints. Other studies, however, have not been able to verify this hypothesis. In this context it can also be mentioned that several studies on former long-distance runners to evaluate whether they are at an increased risk of knee OA have given conflicting results. Furthermore, individuals believed to be occupationally prone to OA, eg, pneumatic drillers, parachutists, divers, etc, have not been found to have a higher incidence than the rest of the general population. However, previous major joint injury appears to be a common cause of OA; and in people with repeated joint use, for example, as in joggers and runners, a history of major joint injury seems to increase the risk of OA.

For the masticatory system, biomechanical factors such as long-term bruxism and extensive occlusal wear have been correlated to an increased rate of TMJ changes. It is not clear, however, if these changes were only adaptive, indicating remodeling, or were really OA ones. Some authors maintain that there is no scientific evidence that the increased mechanical loading during bruxism should lead to OA of the TMJ, and clinical experience shows that many bruxists have no signs of joint degeneration. The important consideration in the development of OA may very well be the condition of the joint tissues, rather than the impact of loading. It is therefore presumed that in the pathologic state, the balance between catabolic and anabolic responses of affected articular tissue is upset, and adaptation yields to disease.[12,15]

Pathogenesis

Animal experimentation, computer-simulated models, and structural changes resulting from disc perforations, displacements, and discectomy[16] underscore the very likely fact that the TMJs are load-bearing joints. Furthermore, the articular discs appear to afford protection against excessive loading.[17]

The work of Radin et al[18] suggests that repetitive impulse loading elicits subtle and clinically undetectable bone changes. These changes consist of an increase in rigidity of subchondral cancellous bone caused by callus formation, secondary to isolated trabecular fatigue fractures. The net effect is an impairment in the joint's capacity to dampen peak dynamic stresses so that damage occurs to the cartilage. While Radin et al emphasize the occurrence of microfractures, which lead to the hardening of subchondral bone and the precipitation of lesions in the articular surfaces, other authors opt for the conviction that OA starts in the articular surfaces per se. Still others claim that simultaneous changes occur both on the surface and subchondrally.[19] Several researchers report that the first affected tissue is hyaline cartilage, with the synovial surface of the cartilage undergoing fragmentation of the collagen network, a change known as fibrillation. Fibrillation is associated with depletion or degradation of the other major cartilage component, the proteoglycans which are largely water-binding molecules. It is not clear whether this change is a cause or a consequence of the disease.

A reduction in resistance to shearing and compressive forces results, and the tissues become softer and prone to deformation. If progressive, this process can lead to degradation of the articular soft tissue surface. When degradation products are produced in large quantities and cannot be efficiently resorbed from the joint cavity by the synovial membrane, an inflammatory response may be elicited and synovitis may develop.[20] Such an inflammation is a potential cause of pain. However, there are many unanswered questions related to pain in OA. Recent medical literature proposes mechanical (eg, increased pressure or destruction) and chemical (eg, inflammatory mediators, such as prostaglandins, kinins, and histamine) stimuli to various components of the joint (bone, periosteum, synovium, and capsule) and periarticular structures as possible causes of pain in and around osteoarthritic joints.[13] Degenerative changes in articular cartilage and synovia might be reflected at the clinical level as impairment of the normal, freely sliding, low-friction qualities of the major joint components. It is believed that joint pain and stiffness, as well as reduced mobility, are frequently a result of secondary inflammation of the capsular tissue. Pyrophosphate is also formed in high concentrations in synovial fluid in joints with OA. The pyrophosphate combines with calcium to form crystals, which may elicit an acute inflammatory response.

A change in function and an increased loading of the articular tissues of the TMJ, which is assumed to follow loss of molar support, or other major changes in the occlusion may stimulate remodeling of tissues. This process involves an increased synthesis of proteoglycans and a thickening of the soft tissue layer due mainly to cartilage formation, which makes the tissue more resistant to compression forces. Remodeling is frequent in the posterior-lateral part of the temporal eminence and in the anterior-lateral part of the condyle, which is supposed to carry the greatest load.[21,22]

Undifferentiated mesenchymal cells in the proliferative layer of the temporal and condylar joint components are thought to play an important part in the remodeling process. The temporomandibular disc, however, lacks this reserve remodeling capacity and is frequently involved first in OA.[23] Long-standing increased compressive forces here lead to thinning, cell necrosis, intercellular matrix degradation, and eventually perforation. An increased vascularization in damaged discs has been observed and interpreted as a sign of an attempt at repair. The thinning of the disc increases the strain on the other opposing components, and if their adaptability is exceeded, OA develops here, too.

Table 1 Diagnostic Criteria for TMJ Arthralgia, Arthritis, and Arthrosis (Group III TMD)*

Group	Symptoms
A. Arthralgia	Pain and tenderness in the joint capsule and/or synovial lining of the TMJ, including (1) Pain in one or both joint sites (lateral pole and/or posterior attachment) during palpation; plus (2) One or more of the following self-reports of pain: pain in the region of the joint, pain in the joint during maximum unassisted opening, pain in the joint during assisted opening, pain in the joint during lateral excursion; and (3) For a diagnosis of simple arthralgia, coarse crepitus must be absent.
B. Osteoarthritis of the TMJ	Inflammatory condition within the joint that results from a degenerative condition of the joint structures. (1) Arthralgia (see A.); plus (2) Either or both of: coarse crepitus in the joint, or tomograms that show 1 or more of the following: erosion of normal cortical delineation, sclerosis of part or all of the condyle and articular eminence, flattening of joint surfaces, or osteophyte formation.
C. Osteoarthrosis of the TMJ	Degenerative disorder of the joint in which joint form and structure are abnormal. Includes: (1) Absence of all signs of arthralgia, ie, absence of pain in the region of the joint, and absence of pain in the joint on palpation during maximum unassisted opening and on lateral excursions (see A.); plus (2) Either or both of: coarse crepitus in the joint, or tomograms showing 1 or more of the following: erosion of normal cortical delineation, sclerosis of part or all of the condyle and articular eminence, flattening of joint surfaces, or osteophyte formation.

*From Dworkin and LeResche[1]; reprinted with permission.
In making diagnoses of disorders in this group, polyarthritides, acute traumatic injuries, and infections in the joint should first be ruled out.

Different authors have given conflicting answers to the question of where OA changes can first be seen and in which joint component. One study[24] indicated that early degenerative changes can occur with similar frequency, but not necessarily at the same time. Thus, severe changes were observed in the condyle, together with an unaffected temporal component, and vice versa. It was also found that there was no complete correlation between microscopic, macroscopic, and radiologic examination of the same joint components. These findings further illustrate the unclear demarcation between remodeling and degeneration processes.[25]

The remodeling process can produce macroscopic changes in the form of the joint without subsequent development of OA; this should be kept in mind in the interpretation of radiographic findings. In severe cases the combined effect of OA and remodeling will result in a severely deformed joint with macroscopic bone exposure. However, the bone tissue in such lesions is covered by a thin connective tissue layer.[24,26] This finding may explain the fact that bony ankylosis is extremely rare, even in severe OA.

A synthesis of recent basic research suggests that the molecular events that may underlie TMJ remodeling and degenerative disease are complex and poorly understood. Structural changes may be the result of a series of cascading molecular events that include neuropeptide synthesis and release, generation of free radicals, cytokine synthesis, increased arachidonic acid metabolism, activation of matrix-degrading enzymes, inhibition and/or reduced synthesis of protease inhibitors, and altered cell-extracellular matrix interactions.[13]

Diagnosis of Osteoarthritis

The Dworkin and LeResche diagnostic criteria for OA, which includes arthralgia, arthritis, and arthrosis, are summarized in Table 1.

Clinical Findings

General symptoms of OA include pain or stiffness in the face and jaws, pain on wide opening, pain on chewing, inability to open wide, locking or catching of the mandible, and joint noise. Clinical signs include tenderness to palpation of the TMJ and/or the muscles of mastication, limited or deviated mandibular movements, pain with movement, locking or subluxation, and joint sounds. It is clear that recorded signs and symptoms are very similar to those of other TMD, with some subtle yet significant exceptions:

1. They almost invariably occur unilaterally.
2. The symptoms appear to worsen as the day goes on.
3. Pain is over the joint per se, especially the distal aspect when the mouth is open.
4. Crepitation (crepitus), as distinct from clicking sounds, is often present, although clicking may also be present.
5. Radiographic changes are frequent.

It should be pointed out that other aspects of TMD can be superimposed upon OA, and that the pain and stiffness are probably due to secondary inflammation of capsular tissue or the other causes discussed previously. The clinical sign of crepitus deserves some emphasis. It is a grating, grinding,

Table 2 Development of Signs and Symptoms of Osteoarthritis in Phases and Stages*

Phase	Stage
1. Clicking	Initial
2. Periodic locking	Initial
3. TMJ pain at rest	Intermediate
4. TMJ pain on function	Intermediate
5. Residual symptoms other than pain	Late
6. Absence of symptoms	Late

*Modified after Rasmussen.[29]
Note that some patients do not experience or report phases 1 or 2 and proceed directly to phase 3. Furthermore, radiographic changes do not appear to be specific to any of the observed changes.

or crunching sound, which may be audible during opening, but especially during lateral mandibular movements. It is better palpated than heard, and while some authors feel that little or no additional information is gained with the use of a stethoscope, other authors consider it a necessity.

Diagnosis of OA is based on patient history and imaging information. It also has been demonstrated to benefit from the application of specific orthopedic tests, which include active jaw movement, passive jaw opening, and palpation.[27] The active movement test includes opening, closing, lateral, and protrusive movements of the mandible carried out by the patient. Passive opening involves application of gentle stretch by the clinician on the incisal edges of the maxillary and mandibular incisors to increase mandibular opening following an active opening movement. Palpation entails bilateral palpation of masseter and temporalis muscles, the insertion of the medial pterygoid muscle, and the lateral and dorsal part of the condyle. These tests yield information that can be synthesized to identify the likely diagnostic subgroup the patient will fall into.

Kopp[23] concluded that the presence of crepitation differentiated patients with TMJ OA from patients with masticatory muscle disorders. He also observed that patients with TMJ OA differed from other patients with TMD with respect to their greater age but not with respect to sex, local symptoms (except joint sounds), duration of symptoms, headache, or symptoms in other joints. The symptoms of pain and dysfunction could of course be an interim phase in a continuum of the trauma/adaptation balance, which seems to underscore the development of the condition.[28]

Rasmussen[29] described the development of the symptomatology of OA (or temporomandibular arthropathy, as referred to by the author) in 3 stages and 6 phases (Table 2). In the first stage, the diagnosis of OA is difficult to separate from other types of TMD, including internal derangement of the TMJ, by means of both clinical and radiographic examination. In the second or intermediate stage, the TMJ becomes painful. The last stage is accompanied by a reduction of symptoms and normalization of function, while radiography reveals increasing deformation and a high frequency of disc perforation.[30]

Similar interpretations of the mainly favorable prognosis of the long-term development of OA have been reported by several authors. A patient illustrating the varying clinical and radiographic development of OA is shown in Figs 1 and 2. In spite of severe osseous changes, certainly including disc perforation, a painless function was maintained in this patient, as it is in most subjects with OA.

The relationship between disc displacement and OA is controversial, with a frequently reported opinion that disc displacement causes OA. It has been proposed that internal derangement is a sign of OA rather than its cause, and it has been concluded that OA is the basic condition that causes TMD. In fact, Stegenga et al proposed a unifying concept for TMD in 1989.[31] They argued that the TMJ was very similar to other joints in the body, in spite of its unique features; that the genesis of OA in non-calcified tissue (hence not readily radiographically detectable) is unlikely to be the result of an occlusal etiology; that the complex 2-way relationships between OA and disc displacements are an acceptable response to the proposal that the latter always leads to the former; and that extracapsular or myofascial problems are only secondary responses to a primary OA process. While such a concept is attractive, it fails to account for the entire spectrum of TMD and their acknowledged multifactorial etiology. Nonetheless, the Dutch work underscores the very close relationship between 2 of the 3 utilities described by Dworkin and LeResche[1] and deserves serious consideration. A report by Lobbezoo-Scholte et al[32] confirmed that in routine clinical practice, history-taking and conventional radiography should be accompanied by a functional examination so as to reduce confusion about an arthrogenous, myogenous, or combined origin of the disorder.

The proposed examination would comprise palpation, active mandibular movements, and passive jaw opening. This research indicates that the active movement test was the most powerful for distinguishing between pairs of diagnostic subgroups.

Fig 1 A 56-year-old woman with movement pain, palpation tenderness, and crepitation in the left TMJ and an eventual diagnosis of OA. She had experienced long-standing mild to moderate pain after extensive mouth opening and intensive chewing. Increased problems after a locking of the TMJ led to the referral to the TMD clinic. Reprinted from Zarb and Carlsson[47] with the permission of Munksgaard.

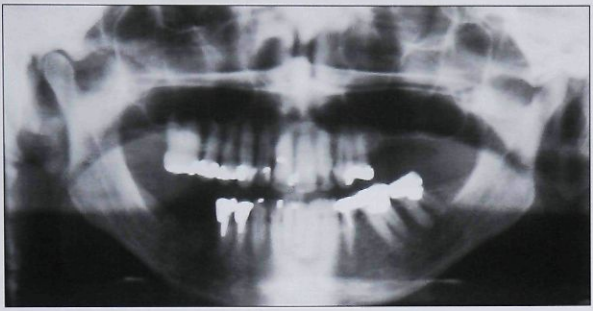
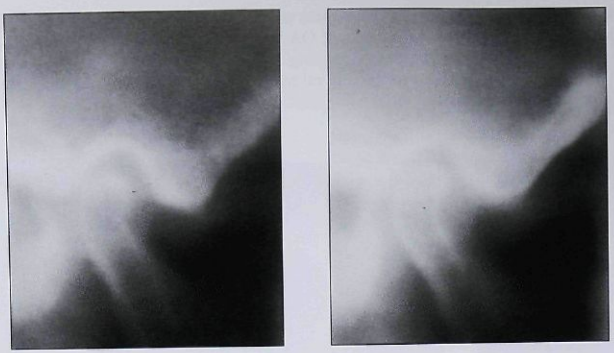


Fig 1a (*Left*) Frontal view of patient indicating location of pain.

Fig 1b (*Above*) Orthopantomogram showing bilateral 30-year-old loss of molars and suggested structural changes of the left TMJ and normal outline of the right TMJ.

Figs 1c and 1d Lateral tomograms of the left TMJ, showing extensive deformation, indicating OA (compare the mainly normal appearance of the right joint in Fig 1b). (*Left*) Lateral section. (*Right*) Central aspect of the joint. Proposed treatment consisted of a short period of splint therapy (which the patient did not like), counseling on the benign character of OA, and the recommendation to avoid extensive jaw movement. After about 4 months the patient had improved substantially and requested no more treatment. A follow-up appointment after 2 years showed that the patient had no problems but still avoided extensive chewing.



Figs 2a and 2b Lateral tomograms of the right TMJ of the patient in Fig 1, indicating increased sclerosis and some condylar surface irregularity (*left:* lateral aspect; *right:* central aspect of the joint). The patient returned after 8 years with movement pain in the right TMJ after a locking episode of that joint. The left joint had crepitation but painless function, and no obvious changes in the radiographic appearance of the left joint occurred during the previous 8 years (see Figs 1c and 1d). The treatment consisted of nonsteroidal anti-inflammatory drugs and information about the condition (the patient did not wish to have a splint). Reprinted from Zarb and Carlsson[47] with the permission of Munksgaard.

Furthermore, palpation and passive opening were also useful for distinguishing between patients and control subjects, and between arthrogenous and myogenous patients.

Laboratory Findings

At this particular stage of reporting, laboratory findings in TMJ synovial fluid appear to be of limited value.[33] This is because collection of such fluid is not routinely practical given the small quantities available, or the total lack of fluid. Ongoing research has shown that release of inflammatory mediators is associated with disease activity,[34] and the enzymatic events involved in the process of OA are being characterized.[20] These products may be useful markers for the evaluation of cartilage matrix degradation in patients with suspected OA. On the other hand, these products may also be associated with adaptive remodeling changes and may not be disease process–specific. The interpretation of such evolving monitoring methods in different genders, age groups, and stages of the disease offers much research scope but is currently not applicable to OA diagnosis.

Radiologic Findings

There are diverse methods for imaging the TMJs (see Pharoah[35]), with tomograms being a particularly popular technique. In both osteoarthritis and osteoarthrosis of the TMJ, tomographic imaging will show 1 or more of the following: erosion of normal cortical delineation, sclerosis of part of or all of the condyle and articular eminence, flattening of joint surfaces, and osteophyte formation (Figs 1 and 2). It must be re-emphasized that 1 or more of the above changes are frequently found in both symptomatic and asymptomatic joints (Table 1). This fact underscores the significance of the clinician's inability to differentiate between the presumed stages/changes in the remodeling process. These changes may be indefinitely asymptomatic, or they may culminate in a symptomatic and thus clinical diagnosis of OA (Table 2). The OA process, confirmed by symptoms and imaging evidence, may then cross an asymptomatic threshold and linger as a memory of an arthritic or symptomatic exacerbation.

It is interesting to note that a recent study of the spines of people without back pain found that nearly two thirds had spinal abnormalities, including herniated or degenerated discs. In a paper

published in *The New England Journal of Medicine*,[36] California researchers concluded that in many cases it may be sheer coincidence, not cause and effect, when a person with back pain is found to have an abnormal disc. The dental literature indicates that a similar conclusion may be relevant for OA of the TMJ.

Management of Osteoarthritis

The apparent multifactorial etiology of TMD demands an eclectic approach to their management. While the general features of this proposed strategy can, with selected modifications, be applied to all 3 major encountered disorders (see Stohler and Zarb[37]), we have fine-tuned the approach here to address the specific management of OA. A proposed therapeutic approach includes *(1)* symptomatic treatment, *(2)* control or reduction of contributory or predisposing factors, and *(3)* treatment of pathologic sequelae. An important overall consideration is the generally favorable prognosis of OA.

Symptomatic Treatment

This comprises a formula of 3 general items: patient reassurance, medication, and physical therapy. Reassurance demands a sympathetic dentist, a simple explanation of the problem and its possible multifactorial etiology, and a carefully explained and justified course of treatment. If OA is diagnosed, then it should be explained that symptoms may worsen before they improve, although in most cases the joint will eventually "heal" or recover. A useful way of illustrating this is to draw a well-shaped graph or curve, which will assist in explaining the prognosis of the condition[38] (Fig 3). It is conceded to the patient that at the time of consultation it may be difficult to assess the patient's precise position on the curve. Therefore, if the OA status is at point A, a period of deterioration will precede improvement; if on the other hand it is at point B, the condition is well on its way to getting better. It must be stressed, however, that with proper management, progress is frequently in the same direction, from left to right. Fortunately, OA patients with TMJ involvement do not appear to suffer from significant functional jaw problems, and it is only acute TMJ pain episodes that tend to necessitate active intervention.

Medication. Pain relief forms a major part of treatment, and all patients with joint pain should be provided with a regular background of analgesics. It should be pointed out, however, that there is no evidence to suggest that the natural history of OA is significantly affected by these drugs, nor, incidentally, that those who feel little pain because of a high pain threshold fare any worse than others. The ratio of hazard to therapeutic effectiveness of many of the available drugs is higher than in most other diseases, so that good judgment in the choice of treatment is required. Occasionally, specific anti-arthritic preparations are used. In patients with severe pain, intra-articular injections of anti-inflammatory agents may be used.

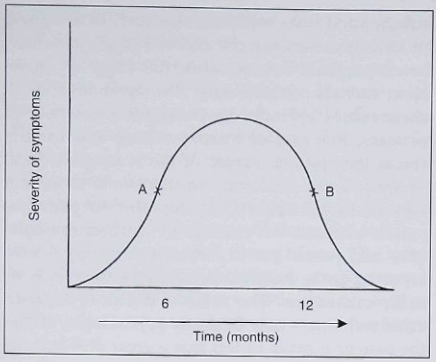


Fig 3 A similar graph may be used to explain the pattern of OA behavior (after Ogus and Toller[38]).

Physical Therapy. Rest, achieved via voluntary or imposed immobilization; heat; and remedial muscle exercises are likely modalities included in a physical therapy regimen. Tissue rest is a functional tenet of arthritic therapy, and with the TMJ it can be achieved by varying degrees of controlled joint immobilization, ranging from a soft diet and voluntary avoidance of excessive mandibular movement, to infrequent use of interarch elastics for very short periods of time (3 to 5 weeks). Such relative jaw immobilization fulfills the objective of protection from "weight bearing" during mastication, although it is probably not useful in the avoidance of other and probably more important traumatic activity, eg, bruxism. Pain seldom arises spontaneously from the rested symptomatic joint; it is usually due to some form of overload or trauma (typically minor) to which the joint is particularly vulnerable. This may be a stretching or "strain" of some soft tissue joint component, a

subchondral bone trabecular fracture, or a nipping of structures between the articular-bearing surface, or else capsule tension when the range of movement exceeds certain limits. The summation of all these minor episodes may suggest a continuous process, but careful history-taking will usually reveal the episodic nature of the symptoms. Heat or short-wave diathermy or ultrasonic treatment may be useful adjuncts in the relief of pain and muscle stiffness. Self-massage of adjacent musculature and joints per se, when tolerated, is also reported to be helpful, particularly since it is so easily carried out. The value of the more sophisticated techniques may be partly psychologic, in that the patient is made to feel that a great deal is being done to ease the discomfort.

The rationale for remedial exercises is to promote normal mandibular function. Several jaw exercises have been proposed, and they all seem to aim at achieving *(1)* the strengthening of the muscle groups controlling affected joints, *(2)* the prevention of future overloading or abuse to the joints, and *(3)* re-education in the use of damaged joints.

Control or Reduction of Contributory or Predisposing Factors

Strong anecdotal evidence suggests that overload from parafunction and/or biomechanical factors are contributory to OA. Consequently, limited dental initiatives, such as consideration of restoration of missing molar support, may be of primary concern to the dentist. Clinical impressions suggest the importance of restoration and maintenance of a functional occlusion, which includes bilateral and an adequate number of centric stops. This objective may require the fabrication of a stabilization appliance or a removable provisional prosthesis to restore unsupported or inadequate posterior dentitions. There is, however, a lack of evidence to support the claim that occlusal therapy per se is of value in the treatment of OA, since a subtle but profound difference exists between the prescription of a stabilization appliance, which presumably alleviates intracapsular effusion in an arthritic joint, and occlusal therapy. The presumed objective of a stabilization appliance is to relieve joint pressure in OA, although there is no evidence to fully support this. The accompanying ensuing uneven interocclusal relationship (which is changeable in the context of reduction in the interarticular inflammation) can then be easily modified on the acrylic resin splint surface in response to effusion changes. This is not unlike the effect of different bandaging techniques as an adjunct to orthopedic management.

It should also be emphasized that the notion of shortened dental arches (eg, bilateral stable quadrant relationships with exclusive bicuspid support) does not appear to automatically imply an increased vulnerability to OA.[39] On the other hand, Pullinger and Seligman[40] referred to a possible correlation between age, number of opposing posterior occlusal units, and occurrence of OA symptoms.

The bite plane or interocclusal stabilization appliance is therefore particularly useful in a mechanical orthopedic sense, as well as in its established role in the reduction of nocturnal and possibly diurnal bruxism. Its versatility is such that it can be designed both to replace missing posterior teeth and to provide an optimal vertical dimension of occlusion. This becomes particularly apt if parafunction is identified as being a contributing etiologic factor. When dental methods appear to be inadequate, help from allied professionals should be prescribed for these patients.

Treatment of Pathologic Sequelae

Most TMD resolve spontaneously or are directed to an earlier resolution via the previously described interventions. The nature of OA suggests a variable symptomatic progression that usually "burns out" in 12 to 24 months. This has also been interpreted as more or less complete repair or resolution of symptoms within 1 to 2 years.[41] Osteoarthritic lesions appear to increase slowly and are frequently asymptomatic. Symptomatic exacerbations appear to be self-limiting, and the previously outlined treatment strategies are supportive in nature and usually provide adequate relief. Above all, these methods aim at minimizing functional disturbances by controlling the risk of overloading or traumatizing the joint. On the other hand, some patients do end up with prolonged discomfort and intractable pain, probably as a result of provoked tissue changes that are by that point clearly pathologic. Such irreversible sequelae are sometimes considered treatable only by TMJ surgery. While few authors have given specific criteria for patient selection for surgical treatment, it seems reasonable to concede that a small percentage of patients will benefit from surgical intervention if the cause is overt TMJ disease. The surgical procedures that have been most frequently employed are condylectomy, condylotomy, discectomy and, more recently, disc repair rather than disc removal (for review see Baker[42]).

Outcome studies of discectomy performed on strict indications have shown excellent long-term results.[43] The prognosis after disc repair has not been so favorable, at least when contributory or predisposing factors have been inadequately controlled.[44]

Current—albeit incomplete—understanding of the pathogenesis of OA and in particular the genesis of disc displacements provides the clinician with more of a rationale for avoiding surgery at all costs. This should be tempered with the recognition that improved diagnosis and understanding of TMJ disease necessitates recognition of the infrequent yet indispensable role to be played by surgery.

Some authors[45] counsel the use of intra-articular injections of anti-inflammatory medications into the TMJs of patients who do not respond to conservative treatment. Both short- and long-term results of single injections of corticosteroids have been reported to be successful in efficient pain reduction, normalized joint function, and no radiographic signs of advanced joint destruction in follow-ups of more than 8 years.[46] An alternative agent, with probably fewer side effects, is sodium hyaluronate. This drug has also been shown to give a significant short- and long-term reduction of subjective symptoms and clinical signs in patients with persistent TMJ problems that had not responded to conservative treatment.[45]

Conclusions

The presumed time-dependent implications of functional or parafunctional loading may elicit adaptive and ultimately even degenerative changes in the TMJs. Under certain conditions—genetic predispositions, trauma, dental morphologic defects, etc—the adaptive or degenerative changes may cross the threshold from an asymptomatic state into a symptomatic one. In a clinical context it is prudent to regard such joint pain as the last link in a long chain of events and to try to determine where stress or stresses on an affected joint can be reduced or eliminated. Non–drug treatment seeks to reduce joint overload so that healing can occur; consequently, rest and exercise are regarded as an important part of a self-care strategy. It is also believed that provisional and reversible dental interventions (such as appliance therapy or restoration of posterior occlusal support) may produce long-range benefits. This sort of prosthodontic intervention is particularly popular as definitive treatment, but has not yet been conclusively shown to be effective in well-controlled clinical trials. It is well documented, however, that OA may not be an irreversible disorder, and while its signs and symptoms fluctuate, they tend to decrease with time and will gradually and frequently disappear. The long-term clinical prognosis of OA is usually favorable, in spite of regularly observed severe radiographic changes.

In the absence of an experimental animal model to test hypotheses of disease etiology, we continue to rely on inferences and observations. The latter have stood us in good therapeutic stead, particularly when clinical discretion and prudence lead to the avoidance of irreversible dental reconstructive procedures.

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