Osteoarthritis of the Temporomandibular Joint Organ and Its Relationship to Disc Displacement

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To overcome disagreements with regard to the relationship between disc displacement and osteoarthritis of the temporomandibular joint (TMJ), the evidence for suggested disease mechanisms and clinical course of these disorders is reviewed. The TMJ behaves as a complex organ in which biochemical and biomechanical processes regulate the physiology of cartilage, bone, synovium, ligaments, and synovial fluid. In this concept, TMJ osteoarthritis is an organ failure involving all its structures. The development of as well as recovery from disease appears to be intimately related to exceeding and supporting the adaptive capacity of the tissues that make up the joint organ. Loss of fibrocartilage and inflammation appear to be major pathobiologic processes, while serious doubts exist about the significance of disc position in joint pathology.
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Although there is consensus that temporomandibular joint (TMJ) disc displacement and osteoarthritis often occur concomitantly,[1–10] the precise relationship between these disorders remains controversial.[11] The most frequently reported relationship is that disc displacement causes osteoarthritis. Disagreement relates mainly to the disease mechanisms and to the associated clinical course, especially when the issue of classification is involved. This review attempts to overcome existing disagreements by defining basic concepts associated with these common TMJ afflictions and exploring the reported evidence against the background of basic biologic principles.

Concepts and Definitions

"Temporomandibular disorders" (TMD) is the widely accepted "umbrella term" coined by Bell[12] to designate the musculoskeletal disorders of the mandibular motor system.[13,14] Many TMD are characterized by similar signs and symptoms, traditionally

described as a triad of pain (in the joint or associated muscles), interferences during movement (frequently associated with joint sounds), and/or restriction of the range of movement.[14] This is probably the reason why these disorders have erroneously been considered a "syndrome" for so many years.

Although the term "TMD" is still used sometimes as if one deals with only a single disorder, it is generally recognized that patients with TMD commonly have a masticatory muscle disorder unrelated to joint pathology or a disease process within the TMJ. Based on this recognition, a distinction between "(mainly) arthrogenous" and "(mainly) myogenous" disorders is frequently made.[15] However, these terms suggest clarity concerning the cause of the disorder, which in fact is obscure in most cases. Moreover, such a distinction can be tricky, in that patients with a primary joint disorder usually have secondary muscle dysfunction, and patients with a primary muscle disorder may exhibit joint symptoms.[16] The terms "articular" and "non-articular" (most commonly "muscular") TMD seem more appropriate,[9,14] because these refer to the location in which the major manifestations of the disorders are observed. Articular disorders may involve responses to stimulation of tendons and muscles (in fact this is usually the case), and, conversely, muscular disorders may eventually give rise to intra-articular responses (at least theoretically). Whatever term is used for these broad categories, they are far too nonspecific to be useful as diagnostic entities.

After the concept of "disc displacement" was reintroduced as central in TMJ pathology,[3,17] the term "internal derangement" became a common diagnostic description of patients with symptoms such as pain, clicking, and restriction of mouth opening. It was proposed (and this view is still widely held) that an internal derangement would inevitably progress to degenerative joint disease.[7]

Parallel to the developments throughout the years, many efforts have been made to classify TMD.[18] The most recent classification is the dual-axis approach of the Research Diagnostic Criteria,[19] where Axis I represents the physical disorders and Axis II the degree of impairment of mandibular function and the patient's psychosocial context. In this system, in addition to muscle disorders, disc derangements and osteoarthritis are classified as separate diagnostic groups and defined by specific criteria (Fig 1). However, disorders such as rheumatic polyarthritis, infective arthritis, metabolic diseases, traumatic arthritis, and phenomena such as adhesion formation, capsular fibrosis, muscle contracture, disc perforation, hypermobility disorders, and tumors are not included in this classification. It is explicitly stated that disorders from more than 1 group (eg, disc displacements and osteoarthritis) may exist simultaneously. This indicates that these disorders are regarded as separate, and possibly independent, diagnostic entities. As one of the limitations of the Research Diagnostic Criteria, Turk noted the apparent interrelationship between the Axis I diagnoses from a study in which 60% of patients diagnosed according to this classification would receive more than 1 diagnosis, with 35% having 3 or more diagnoses.[20] It is therefore justified to explore whether—and if so, to what extent—these disorders should be considered as separate entities or, to state it in reverse, to what extent these disorders are related.

Current Definitions of Internal Derangement and Osteoarthritis

"Internal derangement" is an orthopedic term, defined as "a localized mechanical fault interfering with smooth joint movement."[14,21] Disc displacement represents only 1 of these mechanical joint disorders (Fig 2). Thus, "disc displacement" is not synonymous with "internal derangement," but it is a type of internal derangement of the TMJ.

The "classical" definition of osteoarthrosis is ". . . a non-inflammatory disorder characterized by progressive deterioration and loss of articular cartilage and subchondral bone accompanied by proliferation of new bone and soft tissue."[8p230,22p1] Degenerative changes and inflammatory processes are intimately related, and it is probably extremely rare that a degenerative joint does not display some degree of inflammation of the synovial membrane.[23] Therefore, "osteoarthritis" seems to be the appropriate term to represent this important relationship. It is essential to appreciate that osteoarthritis is a disorder that occurs in joints with as well as without an articular disc or meniscus and is characterized by 2 basic pathologic mechanisms: degeneration and inflammation. This implies that it is unlikely that disc displacement is a prerequisite for osteoarthritis to occur.

The Concept of the Joint Organ

Before reviewing the recent literature with regard to these conditions, a concept should be highlighted that might prove to be essential to overcome differences in opinion about the relationship between osteoarthritis and disc displacements.

AXIS I DISORDERS

Group I	Myofascial pain
	A No limited opening
	B Limited opening
Group II	Disc displacement
	A With reduction
	B Without reduction with limited opening
	C Without reduction without limited opening
Group III	Other joint conditions
	A Arthralgia
	B Osteoarthritis
	C Osteoarthrosis

AXIS II DISORDERS

Group I	Chronic pain grade classification (pain intensity and disability or function impairment)
Group II	Psychologic status: Depression, anxiety, nonspecific physical symptoms

Fig 1 Research Diagnostic Criteria.[19]

Internal derangements
- Structural surface irregularities
- Ankylosing conditions
  - Adherence (sticking, "hesitation")
  - Adhesion formation
  - Fibrous ankylosis
  - Bony ankylosis
- Disc derangements
  - Disc displacement (reducing and permanent)
  - Disc perforation
- Disc-condyle derangements
  - Subluxation
  - Luxation (dislocation)
- Loose body disorders

Capsular derangements
- Hypermobility disorders
- Capsular fibrosis

External derangements
- Muscle shortening disorders
- Disorders associated with pseudoankylosis

Fig 2 Mechanical temporomandibular disorders.

A synovial joint displays the 2 basic characteristics of an organ: it is a body part serving an essential purpose, and it consists of interdependent tissues. The essential purpose served by a joint is "movement to function." In the case of the orofacial region, the essential purpose of the craniomandibular articulation is "movement to serve mandibular function." There would be general agreement that normal mandibular function is characterized by painless, coordinated movement of the joint surfaces without any disturbance (interference) within at least a functional range of motion.

The other important characteristic of an organ is interdependence of the tissues of which it consists. The TMJ essentially consists of a collection of differentiated connective tissues. The extracellular matrices are synthesized as well as degradated by the tissue-specific cells (chondrocytes, osteocytes, synoviocytes, fibrocytes). The function of each of these cells is regulated by very complex feedback mechanisms in which local as well as systemic factors play a role. Important parts of these mechanisms are still obscure, although much progress has been made during the past few years. Even articular cartilage, which traditionally has been described as a relatively inert tissue because it lacks vascularization as well as innervation, has a complex, predominantly enzyme-regulated, internal remodeling system.[24]

All the connective tissues that make up the joint display a dynamic balance between form and function. All have the capacity to adapt to changing functional demands, each within tissue-specific limits.[25] With physiologic loading, there is a balance between synthesis and breakdown within the tissue (ie, between catabolic and anabolic activity). When loading exceeds these physiologic limits, protective and compensatory mechanisms are recruited to prevent, limit, or support repair of the damage. Too low an amount of loading decreases anabolic activity (ie, insufficient matrix is produced), whereas overloading initially induces adaptive responses (such as hypertrophy and hyperplasia), but when the adaptive capacity is exceeded, damage to the cells may result, leading to cell necrosis.

Remodeling of the TMJ associated with normal adaptation to altered functional demands seems to be particularly prominent during late growth and early maturity, which is when the joint components undergo considerable changes in overall shape.[26] However, remodeling continues throughout life, and at more advanced ages, the decreased adaptive capacity of the tissues may be more easily exceeded.

The maintenance of the joint in a functional state involves interdependence of the tissues. The fibrocartilage is completely dependent upon the synovial fluid for its nutrients, metabolic exchange, phagocytosis, and lubrication. Thus, this tissue is intimately related to the synovial membrane, which produces the fluid. These

delicate internal tissues are directly supported by the subchondral bone and the capsular ligaments, respectively. During functional movements of the jaw, the major part of the loading is absorbed by involuntary muscle contractions responding to proprioceptive information from muscles and ligaments. In addition, the mechanical limits of the freedom of motion (compression of joint surfaces, stretching of ligaments) contribute to load absorption. Shearing stress is absorbed by a very effective lubrication mechanism. This supporting system, together with intact neuromuscular coordination, is essential to normal function of the joint.[25] When the capacity of this complex load-absorbing system is exceeded or reduced, the tissues may become (absolutely or relatively) overloaded and possibly damaged. For example, impaired lubrication associated with degenerative and inflammatory tissue changes likely alters the frictional characteristics of the articular surfaces, which may impair the joint's movement capacity and induce gradual stretching of the disc attachments—eventually to an extent that permits disc displacement. Conversely, traumatic stretching or even rupture of these attachments may cause mechanical alterations (eg, disc derangement), as a result of which joint loading is inadequately absorbed, thus increasing the risk of direct damage to the synovium or the fibrocartilage and subchondral bone. Thus, the supporting tissues play a central role in the protection of the delicate intra-articular tissues.

The adaptive capacity of the tissues making up the joint organ, the extent of loading, compensatory mechanisms, and degenerative changes occurring within the joint are all closely related.[11] Failure of the joint organ seems to be analogous to failure of other organs. Absolute overloading increases the functional demand on essentially healthy tissues, just as hypertension may overload the heart. This elicits adaptive changes, and when the adaptive capacity is exceeded, failure may eventually result. Relative overloading refers to a decrease in the functional capacity of the tissues themselves, making the joint organ vulnerable to future demands (which may involve loads of normal magnitude). The analogy with the heart is the development of its failure after, for example, a myocardial infarction.

In this concept, dysfunction is defined as a decreased ability to move the mandible (this being the essential purpose of the TMJ organ) involving all interrelated tissues. Clinically, this manifests as interferences with or restriction of mandibular movement, which is usually accompanied by pain. The pain typically is of the musculoskeletal type,[27] ie, a deep pain that responds to mechanical provocation. Secondary effects, such as referred pain, secondary hyperalgesia, and muscle responses, are common. Joint pain may be related to strained ligaments or changes in the subchondral bone marrow.[28] Inflammation decreases the pain threshold, and under these circumstances, pain is experienced in response to normally painless stimuli.[27]

Mechanical changes may be the result of an intra-articular pathologic process, as opposed to disorders external to the joint proper, which causes abnormalities in motion and function. The former are designated by the commonly used term "internal derangement," and the logical term for extra-articular mechanical disorders would be "external derangement," a term not commonly used but certainly appropriate in this context. As noted earlier, internal derangements include disc displacements, disc-condyle assembly disorders, and hypomobility disorders resulting from articular changes such as ankylosis (Fig 2).

As far as the clinical picture is concerned, differences between most of the TMD and synovial joint disorders in general appear to be related mainly to the presence of the articular disc. Therefore, the next section focuses on research findings specifically addressing the concept of disc derangements.

Disc Displacement

Disease Mechanisms

Use of the term "disc displacement" implies that the disc previously was in a "normal" position, commonly referred to as the "12 o'clock" position, ie, with the posterior band of the disc positioned superior to the condyle. This criterion is based on the conviction that symptoms such as clicking or locking are solely the result of an abnormal disc position. From a study of computer reconstructions of the TMJ components, it appeared that disc positions other than the 12 o'clock position cannot always be considered to induce clinical joint symptoms, and that strict adherence to the 12 o'clock criterion easily leads to overdiagnosis of disc displacements.[29] Based on a consistent discrepancy in estimates of the prevalence of anterior disc position obtained from clinical examination and direct postmortem observation, it has been suggested that "anterior disc position" can be distinguished from "anterior disc displacement."[30,31] The concept that an anterior disc position associated with an otherwise healthy joint should be considered a normal variation is

supported by many autopsy, clinical, and imaging studies, which revealed its presence in approximately 30% of asymptomatic subjects and non-TMD patients.[32–39] These findings, combined with the higher prevalence of disc displacement in older subjects, led Pereira et al to suggest that this condition may, at least sometimes, be regarded as a physiologic process that increases with age.[40,41] Common symptoms such as clicking, to which the body has adequately adapted, may therefore be considered a normal characteristic.[25] Accepting these "normal" variants, disc displacement appears to be a much less common condition than is usually thought.

Many studies have shown that an anterior disc position may elicit responses, particularly in the disc and retrodiscal tissues.[42–49] When a disc is chronically displaced, the anterior band undergoes atrophy or folds over the intermediate zone, while the posterior band becomes flattened and elongated in the superior joint space and enlarged inferiorly. Responses to disc displacement observed in the retrodiscal tissues include increased presence of dense connective tissue, decreased vascularity, and decreased innervation. Isberg and Isaacsson interpreted such changes as tissue adaptation to loading,[44] while Scapino and Blaustein regarded them as signs of pathology related to the altered loading pattern.[42,46,50] However, Pereira et al found similar changes in the retrodiscal area in elderly patients, irrespective of the position of the disc.[49] This suggests that these changes may also be age-related. Whatever may be true, during altered disc position, the retrodiscal tissue may eventually adapt and function as a disc.

On anterior and posterior condylar movements, the richly vascularized retrodiscal tissue is filled with blood and in this way provides a volumetric compensatory mechanism for pressure equilibration and sets up a "pumping" mechanism that is of great importance for the joint's nutrition and lubrication.[51] Interestingly, Wilkinson and Crowley showed that this mechanism appears to remain largely intact with disc displacement—even when degenerative changes are present, because the adaptive or maladaptive changes appear to occur mainly in the anterior part of the retrodiscal tissue.[51]

In a histometric study of 53 postmortem joints, anterior disc position was demonstrated to be associated with deviations in the dimensions of the inferior synovial cavity, while the size of the superior cavity seemed little affected.[52] There was a discrepancy in alignment between the condyle and the disc complex due to elongation of the cap constituted by the disc and its attachments as well as a smaller than normal size of the condyle. This discrepancy may reflect either a constitutional deviation or may have resulted secondarily from remodeling changes. In a study evaluating histologic changes in relation to an anteriorly positioned disc, it appeared that ". . . degenerative and remodeling changes of the condyle [and] the temporal component . . . exhibited associations with age that were not apparently affected by disc position."[53p401] Kondoh et al found a greater prevalence of morphologic changes in the inferior than in the superior surface of the disc, and they found no relationship between surface irregularities in the joint and the position of the disc, although perforations were found more frequently in joints with disc displacement.[54]

In joints from adolescents, rather prominent progressive remodeling changes, but no significant regressive remodeling or degenerative changes, were observed.[53] Thus, an anterior disc position in this younger age group could be related to progressive remodeling associated with growth. This is in agreement with observations that the condyle undergoes considerable change in shape during the period of transition from growth to adulthood.[55] In these cases the anterior disc position could indeed be within the range of normal anatomic variation.

Clinical Course

Retrospective studies support the general idea that an internal derangement is likely to progress to osteoarthritis.[7,56–58] However, this course has rarely been reported in prospective studies. That a disc displacement with reduction may persist for decades implies that a progressive course does not seem to be a general rule.

To diagnose disc displacement, the preferable method appears be a thorough clinical examination.[37] However, an anterior disc displacement without reduction appears to be difficult to diagnose with clinical methods alone. In contrast to the Research Diagnostic Criteria,[19] restriction of movement appears to be absent in almost 50% of the cases, while joint noises are present in about 50%.[59,60] Irrespective of the type of disc displacement, pain appears to be more frequent, condylar translation is less prevalent, and sclerosis is more common in joints with a static disc, suggesting that disc mobility seems to be more important than the type of disc displacement.[61] In view of basic pathologic processes, a better diagnostic description than "disc displacement with or without

reduction" would be "internal derangement with or without adhesion."[14,62]

Kurita et al followed a sample of 40 patients with permanent disc displacement for 2.5 years without treatment.[63] Spontaneous improvement was seen in about 75% of the cases, while the other 25% showed no improvement or required treatment. Of the improved group, almost 60% were asymptomatic. Previous studies also showed that disc displacement and associated problems may resolve on their own in many cases.[64–67]

From the above, it appears that an alternative to the commonly held concept of disc displacement is that an anterior disc position associated with an otherwise healthy or adapted joint should be regarded as a "normal" variation. Adaptation seems to occur often, which may explain the frequent spontaneous clinical improvement. Physical "abnormalities" in disc morphology or position are not necessarily associated with clinical or historic presentation of a TMD.[68] Given the prevalence of disc displacement in a non-patient population, its identification may be more coincidental than causal for a TMD.

Osteoarthritis

Disease Mechanisms

Osteoarthritis basically involves degeneration and inflammation.[11,23,25] Degeneration may be considered a maladaptive response and is therefore closely related to adaptation. Both adaptation and degeneration may give rise to tissue remodeling. Adaptation involves basic changes in the size and activity of cells (hypotrophy and hypertrophy) or in the number of cells (hypoplasia and hyperplasia). These cells produce matrix components, proteolytic enzymes, and inhibitors of proteases, cytokines, and growth factors. All of these are necessary to maintain the balance between catabolism (breakdown) and anabolism (synthesis).[24] Adaptive changes of the joint involve synthesis of tissue and release of breakdown products into the joint fluid, which activate cytokines and phagocytes to clear the breakdown products from the joint. These processes are entirely physiologic and occur continuously.

As opposed to adaptive responses, degeneration of a tissue involves replacement of the original tissue structure by a tissue structure of inferior quality. In the pathologic state, the balance between catabolic and anabolic responses of tissues is upset.[69] In other words, adaptation (intact balance) yields to maladaptive processes. On a cellular level, in addition to adaptive responses, necrosis is present.

Normal tissue turnover involves synthesis and breakdown in a well-regulated balance. A relative increase of breakdown, ie, degenerative activity, leads to accumulation of degradative products that cannot be readily cleared from the joint cavity. Primarily there is an adaptive increase in phagocytotic activity of the synovial membrane, expressed as synovial hyperplasia.[70] When this adaptive capacity is exceeded (ie, when the amount of degenerative products exceeds the capacity of the synovial tissue to clear them from the joint), an inflammatory response may become clinically evident. Thus, in osteoarthritis, an inflammatory reaction reflects increased degenerative activity and is therefore an integral part of the disease process.

Clearly, cartilage breakdown may reflect decreased synthesis of new matrix, increased breakdown of existing matrix, or both. Several mechanisms may be involved in the maladaptive processes occurring in osteoarthritic diseases.[71]

Suppressed synthetic function related to a limitation of cellular function, eg, resulting from insufficient nutrition from the synovial fluid, could contribute significantly to a maladaptive state. In addition, chondrocyte metabolism is exquisitely sensitive to the biomechanical environment.[72] Insufficient loading, static loading, and inappropriate cyclic loading decrease proteoglycan synthesis, whereas the appropriate level of cyclic loading greatly enhances this process.

Matrix breakdown is predominantly proteolytic.[73–75] Cytokines such as interleukin-1 and tumor necrosis factor alpha are known to induce the synthesis and activation of matrix metalloproteases by chondrocytes. Interleukin-1 could not be detected in normal controls, while it was detectable in TMJs with internal derangements and osteoarthritis in comparable amounts.[76] In another controlled study, no detectable tumor necrosis factor levels were found in patients with masticatory muscle disorders, while elevated levels were found in about 50% of patients with disc displacement and in almost all patients with osteoarthritis.[77]

About a decade ago, it was proposed that an imbalance develops between the level of metalloproteases and the level of tissue inhibitors of metalloproteases.[78] This hypothesis was based on the observation that the levels of inhibitor and enzyme are not very far apart in a healthy joint and that enzyme activity increases several times in osteoarthritis, whereas inhibitor levels increase

only about 50%. In the years since the idea was advanced, further supporting evidence for this hypothesis has continued to accumulate.[79,80]

Matrix degradation may be further enhanced by synovial inflammation. Changes in the synovium in osteoarthritis generally reflect limited inflammation, much less than in rheumatoid arthritis.[81] There is no marked accumulation of infiltrating mononuclear cells and plasma cells, but there is some synovial hyperplasia.[70] The synovium is a rich source of hyaluronic acid, a glycosaminoglycan that acts as a boundary lubricant for soft tissues. Although it is less marked than in rheumatoid arthritis, there appears to be an increased hyaluronic acid level in the serum in osteoarthritis, especially in patients with the most destructive joint disease.[82] This observation suggests that an inflammatory process may lead to more rapid destruction of cartilage.

Another enhancing factor related to matrix degradation is the presence of neuropeptides. Nerve terminals containing neuropeptides have been detected in various TMJ tissues,[83–89] especially in the anterior capsular ligament and in the retrodiscal tissues. Traction or compression in these regions stimulates the nerve terminals to release these neuropeptides into the surrounding tissues. This may evoke an inflammatory response and increase the synthesis of cytokines. These cytokines activate proteases and thus contribute to matrix degradation and inhibit repair of damaged matrix.

Milam et al suggested the accumulation of free radicals as a potential mechanism for the initiation of the molecular events that are seen in osteoarthritis in susceptible individuals.[90] Normally, accumulation of free radicals is prevented by endogenous free radical–scavenging mechanisms. However, tissue damage may result if the scavenging capacity is exceeded by an overwhelming production of free radicals, or if the scavenging capacity is compromised. Free radicals may be produced by direct mechanical trauma as well as by several other mechanisms. For example, an increased intra-articular pressure in joints with marked effusion may impede synovial capillary perfusion, resulting in a period of hypoxia.[91] Hypoxia can lead to alterations in the metabolism of affected cell populations, and on reperfusion, metabolically transformed cells may generate free radicals.[92] This mechanism is termed *hypoxia-reperfusion injury*.[93] Another mechanism is the production of hydroxyl radicals and nitrogen dioxide from nitric oxide, of which increased levels have been shown in a controlled study to be involved in cartilaginous degeneration.[94] Also, evidence was provided recently that focal (micro)bleeding resulting from direct trauma or from vessels in inflamed tissues provides a source of redox-active iron that may catalyze the formation of extremely damaging hydroxyl and ferryl radicals.[95]

Damage evoked by the accumulation of free radicals in affected joint tissues can be expanded by the production of extracellular matrix degradation products, by activation of inflammatory cells to synthesize and secrete cytokines, and by the generation of proinflammatory molecules (eg, prostaglandins, bradykinin, histamine) that could contribute to additional microbleeding and pain. Collagen breakdown results in disruption of the 3-dimensional collagenous network,[96] which is normally kept under tension by hydrophilic proteoglycan aggregates. This results in an increase of the volume occupied by the proteoglycans. As a result, the tissue swells and softens (termed *chondromalacia).* An increase in the ratio between proteases and protease inhibitors results in depletion of proteoglycans. Thus, in this stage, an increased content of proteoglycans in the synovial fluid may indicate increased degeneration, which has been demonstrated in several studies.[97–99] The softer cartilage surface is more susceptible to deformation, and the relative load on the underlying subchondral bone increases in these areas. Increased impact loading, especially when repetitive, may cause microfractures, which are known to be powerful inducers of remodeling.[72] The result is increased bone stiffness due to sclerosis. Radin et al have stressed the role of subchondral bone stiffening in the initiation and progression of cartilage damage.[72,100]

Signs of ongoing fibrocartilage disintegration include fibrillation, formation of vertical and horizontal splitting, and subsequent thinning of cartilage.[101] As noted, the increase of breakdown products in the synovial fluid may result in an inflammatory response in the synovial membrane, which may produce an appearance of joint effusion on magnetic resonance imaging, probably representing tissue edema rather than increased fluid production. Joint "effusion" has been shown to be present in 80% of painful joints.[99] Effusion may contribute to restriction of motion and an increase in intra-articular pressure, which in turn may induce a hypoxia-reperfusion injury, generating free radicals.

Because of these changes of the articular surfaces and the synovial fluid, the mechanical properties and lubrication of the joint surfaces alter,

which may cause frictional movement, adherence, and formation of adhesions. Lack of movement may permit adhesions within the capsule and the joint cavity to mature, and eventually capsular fibrosis, adhesive capsulitis, and fibrous ankylosis may result. Depending on the condition of the joint tissues and the degree and duration of loading, adaptive and compensatory changes (eg, muscular responses, changes within the capsule, retrodiscal tissue and subchondral bone) or decompensation (eg, erosions, perforations) may occur.[14,25]

Clinical Course

The presenting clinical signs and symptoms are related to the inflammatory responses and (mechanical) changes of the associated tissues. Importantly, the long-term outcome of osteoarthritis appears to be good. In a series of retrospective studies, patients were examined 30 years after non-surgical treatment for osteoarthrosis.[65] It appeared that the occurrence and extent of radiographic bone change increased,[102,103] while reported symptoms and clinical signs had decreased.[104,105] Long-term evaluation of discectomy patients has shown radiographic bone changes in the joint, despite the reduction in pain experienced by these patients.[106–109] Radiographic changes are found in symptomatic as well as in asymptomatic joints. About 50% to 90% of symptom-free individuals have been shown to have radiographic changes.[110,111] This may reflect the tendency of clinical symptoms of osteoarthritis to subside with time, but radiographic changes may also be the result of remodeling that is associated with growth and recovery or repair, which results in adaptation and normalization of function. On the other hand, an absence of radiographic signs does not rule out the presence of osteoarthritis, since early degenerative changes cannot be detected by radiographic examination.[11,111–113]

When the Research Diagnostic Criteria[19] for osteoarthritis (ie, arthralgia, presence of crepitus, and radiographic changes) are applied, only patients who are in the residual phase of the disease are included. Changes consistent with osteoarthritis were found in 65% of the patients with persistent pain, joint noises, and restriction of opening who underwent arthroscopy.[23] These data suggest that osteoarthritis is a common disorder in patients with signs and symptoms of TMD.

From the above, it may be concluded that there is considerable evidence for biochemical and biomechanical processes underlying osteoarthritic disorders. The course of the disease is characterized by an initial phase (chondromalacia), an intermediate phase of various signs and symptoms (osteoarthritis), and a phase with residual signs in which inflammatory symptoms occur only when the joint is overloaded (residual osteoarthrosis). Therefore, several factors must be assessed to diagnose the disease. The extent of cartilage degradation can be assessed with increasing reliability by means of synovial fluid analysis and by arthroscopic inspection. The reactive synovitis can be assessed clinically and, if necessary, supported by synovial fluid analysis, arthroscopic inspection, and T2-weighted magnetic resonance imaging.[114] Changes in the capsule, disc, retrodiscal tissue, subchondral bone, muscles, and occlusion can be assessed clinically and with proper imaging examinations.

The Relationship Between Disc Displacement and Osteoarthritis

Although there is almost universal consensus that degenerative changes and disc displacement often occur concomitantly and are probably associated, both entities are frequently regarded as separate TMD. Osteoarthritis is regarded as a failure of articular cartilage and subchondral bone, while disc displacement appears to involve primarily the disc proper and its attachment complex. At times, it appears as though clinicians believe that painful joint derangements involve only displacement of an independently functioning disc. However, when we realize that the TMJ is in fact an organ, the result is a more comprehensive frame of reference.

Schiffman et al have provided evidence that suggests that physical "abnormalities" in disc morphology or position are not necessarily associated with a clinical or historic presentation of a TMD.[68] In fact, there appears to be a fairly high prevalence of disc displacement in individuals who have no other signs or symptoms of any TMD.[37,115] When disc displacements are actually thought to be related to disease and are treated, a change in disc position usually cannot be realized; yet patients still report improvement. There are 2 possibilities in these patients: either treatment of a disc displacement may not be indicated because improvement likely occurs on its own, or the disc displacement itself is not actually related to the symptoms. In that case, the identification of a displaced disc, even in a symptomatic patient, may be sheer coincidence.

The available evidence does not establish strong support for a central role of disc displacement.[116] Ohrbach recently stated that the classical concepts of TMJ internal derangement are being challenged by findings focusing on the microscopic and molecular level. Acceptance of these findings means that it no longer seems appropriate to direct treatment primarily at restoration of "normal" anatomic relationships within the joint.[117]

Several authors have demonstrated that degenerative changes in the articular surfaces may be present in a joint with a "normal" disc position.[6,40,55] Pereira et al studied 2 age groups.[40] In the elderly group they noted degenerative changes in 18% of the joints with normal disc position, and in the young group nearly one third of the joints with normal disc position had degenerative changes. Therefore, some factor other than disc displacement and aging is likely involved in the breakdown of the articular surfaces. Possible factors have been previously discussed in this article.

Degenerative and remodeling changes in the condyle and the temporal component exhibit associations with age that are, apparently, not affected by disc position.[53] This does not support previous conclusions that uncorrected internal derangement constitutes a particular risk for the development of osteoarthritis. Rather, degeneration of the joint components other than the disc may be only an accompanying feature of abnormal disc position,[6,41] or it may be considered its cause.[11]

True anterior disc displacement, as distinguished from anterior disc position as an anatomic variant, presumably results from an altered relationship in the size of joint components that previously had been normal.[52] Such a secondary discrepancy could be the result of articular remodeling that could be related either to normal biologic adaptation or to degeneration. Progressive and regressive remodeling of articular tissues associated with adaptive or degenerative changes, respectively, may result in dimensional changes that could account for the discrepancies in the size of the joint components seen in internally deranged joints.[52]

Conclusions

The TMJ appears to behave as a complex organ in which dynamic processes involving biomechanical forces and cytokines regulate the physiology of the cartilage, bone, synovium, ligaments, and synovial fluid. A healthy articular organ in a fully functional state exists as a result of the interdependence and integrity of its tissues. The extracellular matrices of fibrocartilage, bone, and ligaments are assembled and maintained by the cells within them, and these same cells also produce degradative proteinases, cytokines, and other mediators, such as nitric oxide, that can alter the normal balance between synthesis and degradation. In this concept, osteoarthritis should be regarded as an organ failure, rather than an isolated disease state. Its pathophysiology may, therefore, be best understood by examining the nature of the interrelationships among the tissues that make up the joint.

The recent literature has raised serious doubts about the pathologic significance of disc position as the sole cause of pain and joint dysfunction. Joint failure cannot be effectively treated by a concentrated interest of the clinician or the researcher solely on 1 tissue or structure, such as the TMJ disc. It must be appreciated that joint failure involves all the delicate internal structures and their interrelationships. Damage to 1 tissue caused by trauma, inflammation, or degeneration may affect the whole joint. Loss of fibrocartilage and inflammation appear to be major pathobiologic processes common to almost all forms of arthritis, which may eventually lead to failure of the joint organ. In general, synovitis appears to be more pronounced in rheumatoid arthritis than in osteoarthritis, despite a shorter duration of the disease.[118] This may indicate a more aggressive and faster development of the disease in rheumatoid arthritis, which is further substantiated by the more commonly observed erosions in rheumatoid arthritis.[119] There also appear to be similar synovial tissue reactions in (generalized) osteoarthritis and rheumatoid arthritis patients.[120–123] It seems that diseases with different etiopathogeneses evoke similar tissue responses in the TMJ. Cytokines as well as biomechanical effects appear to be involved in both diseases. The influence of cytokines predominates in rheumatoid arthritis. Clinically, synovitis plays a relatively minor role in osteoarthritis, while mechanical signs determine the clinical picture to a more considerable extent. The presence of a disc in the TMJ probably accounts for the relatively prominent mechanical nature of the signs in disorders of the TMJ (including osteoarthritis), as compared with those of other synovial joints.

We have seen that tissue changes in response to loading may involve adaptation and compensation, as well as maladaptation and decompensation. The development of, as well as recovery from, disease states is intimately related to exceeding and supporting the adaptive capacity of the tissues that make up the joint organ. Thus, a central

theme for future studies should be the adaptive capacity of the joint tissues and the distinction between adaptation and maladaptation.

Much progress has been made in identifying cytokines and their functions. This knowledge may provide new therapeutic directions to control tissue damage and promote repair. Treatment of the inflammatory component of osteoarthritis not only may provide pain relief but also may arrest the degenerative changes mediated by cytokines produced as part of the inflammatory process. Therefore, a further field of future research should address the inflammatory process and its relationship to tissue degeneration. When this is understood, the pathology of osteoarthritis and its related manifestations (including disc displacement) might be more effectively regulated.

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