REVIEW ARTICLE

Progressive mandibular retrusion—idiopathic condylar resorption. Part I

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Idiopathic progressive condylar resorption has been recently identified as a clinical problem. Little is known about this condition. Isolated instances have been reported in which the mandible inexplicably moves into a Class II position. Two forms of condylar resorption have been theorized: (1) adult, the mandible recedes after growth completion, and (2) juvenile, potential mandibular growth rate is diminished. The adult form has been clinically documented. To date, the juvenile form has not been proven. Radiographic study in these cases indicates a diminished condylar head volume, decreased ramus height, change in condylar shape, and progressive Class II basal bone relationship. Patients undergoing orthodontic care and/or orthognathic surgery may experience this condition.[1-3] It is not clear whether progressive condylar resorption during and/or after occlusal treatment is associated with treatment factors or events and conditions present before treatment. This article will examine the process of progressive condylar resorption.

A clinical and cellular classification and explanation will be presented that will broaden our concepts of the temporomandibular joint. This article is not meant as *the* answer but rather a spring board to new horizons in our understanding of temporomandibular joint pathophysiology.

DEFINITION OF A NORMAL TEMPOROMANDIBULAR JOINT

A normal temporomandibular joint is typified by the following functional characteristics:

1. The ability of articulating surfaces to move painlessly over one another within a required range of motion.
2. Proper load distribution across a joint.
3. Stability of the joint during function.
4. Support for the dentition in an interdigitated occlusal position.

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The fulfillment of the above criteria is dependent on:

1. The matched congruity of opposing surfaces.
2. The mechanical properties of extracellular matrices of bone, cartilage, fibrocartilage, and connective tissues must meet the functional demands of joint use on both a physiochemical and mechanical level. Sensitive feedback loops that direct cellular interactions, extracellular matrix synthesis, and degradation in normal turnover processes must be present to satisfy the homeostatic requirements in maintaining structural and functional harmony.[4]
3. The intact integrity of the ligaments, muscles, tendons, capsule, nervous system, vascular supply, synovium, and lubrication.[5-7]
4. The tissue mass of the joint structures must remain constant to maintain a stable occlusal relationship.

Two distinct categories of temporomandibular joint remodeling can be envisioned (Fig. 1): (1) functional remodeling and (2) dysfunctional remodeling. Functional remodeling of the temporomandibular joint (TMJ) is characterized by morphologic changes involving the articular structures of the joint that are not associated with any significant alterations in the mechanical function of the joint or occlusion. Functional remodeling is characterized by TMJ morphologic change, stable ramus height, stable occlusion, and normal growth.

Remodeling of the temporomandibular joint is dysfunctional if it adversely affects the mechanical function of the joint and occlusion. Dysfunctional remodeling is distinguished by TMJ morphologic change (decreased condylar head volume), decreased ramus height, progressive mandibular retrusion (adult), or decreased growth rate (juvenile). This condition is characterized by an excessive or sustained physical stress to the articular structures that exceeds normal adaptive capacity or exceeds a decreased adaptive capacity.

The effects of dysfunctional remodeling (condy-

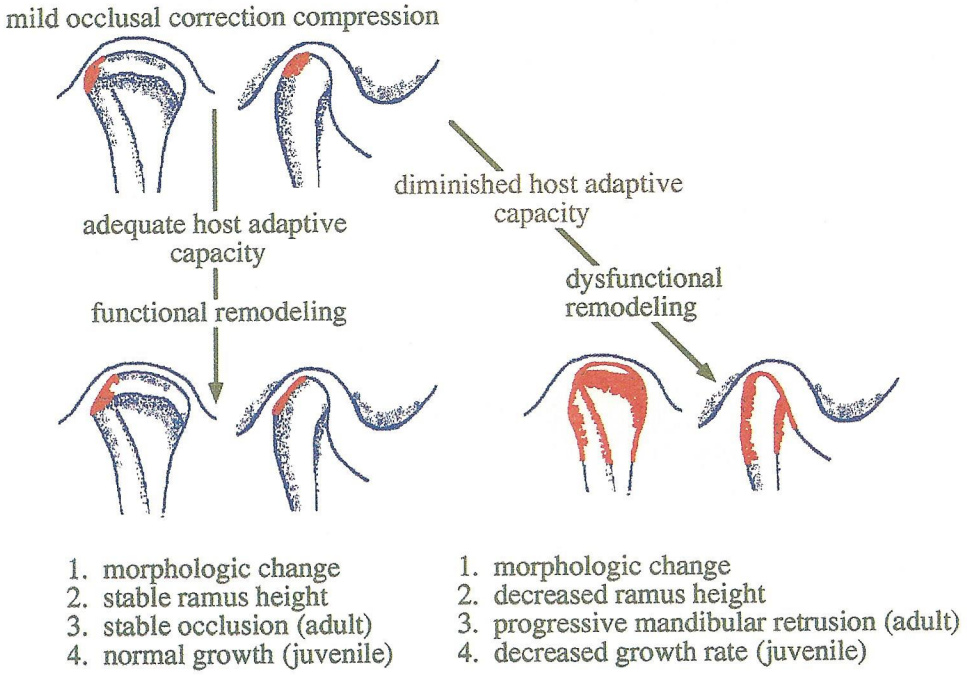

Fig. 1. Mild compression (upper left condyle pair) associated with occlusal correction is depicted. Compression of temporomandibular joint associated with dental care routinely results in functional remodeling. If diminished host adaptive capacity exists, normal loading forces associated with occlusal correction may lead to dysfunctional remodeling. Only condylar changes are depicted in this example. It is possible that meniscal tissues and fossa may also undergo changes affecting support of mandible and dentition.

lar resorption) on adult mandibular position are clear. As the condyles progressively resorb, the mandible progressively retrudes. The cause (condylar resorption) and the effect (progressive mandibular retrusion) are undeniably and clearly linked.

Adult mandibular position +
dysfunctional remodeling (condylar resorption) =
progressive mandibular retrusion

The incidence of adult condylar resorption appears to be rare. Arnett and Tamborello[8] reported 10 cases of progressive Class II secondary to condylar resorption presenting in approximately 800 dentofacial deformities examined over a 10-year period. If condylar resorption was common, adult patients would frequently present with progressive bite changes; this has not been the experience in dentistry.

The linkage of dysfunctional remodeling (condylar resorption) with deficient mandibular growth is far less clear. Multiple variables including genetic coding, development factors (such as airway, allergies, and habits), and dysfunctional remodeling (condylar resorption) may produce deficient mandibular growth. Net mandibular projection is the summation of these three growth elements.

Genetic mandibular length +
developmental influences +
dysfunctional remodeling (condylar resorption) =
net adult mandibular length

Genetic coding surely plays the most prominent role in determining deficient mandibular growth. Just as genetic coding determines mandibular protrusion, coding also must encourage deficient mandibular growth. *Developmental influences* probably retard full development of mandibular length through poorly identified and understood forces. As with the adult, factors that cause *condylar resorption* must play a role in offsetting the otherwise potential growth of the mandible.

Symptoms and signs associated with functional and dysfunctional remodeling are highly variable, presumably because of the variable stimuli creating the changes. Clinical signs and symptoms of dysfunctional remodeling of the temporomandibular joint may include a reduction in masticatory performance, gross structural changes (i.e., decreased

Table I. Factors capable of initiating changes in structure of TMJ

I. Host adaptive capacity factors
A. Age
B. Systemic illnesses
1. General
2. Autoimmune diseases
3. Hyperparathyroid
C. Hormones
1. Sex hormones
a. Estrogen
b. Prolactin
2. Corticosteroids
II. Mechanical stress (compression or stretch)
A. Occlusal therapy
B. Internal derangement
1. Compression
2. Vascular insufficiency
C. Parafunction
D. Macrotrauma
E. Unstable occlusion

ramus height with progressive mandibular retrusion or apertognathia), muscle or joint pain with function, limited mandibular range of motion, and a reduced mandibular growth rate (juvenile). Functional and dysfunctional remodeling represent a spectrum of articular surface changes with condylysis being the most severe, resulting in near or total loss of the condyle.

How do degenerative joint disease, osteoarthrosis, and osteoarthritis relate to functional and dysfunctional remodeling?

First, traditional descriptive categories do not infer the mechanism of disease but describe the end-state based on poorly defined and understood parameters. Dysfunctional remodeling and functional remodeling similarly do not infer mechanism but are well defined and concise. Further, the dysfunctional remodeling category is broad and encompasses the traditional disease descriptions of degenerative joint disease, osteoarthrosis, and osteoarthritis. When these traditional disease descriptions entail joint morphologic change but do not reduce ramus height, alter existing occlusion or result in decreased growth, the traditional disease classification is an example of functional remodeling. Conversely, if the traditional disease term entails joint morphologic change, decreased ramus height, altered occlusion, or decreased growth rate the process would exemplify dysfunctional remodeling.

The temporomandibular joint is believed to be in a constant state of remodeling (cellular and extracellular matrix turnover). The primary function of remodeling is to maintain functional and mechanical relationships between articulating surfaces of the joint. Remodeling is an essential biologic response to normal functional demands, ensuring homeostasis of joint form, and function and occlusal relationships. *In addition, remodeling may result when changes occur in either the host adaptive capacity or when mechanical stresses are placed on the joint structures* (Fig. 1 and Table I). Host factors (i.e., age, systemic disease, hormones) may contribute to dysfunctional remodeling of the temporomandibular joint even when the biomechanical stresses are within a normal physiologic range. Alternatively, excessive mechanical stress (i.e., magnitude or chronicity) may provoke dysfunctional remodeling in the absence of predisposing host factors. Remodeling secondary to host changes or mechanical stress may not maintain homeostasis but may lead to changes in form, function, and occlusion. The *remodeling capacity* responds to *mechanical stresses* imposed on the TMJ. The effectiveness of the host remodeling capacity and the number and extent of the mechanical stimuli determine the degree of TMJ and occlusal morphologic and functional changes.

Host remodeling capacity + mechanical stress =
TMJ morphologic change = occlusal change or stability

Factors that diminish the functional remodeling capacity or increase the biomechanical stress on the TMJ may adversely alter TMJ morphology, function, and occlusion (Table I).

Host remodeling capacity

What factors influence the host remodeling capacity of the temporomandibular joint? Advancing age, systemic illness, and hormonal factors may define the host adaptive capacity of the temporomandibular joint.[9]

1. *Age:* The mean age of progressive condylar resorption, when reported in the publications, is 20.5 years. For unknown reasons progressive resorption occurs in a young age (second and third decade) group.[2,8,10-15] This age of occurrence is distinct from degenerative joint disease, which characteristically occurs in the fifth and sixth decades of life and is secondary to reduced host adaptive capacity[16,17] and diminished cellular density of the articular cartilages.[18]

2. *Systemic illnesses:* Systemic illnesses may influence fibrocartilage metabolism and could affect the adaptive capacity of the temporomandibular joint. These illnesses may include autoimmune disorders, endocrine disorders, nutritional disorders (i.e., anorexia nervosa), metabolic diseases, infec-

tious diseases, cardiovascular diseases, blood dyscrasias, and excessive psychological stress.

In particular, autoimmune diseases have been associated with condylar resorption in many publications.[19-24] The immune response may be directed against specific molecules found in the extracellular matrices of articular tissues in the temporomandibular joint. This immune response can lead to the generation of tissue damaging free radical molecules, as well as the synthesis and activation of matrix degrading enzymes. Alternatively, a decreased blood supply to the mandibular condyle associated with the autoimmune process may account for some of the condylar resorption that is commonly observed in these diseases. Vessel lumen obliteration, vasculitis, fibrotic impingement on small feeder vessels, and atrophy of the muscles surrounding the temporomandibular joint leading to small vessel impingement may contribute to the diminished vascular supply to the mandibular condyle.

Hyperparathyroidism may also affect temporomandibular joint remodeling. Dick studied 39 patients undergoing long-term hemodialysis for renal failure.[25] Six patients (5 of 6 were asymptomatic) had condylar resorption which was presumed related to secondary hyperparathyroidism produced by prolonged hemodialysis.

3. *Hormones:* Hormonal factors may have a marked influence on remodeling of the mandibular condyle.

Estrogen

Arnett and Tamborello[8] have reported on female idiopathic condylar resorption. Over a 10-year period, 10 patients presented with a singular chief complaint of progressive Class II malocclusion development. All the patients were women, in the second and third decade of life. Of the 10 nine had a history of joint loading (seven orthodontic, one extractions, one mandibular trauma without fracture, one unknown cause). Examination of this group revealed progressive, bilateral condylar resorption leading to progressive Class II malocclusions. Forty other cases (all female) of severe condylar resorption (condylysis) have been reported in the literature.[2,3,8,10-15] These clinical experiences indicate that some female patients may be predisposed to dysfunctional remodeling of the temporomandibular joint in response to loading associated with occlusal treatment. This apparent female preponderance for dysfunctional remodeling of the temporomandibular joint suggests a potential role of sex hormones as modulators of this response.

Estrogen is increasingly receiving attention for its potential role in condylar resorption.[8,26-29] Aufdemorte[26] and Milam[27] examined primate temporomandibular joints for estrogen and progesterone receptors. Female baboon temporomandibular joints have estrogen receptors while male baboon temporomandibular joints do not. The presence of estrogen receptors in primate female temporomandibular joints suggests a potential relationship between estrogen-mediated cellular activities and the preponderance of TMJ problems in females. Recently, Abubaker studied human temporomandibular joints for estrogen and progesterone receptors with an immunohistochemical method.[28] He found that 72% of symptomatic females and only 14% of asymptomatic females had immunodetectable estrogen receptors in tissue specimens obtained from their temporomandibular joints. Abubaker concluded, "It is feasible to speculate that the concurrent presence of these receptors and specific circulating hormone levels will lead to connective tissue alterations in the TMJ disk, causing some structural changes in this issue." These findings are consistent with those of Tsai[30] who observed an increase in estrogen and estrogen receptor density in osteoarthritic knee joints relative to normal joints. These findings suggest that symptomatic females may have different responses to estrogen levels based on available target receptors within the temporomandibular joint.

The exact role of estrogen in the pathogenesis of temporomandibular joint disease (i.e., dysfunctional remodeling) is unclear. Estrogen inhibits cartilage synthesis in animal models of osteoarthritis.[31] Estrogen also increases the production of specific cytokines that have been implicated in inflammatory joint diseases.[32-34] Cytokines are soluble peptides produced by a variety of cells including synoviocytes and chondrocytes.[35-38] Certain cytokines can stimulate the synthesis of matrix degrading enzymes by local cell populations.[39,40] These matrix degrading enzymes are likely involved in condylar remodeling. Therefore estrogen can potentially exacerbate dysfunctional remodeling of articular tissues of the temporomandibular joint by inhibiting fibrocartilage synthesis and enhancing extracellular matrix degradation (Fig. 2).

Prolactin

Prolactin, a hormone responsible for initiating postpartum milk letdown, can exacerbate cartilage

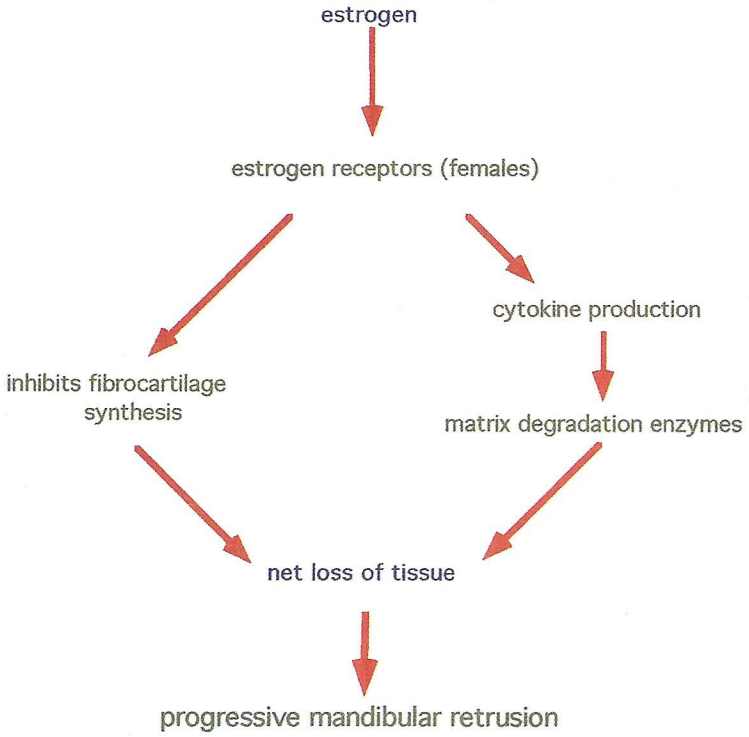

Fig. 2. Cascade of TMJ remodeling events secondary to effects of estrogen and estrogen receptors is illustrated.

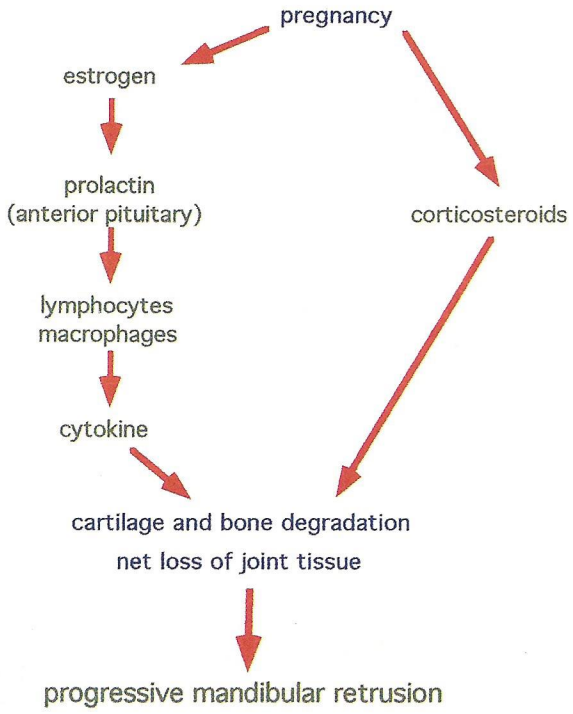

Fig. 3. Cascade of events causing TMJ remodeling secondary to pregnancy is illustrated.

and bone degradation in animal models of inflammatory arthritides (Fig. 3).[41] Prolactin is a potent stimulator of immune functions and enhances cytokine production by lymphocytes and macrophages.[42] Interestingly, prolactin secretion from the anterior pituitary is controlled by estrogen.[43] Bromocriptine, a dopamine receptor agonist, prevents prolactin secretion. Bromocriptine suppresses inflammation in animals with experimentally induced arthritis and the administration of prolactin reverses this effect (i.e., increases inflammation in affected joints).[41,44] Recently, bromocriptine has been used to successfully treat psoriatic arthritis in women.[45] It is conceivable that prolactin may mediate some effects which have previously been attributed to estrogen (Fig. 3). It is likely that prolactin contributes to the accelerated condylysis that has been observed in some pregnant females.

In summary, it appears that female predisposition to dysfunctional remodeling of the temporomandibular joint may be attributed, in part, to the modulation of the biologic responses of articular tissues to functional loading by sex hormones. Other factors that contribute to the sex-based predilection to dysfunctional remodeling of the temporomandibular joint may be identified in the future.

Table II. Four tissue level mechanisms of morphologic change

I. Mechanisms of mechanical stress
A. Physical disruption of cells
B. Impaired cellular vital functions
C. Impeded regional blood flow to cells
D. Neurogenic irritants
1. Neurogenic inflammation
2. Increased sympathetic tone

Corticosteroids

Corticosteroids have been reported as causing joint resorption.[19,20,33,46,47] Furstman[46] reported narrowing of condylar cartilage, osteosclerotic trabeculae, and inhibition of normal calcification when rats were subjected to increased exogenous hydrocortisone. Pellici[47] reported three cases of osteonecrosis involving the femoral head during pregnancy. He associated the osteonecrosis with the effects of increased levels of endogenous corticosteroids associated with pregnancy (Fig. 3). The Pellici findings might explain several unreported cases of TMJ condylysis which have occurred during and shortly after pregnancy. Ogden[23] described complete mandibular condyle resorption occurring after administration of corticosteroids for treatment of rheumatoid arthritis. It is conceivable that changes in corticosteroid levels may, in some individuals, initiate mandibular condylar resorption and attendant progressive Class II.

Mechanical stress factors

Mechanical stress provokes molecular, soft tissue, and osseous adaptive remodeling responses in the normal temporomandibular joint. Stretch and compression of the temporomandibular joint are both forms of mechanical stress (Table I).

What molecular events occur with mechanical stress of the temporomandibular joint?

All forms of mechanical TMJ stress can elicit several different molecular events that yield a decreased tissue volume of the articular surfaces. Tissue changes, if significant, may alter the occlusion.

Change at the molecular level is marked by early, characteristic, and simultaneous events. Excessive stresses may initiate four biologic or molecular level responses (Table II): (1) physical disruption of molecules, (2) impair cellular functions, (3) impede regional blood flow resulting in transient ischemia, and (4) evoke the release of inflammatory peptides from stretched or compressed nerve terminals. All these changes can lead to a net loss

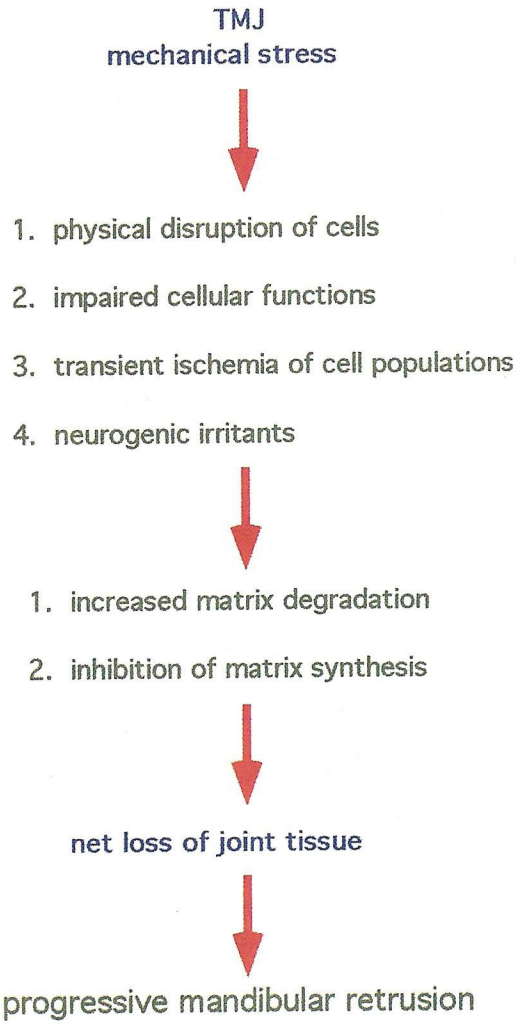

Fig. 4. Sequelae of temporomandibular mechanical stress (compression or stretch) are listed.

of tissue by increasing degradation processes (catabolic) and inhibiting synthetic processes (anabolic) in affected articular tissues (Fig. 4). Thus dysfunctional remodeling may result from excessive mechanical stress that effectively uncouples destructive and synthetic processes in affected articular tissues. Under pathologic conditions, the destructive processes prevail and there is a net loss of articular tissue significant enough to change the occlusal pattern. Healthy temporomandibular joints maintain homeostasis when subjected to normal and episodal excessive stress (such as gum chewing and dental appointments). When compromised (biochemically and biomechanically), the temporomandibular joint may break down when subjected to normal or abnormally elevated mechanical stimulations.

1. *Physical disruption of molecules:* Excessive

mechanical stress can lead to direct physical disruption of molecules in affected tissues leading to cell death and tissue volume decreases. Mechanical disruption of specific molecules in a tissue can also lead to the production of free radicals.[48] Free radicals can, in turn, damage adjacent structures, increasing collateral damage. A direct trauma to the temporomandibular joint may produce both direct and free radical damage to joint molecules and tissues. In the case of normal and abnormal (parafunction) function, the effects of direct and free radical damage are unknown. It is known that the temporomandibular joint is loaded with normal and abnormal function.[49-51] However, it is not clear if functional loading forces generated within the temporomandibular joint are of sufficient magnitude to evoke direct physical damage to molecules in articular tissues.

2. *Impaired cellular functions:* Excessive or prolonged mechanical loading of articular tissues of the temporomandibular joint may also adversely affect nutrient supply to local cell populations. In addition, these forces may perturb certain vital cellular functions as described in recent studies.[52] Thus the synthetic capacity of affected cell populations may be adversely affected by excessive or prolonged mechanical loading. Under these conditions, the balance between catabolic (i.e., destructive) and anabolic (i.e., constructive) biologic processes may be disturbed, leading to a net loss of articular tissue.

3. *Impeded regional blood flow:* Impeded regional blood flow may lead to ischemic reperfusion injury and temporomandibular joint resorption. There is evidence that free radicals, reactive molecules that can destroy tissue and alter cell functions, are produced in excessively loaded joints.[53-55] These *loading* pressures exceed capillary perfusion pressure and can impair blood flow to intracapsular tissues leading to ischemia. Cell populations in ischemic tissues adjust their metabolic pathways to accommodate lower oxygen tensions.[53,56] When blood flow is reestablished in the joint (i.e., reduced intracapsular pressure following relaxation of jaw muscles), oxygen tension rapidly rises in affected tissues. Cells with altered metabolic pathways still functioning generate free radicals under this condition. The free radicals (hydrogen peroxide [H_2O_2], superoxide anion [$O_2^{\bullet}$], hydroxyl anion [$OH^{\bullet}$] may then destroy local tissues. This phenomenon is known as ischemia-reperfusion injury.[53,56] The injury is dependent on both ischemia (which alters the metabolic pathways of affected cell populations) and reperfusion (which supplies oxygen to these cell populations). The oxygen is converted to free radicals as a result of the altered metabolism of previously ischemic cells.

4. *Release of inflammatory peptides:* Neurogenic inflammation and increased sympathetic tone may contribute to net tissue loss following biomechanical stresses to the temporomandibular joint.

Recently, neurogenic inflammation has been cited as possibly mediating condylar morphologic change.[57] Traction or compression of peripheral nerve terminals in the joint may evoke a release of neuropeptides (substance P, cGRP) into surrounding tissues. These neuropeptides can produce inflammation in these tissues, presumably by stimulating the synthesis and activation of inflammatory cytokines.[58] These processes subsequently affect remodeling of the temporomandibular joint. Interestingly, inflammatory cytokines can increase the synthesis of these neuropeptides in a positive feedback mechanism.[59-63] Therefore the inflammatory process produced by the stimulation of peripheral nerve terminals in the TMJ can lead to a self-perpetuating cycle. Under conditions which further enhance cytokine synthesis (i.e., estrogen, free radical generation), this process could be dramatically intensified, leading to an exaggerated degenerative response (condylysis) (Fig. 5).

Compression of the mandibular condyle stimulates direct resorptive remodeling at the site of injury because of local tissue disruption and impaired cellular functions. *In addition, however, the entire mandibular condyle can undergo diminutive change resulting from synovial fluid distribution of free radicals, and inflammatory peptides. Circulation of these by-products of mechanical stress may be essential to condylysis.*

Changes in sympathetic tone may influence bone and cartilage metabolism in the joint by other mechanisms as well.[64] Primary sensory afferent neurons may become responsive to catecholamines (i.e., epinephrine and norepinepherine) under conditions of chronic inflammation or previous trauma.[65-67] Release of these catecholamines from sympathetic nerve terminals in the joint could stimulate these primary sensory efferent neurons evoking the release of inflammatory neuropeptides into surrounding tissues of the joint. Thus heightened sympathetic tone can exacerbate neurogenic inflammation. Conditions that enhance sympathetic tone (i.e., psychological stress, nicotine ingestion, hypoglycemia) could theoretically contribute to degenerative changes in the TMJ by this mechanism (Fig. 6).[58,68,69]

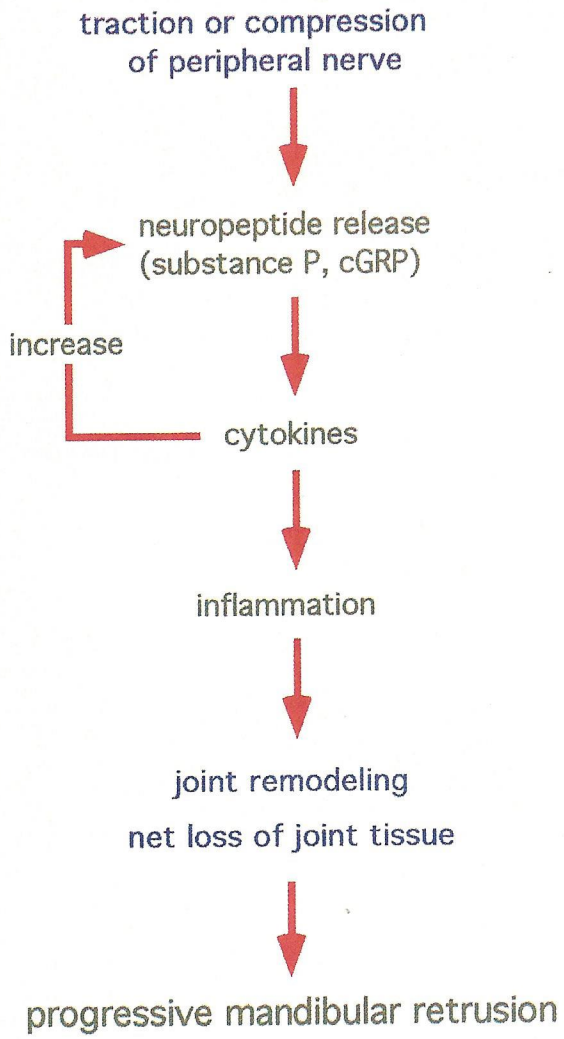

Fig. 5. Cascade of events causing TMJ remodeling initiated by peripheral nerve compression or traction is depicted.

Soft tissues of the temporomandibular joint typically undergo a rapid physical transformation when subjected to chronic or excessive mechanical forces. In separate studies Isberg[70] and Furstman[71] have depicted the soft tissue alterations associated with compressive events. Isberg observed a flattening of the posterior band of the articular disk with changes in its collagen fiber orientation when the mandibular condyle was displaced posteriorly for a period of 5 weeks. Furstman created unstable occlusion in rats by adding vertical occlusal interferences unilaterally. This manipulation led to an increased deposition of fibrous connective tissue and osteoid matrix on the articular surface of the glenoid fossa. In addition, an increased thickness and disorientation of collagen fibers of the articular disk was observed.

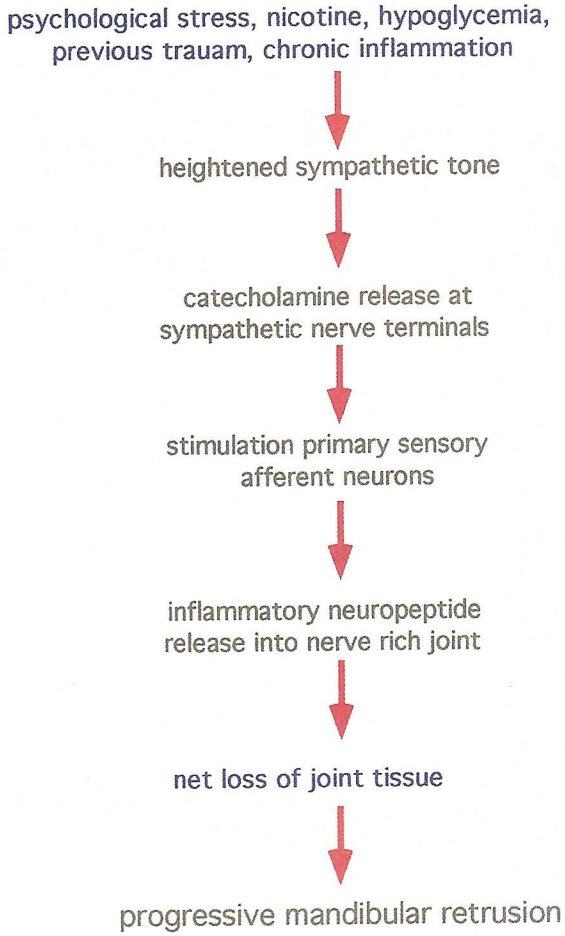

Fig. 6. Multiple factors affect sympathetic tone, thus creating TMJ remodeling.

Ultimately, joint soft tissue changes may contribute to *osseous remodeling.* Multiple studies have assessed the osseous changes that have been associated with condylar compression.[1,8,29,72-78] These studies have shown consistent osseous resorption of the postglenoid spine and posterior condylar surface when the condyle is posteriorized and compressed in the glenoid fossa. Similarly, Arnett and Tamborello[1] have demonstrated morphologic changes of the mandibular condyle associated with posteriorization and medial or lateral condylar torquing during orthognathic surgery.

References appear at the end of Part II.

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Progressive mandibular retrusion—idiopathic condylar resorption. Part II

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How sensitive is the TMJ to changes in mechanical loads?

The structure of the TMJ appears to be very sensitive to functional loading. Mongini demonstrated tomographically condylar changes after occlusal equilibration.[79] Peltola found a greater variation in condylar shape among orthodontically treated patients compared with controls.[80] These morphologic changes presumably represent normal functional remodeling stimulated by changes in mechanical stresses imposed on the temporomandibular joint after occlusal equilibration or orthodontic therapy.

The long-held belief that the TMJ is an immutable object should be abandoned in light of these findings. It appears that any factor that contributes to a change in the biomechanics of the masticatory system can provoke a change in the structure of the TMJ. Conceivably, changes in occlusion (i.e., lost teeth, orthognathic/orthodontic manipulations), excessive parafunctional habits, and articular disk-condyle malrelationships could contribute to remodeling of articular structures of the temporomandibular joint. The tissue response to these potential stressors may be variable and largely dependent on host factors. Therefore one person may exhibit signs and symptoms of aggressive dysfunctional remodeling (i.e., condylysis), while another person, given a similar physical insult to the joint, may be capable of adapting to the changing mechanical stress with functional remodeling.

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What are the mechanical factors capable of initiating change in TMJ structure?

Mechanical factors causing changes in condylar structure (Table I):

1. Occlusal therapy
2. Internal derangement
3. Parafunction
4. Macrotrauma
5. Unstable occlusion

Mechanical factors may occur in isolation or may be interrelated, interdependent, and coexistent. When two or more biomechanical factors coexist, morphologic change is more likely to occur. In addition, when the host adaptive capacity is limited, morphologic changes are accentuated when mechanical factors are active.

The effects of mechanical compression or stretch are influenced by their magnitude and duration and the host adaptive capacity. Temporomandibular joint compression is the most common source of joint structure change.

1. TMJ remodeling in response to the correction of occlusal and/or skeletal malrelationships. Occlusal therapy can produce joint compression by changing the condylar position during or after treatment. Compression results in some degree of resorption of the condyle. The resorption is either functional, maintaining the occlusion or dysfunctional, with attendant change in occlusion. Coexisting remodeling stimuli (internal derangement, parafunction, macrotrauma, unstable occlusion, hormones, systemic diseases or neurogenic conditions) may accentuate condylar remodeling, producing dysfunction. The greater the magnitude and duration of mechanical stress the more likely and larger the

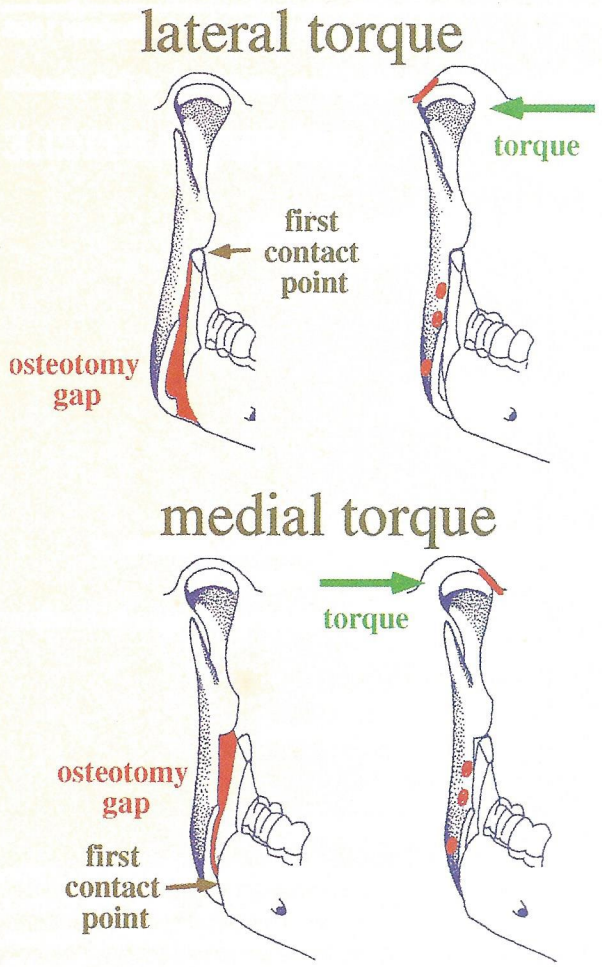

Fig. 7. Medial and lateral torque of mandibular condyle associated with sagittal osteotomy are depicted. When *osteotomy gap* is closed by surgeon, condylar fragment rotates on contact point between condylar fragment and tooth bearing fragment. Resulting medial or lateral condylar compression creates possibility for late (9 to 18 months) condylar resorption and B point relapse.

TMJ structure alterations. Because of the nature of orthognathic surgery, gross positional changes of the mandibular condyle can occur, producing gross condylar morphologic changes. Orthodontic and prosthetic care may also produce condyle position changes but, generally not to the same extent as orthognathic surgery. Orthodontic mechanics used to correct skeletal malocclusions (i.e., Class III and Class II elastics, cross arch elastics) may be capable of greater condylar position changes and associated compression with resorption.

Arnett and Tamborello[1] studied tomographically 61 orthognathic surgery patients to determine the effects of surgically induced condylar position change on the structure of the mandibular condyle. They found that intraoperative condylar torquing or posteriorization leads to condylar resorption and late mandibular relapse (Figs. 7 and 8). Ellis[78] studied rigid and wire fixated sagittal osteotomies of the mandible in monkeys and observed resorption of the postglenoid spine and posterior aspect of the condyle associated with posteriorization of the condyle when rigid fixation was used. Ellis believed that with rigid fixation of the osteotomy sites the condyles was displaced in a posterior direction from soft tissue (i.e., muscle, skin) resistance to the advanced mandible. Because the mandibular condyles provide posterior support for the mandible, these reactive forces were presumably transferred to articulating structures within the joint leading to the observed remodeling. In the second group of animals, Ellis used wire fixation and intermaxillary fixation. In this group, the condyles were typically found in an anterior position in the glenoid fossa. These condyles did not undergo detected morphologic changes. Unlike the rigid fixation group, the posterior displacement forces were apparently absorbed by the intermaxillary fixation rather than the structures of the temporomandibular joint. Ellis concluded that condylar resorption after orthognathic surgery is probably related to compressive phenomena.

2. Articular disk-condyle malrelationships. The relationship between internal derangement (ID) and condylar remodeling in adults is not understood. Internal derangement is found with[3,81-84] and without[82,83,85-87] remodeling. There does not seem to be a clear cause-and-effect relationship between ID and shape changes of the condyle and fossa. For example, deBont[88] has shown that morphologic alterations of the mandibular condyle (osteoarthrosis) may occur in human beings showing a normal articular disk-condyle relationship. In addition, 20% of the human cadavers studied with articular disk-condyle malrelationships did not have condylar changes (osteoarthrosis). On the basis of these observations, deBont concluded that osteoarthrosis (i.e., dysfunctional remodeling) may lead to internal derangement of the temporomandibular joint. In this series, articular disk displacement may be a sign of dysfunctional remodeling and not its cause.

Internal derangement is suspected of causing condylar resorption and, thus, negatively affecting mandibular growth. It is possible that when ID exists genetically coded anterior growth is diminished. However, patients with mandibular defi-

ciency commonly have normal disk position and patients with normal mandibular growth commonly have ID. Therefore no cause and effect can be stated for anterior disk position and mandibular growth deficiency. There is no current evidence that the prevalance of condylar resorption and diminished mandibular growth in children exceeds the rare incidence in adults.

When ID and morphologic alteration occur concurrently the internal derangement may produce condylar morphologic change by compression. Katzberg studied the effects of articular disk displacements on condylar mobility.[89] The mandibular condyle was hypermobile when anterior disk displacement with reduction (ADR) was present. In contrast, condylar translation was markedly decreased with anterior disk displacement without reduction (ADNR). Under this condition, impaired translation of the mandibular condyle may be attributed to a physical obstruction of condylar movement by the displaced articular disk. The obstruction produces compressive forces that are imparted to contacting structures of the joint. Therefore ID may produce condylar remodeling by contributing to compressive loading of articular tissues. This notion is supported by multiple studies[3,81-84] that have shown that condylar remodeling is more likely to be associated with ADNR than ADR (Fig. 9).

Alternatively, deBont and colleagues[88] have proposed that articular disk displacements result from, and are not the cause of, degenerative changes affecting the TMJ. These investigators speculate that decreased joint lubrication leading to changes in the structure of the articular surfaces (irregularities) may increase frictional forces between the mandibular condyle, temporal bone, and articular disk, thus pulling the disk forward with translatory movements. The net result of these adverse changes in the biomechanics of the joint leads to articular disk displacement. The decrease in lubrication and resulting surface irregularity may be the result of molecular level changes. Physical disruption of cells,[48] impaired cellular function,[52] impeded regional blood flow,[53-55] and release of inflammatory peptides[57-63] may lead to decreased lubrication.

Recently, internal derangement (i.e., anterior disk displacement) of the temporomandibular joint has been theorized to produce avascular necrosis (AVN) of the mandibular condyle. This has led to the advocation of disk plication to "normalize" disk position in growing children and mature adults in an attempt to avoid possible AVN.[90-93] The theory is based on the postulation that an anteriorly displaced disk impinges on a terminal arterial branch supplying the mandibular condyle. Advocates of this theory argue that the principal blood supply to the mandibular condyle enters anteriorly near the attachment of the lateral pterygoid muscle.[90-94] However, Boyer studied the blood supply to the TMJ and found an extensive vascular plexus that receives blood vessels most prominently from the posterior aspect of the mandibular condyle. Small feeder vessels also enter the mandibular condyle from the medial, lateral, and anterior aspects of the condyle.[95] This extensive blood supply refutes the claim of a vital dependency on vessels located on the anterior aspect of the mandibular condyle. In addition, there is no evidence to support the notion that an anteriorly displaced articular disk can occlude the anterior blood supply to the TMJ. Therefore it is unlikely that an anteriorly displaced articular disk will produce AVN by critically impairing the blood supply to the mandibular condyle. Recent evidence indicates that AVN of the mandibular condyle is a rare event. The contribution of this phenomenon to condylysis or dysfunctional remodeling of the temporomandibular joint is currently unknown.

Chuong and Piper[92,93] recently focused on the Starling resistor theory of mandibular condyle AVN

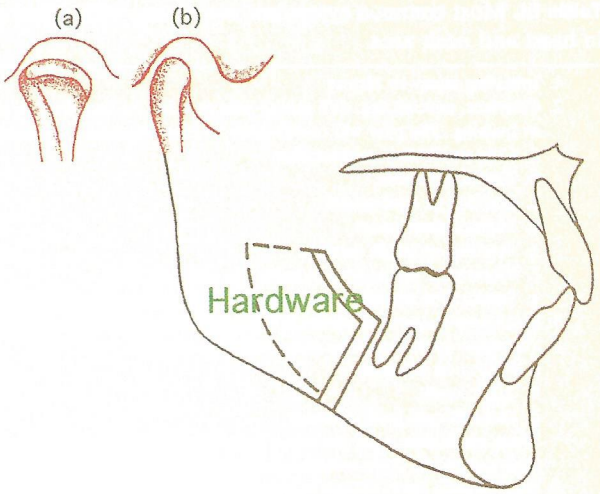

Fig. 8. Sagittal osteotomy advancement of mandible is depicted. Condyle has been placed farther lateral *(a)* and posterior *(b)* than preoperative position and held in that position by hardware (screws, plates or wires) and interdigitation of teeth (CO). Mandibular length is actually excessive. When teeth interdigitate, condyle is driven posterior and lateral, compressing joint tissues. This compression can cause late B point relapse. This same process occurs after LeFort surgery when condyles are positioned posterior to preoperative position.

Table III. Most common symptoms associated with reflex symptomatic dystrophy (RSD) listed in descending frequency in head and neck area

1. Initiating injury-by history
2. Constant, unremitting, dull aching pain that can be described by variable heightened episodes of spontaneous shooting or stabbing, tingling, or burning. The pain will traditionally be exacerbated by, not necessarily all the following:
   a. Cold-particulary cold weather
   b. Emotional stress
   c. Startling or loud noises
   d. High-pitched noises
   e. Startling or bright sunlight
   f. Vibrational noises or movement (i.e., subway ride)
   g. Intolerance of collars, necklaces, clothing or hair next to involved area, producing an unpleasant sensation.
3. Numbness in the distribution of the auriculotemporal nerve and C2-C3.
4. Allodynia (pain caused by nonpainful stimuli) to:
   a. Cold most common
   b. Light touch
   c. Hot and cold least common
   These modalities need not cause pain, but provoke an unpleasant sensation that the patient does not tolerate well. The allodynia to cold is most commonly down-modulated by warm, moist heat application almost immediately.
5. Hyperalgesia (pain caused by painful stimuli). This category insinuated a reduced pain threshold and an increased response that is disproportionate to the stimuli.
6. A tender carotid or facial artery reflex that radiates within the vascular distribution upon palpation.
7. Vasomotor response:
   a. Coldness in the area affected-pallor
   b. Discrete patchy
   c. Feeling of being febrile
8. Sleep loss with or without night sweats
9. Spontaneous lacrimation of the eye(s)
10. Feeling of sinus fullness, sinusitis or rhinitis
11. Light-headedness or dizziness
12. Trophic changes demonstrated by:
    a. Muscular atrophy
    b. Trophedema
    c. Gooseflesh
13. Sudomotor signs
    a. Excessive sweating
    b. Very dry skin
14. Hair loss

Symptoms 1 through 6 may present transiently and cannot be provoked at the time of any one appointment.

(osteonecrosis). This process proposes that unknown factors increase the intramedullary condylar pressure causing decreased arterial inflow. The decreased arterial inflow then results in osteonecrosis (AVN) of the mandibular condyle. To reduce condylar intramedullary pressure and thus increase blood flow to the condyle Chuong advocates corticotomies of the condyle. To date, there is no evidence that increased intramedullary pressure is a factor associated with AVN or osteonecrosis of the mandibular condyle.

3. Parafunction. Parafunction may produce compression that is capable of initiating condylar resorption[12,96-100] or enhancing resorption caused by other factors that initiate this process. It is likely that these forces contribute to condylar resorption by at least two mechanisms. First, direct biomechanical stress can disrupt the integrity of articular tissue and inhibit important synthetic functions of affected cell populations. Excessive biomechanical stress can physically damage molecules in affected tissues. Second, it is possible that tissue damage resulting from excessive loading of the TMJ is secondary to an ischemia-reperfusion injury (Fig. 10).[53,56] Intracapsular pressures have been recorded from symptomatic temporomandibular joints that have approached 200 mmHg with clenching.* These intracapsular pressures exceed the estimated capillary perfusion pressure and can therefore impair blood flow to intracapsular tissues leading to ischemia. Cell populations in ischemic tissues adjust their metabolic pathways to accommodate lower oxygen tensions. When blood flow is reestablished in the joint (i.e., reduced intracapsular pressure after relaxation of jaw muscles), oxygen tension rapidly rises in affected tissues. Cells with altered metabolic pathways still functioning generate free radicals under this condition (Fig. 10). There is evidence that free radicals are produced in excessively loaded joints.[53-55] Parafunction may increase intracapsular

*Personal communication with M.F. Dolwick and D. Nitzan in 1994.

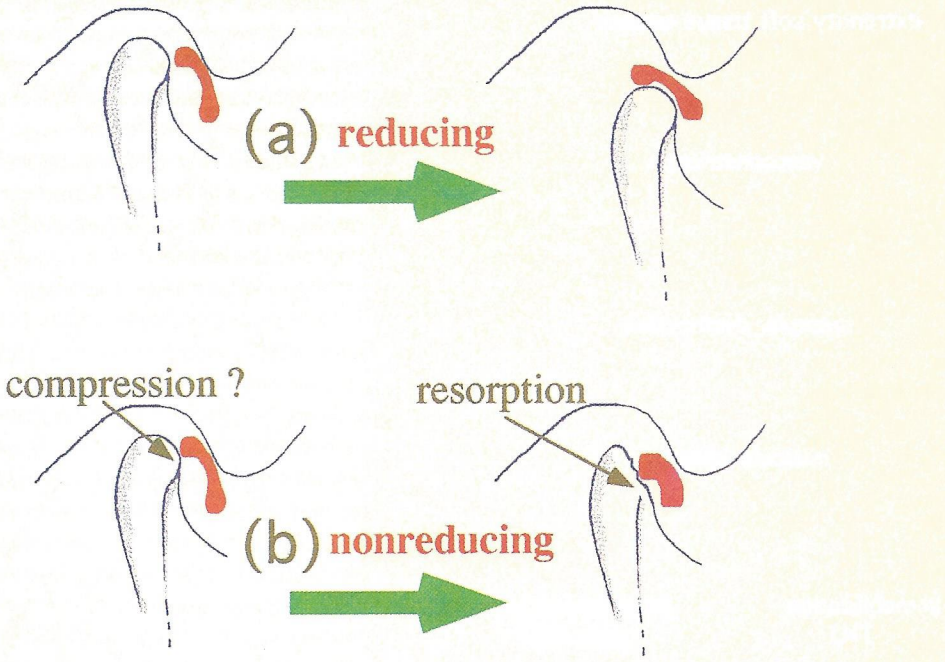

Fig. 9. Identical anterior disk dislocations are depicted *(a)* and *(b)*. In *(a)*, disk reduces into normal position as condyle passes anteriorly. In *(b)*, disk does not allow condyle to pass anteriorly possibly creating anterior condylar compression and osseous resorption.

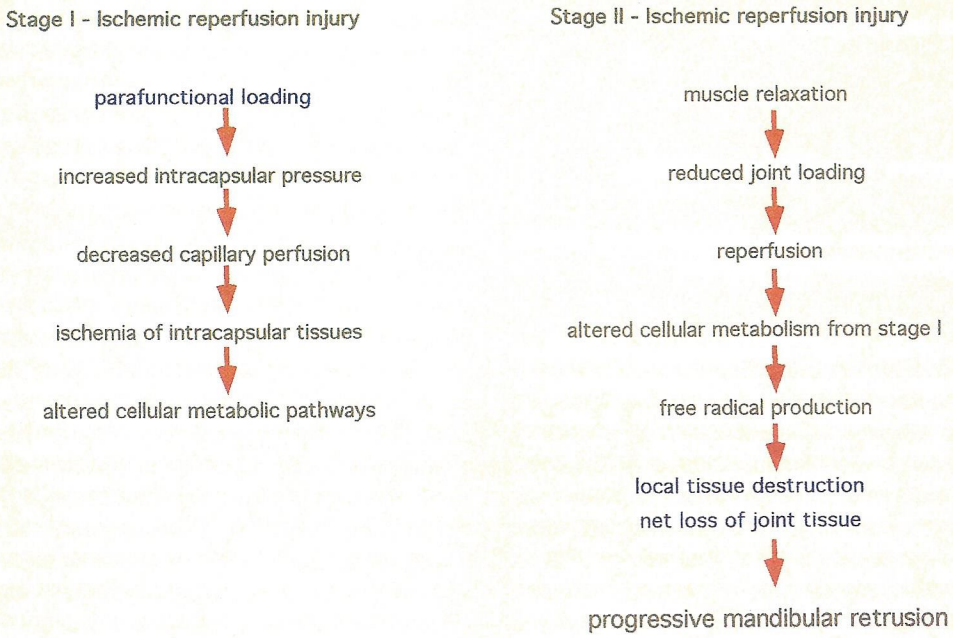

Fig. 10. Cascade of events of stage I and II ischemic reperfusion are shown.

pressures, inhibit capillary perfusion and thus create an ischemic reperfusion injury with attendant loss of temporomandibular tissue volume leading to mandibular retrusion.

4. Macrotrauma. Macrotrauma may also promote condylar resorption.[1,8,13,29] Macrotrauma consists of one episode (compression or stretch) of large magnitude force that is transmitted to articu-

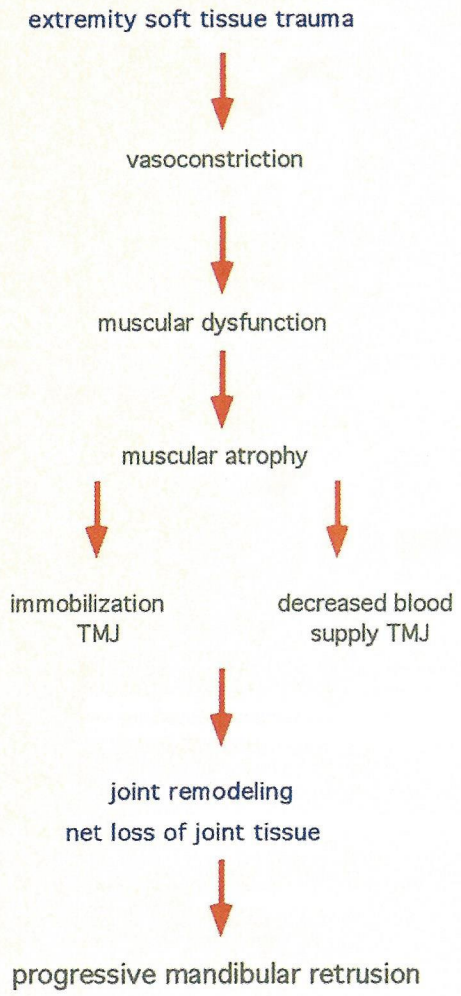

Fig. 11. Cascade of events causing TMJ remodeling initiated by reflex sympathetic dystrophy is depicted.

lar structures of the TMJ. The force is generally of a sufficient magnitude that it is acutely injurious to affected articular tissues. The occlusion is not altered at the time of the macrotrauma. Temporomandibular joint alterations occur over time after the macrotrauma, leading to progressive mandibular retrusion. Resorption of this nature has been associated with removal of third molars,[8] blows to the lower jaw without fracture,[8,13] orthognathic surgery,[1,29,90] joint surgery,[90] whiplash,[90] and protrusive splint therapy.[90]

The mechanism of delayed condylar resorption secondary to macrotrauma is not understood. Several mechanisms may be involved. Condylar resorption can be the result of physical injury or impaired cellular functions. Reflex sympathetic dystrophy (RSD) may be associated with delayed macrotrauma resorption. Reflex sympathetic dystrophy has been recognized as a sequelae of extremity macrotrauma.[101-103] After extremity soft tissue trauma numerous symptoms occur but, most importantly,[1] immobilization of joints, vasoconstriction, muscular dysfunction, muscular atrophy, and osteoporotic bone changes. Recently, RSD has been identified as occurring facially and causing prolonged immobilization and pain of the affected TMJ. It is possible that some of the changes in joint function and structure previously attributed to myofascial pain dysfunction or arthrosis could be by-products of RSD (Table III). The blood supply to the TMJ is derived from multiple feeder vessels originating in the muscles that surround the joint.[95] Atrophy of the muscles surrounding the TMJ secondary to RSD could lead to a diminished blood supply to the joint structures and create an avascular necrotic sequelae. In addition, as reported by Glineburg and Lydiatt,[104,105] TMJ immobilization leads to thinning of the articular cartilage, layer disorganization of the articular fibrocartilage, adhesions, and generalized changes consistent with degenerative joint disease. These changes could conceivably occur secondary to the joint immobilization that occurs with RSD (Fig. 11). These structural changes, as reported by Glineburg and Lydiatt, were proportionate to the length of immobilization, which in the case of RSD can be prolonged. Also, Lydiatt reported that remobilization was followed by an increase in disruptive changes.[105] This could represent a component of ischemia reperfusion injury.

5. Unstable occlusion. Unstable occlusion may contribute to condylar resorption. Two forms of occlusion exist independent of Angle classification: (1) stable and (2) unstable. A stable occlusion does not deflect the condyle position during interdigitation of the dentition (regardless of Angle classification) (Fig. 12). An unstable occlusion produces compressive deflection of the condyle during interdigitation of the teeth (regardless of Angle classification) (Fig. 13). Furstman has described disorientation of collagen fibers in the articular disk and severe osteosclerotic changes of the mandibular condyle that have been associated with the loss of occlusal stability.[71] Gazit and Ehrlich also observed structural changes of the temporomandibular joint that were associated with unstable occlusion including bone resorption and fibrocartilage calcification.[106-108] Posteriorization of the mandibular condyle secondary to occlusal changes (i.e., unstable occlusion) may lead to postglenoid spine and

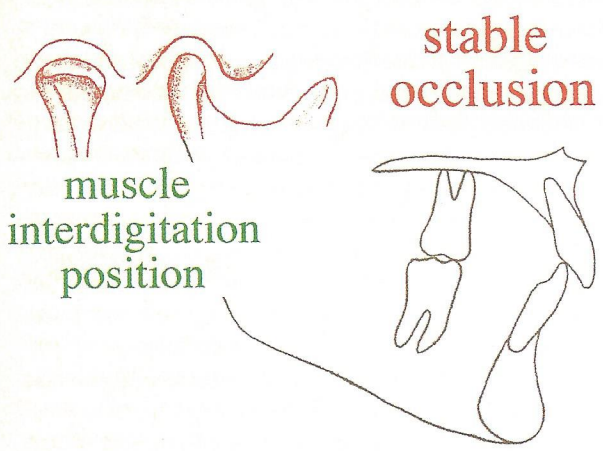

Fig. 12. Stable Class II occlusion is depicted. When teeth maximally interdigitate (CO) under muscular force condyle is not deflected and therefore compressed. Regardless of Angle classification, occlusion is stable if muscular interdigitation of teeth (CO) does not deflect condyle into compression creating condylar resorption. Compressive loading is least likely with Angle Class I occlusion.

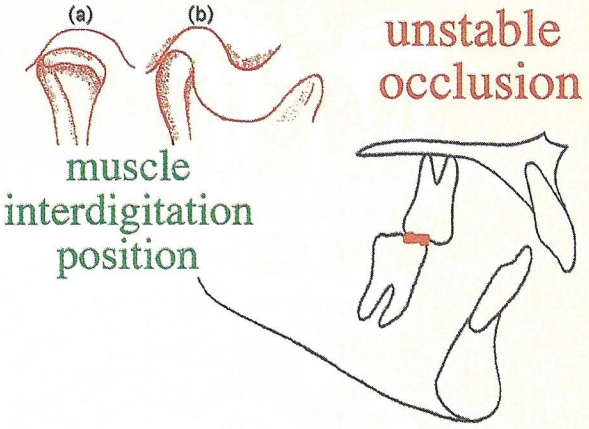

Fig. 13. Unstable occlusion is depicted. When maximal interdigitated position (CO) is produced with muscular force, condyle is deflected into compression. In this example, with molar interdigitation condyle is depicted as loading laterally *(a)* and posteriorly *(b)*. This type of occlusion may be followed by compressive resorption of condyle and resultant mandibular retrusion.

posterior condylar resorption.[1,8,29,70.72-74] In addition, Rasmussen found that, generally, temporomandibular joint arthropathy when followed over time ended with resolution of symptoms and signs. A small group of patients in the Rasmussen study did not undergo resolution of symptoms and signs—these were patients who were missing posterior teeth or posterior stops.[109] It may be that these patients are representative of the effects of unstable occlusion.

Class I occlusion with condylar compression is, by definition, an unstable occlusion (Fig. 14). Correction (orthodontics, orthognathic surgery, prosthetics) of occlusal discrepancies to a Class I dental relationship, if associated with joint compression, can lead to condylar resorption (Fig. 14). Arnett and Tamborello have observed condylar resorption when a Class I occlusion was produced with the mandibular condyles posteriorly displaced after orthognathic surgery.[1,8,29] Orthodontic and prosthodontic therapy can potentially change condylar position, though typically not to the same extent as that observed after orthognathic surgery. Therefore these nonsurgical procedures could contribute to significant condylar remodeling, especially in susceptible patients (i.e., those predisposed to more aggressive tissue response secondary to host factors).

Unstable occlusion can result from dysfunctional remodeling of the mandibular condyle. The

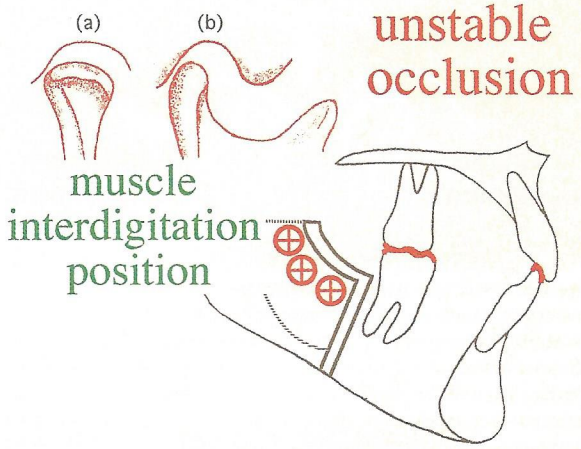

Fig. 14. Postsurgical Class I unstable occlusion is depicted. Mandible was reconstructed with excessive length and condylar torque. When maximal interdigitation (CO) occurs with muscle force condyle is depicted in this example as compressing laterally *(a)* and posteriorly *(b)*. These loaded areas can lead to late condylar resorption and B point relapse despite Angle Class I occlusion.

dysfunctional remodeling can be secondary to host or mechanical factors. When condylar support for the dentition is altered for any reason, muscular interdigitation of the teeth may compress the condyle within the glenoid fossa leading to additional condylar change. The unstable occlusion becomes cocontributor with the original resorption

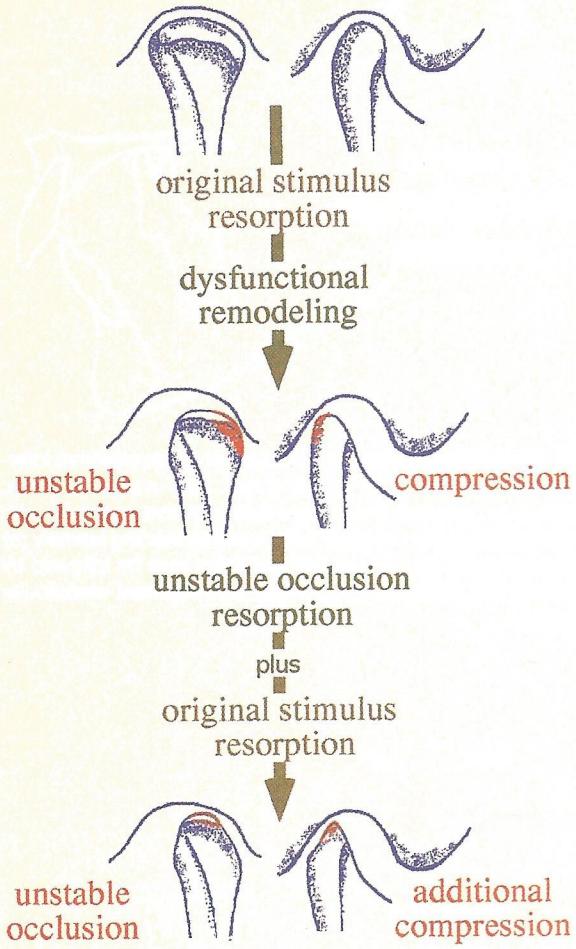

Fig. 15. Any combination of diminished adaptive capacity or biomechanical stress that causes dysfunctional remodeling leads to unstable occlusion. Once unstable occlusion is present, it may cause additional condylar compression with interdigitation (CO) of teeth leading to further resorption. Unstable occlusion is end result of all sources of dysfunctional remodeling.

catalyst in creating the resorptive changes within the temporomandibular joint (Fig. 15).

Treatment of idiopathic condylar resorption (ICR): Treatment for idiopathic condylar resorption is threefold: (1) control or eradication of the etiologic factors, (2) stabilization of the unstable occlusion and TMJ (healing), and (3) correction of the resultant occlusal deformity.

Identification of the cause may be difficult when *host adaptive capacity* is a factor. Systemic illnesses, which may contribute to resorptive changes, should be identified and treated when possible.

Patients in whom estrogen associated factors may be present should be recognized before dental treatment. Estrogen coupled condylar resorption should be suspected with second and third decade women, small decorticated condyles (tomograms), and a history of increasing mandibular retrusion. In this patient population, occlusal correction should be ideally deferred until the end of the third decade or until the joints are proven stable. When occlusal care is rendered, the condyles should not be loaded or resorption may be reinstigated.

Mechanical stresses are probably the most common source of condylar resorption. Occlusal therapy and possible attendant compression of the TMJ should be recognized as causing condylar resorption, especially in combination with decreased host adaptive capacity. If treatment compression is avoided the condyles will most often remain stable during treatment. Internal derangement is a potential source of condylar resorption. When ID is present, nonloading occlusal treatment is mandatory to avoid resorption. Occlusal treatments in the presence of ID should not be started until the joints are proven stable. Disk repositioning procedures have not been shown to be necessary to stabilize the temporomandibular joint. Parafunction should be controlled when it is suspected as an agent in condylar resorption. Night guards, medications, and biofeedback may be necessary when parafunction is causing joint resorption. After macrotrauma, the patient may need anti-inflammatory medications and possibly splint stabilization to control joint changes. Unstable occlusion that results from all sources of condylar resorption must be stabilized to allow the temporomandibular joint to heal. Without stabilization the condyle continues to modify and change the occlusion.

Arnett and Tamborello[8] have proposed a treatment protocol for patients who have undergone resorption with resulting unstable occlusion. This protocol consists of orthodontic preparation for surgery followed by anti-inflammatory medications, stabilizing splints, and precise orthognathic surgery that does not reload the joints. Without this protocol before orthognathic surgery, further condylar resorption routinely occurs in this patient group.[8]

CONCLUSIONS

The cause of condylar remodeling is clearly multifactorial. Remodeling is based on the interaction of two groups of factors: the *host adaptive capacity* and *mechanical stimulus(i).* Regardless of cause, functional remodeling is characterized by adaptation of the articular structures of the TMJ in response to mechanical stress. Functional remodeling preserves structural relationships,

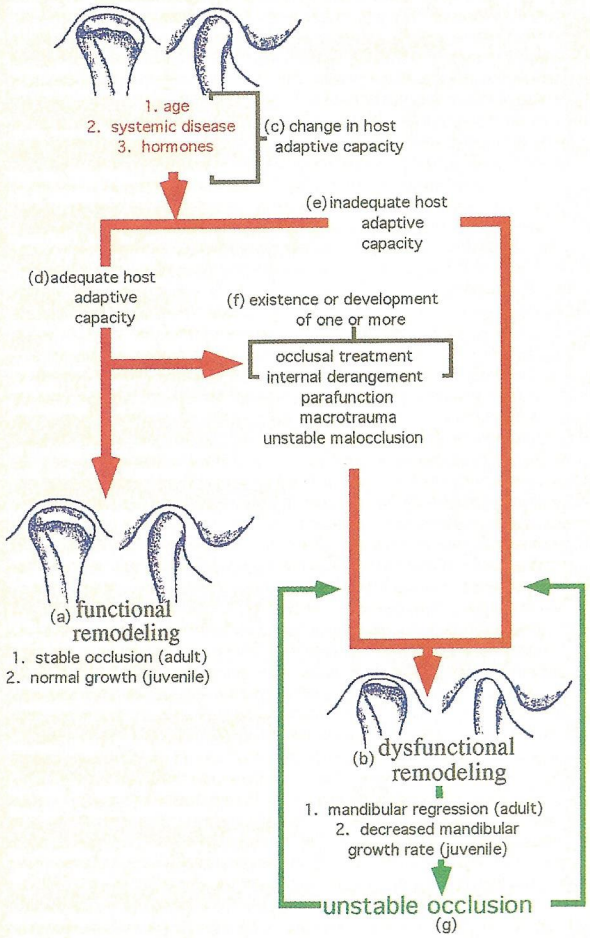

Fig. 16. Functional *(a)* and dysfunctional *(b)* remodeling initiated by changes in adaptive capacity *(c)* are depicted. If change in adaptive capacity is mild and adaptive capacity remains adequate *(d)*, functional remodeling will occur. Dysfunctional remodeling occurs with significantly decreased host adaptive capacity *(e)* or existence or development of compressive or stretch factors *(f)*. End result of all dysfunctional remodeling is unstable occlusion *(g)* that causes additional dysfunctional remodeling.

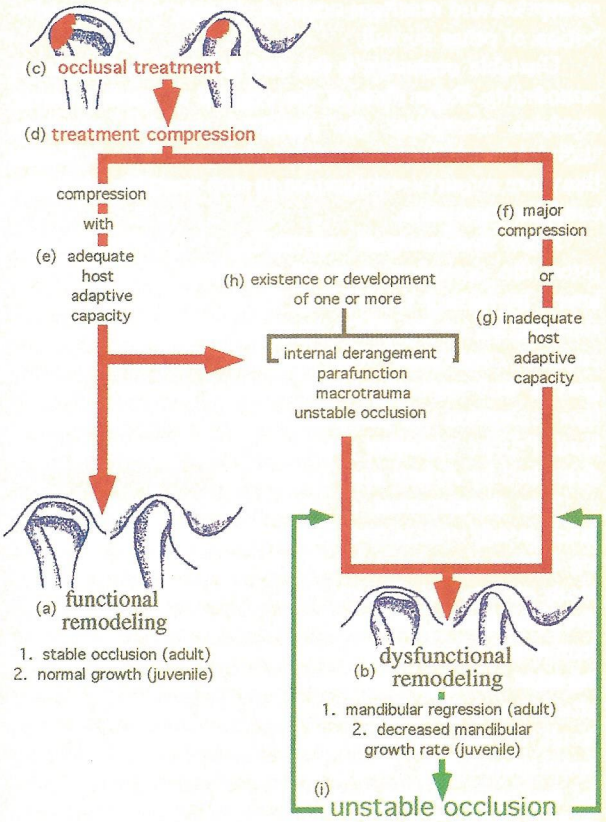

Fig. 17. Functional *(a)* and dysfunctional *(b)* remodeling initiated by occlusal treatment *(c)* are depicted. Functional remodeling occurs routinely with treatment compression *(d)* and adequate host adaptive capacity *(e)*. Dysfunctional remodeling occurs when major compression *(f)* occurs, inadequate host adaptive capacity *(g)* exists or other remodeling stimuli *(h)* exist or develop. Result of all dysfunctional remodeling is unstable occlusion *(i)* that compresses condyle resulting in additional remodeling.

allowing for a continuation of normal biomechanical function of the jaws. Functional remodeling is generally associated with a stable ramus height, stable occlusion (adult), and normal mandibular growth (juvenile). Regardless of cause, dysfunctional remodeling is typically distinguished from functional remodeling by an associated significant alteration in spatial relationships of the jaws. These changes may include a reduction in ramus height and a progressive Class II malocclusion. Condylysis is the extreme of condylar dysfunctional remodeling.

Host factors, such as age, hormonal status, and the presence of systemic disease, can contribute to dysfunctional remodeling of the temporomandibular joint, adversely affecting mandibular growth rate (juvenile) and jaw relationships (Fig. 16). The presence and extent of biomechanical forces impacting the temporomandibular joint alter the effects of the host adaptive capacity on remodeling.

When predisposing host factors are not present, occlusal treatments normally result in functional remodeling (Fig. 17).[79,80] However, dysfunctional remodeling resulting from low level mechanical stress (Fig. 17) (orthognathic surgery, orthodontics, prosthetics) may occur subsequent to an inadequate host adaptive capacity, coincidental internal derangement of the joint, excessive parafunction, macrotrauma, or unstable occlusion (Fig. 17). Dysfunctional remodeling provoked by the treatment of dental skeletal deformities is, to some extent, dependent on the presence of these hidden factors. However, it seems likely that extensive treatment compression[1] is capable of initiating substantial condylar

resorption and resultant occlusal changes without contribution of other stimuli.

Unstable occlusion is the end point of dysfunctional remodeling. Regardless of the original stimulus creating condylar resorption, the resultant unstable occlusion stimulates additional condylar changes. The occlusion therefore requires stabilization to stop unstable occlusion condylar resorption.

After dysfunctional remodeling of the TMJ has altered the occlusion, occlusal correction is usually followed by further remodeling. To prevent this sequelae, stabilization of the TMJs must be accomplished before occlusal correction.[8] The surgical correction must be precise with noncompressive condylar placement or further resorption will occur secondary to the new condylar load. Precise condylar placement techniques have been described by Arnett and Tamborello.[1]

The development of dysfunctional remodeling remains poorly understood. Some of the causative contributing factors have been briefly discussed in this overview. Many attempts at classical descriptive criteria (degenerative joint disease, osteoarthrosis, osteoarthritis, AVN, osteonecrosis) have sought to place overlapping and confusing signs and symptoms into specific disease entities. This article has pointedly avoided that and classified the net result of any pathophysiology—dysfunctional or functional remodeling. Clearly, the key to understanding clinical and radiographic changes lies not within a restrictive central dogma, but the recognition that the degenerative event is probably the result of many predisposing or contributory factors, leading to the development of a common biologic event that presents with variable severity.

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