Risk Factors in the Initiation of Condylar Resorption

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Progressive condylar resorption is a process that involves the temporomandibular joint (TMJ) and the occlusion. During growth, condylar resorption may decrease the projection of the mandible and be unrecognized as the source of a Class II malocclusion. After growth completion, as the condyle resorbs, the occlusion becomes progressively Class II, with or without open bite. Broadly speaking, condylar resorption is initiated or maintained by a combination of systemic factors and any form of TMJ compression, including pressure resulting from dental treatment. (Semin Orthod 2013;19:81-88.)

Two distinct categories of temporomandibular joint (TMJ) remodeling can occur: functional remodeling and dysfunctional remodeling.[1-6] Functional remodeling of the TMJ is an ongoing process involving morphologic changes of the articular structures of the joint that are not associated with any significant alterations in the joints or the occlusion. Therefore, functional remodeling is characterized by TMJ morphologic change, stable ramus height, stable occlusion, and normal growth.

Remodeling of the TMJ is dysfunctional if it adversely affects the joints and the 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 negative type of remodeling is often described as progressive/idiopathic condylar resorption.

The effects of dysfunctional remodeling (condylar 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.[1-6]

The effects of dysfunctional remodeling (condylar resorption) on the growing mandible are also clear. If it occurs during growth, condylar resorption decreases the condylar size and the mandibular growth rate, resulting in mandibular retrusion. The condylar growth is diminished by negative remodeling, and the mandible increasingly becomes Class II, with lack of full forward growth.[1-6] Therefore, factors that diminish the functional condylar remodeling capacity or increase the compression on the TMJ may adversely alter condylar size and the occlusion (Fig 1).

Host Remodeling Capacity

Age, gender, systemic illnesses, and hormonal factors may influence the host adaptive capacity of the TMJ.[1-6]

1. Age and gender—Condylar resorption is more prevalent in younger age-groups, especially among female individuals, and the rea-

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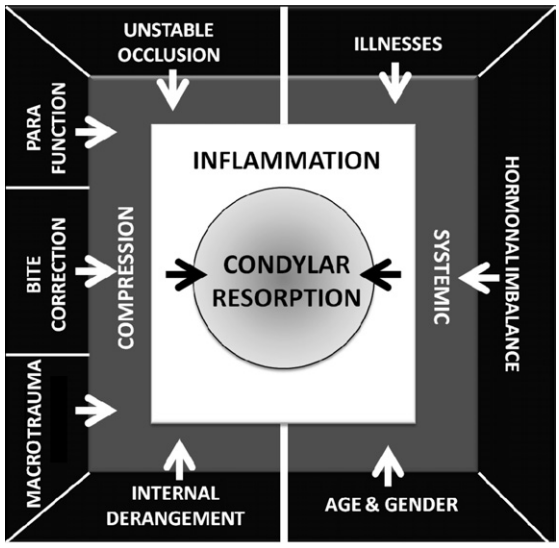

Figure 1. Condylar resorption (center) is a result of TMJ inflammation (white). Inflammation is the sum of joint compression (gray left) and systemic overlay (gray right). Systemic factors (black right), including illnesses, hormonal imbalance, age, and gender, may upmodulate systemic inflammation, which, in turn, upmodulates resorption. Additionally, compression (gray left), when present, upmodulates compressive inflammation, which, in turn, increases condylar resorption. Compression (gray left) is the sum of active factors (black left), which may include unstable occlusion, parafunction, bite correction, condylar displacement, internal derangement, and isolated macrotrauma. To decrease the risk of bite treatment causing condylar resorption, techniques should be used that avoid condylar displacement. Most importantly, in the course of performing orthognathic surgery, surgical techniques are used to decrease treatment compression. Medications inhibit or control inflammation and condylar resorption from either compressive or systemic origins.

sons for these differences in prevalence are unclear.

2. Systemic illnesses—These illnesses may include autoimmune disorders, endocrine disorders, nutritional disorders, and metabolic diseases. In particular, autoimmune diseases have been associated with condylar resorption in numerous publications, as summarized by Arnett et al.[1-6]
3. Hormones—Hormonal factors may have a marked influence on regressive remodeling of the mandibular condyle. Among them are the following:
   a. *Low estrogen*: Arnett and Tamborello have reported observations about a group of female idiopathic condylar resorption patients.[1,6] Examination of this group revealed progressive bilateral condylar resorption leading to progressive Class II malocclusions. Recently, Gunson et al.[6] reported 27 female idiopathic condylar resorption patients who all had low estrogen levels, indicating a possible cause (low estrogen) and effect (condylar resorption). These clinical experiences indicate that some females may be predisposed to dysfunctional remodeling of the TMJ in response to loading associated with occlusal treatment. This apparent female preponderance for dysfunctional remodeling of the TMJ suggests a potential role of sex hormones as modulators of this response (Fig 2).
   b. *Prolactin*: Prolactin, a hormone responsible for initiating postpartum milk letdown, can exacerbate cartilage and bone degradation in animal models of inflammatory arthritides. It is likely that prolactin contributes to the accelerated condylysis that has been observed in some pregnant women. It appears that female predisposition to dysfunctional remodeling of the TMJ may be attributed, in part, to the modulation of the biological responses of articular tissues to joint loading by sex hormones. Other factors that contribute to the sex-based predilection to dysfunctional remodeling of the TMJ may be identified in the future.
   c. *Corticosteroids*: Corticosteroids have been reported as causing joint resorption. It is conceivable that changes in corticosteroid levels may, in some individuals, initiate mandibular condylar resorption and attendant progressive Class II malocclusion.[2-5]

Mechanical Compression Factors

Temporomandibular joint loading produces compressive forces that provoke molecular and soft-tissue adaptive remodeling responses in the normal TMJ.[1-12] Ultimately, under unusual compressive loads, joint soft-tissue changes may contribute to functional or dysfunctional osseous remodeling. Multiple animal studies have as-

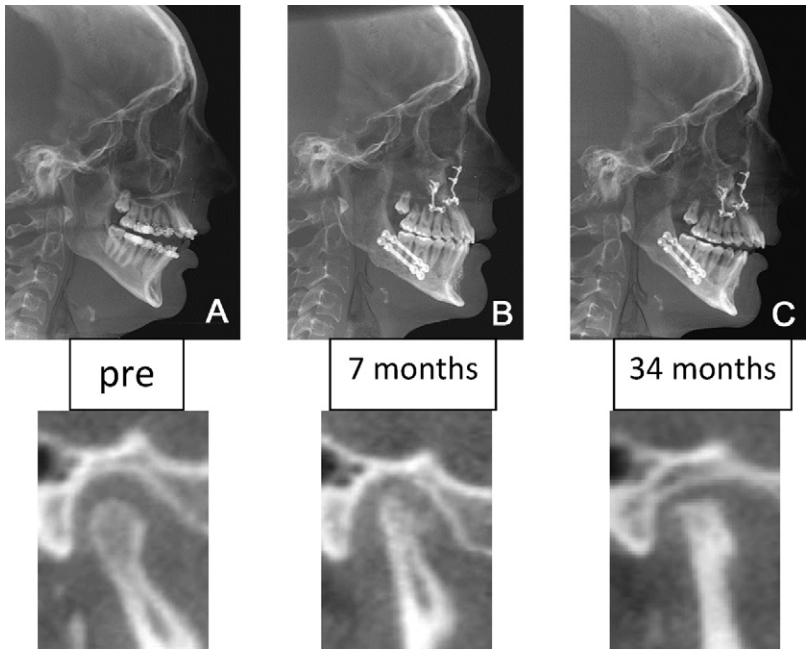

Figure 2. Factors that increase joint compression (unstable occlusion, parafunction, bite correction, macrotrauma, and internal derangement) and/or diminish the remodeling capacity (systemic illnesses, hormonal imbalance, young age, and gender) of the TMJ may cause condylar resorption and mandibular relapse after occlusal correction. Head film and left sagittal tomograms are depicted for a 21-year-old woman. (A) Before orthognathic surgery (pre), (B) 7 months after orthognathic surgery, and (C) 34 months after orthognathic surgery. Note the extreme dysfunctional remodeling depicted in the tomograms from (A) to (B) to (C). The dysfunctional condylar resorption resulted in a Class II occlusion with anterior open bite (C, head film). Dysfunctional condylar resorption is the sum of hidden systemic factors and compression (orthognathic surgery in this case). History, examination, and blood work revealed that the patient did not produce estrogen (Kallman syndrome—hidden systemic factor), which led to profound condylar resorption after normal orthognathic surgery. During this procedure, normal orthognathic surgery (standard techniques) as performed by the authors was designed to produce minor joint compression. Despite minor compression, the patient responded with profound resorption secondary to low estrogen overlay.

sessed the osseous changes that have been associated with condylar displacement. These studies have shown consistent osseous resorption of the postglenoid spine and posterior condylar surface when the condyle is posteriorly displaced and compressed in the glenoid fossa. A summary of compression studies has been published by Arnett et al.[2-5] Similarly, Arnett[11,12] and Tamborello[11] have demonstrated morphologic changes of the human mandibular condyle associated with posteriorization and/or medial or lateral condylar torquing during orthognathic surgery.

Mechanical factors that can cause changes in condylar morphology[2-12] include occlusal therapy, internal derangement, parafunction, macrotrauma, and unstable occlusion; each of these factors is discussed in detail later in the text. Such mechanical factors may occur in isolation, or they may be interrelated, interdependent, and coexistent. When 2 or more biomechanical factors coexist, morphologic change is more likely to occur. Additionally, if the host adaptive capacity is limited, morphologic changes are accentuated when compression factors are active.

TMJ Remodeling in Response to Occlusal Correction Procedures

Animal studies, as summarized by Arnett et al,[2-5,11,12] have shown that, in every instance, sudden change of the mandibular condyle position secondary to occlusal change leads to postglenoid spine and posterior condylar resorption.

In addition, as summarized by Arnett et al,[2-5,11,12] orthodontics, prosthetics, and orthognathic surgery can produce condyle remodeling or

resorption resulting from displacement and compression during treatment; these changes may be either functional, maintaining the occlusion, or dysfunctional, with attendant Class II malocclusion development. Coexisting remodeling stimuli (internal derangement, parafunction, macrotrauma, unstable occlusion, hormones, or systemic diseases) may accentuate condylar remodeling and produce a dysfunctional Class II malocclusion.[-12]

During most orthognathic surgery procedures, gross positional changes of the mandibular condyle can occur, so those procedures have a high risk of producing gross condylar morphologic changes.[11,12] Arnett and Tamborello[11,12] studied the TMJ tomograms from 61 orthognathic surgery patients to determine the effects of surgically induced condylar position change on the morphology of the mandibular condyle. They found that intraoperative condylar torquing or posteriorization leads to condylar resorption and late mandibular relapse.[11,12]

Orthodontic and prosthetic occlusal treatment may also produce condyle position changes, but generally not to the same extent and not as suddenly as orthognathic surgery. Therefore, these nonsurgical procedures can contribute to significant condylar remodeling, especially in susceptible individuals with concurrent predisposing compressive and/or systemic overlays. Class II orthodontic patients seem to be particularly susceptible to condylar displacement, perhaps because predisposing compressive and systemic factors may be the source of the original Class II malocclusion.

Articular Disk Displacement (Internal Derangement)

The relationship between internal derangement (ID) and condylar remodeling in adults is not well understood. Arnett et al[2-5] have summarized the internal derangement literature, with a special focus on the relationship between ID and condylar resorption. ID is very common in the population, and is found with and without bony TMJ remodeling. Thus, there does not seem to be a clear cause-and-effect relationship between ID and shape changes of the condyle and fossa in adult patients.

In the growing patient, ID is suspected of diminishing condylar growth, thereby negatively affecting mandibular growth. It is possible that when ID exists, the anterior growth of the mandible is reduced. However, patients with mandibular deficiency commonly have normal disk position, and patients with normal mandibular growth commonly have ID. As a result, currently no cause-and-effect relationship can be determined for anterior disk position and mandibular growth deficiency.[2-5]

When ID and condylar remodeling do occur concurrently, the ID may produce condylar remodeling by contributing to compressive loading of articular tissues. This notion is supported by multiple studies showing that condylar remodeling is more likely to be associated with nonreducing disk displacement cases than with reducing (clicking) disks.[2-5]

Parafunction

Parafunction may produce joint compression that can enhance resorption caused by other factors that initiate this process. Parafunction is particularly destructive when combined with condylar displacement associated with orthognathic surgery or other major occlusal treatment modalities.[2-5] In such cases, parafunction may increase intracapsular pressures and inhibit capillary perfusion, thereby producing an ischemic injury with attendant loss of temporomandibular tissue volume leading to mandibular retrusion.

Macrotrauma

Macrotrauma to the face may initiate condylar resorption. Macrotrauma consists of one epi-

Table 1. List A: Condyle (1-7) and List B: Fossa (1-5)

		Normal	*CR*
A	Condyle[a]		
	1. Head bulge	Yes	No
	2. Size	Normal	Small
	3. Surface contour	Rounded	Flat surface
	4. Spurs	No	Variable
	5. Cortex irregular	No	Yes
	6. Cortex breaks	No	Yes
	7. Subcortical cysts	No	Variable
B	Fossa[b]		
	1. Depth	Variable	Variable/shallow
	2. Surface contour	Rounded	Flat surface
	3. Cortex irregular	No	Yes
	4. Cortex breaks	No	Yes
	5. Subcortical cysts	No	Yes

[a]Cone beam computed tomography (CT) condyle characteristics associated with a normal condyle and CR are listed.
[b]Cone beam CT fossa characteristics associated with a normal joint and condylar resorption joint (CR) are listed.

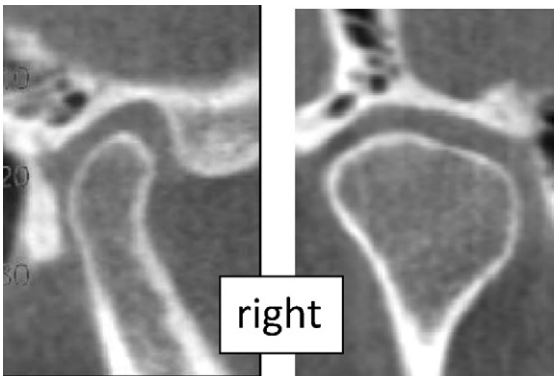

Figure 3. The cone-beam computed tomography (CT) images of a normal right TMJ is shown. Normal joints have condylar head bulge (condyle larger than neck), rounded surfaces, smooth and continuous cortex, and no subcortical cysts.

sode of large-magnitude force that is transmitted to articular structures of the temporomandibular joint. 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, but TMJ alterations can occur over time after the macrotrauma, leading to progressive mandibular retrusion.

Unstable Occlusion

By definition, a stable occlusion is produced when the teeth maximally intercuspate under muscle force and the condyle is not displaced and/or compressed. When a stable occlusion exists, the joints and the occlusion do not change significantly over time. Most natural occlusal relationships between maxillary and mandibular teeth are stable, regardless of the Angle classification. This is true because teeth and TM joints develop an ongoing noncompressed homeostatic relationship, and therefore, it is unlikely that there will be strong compressive forces acting on the TMJ in such stable occlusions. By definition, an unstable occlusion is produced when the teeth intercuspate under muscle force and the condyle is displaced and/or compressed. The compressed joint position then causes some degree of remodeling to occur, depending on the magnitude of position change and coexistent systemic factors. As stated earlier in the text, condylar displacement and compression may occur during occlusal correction procedures as simple as splint therapy or restorative procedures and as complex as orthognathic surgery. However, unstable occlusions also can be produced by nontreatment compressive etiologies such as heavy joint loading (bruxism, clenching) or disk derangements, combined with underlying systemic factors (low estrogen, rheumatoid arthritis, etc). These nontreatment factors can initiate the unstable occlusion, and then the unstable occlusion produces increased compression, resorption, and increased mandibular relapse. Un-

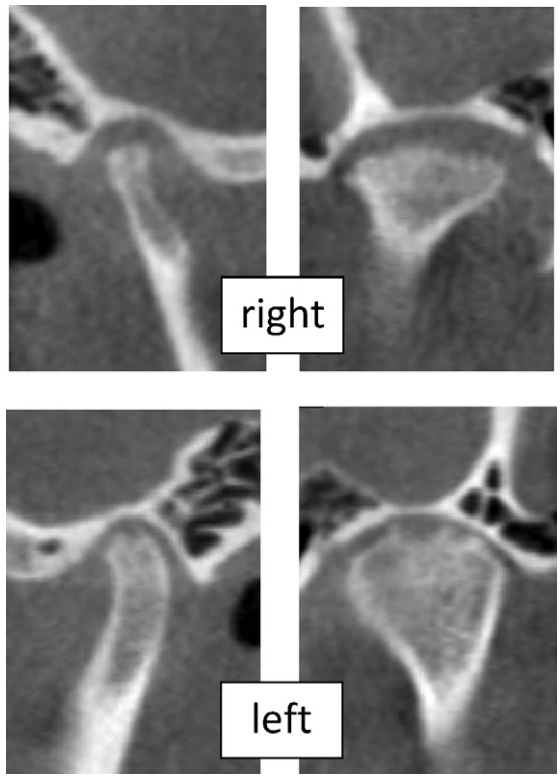

Figure 4. The cone-beam CT images of the right (above) and left (below) TMJ of the same patient on the same day are displayed. The right joint shows evidence of past change as indicated by lack of head bulge, small size, a medial spur, and flat superior surface. Additionally, the right joint demonstrates active changes, including condylar and fossa cortical irregularity, cortical breaks, and a singular subcortical bone cyst in sagittal view. The left TMJ (below) demonstrates normal size and shape, indicating that change has not occurred in the past. However, even without past left joint change, the cortex indicates that the joint is actively changing, as indicated by cortical irregularity, cortical breaks, and subcortical bone cysts. Both joints are in the in the process of condylar resorption, the right preceding the left. The head film and frontal bite photo for this patient are in shown in Figure 5.

der unstable condylar conditions, the condyles are displaced by the malocclusion, thereby creating a cycle of compression and resorption until a stable occlusion is produced and/or cofactors such as low estrogen, rheumatoid arthritis, and so forth are controlled.

Control of Condylar Resorption Associated With Occlusal Treatment

Condylar and fossa characteristics for a normal joint and a condylar resorption (CR) joint are shown in Table 1. A normal joint has a condylar head larger than the neck (head bulge), normal size, and rounded surfaces of the condyle and fossa (Fig 3). Shape changes (Fig 4) generally indicate changes that have occurred in the past; however, these changes may be ongoing as well. Shape changes may include some or all the following: small condyle, no head bulge, flat surfaces, and spurring. The fossa shape changes include flat surfaces and shallowness. Changes in the cortex (Fig 4) of the condyle and fossa indicate that condylar resorption is active, which produces breaks in the cortex, cortex irregularity, and subcortical cysts of the condyle and fossa. The facial skeleton is affected by condylar resorption. If both TM joints resorb symmetrically, the facial skeleton demonstrates a high-angle Class II malocclusion. If only one joint resorbs, or if one precedes the other, the mandible deviates to the smallest joint and cants up on the small side (Fig 5).

When active bilateral condylar resorption is identified or suspected, a rheumatology referral is indicated with appropriate laboratory studies (Table 2) to rule out collagen vascular disorders such as rheumatoid arthritis. Stabilization of progressive condylar resorption cases may be achieved with control of preexisting compressive factors, systemic factors, and the biological degradation caused by both, and this should be done before initiating any corrective surgical procedures. Medications[7,14,15,16] (Table 3) and splint therapy are commonly used before surgical occlusal correction, and may be appropriate for other occlusal correction techniques as well. One thing that has been learned from our experiences with progressive condylar resorption is that occlusal treatment techniques to minimize the possibility of compression-related resorption should be used—even if condylar resorption does not exist. Quite stable joints can be stimulated to resorb if treatment compresses the joint while producing bite correction, especially with orthognathic surgery.

Surgical Management Concepts and Procedures

Arnett and Gunson have presented surgical techniques to limit surgical compression and resultant condylar resorption. These sagittal

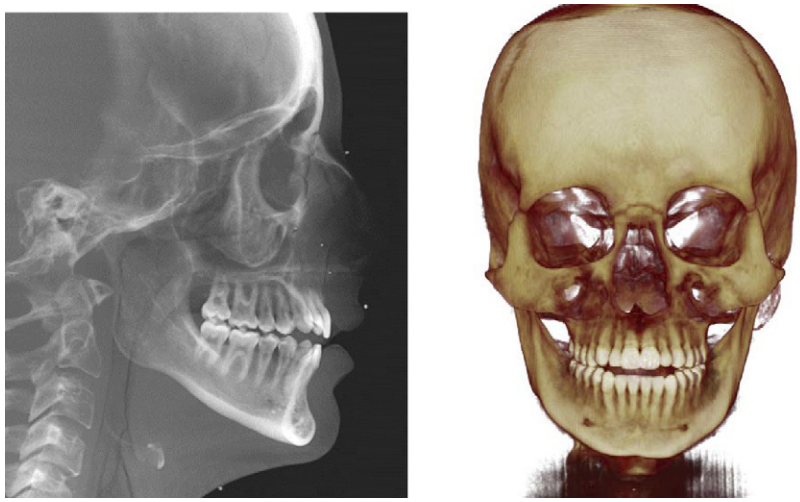

Figure 5. The head film and frontal cone-beam CT images of the patient in Figure 4 are shown. Note the Class II anterior open bite malocclusion. The etiology for this malocclusion is indeterminate but related to compressive and systemic overlay factors. Note that the mandibular midline is to the right and the right mandible is canted up, both findings related to the smaller right joint. (Color version of figure is available online.)

Table 2. Basic Laboratory Studies to Identify Systemic Arthritides are Listed

1	RF—rheumatoid factor Elevated 80% correlation with RA
2	Anti-CCP—anti-cyclic citrullinated peptide antibody 90%-95% correlation with rheumatoid arthritis (RA) Reveals RA earlier than RF More specific to RA than RF
3	ANA—antinuclear antibody Associated with autoimmune disorders, infections, cancer 25%-30% RA have positive ANA
4	Sed rate—erythrocyte sedimentation rate Indicates inflammatory activity
5	CRP—C-reactive protein General marker of inflammation May elevate with RA
6	25-Hydroxyvitamin D Deficiency associated with osteoporosis Deficiency secondary to low sunlight exposure or decreased dietary intake
7	17β-Estradiol Maintenance of bone Low estradiol associated with osteopenia and porosis

osteotomy techniques include bivector condylar seating, no segment clamping, no bicortical screws, plate fixation, a short split, osteotomy stretch, and anterior bone anchor placement. Postsurgical techniques include medications as needed, opening exercises, Class II elastics, and anterior skeletal elastics.[11-13] Condylar torque is associated with segment clamping and bicortical screw fixation of bilateral sagittal osteotomy surgery.[11-13] Joss and Vassalli[17] reviewed the literature comparing stability of sagittal osteotomy advancements when bicortical screws or miniplates were used. He found much greater long-term relapse (condylar resorption) for cases treated with bicortical screws (2%-50.3%), whereas miniplates had comparatively much less relapse (1.5%-8.9%).

Additionally, Gunson and Arnett[7,15] have suggested medical management to stabilize the biology of resorption initiated by inadvertent treatment compression of the TMJ or systemic diseases (Fig 5). The cellular pathways for bone loss at the mandibular condyle require specific cytokines and enzymes. If those cytokines and enzymes are blocked, condylar resorption is minimized or eliminated in spite of systemic factors or compression. Tumor necrosis factor-alpha (TNF-α) is a known cytokine that promotes osteoclastogenesis and osteoclast activity in the joints of patients with inflammatory arthritis. By administering TNF-α inhibitors (etanercept, adalimumab, etc), bone loss at the mandibular condyle may be controlled before, during, and after surgery. Another example is the matrix metalloproteinase (MMP) enzymes, which break down collagen as the final step in articular destruction. Tetracyclines are known MMP inhibitors and have been shown to reduce articular erosions in susceptible patients.

We believe that medical and compressive management of the TMJ significantly reduces the need for total TM joint replacement therapy; a similar shift is occurring in regard to total hip replacement surgery as reported by Hekmat et al.[16] They state that "Potential explanations for the reduced rate of total hip arthroplasty (THA) include reduced rheumatoid arthritis (RA) related joint damage due to better management of RA." In their study, Hekmat et al found that both disease-modifying antirheumatic drug (DMARD) (52%-87%) and TNF-inhibitor drug (0%-20%) use has increased from 1997 to 2005. Furthermore, Hekmat states, "There is extensive

Table 3. Medications Used to Control TMJ Arthritis

Vit D and Ca++	Bone density
C 500 mg and E 400 u	Antioxidants
Omega-3 fatty acid 2-4 G	Potent antioxidant
Doxycycline, 50-100 mg	Anti-inflammatory, MMP, cytokine inhibitor
Feldene, 10-20 mg	Anti-inflammatory, MMP, cytokine inhibitor
Simvastatin, 20 mg	Anti-inflammatory, MMP, cytokine inhibitor
Celebrex, 100 mg	Anti-inflammatory, MMP, cytokine inhibitor
Amitriptyline, 5-15 mg	Antibruxism, mm relax
Klonapin, 0.5-1 mg	Antibruxism
Tiagabine, 2-4 mg	Antibruxism
Botox injection, 36-48 u	Antibruxism
Simvistatin, 20 mg	Autoimmune inhibitor
17β-Estradiol, variable	Potent anti-inflammatory
Etanercept, 50 mg q week	TNF-α inhibitor
Adalimumab, 40 mg q 2 weeks	TNF-α inhibitor

Medications are used to control the biology of condylar resorption whether secondary to compression and/or systemic overlay, whether before or after treatment when active condylar resorption is occurring. All medications and general dosages are listed on left. The biological effects of each medication are listed on the right. Each patient generally will take vitamin D, calcium, doxycycline, and Feldene. If bruxism is a component of loading, one of the bruxism medications will be selected until control of symptoms is achieved. When resorption is active and aggressive as indicated in Figure 1 earlier in the text, additional appropriate medications are added, including an autoimmune inhibitor and, in the case of low estrogen, 17β-estradiol.

evidence for a reduced peripheral joint damage in patients with RA treated with TNF inhibitors, and such treatment could also prevent hip destruction." The parallels to the temporomandibular joint should be obvious, and it is our hope that this more conservative viewpoint becomes widely accepted in our field.

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