Condylar Degeneration and Diseases—Local and Systemic Etiologies

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The temporomandibular joint condyle is susceptible to developing a variety of problems, ranging from simple osteoarthrosis to severe condylar resorption. This article will focus on issues related to the etiology of these conditions. Condylar resorption (also known as condylysis) is an event rarely seen in normal orthodontic practice, but when it occurs, it is a very unhappy occurrence and often puzzling and inexplicable. This phenomenon represents an aggressive and fast-moving form of degenerative joint disease. As will be discussed in this article, many cases of temporomandibular joint degenerative joint disease arise from specific local or systemic etiologies, but others (especially condylysis) have been described as "idiopathic." It is the authors' opinion that the term "idiopathic" might be too easily used as an explanation. When something happens, there is usually a reason, but often it is not known what that reason is. Hence, the term "idiopathic" is used as a quick and easy explanation for the occurrence rather than investigating deeper to find the real cause of the problem. Certainly, the cause cannot be found in every case, but there are more areas to investigate than many are aware of, and some of those will be presented here. (Semin Orthod 2013;19:89-96.)

When the words condylar resorption is mentioned, most orthodontists think of 3 things: (1) idiopathic condylar resorption, (2) condylar changes as a result of traumatic injury to the mandible and/or condyle, or (3) condylar changes as unfortunate sequelae of orthognathic surgery. However, this is a very incomplete list of possibilities to be considered in such cases.

The purpose of this article is to offer a broader and more realistic look at the etiologies of condylar disease and/or morphologic changes to the condyle as well as surrounding hard- and soft-tissue structures. We present this information in such a way as to improve the differential diagnostic capabilities when confronted with unclear symptoms and their potential etiologies. The obvious desired result is more informed decision making in reacting to problems and improving the quality of care of our patients.

After evaluating problems encountered in practice and an evaluation of the literature, we recommend the division of condylar problems into 3 broad categories: (1) localized etiologic factors, such as osteoarthritis and traumatic injuries, (2) systemic etiologic factors, that is, the systemic disease entities that can result in condylar degeneration and other changes, and (3) condylar resorption as either an unfortunate sequelae of orthognathic surgery or as a totally spontaneous occurrence. To help create a systematic approach to diagnosis and treatment, we present the individual elements of each disease, beginning with a general description, expected

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findings on physical examination, common radiographic characteristics, the clinical features, and finally, treatment.

Localized Etiologic Factors

Osteoarthritis

Description

Also known as *osteochondritis dessicans*, this process is described as a result of chronic inflammation of the synovial and discal tissues.[1-3] The clinical outcome is an inflammatory response along with degenerative changes in the cartilage covering and possible necrosis of the bony articular and even subarticular surfaces. The noninflammatory version of this process is often referred to as osteoarthrosis.

Physical Examination

Tenderness of one condyle is the most common characteristic in osteoarthritis. Auscultation may reveal crepitus in the effected joint, and secondary muscular soreness in the masticatory muscles may also be present.

Radiographic Features

Radiographic features may be highly variable, but in the classic presentation, there is an osteophyte on the anteroposterior aspect of the condyle[4] (Fig 1). Irregularities of the cortical plate of the condylar head may be noted, depending on the quality of the image.

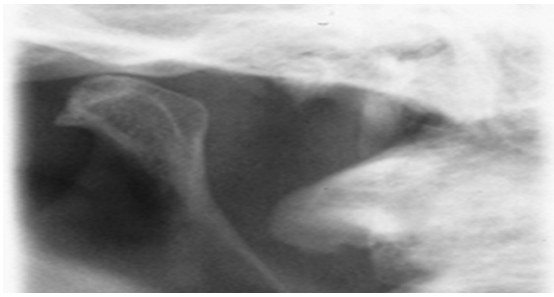

Figure 1. This 49-year-old patient was referred with a chief complaint of "temporomandibular joint." She had unilateral joint pain, and the panoramic radiograph revealed an osteophyte on the anterosuperior aspect of the condyle, characteristic of localized osteoarthritis.

Treatment

Therapy is similar to the orthopedic approach to other joints. Aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly prescribed.[5-7] Muscular symptoms may be treated with muscle relaxants, or drugs from the benzodiazepine family are useful. We most commonly prescribe 1.0 mg of Klonopin (clonazepam) (Genentech USA, Inc., South San Francisco, CA) at bedtime.

Functional Load

In general terms, bone morphology is a function of the load put on it because of bone's inherent characteristics of plasticity.[8-10] The shape and form of the temporomandibular joint (TMJ) condyle are a result of the functional forces placed on it, but in addition, human condylar variability is remarkable.

Physical Examination

Generally unremarkable, but various complaints such as joint soreness or crepitus are the most frequent when abnormal loads are placed on a joint, or when normal loads are placed on a compromised joint.

Radiographic Features

What does a "normal" condyle look like? The variability can be enormous, as would be expected because of the multitude of influencing factors, with genetics being only one of them. Therefore, one must be cautious in deciding how much diagnostic interpretation can be made from a panoramic film, a tomogram, or a cone-beam CT examination.

Case example

Figure 2A represents the condyle of a patient with a severe Class II malocclusion with 8 mm of overjet. She had bilateral joint and facial pain, and she had consulted an oral and maxillofacial surgeon who diagnosed her as having degenerative joint disease secondary to internal derangement of the joint. The pencil line on the film represents where the surgeon had drawn the procedure he proposed: disk plication and "condylar shave"[11] designed to reshape the "eroded" condyle. Instead, the patient chose the course of

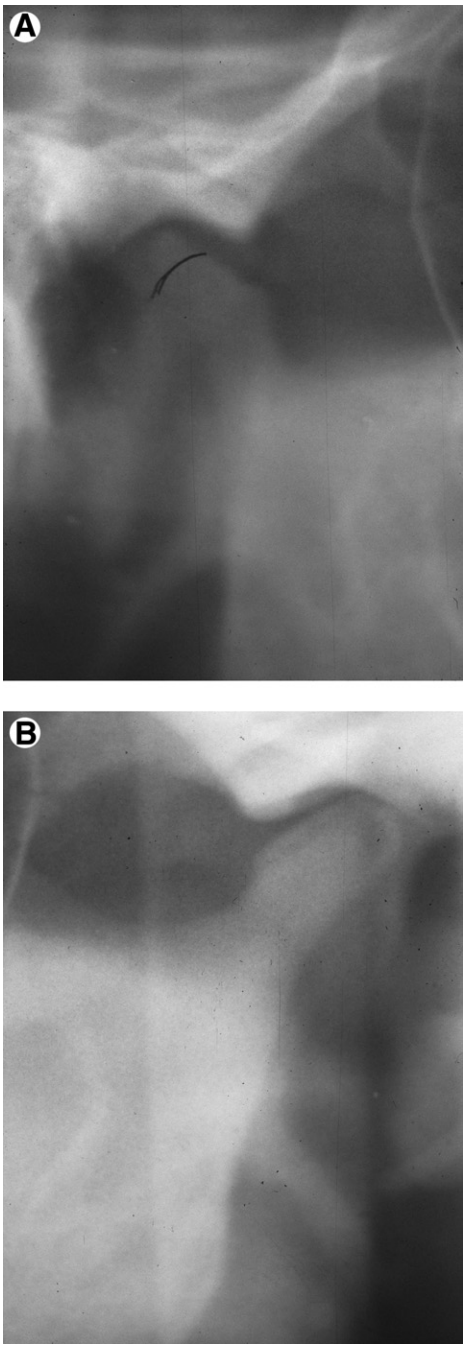

Figure 2. (A) This adult patient presented with a Class II malocclusion with 8 mm of overjet. She was diagnosed as having degenerative joint disease secondary to internal derangement of the joint, resulting in the erosion of the condylar head. A surgeon proposed a "condylar shave" designed to reshape the "eroded" condyle. (B) The same condyle 1 year after treatment with orthodontics and surgical mandibular advancement. With a dramatic change in function, there was a resulting change in the condylar anatomy.

orthodontics and mandibular advancement followed by re-evaluation of the joint. After correction of the skeletal deformity and finishing of orthodontics, her symptoms were reduced dramatically, and she pursued no further treatment. Figure 2B is the same condyle 1 year after treatment was completed; with a dramatic change in function, there was a resulting change in the condylar anatomy.

Traumatic Injury

Description

Traumatic injury to a condyle is the most frequent cause of mandibular asymmetry in growing children. A blow to the mandible that results in traumatic compression of the condylar head against the posterior glenoid fossa can produce an immediate loss of some portion of the cartilage cover of the condylar head, resulting in loss of nourishment and protection along with subsequent bony condylar lysis.[12-14] Fracture of the condylar neck also results in either functional loss of the condylar head itself or interruption of the blood supply, possibly leading to condylar lysis. In trauma cases, limitation of opening or lateral deviation is usually a result of either 1 or both condyles being affected by a traumatic injury. If scarring of the temporomandibular area occurs so that the translation of the mandible is impeded, there will likely also be an interference with normal mandibular growth.

Physical Examination

In adolescents, if restricted range of motion of the affected TMJ occurs, skeletal asymmetry is likely to occur and to get progressively worse as growth continues. The greater the inhibition of translation, the more progressive the deformity will be.

Treatment

The primary guideline in both children and adults is to restore function as soon as possible to avoid ankylosis or the formation of fibrous tissue scarring. Treatment in the child who has experienced condylar trauma often includes the use of a "hybrid" functional appliance,[15] or a unilateral hyperpropulsion device (such as a fixed Herbst appliance[16]); these may be used to guide subsequent growth and maintain as much free-

dom of movement as possible. Any form of surgical treatment should not be used in children with mandibular asymmetry until conservative treatment with some form of growth modification has been attempted and failed.

Case Presentation: Use of a Unilateral Hyperpropulsion Appliance

A 16-year-old girl (Fig 3A) was involved in a motor vehicular accident in which she suffered a zygomatic fracture and a right condylar fracture, resulting in a unilateral Class II malocclusion (Fig 3B). She was placed into full orthodontic appliances and a Herbst appliance, which has the advantage of promoting a growth modification or remodeling effect. The Herbst appliance was activated every 6 weeks on the affected right side only to the point of overcorrection. The propulsion arms of the Herbst appliance were removed for an 8-week period to assess any relapse, and then fixed appliance treatment was resumed to an excellent facial and occlusal outcome (Fig 3D and E). Comparison of the pretreatment and posttreatment films shows the remodeling of the injured condyle (Fig 3F).

Systemic Etiologic Factors

Condylar degeneration or growth interference can occur as a result of many systemic diseases. A review of the literature reveals at least 12 documented diseases that can be associated with condylar resorption. These include the following:

1. Rheumatoid arthritis (RA): a chronic autoimmune inflammatory disorder that typically affects the small joints of the hands and feet. Unlike osteoarthritis, RA affects the lining of the joints, causing pain and swelling that can eventually result in bone erosion and joint deformity.[17]
2. Psoriatic arthritis: a chronic inflammatory disease causing symptoms such as inflammation in joints and overproduction of skin cells, with similar results as RA.[18]

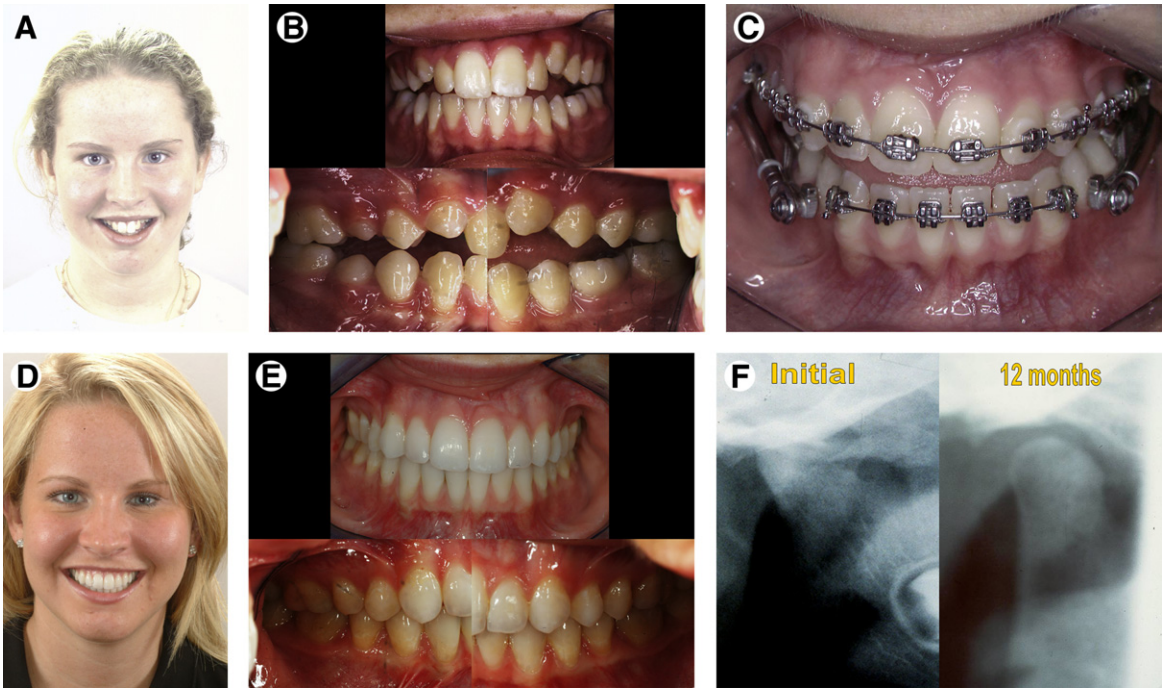

Figure 3. (A) This patient was involved in a motor vehicular accident, with left condylar fracture resulting in a unilateral Class II malocclusion. (B) The unilateral Class II malocclusion. (C) The patient was placed in orthodontics appliances and a Herbst appliance, with the right side periodically activated to protract the injured condyle. (D) The resulting facial symmetry was greatly improved. (E) The final occlusal results. (F) Comparison of the pretreatment and posttreatment films demonstrating remodeling of the injured condyle. (Color version of figure is available online.)

3. Juvenile idiopathic arthritis (JIA)[19-22]: it is an autoimmune disease of unknown etiology that affects 1 in 1000 children in the United States. Previously referenced in the literature as juvenile RA, the change in terms occurred because rheumatologists determined that this condition in adolescents is not simply a "junior" version of RA. Laboratory workups show that these young patients do not have the rheumatoid factor (RF) that is indicative of RA; hence, the term "idiopathic" has become the new label.
4. Exercise-induced osteopenia[23,24]
5. Scleroderma[25-27]
6. Lupus erythematosus[28]
7. Familial Mediterranean fever[29,30]
8. Marfan syndrome[31]
9. Sjogren syndrome[32]
10. Sarcoidosis
11. Mixed connective tissue disease[33]
12. Dermatomyositis[34]

In the following sections, we discuss RA and JIA in detail.

Rheumatoid Arthritis

Clinical Features

The most striking aspect is the change in occlusion, with a progressive anterior open bite, whereas the other common feature is significant change in condylar size and morphology. There is a difference between the adult and adolescent versions of these condylar changes because adults may have actual condylar resorption, which is radiographically detectable, whereas adolescents may experience an attenuation of growth, resulting in severely restricted mandibular growth, but their condyles may appear more normal. These changes at the condylar level result in typical clinical characteristics such as mandibular recession, anterior open bite, and narrowing of the bigonial width.

Radiographic Features

Approximately 30% of RA patients exhibit radiographic changes that are gross enough to be observed on plain films or tomograms. The characteristic cephalometric presentation is flattening of the anterosuperior aspect of the condyles and irregular destruction of the temporal fossa, which is seen as flattening of the fossa that is most often bilateral (Fig 4). If the patient has joint pain but not these radiographic changes, then localized osteoarthritis is a possible differential diagnosis, but systemic RA is always a possibility. Therefore, the history is very important, and the following questions need to be asked: "Is there any history of arthritis in the family?" and "Do any other of your joints hurt?" Other joints need to be examined for swelling or diminished range of motion. Blood assays to identify RF and other inflammation markers are also indicated and are detailed later in this article.

Treatment

Treatment for the TMJ pain and dysfunction tends to be localized and palliative, including aspirin or NSAID therapy, therapeutic exercises, oral appliances, and steroid injections. Intra-articular steroids are probably best for active inflammation. The open bite malocclusion can be treated conventionally with orthognathic surgery, which is the most common approach, but

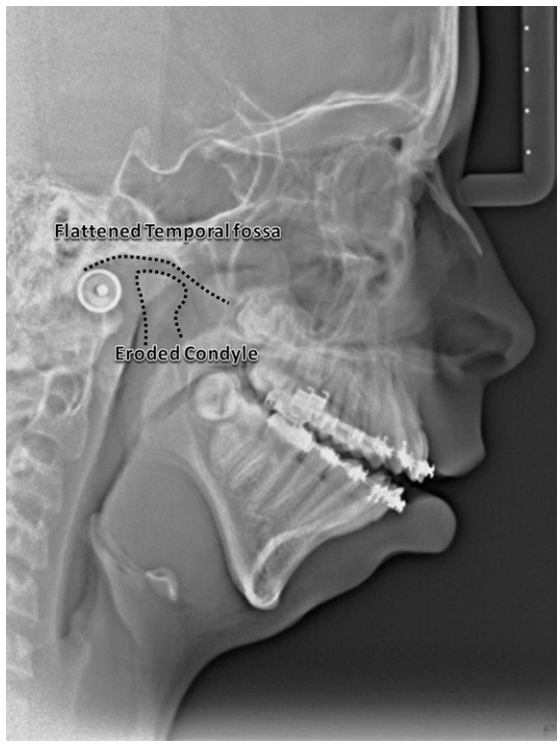

Figure 4. The characteristic cephalometric radiographic presentation of juvenile idiopathic arthritis shows flattening of the anterosuperior aspect of the condyles and irregular destruction of the temporal fossa, seen as flattening of the fossa.

the use of temporary anchorage devices to facilitate orthodontic correction may also be indicated in milder cases. Prosthetic joint replacement may be included as an option. We recommend that before treatment is pursued, the patients should be followed with serial cephalometric radiographs every 6 months until there are 3 consecutive films with no detectable change. Although this is far from being a perfect assessment method, it probably is the most accurate way to determine when the active and destructive disease process is "burned out." Although some recommend technetium radioisotope uptake studies to see whether excessive remodeling activity is continuing on the joint surfaces, these may be difficult to interpret. Magnetic resonance imaging (MRI) with contrast enhancement is both very sensitive and specific for active TMJ inflammation.

Juvenile Idiopathic Arthritis

Clinical Features

Many JIA patients have severe micrognathia due to the disruption of mandibular growth. This is generally bilateral, but can be occasionally unilateral. Complaint of pain is uncommon, particularly in younger children, with the exception of synovitis (inflammation of the synovial lining of the TMJ), which can be treated as a separate issue if necessary. Open bite in JIA patients is rarer than in adult RA patients.

Radiographic Features

The condyles may appear eroded and flattened, and the density of the condyles may appear rarified. The glenoid fossa is shallow, and there may be abnormal articular tubercles. A prominent feature in JIA may include antegonial notching of the body of the mandible. MRI with contrast enhancement can also reveal synovial fluid, bone marrow edema, pannus, disk thinning and displacement, and synovial lining enhancement. A high prevalence of TMJ arthritis at onset of the disease in children with JIA can be detected by MRI, but not by ultrasonography.[35]

Treatment

Again, localized treatment can be administered through intra-articular long-acting corticosteroids, and the clinician also can use aspirin or NSAID therapy. Temporomandibular arthritis can be followed clinically when pain or discomfort is present by measuring mouth opening and range of mandibular movement, and also using MRI with contrast enhancement. In case of painful synovitis, a soft-tissue synovectomy may be possible for the relief of pain. As in the adult with rheumatoid joint destruction, observation and serial cephalometric follow-up for cessation of the disease process is the assessment method of choice, to be followed by correction of the malocclusion.

Blood tests may be administered to confirm or rule out a diagnosis of JIA. Most commonly, erythrocyte sedimentation rate assays may demonstrate the presence of systemic inflammation. A positive antinuclear antibody assay is associated with increased uveitis in children with chronic arthritis. The genetic marker HLA-B27 is associated with enthesitis-related JIA. RF or anti-cyclic citrullinated peptide antibodies are found in $<4\%$ of children with JIA. The use of antinuclear antibody assay can detect those specific antibodies that are proteins commonly associated with autoimmune disease such as arthritis, and the RF is an antibody found in individuals with RA. In JIA cases, however, RF is often not found in children, and therefore, its absence in a test does not mean the patient is negative for JIA.

Conclusions

At the outset of this article, we described 3 broad areas by which we define condylar changes and/or degeneration: (1) problems due to localized etiologic factors, (2) problems due to systemic etiologic factors, and (3) severe resorptive problems due to postoperative factors or of idiopathic origin. This systematic approach to these problems is meant to provide a framework for the differential diagnosis of condylar change, and is meant to help lead clinicians in a logical manner to a reasonable diagnosis before recommending therapy. Although some cases in every category may seem to be of mysterious origin, it is too simple to lean on the crutch of the term "idiopathic." We encourage the reader to follow up with investigation into the literature of other etiologic concepts, such as the potential linkage of hormonal changes to condylar resorption[36-39]

(refer to the article by Arnett and Gunson in this issue).

The topic of postoperative and idiopathic condylar resorption was introduced and discussed briefly in this article, but it was not presented in detail because of the volume of material that would need to be covered,[40-47] and also because it is discussed thoroughly in other articles in this issue. Very often, the response to postoperative condylar resorption (CR) is another recommendation of either orthognathic or TMJ surgery (or both), rather than a careful examination of what may have been potential etiologies of the postoperative event. When the clinician is confronted with the unfortunate event of postoperative condylar degeneration, it is important to react as logically as possible. Therefore, we hope that we have at least provided a framework to facilitate a more thoughtful approach to dealing with these events, thereby leading to appropriate diagnostic and therapeutic methodologies. Although some of the cases are truly mysterious and "idiopathic," not all of them are.

References

1. Milam SB: TMJ osteoarthritis, in Laskin DM, Greene CS, Hylander WL (eds): TMDs: An Evidence-Based Approach to Diagnosis and Treatment. Chicago, IL, Quintessence, 2006, pp 105-123
2. Mercuri LG: Osteoarthritis, osteoarthrosis, and idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am 20:169-183, 2008
3. Campos PS, Freitas CE, Pena N, et al: Osteochondritis dissecans of the temporomandibular joint. Dentomaxillofac Radiol 34:193-197, 2005
4. Marbach T: Arthritis of the temporomandibular joint. Am Fam Physician 19:131-139, 1979
5. Ismail F, Demling A, Hessling K, et al: Short-term efficacy of physical therapy compared to splint therapy in treatment of arthrogenous TMD. J Oral Rehabil 34:807-813, 2007
6. Wilder-Smith CH, Hill L, Spargo K, et al: Treatment of severe pain from osteoarthritis with slow-release tramadol or dihydrocodeine in combination with NSAID's: A randomised study comparing analgesia, antinociception and gastrointestinal effects. Pain 91:23-31, 2001
7. Dionne RA: Pharmacologic approaches, in Laskin DM, Greene CS, Hylander WL (eds): TMDs: An Evidence-Based Approach to Diagnosis and Treatment. Chicago, IL, Quintessence, 2006, pp 347-357
8. Haskin CL, Milam SB, Cameron IL: Pathogenesis of degenerative joint disease in the human temporomandibular joint. Crit Rev Oral Biol Med 6:248-277, 1995
9. Motta AT, Cevidanes LH, Carvalho FA, et al: Three-dimensional regional displacements after mandibular advancement surgery: One year of follow-up. J Oral Maxillofac Surg 69:1447-1457, 2011
10. Kurusu A, Horiuchi M, Soma K: Relationship between occlusal force and mandibular condyle morphology. Evaluated by limited cone-beam computed tomography. Angle Orthod 79:1063-1069, 2009
11. Kurita K, Westesson PL, Eriksson L, et al: High condylar shave of the temporomandibular joint with preservation of the articular soft tissue cover: An experimental study on rabbits. Oral Surg Oral Med Oral Pathol 69:10-14, 1990
12. Arnett GW, Milam SB, Gottesman L: Progressive mandibular retrusion—Idiopathic condylar resorption. Part II. Am J Orthod Dentofacial Orthop 110:117-127, 1996
13. Choi J, Oh N, Kim IK: A follow-up study of condyle fracture in children. Int J Oral Maxillofac Surg 34:851-858, 2005
14. Thorén H, Hallikainen D, Iizuka T, et al: Condylar process fractures in children: A follow-up study of fractures with total dislocation of the condyle from the glenoid fossa. J Oral Maxillofac Surg 59:768-774, 2001
15. Kahl-Nieke B, Fischbach R: Condylar restoration after early TMJ fractures and functional appliance therapy. Part I: Remodelling. J Orofac Orthop 59:150-162, 1998
16. Kahl-Nieke B, Fischbach R, Ruf S, et al. Temporomandibular joint remodeling in adolescents and young adults during Herbst treatment: A prospective longitudinal magnetic resonance imaging and cephalometric radiographic investigation. Am J Orthod Dentofacial Orthop 115:607-618, 1999
17. Gynther GW, Tronje G, Holmlund AB: Radiographic changes in the temporomandibular joint in patients with generalized osteoarthritis and rheumatoid arthritis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 81:613-618, 1996
18. Farronato G, Garagiola U, Carletti V, et al: Psoriatic arthritis: Temporomandibular joint involvement as the first articular phenomenon. Quintessence Int 41:395-398, 2010
19. Ringold S, Cron R: The temporomandibular joint in juvenile idiopathic arthritis: Frequently used and frequently arthritic. Pediatric. Rheumatology 29:7-11, 2009
20. Martini A, Lovell DJ: Juvenile idiopathic arthritis: State of the art and future perspectives. Ann Rheum Dis 69: 1260-1263, 2010
21. Hilderson D, Corstjens F, Moons P, et al: Adolescents with juvenile idiopathic arthritis: Who cares after the age of 16? Clin Exp Rheumatol 28:790-797, 2010
22. Petty RE, Southwood TR, Manners PJ, et al: International League of Associations for Rheumatology Classification of Juvenile Idiopathic arthritis: Second revision, Edmonton. J Rheumatol 2004:390-392, 2001
23. McLean JA, Barr SI, Prior JC: Dietary restraint, exercise, and bone density in young women: Are they related? Med Sci Sports Exerc 33:1292-1296, 2001
24. Lindberg JS, Powell MR, Hunt MM, et al: Increased vertebral bone mineral in response to reduced exercise in amenorrheic runners. West J Med 146:39-42, 1987

25. Defabianis P: Scleroderma: A case report of possible cause of restricted movement of the temporomandibular joint with effects on facial development. J Clin Pediatr Dent 28:33-38, 2003
26. Auluck A, Pai KM, Shetty C, et al: Mandibular resorption in progressive systemic sclerosis: A report of three cases. Dentomaxillofac Radiol 34:384-386, 2005
27. Mugino H, Ikemura K: Progressive systemic sclerosis with spontaneous fracture due to resorption of the mandible: A case report. J Oral Maxillofac Surg 64:1137-1139, 2006
28. Jonsson R, Lindvall AM, Nyberg G: Temporomandibular joint involvement in systemic lupus erythematosus. Arthritis Rheum 26:1506-1510, 1983
29. Tovi F, Gatot A, Fliss D: Temporomandibular arthritis in familial Mediterranean fever. Head Neck 14:492-495, 1992
30. Ince E, Cakar N, Tekin M, et al: Arthritis in children with familial Mediterranean fever. Rheumatol Int 21:213-217, 2002
31. Bauss O, Sadat-Khonsari R, Fenske C, et al: Temporomandibular joint dysfunction in Marfan syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 97:592-598, 2004
32. Fain ET, Atkinson GP, Weiser P, et al: Temporomandibular joint arthritis in pediatric Sjogren disease and sarcoidosis. J Rheumatol 38:2272-2273, 2011
33. Lanigan DT, Myall RW, West RA, et al: Condylysis in a patient with a mixed collagen vascular disease. Oral Surg Oral Med Oral Pathol 48:198-204, 1979
34. Brennan MT, Patronas NJ, Brahim JS: Bilateral condylar resorption in dermatomyositis: A case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 87:446-451, 1999
35. Müller L, Kellenberger CJ, Cannizzaro E, et al: Early diagnosis of temporomandibular joint involvement in juvenile idiopathic arthritis: A pilot study comparing clinical examination and ultrasound to magnetic resonance imaging. Rheumatology (Oxford) 48:680-685, 2009
36. Arnett GW, Tamborello JA: Progressive class II development: Female idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am 2:699-716, 1990
37. Wolford LM, Cardenas L: Idiopathic condylar resorption: Diagnosis, treatment protocol, and outcomes. Am J Orthod Dentofacial Orthop 116:667-677, 1999
38. Gunson MJ, Arnett GW, Formby B, et al: Oral contraceptive pill use and abnormal menstrual cycles in women with severe condylar resorption: A case for low serum 17β-estradiol as a major factor in progressive condylar resorption. Am J Orthod Dentofacial Orthop 136:772-779, 2009
39. Kang SC, Lee DG, Choi JH, et al: Association between estrogen receptor polymorphism and pain susceptibility in female temporomandibular joint osteoarthritis patients. Int J Oral Maxillofac Surg 36:391-394, 2007
40. Kerstens HC, Tuinzing DB, Golding RP, et al: Condylar atrophy and osteoarthrosis after bimaxillary surgery. Oral Surg Oral Med Oral Pathol 69:274-280, 1990
41. Bouwman JP, Kerstens HC, Tuinzing DB: Condylar resorption in orthognathic surgery. The role of intermaxillary fixation. Oral Surg Oral Med Oral Pathol 78:138-141, 1994
42. De Clercq CA, Neyt LF, Mommaerts MY, et al: Condylar resorption in orthognathic surgery: A retrospective study. Int J Adult Orthodon Orthognath Surg 9:233-240, 1994
43. Kobayashi T, Izumi N, Kojima T, et al: Progressive condylar resorption after mandibular advancement. Br J Oral Maxillofac Surg 50:176-180, 2012
44. Borstlap WA, Stoelinga PJ, Hoppenreijs TJ, et al: Stabilisation of sagittal split advancement osteotomies with miniplates: A prospective, multicentre study with two-year follow-up. Part III Condylar remodeling and resorption. Int J Oral Maxillofac Surg 33:649-655, 2004
45. Hwang SJ, Haers PE, Seifert B, et al: Non-surgical risk factors for condylar resorption after orthognathic surgery. J Craniomaxillofac Surg 32:103-111, 2004
46. Hwang SJ, Haers PE, Zimmermann A, et al: Surgical risk factors for condylar resorption after orthognathic surgery. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89:542-552, 2000
47. Cutbirth M, Van Sickels JE, Thrash WJ: Condylar resorption after bicortical screw fixation of mandibular advancement. J Oral Maxillofac Surg 56:178-182, 1998