A Classification System for Conditions Causing Condylar Hyperplasia

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A classification system was developed to place patients with condylar hyperplasia (CH) into categories based on histology, clinical and imaging characteristics, effects on the jaws and facial structures, and rate of occurrence. Four major categories were defined. CH type 1 is an accelerated and prolonged growth aberration of the "normal" mandibular condylar growth mechanism, causing a predominantly horizontal growth vector, resulting in prognathism that can occur bilaterally (CH type 1A) or unilaterally (CH type 1B). CH type 2 refers to enlargement of the mandibular condyle caused by an osteochondroma, resulting in predominantly unilateral vertical overgrowth and elongation of the mandible and face. One of the forms has predominantly a vertical growth vector and condylar enlargement, but without exophytic tumor extensions (type 2A), whereas the other primary form grows vertically but develops horizontal exophytic tumor growth off of the condyle (CH type 2B). CH type 3 includes other rare, benign tumors and CH type 4 includes malignant conditions that originate in the mandibular condyle causing enlargement. The order of classification is based on occurrence rates and type of pathology, where CH type 1A is the most commonly occurring form and CH type 4 is the rarest. This classification system for CH pathology should help the clinician understand the nature of the pathology, progression if untreated, recommended ages for surgical intervention to minimize adverse effects on subsequent facial growth and development in younger patients, and the surgical protocols to comprehensively and predictably treat these conditions.

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Hyperplasia indicates the increased production and growth of normal cells in a tissue or organ without an increase in the size of the cells, but the affected part becomes larger yet retains its basic form. *Condylar hyperplasia* (CH) is a generic term describing conditions that cause excessive growth and enlargement of the mandibular condyle. There are many suggested etiologies of CH, including neoplasia, trauma, infection, abnormal condylar loading,[1] and aberrant growth factors. These condylar pathologies can adversely affect the size and morphology of the mandible, alter the occlusion, and indirectly affect the maxilla, with the resultant development or worsening of dentofacial deformities, such as mandibular prognathism; unilateral enlargement of the condyle, neck, ramus, and body; facial asymmetry; malocclusion; and pain. Some CH pathologies occur more commonly within particular age ranges and genders. Identifying the specific CH pathology will provide insight to its progression if untreated; the clinical, imaging, and histologic characteristics; and treatment protocols proved to eliminate the pathologic processes and provide optimal functional and esthetic outcomes. Adams[2] in 1836 and Humphry[3] in 1856 were 2 of the first to describe and treat CH with a condylectomy.

This report presents a simple but encompassing classification for CH. The acronym CH for this classification includes conditions that create excessive growth and enlargement of the condyle that can cause alterations in the bony architecture of the mandible, malocclusion, and dentofacial deformity. This classification excludes congenital deformities (eg, hemifacial hypertrophy, Sturge-Webber syndrome, fibrous dysplasia) and endocrinal conditions (eg, acromegalia) that

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Table 1. CONDYLAR HYPERPLASIA CLASSIFICATION

CH	Age of Onset	Clinical Characteristics	Imaging	Histology	Treatment Options	Age for Surgery
Type 1A	pubertal growth	bilateral accelerated symmetric or asymmetric growth; self-limiting; can grow into mid-20s; Class III occlusion; prognathic mandible	elongated condylar head, neck, body; normal condylar head shape; MRI: thin discs; asymmetric cases may involve contralateral disc displacement	normally growing condyle; may show slight widening of fibrocartilage on condyle or increased vascularity in proliferative zone	bilateral high condylectomy, disc repositioning, orthognathic surgery; OR wait until growth is complete and then perform orthognathic surgery	youngest age for high condylectomy, orthognathic surgery (F = 14 yr; M = 16 yr)
Type IB	pubertal growth	unilateral accelerated asymmetric growth; self-limiting; can grow into mid-20s; deviated mandibular prognathism; ipsilateral Class III occlusion; anterior and contralateral crossbite	unilateral elongated condylar head, neck, body; deviated prognathism; MRI: thin disc; may have ipsilateral/contralateral disc displacement	normally growing condyle but may show slight widening of fibrocartilage on condyle or increased vascularity in proliferative zone	unilateral high condylectomy, disc repositioning, orthognathic surgery; OR wait until growth is complete and then perform orthognathic surgery	youngest age for high condylectomy, orthognathic surgery (F = 15 yr; M = 17-18 yr)
Type 2	two thirds of cases begin in second decade	unilateral vertical elongation of face and jaws; not self-limiting; can grow indefinitely; ipsilateral posterior open bite	unilateral vertical enlarged condylar head, neck, ramus, body; MRI: ipsilateral disc commonly in place; contralateral TMJ arthritis, displaced disc, 75% of cases	osteochondroma: layer of germinating undifferentiated mesenchymal cells, hypertrophic cartilage, islands of chondrocytes in subchondral trabecular bone; thickened and irregular bony trabeculae	ipsilateral low condylectomy to preserve neck, recontour condylar neck, disc repositioning, orthognathic surgery; nonsalvageable disc; custom-fitted total joint prosthesis	youngest preferred age for low condylectomy, orthognathic surgery (F = 15 yr; M = 17-18 yr)
Type 2A			vertical growth vector; no horizontal exophytic growth off condyle			
Type 2B			enlargement of condyle with exophytic growth off the head			

Table 1. (CONT'D)

Type 3	no specific age	unilateral facial enlargement	varies from normal anatomy of condyle; usually presenting as condylar enlargement	benign tumors, eg, osteoma, neurofibroma, giant cell tumor, fibrous dysplasia, chondroma, chondroblastoma, arteriovenous malformation	treatment dependent on pathology and size	as soon as indicated for the specific pathology
Type 4	no specific age	unilateral facial enlargement	varies from normal anatomy of condyle; usually presenting as condylar enlargement with lytic lesions	malignant tumors, eg, chondrosarcoma, multiple myeloma, osteosarcoma, metastatic lesion, Ewing sarcoma	treatment dependent on malignant pathology	as soon as possible if surgery is indicated

Abbreviations: CH, condylar hyperplasia; F, female; M, male; MRI, magnetic resonance imaging; TMJ, temporomandibular joint.

Wolford, Movahed, and Perez: Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

cause enlargement of the mandible, but not as a direct result of condylar enlargement unless the pathology originates in the mandibular condyle, when it would be classified as CH type 3.

Wolford et al[4] introduced a classification differentiating horizontal from vertical growth vectors that are usually related to specific but different mandibular condylar pathologic conditions. CH type 1 results in a predominantly horizontal vector of mandibular growth (bilateral or unilateral). CH type 2 is an abnormal unilateral excessive vertical growth of the mandible usually accompanied by unilateral compensatory downward growth of the maxilla. The present classification is an updated version more inclusive of pathologies causing CH (Table 1). This report defines the 4 basic categories of CH, with a focus predominantly on the common forms of CH (types 1 and 2) in relation to the clinical, imaging, growth, and histologic characteristics, effects on the jaws and facial structures, and treatment protocols that are highly predictable in the elimination of the pathology and provide optimal treatment outcomes. The classification also reflects the occurrence rate, with CH type 1A being the most common pathology and CH type 4 the rarest.

Brief Description of the CH Classification

CH type 1: Onset usually occurs during puberty; an accelerated and prolonged growth aberration of the "normal" condylar growth mechanism causes condylar and mandibular elongation (prognathism); 60% of patients are female; growth is self-limiting, usually ending by the early to mid-20s, and can occur bilaterally (CH type 1A) or unilaterally (CH type 1B).

CH type 2: A unilateral condylar enlarging pathology is caused by an osteochondroma; it can develop at any age (although 68% are initiated during the second decade); it occurs predominantly in female patients (76%) and with a vertical overgrowth of the mandible. One growth vector causes predominantly vertical elongation and enlargement of the condylar head and neck (CH type 2A) and the other form also has a horizontal exophytic tumor growth off of the condyle (CH type 2 B).

CH type 3: These are other benign tumors causing CH.

CH type 4: These are malignant tumors that originate in the condyle, causing enlargement.

There have been previous classifications applied to CH. Obwegeser and Makek[5] proposed a classification system in 1986 describing 2 different types of CH

FIGURE 1. Case 1. *A to D,* This 15-year-old patient reported a relatively normal jaw and occlusal relation at 11 years, but over the past 3 years developed a Class III skeletal and occlusal relation. The mandible was growing at an accelerated rate secondary to condylar hyperplasia type 1A. This patient was treated in a single surgical stage with *1)* bilateral high condylectomies and articular disc repositioning with Mitek anchors; *2)* bilateral mandibular ramus osteotomies; *3)* multiple maxillary osteotomies; *4)* removal of impacted third molars; *5)* bilateral partial inferior turbinectomies; and *6)* rhinoplasty. *E to H,* At 2 years after surgery, the patient maintains good facial symmetry and balance and a stable Class 1 skeletal and occlusal relation. **(Fig 1 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

anomalies that correlate with the present classification. *Hemimandibular elongation* (HE) described a deformity created by horizontal displacement of the mandible and chin toward the unaffected side without significant vertical elongation (this correlates with the present CH type 1). They stated this entity can be bilateral (CH type 1A) or unilateral (CH type 1B). The other anomaly was *hemimandibular hyperplasia* (HH) that included enlargement on one side of the mandible as a tridimensional anomaly, involving the condyle, ramus, and body, creating a unilateral vertical elongation deformity with the maxilla usually following the mandible, creating a transverse cant in the occlusion and jaws (CH type 2). The investigators made a distinction between "exclusive hyperplasia of the condyle" (CH type 2A) and "osseous tumor and exostosis of the condyle" (CH type 2B). The investigators stated that a mixed form between HE and HH can occur.

Nitzan et al[6] described CH as a unilateral disorder in which the pathology occurs at the head of the condyle, creating facial asymmetry in the vertical or horizontal direction or a combination of both.

CH TYPE 1

CH type 1 occurs in adolescence and the pathologic process is usually initiated during the pubertal growth phase, suggesting a hormonal etiology. Approximately one third of cases may be genetically related, but the other two thirds occur spontaneously.[7] The gender distribution in the present study was 60% female.[8] Normal jaw growth is usually 98% complete in girls at 15 years of age and in boys at 17 to 18 years of age.[9] CH type 1 is an accelerated and excessive growth of the "normal" condylar growth mechanism creating overgrowth of the mandible predominantly in a horizontal vector (mandibular prognathism), with the

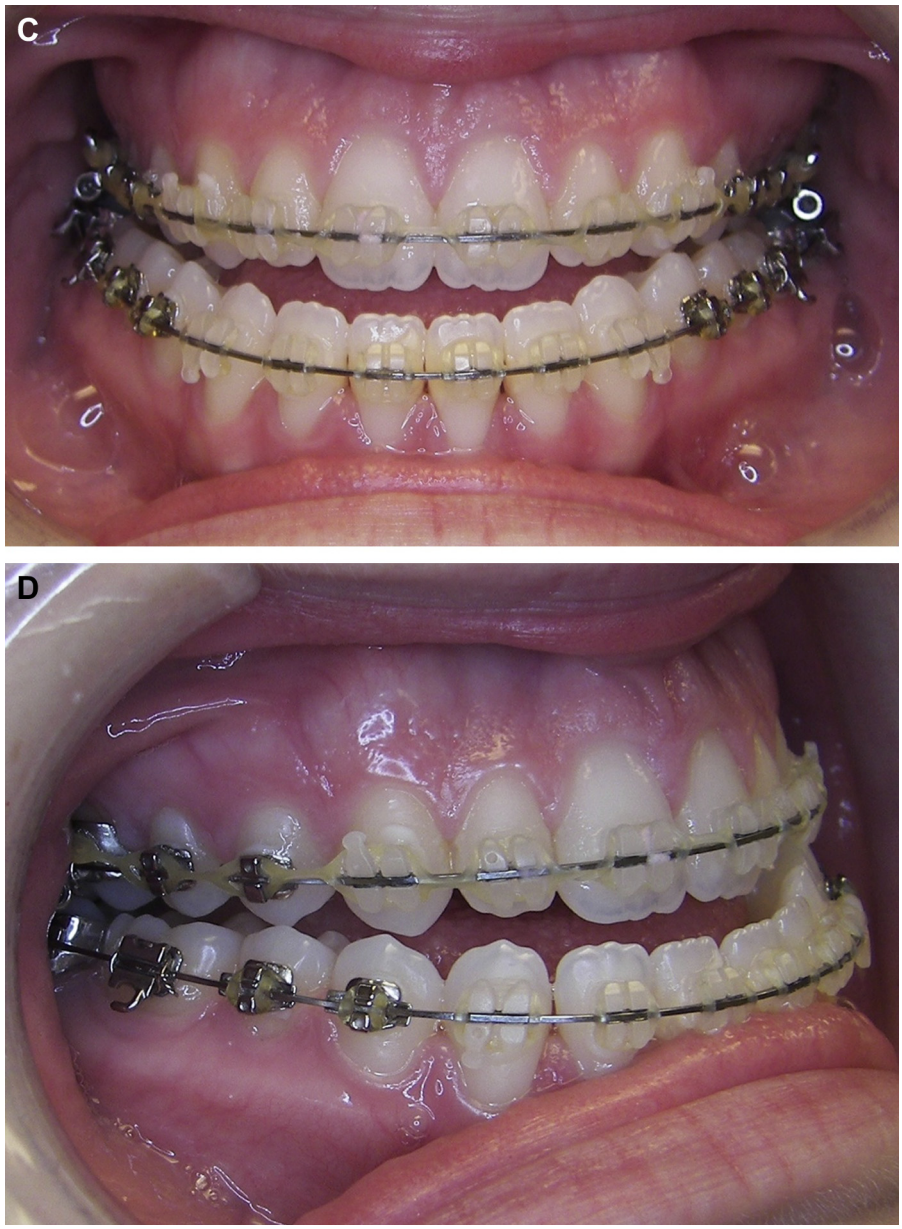


FIGURE 1 (cont'd). (Fig 1 continued on next page.)

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

growth often continuing into the patient's early to mid-20s, but CH type 1 is a self-limiting growth aberration. Patients may begin with a Class I occlusal and skeletal relation as they enter their pubertal growth phase and grow into a Class III skeletal and occlusal relation (Figs 1A-D, 2A) or begin as a Class III and develop into a worse Class III relation. Condylar and mandibular growth can be accelerated bilaterally (CH type 1A) or unilaterally (CH type 1B) in a horizontal or, uncommonly, a vertical vector. The increase in the mandibular growth rate occurs in the condyle but causes elongation of the condylar head and neck and mandibular body, which leads to development of a Class III skeletal and occlusal relation and dental compensations, in which the mandibular incisors can become lingually inclined and the maxillary incisors overangulated.[4,8]

Not all prognathic mandibles are caused by CH; only those exhibiting accelerated, excessive mandibular growth that continues beyond the normal growth years are caused by CH. Differential diagnosis includes *1)* Class III skeletal relation with normal mandibular and maxillary growth, *2)* deficient maxillary growth with a normally growing mandible, or *3)* CH type 1 with or without deficient maxillary growth. Differentiation must be clarified between CH type 1 and maxillary growth deficiency (maxillary hypoplasia). Maxillary hypoplasia also can result in a Class III

FIGURE 1 (cont'd). (Fig 1 continued on next page.)

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

occlusal and skeletal relation in the presence of a normally growing mandible with a resultant progressive worsening occlusion through the normal facial growth process (usually completed at 15 yr for girls, 17 to 18 yr for boys) with stability of the jaw and occlusal relation after that time. Maxillary hypoplasia creating the Class III relation is usually evident years before the pubertal growth phase. Maxillary hypoplasia also can be present, with accelerated mandibular growth (CH type 1) creating a more extreme dentofacial deformity. However, the accelerated mandibular growth is usually not evident until initiated during pubertal growth. Deviated mandibular prognathism is usually related to bilateral CH type 1A, where one side is growing faster than the opposite side, or CH type 1B, where only a unilateral condyle is involved.

Analysis of serial lateral cephalometric and lateral tomograms that include the temporomandibular joint (TMJ), ramus, and mandibular body should allow determination of the growth rates of the maxilla and mandible to differentiate the source of the Class III occlusal and skeletal relation. In CH type I, growth can usually be determined by worsening functional, esthetic, skeletal, and occlusal changes with serial assessments (preferably at 6- to 12-month intervals) consisting of clinical, dental model, and radiographic evaluations.[4,8]

During pubertal growth, the normal yearly growth rate of the mandible measuring from condylion to point B is 1.6 mm for girls and 2.2 mm for boys.[9] Growth significantly greater than the normal rate indicates accelerated growth likely related to CH type 1. With asymmetric condylar growth, the amount of growth may be more difficult to determine from the lateral cephalogram because of the mandible shifting toward the side with less growth, essentially hiding some of the forward growth and elongation on the faster growing side. Thus, using lateral TMJ tomograms that include the mandibular body, ramus, and condyle will allow superimposition of the body, ramus, and posterior teeth to analyze the amount of condylar growth over time for each side. A horizontal mandibular growth vector extending beyond the normal growth years will likely be CH type 1 and the growth can continue into the mid-20s until cessation.

Bone scintigraphy of the TMJs may detect active growth in the more rapidly growing CH type 1 conditions, but in most cases it will be nondiagnostic in determining active CH type 1 growth. Healthy growing TMJs

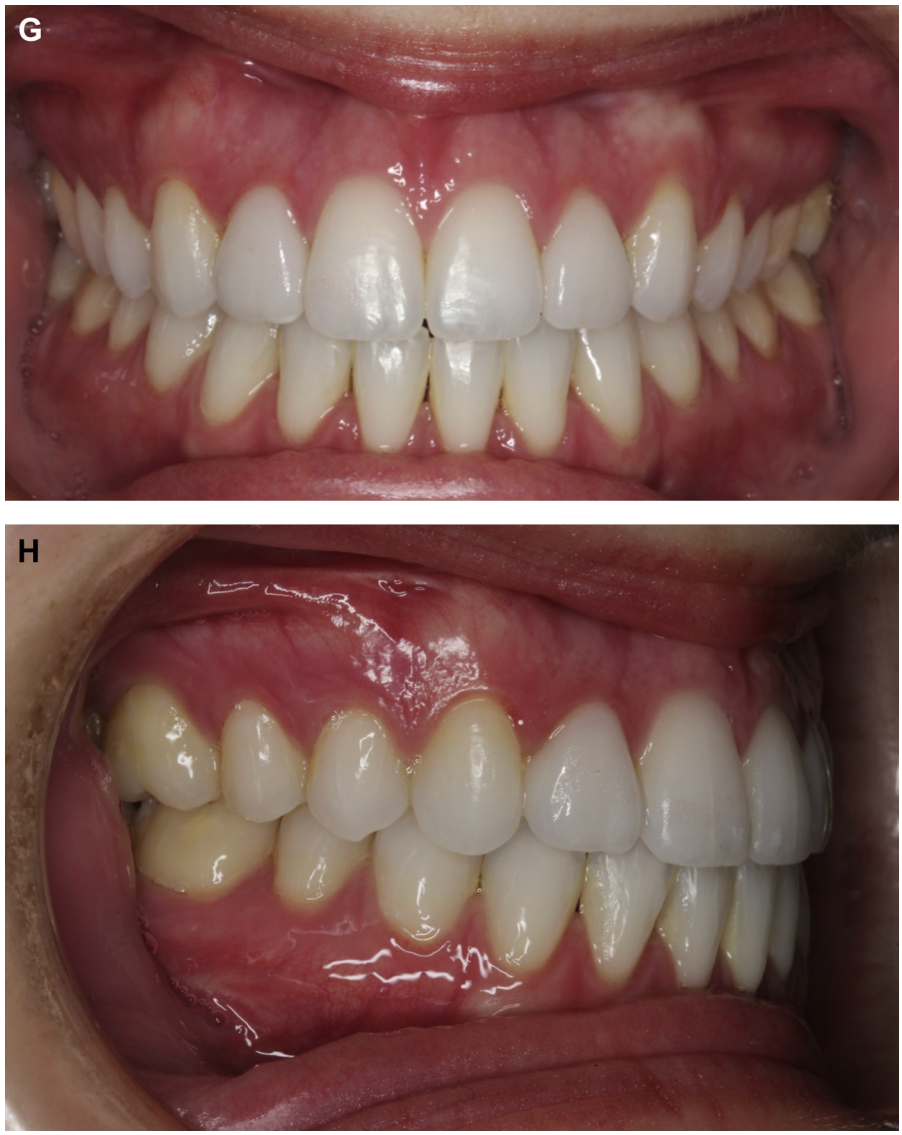


FIGURE 1 (cont'd).

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

normally have some uptake at scintigraphy. The growth rate of CH type 1 is not growing at a tumorous rate, as seen in CH type 2, but only somewhat faster than the normal condylar growth rate; thus, it is usually difficult to differentiate CH type 1 from normal growth, particularly if both joints are involved. In unilateral cases, it also may be difficult to determine an increased uptake on the involved side, particularly if the contralateral TMJ develops a displaced disc and associated arthritic changes (a common contralateral development) because this contralateral TMJ also may have a slight increased uptake. Wrist and hand radiographs provide no insight to CH type 1 because there is no correlation between normal physiologic development and the pathologic process affecting the mandibular condyles.

Histology

A normal condyle is approximately 15 to 20 mm long mediolaterally and 8 to 10 mm wide anteroposteriorly.[10] Histologically, the normal condyle is composed of the following tissue layers, beginning from the outside and progressing inward: fibrous connective tissue layer forming the articulating surface; undifferentiated mesenchyme proliferation layer; intermediate layer; cartilage layer; compact bone; and spongy bone. In CH type 1, histology of the affected condyle commonly resembles a normally growing condyle without any notable pathologic abnormalities (Fig 3A,B). In some cases, the proliferative layer may exhibit greater thickness in some areas. The activity of the proliferative layer may regulate the rate at which the condyle and condylar neck (which is formed from the condyle by remodeling)

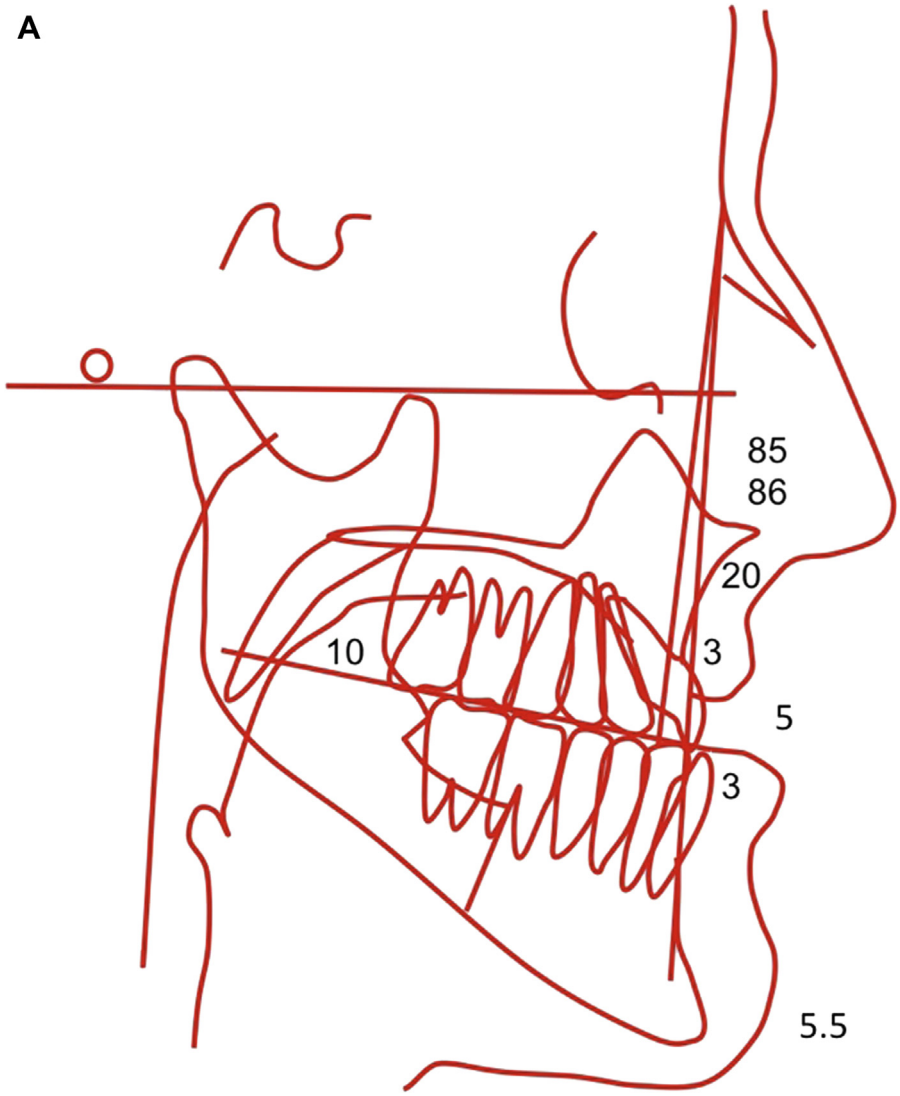


FIGURE 2. Case 1. *A*, Lateral cephalometric radiograph shows the skeletal Class III relation. **(Fig 2 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

will grow. In normal condyles, the formation of cartilage from the proliferative layer and the replacement of cartilage by bone ceases by approximately 20 years of age. The marrow cavity is entirely occluded from the remaining cartilage by the closure of the bone plate.[11] The inability of this plate to close in the presence of an active proliferative cartilage layer may be a major etiologic factor in CH type 1 and may correlate to the authors' observation that cessation of growth related to CH type 1 may not occur until the early to mid-20s.[4,8] Clinically, the authors have observed an increased vertical height of condyle covered by the cartilaginous cap compared with the normal condyle. Conditions that initiate excessive accelerated mandibular growth after the pubertal growth phase (15 yr of age for girls, 17 to 18 yr of age for boys) are most often related to an osteochondroma (CH type 2) or other types of proliferative condylar pathology (CH type 3). As the osteochondroma enlarges, the bone producing islands of cartilage may become further separated from each other so that in the more mature tumors the cartilaginous islands become more difficult to identify histologically.

CH TYPE 1A

Clinical Characteristics

Patients with bilateral active CH type 1A (Fig 1A-D) may have some or all of the following characteristics: *1)* development or progressive worsening of mandibular prognathism; *2)* worsening Class III malocclusion; *3)* anterior and posterior crossbites; *4)* dental compensations, including decreased angulation of lower

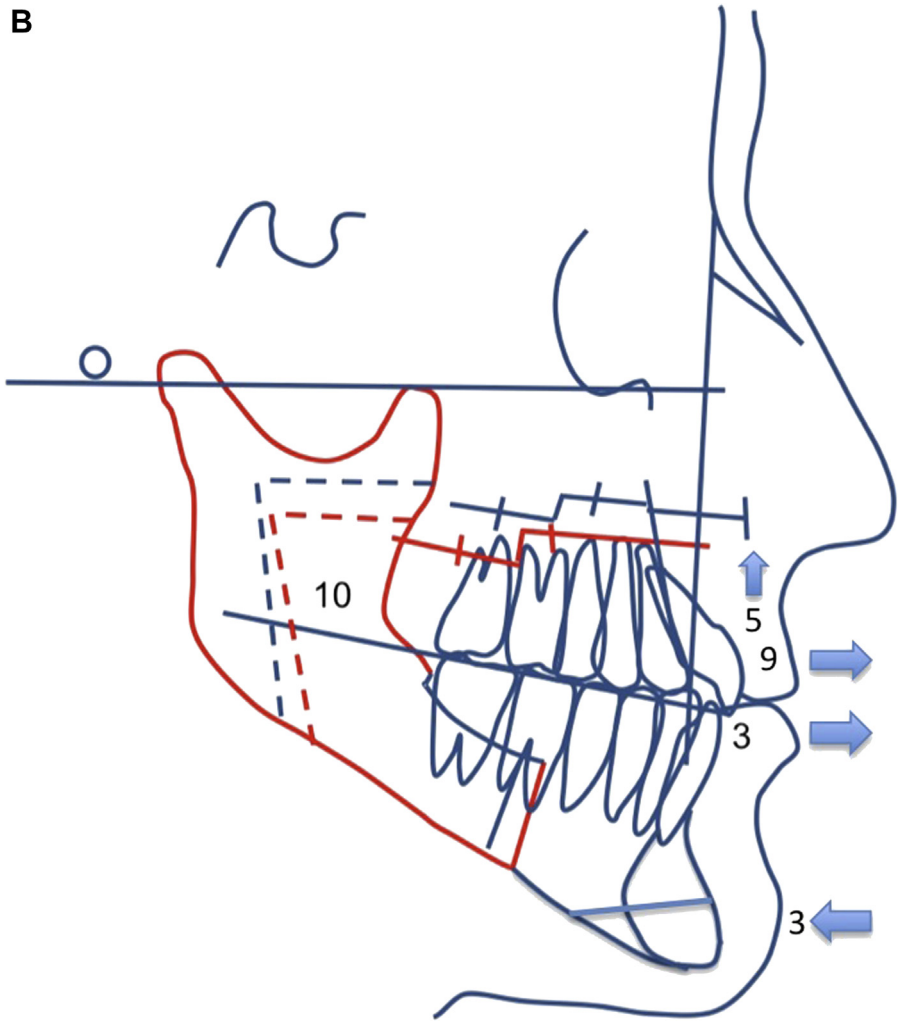


FIGURE 2 (cont'd). *B*, The surgical treatment objectives included *1)* bilateral high condylectomies; *2)* disc repositioning; *3)* bilateral mandibular ramus osteotomies; *4)* multiple maxillary osteotomies; and *5)* rhinoplasty to establish good facial balance and occlusion.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

incisors and overangulation of maxillary incisors; *5)* facial shape more triangular and tapered, often with weakly defined mandibular angles; *6)* masseter muscles have less bulk than normal; and *7)* relatively asymptomatic for TMJ symptoms in patients with symmetric growth of the mandible. In asymmetric cases in which one side grows faster than the other, patients may develop *1)* facial asymmetry, with the mandible shifting toward the slower growing side; *2)* anterior and unilateral posterior crossbite on the slower growing side; *3)* greater Class III occlusion on the faster growing side; *4)* displaced articular disc in the TMJ on the slower growing side and sometimes on the faster growing side; and *5)* TMJ symptoms, such as clicking, TMJ pain, headaches, masticatory dysfunction, or ear symptoms.

Imaging

Lateral cephalometric and tomographic analyses (Fig 2A) will show the following characteristics: *1)* Class III skeletal and occlusal relation, except in patients with high occlusal plane angulation, where the occlusion will be Class III but the skeletal relation could be closer to Class I; *2)* the condylar head and neck are elongated, but the top of the condyle will have a smooth, relatively normal-appearing morphology (Fig 4D-F); *3)* in the coronal view, the top of the condyle will appear more rounded than normal; *4)* the mandibular body is elongated; *5)* the gonial angle may be more obtuse; *6)* the vertical height of the posterior mandibular body may be decreased; *7)* the anteroposterior (AP) thickness of the symphysis and alveolus may be narrower; *8)* the mediolateral width and AP dimension of the rami may be narrower compared with normal; *9)* the cranial base length tends to be decreased and the cranial base angulation (sella to nasion, and nasion to basion) tends to be increased compared with normal; and *10)* the slope of the posterior border of the ramus compared with the Frankfort horizontal plane

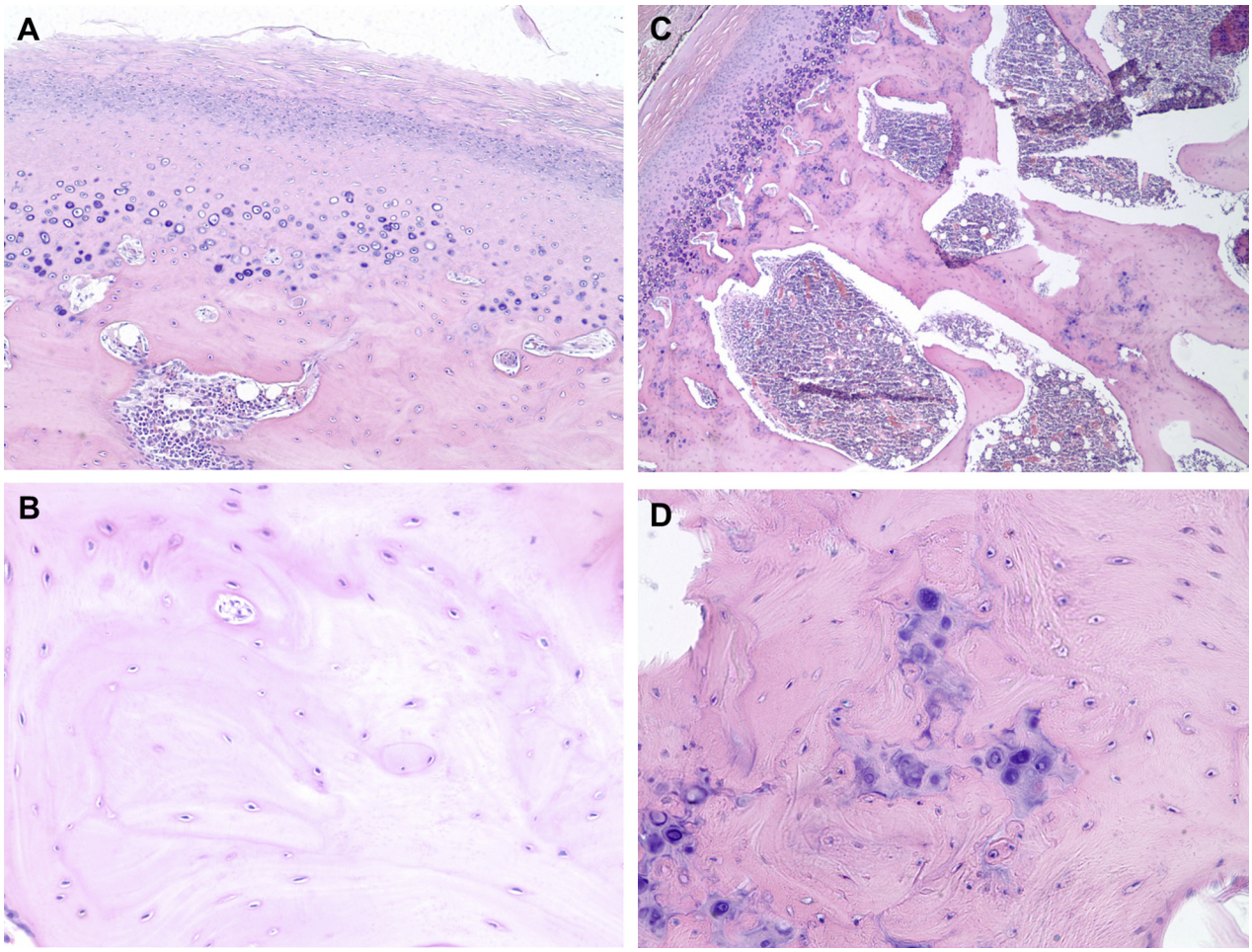


FIGURE 3. Histology using hematoxylin and eosin staining. *A*, *B*, Condylar hyperplasia type 1 (magnifications, ×40 and ×80) may appear very similar to a normally growing condyle without any significant pathologic abnormalities. In some cases, the proliferative layer may exhibit an increased thickness and some subarticular bone hyperplasia. *C*, *D*, Condylar hyperplasia type 2 (magnfications, ×40 and ×80) shows a cartilaginous cap that may not be too dissimilar from the normally growing cartilage, although there can be areas of increased thickness. There are endochondral ossification and cartilaginous islands in the subcortical bone. These deeper regions have isolated aggregates of chondrocytes that resemble a growth plate with endochondral ossification and transition to cancellous bone.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

may be angled forward greater than normal, particularly in the faster growing cases.

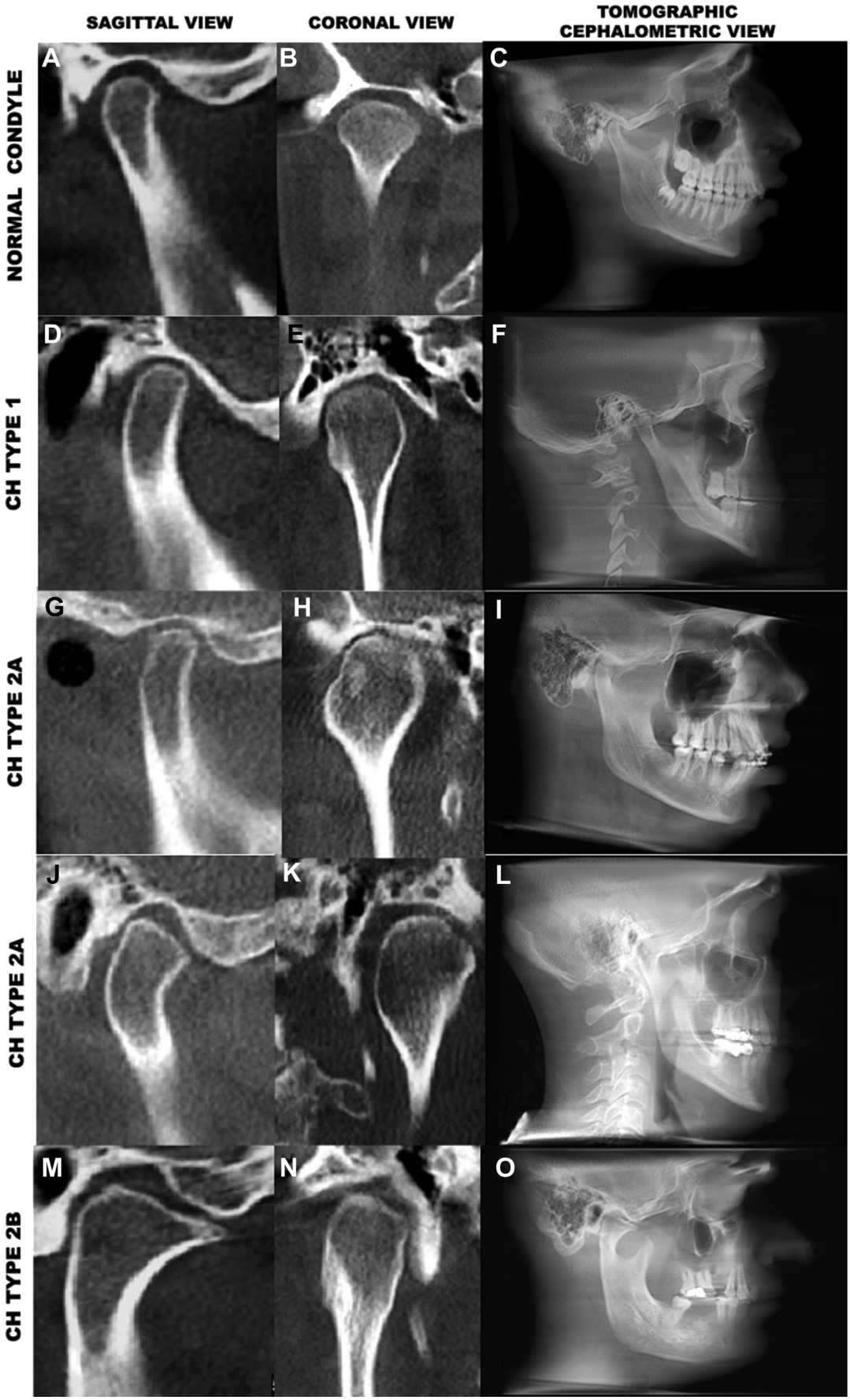
At magnetic resonance imaging (MRI; Fig 5A), the articular discs are commonly thin and may be difficult to identify. Occasionally, the articular discs can be posteriorly displaced (Fig 5B). Posterior disc displacement occurs only in these conditions when there is an increased rate of vertical condylar growth that is faster than the rate of upward migration of the posterior ligament attachment of the disc, thus pulling the disc posterior to the condyle as the condyle continues to grow at an accelerated rate. In asymmetric CH type 1A, disc dislocation can occur unilaterally (greater risk on the slower growing side; Fig 5C) or bilaterally.

Treatment Protocol

Treatment of CH type 1 depends on whether the growth is still active or arrested. Because CH type 1 is self-limiting relative to growth, patients in their mid-20s or older will not have further jaw growth related to CH type 1, so routine orthognathic surgical procedures can usually be performed to correct the

Figure 4. *A to C*, Normal condyle and temporomandibular joint anatomy. *D to F*, Condylar hyperplasia type 1 shows an increased vertical length of the condylar head and neck. In the coronal view, the crown of the condyle may be more rounded than a normal condyle. *G to I*, Condylar hyperplasia type 2A shows increased vertical height of the condylar head and neck. *H*, with greater deformity in the condyle observed in the coronal view. *J to L*, Condylar hyperplasia type 2A with a larger condyle but with a predominantly vertical growth vector. *M to O*, Condylar hyperplasia type 2B shows the exophytic growth from the head of the condyle. In the coronal view, the abnormal growth may not be seen. *O*, There is significant vertical growth and enlargement of the right mandibular condyle, neck, ramus, and body of the mandible. CH, condylar hyperplasia.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.


SAGITTAL VIEW
CORONAL VIEW
TOMOGRAPHIC CEPHALOMETRIC VIEW
NORMAL CONDYLE
CH TYPE 1
CH TYPE 2A
CH TYPE 2A
CH TYPE 2B
A
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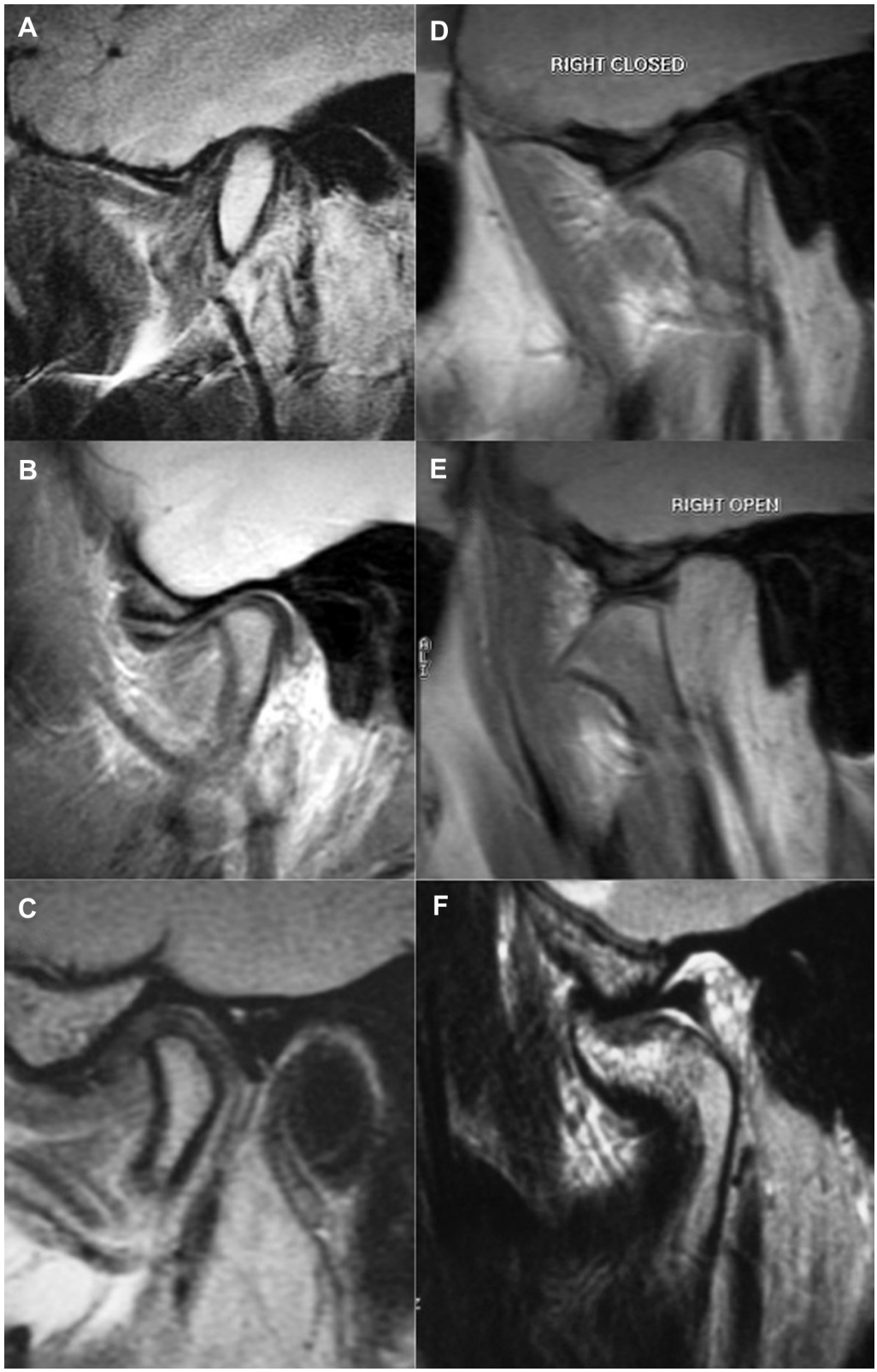

FIGURE 5. Magnetic resonance imaging evaluation. *A*, Condylar hyperplasia type 1 temporomandibular joint with a thin disc that is difficult to identify. *B*, Posteriorly displaced discs predominantly occur in condylar hyperplasia type 1 and occasionally in type 2A. *C*, In condylar hyperplasia type 1 with deviated prognathism and in condylar hyperplasia type 2, the contralateral joint may show a displaced articular disc. *D*, *E*, In condylar hyperplasia type 2B (magnetic resonance image from case 3), the articular disc is seen in position in the closed and open views. *F*, In condylar hyperplasia type 2B, even with large exophytic growth development, the articular disc will commonly be in place. In this particular case, notice the significant downward displacement of the condyle out of the fossa created by the large exophytic tumor growth.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

dentofacial deformity and malocclusion. However, if the TMJ discs are displaced and salvageable, the discs may require repositioning. If the patient is still a teenager or even in the early 20s, the growth process can be active and progressive. If there is active growth and only the orthognathic surgery is performed, then there will be predictable relapse, with the mandible growing out into a Class III skeletal and occlusal relation.[4,8] If growth is active, then there are 2 predictable treatment options.

Option 1. The Wolford surgical protocol (Fig 6) for active CH type 1A includes *1)* a high condylectomy removing 4 to 5 mm of the top of the condylar head on the involved side (both sides for bilateral CH), including the medial and lateral pole areas, which will arrest any further AP growth of the mandible; *2)* repositioning the articular discs using the Mitek anchor (Mitek Products, Inc, Westwood, MA) technique (Fig 7A-D); *3)* performing the appropriate orthognathic surgical procedures, including bilateral mandibular ramus osteotomies and maxillary osteotomies if indicated (Fig 2B); and *4)* additional ancillary procedures as indicated (third molar removal, genioplasty, turbinectomies, rhinoplasty, etc). This protocol will provide predictable and stable results (Fig 1E-H). These procedures can be performed in 1 operation or divided into 2 or more operations depending on the surgeon's skills and experience, but the TMJ surgery should be performed first.

Option 2. Surgery is delayed until growth is complete and then only orthognathic surgery is performed. However, because these cases often continue to grow into the mid-20s, the surgery would be delayed until it is confirmed that the growth has stopped. The longer the abnormal growth is allowed to proceed, the worse the facial deformity, asymmetry, and dental compensations will become, affecting the dentoskeletal development and producing excessive soft tissue development. This may increase the difficulties in obtaining an optimal functional and esthetic result, in addition to the adverse effects on the occlusion, dental compensations, mastication, speech, and psychosocial development. If the TMJ articular discs are displaced, then TMJ surgery may still be indicated to reposition the discs.

The Wolford surgical protocol for treating active CH type 1A is a highly predictable treatment that will stop the abnormal growth and allow completion of the orthognathic surgery at the same operation, with long-term stable functional and esthetic outcomes.[4,8] Except in select cases, surgical correction of CH type 1A requiring 2-jaw surgery should be deferred until at least 14 years of age in girls and 16 years of age in boys, when normal maxillary and mandibular growth are closer to completion. Because no further AP growth of the mandible and maxilla can be expected after high condylectomies and Le Fort I osteotomies, residual maxillary vertical growth results in a downward and backward rotation of the maxillomandibular complex, but the occlusion should remain stable.[12-15] If only the high condylectomies and mandibular osteotomies are performed (no maxillary osteotomies), then surgery should be delayed until 15 years of age for girls and 17 to 18 years of age for boys, because the maxilla can continue to grow in the AP and vertical directions, potentially developing a Class II occlusion if surgery is performed earlier because of cessation of mandibular growth from the high condylectomies.

In comparative studies of active CH type 1, Wolford et al[4,8] divided patients into 2 groups; group 1 patients were treated with conventional

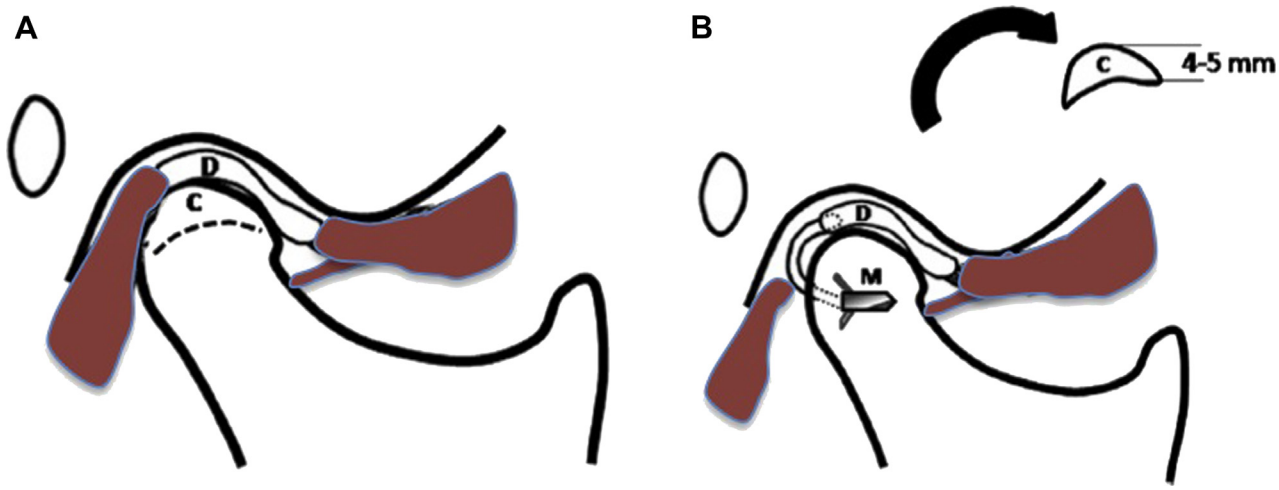

FIGURE 6. *A*, Schematic illustration shows the level of high condylectomy removing the top 4 to 5 mm for treatment of condylar hyperplasia type 1. *B*, For condylar hyperplasia type 1 in active growth, a high condylectomy will arrest any further anteroposterior mandibular growth. The articular disc is repositioned and stabilized on the condyle with a Mitek anchor.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

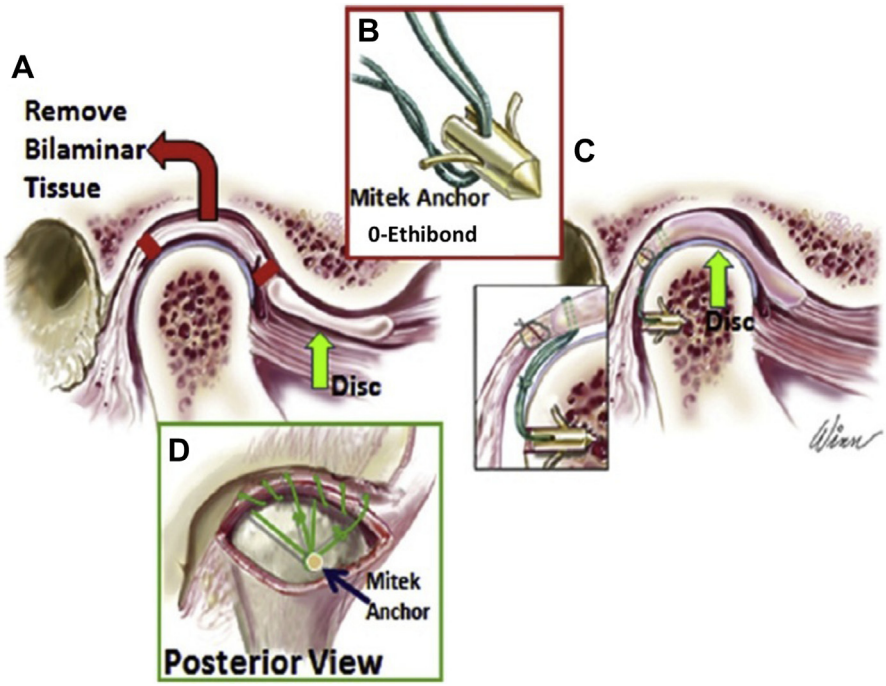

FIGURE 7. The Mitek anchor system is used to stabilize the articular disc in position after a high or low condylectomy on the ipsilateral side and reposition the contralateral articular disc if dislocated. *A*, Excessive bilaminar tissue is removed and the disc is mobilized. *B*, The Mitek anchor is threaded with 2 strands of 0-Ethibond suture (Ethicon, Inc, Somerville, NJ). *C*, The anchor is inserted in the posterior head of the condyle and sutures are attached to the disc. *D*, Posterior view showing the anchor location and suturing technique.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

orthognathic surgery only and the mandible continued to grow after surgery, whereas group 2 patients also underwent high condylectomies and orthognathic surgery and had resultant stable Class I occlusal and skeletal outcomes. These studies confirm that high mandibular condylectomies stop mandibular AP growth, providing a means to achieve predictable and stable treatment outcomes.

CH TYPE 1B

Clinical Characteristics

CH type 1B (Fig 8A-F) is the unilateral form with the following common characteristics: *1)* usually grows in a horizontal direction, although there occasionally can be a vertical directional component; *2)* the left-to-right vertical facial heights are usually relatively symmetric, but the ipsilateral side sometimes can be vertically longer; *3)* the mandible becomes prognathic and deviated toward the contralateral side; *4)* increased worsening of the ipsilateral Class III occlusion while the contralateral side usually remains Class I; *5)* crossbites develop anteriorly and on the contralateral side; *6)* the mandibular dental midline and chin shift off the facial midline toward the contralateral side; *7)* the mandible continues to grow more asymmetric beyond the normal growth years but usually will complete its growth in the early to mid-20s; and *8)* unilateral CH may cause articular disc displacement, particularly on the contralateral side, creating typical TMJ symptoms.

Imaging

Radiographic imaging for patients with CH type 1B (Figs 4D-F, 9A) commonly show the following features: *1)* mandibular deviated prognathism; *2)* mandibular asymmetry primarily in a transverse direction (however, there can occasionally be a vertical component) with the mandible and chin shifted toward the contralateral side; *3)* condylar head and neck on the involved side are longer compared with the contralateral side; *4)* the ipsilateral body of the mandible may be more bowed and the contralateral side may be more flat, creating significant asymmetry in the axial plane of the mandible; and *5)* bone scans may or may not be of value in diagnosing CH type 1B because the growth rate, although accelerated, is still relatively slow but continuous on the involved side. In addition, if there is a displaced disc and mild arthritis on the contralateral side, there may not be much differentiation in the amount of isotope uptake comparing one side with the other. Serial radiographs (lateral cephalograms, cephalometric tomograms, etc), dental models, and clinical evaluations are usually the best diagnostic methods to determine if the growth process is still active.

MRI may show a displaced articular disc on the contralateral side with mild arthritis (Fig 5C) and

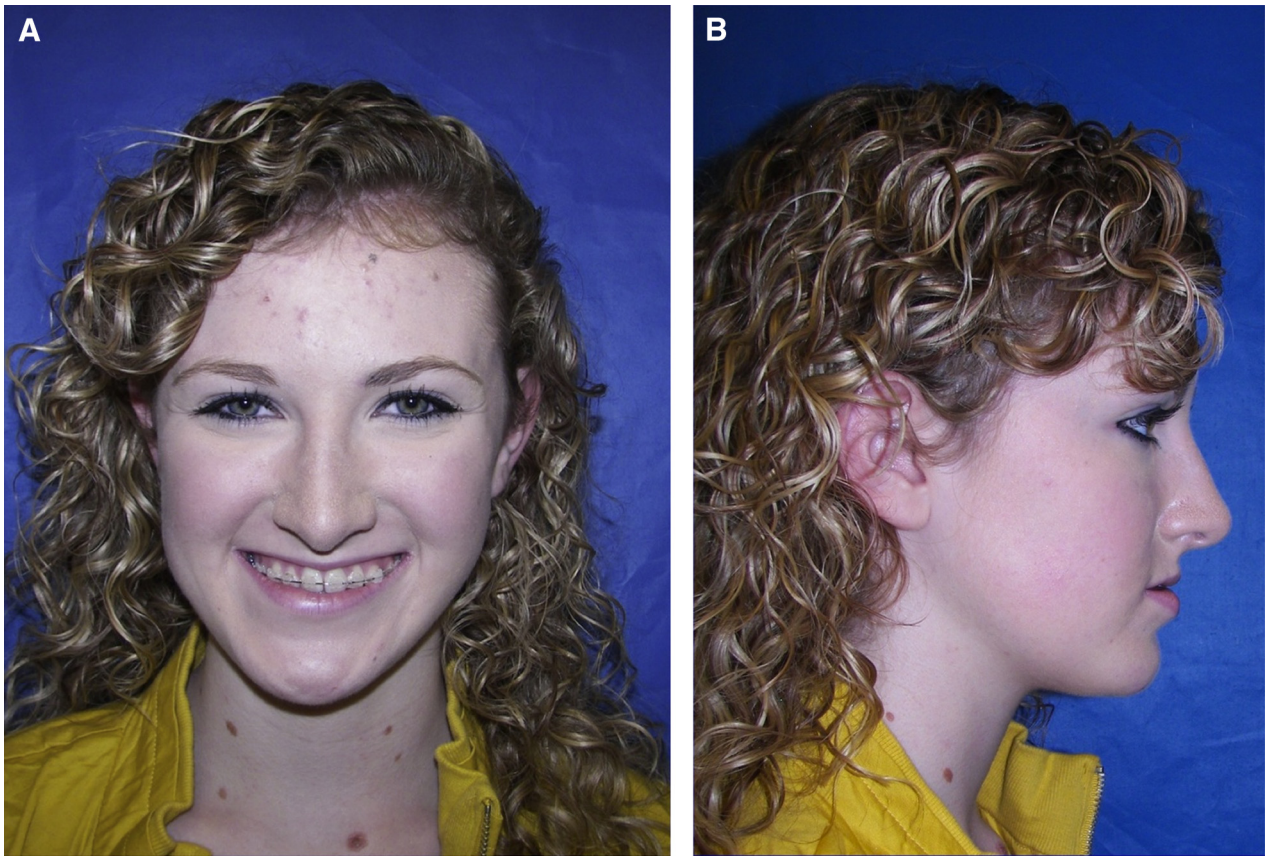

FIGURE 8. Case 2. *A to D*, A 15-year-old female patient with left-sided condylar hyperplasia type 1B, bilateral displaced articular discs, and significant temporomandibular joint pain and headaches. She was treated in a single surgical stage with *1)* unilateral left high condylectomy; *2)* bilateral disc repositioning; *3)* bilateral ramus osteotomies; *4)* multiple maxillary osteotomies; *5)* nasal turbinectomies; and *6)* third molar removal (×4). *E to H*, At 3 years after surgery, the patient has good facial balance, stable skeletal and occlusal relations, and is pain free. **(Fig 8 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

sometimes disc displacement on the ipsilateral side of CH type 1B. The disc on the ipsilateral side is often thinner than a normal disc, making it sometimes difficult to identify at MRI. Occasionally, there can be a posteriorly displaced disc on the ipsilateral side (Fig 5B).

Treatment Protocol

The treatment options for CH type 1B are similar to those for CH type 1A, in which patients with confirmed nongrowth can be treated with traditional orthognathic surgery. The TMJs need to be addressed only if the articular discs are displaced; a relatively common occurrence is facial asymmetries. If active growth is confirmed, then there are 2 options for treatment.

Option 1. The Wolford surgical protocol for active CH type 1B consists of *1)* unilateral high condylectomy to arrest the aberrant condylar growth and disc repositioning (Figs 6, 7); *2)* contralateral disc repositioning if indicated; *3)* orthognathic surgical procedures, often requiring 2-jaw surgery to optimize the functional and esthetic outcomes; and *4)* other ancillary procedures as indicated. This protocol predictably stops ipsilateral mandibular growth and provides highly predictable and stable outcomes, with normal jaw function and good esthetics (Figures 8E to H).[4,8]

Option 2. Surgery is delayed until growth is complete, which could be in the early to mid-20s, and then only orthognathic surgery is performed. However, the longer the abnormal growth is allowed to proceed, the worse the facial deformity, asymmetry, occlusion, and dental compensations will become, in addition to warping of the mandible and ipsilateral excessive soft tissue development. This will increase the difficulties in obtaining optimal functional and esthetic results, in addition to the adverse effects on occlusion, dental compensations, mastication, speech, and psychosocial development. If the TMJ articular discs are displaced (a common occurrence), these patients also may have TMJ pain, headaches, and myofascial pain. TMJ surgery may still be indicated to reposition the discs, stabilize the joints, and eliminate the pain factors.

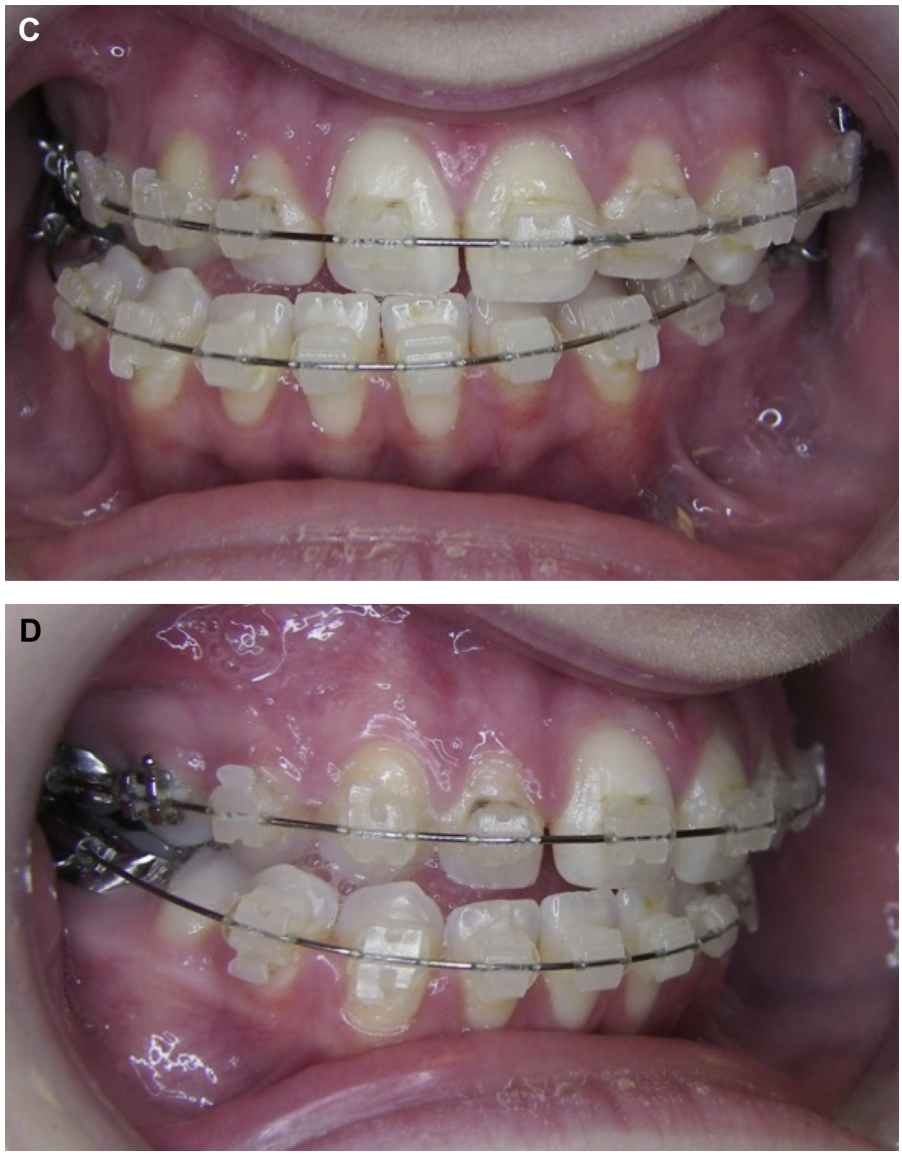

FIGURE 8 (cont'd). (Fig 8 continued on next page.)

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Surgery for CH type 1B cases should be deferred until 15 years of age for girls and 17 to 18 years of age for boys. However, the severity of the deformity may warrant surgery at a younger age. If the ipsilateral high condylectomy is performed when normal jaw growth is still occurring (<15 yr of age in girls and <17 to 18 yr of age in boys), then there is the risk of the unoperated contralateral condyle continuing with normal growth and shifting the mandible toward the ipsilateral side until normal growth cessation. In CH type 1B, the contralateral TMJ articular disc is commonly displaced. Therefore, disc repositioning with the Mitek anchor technique would be indicated but without the high condylectomy. However, if surgery is indicated at a younger age during active growth, then a high condylectomy also can be performed on the contralateral side so that the mandible will not grow and will remain symmetrical.

CH TYPE 2

CH type 2 is a unilateral mandibular condylar enlargement caused by an osteochondroma that vertically lengthens the ipsilateral mandible, can shift it toward the contralateral side, and is not self-limiting in relation to growth. The growth rate for this pathology varies from slow to moderate, but some cases can have a more rapid growth rate. Confirmation of the condylar pathology usually requires histologic

FIGURE 8 (cont'd). (Fig 8 continued on next page.)

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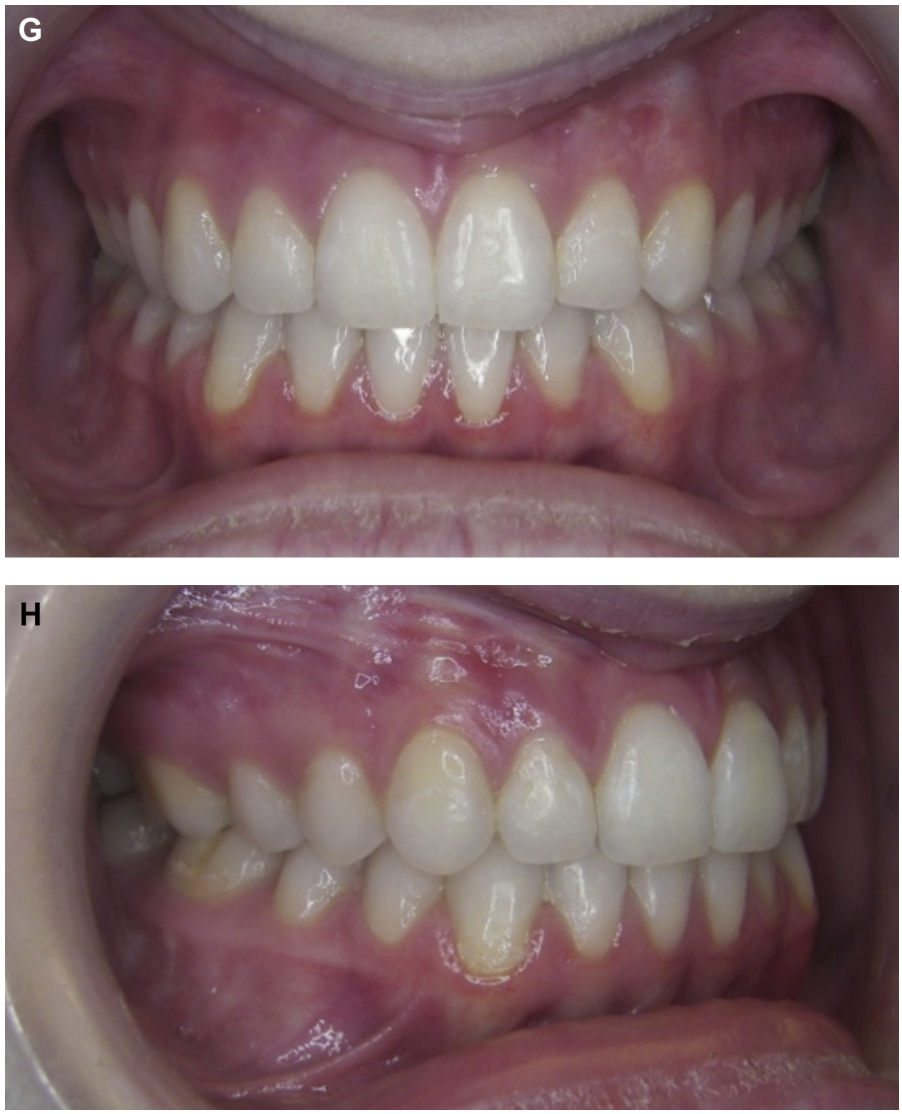
assessment. Osteochondromas are one of the most common benign tumors of bone, representing approximately 35 to 50% of all benign tumors and 8% to 15% of all primary bone tumors, and the most common tumor of the mandibular condyle.

Histology

Osteochondromas (CH type 2) include a cartilaginous cap similar to that seen in a normal growth cartilage, endochondral ossification, cartilaginous islands in the subcortical bone, and a marrow space contiguous with the underlying bone (Fig 3C,D). It has been reported that the cartilaginous cap may be 1 cm or greater in thickness in the axial skeleton. However, it tends to be thinner in the maxillofacial region and may even be absent in long-standing cases. Gray et al[16,17] reported that the bony trabeculae are often thickened and irregular, resulting in a consistently large volume of trabecular bone and a larger than normal percentage of surfaces covered in osteoids. They also pointed out the presence of an uninterrupted layer of undifferentiated germinating mesenchymal cells, hypertrophic cartilage, and islands of chondrocytes in the subchondral trabecular bone and they made the direct correlation between the scintigraphic activity and the frequency of cartilage islands at deep in the trabecular bone. The cartilage islands are mini growth centers producing bone, causing enlargement of the condyle.

Clinical Characteristics

Common clinical features of CH type 2 (Fig 10A-D) include: *1)* it develops at any age, but for most cases, in the second decade (68% of cases); *2)* it occurs predominantly in female patients (76% of cases); *3)* there is increased unilateral mandibular height involving the condyle, neck, ramus, body, and dentoalveolus of the ipsilateral mandible; *4)* there is increased soft tissue volume on the ipsilateral side of the face, including elongation of the muscles of mastication; *5)* there is a low mandibular plane angle facial-type morphology; *6)* there is chin asymmetry vertically and transversely, with shifting toward the contralateral side; *7)* there is compensatory downward growth of the ipsilateral maxillary dentoalveolus; *8)* there is a lateral open bite on the ipsilateral side, particularly in more rapidly growing pathology (Fig 10C,D); *9)* labial tipping of the mandibular ipsilateral posterior teeth and lingual tipping of the contralateral posterior teeth may occur; *10)* there is a transverse cant in the occlusal plane; *11)* mandibular anterior teeth crowns may be tipped



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toward the ipsilateral side and the long axis of the roots may be angled toward the contralateral side; and *12)* commonly contralateral TMJ arthritis and articular disc dislocation (75% of cases) can occur from the functional overload caused by the ipsilateral pathology accompanied by symptoms, such as clicking, popping, TMJ pain, headaches, etc.

Imaging

Radiographic features will include *1)* an enlarged, elongated, deformed ipsilateral condyle (CH type 2A; Fig 4G-L) and commonly there may be exophytic extensions of the tumor off of the condyle (CH type 2B; Fig 4M-O); *2)* increased AP and mediolateral thickness of the ipsilateral condylar neck; *3)* increased vertical height of the ipsilateral mandibular condyle, neck, ramus, body, symphysis, and dentoalveolus (Fig 11A); *4)* increased vertical height of the ipsilateral maxillary dentoalveolus; *5)* transverse cant in the occlusal plane; *6)* facial asymmetry; *7)* posterior border of the ipsilateral mandibular ramus may be more vertical than normal; *8)* coronoid process is usually normal in size and may be displaced below the zygomatic arch with elongation of the temporalis muscle; *9)* loss of antegonial notching with downward bowing of the inferior border on the mandible; *10)* the inferior alveolar nerve canal may be positioned adjacent to the apices of the teeth but more commonly toward the inferior border of the mandible; and *11)* chin vertically longer on the ipsilateral side and may be prominent in profile.

MRI may show a displaced articular disc on the contralateral side (76% of the cases) and associated arthritic

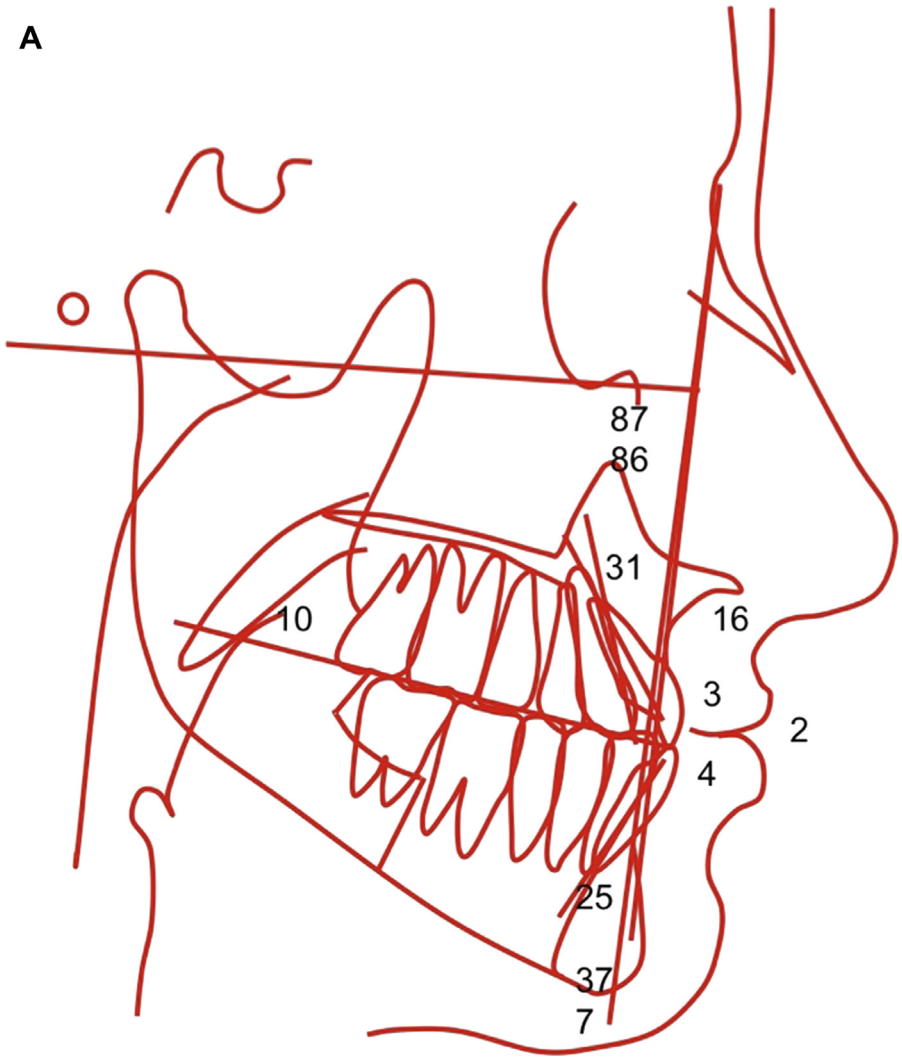


FIGURE 9. Case 2. *A*, Cephalometric analysis does not reflect the severity of this patient's left-sided condylar hyperplasia type 1B because the growth vector is predominantly horizontal, with the mandible deviating off to the right side. **(Fig 9 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

condylar changes (Fig 5C). The disc is commonly in position on the ipsilateral side (Fig 5D-F), although it also can be displaced. Unless the tumor is very slow growing, bone scintigraphy will usually show increased uptake, particularly in the more active tumors.

CH type 2 can be subdivided into 2 primary groups based on tumor morphology. CH type 2A indicates an enlargement of the condylar head and neck with a predominant vertical growth vector of the osteochondroma without significant exophytic tumor development (Fig 4G-L). There can be unevenness or lumpiness on the condyle. CH type 2B indicates exophytic tumor extensions off the condyle, usually forward and medially, with the head becoming significantly enlarged and deformed (Fig 4M-O). The exophytic growth also can occur posteriorly and laterally, but is less common. These tumors usually have a significant vertical growth vector, but the exophytic growths, when relatively large, can disarticulate the condyle down and out of the fossa, creating a greater exaggeration of the ipsilateral vertical height of the jaws and face (Fig 5F). When performing surgery to remove the tumors, the incision for removal of CH type 2A can usually be smaller compared with a large CH type 2B that may be difficult to remove and require greater access. CH type 2B may have a greater risk of intraoperative and postsurgical vascular and neurologic complications.

The different growth patterns of the tumors for CH types 2A and 2B may be related to the anatomic origin of the tumor on the condylar head, rate of growth, and elongation adaptation of the muscles of mastication and other soft tissues on the ipsilateral side. The constraints of the rate of muscular and soft tissue adaptation and elongation compared with the rate of tumor

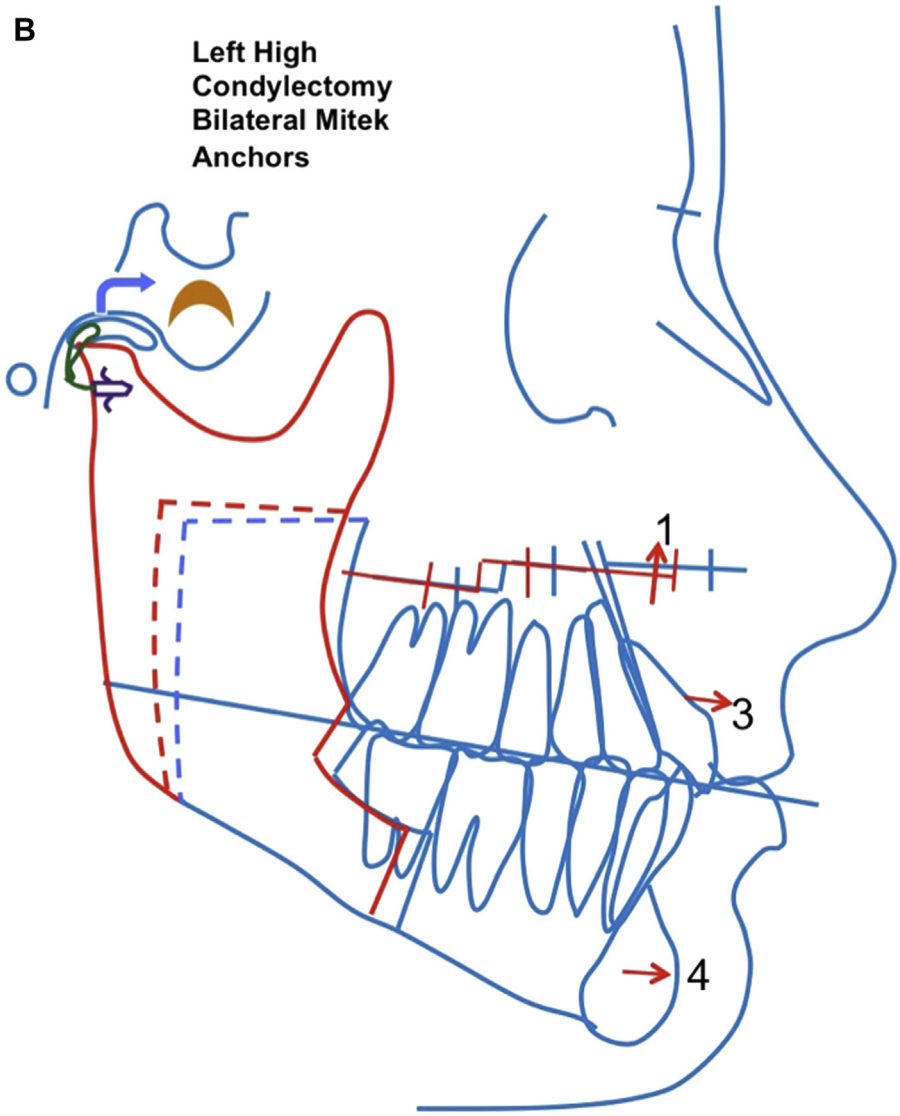


FIGURE 9 (cont'd). *B*, Surgical treatment consisted of *1)* left high condylectomy; *2)* bilateral articular disc repositioning; *3)* bilateral mandibular ramus sagittal split osteotomies; and *4)* maxillary osteotomies to establish good facial balance with skeletal and occlusal stability.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

growth may redirect the tumor development in the direction of least resistance (anteriorly and anteromedially). The roof and posterior wall of the fossa and the lateral and medial capsular ligaments may act as barriers, directing the growth forward. The authors have treated cases with the rare development of the exophytic growth extending laterally and posteriorly.

Treatment Protocol

There are 2 basic treatment approaches for managing CH type 2. Because this pathology is usually progressive and deforming, the 2 options include a condylectomy to remove the tumor.

Option 1. The Wolford surgical protocol (Figs 11B, 12) for CH type 2 includes *1)* a low condylectomy removing the ipsilateral condyle at the condylar base and preserving the condylar neck (Fig 12A); *2)* reshaping the condylar neck to function as the new condyle; *3)* repositioning the articular disc over the top of the condylar neck and stabilization; *4)* repositioning the articular disc on the contralateral side, when displaced; *5)* orthognathic surgery to correct the associated maxillary and mandibular deformities; and *6)* inferior border ostectomy on the ipsilateral side to re-establish the vertical height balance of the mandibular ramus, body, and symphysis if indicated (Figs 11B, 12B). This last procedure will require dissection and preservation of the inferior alveolar nerve if it is located low in the mandible where the ostectomy will be performed. This protocol will provide predictable and

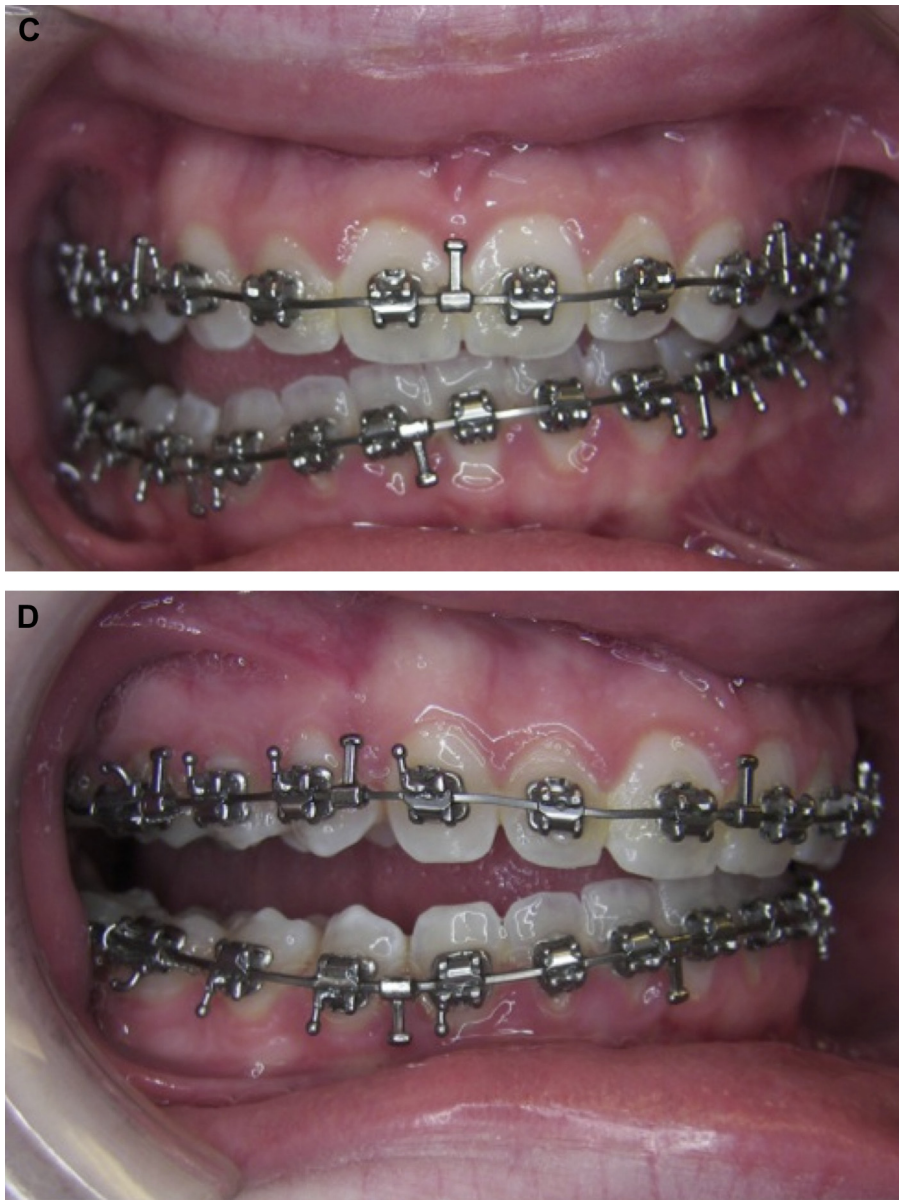
FIGURE 10. Case 3. *A to D,* A 16-year-old female patient presented with condylar hyperplasia type 2A. She first became aware of developing facial asymmetry 4 to 5 years before the first appointment. She developed significant elongation of the right side of the face, right-side posterior open bite, pain, and headache. Surgical treatment in a single stage included *1)* right low condylectomy; *2)* bilateral temporomandibular joint disc repositioning; *3)* bilateral mandibular ramus osteotomies; *4)* multiple maxillary osteotomies; and *5)* right inferior border ostectomy with preservation of the inferior alveolar nerve. *E to H,* The patient 3.5 years after surgery shows good facial balance and a stable skeletal and occlusal result. **(Fig 10 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

stable outcomes and optimize the functional and esthetic results (Fig 10E-H). This treatment approach will allow removal of the tumor, yet still use the enlarged condylar neck as the new condyle. The articular disc on the ipsilateral side and frequently on the contralateral side (if that disc is displaced) will require repositioning and stabilization (Fig 7) to provide the best treatment outcome in relation to function, esthetics, and elimination of any associated pain and dysfunction.[18] If the disc is nonsalvageable, then the authors prefer a custom-fitted total joint prosthesis to reconstruct the ipsilateral or contralateral TMJ.

Option 2. The most popular approach for treating CH type 2 is performing only an ipsilateral condylectomy, partial or complete, without any orthognathic surgery. With a partial condylectomy, usually no other surgery is recommended. When the condylectomy results in significant functional and occlusal instability, or where the entire condyle and neck have been removed, then condyle reconstruction techniques may include TMJ total joint prosthesis, sliding ramus osteotomy, rib graft, sternoclavicular graft, free bone graft, or pedicled osseous graft. When only the ipsilateral condyle is addressed, with no additional orthognathic surgery included, the patient is often left with compromised functional and esthetic results because significant facial asymmetry and possibly malocclusion will remain. This is particularly the case if significant ipsilateral downward growth of the maxilla and vertical elongation of the ipsilateral mandibular body and ramus have occurred. Some of these patients may require secondary orthognathic procedures to achieve a functional occlusion and to restore good facial balance and esthetics. Without maintaining a functional ipsilateral articular disc when using the autogenous condylar reconstruction techniques, significant postsurgical TMJ dysfunction may develop.

When CH type 2 is identified during the normal growth years, then surgery should be deferred, if possible, until 15 years of age for girls and 17 to 18 years of



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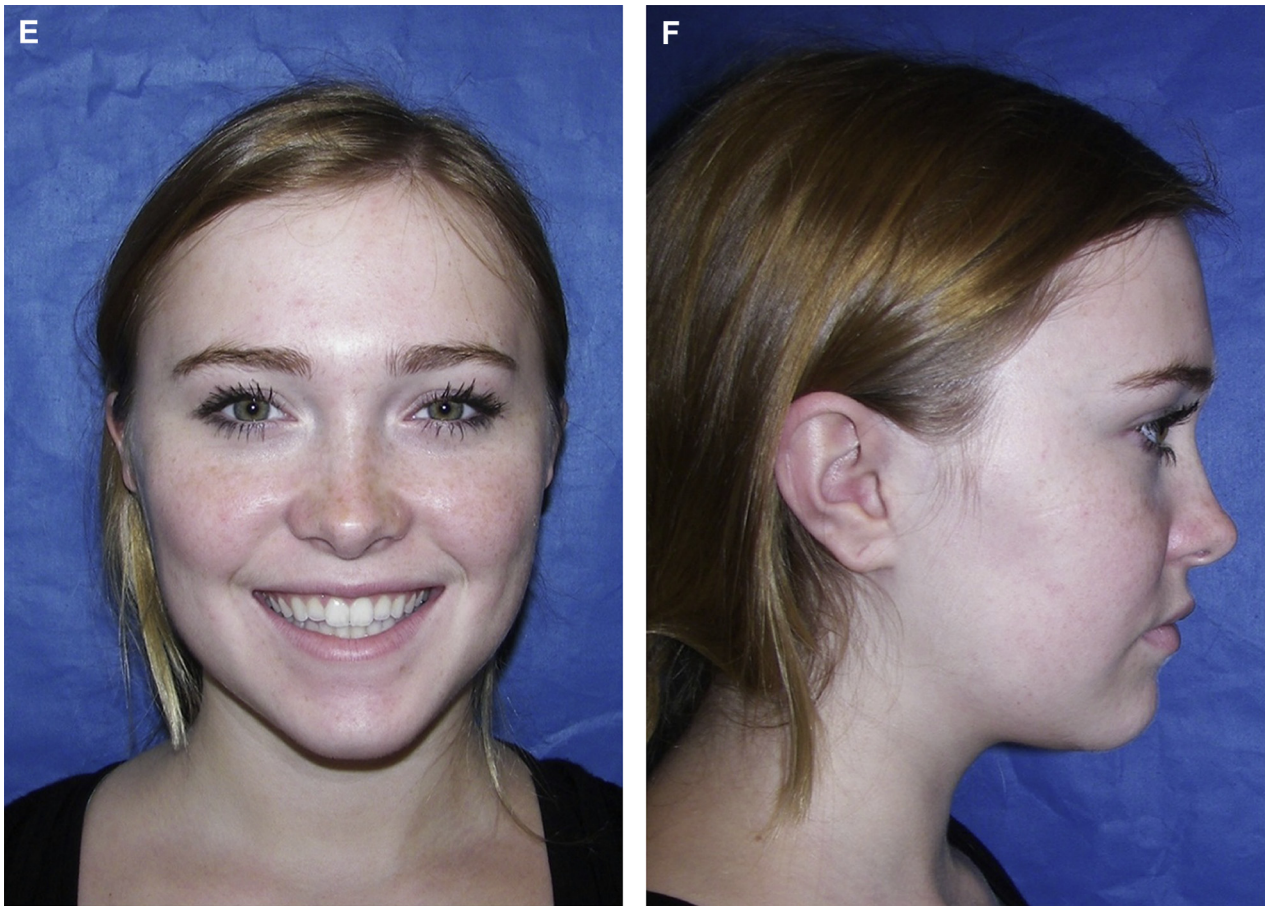
Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

age for boys, after normal jaw growth is relatively complete. However, the severity of the deformity may warrant surgery at a younger age. If the ipsilateral low condylectomy is performed when normal jaw growth is still occurring (<15 yr of age in girls and <17 to 18 yr of age in boys), then there is the risk of the contralateral condyle continuing with normal growth, shifting the mandible toward the ipsilateral side until growth cessation. In CH type 2, the contralateral TMJ articular disc is commonly displaced (76% of cases) and, therefore, disc repositioning would be indicated. However, if surgery is indicated at a younger age, then a high condylectomy can be performed on the contralateral side so that no further growth will occur and the mandible will remain symmetric. Another option would be to perform the unilateral condylectomy and plan for orthognathic surgery as a second stage after cessation of growth.

Ideal facial balance may be difficult to achieve after surgery because of the excessive amount of soft tissue development that occurs with unilateral elongation and the transverse asymmetry that develops. In addition, the ipsilateral mandibular body becomes more curved and the contralateral body contour is flatter. Thus, with vertical shortening of the ipsilateral bony structures and rotation of the chin toward the ipsilateral side, there is excessive soft tissue bulk, including the masseter muscle, that will make the ipsilateral side



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Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

appear fuller, even with the most accurate skeletal correction.

CH TYPES 3 AND 4

CH type 3 includes rare benign tumors originating in the mandibular condyle, such as *1)* osteoma, *2)* chondroblastoma, *3)* osteoid osteoma, *4)* osteoblastoma, *5)* aneurysmal bone cyst, and *6)* central giant cell granuloma. CH type 4 would include malignant tumors originating in the mandibular condyle, such as *1)* chondrosarcoma, *2)* osteosarcoma, and *3)* metastatic carcinoma.

Clinical Features

Benign and malignant tumors affecting the mandibular condyle may have a relatively slow or more rapid progressive development, firm enlargement, and not typically painful until advanced. If numbness, paralysis, and significant pain develop, regardless of whether there is significant facial enlargement, then malignancy must be strongly considered. Unilateral facial enlargement occurs with expansile hard and soft tissue tumors (benign or malignant).

Imaging

Radiography will usually show bone destruction or deposition associated with the condyle, with expansion when the skeletal structures are involved. MRI, computed tomographic scans, and bone scans may show bone or soft tissue involvement.

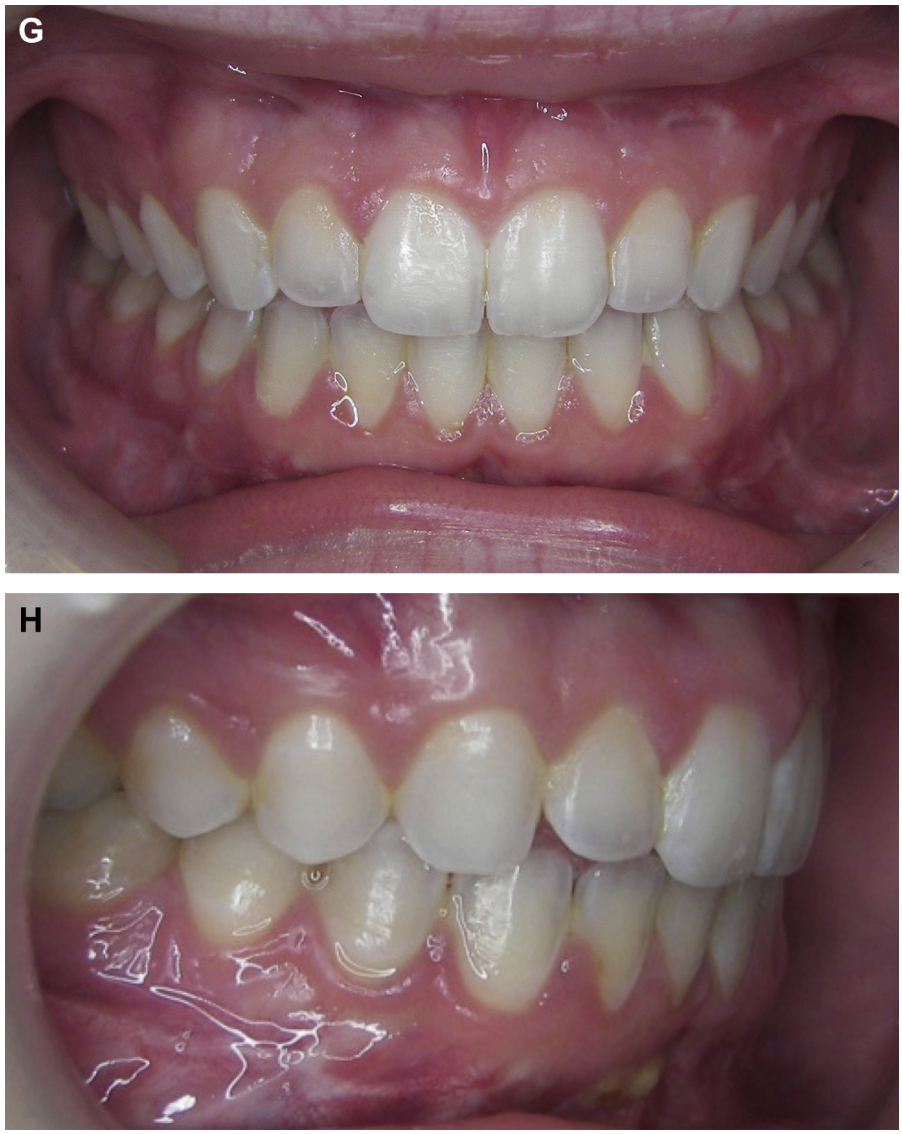
Treatment Considerations

Treatment will depend on the nature and extent of the pathology (benign or malignant), the structures involved, the age of patient, and other medical conditions present. Treatment may include removal of the tumor or pathologic process, with appropriate adjunctive treatment for the specific neoplasia.

Discussion

NORMAL FACIAL GROWTH

Two main types of bone play an integral role in the development of the facial skeleton: intramembranous and endochondral ossification. The facial structures are a hybrid of osteogenic mechanisms; the cranial vault, upper face, midface, and most of the mandible



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arise from intramembranous ossification, whereas the cranial base and mandibular condyle arise from endochondral sources. Developing bone receives genetic, chemical, hormonal, and mechanical signals that induce proliferation and differentiation and affect the contour, shape, and volume of bone in any given area. The mandible is the last element to skeletally mature and is a keystone in the development of the facial skeleton, providing normal occlusion and the maintenance of anteroposterior and vertical facial balance.[19] Alteration or abnormal inflection of any of these stimuli could induce abnormal condylar growth.

Growth of cartilage is interstitial in nature; chondrocyte multiplication and extracellular matrix synthesis are primarily controlled by interstitial genetics and epigenetic factors. During the growth of primarily cartilage, the matrix effectively isolates the dividing chondrocytes from the environmental influences. Chondral growth of TMJ cartilage is distinctly different. Because secondary cartilage grows primarily by apposition, with proliferation occurring in progenitor cells that are surrounded by substantially fewer matrixes, mechanical stimulation alters the growth and differentiation of condylar secondary cartilage cells.[20] Hormonal stimulation from growth hormones and sex-based hormones may be a major factor in the development of CH types 1 and 2. In the authors' studies of CH type 1,[4,8] the onset is during the pubertal growth phase (usually 11 to 15 yr of age) and is more common in female patients (60%), which could indicate possible female and growth hormonal stimulating factors. The authors' studies

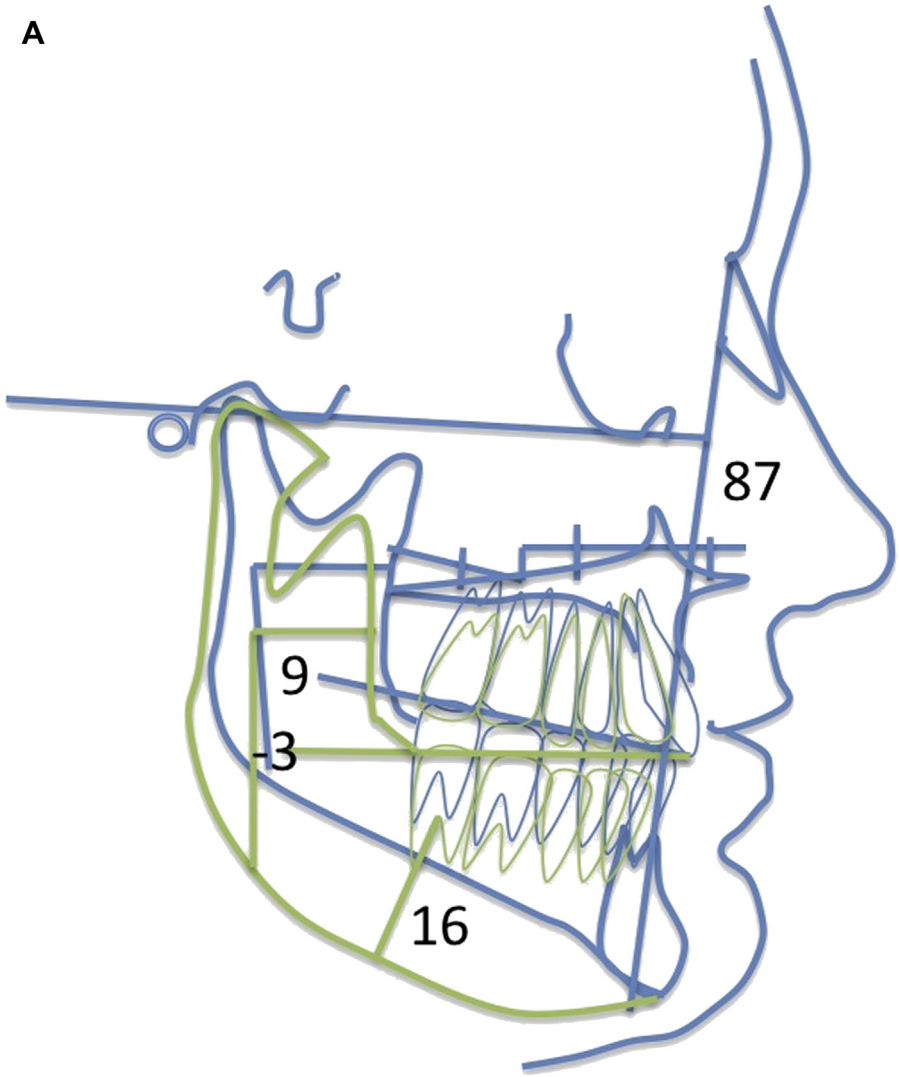

FIGURE 11. Case 3. *A*, Cephalometric analysis shows severe vertical elongation of the right side of the mandible at the inferior border and at the occlusal plane. **(Fig 11 continued on next page.)**

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

of CH type 2 show that 68% of these cases develop during the second decade (10 to 20 yr of age) and predominantly in female patients (76%), indicating a more probable relation to female hormonal stimulus[21] perhaps in conjunction with growth hormones.

Although the specific cause of CH types 1 and 2 has not been clearly identified, the strong predilection for initiation during the teenage years and the dominant occurrence of CH types 1 and 2 in female patients support a theory of female and growth hormonal mediation. Estrogen receptors have been identified in the TMJs of female primates,[22,23] human TMJ tissues that appear to correlate to TMJ symptoms,[24] and arthritic knee joints.[25] Estrogen is known to mediate cartilage and bone metabolism in the female TMJ. An increase in receptors may predispose to the initiation of CH conditions. Further studies in this area are indicated to determine cause and effect.

HISTORICAL TREATMENT OF CH

Adams[2] in 1836 and Humphry[3] in 1856 reported that excision of the condyle in a unilateral CH (these cases were CH type 2B) would arrest the condylar hyperplastic growth process. Gruca and Meisels[26] (1926) supported this treatment approach and stated, "The earlier the operation is done, the smaller the deformation will develop in the upper jaw and position of the teeth. The early resection is not only advisable, but even absolutely indicated." Rushton[11] (1946) also supported this concept, but emphasized that the deformity must be progressive and that a condylectomy of the affected side at a young age, before completion

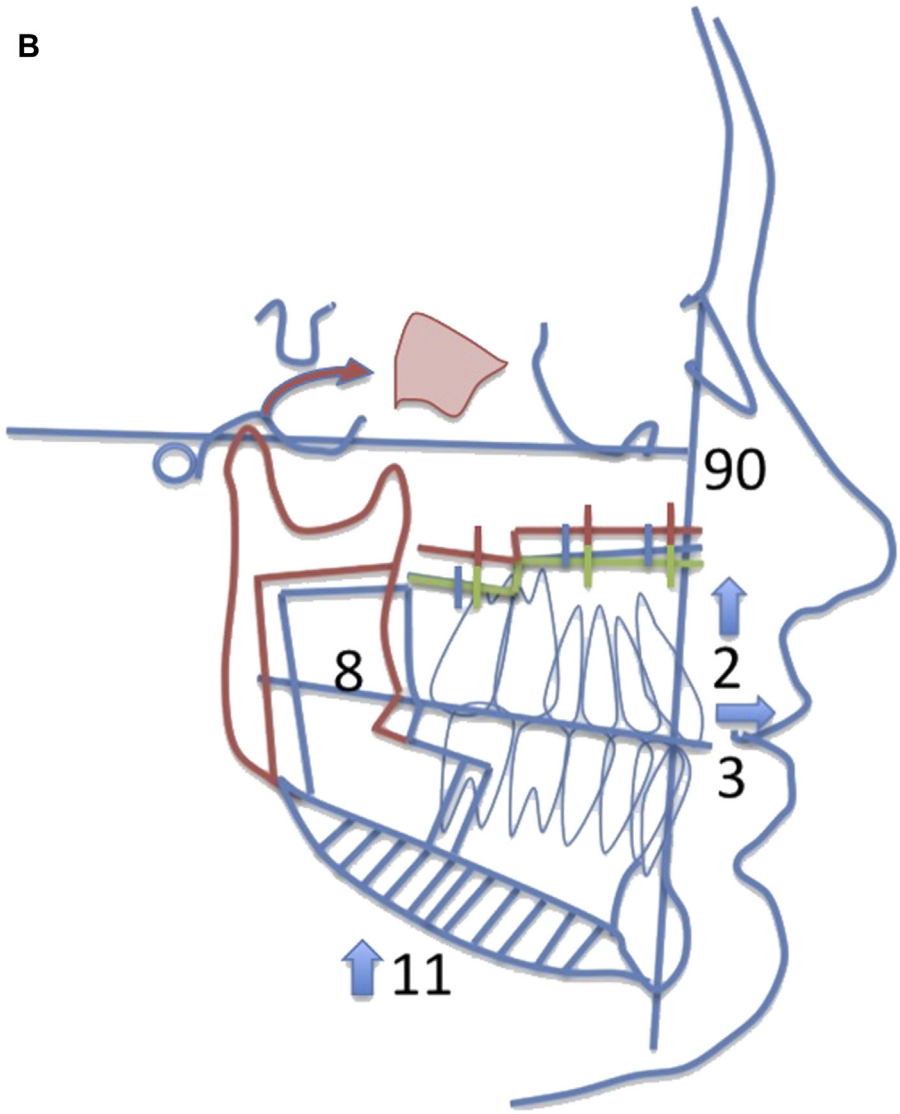

FIGURE 11 (cont'd). *B,* Surgical treatment included *1)* right low condylectomy; *2)* bilateral temporomandibular joint articular disc repositioning; *3)* bilateral mandibular ramus osteotomies; *4)* multiple maxillary osteotomies; and *5)* resection of inferior border of the mandible on the right side with preservation of the inferior alveolar nerve.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

of normal growth, would foreshorten that side of the jaw permanently. This early treatment philosophy for CH was to remove as much condyle as necessary to correct the bite problem but not to overcorrect it. During the past 170 years, many surgeons have advocated and shown that condylectomy for CH type 2 is a predictable method to stop the pathologic growth process.

The authors previously reported that mandibular setback surgery for correction of mandibular prognathism in nongrowing patients without active CH is a very stable procedure.[27] Fourteen nongrowing patients with mandibular prognathism (11 female and 3 male) treated with orthognathic surgery only were evaluated at an average age of 24 years and average postsurgical follow-up of 29 months. The results were stable, with no statistically significant AP changes at point B from immediately after surgery to longest follow-up. This shows that orthognathic surgery for nongrowing patients with mandibular prognathism is a stable and predictable procedure. However, numerous studies have reported relapse for mandibular setback, ranging from 20% to 91% of the amount of posterior surgical movement.[28-36] It is likely that this large percentage of relapse is due to unrecognized and untreated active CH type 1. Understanding the etiology of CH type 1, the nature of the pathology and deformity, clinical presentation, options available for treatment, and timing of treatment is important for achieving optimal treatment outcomes. In 1979, Wolford[37] used a technique to predictably eliminate mandibular growth in CH type 1 by performing high condylectomies with

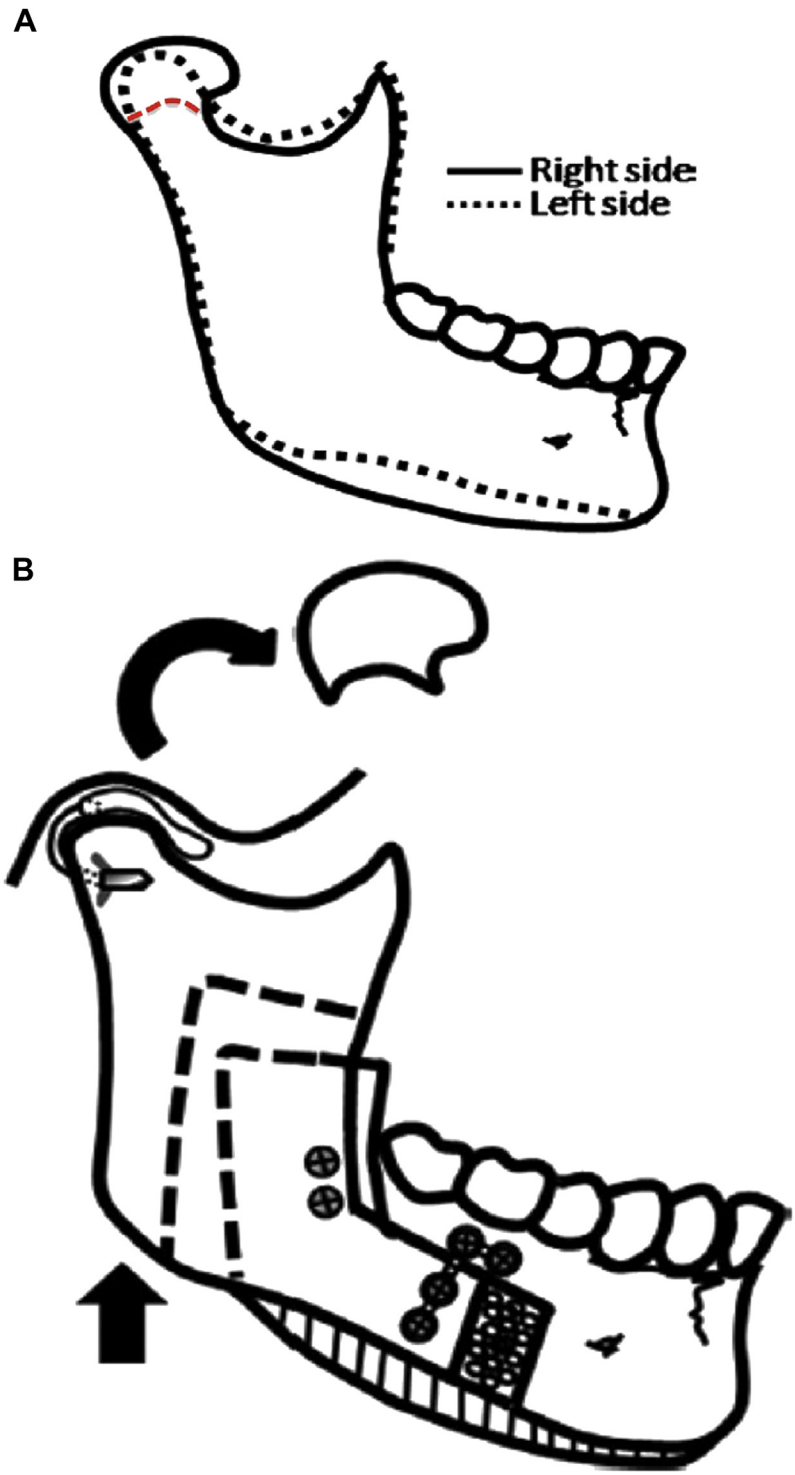

FIGURE 12. *A*, Schematic for the treatment of condylar hyperplasia type 2 includes a low condylectomy, preserving the condylar neck as outlined to remove the osteochondroma. *B*, The condyle is removed, the condylar neck is recontoured, the articular disc is repositioned with a Mitek anchor, sagittal split osteotomies are completed, and an inferior border ostectomy is performed with preservation of the inferior alveolar nerve. Most of these cases also had an indication for maxillary osteotomies.

Wolford, Movahed, and Perez. Classification System for Condylar Hyperplasia. J Oral Maxillofac Surg 2014.

simultaneous orthognathic surgery to correct the associated jaw deformity. The high condylectomy (Fig 6) arrests the excessive and disproportionate growth of the mandible by surgically removing one of the important mandibular growth centers and the site responsible for the CH type 1 pathologic growth process.[4,8]

Wolford et al[4] reported on 54 patients diagnosed with active CH type 1. Group 1 (n = 12) was treated with orthognathic surgery only, whereas group 2 (n = 42) underwent high condylectomies, articular disc repositioning, and orthognathic surgery. The results showed the average ages at surgery were 17.5 years for group 1 and 16.6 years for group 2. The average presurgical follow-ups were 12.5 months for group 1 and 12.1 months for group 2 and confirmed active CH type 1. The average postsurgical follow-up for group 1 was 5.6 years and that for group 2 was 5.1 years. Before surgery there were no statistically significant differences ($P > .05$) in the 2 groups for maximal incisal opening (MIO), lateral excursion (LE), and subjective jaw function. However, group 2 showed a statistically significant ($P < .05$) difference with more active presurgical mandibular growth. At long-term postsurgical follow-up, no differences were found in LE and subjective jaw function. Group 2 showed a statistically significant greater increase in MIO ($P < .01$) and skeletal and occlusal stability ($P < .05$) at long-term follow-up. All patients in group 1 grew back into skeletal and occlusal Class III relations requiring secondary intervention. All patients in group 2 remained stable in a Class I skeletal and occlusal relation. This study confirms the predictability of this surgical protocol for treating CH type 1.

For CH type 2, numerous case reports have reported successful management with condylectomy. The authors previously reported a series of 6 cases of mandibular condylar osteochondroma treated with the protocol described in the present report, including a low condylectomy, recontouring the condylar neck to function as the new condyle, repositioning the articular disc on top of the "new" condyle, and performing the appropriate orthognathic surgery to correct the associated dentofacial deformity.[38] The average follow-up was 52 months (range, 22 to 108 months), with no tumor recurrence. The 6 patients had stable outcomes with a Class I occlusion, good facial balance, average incisal opening of 54 mm (range, 45 to 61 mm), good excursion movements with an average of 5.8 mm (range, 2 to 10 mm), average postsurgical pain at 0.3 (range, 0 to 2, where 0 = no pain, 10 = worst pain imaginable), and jaw function at 1.6 (range, 0 to 3, where 0 = normal function, 10 = inability to move jaw, frozen jaw).

Yang et al[39] presented a study of 17 patients with mandibular condylar osteochondroma treated with a condylectomy at the sigmoid notch and vertical ramus osteotomy to move the posterior border of the mandible into the glenoid fossa fixated with bone plates. They did not correct the facial asymmetry because no orthognathic surgery was performed in these patients. The articular disc was preserved, there were no reoccurrences of the tumor, and all patients reportedly had improved jaw function at longest follow-up.

Ord et al[40] reported on 8 cases treated with subsigmoid osteotomy, removal of the tumor and repositioning of the proximal segment in 5 cases, and a condylectomy with rib graft reconstruction in 3 cases. No orthognathic surgical procedures were performed to correct the dentofacial deformity. There were no recurrences reported, but 4 cases were followed for only 3 to 6 months after surgery. Postsurgical function was not reported. They reviewed the literature and showed that 32 of the 57 cases reported involved female patients (56%). This differs from the present series, in which 76% of the cases involved female patients.[21]

Wolford et al[21] recently reported on a series of 37 patients with CH type 2. There were 28 female (76%) and 9 male (24%) patients treated with the Wolford protocol who were followed for an average of 48 months (range, 12 to 288 months). Only 1 case had a reoccurrence and that patient was initially diagnosed with CH type 1 based on his facial morphology, imaging presentation, and growth pattern. He underwent a high condylectomy (removing the top 4 to 5 mm of the condyle) with disc repositioning and 2-jaw orthognathic surgery. However, the histologic report showed an osteochondroma and the tumor continued to grow, requiring a repeat surgery 14 months later with a low condylectomy and partial repeat of the orthognathic surgery, with a stable outcome at 4 years after surgery. This case confirms the importance of performing a low condylectomy to eliminate the tumor. In this study, the 37 patients had statistically significant improvement in pain, headaches, jaw function, diet, and disability. Importantly, 76% of the patients had contralateral TMJ arthritis and disc dislocation from the functional overload created from the ipsilateral tumor, requiring contralateral disc repositioning. At longest follow-up, there was a nonsignificant decrease in incisal opening of 2.3 mm (average postsurgical incisal opening, 44.9 mm) and a significant decrease in excursive movements of 2.5 mm on the right and 2.2 mm on the left (average postsurgical excursions, 5.3 mm to the right and 5.0 mm to the left).

This report describes a classification for CH conditions, based on occurrence rate and type of pathology. CH type 1A is the most commonly occurring form and CH type 4 is the rarest. Classifying CH pathology in this manner will help the clinician understand the nature of the pathology, progression if untreated, recommended ages for surgical intervention to minimize an adverse effect on subsequent facial growth and development in younger patients, and the surgical protocols to comprehensively and predictably treat these conditions.

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