

Efficacy of high condylectomy for management of condylar hyperplasia

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The purpose of this study was to compare the treatment outcome and long-term stability of 2 groups of young adult patients diagnosed with active condylar hyperplasia and treated with 2 different surgical methods. Thirty-seven patients (19 females and 18 males) met the criteria for inclusion in the study. Group 1 (n = 12; average age at surgery, 17.5 years) was treated with orthognathic surgery only, while group 2 (n = 25; average age at surgery, 16.7 years) had high condylectomy, articular disc repositioning, and orthognathic surgery. All patients underwent standardized clinical and radiographic examination at initial consultation, immediately before surgery, immediately after surgery, and at longest follow-up. Objective evaluation of temporomandibular joint (TMJ) function included maximum incisal opening and lateral excursions. Subjective evaluations were performed in group 2 for TMJ pain, jaw function, and diet. Lateral cephalometric radiographs were evaluated for presurgical and postsurgical mandibular growth. There were no statistically significant differences ($P > .05$) between the 2 groups for maximal incisal opening, lateral excursions, or subjective jaw function before surgery. Presurgical growth differed significantly ($P < .05$), with group 2 showing more active growth. At the long-term follow-up, no differences were found in lateral excursions or subjective jaw function. There was a statistically significant difference in maximum incisal opening ($P < .01$), with a greater increase in group 2, as well as a statistically significant difference ($P < .05$) in cephalometric stability, with group 2 being much more stable at long-term follow-up. All patients in group 1 grew back into skeletal and occlusal Class III relationships and required secondary intervention. Only 1 patient in group 2 required secondary surgery, involving maxillary surgery to correct postsurgical transverse maxillary relapse; the mandible was stable at long-term follow-up. The results of this study showed that patients with active condylar hyperplasia treated with high condylectomy, articular disc repositioning, and orthognathic surgery have stable, predictable outcomes compared with those treated with orthognathic surgery alone. (Am J Orthod Dentofacial Orthop 2002;121:136-51)

Condylar hyperplasia (CH) is a pathological condition that presents a challenge to both orthodontists and oral and maxillofacial surgeons because of its progressiveness and the severe dentofacial deformity it can create. First described by Adams¹ in 1836, CH causes overdevelopment of the mandible,

usually creating significant functional and esthetic deformities. In 1946, Rushton² reviewed 29 reported unilateral cases, and by 1968, a total of 150 cases had been reported in the literature, most of which were isolated.³ However, CH is much more common than clinicians realize, and failure to recognize this condition can result in unfavorable functional and esthetic treatment results.

A sound understanding of the etiology, nature of the deformity, clinical presentation, options for treatment, and timing of treatment is required to achieve optimal treatment outcomes. The high condylectomy (Fig 1) arrests the excessive and disproportionate growth of the mandible by surgically removing a principal mandibular growth site. No large studies are reported in the literature regarding diagnosis and surgical treatment of CH. The purpose of this study was to compare the treatment outcome and long-term follow-up stability of patients diagnosed with condylar hyperplasia, with 1 group treated with conventional orthognathic surgery and another group treated with high condylectomy and articular disc repositioning with simultaneous orthognathic surgery.

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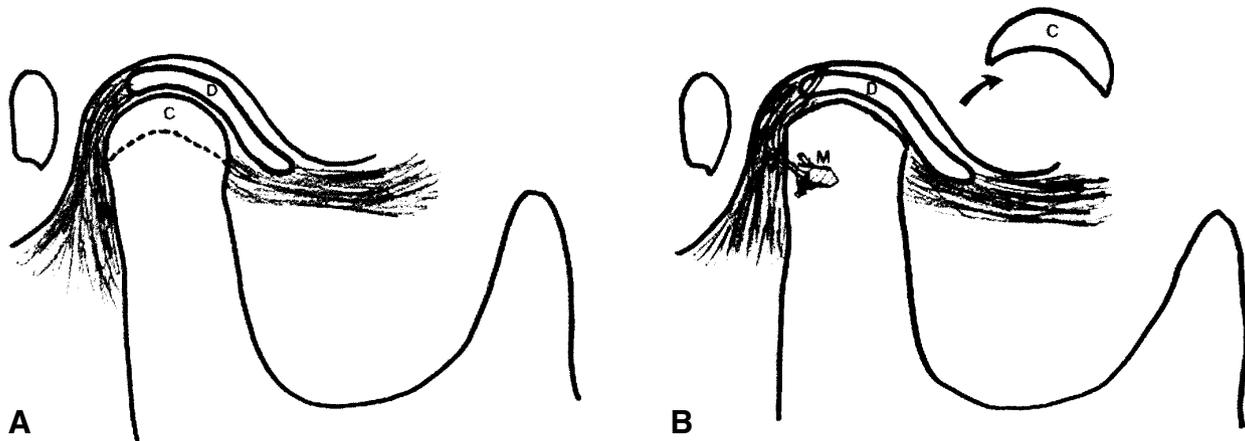


Fig 1. A, High condylectomy involves removing top 3-5 mm of condylar head (C), including lateral and medial poles. **B**, Top portion of condylar head has been removed. TMJ articular disc (D) has been repositioned over condylar stump and secured in position with Mitek mini anchor. Cortical bone will reform over head of condyle.

CH usually develops during puberty and rarely begins after the age of 20.⁴ The identification of sex hormone receptors in and around the temporomandibular joint (TMJ) and the pubertal onset of CH strongly suggest a hormonal influence in the etiology. Trauma,⁵⁻⁹ infection,^{1,5,10,11} heredity,^{3,4,12-15} intrauterine factors,^{6,16,17} and hypervascularity^{15,18,19} have also been implicated as causative factors. Approximately one third of bilateral CH patients have a family history of the condition.⁴

Two basic growth vectors occur with CH: horizontal growth vector (type 1) and vertical growth vector (type 2). The prevalence ratio between types 1 and 2 is approximately 15:1. Distinct radiographic and clinical features differentiate the 2 types. A normal condyle is approximately 15 to 20 mm in mediolateral dimension, and 8 to 10 mm wide anteroposteriorly.²⁰ In type 1 CH, although the condyle usually retains a relatively normal architecture, an increase in length of the condylar head, neck, and mandibular body is commonly seen.^{2,3,21-23} Type 2 CH may demonstrate a condylar head and neck that are much larger in length and diameter than normal, and the medial and lateral poles may be prominent, but the condylar surface is smooth and the contour uniform. Unusual morphological characteristics of the condylar head, such as bony outgrowths, globular enlargements, saddle-shaped cavities, hockey-stick-like exostoses, and bulbous enlargements, are most likely not CH but, rather, an osteochondroma or other pathological condition of the condyle.

Histological observations of the proliferative layer in a CH condyle demonstrate a greater thickness in some areas and lesser in others, but cartilage-producing

cells are everywhere at its lower border. In some regions, the cartilage is very thick and is being actively generated and replaced by new bone.² The activity of the proliferative layer appears to regulate the rate at which the condyle and the condylar neck (formed from the condyle by remodeling) will grow. In normal condyles, the formation of cartilage from the proliferative layer and the replacement of cartilage by bone cease by approximately 20 years of age. The marrow cavity is entirely occluded from the remaining cartilage by the closure of the bone plate.² The inability of this plate to close in the presence of an active proliferative layer may be a major etiologic factor in CH and may correlate to our observation that cessation of growth related to CH may not occur until the middle to late 20s.²³ Conditions that initiate excessive accelerated mandibular growth after the age of 20 are most often related to an osteochondroma, an osteoma, or another type of proliferative condylar pathology.

Type 1 CH is not well accepted as a form of CH by many clinicians. It is usually termed symmetrical or deviated prognathism, laterognathia, or mandibular hyperplasia. However, the basic cause of many mandibular prognathic cases is type 1 CH, ie, excessive mandibular growth originating in the mandibular condyles. Type 1 CH occurs with equal frequency in males and females, as well as unilaterally and bilaterally. These patients usually demonstrate a Class I or mild Class III skeletal and occlusal relationship before the onset of CH and develop into a Class III or severe Class III relationship, respectively, as their growth accelerates. Type 1 CH rarely occurs in skeletal Class II patients. If 1 side of the mandible grows more rapidly

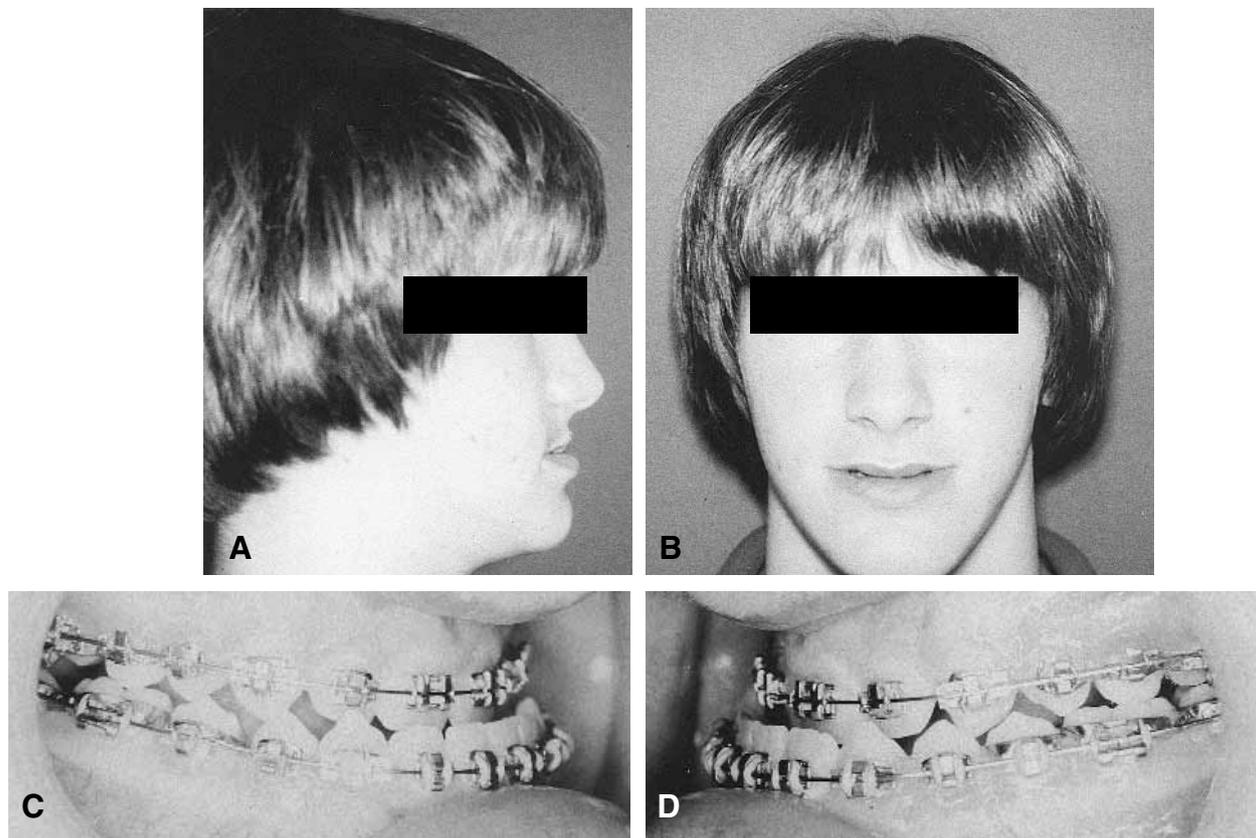


Fig 2. Case 1. **A** and **B**, This 15-year-old male had symmetric mandibular prognathism due to active type 1 CH. **C** and **D**, Full Class III occlusal relationship at T2.

than the contralateral side, deviate prognathism develops. Common clinical and radiographic characteristics observed in bilateral, symmetrically growing type 1 CH patients (Figs 2-4) usually include (1) increased length of the condylar head and neck, without a significant volumetric increase in the size of the condylar head; (2) accelerated mandibular growth; (3) mandibular growth continuing beyond the normal growth years; (4) worsening Class III skeletal and occlusal relationship; (5) worsening esthetics; (6) obtuse gonial angles; (7) decreased angulation of the lower incisors and possibly increased angulation of the upper incisors (dental compensations); (8) decreased vertical height of the posterior mandibular body; (9) high mandibular plane angle; and (10) narrow anteroposterior (A-P) dimension of the symphysis in more severe cases. Additionally, unilateral cases (Figs 5-7) may have (1) TMJ articular disc displacement; (2) worsening facial and occlusal asymmetry, with the mandible progressively shifting toward the contralateral side; (3) unilateral posterior crossbite on the contralateral side; (4) transverse bowing of the mandibular body on the

affected side; and (5) transverse flattening of the mandibular body on the contralateral side.

The differential diagnosis for type 1 CH includes (1) maxillary hypoplasia; (2) mandibular prognathism without CH (patients start out as skeletal Class III in early childhood and maintain harmonious growth between maxilla and mandible, with growth ceasing at the normal age); (3) dislocation of the condyles anterior to the articular eminence; (4) dental interferences or habitual posturing, causing anterior positioning of the mandible; (5) acromegaly; (6) macroglossia; and (7) other TMJ pathology such as osteochondroma, osteoma, or contralateral condylar resorption.

Type 2 CH is sometimes called hemimandibular hypertrophy. However, it can be a form of CH and usually occurs unilaterally. The more severe the pathology, the greater the clinical asymmetry and the degree of morphological alterations. Most patients have a low mandibular plane angle before the onset of CH. Specific characteristics of type 2 CH (Figs 8-10) include (1) unilateral elongation of the face, causing facial asymmetry and worsening esthetics; (2)

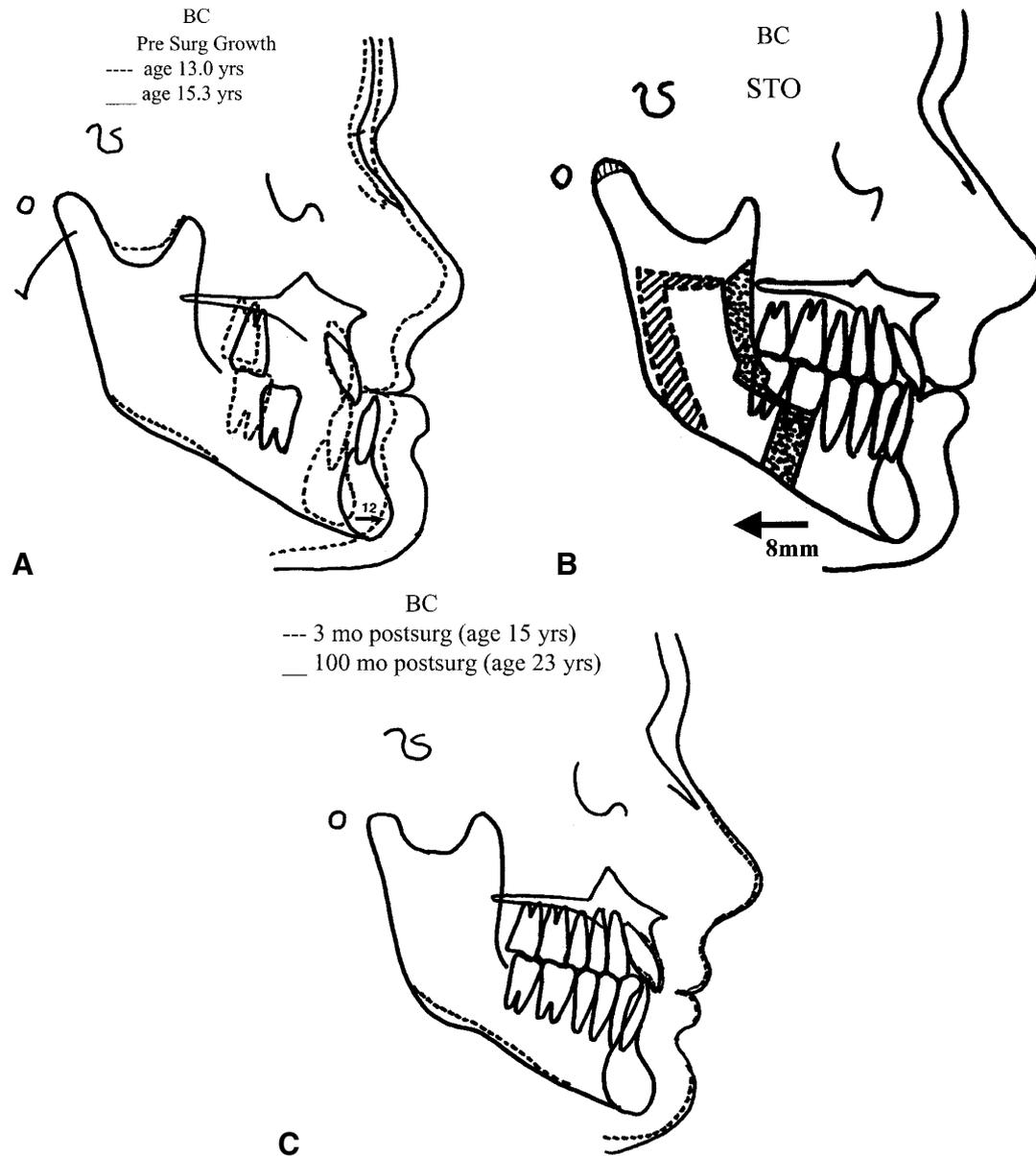


Fig 3. Case 1. **A**, Superimposition of cephalometric tracing from age 13 (*dotted line*) to age 15 years 3 months (*solid line*) demonstrates 12 mm of mandibular growth, changing patient from skeletal Class I position to full Class III position. **B**, Prediction tracing illustrates performance of bilateral high condylectomies, repositioning of articular discs, and bilateral mandibular ramus osteotomies to set mandible posteriorly into proper relationship with maxilla. **C**, Superimposition of 3-month postsurgical cephalometric tracing (*dotted line*) and 8 year 4 month postsurgical tracing (*solid line*) demonstrates excellent stability of surgical result.

increased length, size, and diameter of the condylar head and neck; (3) increased vertical height of the entire mandible on the involved side (except for the coronoid process); (4) open bite on the involved side; (5) compensatory vertical overdevelopment of the maxilla on the involved side; and (6) dental compensations.

The differential diagnosis for type 2 CH includes (1) osteochondroma or other condylar enlarging pathology; (2) hemifacial hypertrophy; (3) contralateral condylar hypoplasia, resorptive TMJ pathology, or condylar fracture; and (4) other pathology such as unilateral fibrous dysplasia, Sturge-Weber syndrome, arteriovenous malformation, hemangioma, or lymphangioma.

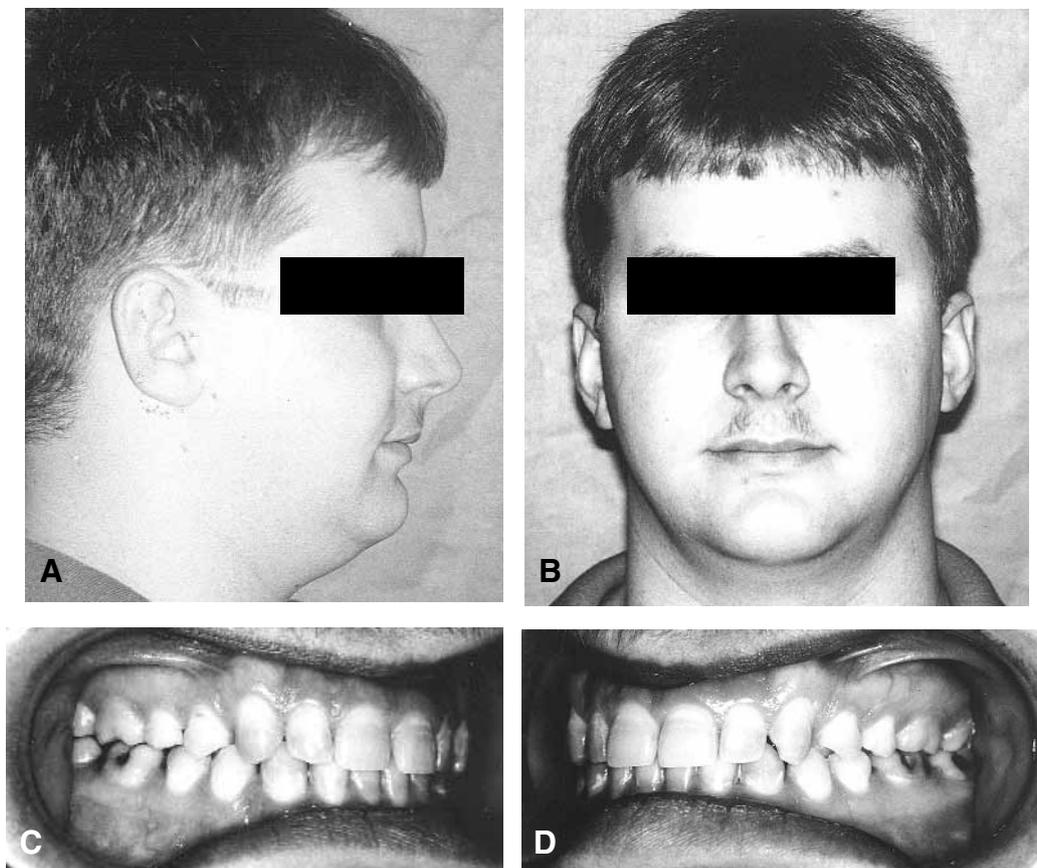


Fig 4. A-D, Patient at 8 years 4 months after surgery, demonstrating good facial balance and very stable Class I occlusal result.

The most common and similar pathology to type 2 CH is an osteochondroma of the condyle. The radiographic appearance of the 2 processes can be similar, unless the osteochondroma is large, with exophytic growths. Clinically, the condylar surface of an osteochondroma is lumpy and irregular, unlike the smooth surface seen in CH. Histopathologically, unlike CH, an osteochondroma usually has islands of cartilage within the medullary bone of the condyle; however, in slow-growing lesions, these may be difficult to identify. Treatment of an osteochondroma usually requires a complete condylectomy and TMJ reconstruction to stop the abnormal growth, whereas CH requires only a high condylectomy (removal of the top 3 to 5 mm of the condyle).

Active CH growth can usually be determined by worsening functional and esthetic changes with serial assessments (preferably at 6- to 12-month intervals) consisting of (1) clinical evaluation; (2) dental model analysis with orthodontically trimmed models or models mounted in centric relation; (3) radiographic evaluation by superimposition, including (a) lateral cephalometric radiographs, (b) frontal cephalometric

radiographs (particularly helpful in unilateral CH cases), and (c) lateral cephalometric tomograms that include the TMJ, the mandibular ramus, and the body. The normal pubertal mandibular growth rate is approximately 2 mm for males and 1.5 mm for females, measuring from condylion to Point B.

Bone scanning with Technetium 99M pyrophosphate or Technetium 99M methylene diphosphonate may detect active growth in the condyle.²⁵⁻³⁰ This may be most effective in unilateral cases, especially if applied after the normal growing years, when condylar growth should have ceased. We have found bone scans to be inconclusive in younger patients and those with slow-growing CH. Hand-wrist films have no value in CH because the mandible can continue to grow well beyond the normal growth years.

PATIENTS AND METHODS

This retrospective study included treatment records of all patients diagnosed with active CH treated by the senior author (L.M.W.). Criteria for inclusion in the study were (1) confirmed active bilateral or unilateral CH based on serial clinical evaluations and radio-

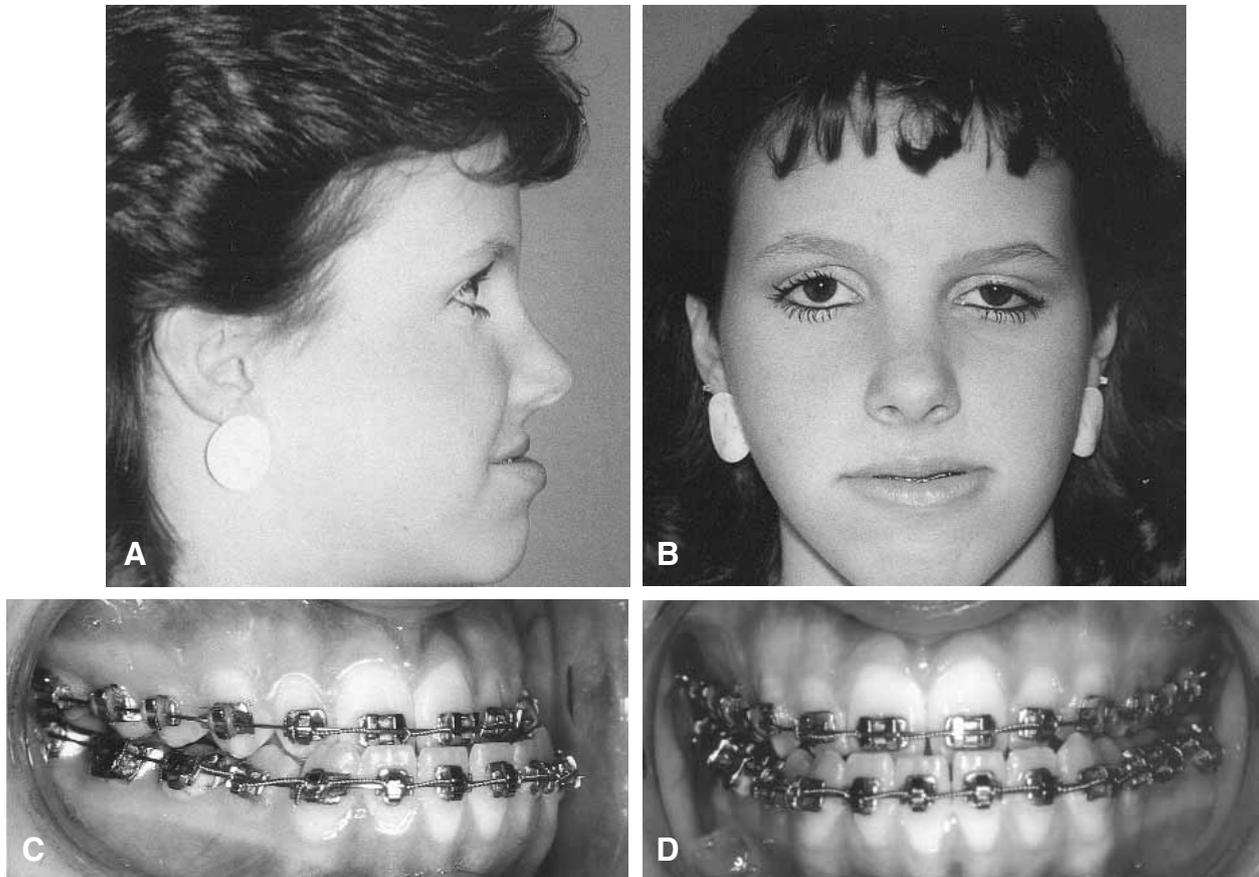


Fig 5. Case 2. **A** and **B**, This 16-year-old female had deviated mandibular prognathism toward left that was worsening as result of unilateral right type 1 CH. **C** and **D**, Deviation of mandibular dental midline to left with anterior and left posterior crossbite. Class III occlusion was greatest on right side.

Table I. General data on 37 patients divided into 2 groups

	Sex	Age	Affected side	Presurgical follow-up	Postsurgical follow-up
Group 1 (n = 12)	8 F, 4 M	17.5 years (range, 13 to 25)	12 bilat	12.5 months (range, 5 to 43)	5.65 years (range, 2 to 11.2)
Group 2 (n = 25)	12 F, 13 M	16.7 years (range, 13 to 24)	13 unilat 12 bilat	12.3 months (range, 4 to 45)	5.3 years (range, 2.8 to 16.9)

graphic tracings with superimposition (serial lateral cephalograms and lateral cephalometric tomograms) demonstrating progressive excessive mandibular growth; (2) orthognathic surgery performed to correct the associated Class III dentofacial deformity, including bilateral mandibular ramus sagittal split osteotomies; (3) treatment with or without high condylectomy and articular disc repositioning concomitantly with the orthognathic surgery; and (4) at least 2 years of post-surgical follow-up.

Thirty-seven patients (19 females and 18 males) met the criteria for inclusion in the study for active type 1 CH (Table I). We also evaluated 2 separate cases of active unilateral type 2 CH treated with a combination

of orthognathic surgery, unilateral condylectomy with articular disc repositioning, and horizontal ostectomy of the inferior border of the mandible on the involved side with preservation of the inferior alveolar nerve. Of the 37 patients with type 1 CH, 23 had bilateral and 14 had unilateral CH. All patients underwent standardized clinical and radiographic examination at the following intervals: initial consultation (T1), immediately presurgical (T2), immediately postsurgical (T3), and longest follow-up (T4). A single clinician (L.M.W.) performed all clinical examinations. TMJ function was objectively evaluated, and maximum interincisal opening (MIO) and lateral excursions (LEs) were measured on all patients. For those undergoing TMJ surgery, additional

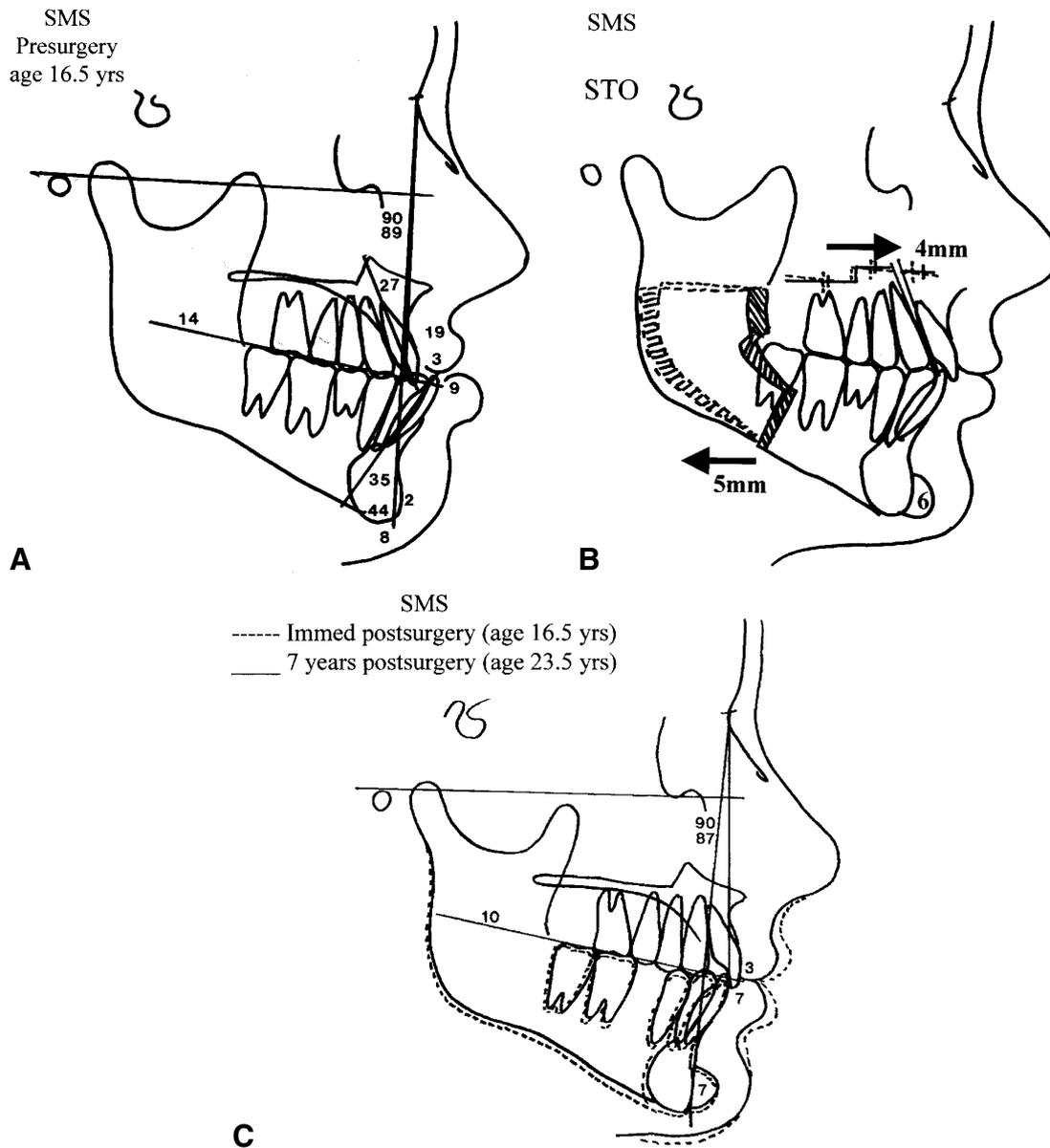


Fig 6. Case 2. **A**, Presurgical lateral cephalometric tracing demonstrates skeletal and occlusal Class III relationship. **B**, Prediction tracing illustrates intended surgery, including right TMJ high condylectomy with repositioning of articular disc, bilateral mandibular ramus osteotomies, maxillary advancement, and chin augmentation with alloplast. **C**, Superimposition of immediate postsurgery (*dotted line*) and 7-year follow-up (*solid line*) cephalometric tracings demonstrates outcome stability.

evaluations included subjective evaluations with numerical visual analog scales (VAS) to assess TMJ pain (0 = no pain, 10 = worst pain imaginable), jaw function (0 = normal function, 10 = no function), and diet (0 = no restrictions, 10 = liquids only). A calibration error test was performed for each parameter in 10 different cephalograms. Correlations were calculated for intraexaminer and interexaminer reliability. The calibration showed a high correlation ($R > 0.96$) for

both intraexaminer and interexaminer reliability. Standard error was less than 0.45 for each parameter. Lateral cephalograms at T1, T2, T3, and T4 were traced and superimposed by a single examiner (O.R-F.) to calculate presurgical change (T2-T1), surgical change (T3-T2), and long-term stability (T4-T3). The lateral cephalometric radiographs were assessed on all patients for (1) mandibular position in relation to the cranial base (FH-NB), (2) condylion-pogonion length

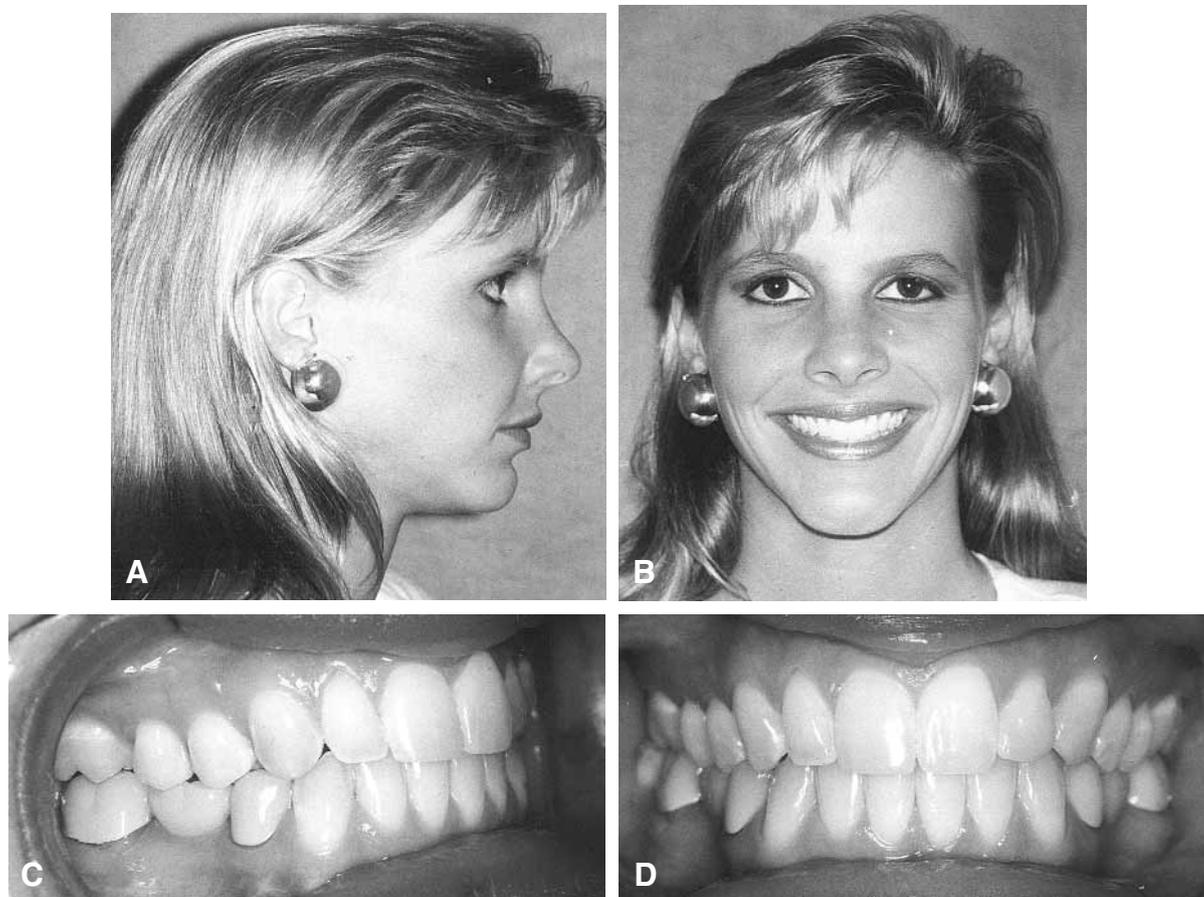


Fig 7. Case 2. **A-D**, Patient at 7 years after surgery, demonstrating good long-term function and esthetic stability.

(Co-Pg), and (3) condylion-Point B length (Co-Pt B). The Student *t* test was used to detect differences between groups and between time intervals, at a significance level of $P \leq .05$.

RESULTS

Because the statistical analysis showed no significant sex differences or differences between unilateral and bilateral patients ($P > .05$), the 2 samples were pooled for subsequent analyses (Tables II-IV).

Patients in group 1 were treated with orthognathic surgery only; patients in group 2 were treated with orthognathic surgery, high condylectomy, and articular disc repositioning on condyles demonstrating active CH.

In group 1 ($n = 12$), the average age at surgery was 17.5 years, the average presurgical follow-up was 12.5 months, and the average postsurgical follow-up was 5.6 years. In group 2 ($n = 25$), the average age at surgery was 16.7 years, the average presurgical follow-up was 12.3 months, and the average

Table II. Objective clinical data on 37 patients divided into 2 groups

	Mean MIO (mm)	Mean LE (mm)
Group 1 ($n = 12$)		
T2	46.8 (range, 26 to 53)	7.0 (range, 5 to 9)
T4	46.7 (range, 43 to 50)	7.5 (range, 6 to 10)
Group 2 ($n = 25$)		
T2	39.5 (range, 26 to 49)	7.7 (range, 3 to 10)
T4	49.7* (range, 31 to 62)	7.8 (range, 4 to 12)

*Statistically significant at $P < .01$ level.

MIO, Maximum interincisal opening; LE, lateral excursions.

postsurgical follow-up was 5.3 years. All patients in group 1 grew back into skeletal and occlusal Class III relationships and required secondary intervention to correct the resultant deformity. Only 1 patient in group 2 required secondary surgery, involving maxillary surgery to correct a postsurgical transverse maxillary relapse; the mandible, however, was stable at T4.

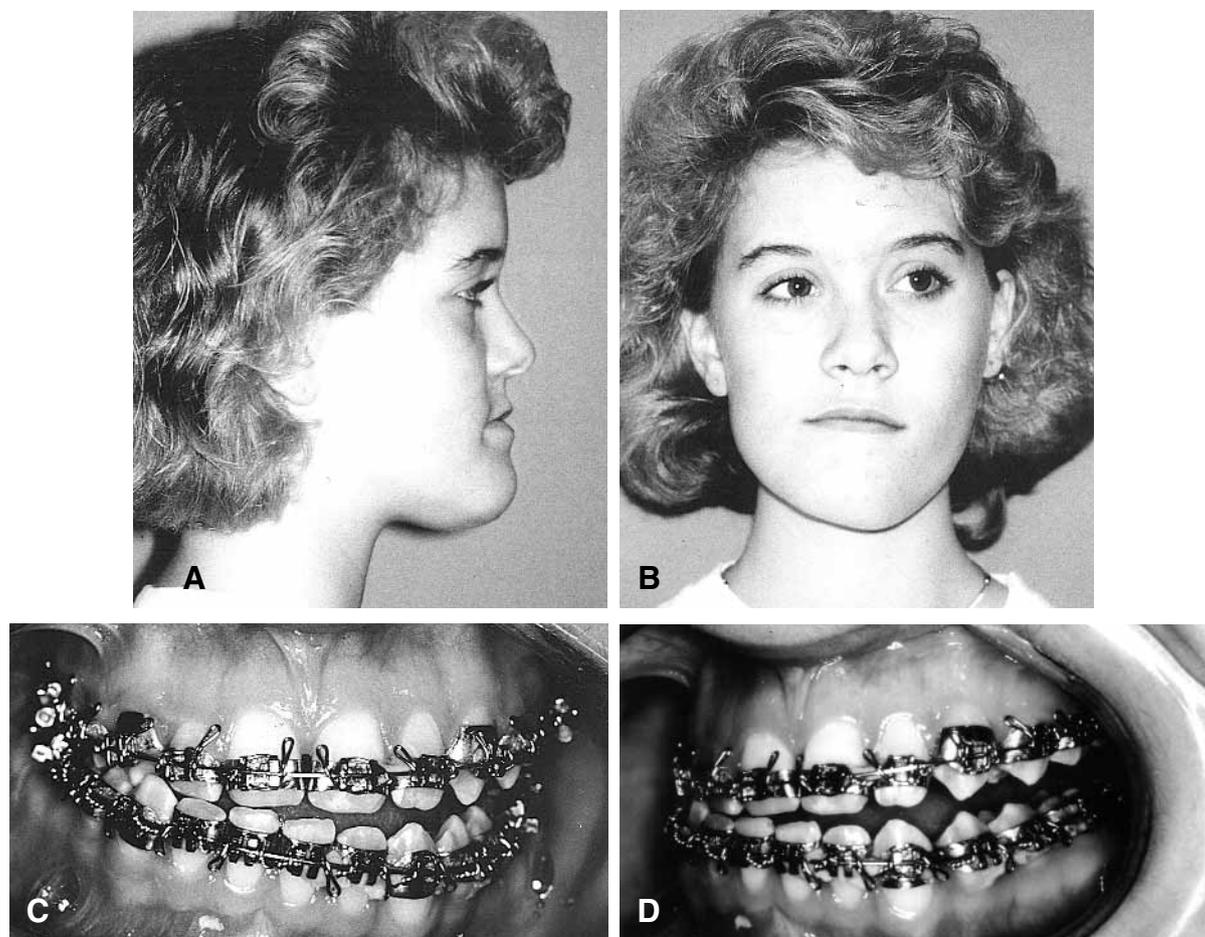


Fig 8. Case 3. **A** and **B**, This 16-year-old female had type 2 CH with severe elongation of left side of face. **C** and **D**, Open bite was created on left side by type 2 CH, with compensatory down-growth of left maxilla.

Table III. Cephalometric data on 37 patients divided into 2 groups

	Mean FH-NB (degrees)	Mean Co-Pg (mm)	Mean Co-Pt-B (mm)
Group 1 (n = 12)			
T2-T1	1.35 (range, -1 to 5)	2.4 (range, 1 to 5)	2.3 (range, 1 to 5)
T3-T2	-4.1 (range, -1 to -7)	-4.7 (range, -1 to -9)	-5.8 (range, -3 to -12)
T4-T3	2.8* (range, 0 to 5)	3.5† (range, 1 to 11)	3.6† (range, 2 to 7)
Group 2 (n = 25)			
T2-T1	2.0 (range, 1 to 5)	4.5* (range, 1 to 14)	3.6* (range, 1 to 11)
T3-T2	-4.6 (range, -1 to -7)	-5.2 (range, -1 to -8)	-4.5 (range, -1 to -10)
T4-T3	1.2 (range, 0 to 2)	0.8 (range, 0 to 3)	0.4 (range, 0 to 2)

*Statistically significant at $P < .05$ level.

†Statistically significant at $P < .01$ level.

Objective clinical data

In group 1, MIO was 46.8 mm at T2 and 46.7 mm at T4. In group 2, MIO was 39.5 mm at T2 and 49.7 mm at T4. Although there was a difference between groups

1 and 2 in MIO at T2, group 2 started with lower values than group 1, but the difference was not statistically significant ($P > .05$). At T4, group 1 showed a nonstatistically significant decrease of MIO ($P > .05$), and group

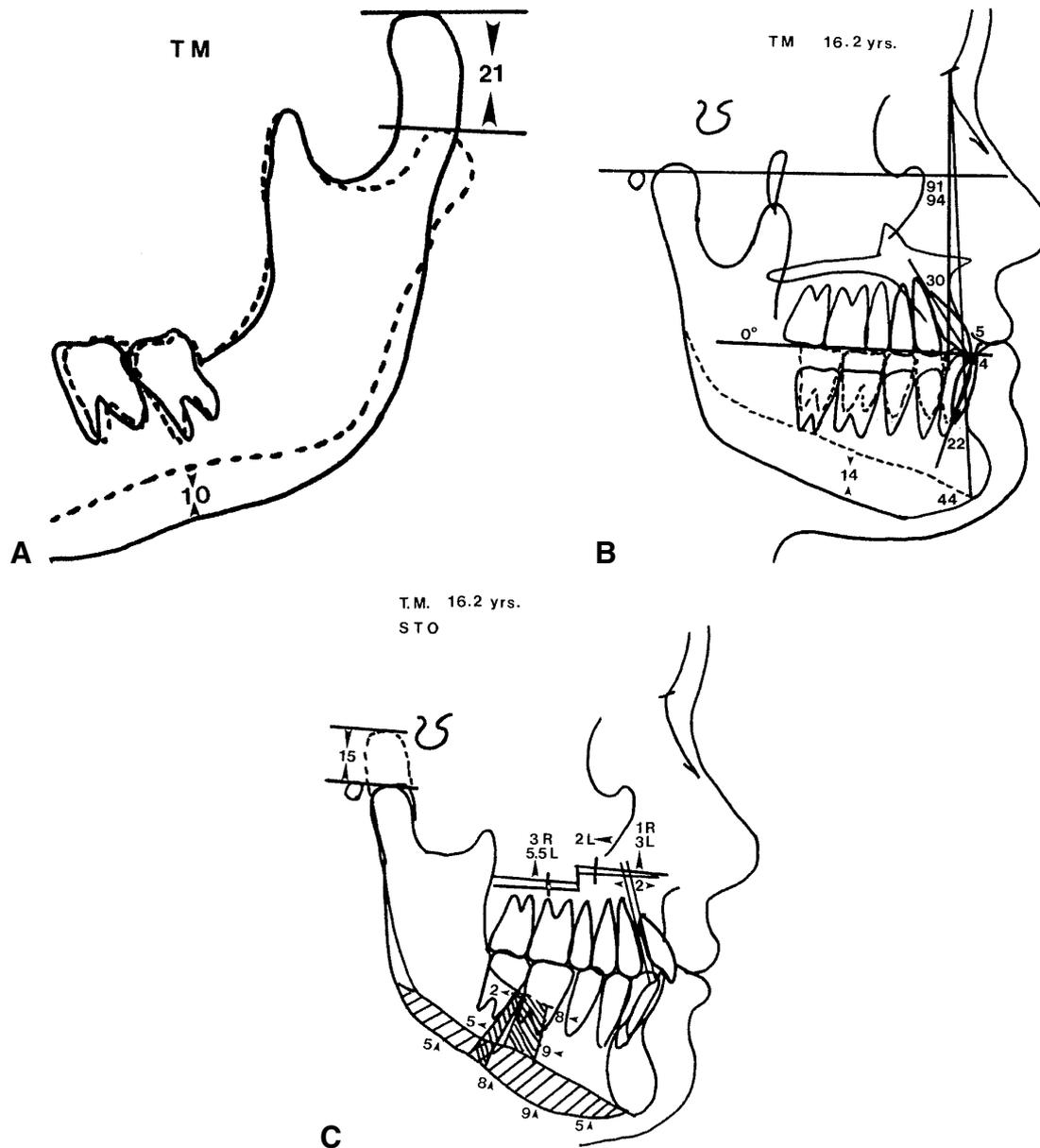


Fig 9. Case 3. **A**, Excessive elongation of left mandibular condyle compared with relatively normal right condyle. **B**, Presurgical cephalometric radiograph demonstrates significant increased vertical elongation of mandible on left compared with level of inferior border on right side. Mandibular occlusal plane on left side is significantly lower than right side. **C**, Prediction tracing illustrates surgery to be performed, including low condylectomy (to help correct severe vertical discrepancy), double jaw orthognathic surgery, and resection of left inferior border of mandible, to improve facial harmony.

2 showed a statistically significant increase ($P < .01$). The LE of group 1 were 7.0 mm at T2 and 7.5 mm at T4. The LEs of group 2 were 7.7 mm at T2 and 7.8 mm at T4. There was no significant difference between the groups when comparing LEs at T2 and T4 ($P > .05$) (Table II).

Cephalometric data

Group 1 had a change of 1.35° in Frankfort horizontal-NB angle from T1 to T2, a change of -4.1° from T2 to T3, and a change of 2.8° from T3 to T4. Group 2 had a change of 2.0° in Frankfort horizontal-NB angle from T1 to T2, a change of -4.6° from T2 to T3, and a change

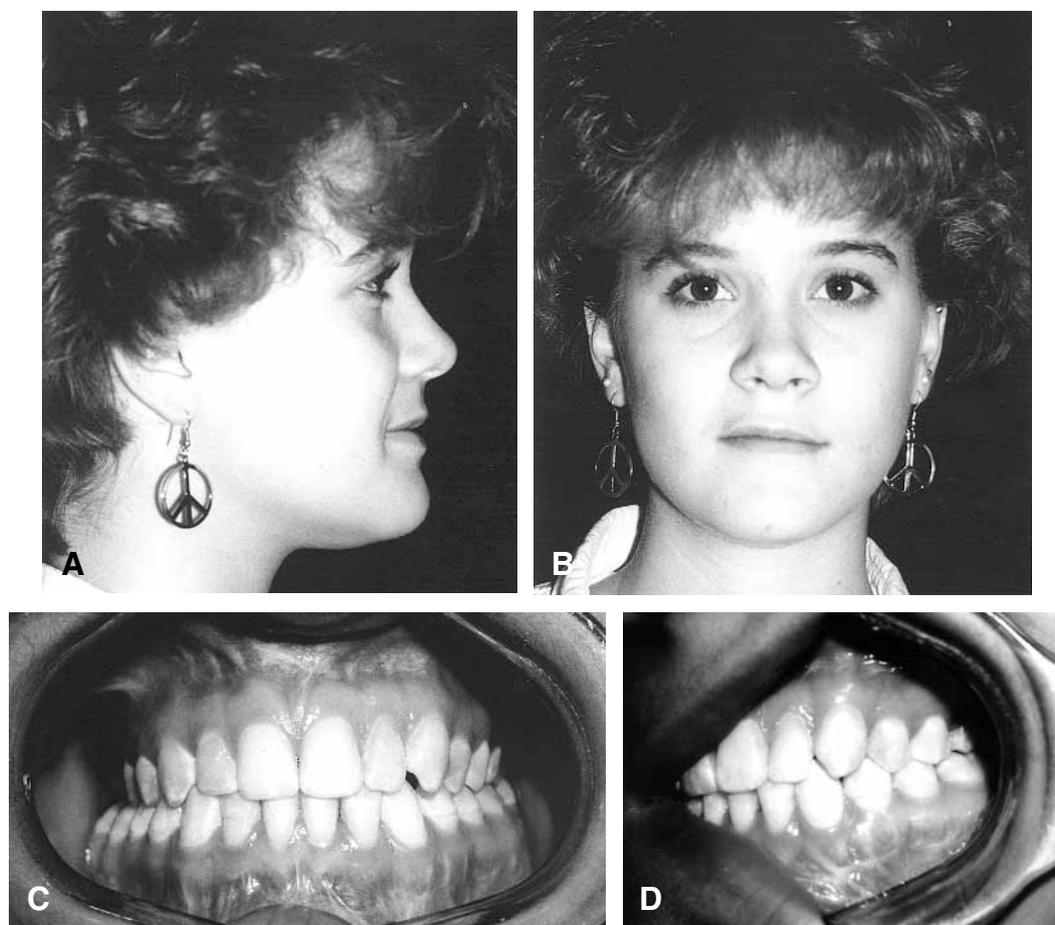


Fig 10. Case 3. **A-D**, Patient at 3 years posttreatment, demonstrating significantly improved facial harmony and good stability of occlusal result.

Table IV. Subjective data (VAS score) for group 2 (n = 25)

	Mean TMJ pain (0 = no pain; 10 = worst pain)	Mean jaw function (0 = normal function; 10 = no function)	Mean diet (0 = no restriction; 10 = liquids only)
Presurgical (T2)	0.6 (range, 0 to 2)	3.6 (range, 0 to 5)	0.7 (range, 0 to 1)
Postsurgical (T4)	0.3 (range, 0 to 2)	2.4 (range, 0 to 5)	0.5 (range, 0 to 1)

of 1.2° from T3 to T4. There was no significant difference ($P > .05$) between the groups when comparing growth before surgery (T1-T2) and the surgical change ($P > .05$). However, there was a significant difference ($P < .02$) between the groups in the amount of change from T3 to T4; some change might be related to splint removal, with forward and upward rotation of the mandible, and settling of the occlusion.

Group 1 had a change in Co-Pg of 2.4 mm from T1 to T2, a change of -4.7 mm from T2 to T3, and a change of 3.5 mm from T3 to T4. Group 2 had a change in Co-

Pg of 4.5 mm from T1 to T2, a change of -5.2 mm from T2 to T3, and a change of 0.8 mm from T3 to T4.

Group 1 had a change in Co-Pt B of 2.3 mm from T1 to T2, a change of -5.8 mm from T2 to T3, and a change of 3.6 mm from T3 to T4. Group 2 had a change in Co-Pt B of 3.6 mm from T1 to T2, a change of -4.5 mm from T2 to T3, and a change of 0.4 mm from T3 to T4 (Table III).

For the Co-Pg and Co-Pt B measurements at T2, there was a statistically significant difference ($P < .05$) between the groups, with group 2 having more active

growth. A nonstatistically significant difference was seen when comparing the surgical changes ($P > .05$). There was a significant difference ($P < .01$) between the groups in the amount of growth from T3 to T4, with group 1 showing greater growth.

Subjective clinical data

These data (Table IV) were available only for the patients who underwent high condylectomy and disc repositioning (group 2), because it is part of our standard records for all patients having TMJ surgery; it is not used for orthognathic surgery patients without TMJ symptoms or pathology. No patients in group 1 had TMJ dysfunction or pain at T2 or T4. The numerical visual analog scale (VAS) indications are 0 = no pain and best function, and 10 = worst pain and worst function.

TMJ pain was not a common symptom at T2. The average VAS scores for group 2 were 0.6 (range, 0-2) at T2 and 0.3 at T4. Two patients had TMJ discomfort for 4 to 6 months at T3, but both were free of pain at T4. No statistically significant difference was found when comparing T2 and T4 values ($P > .05$).

The average VAS scores for jaw function for group 2 were 3.6 at T2 and 2.4 at T4, indicating a slight but not statistically significant improvement ($P > .05$).

No significant dietary restrictions were reported by any patient at T2 or T4. The average VAS scores for group 2 were 0.7 at T2 and 0.5 at T4 ($P > .05$).

The 2 cases of type 2 CH followed the same behavior as the group 2 patients at T2 and T3, with good stable outcomes.

DISCUSSION

It is important to identify the type of growth pattern occurring in CH and to determine if growth is active or inactive. CH usually begins during the second decade of life around the pubertal growth phase and can continue into the middle or late 20s. The specific growth pattern of the condyles, in terms of magnitude, rate, and direction, can influence the timing of surgery and the types of corrective surgical procedures. The basic orthodontic goals are the same as in any conventional orthognathic surgical patient—to align and level the teeth over the basal bone and to remove dental compensations, regardless of the magnitude of skeletal and dental malalignment. In our experience, type 1 CH is much more common than type 2 CH (approximately 15:1 ratio); however, type 1 CH is often undiagnosed because of the lack of understanding that this aberrant condylar growth pattern can create mandibular prognathism. It is often perceived that mandibular prognathism is associated with growth disturbances in the

mandibular body. Although the mandibular body can be affected, the primary stimulus creating the deformity is usually the result of condylar hyperplastic growth.²³ During surgery, we have observed an increased vertical height of the condyle that is covered by the cartilaginous cap. Also, the condylar bone seems softer and more vascular in CH patients, compared with the condyles of age-matched non-CH patients. Patients with arrested CH (the abnormal condylar growth has stopped and become stable) can usually be treated with routine orthodontics and orthognathic surgery. However, active CH cannot be predictably controlled with orthodontics or orthopedic mechanics. There are 3 surgical options for active CH patients. Based on our experience and supported by the results of this study, our choice is option 3.

Treatment option 1. With treatment option 1, corrective surgery is deferred until growth is complete; this often means waiting until the middle or late 20s. Consequently, the patient may suffer from functional problems (mastication and speech), worsening esthetic disfigurement, pain, and psychosocial stigmata associated with a severe facial deformity.^{3,12} Additionally, the magnitude of the deformity, if allowed to fully manifest by this delay in treatment, may preclude an ideal result later. This hyperplastic condylar growth may result in severe deformation of the mandible. Compensatory changes will occur in the maxilla, dentoalveolar structures, and associated soft tissue structures, significantly compromising the clinical treatment outcome.

Treatment option 2. With treatment option 2, orthognathic surgery only is performed during active CH growth, with consideration for overcorrection of the mandible. The accelerated mandibular condylar growth will continue after surgery, and repeat surgery will be needed if the estimated overcorrection is greater or lesser than necessary. Early intervention may benefit the patient, relative to function, esthetics, and psychosocial concerns. With this option, surgery is best performed after most of the maxillary growth is complete (females, 15 years; males, 17 years), to help in estimating how much overcorrection is necessary. Group 1 patients were treated with orthognathic surgery only and were placed in the best occlusion fit at the time of surgery. All patients in group 1 grew into Class III occlusal and skeletal relationships and required additional surgical intervention.

Treatment option 3. Treatment option 3 was used for all patients in group 2, by surgically eliminating further mandibular CH growth with a high condylectomy (removing 3-5 mm of the superior aspect of the condylar head including the medial and lateral poles) (Fig 1) and simultaneous orthognathic surgery.²³ The TMJ

articular disc was repositioned and stabilized to cover the articulating surface of the "new" condyle. Cortical bone reformed over the top of the condyle. We used a mini anchor (Mitek, Norwood, Mass) to stabilize the articular disc to the condyle.³¹⁻³³

The high condylectomy and the disc repositioning procedure can be combined with simultaneous orthognathic surgery to correct the jaw deformity.³⁴ The surgeon may perform this procedure in 1 or 2 stages. If the surgeon is less experienced, the high condylectomy and the disc repositioning can be performed in stage 1 surgery, followed by orthognathic surgery later. When orthognathic and TMJ surgeries are performed in 1 operation, we recommend using the sagittal split ramus osteotomy for the mandible as the procedure of choice because it provides positional control of the condyle, and maintains maximal soft tissue attachments and vascularity to the proximal segment. Other techniques, such as the inverted L or vertical ramus osteotomies, require increased stripping of the periosteum and may lead to vascular compromise of the proximal segment, as well as causing difficulties with positional control of the condyle.

In our experience, surgical correction of bilateral CH can predictably be performed from the ages of 13 in females and 15 in males. The vector of facial growth will change to a vertical direction because the A-P mandibular growth is stopped, but the maxillary vertical alveolar growth will continue until maturation. In unilateral cases, we recommend delaying surgery until the ages of 15 for females and 17 for males, when most of the normal facial growth is complete. A unilateral high condylectomy will arrest growth on the operated side, but normal growth can continue on the contralateral side and could cause development of facial and occlusal asymmetry later if the surgery is performed at a younger age.

Although controversies exist about the stability of mandibular setback to correct mandibular prognathism, we have demonstrated good stability and predictability of results in patients without active CH.³⁵ Numerous other studies have reported relapse for mandibular setback ranging from 20% to 91% of the amount of posterior movement.³⁶⁻⁴⁴ It is possible that the high percentage of relapse is in part due to undiagnosed and untreated active CH in some patients. The results from our study suggest that including the high condylectomy in the treatment of active CH patients significantly improves long-term outcomes. All group 1 patients with active CH treated with orthognathic surgery only grew into Class III occlusal and skeletal relationships and required additional surgical intervention. On the other hand, the patients in group 2 remained stable, with 1

exception involving maxillary transverse relapse with Class II occlusal tendency at T4. The patient required a secondary maxillary surgical intervention, but the mandible remained stable. No patients in group 2 demonstrated any significant growth of the mandible at T3, except for expected appositional growth at pogonion. Long-term follow-up revealed no undesirable changes in subjective and objective jaw function, with maintenance or a slight increase in average MIO and lateral excursion values. No patient reported any significant TMJ pain or dietary restrictions at T4. The 2 patients with type 2 CH showed the same results as the group 2 type 1 CH patients with the same stable outcomes.

Case presentations

Case 1. This 15-year-old male had been followed for 2 years 3 months at T2. Significant accelerated disproportionate growth of the mandible, secondary to type 1 bilateral CH was documented (Fig 2, A-D). The mandible grew forward by 12 mm at pogonion during that 27-month period (Fig 3, A). His diagnosis included (1) bilateral mandibular active type 1 CH, (2) mandibular prognathism, (3) Class III malocclusion, and (4) posterior and anterior crossbites. Treatment included (1) presurgical orthodontics to align and level the arches, placing the teeth over basal bone; (2) surgery (Fig 3, B): bilateral TMJ high condylectomy, repositioning of the articular discs over the condylar stumps, and bilateral mandibular ramus osteotomies to posteriorly reposition the mandible by 8 mm; and (3) postsurgical orthodontics to finish and retain. At 8 years and 4 months after surgery, the patient maintains good skeletal and occlusal stability with good facial balance (Figs 3, C, and 4, A-D).

Case 2. This 16-year-old female had been followed for 2 years 7 months before surgery with Class III mandibular and occlusal asymmetry that was becoming progressively worse (Fig 5, A-D). During this time, her mandibular dental midline shifted more to the left. Her diagnosis included (1) unilateral right type 1 CH, (2) mandibular deviated prognathism, (3) A-P maxillary deficiency (Fig 6, A), (4) Class III malocclusion (right side worse than left), and (5) anterior and left posterior crossbite. Treatment included (1) presurgical orthodontics to align and level the teeth over basal bone; (2) surgery (Fig 6, B): right high condylectomy and disc repositioning, bilateral mandibular ramus osteotomies to rotate the mandible to the right 4 mm and set it posteriorly 5 mm, segmented LeFort I maxillary osteotomy to advance 4 mm and widen 4 mm, and alloplastic chin augmentation of 6 mm; and (3) postsurgical orthodontics to refine the occlusion and retention. At 7 years after

surgery, she demonstrates good facial balance with good skeletal and occlusal stability. (Figs 6, C, and 7, A-D).

Case 3. This 16-year-old female came for correction of her severe dentofacial deformity (Fig 8, A-D). Her diagnosis included (1) unilateral left active type 2 CH, (2) elongation of the left side of the face, (3) excessive elongation and enlargement of the left mandibular condylar head and neck (Fig 9, A), (4) increased mandibular body height on the left side, (5) low mandibular plane angle facial type, (6) compensatory downward growth of the left maxilla (Fig 9, B), (7) left-side open bite, and (8) Class III canine relationship on the left. Treatment included (1) presurgical orthodontics to align and level the teeth over the basal bone; (2) surgery (Fig 9, C): left condylectomy, removing 15 mm of vertical height and reshaping the condylar neck to function as a condyle with repositioning of the articular disc over the condyle, bilateral mandibular ramus osteotomies to reposition the mandible posterior by 9 mm on the left and 5 mm on the right, multiple maxillary osteotomies to level the transverse occlusal plane and to superiorly reposition the left posterior aspect by 5.5 mm and the right side by 3 mm, left inferior border osteotomy, removing 5 to 9 mm to correct the vertical overgrowth of the mandibular body, with preservation of the inferior alveolar nerve (Fig 9, C); and (3) postsurgical orthodontics to finish and retain. At 3 years after surgery, the patient shows the establishment and maintenance of facial balance and occlusal stability (Fig 10, A-D). In severe cases of type 2 CH, a low condylectomy may be indicated to help correct the severe vertical facial imbalance, although only a high condylectomy is necessary to arrest the growth process in CH.

CONCLUSIONS

The purpose of this study was to compare the treatment outcomes and the stability at T4 between patients diagnosed with active CH treated with conventional orthognathic surgery (group 1) and patients treated with high condylectomy and articular disc repositioning (Fig 1) in conjunction with orthognathic surgery (group 2). The results showed a statistically significant difference between CH patients treated conventionally and those who have the additional high condylectomy, demonstrating that a more stable outcome can be achieved at T4 with high condylectomy and articular disc repositioning. Precise removal of the top 3 to 5 mm of the condylar head in CH patients effectively arrests further A-P mandibular growth, without causing any long-term adverse impact on jaw function. Performing a high condylectomy and orthognathic surgery in 1 operation is a stable procedure with a very predictable outcome for surgical treatment of unilateral or bilateral active CH.

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COMMENTARY

This paper reports the use, in a single private practice, of a high condylectomy (removal of the upper 3-5 mm of the mandibular condyles) combined with a bilateral sagittal split osteotomy to manage excessive (though not necessarily unilateral) mandibular growth.

The difficulties with the paper begin with the diagnosis of condylar hyperplasia (CH) for the patients included in this report. A reader who expects CH to mean what it did in essentially all the papers cited in the review of the literature eventually realizes that, for these authors, the term means something different. Traditionally, CH has been used to describe unilateral overgrowth of the mandible accompanied by enlargement or elongation of the condyle on 1 side. The term *hemi-mandibular hypertrophy* has been used to describe such cases in recent years. For this paper, however, 14 of the 27 patients in the sample had symmetric excessive mandibular growth, and only 2 had what the authors called type 2 CH, which they referred to as the classically described form of CH.

The second problem is the diagnosis of CH for this group of patients. As the authors note, what they call "type 1 CH" (25 of the 27 patients in their sample) "is not well accepted as a form of CH by many clinicians." No information is provided as to exactly how the diagnosis of type 1 CH was made or how these patients—the candidates for high condylectomy in addition to ramus osteotomy to set back the mandible—are differentiated from patients who receive ramus osteotomy alone. Because these authors say they have previously demonstrated that mandibular setback surgery to correct mandibular prognathism in nongrowing patients is a very stable procedure (a finding that seems to be unique to them), not all their patients are treated with the high condylectomy. They suggest that other results with mandibular setback are not as good as theirs because other workers have not recognized active CH. That would be easier, of course, if there were recognized ways to do that. The authors say that bone scans are of limited or no value and recommend cephalometric evaluation but do not indicate exactly what one should look for or how to interpret the findings. The reader suspects that, in this practice, if the patient is young and is experiencing more growth in the mandible than in the maxilla, a diagnosis of CH is made just because the jaw is growing; if the patient is old enough for growth to have stopped, he or she either does not have CH or has *arrested CH*, another term used in the paper.

It appears that the clinician on whose practice this paper is based believes that the usual cause of excessive mandibular growth is in fact hyperplasia at the

condyles. This becomes the rationale for combining high condylectomy to remove a growth site in the mandible along with surgery to shorten the long mandible. Such a concept ignores most of the work in recent years on mandibular growth and its control.

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AUTHOR'S RESPONSE

The primary objective of this paper is to educate orthodontists of the causative factors in specific patients with Class III skeletal and occlusal relationships. The excessive Class III growth created by type 1 condylar hyperplasia (CH) can develop during puberty and continue well beyond the normal growth years. Most orthodontists have experienced cases in which the patients "grow out" from their treatment. It would benefit the orthodontist to identify these patients and understand treatment options.

The editor states that there is a lack of information in the paper regarding the diagnosis of type 1 CH or the differentiation from patients who received a ramus osteotomy only. However, this information is clearly provided. The distinct clinical features of types 1 and 2 CH are given on pages 138 and 139, including differential diagnoses. At the bottom of page 140, methods to determine active CH are presented. Diagnosis requires serial records (at 6-to-12 month intervals) to analyze clinical, dental model, and radiographic changes. Some patients may require 2 or 3 years of evaluation to confirm the presence of CH. If the rate of mandibular growth is excessive during and beyond the normal growth years, then CH is present, if the differential diagnosis is negative for other abnormal conditions.

Assessing patients in this manner allows the clinician to distinguish between the different types of Class III patients.

Surgery can provide highly predictable and stable results in treating patients with types 1 and 2 CH (high condylectomies and osteotomies), as well as treating normal growing and nongrowing patients (osteotomies only). Other clinical researchers have recognized relapse rates of 20% to 90% in treating mandibular prognathism. If their technical execution of the surgical procedures is not at fault, then the relapse problems could be due to unrecognized type 1 CH.

Our study included a control group of patients (group 1) diagnosed with CH, but whose treatment consisted of orthognathic surgery without high condylectomy. Although the patients of group 1 were an average of 10 months older than those in group 2 (and should have had most of their growth complete), all patients of group 1 grew back into Class III skeletal and occlusal relationships. However, the patients of group 2 were treated with high condylectomies and orthognathic surgery, and all maintained a Class I skeletal and occlusal relationship postsurgery for an average follow-up of 5 years. This demonstrates that high condylectomies and orthognathic surgery are very effective in treating prognathic patients who have active type 1 CH and those who have type 2 CH with highly predictable and stable outcomes. Orthodontists should be aware of these factors for optimal treatment options.

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