UNESP - UNIVERSIDADE ESTADUAL PAULISTA
FACULDADE DE ODONTOLOGIA DE ARARAQUARA

ADRIANO PORTO PEIXOTO

INFLUÊNCIA DA HIPERPLASIA CONDILAR NO CRESCIMENTO CRANIOFACIAL E ESTABILIDADE DO TRATAMENTO DE PACIENTES CLASSE III PÓS SURTO DE CRESCIMENTO PUBERTÁRIO.

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Tese apresentada ao Programa de Pós-Graduação em Ciências Odontológicas - Área de Ortodontia, da Faculdade de Odontologia de Araraquara, da Universidade Estadual Paulista "Júlio de Mesquita Filho" para a obtenção do título de Doutor em Ciências Odontológicas.

Orientador:
Prof. Dr. João Roberto Gonçalves

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Araraquara, 4 de março de 2013.

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"Talvez não tenha conseguido fazer o melhor, mas lutei para que o melhor fosse feito. Não sou o que deveria ser, mas Graças a Deus, não sou o que era antes".

Martin Luther King

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Dedico esse trabalho...

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RESUMO

Peixoto AP. Influência da hiperplasia condilar no crescimento craniofacial e estabilidade do tratamento de pacientes classe III pós surto de crescimento pubertário [tese de doutorado]. Araraquara: Faculdade de Odontologia da UNESP; 2013.

Resumo

A estabilidade do tratamento cirúrgico da classe III tem sido uma preocupação com relatos de pequenas ou significativas alterações mandibulares pós-cirúrgicas, Alguns pacientes afetados pela instabilidade do tratamento podem manter relação oclusal aceitável com compensações ortodônticas pós-cirúrgicas, mas outros irão desenvolver alterações oclusais e esqueléticas de forma significativa, necessitando de intervenção adicional ortodôntica e cirúrgica gerando custo adicional e aumento da morbidade. Muitas variáveis têm sido apontadas como preditoras da instabilidade do tratamento, mas poucos estudos sugerem a hiperplasia condilar (HC) como um fator de risco para a correção cirúrgica da Classe III. 51 pacientes classe III foram selecionados consecutivamente de uma população de pacientes operados entre 1996 e 2007 por um único cirurgião. Telerradiografias em norma lateral foram selecionadas nas seguintes fases: diagnóstico inicial, imediatamente antes da cirurgia, imediatamente após a cirurgia e com pelo menos um ano de acompanhamento pós-cirúrgico. Um total de 19 pontos de referência foram identificados e digitalizados através da utilização do software DFPlus (Dentofacial Software Inc, Toronto, Ontario, Canada). Estes pontos de referência

foram utilizados para calcular 21 medidas lineares e/ou angulares e 10 medidas horizontais e verticais. A amostra que apresentava classe III associada a hiperplasia condilar obteve maior taxa de crescimento mandibular total pós puberal (2,6 mm) e maior comprimento do corpo (2,2 mm). Diante de uma má oclusão esquelética de classe III associada a HC, a adoção de uma abordagem de tratamento diferenciada é de fundamental importância para a obtenção de resultados estáveis, embora os tratamentos que envolvam a manipulação da articulação temporomandibular (ATM) sejam vistos com cautela por muitos profissionais da área de cirurgia, para algumas patologias articulares, como a hiperplasia condilar, é necessário a intervenção cirúrgica na ATM em conjunto com a cirurgia ortognática para que resultados previsíveis em longo prazo sejam obtidos, evitando assim uma segunda cirurgia para a correção definitiva do problema.

Palavras-chave: côndilo mandibular; má oclusão de Angle classe III; cirurgia ortognática

ABSTRACT

Peixoto AP. Influence of condylar hyperplasia on post pubertal class III patients craniofacial growth and treatment stability [tese de doutorado]. Araraquara: Faculdade de Odontologia da UNESP; 2013.

Abstract

Class III surgical treatment stability has been a concern with reports of minor or significant post-surgical mandibular change. Most of the patients affected by treatment instability may retain acceptable oclusal relation while some of the skeletal position changes. Other affected patients have to undergo further treatment generating additional cost and increased morbidity. Many variables have been identified as predictors of treatment instability, but few reports suggest condylar hyperplasia as a risk factor for class III surgical correction. 51 class III patients records were consecutively selected from the patient population operated between 1996 and 2007 by the senior author. Lateral cephalometric radiographs were selected at the following periods: initial, immediate before surgery, immediate after surgery and at least one year follow-up). A total of 19 landmarks were identified and digitized using DFPlus software (Dentofacial Software Inc, Toronto, Ontario, Canada). These landmarks were used to compute 21 traditional linear and/or angular measurements and 10 horizontal and vertical measurements. During the observational period in the present sample condylar hyperplasia

promoted higher post pubertal total mandibular growth rate (2.6 mm) and higher corpus length (2.2 mm). Appropriate condilar hyperplasia diagnosis and customized approach are mandatory in order to obtain stable results for Class III surgical treatment. Although treatments that involving manipulation of the temporomandibular joint to be viewed with caution by many professionals in the field of surgery, for some articular pathologies, such as condylar hyperplasia, it is necessary to approach the TMJ together with orthognathic surgery so that stable long term results are obtained, thereby avoiding a second surgery for the final remediation of the problem.

Key words: mandibular condyle; malocclusion, Angle Class III; orthognathic surgery

SUMÁRIO

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INTRODUÇÃO

1 INTRODUÇÃO

Pacientes com más oclusões esqueléticas de Classe III são tratados convencionalmente através da associação entre ortodontia e cirurgia ortognática. Porém, a estabilidade dos resultados obtidos com o tratamento muitas vezes tem se apresentado pouco previsível, com relatos de pequena ou significativa mudança mandibular pós-cirúrgica[2,3,12,21-22].

Alguns pacientes afetados pela instabilidade do tratamento podem manter relação oclusal aceitável com compensações ortodônticas pós-cirúrgicas, mas outros irão desenvolver alterações posicionais oclusais e esqueléticas adversas, de magnitude significativa. Esses pacientes podem necessitar de intervenção adicional ortodôntica e cirúrgica gerando custo adicional e aumento da morbidade.

O crescimento anormal do côndilo mandibular pode afetar gravemente a oclusão e a harmonia facial. Algumas condições patológicas podem predispor essas alterações, como malformações congênitas associadas a distúrbios de crescimento, microssomia hemifacial (ou espectro óculo-aurículo-vertebral ou Síndrome de Goldenhar), distúrbios de crescimento primário, doenças ou lesões adquiridas que estão associados com as desordens do crescimento[18].

Muitas variáveis têm sido apontadas como preditoras da instabilidade do tratamento, porém, poucos estudos têm sugerido a hiperplasia condilar como um fator de risco para a correção cirúrgica da classe III[28-29].

A Hiperplasia condilar (HC), condição patológica descrita pela primeira vez em 1836 por Adams, caracteriza-se pelo desenvolvimento excessivo do processo condilar, criando assim, importantes deformidades estéticas e funcionais que resultam na projeção para frente do corpo e ramo mandibular do lado afetado com uma inclinação para baixo dos planos oclusal e mandibular e mordida cruzada[14,28].

Obwegeser, Makek[17], diferenciam o termo "hiperplasia condilar" em três tipos: hiperplasia hemimandibular (HH), alongamento hemimandibular (AH) e uma forma híbrida. Hiperplasia hemimandubular (HH), é caracterizada por um lado da mandíbula tridimensionalmente aumentado, normalmente com o crescimento anormal da mandíbula terminando ao mesmo tempo em que termina o crescimento corporal. O alongamento hemimandibular (AH) é caracterizado por um deslocamento horizontal da mandíbula para o lado contralateral. Os ramos horizontais em ambos os lados estão no mesmo nível.

Wolford[28] desenvolveu uma classificação para a hiperplasia condilar através da diferenciação etiológica entre os processos patológicos. O sistema de classificação proporciona uma indicação das alterações histológicas, a natureza patológica do processo de crescimento anormal e considerações do tratamento que são mais previsíveis para eliminar a patologia, bem como corrigir a deformidade oclusal e facial. A classificação também reflete a taxa de ocorrência da HC tipo 1A, sendo a patologia que mais comumente ocorre.

HC tipo 1: Esta condição se desenvolve durante a puberdade, sendo caracterizada por um crescimento acelerado e prolongado além do mecanismo normal de crescimento condilar, podendo ocorrer bilateralmente (CH tipo 1A) ou unilateralmente (HC tipo 1B), é auto-limitante, mas o crescimento pode continuar em média até os 20 anos.

HC tipo 2: Estas patologias condilares são causadas por tumores benígnos, podendo se desenvolver em qualquer idade. São aumentos condilares unilaterais que causam deformidades verticais com alongamento facial. Os tumores condilares mandibulares mais comuns que ocorrem são osteocondroma (HC tipo 2A) e osteoma (HC tipo 2B).

HC tipo 3: São outras condições raras, sejam elas benígnas (HC tipo 3A) ou malígnas (tipo HC 3B) que podem causar alargamento condilar.

Hiperplasia Condilar tipo 1 é o foco deste trabalho. Clinicamente, pacientes com HC tipo 1 com crescimento ativo podem apresentar uma piora progressiva da sua mordida e aparência facial, com a mandíbula se tornando mais prognata. HC do tipo 1A pode apresentar-se com o crescimento mandibular acelerado de forma simétrica, ou um lado pode crescer mais rapidamente do que o lado oposto criando assimetria mandibular (prognatismo com desvio mandibular) assim como também ocorrerá com a HC tipo 1B (HC unilateral). A oclusão geralmente reflete uma relação de classe III com mordida cruzada anterior e posterior.

Radiograficamente, a cabeça e o pescoço da mandíbula são alongados, assim como o corpo da mandíbula. A cabeça do côndilo terá uma aparência relativamente normal. Em condições de crescimento acelerado, o ângulo goníaco pode tornar-se mais obtuso e a altura vertical do corpo mandibular posterior pode estar reduzida verticalmente, contribuindo para uma morfologia elevada do plano facial mandibular. Os incisivos inferiores podem estar inclinados lingualmente e nos casos mais graves, podem se desenvolver espaços entre os dentes anteriores.

Sua etiologia é controversa e não totalmente esclarecida. Algumas teorias para explicar as causas da hiperplasia condilar sugerem trauma[4, 9,25-28], hereditariedade[4,9,25-28], infecção[4,25,28], neoplasia[9,25], distúrbios hormonais[4,26-28] e fatores intra-uterinos[28] .

O crescimento condilar excessivo e/ou por tempo mais prolongado do que o esperado é provavelmente causado pela atividade persistente de células pré-cartilaginosas da zona de crescimento condilar[11], embora outros autores sugerem uma mudança local no controle dos hormônios de crescimento celular[5].

Seu surgimento ocorre geralmente durante a puberdade sendo que raramente inicia-se após os 20 anos[8-9].

Crescimento mandibular acelerado iniciado ao final da adolescência ou mais tarde, após os 20 anos de idade, está mais freqüentemente relacionado à HC tipo 2 (osteocondroma ou osteoma)[28].

A Hiperplasia condilar pode ocorrer unilateralmente ou bilateralmente, embora os casos unilaterais sejam mais freqüentemente descritos na literatura[4,26].

Alguns estudos indicam uma prevalência ligeiramente aumentada para o gênero feminino[5,9,11,16,24,27].

Na Hiperplasia condilar do tipo 1, os pacientes podem apresentar sintomas de DTM (dor na ATM, dores de cabeça, ruídos articulares, etc), mais comumente vistos na HC do tipo 1B, do tipo 2, ou na HC tipo1A com crescimento assimétrico. Sintomas na ATM são menos comuns em casos simétricos de HC tipo 1A.

A taxa de crescimento puberal normal para mulheres é de 1,6 mm por ano (medindo de condílio ao ponto B), e geralmente até a idade de 15 anos cerca de 98% está completa. Para os homens, a taxa de crescimento é de 2,2 mm por ano, com 98% de crescimento completo entre 17 a 18 anos[23].

Para a HC do tipo 1, esta taxa de crescimento pode estar acelerada. Alexander et. al.[1] em 2009, relatam que a taxa normal de crescimento mandibular pós-puberal para a classe III esperada entre 15 e 16 anos é de 1,5 mm a 2 mm, em média, para homens e mulheres, medindo do condílio ao gnátio No entanto, eles não separaram os pacientes classe III com HC ativa dos pacientes sem HC.

O diagnóstico da HC ativa pode ser determinado através de documentação seriada, preferencialmente em intervalos de no mínimo 12 meses, constando de: avaliação clínica, modelos de estudo obtidos em relação cêntrica e sobreposições radiográficas.

Superposição de séries radiográficas (telerradiografias) ou medições digitais de tomografias do côndilo e corpo da mandíbula podem ser empregadas

como diagnóstico de HC ativa do tipo 1, caso seja observada aumento em comprimento das estruturas esqueléticas .

Cintilografia óssea[10-11,19] e SPECT (tomografia computadorizada por emissão de fóton único)[20], também tem sido empregados para o diagnóstico.

A cintilografia óssea com pirofosfato de tecnécio, tecnécio de metileno 99 ou cloreto de metileno difosfanato 99 podem detectar crescimento ativo no côndilo através da identificação de áreas com maior atividade dos osteoblastos através da captação do marcador radioativo.

A cintilografia, no entanto, parece ser mais eficaz em casos unilaterais e, se aplicado somente após o crescimento normal do indivíduo ter cessado. No entanto, a cintilografia óssea pode ser inconclusiva em pacientes mais jovens. As radiografias de mão e punho não têm valor no diagnóstico da HC, pois a mandíbula continua a crescer além do período de crescimento normal[28].

Os objetivos básicos do tratamento ortodôntico para uma má oclusão esquelética de classe III associada a HC são os mesmos a serem adotados a um paciente classe III sem HC em preparo para uma cirúrgica ortognática convencional (alinhamento e nivelamento dos dentes ao longo da base óssea, visando remover compensações dentárias, independentemente da magnitude do desalinhamento esquelético e dentário)[28].

No entanto, a HC ativa não pode ser tratada de forma previsível apenas com ortodontia e cirurgia ortognática convencional.

Falha em reconhecer esta entidade patológica, pode resultar em resultados funcionais e estéticos desfavoráveis após o tratamento com ortodontia e cirurgia ortognática, caso o fator HC seja ignorado[29].

O diagnóstico e o tratamento de pacientes com hiperplasia condilar unilateral ou bilateral ainda não é um consenso entre clínicos e pesquisadores[6-7,13,15,29].

A falta de evidências científicas comprovadas através de ensaios clínicos bem controlados limita o desenvolvimento e adoção de um protocolo de tratamento de consenso clínico.

Deste modo, devido à falta de evidências científicas comprovadas através de ensaios clínicos bem controlados, tornam-se necessários estudos que propiciem o desenvolvimento e adoção de um protocolo de tratamento que se baseie no adequado diagnóstico dos fatores que podem estar associados às más oclusões esqueléticas de classe III, para que se obtenham resultados estáveis em longo prazo.

PROPOSIÇÃO

2 PROPOSIÇÃO

2.1 OBJETIVO GERAL

Avaliar comparativamente a influência da hiperplasia condilar no crescimento mandibular pós pubertário de pacientes com más oclusões esqueléticas de classe III e a estabilidade da cirurgia ortognática associada ou não a condiloplastia simultânea.

2.2 OBJETIVOS ESPECÍFICOS

Os objetivos específicos da presente pesquisa serão testar as seguintes hipóteses (H0):

1. A hiperplasia condilar não influencia o crescimento mandibular pós pubertário de pacientes com más oclusões esqueléticas de Classe III associada a HC;

2. Pacientes com más oclusões esqueléticas de Classe III tratados com cirurgia ortognática exclusiva, apresentam a mesma estabilidade, independentemente da presença de hiperplasia condilar.

3. A condiloplastia associada à cirurgia ortognática no tratamento de pacientes Classe III esquelética com hiperplasia condilar, não promove incremento na estabilidade dos resultados obtidos;

ARTIGO 1

3- ARTIGO 1

Influence of condylar hyperplasia on post-pubertal growth in class III patients.*

*Artigo a ser enviado para publicação no periódico:

Journal of Oral and Maxillofacial Surgery

Purpose: The aim of this study was to evaluate the influence of condylar hyperplasia in the post pubertal mandibular growth of class III patients one year before surgery.

Materials and Methods: 40 skeletal Class III patients consecutively selected were paired in two Groups (Group WCH = without condylar hyperplasia and Group CH = active unilateral or bilateral condylar hyperplasia) according to gender, age and craniofacial pattern. Lateral cephalograms were traced and digitized using DFPlus software at the initial observation (T1) and immediate before surgical treatment (T2). Significant cephalometric differences during the one year follow up period (T2-T1) were compared between the two paired Groups. Analysis of covariance was used to adjust for the potential confounding factors (age and gender at the initial observation).

Results: During the observational period, Group WCH mean differences were smaller than Group CH for all variables that indicated sagital mandibular position: SNB (mean = -1.1; sd = 0.17 degrees), SN.Pog (mean = 0.72, sd = 0.16 degrees), Go-Me (mean = -1.99, sd = 0.39 mm), Ar-Gn (mean = -2.5, sd = 0.37 mm) and S-Gn (mean = -1.9, sd = 0.45 mm).

Conclusions: After controlled by age and gender differences between Groups, our paired sample showed that condylar hyperplasia promotes higher total mandibular (Ar-Gn) and corpus length (Go-me) growth for skeletal Class III patients during post-pubertal period.

Key words: diagnosis; mandibular condyle; growth & development; orthognathic surgery; malocclusion, Angle Class III

Introduction

While condylar growth does not have a major role in normal overall mandibular growth as previously thought, abnormal condylar growth has a dramatic effect on facial morphology. Abnormal mandibular condylar growth can severely affect the occlusion and facial harmony. This is easily demonstrated by many unilateral abnormal condylar conditions as hemifacial microsomia (oculo-auriculo-vertebral spectrum) and acquired diseases or injuries that are associated with growth disorders[1].

Class III surgical treatment stability has been a concern with reports of minor or significant post-surgical mandibular instability[2-6]. While many variables have been pointed out as predictors for treatment instability, very few reports have been suggested condylar hyperplasia as a risk factor of class III surgical correction[7-9].

Obwegeser and Makek[10], differentiate the term "condyle hyperplasia" in three different types: hemimandible hyperplasia (HH), hemimandible elongation (HE) and a hybrid form. Hemimandible hyperplasia (HH) which is characterized by an increased three-dimensional side of the jaw, usually with the abnormal growth of the jaw ending at the same time as it completes the overall growth. Hemimandibular elongation (HE) is characterized by horizontal displacement of

the mandible to the contralateral side. The vertical position of the mandibular body and occlusal plane horizontal branches on both sides are on the same level.

Wolford's Condylar Hyperplasia (CH) Classification[8]

The term condylar hyperplasia (CH) indicates an enlarged condyle of which there are a number of pathological etiologies. Wolford developed a classification for condylar hyperplasia differentiating between the etiologies of the pathological processes. The classification system provides an indication of the histological findings, nature of the pathology, abnormal growth process and treatment considerations that are most predictable to eliminate the pathology as well as correct the occlusal and facial deformity. The classification also reflects the occurrence rate with CH type 1A, being the most commonly occurring pathology.

CH type 1: This condition develops during puberty, is an accelerated and prolonged growth aberration of the normal condylar growth mechanism, can occur bilaterally (CH type 1A) or unilaterally (CH type 1B), and is self limiting, but growth can continue into the med 25's.

CH type 2: These condylar pathologies are caused by benign tumors, can develop at any age, and are unilateral condylar enlargements that cause unilateral vertical facial elongation deformities. The most common occurring mandibular condylar tumors are osteochondroma (CH type 2A) and osteoma (CH type 2B).

CH type 3: These are other rare benign (CH type 3A) and malignant (CH type 3B) conditions that can cause condylar enlargement.

CH type 1 is the focus of this paper. It is characterized by accelerated and prolonged growth of the "normal" growth mechanism of the condyle developing elongation of the condylar head and neck as well as the body of the mandible. This creates significant functional and esthetic deformities which commonly results in a horizontal forward projection of the mandible, body and ramus on the affected side(s), anterior and posterior crossbites, and sometimes increased inclination of the occlusal plane[8, 11].

Clinically, CH type 1 patients with active growth may present with a progressive worsening of their bite and physical appearance with the mandible becoming more prognathic. CH type 1A can present with symmetric accelerated mandibular growth, or one side can grow faster than the opposite side creating mandibular asymmetry (deviated prognathism) as will also occur with CH type 1B (unilateral CH). The occlusion will usually reflect classes III relationship with anterior and posterior cross bites.

Radiographically, the condylar head and neck are elongated as well as the mandibular body. The crown of the condyle will have a relatively normal appearance. In the faster growing condition, the gonial angle may become more obtuse and the vertical height of the posterior mandibular body may be decreased contributing to a high mandibular plane facial morphology. The lower incisors may be in lingual inclination and in the more severe cases; spaces may develop between the anterior teeth.

Its etiology is controversial and not completely understood. Some theories to explain the causes of condylar hyperplasia suggest trauma[8, 12-16], heredity[8, 12-16],

infection[8, 12, 14], neoplasia[13, 14] , hormonal disorders[8, 12, 15, 16] and intrauterine factors[8].

Condylar hyperplasia can occur bilaterally or unilaterally. However, unilateral cases are more frequently described[12, 14, 15], bilateral affected patients experience similar relevance[8, 9]. Some studies indicate a slightly increased prevalence of the female gender[13, 17, 18].

Condylar hyperplasia onset usually occurs during puberty and rarely begins after the twenties[13, 19].

Accelerated mandibular growth emergence after the age of 20 is most often related to an osteochondroma or osteoma[8] . Occasionally patients may present with TMJ symptoms, although they are unusual.

The diagnosis can be determined through serial documentation, preferably at intervals of at least 12 months, consisting of: physical examination, study models obtained in centric relation and radiographic overlays.

Bone scintigraphy[20-22] and SPECT (single photon emission computer tomography)[23] , have been recomended for diagnosis also.

Bone scintigraphy with technetium pyrophosphate, technetium 99 methylene or methylene difosfanato 99 can detect active growth in the condyle through the identification of areas with increased osteoblastic activity through the uptake of the radioactive marker. Scintigraphy, however, seems to be more effective in unilateral cases and, if applied only after the individual's normal growth has ceased. However, bone scans can be inconclusive in younger patients. Hand and

wrist radiographs have no value in the diagnosis of CH as the jaw continues to grow beyond normal growth period[8].

The lack of scientific evidence presented in well-controlled clinical trials limits the development and adoption of a consensus clinical treatment protocol. The diagnosis and treatment of patients with bilateral condylar hyperplasia is not yet a consensus among clinicians or researchers[9, 24]. The aim of this study was to evaluate the influence of condylar hyperplasia in the post pubertal mandibular growth of class III patients one year before surgery and contribute to the present knowledge regarding the relevance of condylar hyperplasia diagnosis on the treatment of skeletal Class III patients.

Materials and Methods

One hundred and fifty one class III patient records were consecutively evaluated from the patient population operated between 1996 and 2007 by the senior author (LMW). From the initial sample 104 patients had adequate records (lateral cephalometric radiographs). Among them, 56 patients were diagnosed as active condylar hyperplasia by scintigraphy[13], serial clinical assessments, dental model and radiographic evaluations with lateral cephalograms superimpositions[8, 9]. Forty-eight patients (29 females, 19 males) met the inclusion criteria described bellow for this retrospective study. Eight patients diagnosed as active condylar hyperplasia refused to have high condylectomies and were analyzed in a separate paper. The remaining 40 patient records were paired according to gender, age and craniofacial pattern in two groups. Group WCH had no condylar hyperplasia and

was submitted to orthognathic surgery only and Group CH was diagnosed with active condylar hyperplasia and had high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure.

The following inclusion criteria were adopted: 1) patients with skeletal Class III malocclusion based on clinical observation and dental models (molar relationship); 2) at least one year follow-up before surgery; 3) at least one year follow-up after surgery (to be addressed in a separate paper); 4) females at least 13 and maximum 23 years old and males at least 17 and maximum 23 years old; 5) adequate records at the following periods: initial, immediate before surgery, immediate after surgery and at least one year follow-up. Patients were rejected based on the following criteria: 1) absence of one or more inclusion criteria 2) previous surgical intervention in the craniofacial area; 3) presence of craniofacial syndrome; 4) inadequate or poor-quality records (radiographs). Initial evaluation (T1) and pre surgical (T2) lateral cephalometric radiographs were selected for the present study.

This research has been approved by Research Ethics Committee of the Araraquara Dental School under protocol number 73/10.

Cephalometric Measurements

Standardized lateral cephalometric radiographs (Quint sectograph; American Dental Co, Hawthorne, CA) were randomly traced and digitized twice by a single investigator (APP) approximately 1 week apart. The average values for the 2 replicates were used to decrease landmark technical errors. A total of 19

landmarks were identified (Fig 1) and digitized using DFPlus software (Dentofacial Software Inc, Toronto, Ontario, Canada). These landmarks were used to compute 21 traditional linear and/or angular measurements and 10 horizontal and vertical measurements. After collection, all data were transferred to SPSS software (version 16.0, SPSS, Chicago, I11) for statistical analysis.

Statistical Analysis

Dental and skeletal cephalometric differences and similarities among the groups were evaluated with Student t-test. Levene test was used to check homoscedasticity (lack of significant difference variances between groups).

Considering individuals in the sample had a large range of age and some might be still growing, the observed measurement changes could be correlated with age and associated with gender, significant average changes in each group were evaluated by analysis of the covariance model:

$$y_{ijk} = \mu + \mu_{1i} X_{1i} + \mu_2 X_2 + \beta Z_{ijk} + \varepsilon_{ijk}$$

y_{ijk} = y variable of the k individual difference from group i and gender j during T1 and T2.

μ = constant;

μ_{11} = effect attributed to bilateral hyperplasia and and

$$X_{11} = \begin{cases} 1 & \text{to individual from bilateral hyperplasia} \\ 0 & \text{in another case} \end{cases}$$

μ_{12} = effect attributed to unilateral hyperplasia and

$$X_{12} = \begin{cases} 1 & \text{to individual from unilateral hyperplasia} \\ 0 & \text{in another case} \end{cases}$$

μ_2 = effect due to gender and

$$X_2 = \begin{cases} = 0 & \text{for males} \\ = 1 & \text{for females} \end{cases}$$

β = coefficient of the covariate age (Z).

Method error was evaluated in 20% of the sample randomly chosen to assess intraexaminer reliability using ICC (Intraclass Correlation Coefficient) analysis. All measurements were done twice one week apart.

Results

Reproducibility tested by Intraclass Correlation Coeficient (ICC) showed high confiability (range 0.989 – 0.999) for all variables.

Group Similarities and Differences

All groups were successfully paired. Seven males and thirteen females in each group had overall initial (T1) mean age of 17.4 and 17.2 years old in Group WCH and CH respectively. Among the females, mean age were 16.4 years old for both groups and among the males mean age were 19.2 and 18.7 years old for Group WCH and CH respectively. Follow-up period were 12.9 and 13.3months for Group WCH and CH respectively (Table 1).

The sample was appropriated paired according to skeletal and dental characteristics. Cephalometric comparison between Group WCH (class III only) and Group CH (class III with unilateral or bilateral condylar hyperplasia) showed no significant differences among the variables average values at the initial observation period (Table 2).

Net Skeletal Changes

Mean follow-up period was 12.9 and 13.3 months for Group WCH and Group CH respectively (Table 1). During the observational period, Group WCH mean skeletal changes were smaller ($p<0.001$) than Group CH for all variables indicating sagital mandibular position: SNB (1.1±0.17), SN.Pog (0.72±0.16), Go-Me (1.99±0.39), Ar-Gn (2.5±0.37) and S-Gn (1.9±0.45). Overbite (OB) mean skeletal changes was bigger ($p<0.036$) for Group WCH than Group CH (Table 3).

Adjusted model for gender, age and condylar hyperplasia (unilateral, bilateral or absence)

Since Group CH (condylar hyperplasia) joined unilateral (n=10) and bilateral (n= 10) affected patients, this group was evaluated in two distinct subgroups for cephalometric differences during the follow-up period.

In order to evaluate possible influence of gender and age between groups (presence or absence of condylar hyperplasia) an analysis of covariance was performed (Table 4a). The adjusted models showed that SN.Pog and Go-Me changes were explained exclusively by group differences (presence of condylar

hyperplasia). Group and age at the initial of the evaluation explained SNB, Ar-Gn and S-Gn changes. The behavior of the variable S-Ar was explained exclusively by patient age at the beginning of the evaluation.

The data in Table 4b indicate that in conditions of equal gender and age we expect a change in variable SNB for groups with bilateral and unilateral CH of about 1 degree (μ_{11} = 1.09 and μ_{12} = 1.13 respectively) compared to the group without CH. For the variable SN.Pog expect a reduction for groups with bilateral and unilateral CH of about 0.7 degrees (μ_{11} = - 0.79 and μ_{12} = - 0.67 respectively) in relation to the group without CH.

For variables Go-Me, Ar-Gn and S-Gn, increases are expected (μ_{11} = 2.29 and μ_{12} = 1.61) (μ_{11} = 2.38 and μ_{12} = 2.53) (μ_{11} = 1.88 and μ_{12} = 1.83) respectively in relation to the group without CH in conditions of equal gender and age. To the variable S-Ar, with increasing age can be expected to increase by approximately 0.08 mm (β = 0.08).

The covariance test to evaluate group and subgroups behavior showed that Group WCH (no condylar hyperplasia) differed significantly from bilateral condylar hyperplasia and unilateral condylar hyperplasia subgroups (SNB, SN.Pog, Go-Me, Ar-Gn and S-Gn). Unilateral and bilateral condylar hyperplasia subgroups did not differ significantly (Table 4a).

Discussion

To our knowledge, this is the first retrospective study that compared paired class III post-pubertal growth and the influence of condylar hyperplasia. The sample used was carefully paired according to gender, age, craniofacial pattern and follow-up period following a strict protocol made to avoid bias. Among 151 patient records that met inclusion criteria selection, 48 could be paired and 2 groups of 20 were used for the present study. Age, gender, craniofacial pattern and follow-up period mean differences among the groups showed minor differences.

Main limitations of the study were the lack of skeletal age evaluation, inclusion of unilateral and bilateral condylar hyperplasia patients in the same sample and bidimensional analysis method. Indeed those parameters should have been addressed but it was not possible due to the retrospective character of the study.

Hand-wrist radiographs were not available for the present sample. Cervical vertebral maturation analysis was inconclusive and reliability was questioned and therefore was not used. Cervical vertebral maturation assessment to determine skeletal maturation has been questioned before for the average age used in the present sample[25, 26].

Sagital evaluation through lateral cephalometric analysis misses unilateral growth magnitude. Besides the fact of half of the condylar hyperplasia group was unilateral, sagital evaluation was enough to show significant mandibular growth during the observational period.

In the present study, Group WCH (non active condylar hyperplasia) experienced no significant overall mandibular growth (Ar – Gn) during the period of evaluation (12.9 months) while Group CH (active condylar hyperplasia) showed 2.61 mm (sd = 1.59) average growth during a similar period of time (13.3 months). Active condylar hyperplasia affected significantly total mandibular growth in the present paired sample comparison. The average total mandibular growth observed in the active condylar hyperplasia group is observed only in much younger class III samples in an annually rate[27, 28]. The annual amount of residual mandibular growth in skeletal class III patients after the pubertal spurt (15-16 years old) has been emphasized and estimated to be 1.5 to 2.0 mm respectively for girls and boys[27].

Corpus length (Go-Me) changes has been pointed out as a matter of mandibular remodeling rather than condylar growth. Posterior ramus bone deposition would explain such corpus growth rather than condylar activity[28]. Surprisingly, in our sample, Group CH (condylar hyperplasia) corpus length (Go-Me) changed significantly over the period of observation an average of 2.18 mm. Such average growth rate observed in Group CH only (17.2 years old at the beginning of observation), could not be explained by condylar hyperplasia only since total mandibular change (Ar-Gn = 2.61, sd = 1.59) observed was just 0.43 mm greater than corpus length (Go-Me = 2.18, sd = 1.68). Ramus height (Ar-Go) showed no significant change in either group. A possible explanation could be posterior ramus remodeling exacerbated by condylar hyperplasia, as a response of altered function and soft tissue stretching.

In the vertical dimension, both groups showed similar behavior of the anterior facial height (N-Me) with no significant changes observed.

The influence of unilateral and bilateral condylar hyperplasia patients in Group CH, age at the beginning of the evaluation and gender were assessed by covariance analysis.

The analysis of the adjusted model for bilateral or unilateral condylar hyperplasia did not affect the average results significantly (Table 5). It was observed that the lack of condylar hyperplasia (Group WCH) showed overall significantly less mandibular change than bilateral and unilateral condylar hyperplasia patients (SNB, SN.Pog, Go-Me, Ar-Gn and S-Gn). Although the present study was not designed to evaluate the influence of condylar hyperplasia affected side, it was possible to observe that unilateral and bilateral condylar hyperplasia affected mandibular average sagital changes similarly (Table 5). The vast majority case reports or clinical series of mandibular condylar hyperplasia studies seen in the literature addressed unilateral condylar hyperplasia patients[15, 16, 29, 30].

Indeed, very few clinicians and researchers search and diagnose bilateral condylar hyperplasia. Failing in properly identification of bilateral condylar hyperplasia leads to relapse after surgical correction that may be interpreted as normal growth after surgery and earlier operation than ideal for predictable results[8, 9].

Difficulty in bilateral condylar hyperplasia patient identification might be related to the scarce of bilateral condylar hyperplasia reports in comparison with

unilateral affected patient reports. The latter form of condylar hyperplasia, in fact, present much more visible and distinct than normal class III mandibular growth.

Total group mean age at the beginning of evaluation was 17.4 and 17.2 years old for Group WCH and Group CH respectively. Female patients mean age were 16.4 years old in both groups while male patients were 19.2 and 18.7 years old for Group WCH and Group CH respectively.

There are few studies that addressed post-pubertal mandibular growth mainly for the timeframe and age evaluated in the present study[27, 28, 31-33].

In the present sample, female mean age (16.4 years old) would be between CVM 5 and CVM 6 at the beginning of the observational period. All male patients included would be classified according to CVM 6 or older[32].

The covariance analysis of the adjusted model for age, gender and condylar hyperplasia showed that only the variables SN.Pog and Go-Me significant changes were explained by the presence of condylar hyperplasia alone. SNB, Ar-Gn and S-Gn significant changes were explained by the presence of condylar hyperplasia and age at the beginning of the evaluation (Table 4). Indeed, an older sample could be less influenced by age than the present sample but active condylar hyperplasia decreases in older patients and most of the patients who seek class III surgical treatment do not want to postpone treatment further than the present sample average age due to aggressive facial deformity and social consequences. Regardless the fact that the present sample inclusion criteria did not allow us to attribute all significant changes to the presence of condylar hyperplasia, two variables that well expressed total mandibular change (SN.Pog)

and mandibular body change (Go-Me) were explained by the presence of condylar hyperplasia only in the adjusted model. SN.Pog changed approximately 20 times more among the bilateral or unilateral condylar hyperplasia patients than class III only patients. Go-Me changed approximately 10 times more among the bilateral or unilateral condylar hyperplasia patients than class III only patients (Table 5).

The present sample was too small to evaluate gender differences and the main purpose of the study was to address group differences in both genders instead. However, the adjusted model showed that gender did not affected significantly the results of the overall sample (Table 4).

Class III treatment stability has been demonstrated to be less predictable than expected either after surgical[2, 4, 34] or non-surgical treatment[35-39].

Class III surgical treatment is affected by patient age and growth after operation, mainly if bimaxillary surgery is performed. It is well established that, when performed in growing children, bimaxillary surgery promotes significant subsequent mandibular and maxillary growth difference[34]. Sagital mandibular growth seems to be less affected than maxillary growth after surgery. Long term outcome in this situation will be sagital relapse of surgical achievements.

Professionals, patients and family postpone surgical treatment to avoid such instability related to craniofacial growth and this decision evolves a high psychosocial price that can be useless if condylar hyperplasia diagnosis is missed.

In the present sample condylar hyperplasia promoted higher post pubertal total mandibular growth rate (Ar-Gn = 2.61 mm) and higher corpus length (Go-

Me = 2.2 mm) than a group paired by age, gender and craniofacial pattern (Table 3).

Bidimensional evaluation based on superimposition or sagital and vertical measurements has brought a great deal of contribution to orthodontics and orthognathic surgery. Recent developments on tridimensional evaluations would bring better and more accurate information in the present study but the sample selected had no scans available. A prospective, controlled clinical trial should be conducted to address condylar hyperplasia influence evaluated with tridimensional surface volumes superimpositions.

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Tables

Table 1. Sample Demographic Data

PATIENTS PER GROUP		INITIAL AGE (years)		FOLLOW-UP (months)	
		GROUP 1	GROUP 2	GROUP 1	GROUP 2
		Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)
FEMALE	(13)	16.4 (2.9)	16.4 (2.6)	12.3 (0.5)	13.8 (2.5)
MALE	(7)	19.2 (1.6)	18.7 (1.6)	14.1 (3.2)	12.3 (0.6)
Total	(20)	17.4 (2.8)	17.2 (2.5)	12.9 (2.0)	13.3 (2.2)

Table 2. Descriptive measurements at the initial of the evaluation mean differences and Student t test for means equals.

Variable	Group 1		Group 2		difference		t test
	mean	sd	mean	sd	mean	sd	p-value
SNA [1]	81,51	2,17	82,16	5,57	-0,65	1,34	0,631
SNB	82.96	3.34	84.56	4.36	-1.60	1.23	0.201
ANB	-1.45	2.69	-2.39	2.88	0.95	0.88	0.290
SN.OP	15.13	6.32	13.83	6.79	1.30	2.07	0.535
SN.MP	35.07	6.78	34.93	7.08	0.14	2.19	0.949
SN.Pog	62.55	4.29	61.94	4.91	0.62	1.46	0.676
OJ	-0.98	2.84	-2.37	4.14	1.39	1.12	0.223
OB	0.99	2.57	1.26	3.31	-0.28	0.94	0.771
U1.SN	111.15	5.85	111.90	7.58	-0.75	2.14	0.728
L1.GoMe	87.51	8.19	85.09	7.89	2.42	2.54	0.347
L1.SN	57.43	9.96	59.98	10.93	-2.55	3.31	0.446
S-Go	81.17	7.66	79.29	7.53	1.87	2.40	0.440
N-Me	126.66	7.85	123.84	10.25	2.82	2.89	0.335
S-Ar	33.36	3.97	32.48	4.75	0.88	1.39	0.529
Ar-Go	51.92	5.43	51.67	5.75	0.25	1.77	0.888
Go-Me	78.10	4.36	77.22	5.90	0.88	1.64	0.595
Ar_Gn	121.35	7.13	121.22	8.55	0.13	2.49	0.959
S_Gn	145.34	8.18	143.53	10.33	1.81	2.95	0.544
ArGoMe	131.90	6.18	133.74	6.34	-1.84	1.98	0.357
SN.Ar	119.27	5.48	120.88	5.66	-1.61	1.76	0.367
ANS-Me	72.85	6.15	73.11	6.01	0.27	0.76	0.969

[1] heteroscedastic variable. Unequal variance t-test was used.
* Mean of group is statistically significantly different from zero

Table 3. Descriptive measures of the changes observed in both groups, differences in changes of two groups and t test for equality of means.

Variable	Group 1		Group 2		difference		t teste		
	mean	sd	mean	sd	mean	sd	t	df	p
SNA [1]	0,35 *	0,42	0,82 *	1,60	-0,47	0,37	-1,27	21,6	0,216
SNB	0,09	0,46	1,18 *	0,59	-1,10	0,17	-6,58	38	**<0,001**
ANB [1]	0,27 *	0,46	-0,37	1,34	0,64	0,32	2,01	23,4	0,056
SN.OP	0,52	1,65	-0,28	2,35	0,79	0,64	1,23	38	0,226
SN.MP [1]	0,01	0,52	-0,39	1,00	0,39	0,25	1,55	28,4	0,132
SN.Pog [1]	-0,03	0,24	-0,75 *	0,66	0,72	0,16	4,62	23,9	**<0,001**
OJ	-0,69	1,89	-0,53	2,09	-0,17	0,63	-0,26	38	0,795
OB	0,61 *	0,85	-0,13	1,23	0,73	0,34	2,18	38	**0,036**
U1.SN	0,32	3,51	1,35	4,45	-1,04	1,27	-0,82	38	0,419
L1.GoMe	2,41	4,65	0,86	2,49	1,55	1,18	1,31	38	0,197
L1.SN	-2,42 *	4,78	-0,47	2,76	-1,96	1,23	-1,58	38	0,121
S-Go [1]	0,01	0,46	0,21	1,25	-0,20	0,30	-0,67	24,1	0,509
N-Me [1]	0,12	0,53	0,78	2,17	-0,66	0,50	-1,31	21,2	0,204
S-Ar	0,02	0,47	0,12	0,43	-0,10	0,14	-0,67	38	0,509
Ar-Go	-0,01	0,74	0,33	1,25	-0,35	0,33	-1,08	38	0,289
Go-Me [1]	0,20	0,49	2,18 *	1,68	-1,99	0,39	-5,07	22,2	**<0,001**
Ar-Gn [1]	0,11	0,46	2,61 *	1,59	-2,50	0,37	-6,75	22,1	**<0,001**
S-Gn	0,08	1,08	1,98 *	1,70	-1,90	0,45	-4,23	38	**<0,001**
ArGoMe	-0,02	0,98	0,33	1,54	-0,35	0,41	-0,87	38	0,389
SN.Ar	0,05	0,78	0,20	0,85	-0,16	0,26	-0,62	38	0,540
ANS-Me[1]	0,27	0,76	0,66	1,77	-0,39	0,43	-0,89	25,9	0,380

[1] The variable is heteroscedastic. The Student t test for populations with unequal variances was used.

* Average of the changes statistically different from zero

Table 4a. Covariance model adjusted for hyperplasia, gender and age.

Variable	Effect attributable to						Model		power	R^2
	hyperplasia		gender		age					
	$F_{(2,35)}$	p-value	$F_{(1,35)}$	p-value	$F_{(1,35)}$	p-value	$F_{(4,35)}$	p-value		
SNA	1.97	0.169	3.02	0.091	3.331	0.076	2.01	0.129	0.474	0,205
SNB	48.25	**0.000**	1.45	0.236	5.01	**0.031**	17.28	**0.000**	1.000	**0,590**
ANB	3.83	0.058	1.86	0.181	0.90	0.349	2.01	0.131	0.472	0,202
SN.OP	1.38	0.248	0.13	0.721	0.25	0.620	0.57	0.637	0.156	0,111
SN.MP	2.50	0.123	0.95	0.336	0.88	0.354	1.20	0.324	0.294	0,102
SN.Pog	22.79	**0.000**	3.40	0.074	1.59	0.215	8.61	**0.000**	0.989	**0,423**
OJ	0.07	0.792	0.03	0.868	0.04	0.839	0.04	0.990	0.056	0,003
OB	4.39	0.043	0.08	0.781	0.82	0.372	1.81	0.162	0.432	0,151
U1.SN	0.60	0.445	0.02	0.900	0.21	0.651	0.28	0.836	0.099	0,028
L1.SN	2.34	0.135	0.05	0.816	0.14	0.711	0.92	0.441	0.231	0,071
S-Go	0.36	0.554	1.20	0.280	2.09	0.157	0.92	0.441	0.232	0,079
N-Me	1.59	0.216	3.54	0.068	5.73	0.022	2.79	0.054	0.623	0,190
S-Ar	0.84	0.366	0.00	0.972	9.97	**0.003**	4.43	**0.010**	0.837	**0,288**
Ar-Go	1.00	0.324	0.51	0.481	3.84	0.058	1.71	0.183	0.408	0,145
Go-Me	25.74	**0.000**	0.67	0.419	3.59	0.066	10.14	**0.000**	0.996	**0,480**
Ar-Gn	48.47	**0.000**	2.80	0.103	5.50	**0.025**	18.74	**0.000**	1.000	**0,611**
S-Gn	18.71	**0.000**	2.17	0.149	6.09	**0.018**	8.70	**0.000**	0.990	**0,420**
ArGoMe	0.89	0.351	0.25	0.619	2.03	0.162	0.94	0.429	0.237	0,130
SN.Ar	0.35	0.557	0.11	0.737	0.04	0.838	0.22	0.884	0.087	0,073
ANS-Me	0.68	0.415	2.13	0.153	2.57	0.117	1.34	0.276	0.327	0,101

Table 4b. Significant coefficients of the adjusted models.

Variable	Coefficients				
	μ	μ_{11}	μ_{12}	μ_2	β
SNB	-1,42	1,09***	1,13***	0,22	0,08*
SN.Pog	0,98	-0,79***	-0,67**	-0,36	-0,04
S-Ar	-1,42*	0,03	0,20	-0,03	0,08**
Go-Me	2,91	2,29***	1,61**	-0,23	-0,15
Ar-Gn	3,79*	2,38***	2,53***	-0,73	-0,18*
S-Gn	4,57*	1,88**	1,83**	-0,74	-0,23*

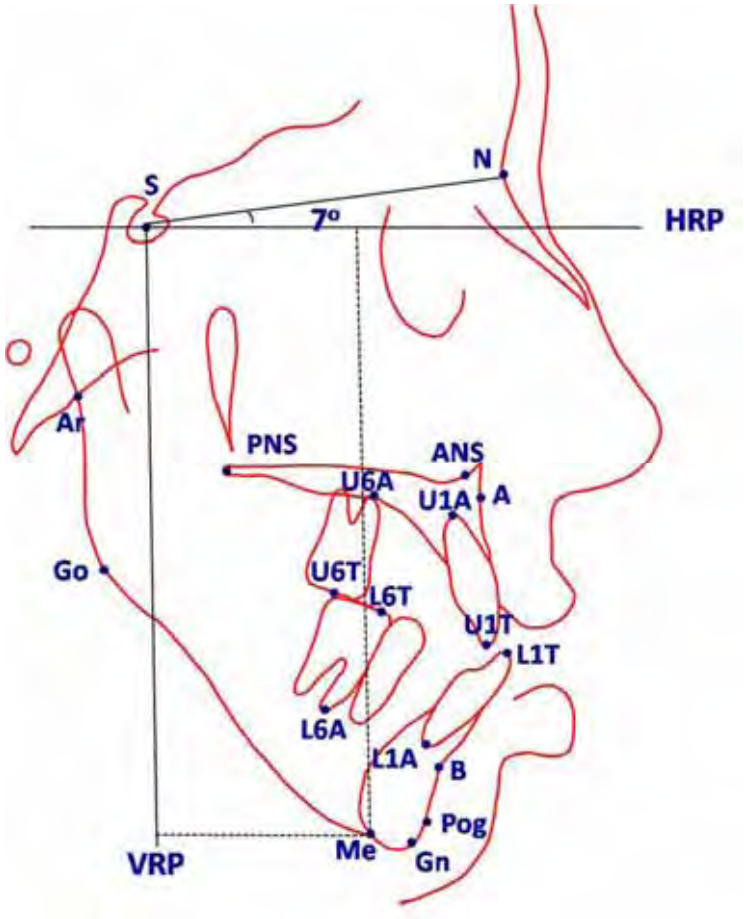
* p-value < 0,05; ** p-value<0,01, *** p-value < 0,001

Table 5. Descriptive measurements of skeletal changes of three groups and results of Bonferroni test for multiple comparison of means.

Variable	without (n=20)	bilateral (n=10)	unilateral (n=10)
	Mean (sd)	Mean (sd)	Mean (sd)
SNA	0.35 (0.42)	0.32 (1.44)	1.32 (1.66)
SNB	**0.09** ^{A} (0.46)	**1.13** ^{B} (0.56)	**1.23** ^{B} (0.64)
ANB	0.27 (0.46)	-0.78 (1.12)	0.05 (1.46)
SN.OP	0.52 (1.65)	-1.02 (2.77)	0.47 (1.65)
SN.MP	0.01 (0.52)	-0.46 (0.82)	-0.31 (1.20)
SN.Pog	**-0.03** ^{A} (0.24)	**-0.76** ^{B} (0.62)	**-0.74** ^{B} (0.72)
OJ	-0.69 (1.89)	-0.58 (1.50)	-0.47 (2.64)
OB	0.61 (0.85)	-0.35 (0.82)	0.10 (1.56)
U1.SN	0.32 (3.51)	1.74 (2.64)	0.96 (5.87)
L1.SN	-2.42 (4.78)	-0.55 (3.14)	-0.38 (2.49)
S-Go	0.01 (0.46)	0.15 (1.58)	0.26 (0.90)
N-Me	0.12 (0.53)	0.86 (1.87)	0.69 (2.54)
S-Ar	0.02 (0.47)	0.04 (0.35)	0.20 (0.50)
Ar-Go	-0.01 (0.74)	0.17 (1.41)	0.50 (1.12)
Go-Me	**0.20** ^{A} (0.49)	**2.55** ^{B} (1.09)	**1.81** ^{B} (2.11)
Ar-Gn	**0.11** ^{A} (0.46)	**2.64** ^{B} (1.62)	**2.58** ^{B} (1.65)
S-Gn	**0.08** ^{A} (1.08)	**2.11** ^{B} (1.64)	**1.85** ^{B} (1.83)
ArGoMe	-0.02 (0.98)	-0.12 (0.98)	0.78 (1.89)
SN.Ar	0.05 (0.78)	-0.07 (0.99)	0.48 (0.63)
ANS-Me	0.05 (0.78)	-0.07 (0.99)	0.48 (0.63)

Caption to illustration

Figure 1. Landmarks used for cephalometric assessment. The horizontal reference plane (HRP) was constructed at -7° to the SN plane. The vertical reference plane (VRP) was constructed perpendicular to the HRP, through sella (S). Dotted lines indicate the method of measuring Me relative to reference planes HRP and VRP. N (nasion); S (sella turcica); Ar (articulare); ANS (anterior nasal spine), a point posterior to the tip of the median, sharp bony process of the maxilla, on its superior surface, where the maxilla process first enlarges to a width of 5 mm); PNS (posterior nasal spine); U1T (upper incisor tip); U1A (upper incisor apex); U6T (upper molar mesial cusp tip); U6A (upper molar mesial apex); L1T (lower incisor tip); L1A (lower incisor apex); L6T (lower mesial cusp tip); L6A (lower molar mesial apex); A (A point); B (B point); Pog (pogonion); Me (menton); Go (gonion); Gn (gnathion)



ARTIGO 2

4- ARTIGO 2

High condylectomy allow predictable results for patients with unilateral or bilateral Condylar hyperplasia.*

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Purpose: To evaluate the stability of orthognathic surgery in class III patients with and without condylar hyperplasia and the affect of high condylectomy, articular disc repositioning and simultaneous orthognathic surgery in patients with active condylar hyperplasia.

Materials and Methods: Sixty-three patients (42 females and 21 males) were paired according to gender, age and craniofacial pattern in three groups. Group WCH (without condylar hyperplasia) was submitted to orthognathic surgery only, Group BCH (bilateral condylar hyperplasia) and Group UCH (unilateral condylar hyperplasia) were submitted to high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure. Lateral cephalometric radiographs were taken immediately before surgery (T1), immediate after surgery (T2) and longest follow-up (T3). 19 landmarks were identified and digitized using DFPlus software.

Results: Initial observation (T1) showed no significant differences between the three groups. Longest follow-up after surgery showed no differences among Group WCH (mean = 28.1; sd = 26.7 months); Group BCH (mean = 15.9; sd = 7.7 months); and Group UCH (mean = 29.7; sd = 31.8 months) even after covariance model adjustment for the presence of hyperplasia, gender, age and follow-up.

Conclusions: Treatment protocols used for all three groups were predictable and stable. Appropriate condilar hyperplasia diagnosis and customized approach are mandatory in order to obtain stable results for Class III surgical treatment.

Key words: diagnosis; mandibular condyle; growth & development; orthognathic surgery; malocclusion, Angle Class III

Introduction

Patients with skeletal Class III malocclusion are successfully treated with orthodontics and orthognathic surgery. Treatment stability, however, has been questioned[1-8].

Many variables have been identified as predictors of treatment instability, but few reports suggest condylar hyperplasia as a risk factor for class III surgical correction[9-13].

Condylar hyperplasia (CH), a pathological condition, first described in 1836 by Adams[14], is characterized by the development of the condyle above normal, thereby creating, significant functional and cosmetic deformities that result in forward projection of the mandibular body and ramus on the affected side, occlusal plane/mandibular plane cant and contralateral crossbite[11,15].

Abnormal mandibular condyle growth or pathological conditions can severely affect the occlusion and facial balance including congenital malformations (i.e. oculo-auriculo-vertebral spectrum, Treacher-Collins syndrome), primary growth disorders, acquired diseases or injuries that are associated with growth abnormalities[16] and condylar hyperplasia conditions.

Some patients affected by treatment instability may retain acceptable oclusal relation with post surgical orthodontic compensations but others will

develop significant adverse occlusal and skeletal positional changes. These patients may require further orthodontic and surgical intervention to correct the residual mal-occlusion and skeletal mal-alignment generating additional cost and increased morbidity.

Obwegeser and Makek[17], differentiate the term "condyle hyperplasia" into three different types: 1) Hemimandibular hyperplasia, 2) Hemimandibular elongation, and 3) Hybrid form. Hemimandibular hyperplasia is characterized by an increased three-dimensional side of the jaw, usually with the abnormal growth of the jaw ending at the same time as it completes the overall growth. Hemimandibular elongation was described as a horizontal displacement of the mandible to the contralateral side with little or no vertical effect.

Wolford[11] developed a classification for condylar hyperplasia according to its etiology. Condylar hyperplasia (CH) type 1A, is the most commonly occurring pathology, may occur bilaterally (CH type 1A) or unilaterally (CH type 1B). CH type 2 is caused by benign tumors, can develop at any age, and is unilateral condylar enlargements that cause unilateral vertical facial elongation deformities. The most common occurring mandibular condylar tumors are osteochondroma (CH type 2A) and osteoma (CH type 2B). CH type 3, are related to rare benign (CH type 3A) or malignant (CH type 3B) conditions that can cause condylar enlargement.

The etiology of condylar hyperplasia is controversial and not completely understood with possible causes including trauma[11, 18-22], heredity[11, 18-22], infection[11, 18, 20], neoplasia[19, 20], hormonal disorders[11, 18, 21, 22] and intrauterine

factors[11]. The onset of CH type 1 occurs during pubertal growth strongly indicating a hormonal induced etiology. Excessive condylar growth for a longer period of time than normal and/or increased growth rate are probably caused by persistent activity of pre-cartilaginous zone growth. Accelerated mandibular growth initiates in the late teens or later is most often related to CH type 2 (osteochondroma or osteoma)[11].

CH type 1 can occur bilaterally or unilaterally. Although bilateral cases occur more often, unilateral cases are more frequently described in the literature[18, 20, 21]. Female gender has been associated as slightly more prevalent[19, 23, 24].

Diagnosis of active CH can usually be determined through serial documentation, preferably at 12 months interval, consisting of: physical examination, study models obtained in centric relation and radiographic overlays. Superimposition of serial lateral cephalograms of the condyle and body of the mandible can help on diagnose of active CH if increased lengthening of the skeletal structures is confirmed.

It is important to identify if mandibular growth is active or inactive even in young adult patients and adopting a treatment protocol that is appropriate to the problem minimizing possible relapse.

Failure to recognize CH can result in unfavorable functional and esthetic treatment results following orthodontics and orthognathic surgery due to post-surgical instability[12].

Treatment protocol of patients with unilateral or bilateral condylar hyperplasia is not yet a consensus among clinicians or researcher[12, 25-28].

The aim of this study was to evaluate patients with skeletal Class III malocclusions treated with orthognathic surgery exclusively, showed the same stability regardless of the presence of condylar hyperplasia.

Material and methods

One hundred and fifty one class III patients records were consecutively selected from the patient population operated between 1996 and 2007 by the senior author (LMW).

From the initial sample, 104 patients had adequate records (lateral cephalometric radiographs). Among them, 56 patients were diagnosed as active condylar hyperplasia by scintigraphy[19], serial clinical, dental model and radiographic evaluations with cephalometric superimposition analysis[11, 12] and 48 were diagnosed as skeletal class III malocclusion with no condylar hyperplasia. Sixty-three patients (42 females and 21 males) met the inclusion criteria described bellow for the present retrospective study. This sample was paired according to gender, age and craniofacial pattern in three groups (Table 1).

Group WCH had no condylar hyperplasia and was submitted to orthognathic surgery only, Group BCH was diagnosed with bilateral condylar hyperplasia and had high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure and Group UCH was diagnosed with unilateral condylar hyperplasia and had high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure.

The following inclusion criteria were adopted: 1) patients with skeletal Class III malocclusion based on clinical observation and dental models (molar relationship); 2) at least 6 months follow-up after surgery; 3) females at least 14 years old and males at least 16 years old; 4) adequate records at the following periods: immediate before surgery (T1), immediate after surgery (T2) and longest follow-up (T3). Patients were rejected based on the following criteria: 1) absence of one or more inclusion criteria 2) previous surgical intervention in the craniofacial area; 3) presence of craniofacial syndrome; 4) inadequate or poor-quality records (radiographs).

Surgical Technique

High condylectomies were performed according to Wolford & LeBanc (1985)[11, 29] and consisted of removing 3-5 mm of the superior aspect of condylar head including the medial and lateral poles. The articular disc was then repositioned and stabilized to cover the articulating surface of the "new" condyle. A mini anchor (Mitek, Norwood, Mass) was used to stabilize the articular disc to the condyle[30-32]. Maxillo-mandibular osteotomies and TMJ surgeries were perfomed in one operation.

Horizontal measurments (N, Ar, ANS, PNS, A, B, U1T, U1A, U6T, U6A, L6T, L6A, L1T, L1A, Pog, Gn, Me, Go), vertical (N, Ar, ANS, PNS, A, B, U1T, U1A, U6T, U6A, L6T, L6A, L1T, L1A, Pog, Gn, Me, Go), length (S-Go, N-Me, S-Ar, Ar-Go, Go-Me, Ar-Gn, S-Gn and ANS-Me) and angles (SNA, SNB, ANB,

SN.PP, SN.OP, SN.MP, SN.Pog, U1.SN, L1.SN, ArGoMe and SN.Ar) were evaluated.

Cephalometric Measurements

Standardized lateral cephalometric radiographs (Quint sectograph; American Dental Co, Hawthorne, CA) were randomly traced and digitized twice by a single investigator (APP) approximately 1 week apart. The average values for the 2 replicates were used to decrease landmark technical errors. A total of 19 landmarks were identified (Fig 1) and digitized using DFPlus software (Dentofacial Software Inc, Toronto, Ontario, Canada).

This research has been approved by Research Ethics Committee of the Araraquara Dental School under protocol number 73/10.

Statistical Analysis

Dental and skeletal cephalometric differences and similarities among the groups were evaluated with student-t test and ANOVA. Levene test was used to check homoscedasticity (lack of significant difference variances between groups). Whenever variances differences were detected, ANOVA test were replaced by Brown-Forsythe test.

Considering that individuals in the sample had a large range of follow-up and age and some might be still growing, the observed measurement changes could be correlated with this variables and associated with gender, significant average changes in each group were evaluated by a covariance model:

$$y_{ijk} = \mu + \mu_{1i}X_{1i} + \mu_2 X_2 + \beta Z_{1ijk} + \gamma Z_{2ijk} + \varepsilon_{ijk}$$

y_{ijk} = y variable of the k individual difference from group i and gender j during T2 and T3.

μ = constant;

μ_{11} = effect attributed to bilateral hyperplasia and

$$X_{11} = \begin{cases} 1 & \text{to individual from bilateral hyperplasia} \\ 0 & \text{in another case} \end{cases}$$

μ_{12} = effect attributed to unilateral hyperplasia and

$$X_{12} = \begin{cases} 1 & \text{to individual from unilateral hyperplasia} \\ 0 & \text{in another case} \end{cases}$$

μ_2 = effect due to gender and

$$X_2 = \begin{cases} = 0 & \text{for males} \\ = 1 & \text{for females} \end{cases}$$

β = coefficient of the covariate age (Z),

γ = coefficient of covariate follow-up time (Z_2) and

ε_{ijk} = is a random error with normal distribution with mean zero.

Method error was evaluated in 20% of the sample randomly chosen to assess intraexaminer reliability using ICC (Intraclass Correlation Coeficient) analysis. All measurements were done twice one week apart.

Results

Method reproducibility was tested by Intraclass Correlation Coeficient (ICC). All variables showed high confiability (range 0.989 – 0.999).

All groups were successfully paired. Twenty-one males and forty-two females in each group had overall initial (T1) mean age of 18.3, 16.8 and 18.3 years old in Group WCH, BCH and UCH respectively. Among the females, mean age were 17.3, 15.9 and 17.6 years old and among males mean age were 20.4, 18.7 and 19.7 years old for Group WCH, BCH and UCH respectively. Average follow-up period were 28.1, 15.9 and 29.7 months for Group WCH, BCH and UCH respectively (Table 1).

The sample was appropriated paired according to skeletal and dental characteristics. Cephalometric comparison between Group WCH (class III only), Group BCH (class III with bilateral condylar hyperplasia) and Group UCH (class III with unilateral condylar hyperplasia) showed no significant differences among the variables mean values at the initial observation period, except for the variable OJ, where the group with BCH was found to be different from groups WCH and UCH (Table 2).

Net Skeletal Changes

Mean follow-up period was 28.1, 15.9 and 29.7 months for Groups WCH, BCH and UCH respectively (Table 1). During the observational period (T3-T2), Group WCH (no condylar hyperplasia) mean differences were bigger ($p<0.01$) than Group BCH (bilateral condylar hyperplasia) only for the variable OJ. ANB

mean difference was smaller ($p<0.024$) for Group WCH than Group BCH (Table 3).

Adjusted model for gender, age, condylar hyperplasia (unilateral, bilateral or absence) and follow-up

In order to evaluate possible influence of gender, age, group differences (presence or absence of condylar hyperplasia) and follow-up length differences, a analysis of covariance was performed (Table 4). The proposed model explained only the changes that occurred in OJ and Ar-Gn. It was observed that hyperplasia was a significant factor that influenced the changes occured in OJ while changes in Ar-Gn were influenced by gender and time of observation.

Discussion

This is the first study to investigate the stability of surgical treatment of Class III patients with and without condylar hyperplasia. Previous studies showed the relevance of condylar hyperplasia diagnosis and appropriate treatment to obtain stable result[9-13, 33] although the present study presented groups comparison paired by age, gender, presence or absence of condylar hyperplasia according to skeletal and dental characteristics (Table 2). The inclusion of a control group without CH showed that patients with CH and treated appropriately (with high condylectomies simultaneously to orthognathic surgery) can have similar predictable results as Class III patients treated with orthognathic surgery only.

Among 21 variables studied, only two variables (ANB and OJ) showed significant differences among the Groups studied. During the overall evaluation period ANB decreased more in Group WCH than Group BCH and OJ in Group BCH decreased more than the other two Groups (less than 1 mm.). In 2 previous studies[11, 12] that addressed the influence of CH that evaluated the treatment and stability of patients with condylar hyperplasia, it was also observed some post-surgical minor instability. The distance Co-Pog relapsed 0.6 to 0.8 mm. and Co-B relapsed 0.3 to 0.4 mm[11, 12].

Mentioned instability was significantly less than reported by studies that ignored the existence of condylar hyperplasia and probably included these patients as regular skeletal Class III in their sample[2, 3, 5-7, 34, 35].

As active mandibular growth lasts longer than the maxilla, age and gender at surgery can be confounders to observed stability and the influence of CH. Indeed, the covariance model in present sample showed that females and shorter follow-up tended to have less instability than males (factor gender $\mu_2 = -0.88$) and longer follow-up (factor follow-up $\gamma = -0.13$) at least for one variable (Ar-Gn). Similarly, the presence of unilateral CH influenced OJ negatively (factor $\mu_{12} = -0.89$), it is expected a reduction of about 0.9 mm (factor $\mu_{12} = -0.89$) in OJ of patients with unilateral CH.

A bigger sample, gender specific and narrower range of follow-up would clarify these findings better.

Main limitations of the present study were the lack of skeletal age evaluation, presence of three patients under 15 years old and bidimensional

analysis method. Indeed those parameters should have been addressed but it was not possible due to the retrospective character of the study.

Sagital evaluation through lateral cephalometric analysis misses the whole magnitude of unilateral abnormal growth.

In order to obtain stable results following surgical treatment of class III patients, it is of fundamental importance to identify the presence or absence of condylar hyperplasia and whether it is still active (Fig 2)

Prospective studies with longer follow-up, gender specific and the inclusion of a group diagnosed with CH but treated with orthognathic surgery only, would collaborate the present knowledge regarding the influence of CH on Class III surgical treatment stability.

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Tables

Table 1. Sample Demographic Data

PATIENTS (nº)	INITIAL AGE (years)			FOLLOW-UP (months)		
	Mean (sd)	min	max	Mean (sd)	min	max
Group WCH						
Female (14)	17,3 (2,6)	14,5	24,0	26,7 (25,0)	6,2	86,3
Male (7)	20,4 (1,7)	18,8	23,4	30,8 (31,8)	11,9	87,7
All (21)	18,3 (2,7)	14,5	24,0	28,1 (26,7)	6,2	87,7
Group BCH						
Female (14)	15,9 (1,6)	14,1	19,5	16,3 (8,0)	12,0	43,1
Male (7)	18,7 (2,2)	16,3	23,0	15,2 (7,5)	11,7	32,2
All (21)	16,8 (2,2)	14,1	23,0	15,9 (7,7)	11,7	43,1
Group UCH						
Female (14)	17,6 (2,9)	14,8	25,8	28,7 (33,3)	7,2	120,6
Male (7)	19,7 (3,4)	16,6	26,6	31,8 (31,1)	11,5	84,2
All (21)	18,3 (3,2)	14,8	26,6	29,7 (31,8)	7,2	120,6

WCH = Without Condylar Hyperplasia;
BCH = Bilateral Condylar Hyperplasia;
UCH = Unilateral Condylar Hyperplasia

Table 2. Descriptive measures of the three groups at the initial of the evaluation and results of analysis of variance to test the hypothesis of equality of means of three groups.

Variable	Group WCH (n=21)		Group BCH (n=21)		Group UCH (n=21)		ANOVA	
	Mean (sd)		Mean (sd)		Mean (sd)		F	p-value
SNA	80.25	3.40	81.50	3.80	82.48	4.36	1.75	0.183
SNB	81.87	4.04	83.93	3.31	83.20	4.76	1.39	0.258
ANB	-1.60	2.61	-2.42	3.85	-0.73	3.02	1.47	0.239
SN.PP	7.79	3.87	6.03	2.81	6.59	2.74	1.66	0.198
SN.OP	16.15	6.93	16.61	5.23	14.72	6.23	0.53	0.589
SN.MP	36.50	7.83	38.08	5.26	35.01	6.66	1.11	0.336
SN.Pog	63.85	5.01	63.24	3.63	62.82	4.74	0.28	0.760
OJ	-1.85[a]	2.23	-3.63[b]	3.23	-0.33[a]	2.69	7.61	**0.001**
OB [1]	1.67	2.57	1.95	2.91	0.32	1.28	2.84	0.069
U1.SN	110.06	6.59	110.93	5.35	112.96	6.88	1.17	0.318
L1.SN	54.86	8.27	54.96	10.69	56.32	9.57	0.15	0.858
S-Go	79.92	7.22	78.12	7.02	78.62	7.00	0.36	0.700
N-Me	127.19	9.32	127.86	9.33	124.26	9.54	0.87	0.424
S-Ar	33.10	3.82	32.21	3.30	33.02	4.18	0.35	0.704
Ar-Go	51.20	5.08	50.11	5.11	50.04	4.63	0.37	0.695
Go-Me	78.47	4.16	79.70	5.98	78.75	6.07	0.29	0.748
Ar-Gn	121.02	7.48	122.76	8.01	119.93	7.36	0.73	0.485
S-Gn	143.90	8.71	145.23	9.69	143.15	8.27	0.29	0.746
ArGoMe	132.05	6.39	135.18	5.62	131.26	6.01	2.49	0.092
SN.Ar	122.01	6.23	119.61	7.41	121.28	5.07	0.80	0.454
ANS-Me	72.04	7.09	74.96	6.90	70.21	6.05	2.68	0.076

Note: Differents letters reply satatistically differents means (N-K-S test ou Tamhane)

[1] Heteroscedastic variables. The Brown-Forsythe test was used.

WCH = Without Condylar Hyperplasia; BCH = Bilateral Condylar Hyperplasia; UCH = Unilateral Condylar Hyperplasia

Table 3. Descriptive measurements and ANOVA for test of equality of means in the three groups during the overall evaluation period (T3-T2).

Variable	Group WCH (n=21)		Group BCH (n=21)		Group UCH (n=21)		ANOVA		power	R^2
	Mean (sd)		Mean (sd)		Mean (sd)		F	p-value		
Follow-up (months)	28.07	26.70	15.93	7.68	29.70	31.82				
SNA	-0.10	0.75	0.61	1.29	0.22	0.88	2.60	0.082	0.500	0.08
SNB	0.07	0.51	0.01	0.73	-0.03	0.67	0.14	0.871	0.070	0.01
ANB	-0.17^{A}	0.62	0.59^{B}	0.94	0.28^{AB}	1.00	3.99	**0.024**	0.693	0.12
SN.PP	0.14	2.19	1.09	1.53	0.43	1.74	1.44	0.244	0.297	0.05
SN.OP	-0.50	1.75	-0.26	2.98	-0.16	1.91	0.13	0.882	0.068	0.01
SN.MP	0.29	1.25	0.53	1.16	0.20	1.28	0.40	0.670	0.112	0.01
SN.Pog	-0.09	0.46	-0.01	0.50	-0.08	0.63	0.12	0.890	0.067	0.01
OJ	-0.23^{A}	0.83	-0.96^{B}	0.98	-0.10^{A}	1.04	4.94	**0.010**	0.789	0.14
OB [1]	-0.65	0.57	-0.39	1.20	-0.74	1.04	0.76	0.474	0.173	0.03
U1.SN	-1.41	3.71	-2.23	2.75	-0.79	1.91	1.33	0.272	0.277	0.04
L1.SN	1.57	3.63	-0.75	3.47	0.29	2.61	2.65	0.079	0.508	0.08
S-Go	-0.66	1.50	-0.70	1.87	-0.44	1.20	0.17	0.841	0.076	0.01
N-Me	-0.18	0.99	-0.01	1.50	0.01	1.58	0.12	0.890	0.067	0.01
S-Ar	-0.08	0.57	-0.09	0.61	0.20	0.78	1.32	0.276	0.274	0.04
Ar-Go	-0.60	1.39	-0.68	1.73	-0.59	1.38	0.03	0.975	0.054	0.01
Go-Me	0.28	1.26	0.24	1.59	0.48	1.22	0.18	0.835	0.077	0.01
Ar-Gn	-0.06	0.75	-0.31	1.13	-0.20	1.03	0.35	0.709	0.103	0.01
S-Gn	-0.15	1.73	-0.30	1.75	-0.90	1.25	1.30	0.281	0.270	0.04
ArGoMe	0.51	1.76	0.76	1.95	0.47	1.57	0.17	0.848	0.074	0.01
SN.Ar	-0.05	0.51	-0.48	1.07	0.10	1.33	1.75	0.182	0.354	0.01
ANS-Me	0.49	1.64	-0.16	1.06	-0.03	1.38	1.28	0.284	0.268	0.04

Table 4. Covariance model adjusted for hyperplasia, gender, age and follow-up.

Variable	Effect attributable to								Model		power	R^2
	hyperplasia		gender		age		follow up					
	$F_{(2,35)}$	p-value	$F_{(1,35)}$	p-value	$F_{(1,35)}$	p-value	$F_{(1,35)}$	p-value	$F_{(4,35)}$	p-value		
SNA	1.54	0.224	0.26	0.614	1.53	0.221	0.25	0.619	1.35	0.258	0.441	0.11
SNB	0.20	0.818	0.83	0.366	0.21	0.650	0.77	0.385	0.28	0.923	0.113	0.02
ANB	3.07	0.054	0.02	0.881	0.99	0.325	0.00	0.965	1.77	0.134	0.567	0.13
SN.PP	1.81	0.173	1.90	0.174	0.33	0.570	2.80	0.100	1.44	0.224	0.471	0.11
SN.OP	0.14	0.867	2.36	0.130	0.24	0.629	0.50	0.483	0.54	0.748	0.185	0.05
SN.MP	0.64	0.531	1.03	0.314	0.01	0.930	1.58	0.215	0.55	0.737	0.189	0.05
SN.Pog	0.09	0.914	0.05	0.819	0.27	0.607	0.23	0.635	0.23	0.949	0.1	0.02
OJ	4.41	**0.017**	0.82	0.368	1.18	0.282	0.31	0.583	2.46	**0.044**	0.733	0.18
OB	0.30	0.746	1.10	0.299	1.71	0.196	0.03	0.859	0.87	0.508	0.288	0.07
U1.SN	0.64	0.529	3.38	0.071	0.00	0.985	3.07	0.085	1.46	0.216	0.477	0.11
L1.SN	3.55	0.035	0.24	0.623	0.89	0.351	1.85	0.179	1.51	0.201	0.491	0.12
S-Go	0.54	0.587	2.78	0.101	0.01	0.906	4.98	0.030	1.25	0.297	0.412	0.10
N-Me	0.10	0.902	0.77	0.385	0.03	0.861	0.68	0.412	0.24	0.943	0.104	0.02
S-Ar	1.18	0.315	0.04	0.835	0.01	0.914	0.02	0.876	0.52	0.764	0.179	0.04
Ar-Go	0.43	0.651	6.00	0.017	0.08	0.784	7.91	0.007	2.10	0.078	0.654	0.16
Go-Me	0.16	0.851	0.01	0.930	0.37	0.545	0.41	0.524	0.31	0.903	0.122	0.03
Ar-Gn	1.37	0.263	9.66	**0.003**	0.55	0.463	6.80	**0.012**	2.40	**0.048**	0.721	0.17
S-Gn	1.66	0.199	0.15	0.700	2.92	0.093	0.01	0.905	1.19	0.325	0.392	0.10
ArGoMe	0.21	0.815	0.44	0.511	0.07	0.797	0.47	0.496	0.23	0.950	0.1	0.02
SN.Ar	1.70	0.191	4.17	0.046	0.40	0.528	0.89	0.349	1.75	0.139	0.561	0.13
ANS-Me	2.32	0.108	1.587	0.213	0.95	0.334	4.27	0.043	1.44	0.223	0.471	0.11

Table 5. Significant coefficients of the adjusted models.

Variable	Coefficients					
	μ	μ_{11}	μ_{12}	μ_2	β	γ
OJ	-0,65	-0,14	-0,89**	0,27	-0,01	0,03
Ar-Gn	2,94	0,14	-0,36	-0,88**	-0,01	-0,13**

* p-value < 0,05; ** p-value<0,01, *** p-value < 0,001

Captions to illustrations

Figure 1. Landmarks used for cephalometric assessment. The horizontal reference plane (HRP) was constructed at -7° to the SN plane. The vertical reference plane (VRP) was constructed perpendicular to the HRP, through sella (S). Dotted lines indicate the method of measuring Me relative to reference planes HRP and VRP. N (nasion); S (sella turcica); Ar (articulare); ANS (anterior nasal spine), a point posterior to the tip of the median, sharp bony process of the maxilla, on its superior surface, where the maxilla process first enlarges to a width of 5 mm); PNS (posterior nasal spine); U1T (upper incisor tip); U1A (upper incisor apex); U6T (upper molar mesial cusp tip); U6A (upper molar mesial apex); L1T (lower incisor tip); L1A (lower incisor apex); L6T (lower mesial cusp tip); L6A (lower molar mesial apex); A (A point); B (B point); Pog (pogonion); Me (menton); Go (gonion); Gn (gnathion).

Figure 2. Conceptual scheme to be evaluated in a patient with progressive facial asymmetry and indication of surgical orthodontic treatment.

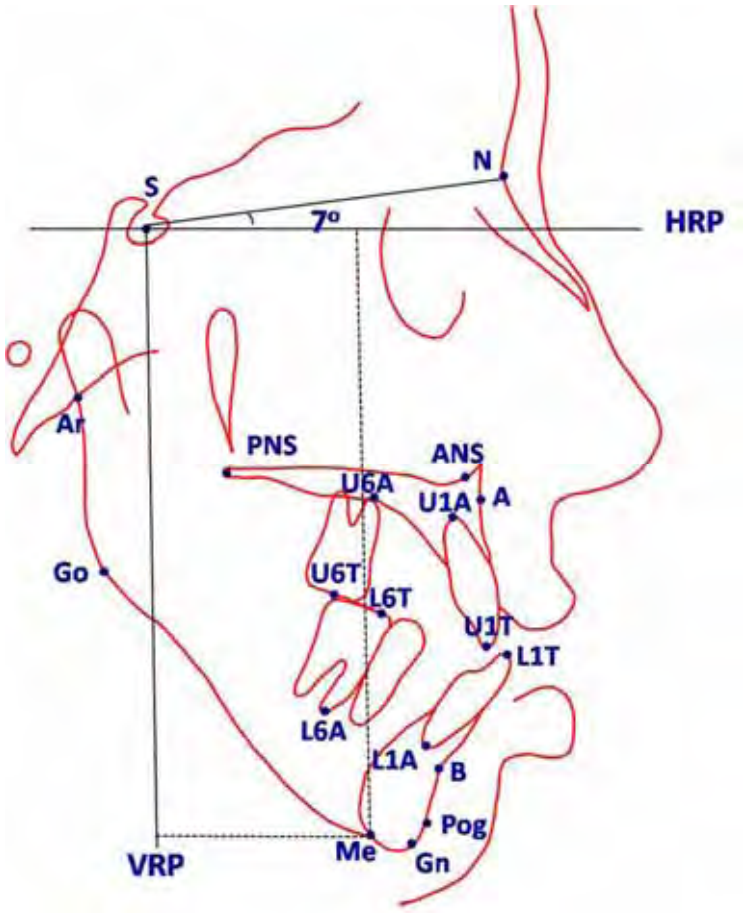
S
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Figure 1

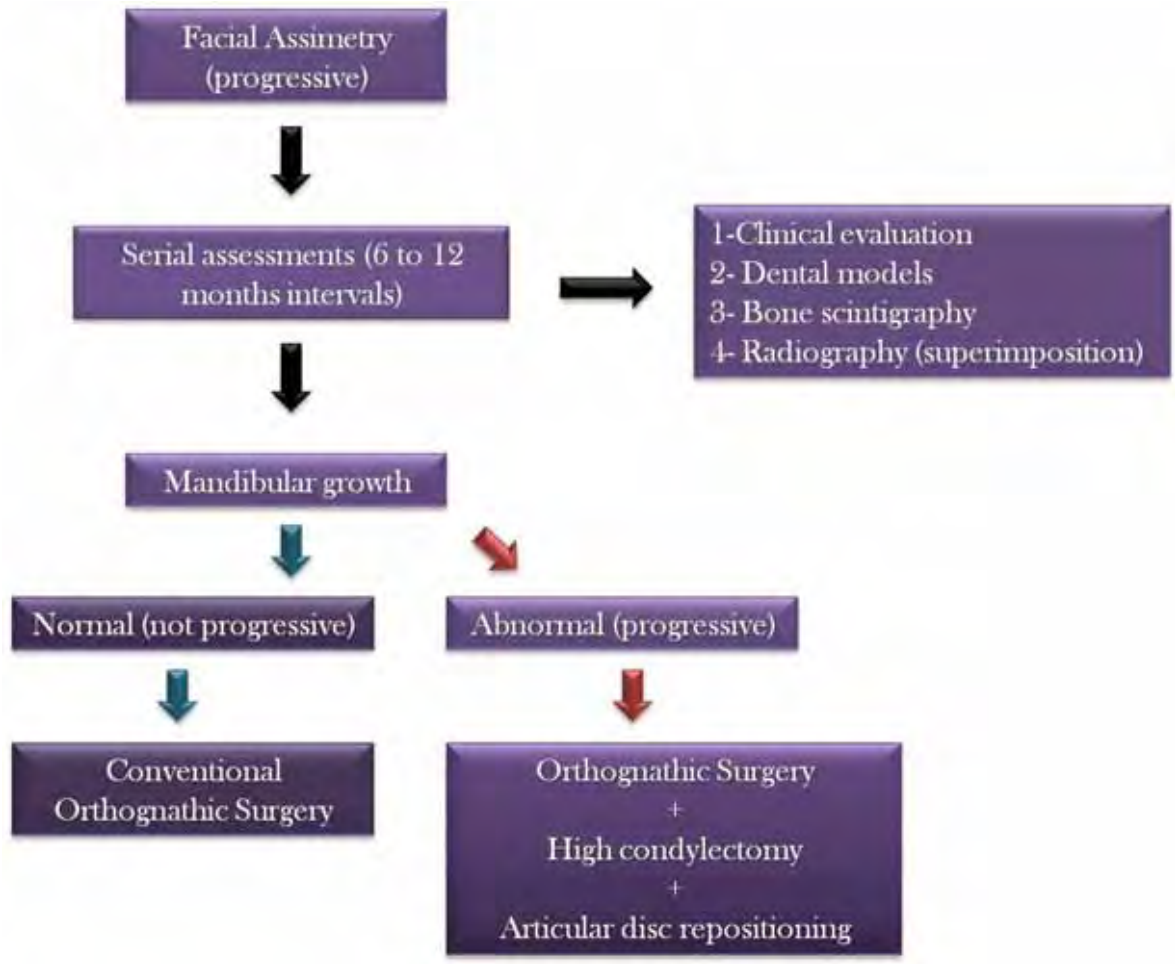
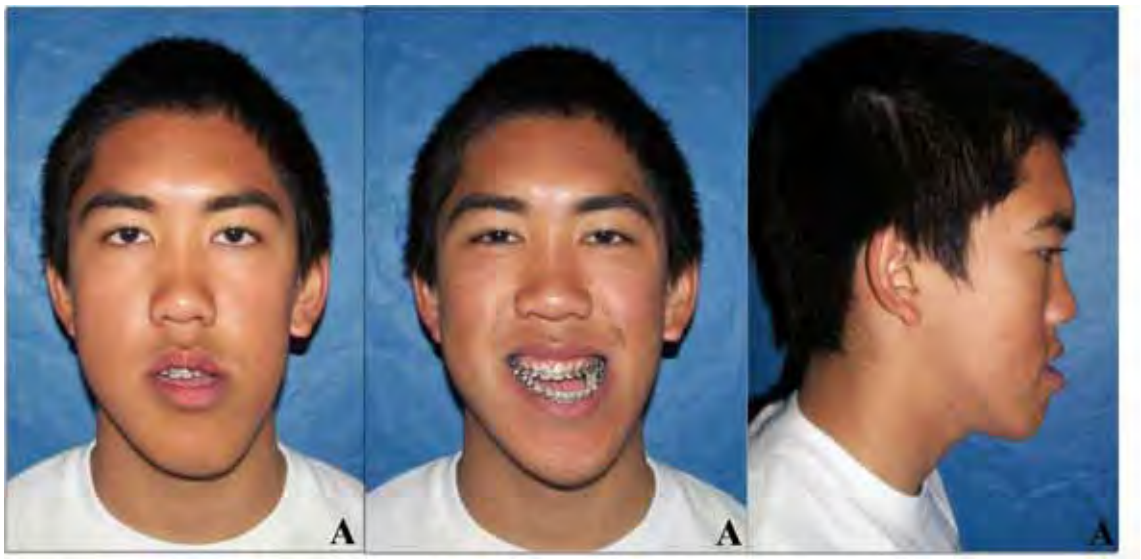

Facial Assimetry (progressive)
Serial assessments (6 to 12 months intervals)
1-Clinical evaluation
2- Dental models
3- Bone scintigraphy
4- Radiography (superimposition)
Mandibular growth
Normal (not progressive)
Abnormal (progressive)
Conventional Orthognathic Surgery
Orthognathic Surgery
+
High condylectomy
+
Articular disc repositioning

Figure 2

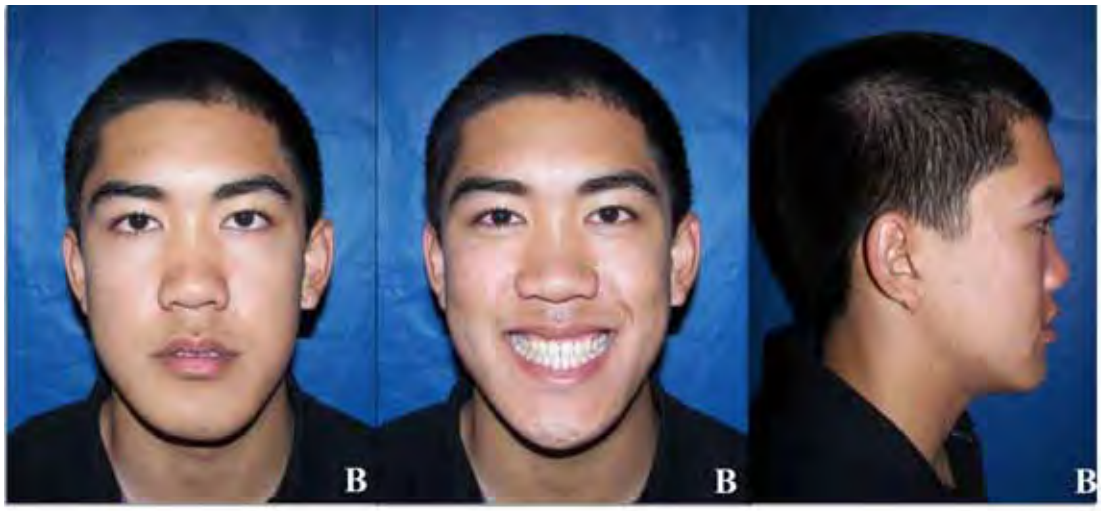
Clinical Cases

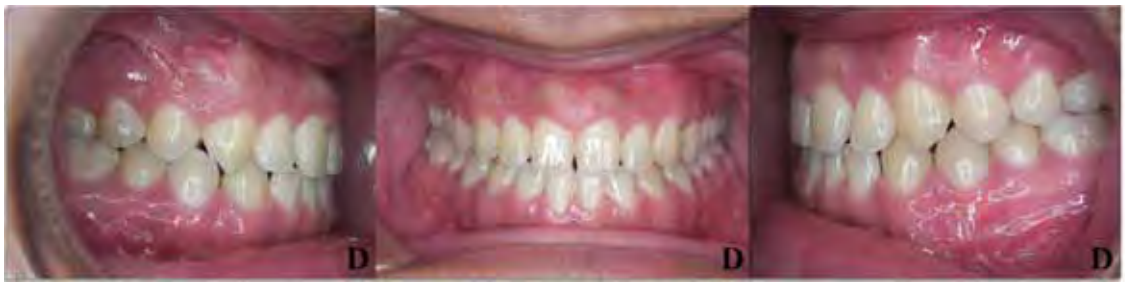
Clinical Case 1

Skeletal Class III malocclusion associated with assimetric mandibular prognathism due to unilateral Condylar Hyperplasia. Deviation of mandibular dental midline to left with severe anterior crossbite. Class III occlusion was greatest on the right side. *Facial photographs*: A, B (pre and post treatment: frontal view, frontal smile and right profile) and *Intrabucal photographs*: C, D (pre and post treatment: right side, frontal and left side).



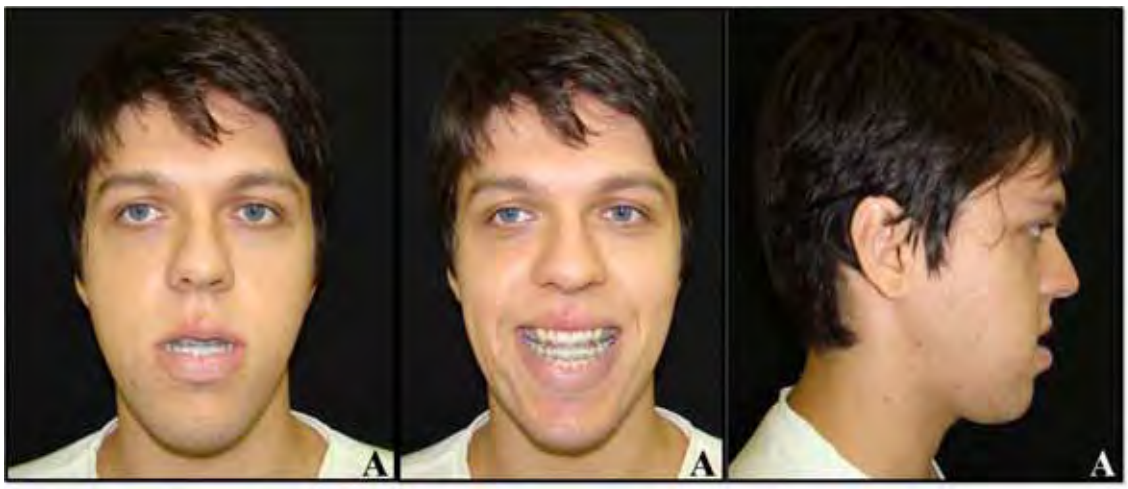
Clinical Case 1

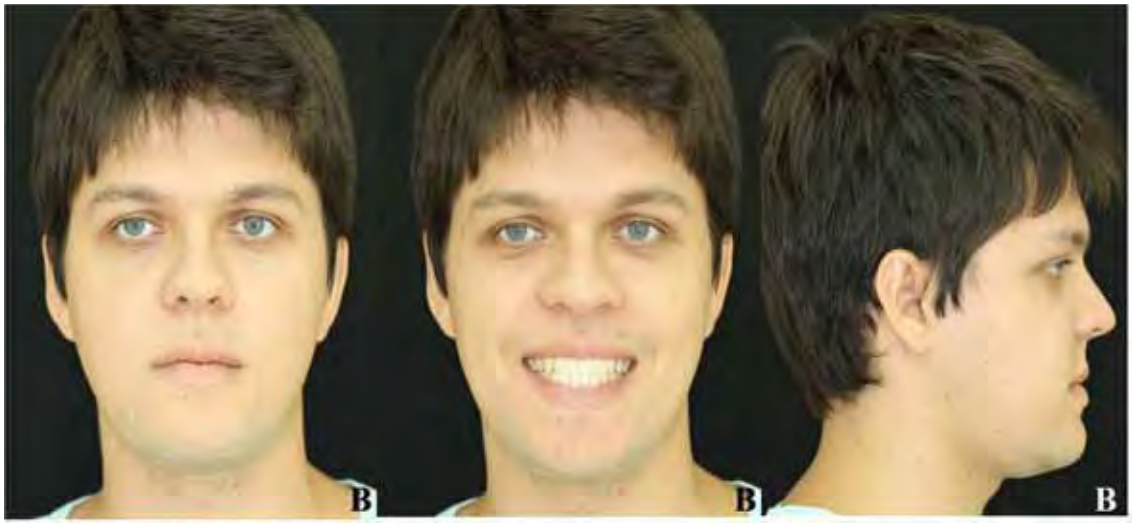




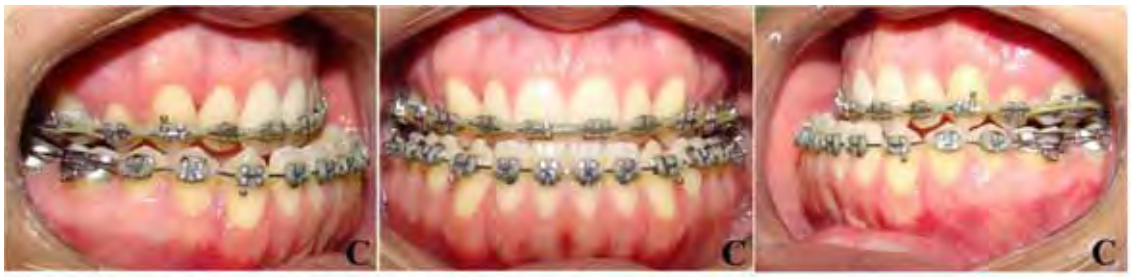
Clinical Case 2

Skeletal Class III malocclusion associated with simetric mandibular prognathism due to bilateral Condylar Hyperplasia. Anterior and posterior crossbite. *Facial photographs*: A, B (pre and post treatment: frontal view, frontal smile and right profile) and *Intrabucal photographs*: C, D (pre and post treatment: right side, frontal and left side).





Clinical Case 2



ARTIGO 3

5- ARTIGO 3

Stability of skeletal class III surgical treatment. Affect of high condylectomy.*

*Artigo a ser enviado para publicação no periódico:

Journal of Oral and Maxillofacial Surgery

Purpose: To evaluate the role of high condylectomy on surgical treatment stability of Class III patients with condylar hyperplasia.

Materials and Methods: Twenty-four patients (9 females and 15 males) were paired according to gender, age, skeletal and dental characteristics in three groups. Group 1 (no condylar hyperplasia) underwent orthognathic surgery only, Group 2 (with unilateral or bilateral condylar hyperplasia) underwent high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure and Group 3 (with unilateral or bilateral condylar hyperplasia) underwent orthognathic surgery only. Lateral cephalometric radiographs were selected at the immediately before surgery (T1), immediate after surgery (T2) and longest follow-up (T3).

Results: Cephalometric comparison between the 3 groups showed no significant differences among the variables at the initial observation period (T1). During the observational period (T3-T2), patients in Group 3 showed significant relapse at SNB (mean = 2.18, sd = 1.39 degrees), ANB (mean = -2.68, sd = 2.24 degrees), SN.Pog (mean = -1.48, sd = 1.66 degrees), OJ (mean = -2.99, sd = 1.64 mm), OB (mean = 1.45, sd = 1.16 mm), Ar-Go (mean = 2.23, sd = 2.66 mm), Ar-Gn (mean = 3.76, sd = 1.48 mm), S-Gn (mean = 2.3, sd = 2.34 mm) and ANS-Me (mean = 2.06, sd = 2.2 mm) demonstrating that treatment adopted was insufficient for stable results within this Group. Groups 1 and 2 remained stable one year after surgery.

Conclusions: Orthognathic surgery for correction of skeletal class III malocclusion is a stable procedure for patients without condylar growth abnormalities and for patients undergoing simultaneous high condylectomies and

articular disc repositioning. Those patients with preoperative condylar hyperplasia who underwent double-jaw surgery and no TMJ intervention experienced significant relapse.

Key words: diagnosis; mandibular condyle; growth & development; orthognathic surgery

Introduction

Many factors have been identified as predictors for treatment instability of class III surgical correction: surgical technique single jaw versus double-jaw procedures[1-5], bi-cortical osteosynthesis or mono-cortical osteosynthesis[6, 7], presence or absence of orthodontic treatment prior to surgery[8], age at surgery[9, 10], amount of surgical alteration of the mandibular position[11], positioning of the proximal segment[12], condylar positioning in the glenoid cavity[13,14], neuromuscular adaptation[14, 15], magnitude of surgical change[16, 17], method of maxillomandibular fixation[18, 19], post-surgical growth[20], surgeon experience[19], condylar positioning and proximal segment rotation with clockwise rotation of the distal segment[21]. Few reports have suggested Condylar Hyperplasia (CH) as a risk factor for class III surgical correction[22-26].

CH is an abnormal growth condition affecting the mandibular condyles, unilateral or bilateral, creating accelerated and excessive overgrowth of the mandible, often continuing into the patient's mid 20's[24].

Wolford[24, 25] have demonstrated that orthognathic surgery alone for patients with skeletal Class III malocclusions where CH is associated, show poor results, as the presence of relapses requiring a new surgical intervention associated to high condylectomy in order to obtain long-term predictable results.

Healthy TMJs are essential to obtain long-term stable results in orthodontics and orthognathic surgery. If TMJs are not stable and healthy, treatment outcomes may be unsatisfactory relative to function, esthetics, stability and pain[22].

The lack of appropriate diagnostic steps, treatment planning, selection of the surgical procedures and adequate postoperative control will usually provide unpredictable outcomes.

This study sought to evaluate wheter patients with skeletal class III malocclusions treated with orthognathic surgery exclusively, showed the same stability regardless of the presence of condylar hyperplasia.

Material and methods

One hundred and fifty one class III patients records were consecutively selected from the patient population operated between 1996 and 2007 by the senior author (LMW). From the initial sample 104 patients had adequate records (lateral cephalometric radiographs). Among them, 56 patients were diagnosed as active condylar hyperplasia by scintigraphy[27], serial clinical, dental model and radiographic evaluations with cephalometric superimposition analysis[24, 25]. Forty-eight were diagnosed as skeletal class III malocclusion with no condylar

hyperplasia. Twenty four patients (9 females and 15 males) met the inclusion criteria described bellow for the present retrospective study and were paired according to gender, age and craniofacial pattern in three groups (Table 1).

Group 1 had no condylar hyperplasia and underwnt to orthognathic surgery only, Group 2 was diagnosed with active bilateral (6 cases) and unilateral (2 cases) condylar hyperplasia and underwent high condylectomy, articular disc repositioning and orthognathic surgery in the same procedure and Group 3 was diagnosed with active bilateral (7 cases) and unilateral (1 case) condylar hyperplasia underwent to orthognathic surgery only because refused to have high condylectomies.

The following inclusion criteria were adopted: 1) patients with skeletal Class III malocclusion based on clinical observation and dental models (molar relationship); 2) at least one year follow-up after surgery; 3) females at least 14.0 years old and males at least 14.9 years old; 4) adequate records at the following periods: immediate before surgery (T1), immediate after surgery (T2) and longest follow-up (T3). Patients were rejected based on the following criteria: 1) absence of one or more inclusion criteria 2) previous surgical intervention in the craniofacial area; 3) presence of craniofacial syndrome; 4) inadequate or poor-quality records (radiographs).

High condylectomies were performed according to Wolford & LeBanc (1985)[24, 28] and consisted of removing 3-5 mm of the superior aspect of the condylar head including the medial and lateral poles. The articular disc was then repositioned and stabilized to cover the articulating surface of the “new” condyle.

A mini anchor (Mitek, Norwood, Mass) was used to stabilize the articular disc to the condyle[29-31]. Maxillo-mandibular osteotomies and TMJ surgeries were perfomed in one operation.

This research has been approved by Research Ethics Committee of the Araraquara Dental School under protocol number 73/10.

Cephalometric Measurements

The following horizontal measurments (N, Ar, ANS, PNS, A, B, U1T, U1A, U6T, U6A, L6T, L6A, L1T, L1A, Pog, Gn, Me, Go), vertical measurments (N, Ar, ANS, PNS, A, B, U1T, U1A, U6T, U6A, L6T, L6A, L1T, L1A, Pog, Gn, Me, Go), length (S-Go, N-Me, S-Ar, Ar-Go, Go-Me, Ar-Gn, S-Gn and ANS-Me) and angles (SNA, SNB, ANB, SN.PP, SN.OP, SN.MP, SN.Pog, U1.SN, L1.SN, ArGoMe and SN.Ar) were evaluated.

Standardized lateral cephalometric radiographs (Quint sectograph; American Dental Co, Hawthorne, CA) were randomly traced and digitized twice by a single investigator (APP) approximately 1 week apart. The average values for the 2 replicates were used to decrease landmark technical errors. A total of 19 landmarks were identified and digitized using DFPlus software (Dentofacial Software Inc, Toronto, Ontario, Canada).

Statistical Analysis

Dental and skeletal cephalometric differences and similarities among the groups were evaluated with student-t test and ANOVA. Levene test was used to

check heteroscedasticity (variance of a variable is not constant). Whenever variances differences were detected, ANOVA test were replaced by Brown-Forsythe test.

The samples were subjected to the Kolmogorov-Smirnov test to examine the hypothesis that the populations had normal distribution. All measurements, within the three timepoints, showed no statistical evidence to reject the hypotheses that they were normally distributed. This allowed, even with small samples, parametric statistical methods were employed.

Method error was evaluated in 20% of the sample randomly chosen to assess intraexaminer reliability using ICC (Intraclass Correlation Coeficient) annalysis. All measurements were done twice one week apart.

Results

Method reproducibility tested by Intraclass Correlation Coeficient (ICC) showed high confiability (range 0.989 – 0.999) for all variables.

Group Similarities and Differences

All groups were successfully paired. Among the females, mean age were 14.6, 14.9 and 14.4 years old and among the males mean age were 19.5, 17.2 and 17.0 years old for group 1, 2 and 3 respectively. Average follow-up period were 29.9, 36.8 and 34.2 months for group 1, 2 and 3 respectively (Table 1).

The sample was appropriated paired according to gender, skeletal and dental characteristics. Cephalometric comparison between Group 1 (class III

only), Group 2 and 3 (class III with unilateral and bilateral condylar hyperplasia) showed no significant differences among the variables at the initial observation period, except for S-Ar that was larger in Group 1 than Group 2 (Table 2).

Net Skeletal Changes

Significant changes were observed in Group 1 for the variables Go-Me (mean = 0.64, sd = 0.75 mm), Ar-Gn (mean = 0.43, sd = 0.31 mm) and ANS-Me (mean = 1.60, sd = 1.56 mm) (Table 3). Group 2, significant changes were observed only at OJ (mean = -0.95, sd = 1.09 mm) (Table 4) while in Group 3, the variables SNB (mean = 2.18, sd = 1.39 degrees), ANB (mean = -2.68, sd = 2.24 degrees), SN.Pog (mean = -1.48, sd = 1.66 degrees), OJ (mean = -2.99, sd = 1.64 mm), OB (mean = 1.45, sd = 1.16 mm), Ar-Go (mean = 2.23, sd = 2.66 mm), Ar-Gn (mean = 3.76, sd = 1.48 mm), S-Gn (mean = 2.3, sd = 2.34 mm) and ANS-Me (mean = 2.06, sd = 2.2 mm) showed significant changes during the postoperative follow-up (Table 5).

Follow-up Group Differences (T3-T2)

Mean follow-up period was 29.9, 36.8 and 34.2 months for Groups 1, 2 and 3 respectively (Table 1). During the observational period (T3-T2), Group 3 (bilateral and unilateral condylar hyperplasia with no TMJ intervention) mean differences were larger than Group 1 (no condylar hyperplasia) and Group 2 (bilateral and unilateral condylar hyperplasia treated with simultaneous high condylectomy) for the variable SNB, ANB, OJ, OB and Ar-Gn (Table 6).

Group 3 also showed larger significant post-surgical changes (T3-T2) for the variables SN.Pog, Ar-Go and S-Gn than observed in Groups 1 and 2, although the multiple-choice tests (Brown-Forsyte) were unable to identify them (Table 6).

Discussion

This is the first study that includes a group without Condylar Hyperplasia (control) to compare the effectiveness of high condylectomy in the stability of surgical correction of skeletal Class III patients. It is also the first study that assessed Class III surgical treatment stability and the influence of condylar hyperplasia with paired samples controlled by age, gender, follow-up length, skeletal and dental cephalometric similarities.

Adittionally, Groups 2 and 3 comprised similar frequency of unilateral and bilateral CH. Certainly, differences regarding the age at surgery, gender and follow-up length between Groups could had influenced previous studies as multiple confounders.

Our results showed that appropriate diagnosis of Class III patients without condylar hyperplasia lead to improved surgical treatment stability than previously showed[1, 3-5, 8, 9, 32] and confirm previuous findings[22-25, 33] regarding the relevance of simultaneous high condylectomy for the surgical treatment stability of Class III patients with active condylar hyperplasia.

Bilateral condylar hyperplasia patients treated with bilateral high condylectomy have been presented as highly predictable even in young patients[24, 25].

Although condylar growth does not drive mandibular growth in normal conditions, high condylectomies seems to disturbe enough remanescent growth to avoid surgical relapse in patients with condylar hyperplasia[24, 25]. Young patients with unilateral condylar hyperplasia will not have the same predictable outcome due to contralateral side normal growth after surgery. In our sample, two young females (14.1 and 14.2 years old) and one male (14.9 years old) with bilateral condylar hyperplasia underwent orthognathic surgery with high condylectomies (Group 2) and end up with stable results at least one year following surgery. Unilateral condylar hyperplasia patients have their surgeries postponed in order to avoid contralateral side relapse (one female patient with 16.4 years old and 1 male patient with 14.9 years old).

It's not rare the imperative need of early operations in order to avoid psicological implications for severe Class III patients. In our sample all the three Groups have young patients probably under post-pubertal active growth.

During the observational period (T3-T2), patients in Group 3 showed significant relapse for variables SNB (mean = 2.18, sd = 1.39 degrees), ANB (mean = -2.68, sd = 2.24 degrees), SN.Pog (mean = -1.48, sd = 1.66 degrees), OJ (mean = -2.99, sd = 1.64 mm), OB (mean = 1.45, sd = 1.16 mm), Ar-Go (mean = 2.23, sd = 2.66 mm), Ar-Gn (mean = 3.76, sd = 1.48 mm), S-Gn (mean = 2.3, sd = 2.34 mm) and ANS-Me (mean = 2.06, sd = 2.2 mm) (Table 5), demonstrating failure on treatment emploied with the recapture of mandibular growth and surgical relapse. This patients probably had to undergo a new surgical procedure associated with high condylectomy associated to new orthognathic surgery or had

to waite until natural condylar growth arrest to have orthognathic surgery only with predictable results as we observed in Groups 1 and 2.

Our results are consistent with previous studies where significant relapses were observed in some representative measures of mandibular length as the Co-Pg (mean 3.5 mm) and Co-B (mean 3.6 mm) with the average postsurgical follow-up of 5.6 years (ranging from 2 to 11.2 years) [24, 25].

The sample for this study was limited to 8 patients per group due to the fact that only 8 patients with active condylar hyperplasia refused to undergo the proposed treatment (high condylectomy associated to simultaneous orthognathic surgery). Although with small sample size the consistency of observed Group behaviour did not let doubts regarding the greater instability that occurred with patients from Group 3.

Prospective studies with longer follow-up, skeletal age evaluation, and tridimensional analysis methods are necessary to corroborate the protocols used today in relation to the diagnosis, treatment and stability of Class III skeletal malocclusions associated with Condylar Hyperplasia.

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Tables

Table 1. Sample Demographic Data

PATIENTS (nº)	AGE AT SURGERY (years)			FOLLOW UP (months)		
	Mean (sd)	min	max	Mean (sd)	min	max
Group 1						
Female (3)	14.6 (0.2)	14.5	14.9	41.2 (39.4)	13.0	86.3
Male (5)	19.5 (0.5)	18.8	19.9	23.1 (23.6)	11.9	65.3
All (8)	17.7 (2.6)	14.5	19.9	29.9 (29.2)	11.9	86.3
Group 2						
Female (2B+1U)	14.9 (1.3)	14.1	16.4	47.2 (37.3)	12.2	86.4
Male (4B+1U)	17.2 (1.6)	14.9	18.8	30.5 (31.2)	11.6	84.2
All (8)	16.3 (1.8)	14.1	18.8	36.8 (32.1)	11.6	86.4
Group 3						
Female (2B+1U)	14.4 (0.8)	14.0	15.4	39.3 (17.4)	19.3	50.4
Male (5B)	17.0 (1.9)	14.9	18.8	31.1 (26.1)	12.0	67.5
All (8)	16.0 (2.0)	14.0	18.8	34.2 (22.2)	12.0	67.5

B = bilateral. U = unilateral

Group 1 (Class III only); Group 2 and 3 (bilateral and unilateral Condylar Hyperplasia)

Table 2. Descriptive measures of the three groups and results of analysis of variance to test the hypothesis of equality of means of three groups before surgery (T1).

Variable	Group 1 (n=8)	Group 2 (n=8)	Group 3 (n=8)	ANOVA	
	Mean (sd)	Mean (sd)	Mean (sd)	F	p-value
SNA	79.74 (3.98)	81.15 (5.76)	78.50 (5.65)	0.52	0.601
SNB	81.66 (4.56)	82.86 (2.81)	80.79 (4.58)	0.52	0.599
ANB	-1.94 (3.38)	-1.69 (3.69)	-2.29 (4.39)	0.05	0.952
SN.PP	7.61 (4.74)	5.18 (2.68)	7.24 (3.44)	1.00	0.386
SN.OP	15.48 (9.03)	13.95 (4.85)	20.56 (7.52)	1.78	0.193
SN.MP	36.06 (9.35)	39.53 (4.07)	42.95 (6.96)	1.87	0.179
SN.Pog	63.78 (6.05)	63.11 (2.52)	67.45 (5.06)	1.91	0.172
OJ	-1.90 (1.81)	-3.28 (3.50)	-4.86 (5.12)	1.27	0.303
OB	2.36 (2.57)	2.21 (3.51)	3.18 (4.07)	0.18	0.835
U1.SN *	110.60 (7.39)	113.85 (2.48)	105.88 (10.33)	2.31	0.137
L1.SN	56.39 (10.83)	53.01 (5.51)	47.73 (8.38)	2.10	0.147
S-Go	83.40 (9.45)	76.30 (5.50)	75.89 (6.27)	2.69	0.091
N-Me	130.23 (9.13)	129.04 (7.91)	129.31 (13.68)	0.03	0.973
S-Ar	35.70^{A} (3.59)	30.98 ^{B} (2.77)	32.93 ^{AB} (3.69)	3.96	**0.035**
Ar-Go	52.85 (7.22)	49.76 (4.31)	46.18 (5.81)	2.56	0.101
Go-Me	78.34 (5.45)	81.46 (6.87)	76.55 (4.46)	1.53	0.239
Ar-Gn	123.39 (10.38)	124.75 (7.56)	116.70 (9.64)	1.73	0.202
S-Gn	148.45 (11.88)	147.51 (10.16)	141.41 (12.79)	0.86	0.438
ArGoMe	133.71 (7.17)	138.43 (7.08)	136.06 (8.39)	0.78	0.473
SN.Ar	122.03 (7.54)	119.56 (6.78)	119.79 (8.20)	0.26	0.772
ANS-Me	73.94 (6.28)	76.78 (6.74)	75.89 (9.90)	0.28	0.761

Note: Differents letters reply satatistically differents means (N-K-S test or Tamhane)
* heteroscedastic variable - ANOVA test was replaced by Brown-Forsyte

Table 3. Descriptive measures of the Group 1 (times 2 and 3), and results of Student t-test of the hypothesis of equality of means is equal to zero.

Variable	T2	T3	T3-T2	t-test	
	Mean (sd)	Mean (sd)	Mean (sd)	t	p-value
SNA	82.58 (5.14)	82.58 (5.40)	0.00 (0.87)	0.00	0.999
SNB	81.84 (4.39)	82.04 (4.16)	0.20 (0.52)	1.10	0.309
ANB	0.74 (1.70)	0.53 (1.90)	-0.21 (0.77)	-0.78	0.461
SN.PP	7.33 (4.61)	6.33 (3.12)	-1.00 (2.60)	-1.09	0.312
SN.OP	9.99 (4.41)	10.21 (3.79)	0.23 (1.77)	0.36	0.730
SN.MP	32.28 (5.88)	32.83 (5.78)	0.55 (1.36)	1.14	0.292
SN.Pog	62.08 (4.12)	62.08 (4.24)	0.00 (0.42)	0.00	1.000
OJ	3.40 (1.03)	2.85 (0.64)	-0.55 (0.84)	-1.85	0.107
OB	-1.18 (0.88)	-1.68 (1.02)	-0.50 (0.60)	-2.34	0.052
U1.SN	112.11 (6.39)	109.45 (6.95)	-2.66 (3.31)	-2.27	0.057
L1.SN	60.90 (8.70)	61.76 (6.62)	0.86 (2.91)	0.84	0.430
S-Go	84.85 (10.02)	84.34 (10.19)	-0.51 (1.36)	-1.06	0.323
N-Me	127.30 (6.54)	127.73 (6.39)	0.43 (0.98)	1.22	0.261
S-Ar	35.75 (3.78)	35.69 (3.33)	-0.06 (0.56)	-0.32	0.761
Ar-Go	54.23 (7.93)	53.76 (8.04)	-0.46 (1.18)	-1.11	0.305
Go-Me	78.36 (6.56)	79.00 (6.42)	0.64 (0.75)	2.41	**0.047**
Ar-Gn	122.85 (8.63)	123.28 (8.45)	0.43 (0.31)	3.87	**0.006**
S-Gn	148.23 (10.75)	148.38 (10.62)	0.15 (0.41)	1.02	0.340
ArGoMe	129.76 (5.66)	130.43 (5.14)	0.66 (1.88)	1.00	0.352
SN.Ar	121.76 (7.77)	121.73 (7.75)	-0.04 (0.47)	-0.22	0.830
ANS-Me	69.51 (3.87)	71.11 (3.33)	1.60 (1.56)	2.89	**0.023**

Table 4. Descriptive measures of the Group 2 (times 2 and 3), and results of Student t-test of the hypothesis of equality of means is equal to zero.

Variable	T2	T3	T3-T2	t-test	
	Mean (sd)	Mean (sd)	Mean (sd)	t	p-value
SNA	85.30 (4.33)	86.14 (4.90)	0.84 (1.41)	1.68	0.136
SNB	82.29 (3.01)	82.44 (3.16)	0.15 (0.61)	0.69	0.510
ANB	2.99 (3.02)	3.68 (2.93)	0.69 (1.00)	1.95	0.093
SN.PP	5.68 (5.33)	5.91 (4.95)	0.24 (1.91)	0.35	0.736
SN.OP	9.83 (4.45)	10.29 (4.09)	0.46 (1.21)	1.08	0.314
SN.MP	36.64 (4.13)	36.79 (4.06)	0.15 (0.86)	0.50	0.635
SN.Pog	61.91 (2.64)	61.86 (2.61)	-0.05 (0.35)	-0.41	0.695
OJ	3.65 (0.80)	2.70 (0.94)	-0.95 (1.09)	-2.47	**0.043**
OB	-1.63 (0.86)	-1.71 (0.96)	-0.09 (1.20)	-0.21	0.843
U1.SN	112.21 (3.98)	109.60 (2.78)	-2.61 (3.37)	-2.19	0.064
L1.SN	57.28 (7.90)	57.61 (7.23)	0.34 (1.93)	0.49	0.636
S-Go	77.73 (6.96)	77.63 (6.90)	-0.10 (0.82)	-0.34	0.742
N-Me	126.53 (6.48)	126.61 (6.99)	0.09 (1.00)	0.25	0.813
S-Ar	31.46 (3.12)	31.69 (3.06)	0.23 (0.63)	1.02	0.343
Ar-Go	50.79 (5.45)	50.71 (5.48)	-0.07 (0.78)	-0.27	0.793
Go-Me	80.41 (6.97)	80.36 (6.87)	-0.05 (0.76)	-0.19	0.857
Ar-Gn	123.46 (7.56)	123.46 (7.46)	0.00 (1.07)	0.00	1.000
S-Gn	146.55 (8.43)	146.48 (8.89)	-0.08 (2.35)	-0.09	0.931
ArGoMe	135.64 (7.69)	136.25 (8.11)	0.61 (1.22)	1.42	0.198
SN.Ar	119.36 (7.05)	119.63 (7.28)	0.26 (1.37)	0.54	0.606
ANS-Me	73.45 (5.22)	73.44 (5.61)	-0.01 (0.93)	-0.04	0.971

Table 5. Descriptive measures of the Group 3 (times 2 and 3), and results of Student t-test of the hypothesis of equality of means is equal to zero.

Variable	T2	T3	T3-T2	t-test	
	Mean (sd)	Mean (sd)	Mean (sd)	t	p-value
SNA	81.95 (4.32)	81.45 (4.19)	-0.50 (1.43)	-0.99	0.356
SNB	78.18 (4.75)	80.35 (4.28)	2.18 (1.39)	4.41	**0.003**
ANB	3.78 (1.98)	1.10 (1.54)	-2.68 (2.24)	-3.38	**0.012**
SN.PP	8.20 (4.67)	7.60 (4.34)	-0.60 (1.92)	-0.89	0.405
SN.OP	16.59 (5.43)	16.39 (3.13)	-0.20 (4.46)	-0.13	0.903
SN.MP	41.06 (4.88)	40.11 (4.78)	-0.95 (3.35)	-0.80	0.448
SN.Pog	67.83 (4.08)	66.35 (3.43)	-1.48 (1.66)	-2.52	**0.040**
OJ	4.34 (1.06)	1.35 (2.21)	-2.99 (1.64)	-5.14	**0.001**
OB	-2.09 (1.03)	-0.64 (1.42)	1.45 (1.16)	3.52	**0.010**
U1.SN	105.40 (11.00)	106.18 (6.96)	0.77 (5.15)	0.43	0.683
L1.SN	52.95 (6.16)	53.43 (7.10)	0.48 (5.30)	0.25	0.807
S-Go	77.05 (6.96)	78.69 (7.74)	1.64 (2.58)	1.80	0.115
N-Me	126.95 (11.66)	128.33 (12.85)	1.38 (2.63)	1.48	0.182
S-Ar	33.56 (4.46)	33.45 (4.15)	-0.11 (0.62)	-0.51	0.625
Ar-Go	46.28 (7.36)	48.50 (8.47)	2.23 (2.66)	2.37	**0.050**
Go-Me	73.64 (2.63)	75.20 (4.48)	1.56 (2.82)	1.57	0.161
Ar-Gn	112.65 (9.02)	116.41 (10.09)	3.76 (1.48)	7.20	**0.000**
S-Gn	140.36 (13.06)	142.66 (14.09)	2.30 (2.34)	2.78	**0.027**
ArGoMe	132.60 (7.06)	133.54 (6.85)	0.94 (2.45)	1.08	0.314
SN.Ar	119.09 (8.39)	119.04 (8.42)	-0.05 (0.87)	-0.16	0.876
ANS-Me	71.79 (7.62)	73.85 (9.36)	2.06 (2.20)	2.65	**0.033**

Table 6. Descriptive measurements and Student-t test for equality of means in the three groups during the overall evaluation period.

Variable	Group 1 (n=8)	Group 2 (n=8)	Group 3 (n=8)	ANOVA	
	Mean (sd)	Mean (sd)	Mean (sd)	F	p-value
SNA	0.00 (0.87)	0.84 (1.41)	-0.50 (1.43)	2.293	0.126
SNB	0.20 ^{A} (0.52)	0.15 ^{A} (0.61)	2.18 ^{B} (1.39)	12.399	**0.000**
ANB	-0.21 ^{A} (0.77)	0.69 ^{A} (1.00)	-2.68 ^{B} (2.24)	11.006	**0.001**
SN.PP	-1.00 (2.60)	0.24 (1.91)	-0.60 (1.92)	0.680	0.517
SN.OP *	0.23 (1.77)	0.46 (1.21)	-0.20 (4.46)	0.110	0.897
SN.MP *	0.55 (1.36)	0.15 (0.86)	-0.95 (3.35)	1.050	0.385
SN.Pog *	0.00 (0.42)	-0.05 (0.35)	-1.48 (1.66)	5.534	**0.029**
OJ	-0.55 ^{A} (0.84)	-0.95 ^{A} (1.09)	-2.99 ^{B} (1.64)	8.930	**0.002**
OB	-0.50 ^{A} (0.60)	-0.09 ^{A} (1.20)	1.45 ^{B} (1.16)	8.011	**0.003**
U1.SN	-2.66 (3.31)	-2.61 (3.37)	0.77 (5.15)	1.906	0.174
L1.SN	0.86 (2.91)	0.34 (1.93)	0.48 (5.30)	0.044	0.957
S-Go	-0.51 (1.36)	-0.10 (0.82)	1.64 (2.58)	3.404	0.052
N-Me *	0.43 (0.98)	0.09 (1.00)	1.38 (2.63)	1.206	0.336
S-Ar	-0.06 (0.56)	0.23 (0.63)	-0.11 (0.62)	0.731	0.493
Ar-Go *	-0.46 (1.18)	-0.07 (0.78)	2.23 (2.66)	5.574	**0.021**
Go-Me *	0.64 (0.75)	-0.05 (0.76)	1.56 (2.82)	1.729	0.231
Ar-Gn *	0.43 ^{A} (0.31)	0.00 ^{A} (1.07)	3.76 ^{B} (1.48)	29.754	**0.000**
S-Gn	0.15 (0.41)	-0.08 (2.35)	2.30 (2.34)	3.685	**0.043**
ArGoMe	0.66 (1.88)	0.61 (1.22)	0.94 (2.45)	0.067	0.936
SN.Ar	-0.04 (0.47)	0.26 (1.37)	-0.05 (0.87)	0.261	0.773
ANS-Me *	1.60 (1.56)	-0.01 (0.93)	2.06 (2.20)	3.493	0.056

Note: Differents letters reply satatistically differents means (N-K-S test or Tamhane)

* heteroscedastic variable - ANOVA test was replaced by Brown-Forsyte

CONSIDERAÇÕES FINAIS

6 CONSIDERAÇÕES FINAIS

O presente estudo avaliou a influência da hiperplasia condilar no crescimento mandibular pós-pubertário de pacientes portadores de más oclusões esqueléticas de classe III com e sem Hiperplasia Condilar e a estabilidade do tratamento cirúrgico implementado, através da avaliação retrospectiva de Telerradiografias de pacientes que já haviam finalizado o tratamento Orto-cirúrgico. Foi demonstrado que:

1) ▪ Para o grupo com Hiperplasia Condilar, um maior crescimento mandibular total (Ar-Gn = 2.61 mm) e um maior comprimento do corpo mandibular (Go-Me = 2.2 mm) foi observado durante o período pós-pubertal;

2) ▪ Para a obtenção de resultados estáveis no tratamento cirúrgico de pacientes da classe III, é de fundamental importância identificar a presença ou ausência de Hiperplasia Condilar e se ela ainda está ativa, além da adoção de uma abordagem de tratamento personalizada;

3) ▪ A cirurgia ortognática para a correção da má oclusão esquelética de Classe III é um procedimento estável para pacientes sem anormalidades crescimento condilar e para pacientes com diagnóstico

de Hiperplasia Condilar ativa submetidos simultaneamente a cirurgia ortognática, condilectomia alta e reposicionamento do disco articular. Pacientes com Hiperplasia Condilar que foram submetidos somente à cirurgia ortognática sem nenhuma intervenção da ATM, experimentaram recidiva significante.

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* De acordo com o manual da FOAr/UNESP, adaptadas das normas Vancouver. Disponível no site: http://www.nlm.nih.gov/bsd/uniform_requirements.html

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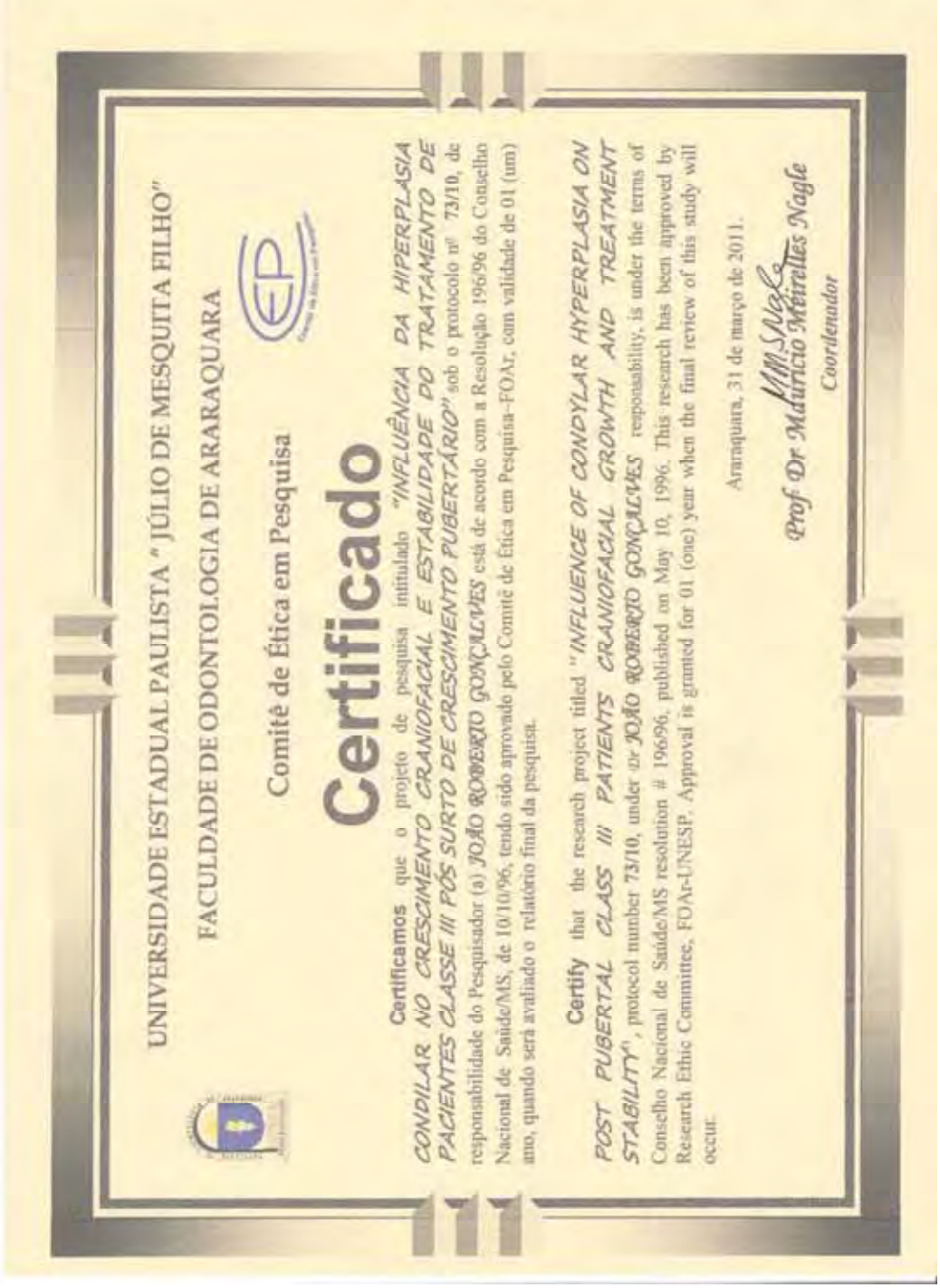
ANEXOS

Certificado do Comitê de Ética



UNIVERSIDADE ESTADUAL PAULISTA "JÚLIO DE MESQUITA FILHO"

FACULDADE DE ODONTOLOGIA DE ARARAQUARA

Comitê de Ética em Pesquisa

Certificado

Certificamos que o projeto de pesquisa intitulado *"INFLUÊNCIA DA HIPERPLASIA CONDILAR NO CRESCIMENTO CRANIOFACIAL E ESTABILIDADE DO TRATAMENTO DE PACIENTES CLASSE III PÓS SURTO DE CRESCIMENTO PUBERTÁRIO"* sob o protocolo nº 73/10, de responsabilidade do Pesquisador (a) *JOÃO ROBERTO GONÇALVES* está de acordo com a Resolução 196/96 do Conselho Nacional de Saúde/MS, de 10/10/96, tendo sido aprovado pelo Comitê de Ética em Pesquisa-FOAr, com validade de 01 (um) ano, quando será avaliado o relatório final da pesquisa.

Certify that the research project titled *"INFLUENCE OF CONDYLAR HYPERPLASIA ON POST PUBERTAL CLASS III PATIENTS CRANIOFACIAL GROWTH AND TREATMENT STABILITY"*, protocol number 73/10, under Dr *JOÃO ROBERTO GONÇALVES* responsability, is under the terms of Conselho Nacional de Saúde/MS resolution # 196/96, published on May 10, 1996. This research has been approved by Research Ethic Committee, FOAr-UNESP. Approval is granted for 01 (one) year when the final review of this study will occur.

Araraquara, 31 de março de 2011.

Prof Dr Maurício Meirelles Nagle

Coordenador

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Araraquara, 04 de março de 2013.

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