Management of anterior open bite

Case-report

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Forord

Kunnskap om vekst, okklusjonsutvikling og kjeve- og ansiktsanalyse (kefalometri) er det essensielle grunnlaget for all kjeveortopedi. Det gjelder å forstå hva slags ansikt man har for seg i hvert enkelt tilfelle, hvor feilen "sitter", samt hvilke behandlingsmuligheter og begrensninger man har. Dessuten er det viktig å påvise hvilke dysfunksjoner som måtte forekomme, deres sannsynlige etiologi, hva de gjør med tannstilling og funksjon, samt hvordan man eventuelt kan avhjelpe problemet.

Anteriort åpent bitt er en malokklusjon med multifaktoriell etiologi som er vanskelig å behandle, og hvor behandlingsresultatet ofte ikke er stabilt. Med denne oppgaven ønsket vi å finne ut hvorfor det er slik, og om det finns noen behandlinger med god langtidsprognose.
Oppgaven er en litteraturstudie om anteriort åpent bitt og behandling av denne malokklusjonen. Litteraturstudiet er supplert med en kasuspresentasjon.

Vi vil benytte anledningen til å takke våre veiledere Maud Els-Marie Andersson, Gisela Vasconcelos og Espen Færøvig for god hjelp og veiledning i arbeidet med denne oppgaven.

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Introduction

Anterior open bite (AOB) is a malocclusion that occurs in the vertical plane defined by lack of vertical overlap between the upper and lower incisors when the mandible is brought into full occlusion *(Nanda 2005)*. AOB is also defined by a reversed overjet (<0 mm) *(Thilander, Bjerklin et al. 2018)*. AOB is less common than deep bites, and the demand for treatment is approximately 17% *(Proffit, 2012)*. The prevalence of AOB in the general population ranges from 1.5% to 11% and varies in relation to age and ethnicity *(Zuroff, Chen et al. 2010)*. The diagnosis and successful treatment of AOB are among the most challenging and difficult tasks for clinicians involved in orthodontic treatment *(Kim 1974)*.

Many potential etiological factors are associated with AOB, including a combination of heredity, unfavorable growth, environmental factors, tongue and thumb behaviors, abnormal orofacial muscle function and the interaction of these factors with skeletal growth *(Linder-Aronson and Woodside et al. 1986)*. AOB can also result in functional disorders, such as difficulties in speech, cutting, chewing, and swallowing, and can affect psychosocial development due to its esthetic impact *(Teittinen, Tuovinen et al. 2012; Dimberg, Lennartsson et al. 2013)*. All children experience AOB during the transition from primary to permanent dentition *(Dimberg, Lennartsson et al. 2013)*. In younger children, oral habits are the major cause of AOB, however, many of these simple AOB cases resolve by adolescence *(Worms, Meskin et al. 1971)*. Incompetent lips and hyperactive mentalis muscle activity are common findings in individuals with skeletal AOB *(Lopez-Gavito et al. 1985; Ngan and Fields 1997; Hart et al. 2015)*. The choice of treatment protocol depends on many factors, including occlusal esthetic aspects, morphologic characteristics, and the severity of the skeletal anomaly *(Bjørk 1955; Sassouni and Nanda 1964; Schudy 1964; Enlow, Kuroda et al. 1971; Isaacson et al. 1971; Bjørk and Skieller 1972, 1983; Nahoum, Horowitz et al. 1972; Nahoum 1975, 1977; Schendel et al. 1976; Frost et al. 1980; Cangialosi 1984; Beckmann, et al. 1998; Kuitert et al. 2006)*.

Classification

Open bite (OB) and AOB occur in many varieties, but there is no universal consensus about their classification *(Subtelny, Sakuda et al. 1964)*. Morphologically, AOB can be categorized as being of dentoalveolar or skeletal origin *(Greenlee et al. 2011)*. Various terms have been used interchangeably to differentiate between OB and AOB, such as: by describing angles, growth tendencies, occlusal traits, implying etiology, dolichofacial, leptoprosopic, hyperdivergent, skeletal open bite, high angle, backward rotator (posterior rotation), adenoid faces, "Long face syndrome" with vertical maxillary excess. Cook, Sellke et al. (1994) classified AOB into skeletal, habit or dental, abnormal tongue function, iatrogenic, and pathological factors. They further divided AOB into dental (abnormal eruption of the incisors), dentoalveolar (change in the vertical growth of the alveolar component), and skeletal (unfavorable vertical growth pattern). AOB has also been divided into Simple (from canine to canine), Compound (from premolar to premolar), and –Infantile) (from molar to molar) *(Isaacson et al., 1971; Worms, Meskin et al. 1971)*. According to Yamaguchi (2010) skeletal AOB can be divided into either long face caused by clockwise (backward) rotation of the mandible or skeletal deformation (such as tipping of the maxilla and diversion of the gonial angle of the mandible). Richardson (1981) created a clinical classification of AOB based on its etiology: transitional (due to eruption of teeth), due to habits (finger, thumb or digit sucking), due to local pathology (supernumerary teeth, ankyloses, cysts), non-pathological skeletal group, skeletal open bites (improving), deteriorating (improving but later), and deteriorating skeletal (de novo) open bites. Only 13% of patients with skeletal OB had a tendency for AOB with a vertical space between the incisors perpendicular to the occlusal plane. Skeletal OB is often camouflaged by overeruption of the anterior teeth *(Dung, Smith et al. 1988)*.

Etiology

The cause of AOB is generally multifactorial and can be due to a combination of skeletal, dental and soft tissue effects. Skeletal growth and maturation are under the control of a great many factors, such as genetic, endocrine, environmental, and functional. These factors can act alone or in combinations *(Subtelny, Sakuda et al. 1964; Mizrahi 1978)*. In younger

children, the major causes of AOB are non-nutritive sucking habits, increased tongue size and tongue position. An overbite established at 8 years of age is most likely to increase until 12 years of age and then decrease from 12-18 years of age *(Bergersen 1988)*. Some AOBs persist into the late stages of growth, and in some patients a persistent AOB develops during the adolescent stage of development, even though serial records have shown no evidence of an AOB at the preadolescent stage. This may indicate that other factors can influence the development of an AOB, factors which may be difficult to diagnose and differentiate at earlier stages of growth. AOB can be due to many causes, including both hereditary and environmental factors. Environmental factors include tongue thrust, prolonged thumb or finger sucking, mouth breathing, atypical deglutition, disproportionate neuromuscular growth and weak masticatory muscle force *(Bjørk, Krebs et al. 1964; Moss and Salentijn 1971; Bjørk and Skieller 1972; Speidel, Isaacson et al. 1972; Schendel et al. 1976; Mizrahi 1978; Ngan and Fields 1997; Yamaguchi 2010)*.

Many potential etiological factors have been considered, including breathing deficiency due to obstructive pharyngeal airways caused by hypertrophied adenoids (enlarged lymphatic tissue) *(Subtelny, Sakuda et al. 1964)*. Skeletal AOB may be related to the morphology and behavior of the tongue and lips *(Yamaguchi 2010).* AOB is also related to excessive growth of the dento-alveolar complex, especially in the posterior molar region *(Sassouni 1969)*, or backward mandibular growth rotation *(Bjørk 1969; Bjørk and Skieller 1983)*.

Posterior face height (Sella-Gonion) versus Anterior face height (Nasion-Menton) are used to measure vertical growth pattern *(Jarabak and Fizzell 1972).* AOBs are often registered in individuals with a Dolichofacial pattern *(Ricketts 1968)* and are also found in individuals with a hypodivergent facial pattern *(Schudy 1963)*.

Skeletal OB has been described with an unfavorable growth pattern with divergent basal bones e.g. steepness of mandibular plane, increased gonial angle, short mandibular ramus, increased anterior lower face height, backward rotation of the mandible *(Subtelny, Sakuda et al. 1964)*. In AOB there is undergrowth of the anterior segment combined with excessive growth of the posterior alveolar portion. A patient with AOB may have a long and narrow

face with marked convex profile. Lower facial height is a feature under strong genetic influence *(Schudy 1964; Sassouni 1969; Nanda 1988)*.

Prevalence

The prevalence of AOB in different studies varies according to the methods of assessment, ethnicity differences, and the chronological age of the sample. Among the barriers to accurately estimating the prevalence of AOB are: (a) heterogeneity of open bites; (b) the lack of standard criteria for the collection of data; and in particular (c), the lack of/and or failure to apply an internationally comparable classification system for open bite and differences in the developmental status of the cohort *(Thilander and Myrberg 1973; Thilander, Bjerklin et al. 2018)*. In the primary dentition it is reported that 50% of 3-year-old children have AOB *(Cozza, Mucedero et al. 2005; Dimberg 2015)*. However, most of these AOB cases self-correct as sucking habits cease, and thus, only about 4% of school children and adolescents show AOB *(Worms, Meskin et al. 1971; Cozza et al. 2005)*. In the U.S., AOB occurs in approximately 16% of African-American children, but only 4% of Caucasian. Among all of the skeletal characteristics associated with AOB. AOB is more prevalent during the early stages of growth than at the later stages. This observation can be explained by the prevalence of sucking habits at early ages, by the growth of lymphatic tissue, and by the still inadequate growth of the skeletal jaws. Followed by raising of the tongue and closing of the lips, and leading to changes in dentition *(Linder-Aronson, Woodside et al. 1986; Behlfelt et al. 1990)*. Skeletal OB may be camouflaged by overeruption of the anterior teeth *(Dung, Smith 1988)*.

Cephalometric analyses

Bjørk (1955) designed a cephalometric analysis that defined whether malocclusions are of dentoalveolar or skeletal origin, focusing on the role of the interaction between the sagittal and the vertical development and its impact on the craniofacial skeleton. Face height is measured by using the nasion sella line (NSL, equivalent to the cranial base) in vertical relation to the nasal line (NL) (nasal floor and the upper part of the maxilla) and the mandibular line (ML) (corresponding to the mandibular lower border). The angle between the NSL and ML, as well as the NSL and NL is a measure of the face height. In skeletal OB with an increased anterior face height, the angle between NSL and ML and there a high-

angle (hyperdivergent) between the mandibular plane and to the cranial base. The Bjørk (1955) indicators provide a useful method to for predicting vertical growth patterns. However, these indicators are based on patients with extreme variations in vertical facial growth; thus, they may be of limited value in patients with less severe open-bite skeletal patterns. Posterior growth rotation of the mandible occurs when there is a discrepancy between the amount of growth in anterior and posterior facial heights. The ANB angle, the facial plane angle and the Y-axis angle show little of the depth of overbite *(Schudy 1968)*. Any elongation of the posterior teeth will induce a clockwise rotation of the mandible *(Ari-Viro and Wisth 1983; Lee et al. 1987)*. Vertical skeletal dysplasias may be inherited, and vertical dysplasias can be associated with either a Class I, Class II or Class III skeletal relationship *(Sassouni 1969; Mizrahi 1978)*. The face grows from two to three times as much vertically as anteroposteriorly and thus becomes longer over time. It was Schudy in 1963 who introduced the term "hyper- and hypodivergent" in relation to facial growth patterns. In cases with a low-angle (hypodivergent), the mandible plane in relation to the cranial base is reduced (NSL/ML<27°), and in high-angle cases (hyperdivergent) this angle is increased (NSL/ML>39°). Hyperdivergent individuals showed an increased anterior face height due to a backward and posterior growth rotation of the mandible *(Schudy 1964, 1965, 1968).* It is observed that there are deep overbites in the hyperdivergent facial type. Similarly, AOBs are also found in the hypodivergent facial type. According to Sassouni (1969), in the hyperdivergent facial type the four bony planes of the face are in steep relation to each other, bringing the center close to the profile. The posterior vertical chain of muscles is accurate, and the masseter muscle is posterior to the buccal teeth, thus creating a mesial component of forces responsible for the dental protrusion, or the cranial base angle is obtuse. According to Schudy, the ratio of depth to height affects not only the facial type but also degree of overbite in AOB *(Schudy 1963; Sassouni and Nanda 1964).* Worms, Meskin et al. (1971) reported that in general, the overbite increases during the mixed dentition period, and 80% of AOB cases self-correct from a 7-to-9-year-old to a 10-to-12-year-old sample. There seems to be a slight decrease in overbite from 12 to 18 years of age and some correlation between the overbite at different ages *(Baumrind et al. 1981).*

Predicting a patient's growth potential

Growth patterns are established early in life, and maintained over time in most people *(Hartsfield et al. 2002)*. Control of the vertical growth pattern is difficult, and changes in the dentoalveolar complex may directly affect the most representative skeletal features of an OB. For example, controlling the vertical eruption of the molars allows the mandible to rotate in a counterclockwise direction, reducing the excessively vertical skeleton facial pattern. Therefore, a skeletal open-bite pattern could be evident in the early mixed dentition. Early morphological signs among patients with vertical growth potential include inclination of the condylar head, curvature of the mandibular canal, shape of the lower border of the mandible, inclination of the symphysis, anterior lower facial height, inter incisal angle, inter premolar and inter molar angle *(Bjørk and Skieller 1983)*.

The "overbite depth indicator index" of the primary dentition (ODI)

It has been suggested that the overbite depth indicator (ODI) index can be a good predictor in the primary dentition of a skeletal OB tendency in adolescence *(Kim 1974; Klocke, Nanda et al. 2002).* This index is a composite of cephalometric measurements involving the mandibular and palatal plane. A value of 68° or less indicates a skeletal open bite tendency. Another method for predicting skeletal growth is the ratio of the upper anterior face height to the lower anterior face height (AUFH:ALFH). If the patient has an AOB and the ratio is less than 0.65, then it might be impossible to correct the AOB with only orthodontic treatment *(Nahoum 1975)*. Skeletal signs have been identified in Class I and Class II open bite subjects in the early transitional dentition, although self-correction of the OB has been demonstrated *(Bjørk 1969).*

Long Face Syndrome

Individuals who have excessive vertical growth show a characteristic facial appearance and have been described as having "long-face syndrome" *(Proffit 2012)*. Because the excessively long lower face is often accompanied by an AOB, this condition has also been categorized as "skeletal OB". However, not all long-faced patients have AOB and not all AOB patients are long-faced. Individuals with "long face syndrome" may have a short upper lip with excessive maxillary incisor exposure *(Bell 1971; Nahoum 1977)*. The extent of the dentoalveolar AOB depends on the extent of the eruption of the teeth, e.g. supraocclusion of the molars and

infraocclusion of the incisors can be the primary etiologic factors. Skeletal AOB is mostly of hereditary origin, although abnormal functions and altered soft tissues posture may also contribute to open bite *(Straub 1960; Sassouni 1969; Mizrahi 1978; Proffit and Fields 1983)*, and Straub (1960) found in their studies a correlation between orofacial musculature and facial structure, suggesting a relationship between tongue position and AOB pattern. This pattern can be seen in patients with an extended head posture due to prolonged obstruction of nose breathing, which can contribute to the further development of a skeletal OB.

Transitional AOB

AOB often occurs when the permanent teeth are erupting. Growth of the dentoalveolar process and up-righting of incisors initiate a spontaneous closure of AOB. AOB that is apparent in the pre-pubertal stage may close in the pubertal stage and then reappear in the post-pubertal stage. This is due to the interplay between vertical facial growth and compensating dentoalveolar growth, which is sufficient to close the open bite *(Subtelny, Sakuda et al. 1964).*

Tongue thrust and thumb-sucking

Tongue thrust and thumb-sucking have been suggested as etiologic factors of AOB. Recommended treatment for AOB related to tongue thrust are myofunctional therapy, palatal crib and lingual prongs on the incisors *(Straub 1960; Subtelny, Sakuda et al. 1964; Parker 1971)*. After long-term tongue thrust, the maxillary may become narrow, the molars passively super erupt, and the mandible rotates clock-wise, all factors that contribute to an increase in anterior face height. Immediately after the crib placement, the overjet increases due to the tip of the tongue being positioned posteriorly during all stages of deglutition. Additionally, the anterior and middle parts of the dorsum of the tongue are at a lower position, reflecting a compensatory functional change for bolus propulsion and airway protection. Taslan, Biren et al. (2010) reported a decrease in resting tongue pressure and swallowing pressure after 10 months of tongue crib treatment in individuals with AOB. This may be an adaptation in response to the altered environmental changes *(Cozza et al. 2006)*. Persistence of AOB may be associated with neuromuscular imbalance or a divergent growth pattern. Re-opening of a corrected AOB at long-term follow up is a common finding, not only

after orthodontic treatment, but also after surgical orthodontic therapy *(Lopez-Gavito, et al. 1985; Fischer, von Konow et al. 2000)*. When thumb- and/or finger-sucking is the obvious cause of the open-bite, there will probably be inadequate development of the anterior alveolar processes as well as incomplete eruption of the incisor teeth. It is not unusual to see the anterior aspect of the tongue enter the open-bite area during swallowing.

Airway

Individuals with obstructive sleep apnoea and mouth breathing have characteristic cephalometric patterns such as: long face and increased lower anterior facial height, AOB and cross-bites *(Ricketts 1968; Linder-Aronson 1970; Harvold et al. 1972; Linder-Aronson 1974; Fränkel and Fränkel 1983; Kikuchi et al. 2002).* Enlarged tonsils create a mechanical obstruction in the oro-pharynx posterior to the root of the tongue. The tongue may be forced to move forward, away from its normal position, which can cause nasal airway obstruction, leading to induced mouth breathing. The lower tongue position can also increase the inter-maxillary space leading to overdevelopment of the buccal segment, which increases the height of the lower third of the face and cause an AOB. Improvements in dental position have been registered in children after adenoidectomy and the reduced size of the adenoids after surgery allows switching from oral to nasal respiration, followed by raising of the tongue and closing of the lips, and leading to changes in dentition *(Subtelny, Sakuda et al. 1964; Linder-Aronson, Woodside et al. 1986)*. In one study, children with obstructive sleep apnoea due to enlarged tonsils had a significantly more posteriorly inclined mandible; a more anteriorly inclined maxilla, a greater lower anterior face height and retroclined upper and lower incisors *(Zettergren-Wijk, Forsberg et al. 2006).* Mouth breathers may be divided into either “Adenoid faces”, Skeletal OB, or “Long face syndrome”. Adenoid faces are characterized by: long narrow face; narrow nose and nasal airway; flaccid lips with short upper lip; and upturned nose exposing nares frontally. Skeletal OB or “Long Face syndrome” are characterized by excessive eruption of posteriors; constricted maxillary arch; and excessive overjet. The pressure from the tongue may be unfavorable to skeletal and dentoalveolar development, e.g. the mandible grew vertically in its posterior and anterior regions as well as in its anteroposterior dimension. There is no clear-cut relationship between breathing and AOB. The majority of individuals with a long face have no evidence

of nasal obstruction or AOB *(Linder-Aronson, Woodside et al. 1986; Shanker et al. 2004)*. Behlfelt et al. (1990) reported that young children with enlarged tonsils use mouth-breathing 59% of the time during the day and 82% during the night. Procedures that promote better breathing through the nose (turbinate surgery, adenoid and tonsil removal, allergy treatment) may help to reestablish normal growth patterns *(Harvold et al. 1981; Linder-Aronson, Woodside et al. 1986).* However, the growth direction of the mandible among patients varies greatly after any of these procedures *(Linder-Aronson, Woodside et al. 1993)*. This variability makes the decision to intervene with a respective surgical procedure difficult. Therefore, the diagnosis of upper airway obstruction and the decision for surgical intervention should always be made by an appropriate team of specialists.

Treatment in patients with AOB

Treatment of skeletal AOB is "vertical growth modification" in growing patients and "intrude posterior teeth" in non-growing patients. The goal of treatment is to inhibit the vertical development with intrusion of the buccal dentoalveolar segments. Achieving an ideal treatment outcome for AOB depends on an accurate diagnosis in three dimensions, a good understanding of the interaction between the neuromuscular components of the orofacial region and the craniofacial skeleton, and the ability to provide individualized treatment mechanics *(Fränkel and Fränkel 1983; Kuster and Ingervall 1992)*, by either eliminating the cause or correcting dentofacial changes, with the objective of improving mastication, respiratory function and swallowing *(Schudy 1964; Sassouni 1969, Nanda 1988; Ali, Pitson et al. 1993; Gottlieb et al. 2003)*. Factors that might interfere with reaching stability are tongue size, muscular dystrophy, amelogenesis imperfect, and idiopathic condylar resorption. However, according to Lentini-Oliveira, Carvalho et al. (2014), treatment protocols have been shown to have low scientific evidence for treatment stability. The treatment of skeletal AOB is often postponed until growth has terminated because the prognosis for stability is highly dependent on the remaining growth. Different factors considered in treatment planning are: overbite and the steepness of the mandible *(Schudy 1964; Creekmore 1967)*, the degree of the gonial angle *(Jensen and Palling 1954),* the degree of occlusomandibular plane angle *(Schudy 1963)*, and the ratio between the anterior and posterior facial heights *(Wylie 1946; Wylie and Johnson 1952)*. Different treatment options for a dentoalveolar AOB

are: headgear, posterior bite blocks, second molar extraction, intrusion of the buccal dentoalveolar segments by first molar extraction, premolar extraction, first or second premolar extraction due to crowding and/or overjet *(Hart et al. 2015)*.

Temporomandibular Pain & Dysfunction

The posterior rotation of the mandible in children with juvenile idiopathic arthritis (JIA) involving the temporomandibular joint (TMJ) differs from the posterior growth rotation of the mandible seen in the "Long face syndrome" *(Bjørk and Skieller 1983; Pepicelli, Woods et al. 2005)*. This leads to a weaker bite force, and it increases the OB and development of an AOB *(Kim et al. 2000)*. The inflammatory destruction of the mandibular condylar cartilage/condyle in JIA patients places the condyle at a more anterior position in the glenoid fossa *(Peltomäki et al. 2015)*. During periods of dysplastic mandibular growth, apposition of bone gradually takes place at the gonial angle, leading to antegonial notching *(Bjørk and Skieller 1983; Peltomäki et al. 2015)*.

Search strategy

The paper comprises a review of the scientific literature on the malocclusion "Open bite". To identify studies of etiology and the morphologically description of "Open bite", a literature survey was done by applying Electronic databases: the Medline database (Entrez PubMed, http://www.ncbi.nlm.nih.gov/pubmed), and the Cochrane Library were used. The survey covered the period from 1950 to 2019 and used the MeSH terms:"open bite" or "malocclusion" or "orthodontics".

Summary

The diagnosis and successful treatment of AOB are among the most challenging and difficult tasks for clinicians involved in orthodontic treatment *(Kim 1974)*.

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Case report

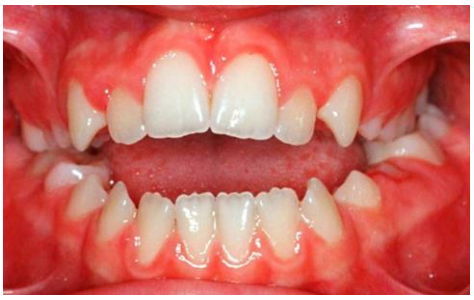
Masterthesis
Oslo 2020

By Eirik Bergan-Skar og Hanna Alexandra Adelved

Supervisors: Maud Els-Marie Andersson, Gisela Vasconcelos, Espen Færøvig

UiO : University of Oslo

UiO : Det odontologiske fakultet



Name: E.H.

10 yrs. and 5 mnd. old

Gender: Female

Ethnicity: Norwegian

Helfo: Gr. B open bite, with 75%

Patient overview

Profile: Convex profile, incompetent lips with tongue and lip dysfunction
Dental: Angle Cl. II. Overjet: +5 mm, overbite: -7 mm.
Maxillary space condition + 3mm total
Mandibular space condition +1 mm total
Maxillary incisor protrusion (Is to Apg: 7.8 mm)
Maxillary incisor proclination (ILs/Apg: 35.0º)
Mandibular incisor protrusion (Ii to Apg: 3.7 mm)
Mandibular proclination (Ili/Apg: 24.0º).
Slightly low interincisal angle (Ili/Ils:115.0º)
Skeletal sagittal: Quite normal maxilla (SNA: 79.8º)
Tendency retrognathic mandible (SNB: 75.3º)
ANB : 4.4
Skeletal vertical: Posterior rotation of mandible (ML/NSL: 44.3º)
Increased basal vertical jaw relation (ML/NL: 37.9º)

Patient main concern: Dissatisfied with function and esthetics

Treatment type: Late mixed corrective and comprehensive treatment

Appliances: HHG and TPB with spurs
Fixed appliance (Edgewise 0.022” technique) in both jaws**.**

Treatment result

Treatment start: April 2011

Treatment end: September 2013

Treatment duration: 2 years 5 months

Treatment result

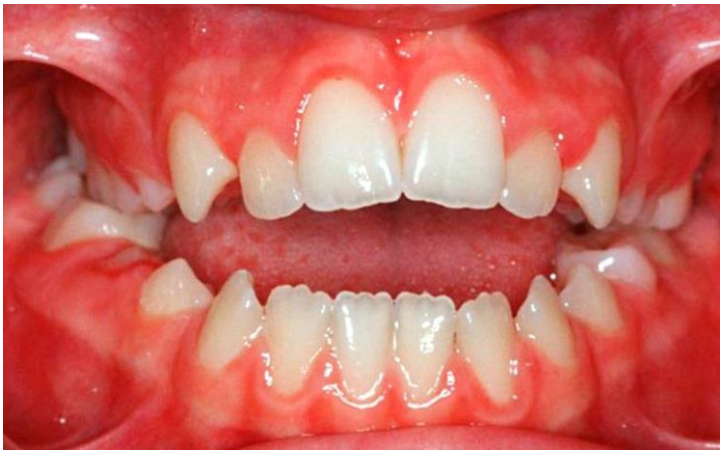
Occlusion: Good
Molar relationship: Class I
Canine relationship: Class I
Overjet: 2 mm
Overbite: 1 mm
Facial profile: Straight, full and competent lips
Function: Good function, canine guidance on laterotrusion.
No signs or symptoms of TMD

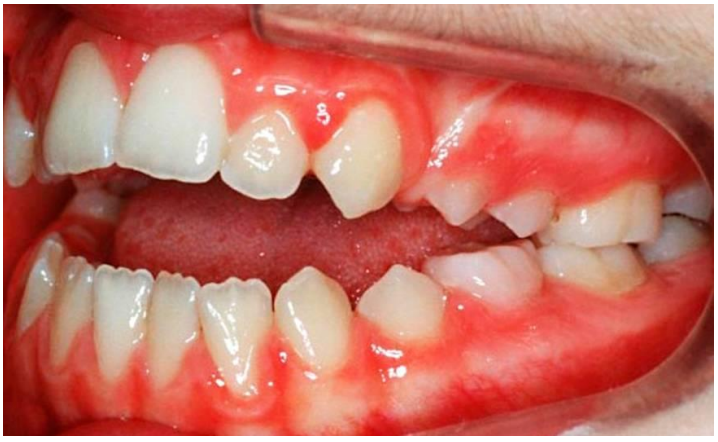
Retention

Maxilla	Bonded retainer 13-2-1-1-2-23 ZMRW 0.0215” Penta-one twisted wire. Labial buttons Vacuum pressed Stavac plate 2.0 mm
Mandible	Bonded retainer 33-2-1-1-2-43 ZMRW 0.0215” Penta-one twisted wire. Labial buttons

Pre-treatment intra-oral photos

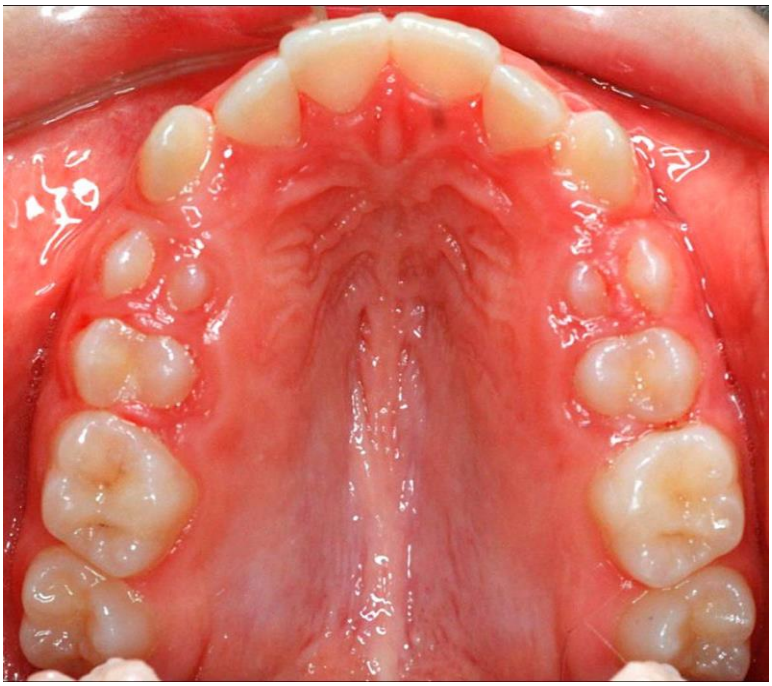
10 years 5 mnd

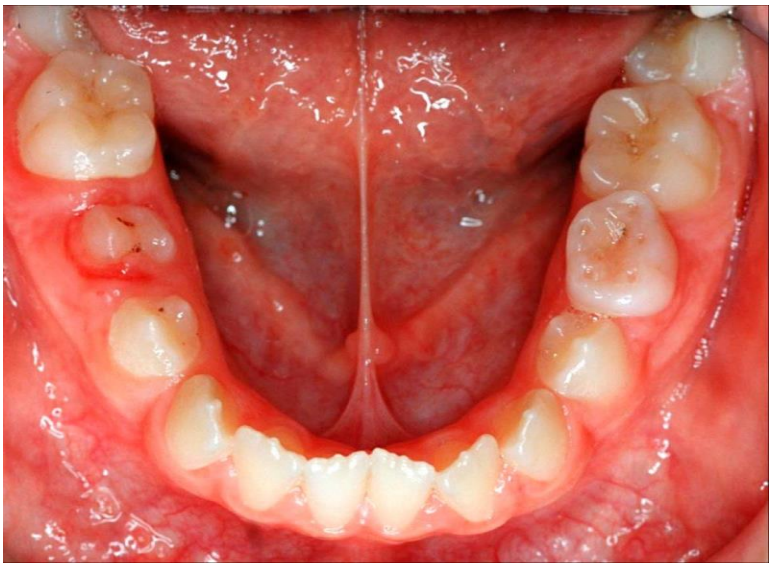




Pre-treatment intra-oral photos

10 years 5 mnd

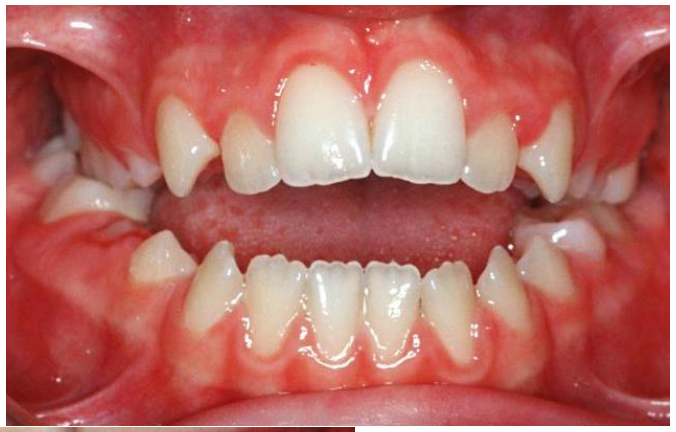


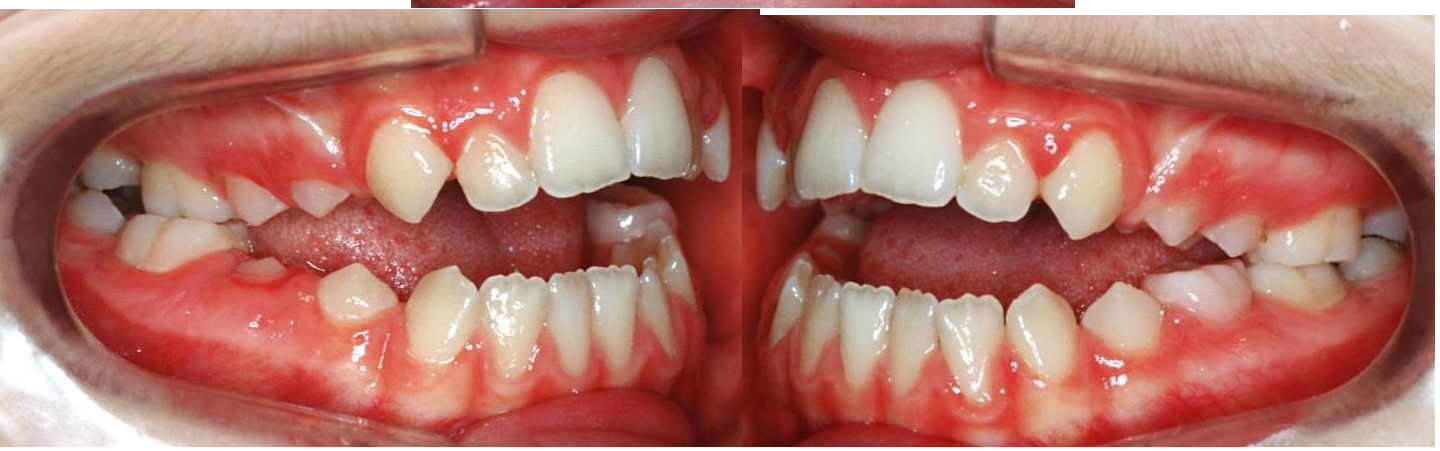


Pre-treatment intra-oral photos
10 years 5 mnd

Frontalt åpent bitt

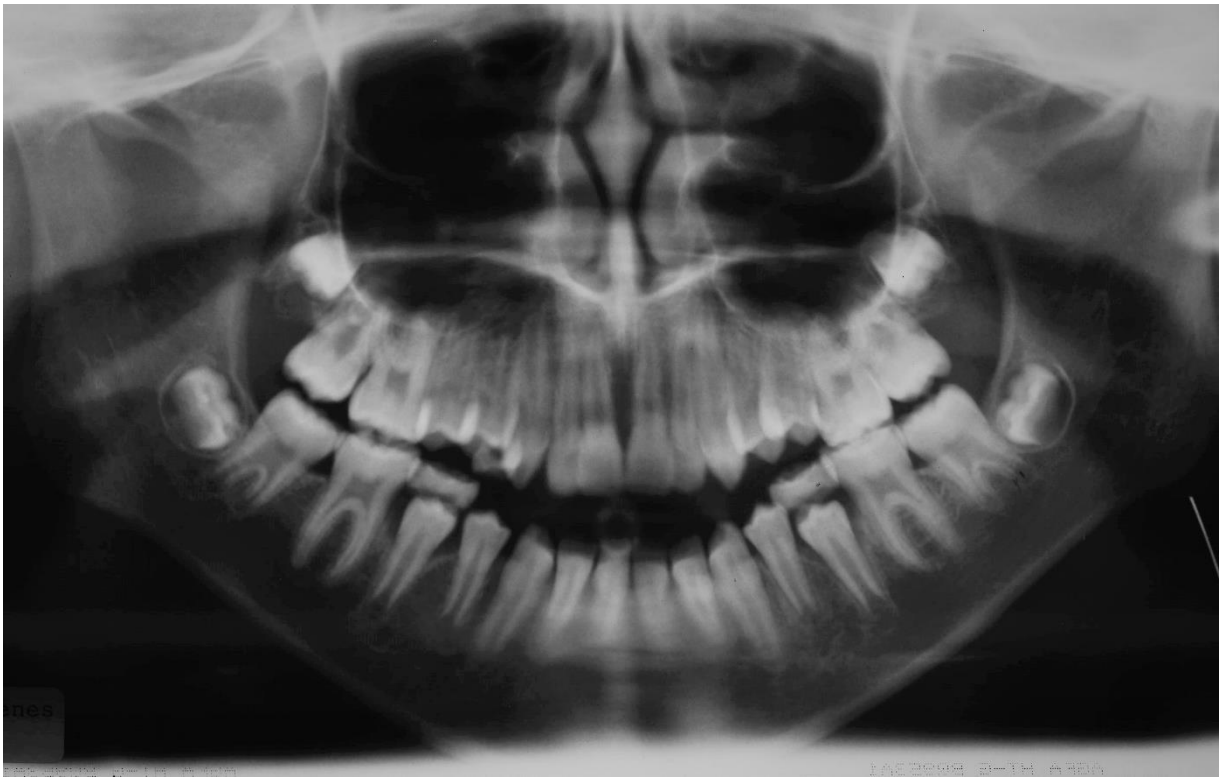
Jente i sent blandigstannsett





Pre-treatment OPG

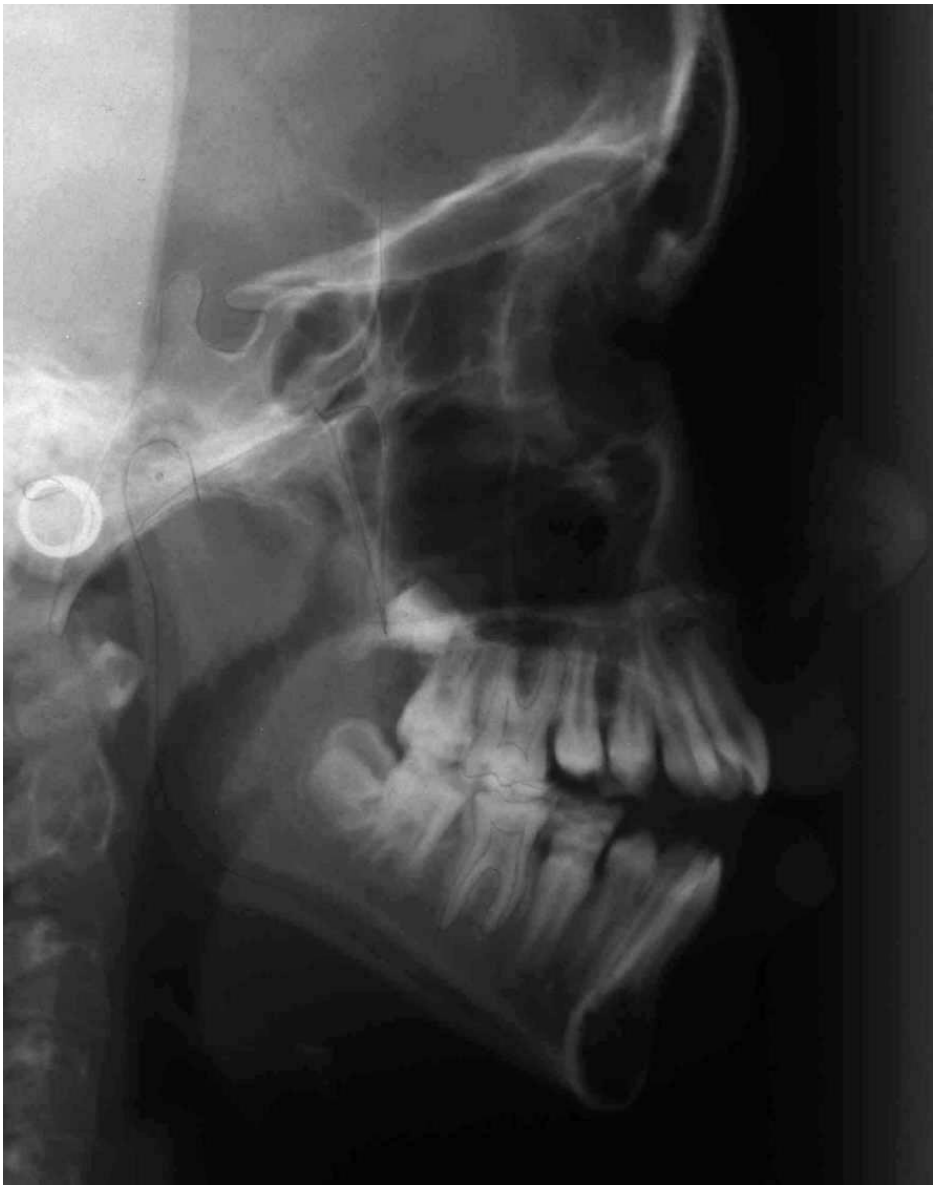
10 years 5mnd



Radiographic Examination	
Dental development	Late mixed dentition
Occlusion	Only occluding on molars
Agenesis	-
Impacted	-
Ectopic	-
Abnormal root morphology	-
Other	17,27,37,47 visible development

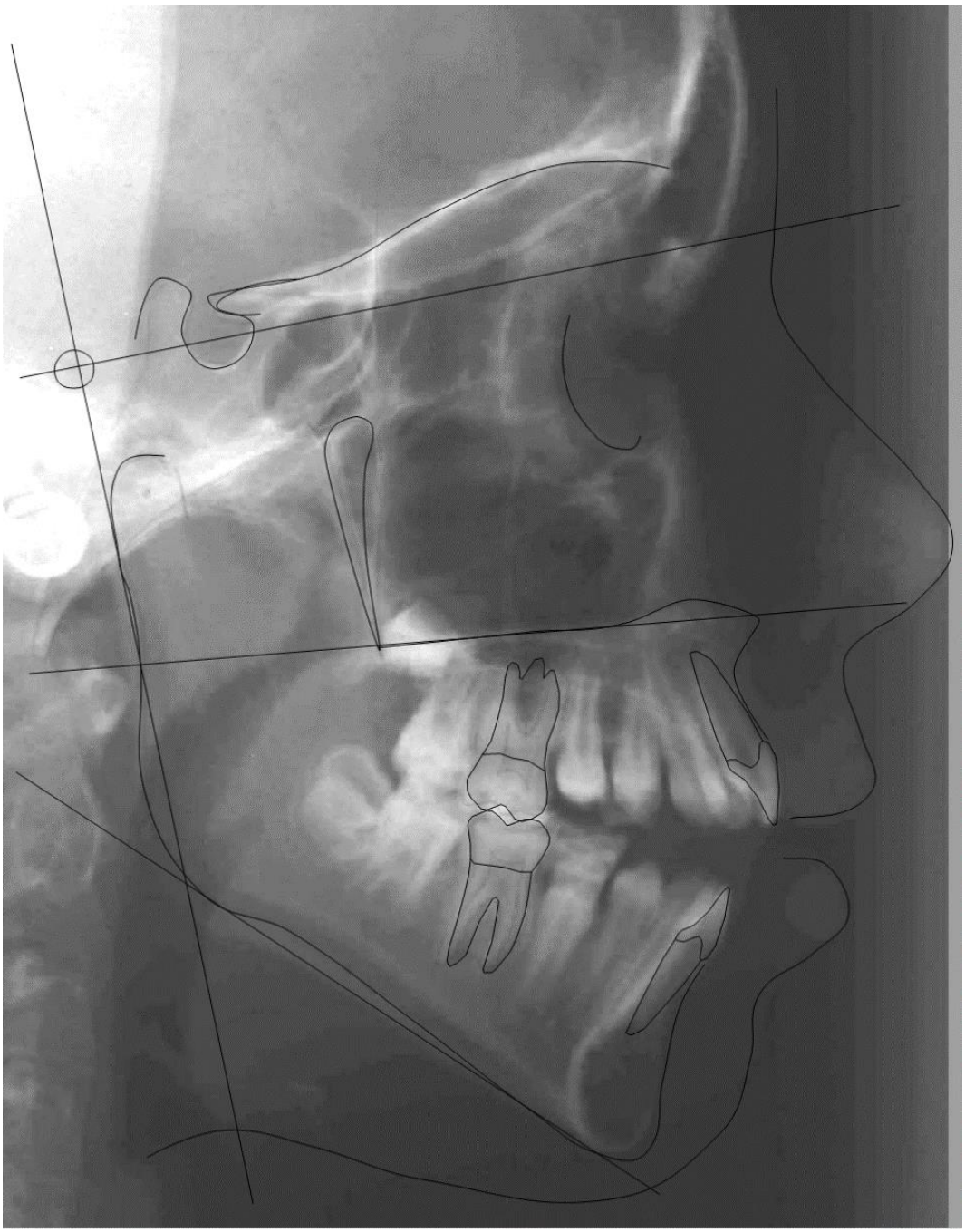
Pre-treatment lateral cephalogram

10 years 5 mnd



Pre-treatment lateral tracing

10 years 5 mnd

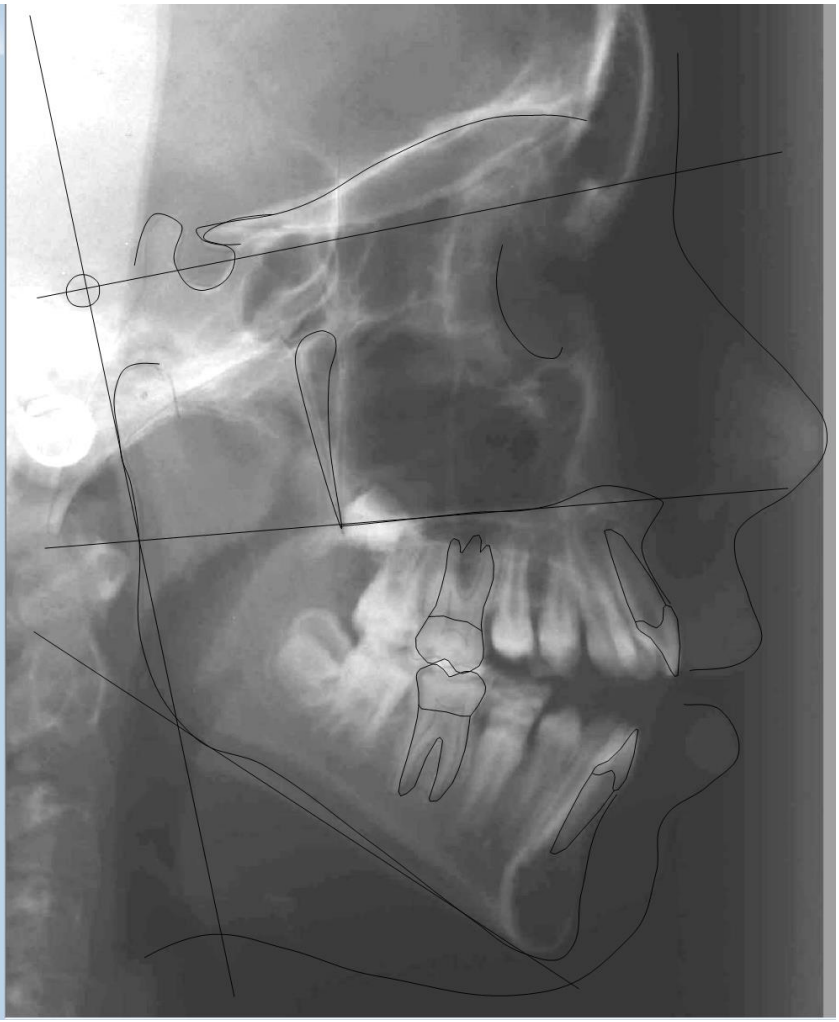


Pre-treatment lateral cephalogram analysis

UIO_OsloSurg-2.0

Kef navn	Orginal	Norm	Enhet	Avv O
NSBa	131.0	130±5	°	
SNA	79.8	82±3	°	
SNB	75.3	79±3	°	-
ANB	4.4	2-3	°	+++
FH/NSL	22.1	5-7	°	+++++°
NL/NSL	6.5	7±3	°	
ML/NSL	44.3	33±4	°	++
ML/NL	37.9	26	°	
FA/NBa	82.7	90±3.5	°	--
A to N-Pg	4.8	2±2	mm	+
UFH	43.3	51±3	mm	--
LFH	66.7	64±4	mm	
U:L Facial Hgh	65.0	80	%	
A-N | FH	10.7	0-1	mm	+++++°
Pg-N | FH	11.6	-4-0	mm	+++++°
Co-A	77.4		mm	
Co-Pgn	102.9		mm	
Max-Mand diff	25.5	25-27	mm	
Ii to A-Pg	3.7	1±2	mm	+
Is to A-Pg	7.8	1±2	mm	+++
ILi/A-Pg	24.0	22±4	°	
ILs/A-Pg	35.0	26±4	°	++
InterIncisal	121.1	131±6	°	-
ILi/ML	90.7	94±4.5	°	
ILs/FH	126.0	107	°	
ILs/NSL	103.9	105±6	°	
PLi-EL	-1.6	0	mm	
PLs-EL	-0.4	-2	mm	
PLi-SL	-1.0	0	mm	
PLs-SL	0.5	0	mm	
GL-RN	56.4	60-66	mm	--
RN-GNs	69.2	60-66	mm	++
RN-LLV	37.4	30-33	mm	+++
LLV-GNs	31.8	30-33	mm	
RN-STs	20.6	22±2	mm	
STi-GNs	43.8	44±4	mm	
STs-STi	4.7	0-3	mm	++
Nasolabial	134.6	102±8	°	++++
GL-RN-PGs	24.5	12±4	°	+++

Navn	Sag [mm]	Ver [mm]	Rot [°]
Audio			
Mandible			
Maxilla			
Orbit			
Orbital roof			
Pterygoid fossa			
Sella turcica			
Inc, low			
Inc, upp			
Molar, low			
Molar, upp			
A			
Ar			
B			
Ba			
Co			
GL			
Gn			
iGo			
Ii			



Pre-treatment lateral cephalogram analysis

	Ceph. name	Normal values	Original	interpretation
DENTAL	Ii to APg	1±2 mm	3.7	Protrusive lower incisors
	Ili/APg	22±4°	24.0	Slightly protruded lower incisors
	ILi/ML	94±4.5°	90.7	90° on mandibular plane
	Is to APg	3.5±2 mm	7.8	Highly protrusive upper incisors
	ILs/APg	26±4°	35.0	Highly proclined upper incisors
	ILs/FH	107°	126.0	Proclined in rel to FH
	Ili/Ils Interincisal	131±6°	121.1	Reduced angle due to maxillary proclination
SKELETAL SAGITTAL	NSBa	130±5°	131.0	
	SNA	82±3°	79.8	Quite normal maxillary prognatism
	SNB	79±3°	75.3	Tendency to retrognathic mandible
	ANB	2-3°	4.4	Balanced sagittal relationship
	A to NPg	2±2 mm	-2.0	Skeletal facial concavity
	Wits	0-4 mm	1.7	Average sagittal relation
	A-N | FH	0-1 mm	2.1	Anterior rotated maxilla to ant crania base
	Pg-N | FH	-4-0 mm	7.9	Prognatic mandibula rel. to anterior cranial base
SKELETAL VERTICAL	FH/NSL	5-7°	22.1	Posterior inclination of the face in rel to anterior cranial base
	NL/NSL	7±3°	6.5	
	ML/NSL	33±4°	44.3	Posterior rotation of the mandible
	ML/NL	26°	37.9	Increased basal vertical jaw relation
	FA/NBa	90±3.5°	82.7	
	n-sp	51±3 mm	43.3	
	sp-gn	64±4 mm	66.7	
	U:L facial height	80%	65%	Reduced ratio due to small UFH in relation to LFH
SOFT TISSUE	PLi-EL	0 mm	-1.6	Retruded lower lip
	PLi-SL	0 mm	-1.0	
	PLs-EL	-2 mm	-0.4	Normal upper lip
	PLs-SL	0 mm	0.5	

Pre-treatment study cast analysis

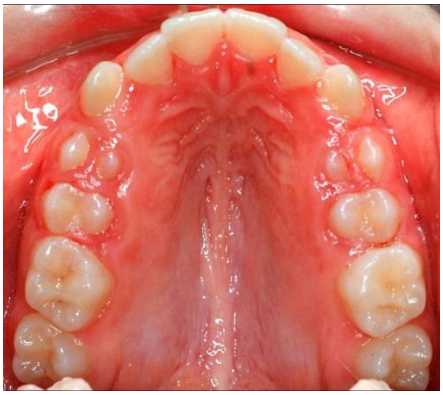
Dental status

		16	15	14	13	12	11	21	22	23	24	25	26		
		46	75	44	43	42	41	31	32	33	34	85	36		

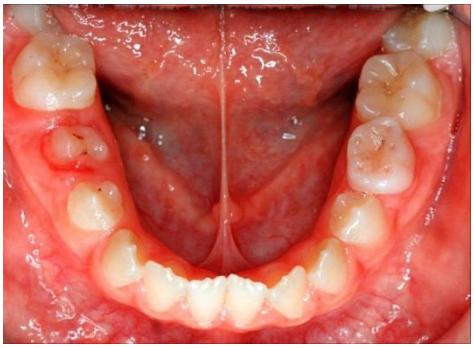
Norma Lateralis	
Occlusal analysis:	Class II
Incisor classification:	
Curve of Spee:	Reversed
Overjet:	5 mm
Overbite:	-7 mm
Canine left:	Cl. II
Canine right:	Cl. II
Molar right:	Cl. II
Molar left:	Cl. II

Space conditions

Maxilla	Right	Ant	Left	Total
Available	14,0	49,0	14,0	77,0
Needed	12,0	50,0	12,0	74,0
Difference	+2,0	-1,0	+2,0	+3,0



Mandible	Right	Ant	Left	Total
Available	18,0	30,0	17,0	65,0
Needed	15,0	34,0	15,0	64,0
Difference	+3,0	-4,0	+2,0	+1,0



Bolton Analysis

Anterior ratio: 0,77. Norm: 0,75-0,80. *No discrepancy*
Over-all ratio: 0,93. Norm: 0,88-0,95. *No discrepancy*

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Diagnosis

Soft tissue profile	Convex profile, incompetent lips
Angle Cl	Cl. II
Overjet	+5 mm
Overbite	-7 mm Large anterior open bite
Space requirements	Upper jaw: -1 mm anterior Lower jaw: -4 mm anterior
Agenesis	-
Anomalies in tooth form and position	-
Midline deviations	-
Transverse discrepancies	-
Incisor position and inclination	Maxilla: Protrusion and proclination Mandibula: Moderate retroclinationclination
Skeletal deviations	Quitr normal maxilla Tendency to retrognathic mandible Posterior rotation of the mandible with increased basal vertical jaw relation
Functional problems	Earlier tumb sucking
Trauma	Earlier trauma to maxillary 51,61
Radiologic anomalies	
Other	Difficulties in biting and chewing
Bolton	No discrepancies

Growth prognosis

The patient is 10 years old at the pre-treatment evaluation. We can expect a lot of continuing growth and development

Prioritized problem list

1. Normalizing the transverse problems
2. Close the anterior open bite as much as possible
3. Improve function by improved biting, chewing and swallowing

Etiologic factors

Frontal open bites are normally related to habits, function and genetics

The cross bite is a common side effect and or contributor to the open bite

The patient and the parents can not say if any of her proximate family has a similar malocclusion. Se has a protruded tongue, which can contribute or is just adapting to the situation..?

The patient has a history of thumb sucking habit up to 6 mnd ago

Treatment Need

Esthetic:	The patient is very shy and reluctant to her open bite with impaired smile esthetics
Prophylactic	Already history of traumatic injury to maxillary temporary incisor
Functional:	Achieve a better ability to bite off food in the anterior part of the dentition and improve chewing With better digestion
HELFO	Gr. B open bite, with 75% refund

Treatment Aims

Achieve neutral occlusion on molars and canines; close anterior open bite and relieve crossbite to improve function and esthetics

Discussion of treatment plan

Treatment options:

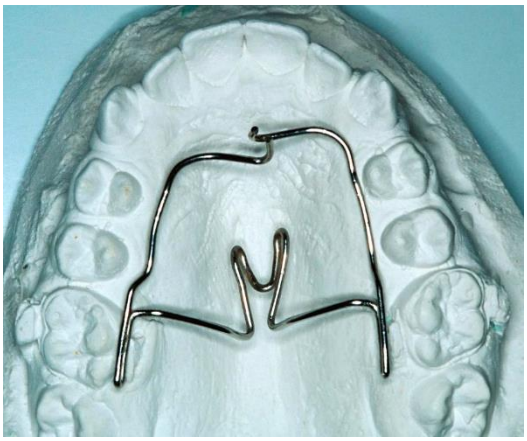
a) HHG and TPB with spurs and tongue training
b) Fixed appliances in both jaws, non extraction treatment
c) Fixed appliance, extraction 4 premolars
d) No treatment

The patient was dissatisfied with her function and her appearance with the large frontal open bite. She also wanted to remove the crossbite and to align the teeth in both jaws. To accommodate to the patients needs it would be necessary to improve her tongue and swallowing dysfunctions first, to make it possible to overcome these forces and to maintain the results for the long term.
From the guiding directive we can see that there is no need for extraction in any of the jaws to relieve the crowding. Extraction in the upper an/or lower jaw could have been an option to help the vertical closing and to reduce the overjet. Extraction treatment was kept as an option during the treatment but is was decided to start with HHG and TPB for intrusion of molars and evaluate the response during the treatment.

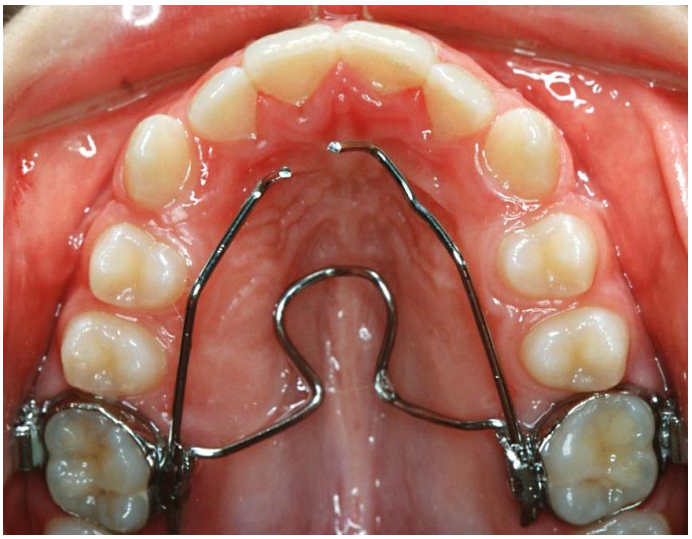
No treatment did not seem like an option since there are clearly an objective and subjective treatment need

Treatment Progress

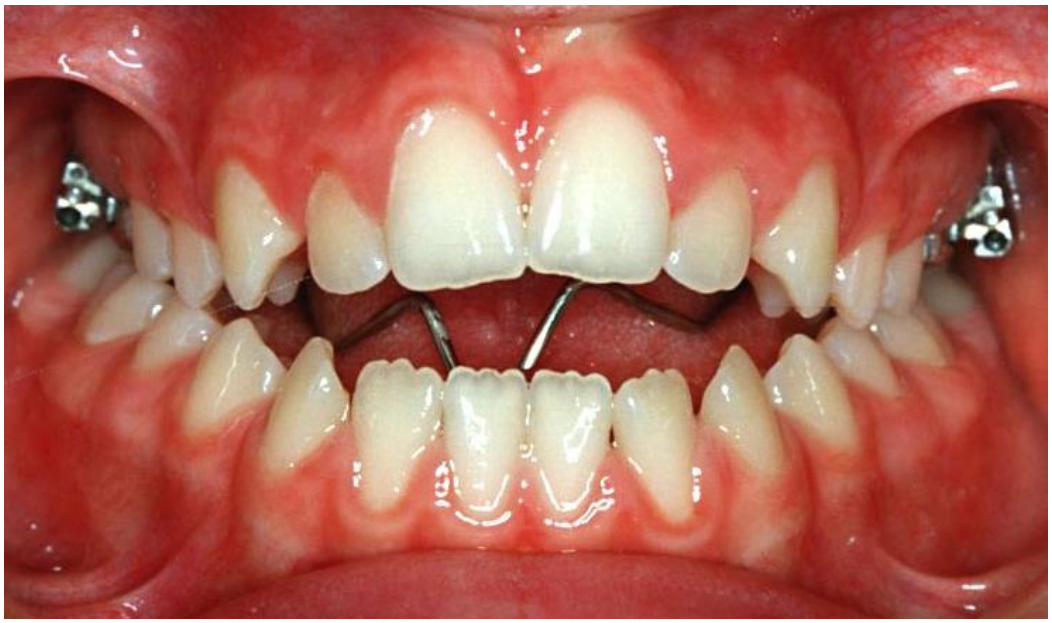
2011	
September	Pre-treatment records
December	Cemented bands 16,26 and adapted standard TPB
2012 January	HHG for night and 5 hours daily
February	Cut and shorten the outer bows for more intrusive effect Impression for adapting TPB with tongue spurs
March	Inserted the new TPB with tongue spurs Information together with the mother
May	Bonded 36-5-4-3-2-1-1-2-3-4-5-46 and .014 Sentalloy Continue HHG at nights and keeping the TPB with spurs
September	.016 x.016 SS in the Mandible
December	Bonded 16-5-4-3-2-1-1-2-3-4-5-26 and .014 Sentalloy Control and adaptation of TPB ang HHG



TPB for expantion and tongue «spurs»



TPB preventing anterior tounge posture



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Treatment Progress

2013	
January	.016 x 022 SS in Maxilla + control of TPB and HHG
March	.017 x 025 SS in Maxilla with intrusion steps for molars Adjustment of the TPB and spurs for better tongue prevention
April	Kobajachi hooks for using vertical elastics in squares both sides Adapting steps to work in the same direction
May	More expansion in maxillary lateral segments with additional torque control and a lot of motivation for the vertical elastics And HHG night and 3 hours daily
June	.017 x 025 SS lower jaw for better protrusion and torque Copy steps and intrusion of molars More motivation and repetition of the tongue placement when swallowing She has a much better and more normal tongue function
August	Rebonded 13 and 23 higher gingivally Repeating motivation with elastics and HHG The vertical and sagittal relations are looking much better Next time impression for retainers
September September September 	Bonded retainer 13-2-1-1-2-23 and debonded upper jaw Bonded composite buttons 13,23 labially Additional 2 mm Hard Stavac in Maxilla and grinded out for the buttons labially Bonded retainers 33-2-1-1-2-43 and debonded lower jaw Bonded composite buttons 33,43 labially Instructed for vertical elastics between buttons to be used every nights Post treatment records and follow up instruction for retention protocol with Stavac and vertical elastics at nughts

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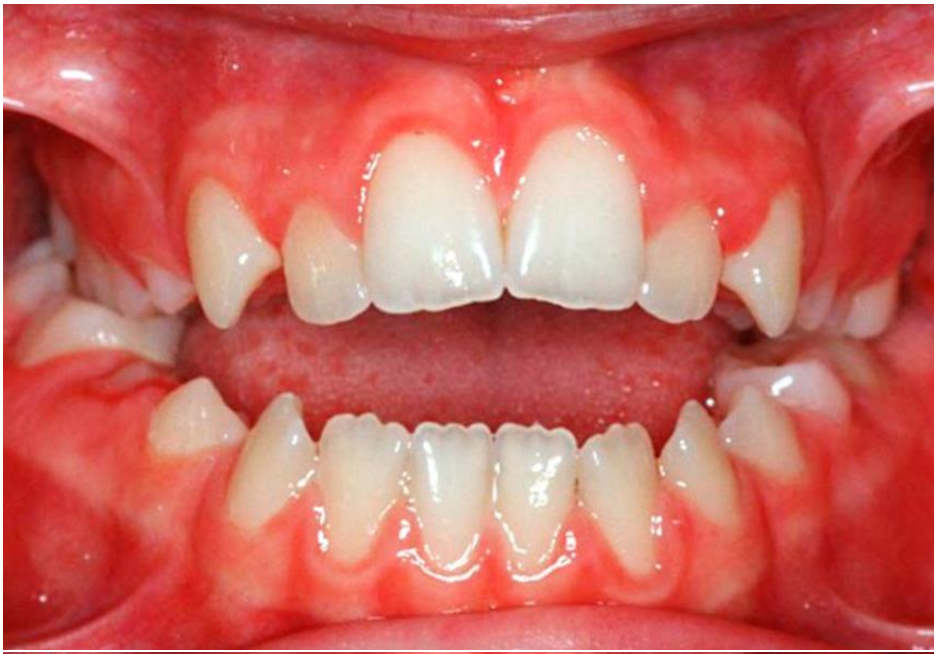
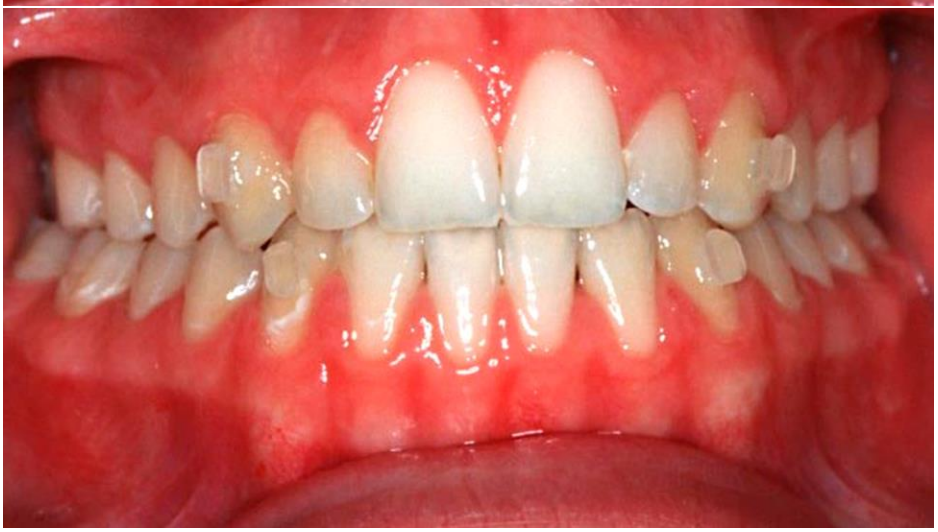
Treatment Progress

2018	
May	Maxillary anterior segment needs more extrusion. By rebonding brackets 12-22 it will increase overbite. U.a.: rebond brackets 13-23 with more gingival position. 016x022 Bioforce. Pc 16-26. L.a.: interproximal reduction 33-43, pc 36-46.
June	All spacings in upper jaw are closed. More vertical adjustment 13-12 is desirable. U.a.: Kobayashi 12 for vertical intermax elastics. Pc 16-26 L.a.: Pc 36-46. Patient is instructed in vertical intermaxillary elastics 13-12-43 to wear at night.
September	Levelled and harmonic arches. Vertical relation is good. U.a.: finishing steps 14 and 24, interprox. Reduction 11 and 21 to get tighter contact point. Pc 11-21 and 16-26. L.a.: labial step 33. Alginate impression for pre-customized retainers. Plan debonding in two weeks.
	Remove fixed appliances in both jaws, 17-27 and 36-46. Remove composite remnants from buccal surfaces and polish enamel. No signs of demineralization in enamel. Patient is unhappy with relation between 11 and 12. This is due to the increased labial-palatal width 11, which has been sliced incisal and slightly extruded after trauma. Explain to the patient that this can not be corrected with orthodontic treatment, but will rather need prosthodontic correction. He is not interested in prosthodontic referral. We agree to increase the labial-palatal width on the mesial part of 12 to level the buccal surface with 11. Patient is happy with this solution. U.a.: Bond pre-customized retainer 0.0215 Penta One to palatal surfaces of 13, 12, 11, 21, 22, 23. Total etch, Transbond Supreme LV. Alginate impression, production and try in of vacuum pressed plate Stavac 0.75 mm. L.a.: Bond pre-customized retainer 0.0215 Penta One to palatal surfaces of 33, 32, 31, 41, 42, 43. Total etch, Transbond Supreme LV. Post-treatment records.

Before



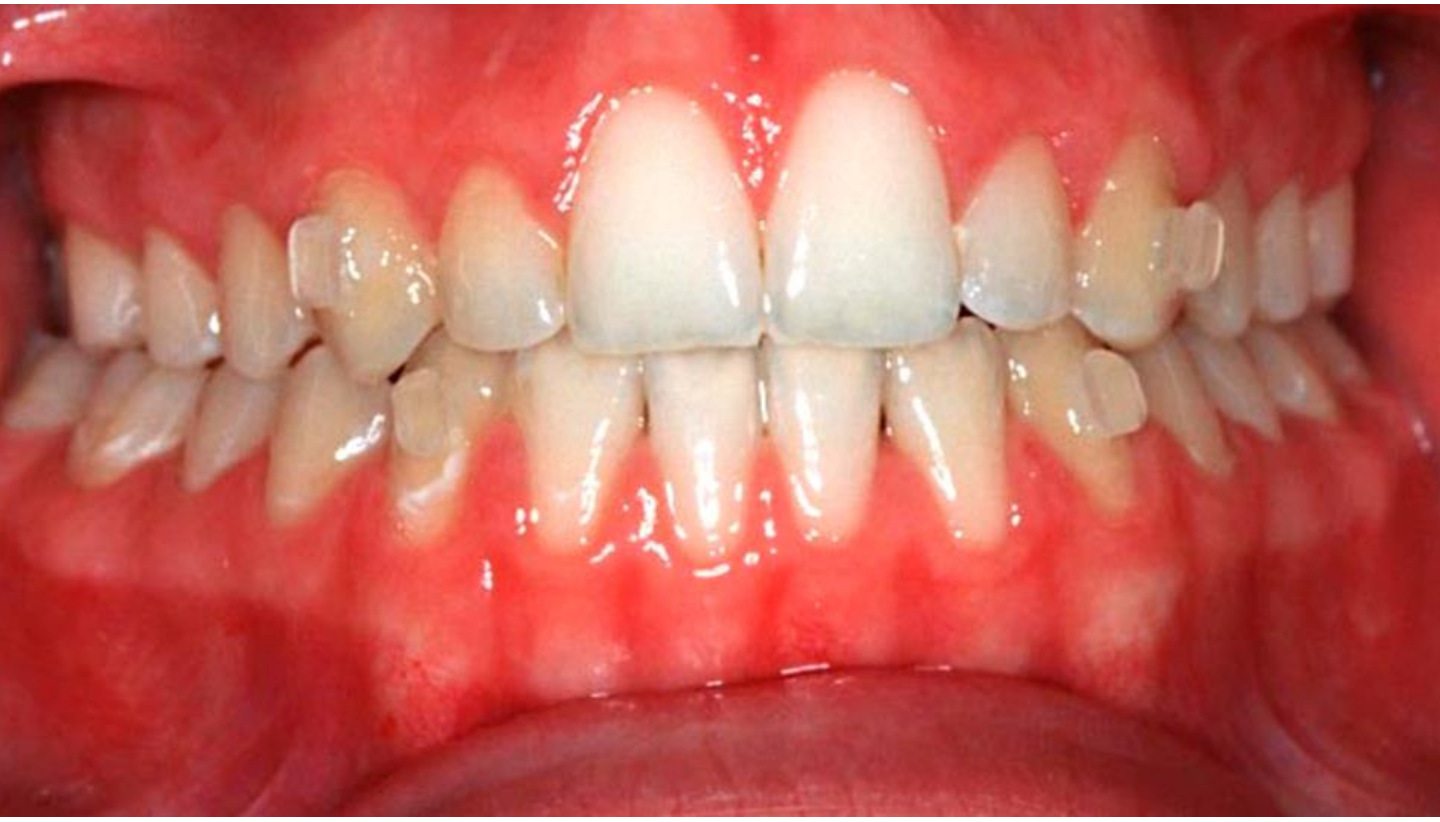
After



Labiale buttons for vertical elastics during nights

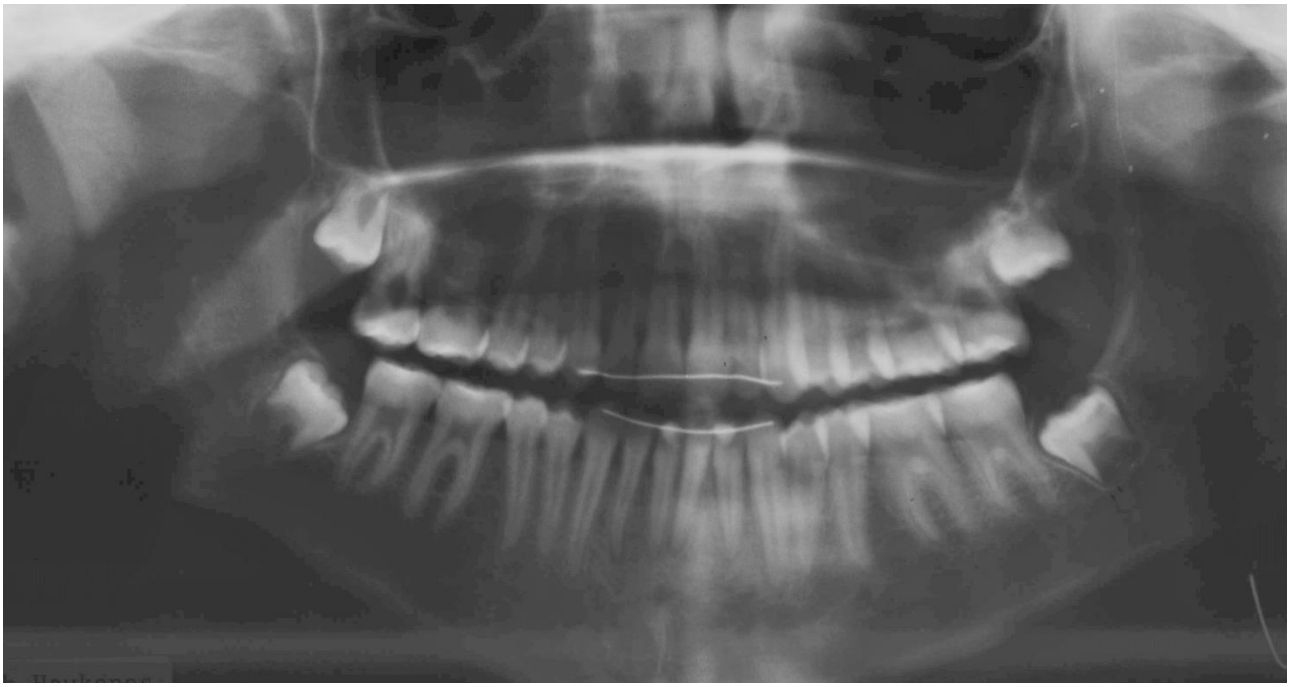
In addition to lingual retainers 13-23,33-43

Gives increased vertical retention and stability



Post-treatment OPG

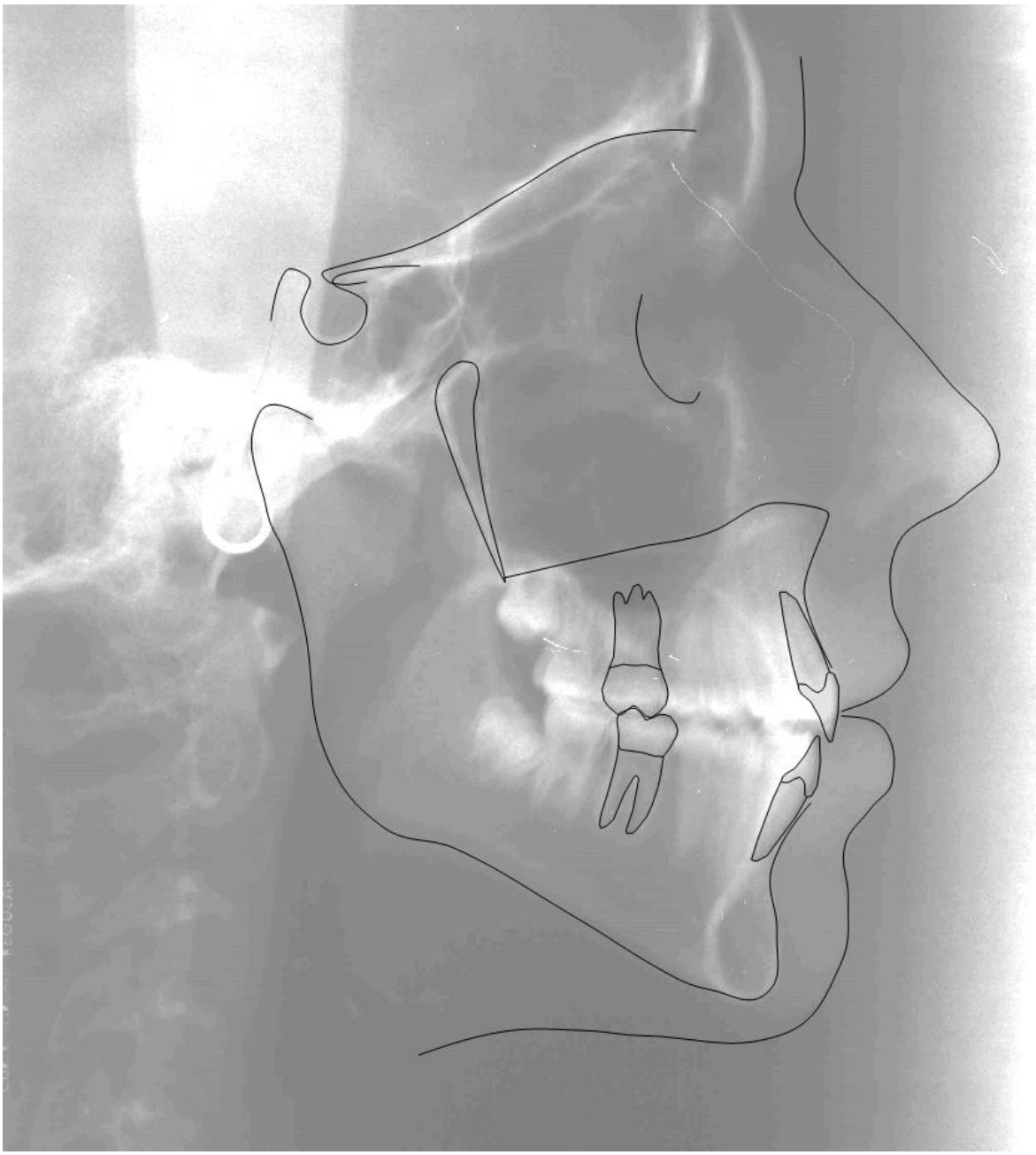
September 2018



Radiographic Examination

Acceptable root angulations. No signs of pathology, no structural changes.
Teeth 18,28,38,48 under development
Bonded retainers 13-2-1-1-2-23 and 33-2-1-1-2-43

Post-treatment lateral cephalogram
September 2018



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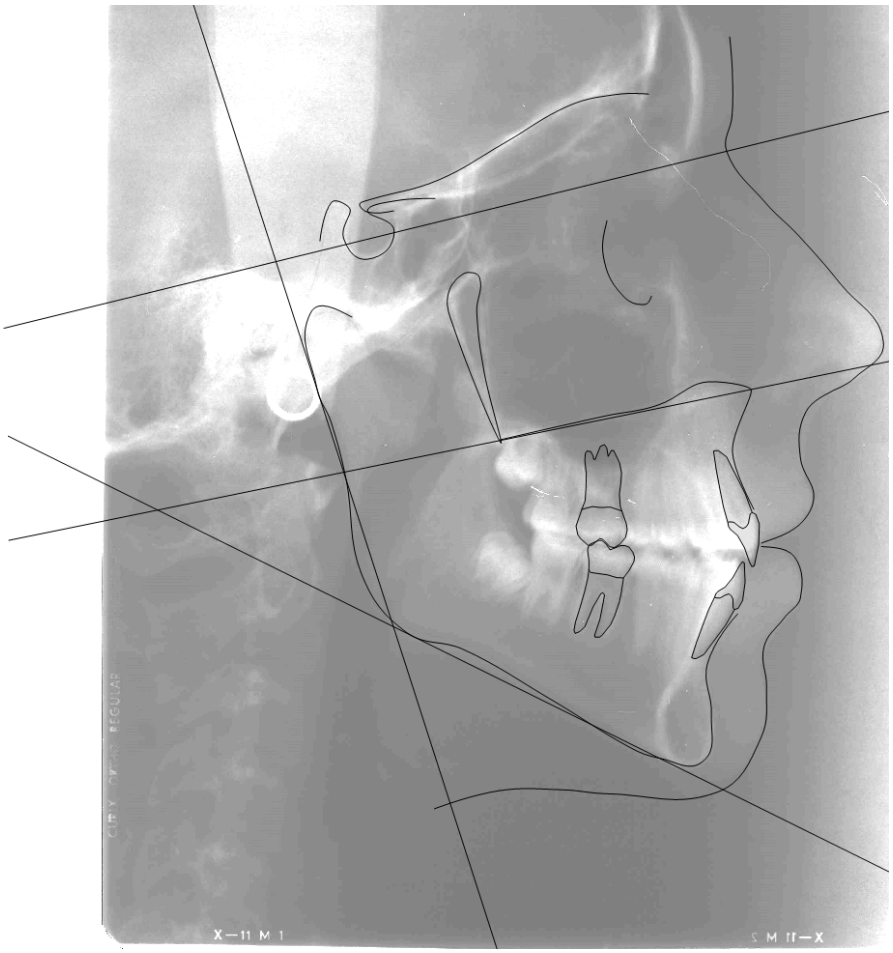
Post-treatment lateral Ceph tracing
September 2018

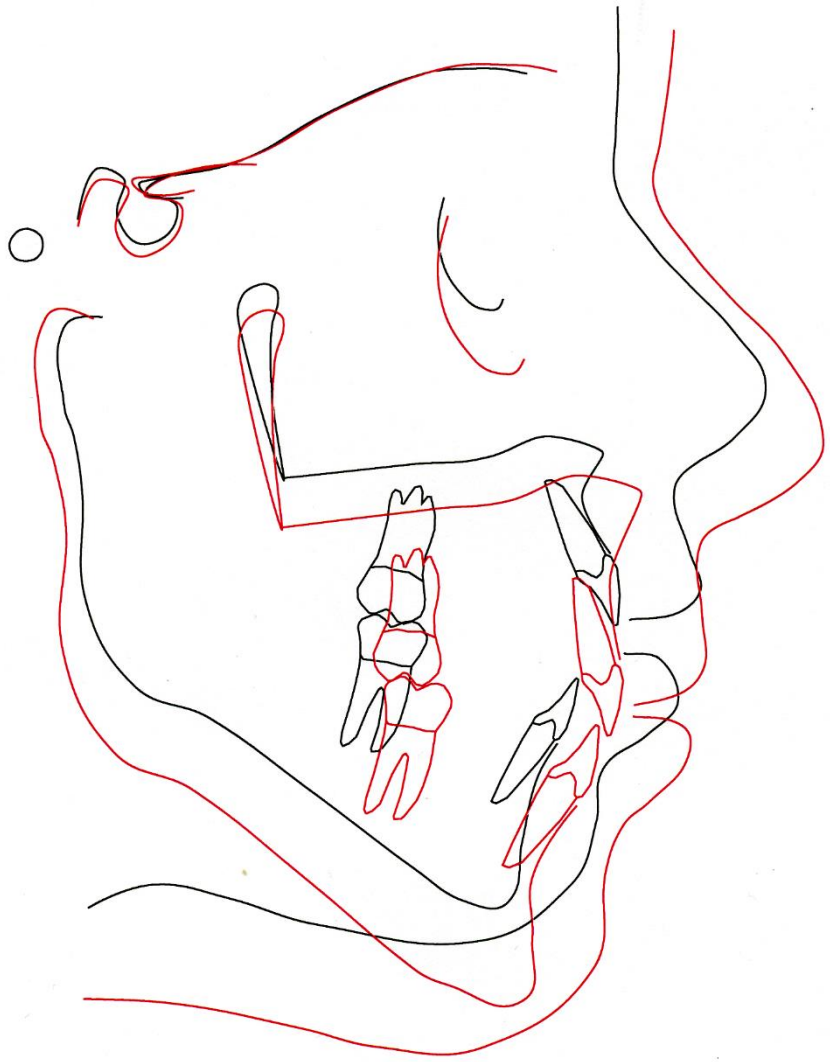
UIO_OsloSurg-2.0

Kef navn	Orginal	Norm	Enhet	Avv O
NSBa	127.3	130±5	°	
SNA	83.4	82±3	°	
SNB	77.5	79±3	°	
ANB	5.8	2-3	°	+++++*
FH/NSL	8.2	5-7	°	++
NL/NSL	2.3	7±3	°	-
ML/NSL	40.1	33±4	°	+
ML/NL	37.8	26	°	
FA/NBa	82.3	90±3.5	°	--
A to N-Pg	5.7	2±2	mm	+
UFH	50.1	51±3	mm	
LFH	76.3	64±4	mm	+++
U:L Facial Hgh	65.7	80	%	
A-N | FH	1.7	0-1	mm	++
Pg-N | FH	-7.8	-4-0	mm	--
Co-A	94.6		mm	
Co-Pgn	127.6		mm	
Max-Mand diff	32.9	25-27	mm	+++++*
Ii to A-Pg	3.5	1±2	mm	+
Is to A-Pg	5.6	1±2	mm	++
ILi/A-Pg	23.6	22±4	°	
ILs/A-Pg	20.9	26±4	°	-
InterIncisal	135.5	131±6	°	
ILi/ML	90.7	94±4.5	°	
ILs/FH	101.9	107	°	
ILs/NSL	93.7	105±6	°	-
PLi-EL	-0.3	0	mm	
PLs-EL	-3.9	-2	mm	
PLi-SL	1.2	0	mm	
PLs-SL	-1.5	0	mm	
GL-RN	74.3	60-66	mm	+++
RN-GNs	77.7	60-66	mm	++++
RN-LLV	51.6	30-33	mm	+++++*
LLV-GNs	26.1	30-33	mm	---
RN-STs	25.7	22±2	mm	+
STi-GNs	51.3	44±4	mm	+
STs-STi	0.7	0-3	mm	
Nasolabial	132.2	102±8	°	+++
GL-RN-PGs	22.4	12±4	°	++

Navn	Sag [mm]	Ver [mm]	Rot [°]
Mandible			
Maxilla			
Orbit			
Orbital roof			
Pterygoid fossa			
Sella turcica			
Inc, low			
Inc, upp			



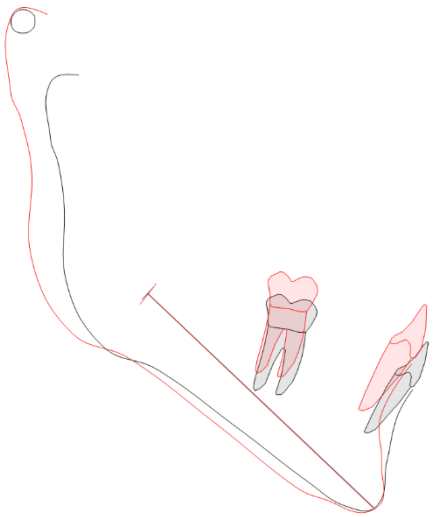
SUPERIMPOSITION:
Overall



Pre-treatment

Post-treatment

SUPERIMPOSITION:
Maxilla and mandible



Pre-treatment

Post-treatment

What did I learn from this case?

From this case I have gained knowledge about orthodontic treatment of anterior open bite I have experienced that treating patient who is genuine interest in their treatment can be very pleasant and stimulating.

It is important to have a proper diagnose before you start with the treatment plan
Generally speaking anterior open bite is a difficult malocclusion to treat

There is a big difference in treating younger patients that are very motivated in reverse to those thar really do not care and do not understand that they have a problem.

It will be exciting to follow this patient for the future to see how stable the treatment result will be…

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