MANAGEMENT OF DEEPBITE AND POSTERIOR OPEN BITE IN CLINICAL PRACTICE

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PREFACE

It was a privilege to be the keynote speaker for the 49th Robert E. Moyers Symposium. Just as we were all hoping that the cloud of COVID-19 was finally lifting, so too I hoped that we could lift some of the clouds surrounding two orthodontic topics that are seldom addressed at our conferences: anterior deep bite and posterior open bite. And just as my keynote lecture was intended to create a framework for the Moyers Symposium lecturers, this preface is intended to present a framework for the chapters that follow. I hope you will enjoy these lecture summaries, and I would like to thank Dr. Park and Dr. Kim-Berman for their excellent assistance as co-editors. It was a pleasure working with both of you.

Anterior deep bite is a common problem, and we are usually successful in managing mild-to-moderate deep bites. However, very severe deep bites are less common and can be a significant challenge to treat, especially when they are accompanied by the loss of posterior teeth.

Some of the questions that I posed to the audience regarding anterior deep bite included:

- What is the prevalence of deep bite for various age categories?
- What are the different “flavors” of deep bite?
- What is the natural history of deep bite from mixed dentition to adolescence to adulthood?
- Do different mechanics result in different mechanisms of deep bite correction in adolescents and adults?
- How can deep bites be prevented/corrected with aligners?
- Can loss of vertical dimension be restored prosthetically?
- How stable is deep bite correction?

The following chapters address many of these questions, and also address esthetic and functional considerations, as well as correction with temporary anchorage devices.

Regarding posterior open bite, I have never been to a meeting where this was the primary topic. This is probably because posterior open bites are so challenging to correct, and we should be very thankful that they are extremely rare. Most of us only treat a handful of them in our entire careers.

For posterior open bites, many questions come to mind:

- What is the etiology of a particular posterior open bite?
- How can posterior open bites be diagnosed and managed?
- How stable is posterior open bite correction?
- How can we prevent posterior open bite and anterior deep bite in aligner patients?

Once again, in the chapters that follow, you will find the answers to some of these difficult questions. In particular, Dr. Frazier-Bowers presents information regarding ankylosis and failure of eruption that is extremely helpful in identifying the etiology of posterior open bites.

Additional chapters touch on many interesting topics, including the role of cilia on bone remodeling, machine learning, 3D printing, mandibular asymmetry, anterior open bite treatment, maxillary expansion, impacted canines, and clefts. As you can see, an impressive panel of experts shared their knowledge with all the attendees.

This annual symposium honors the career and accomplishments of Dr. Robert Moyers, who continues to have a profound influence on the orthodontic profession. On behalf of my co-editors, we thank him for his contributions to Orthodontics. We also give our sincerest thanks to the speakers who participated, and the doctors who attended. Although our numbers may have been down slightly due to COVID-19, it was a great meeting with an unrivaled enthusiasm and passion for learning!

The *49th Annual Moyers Symposium* and the *47th Annual International Conference on Craniofacial Research (Presymposium)* were held at the University of Michigan on Friday, March 4, 2022, through Sunday, March 6, 2022. This meeting was sponsored by the Department of Orthodontics and Pediatric Dentistry, School of Dentistry, University of Michigan. The proceeding of this annual meeting is memorialized in the 58th volume of the Craniofacial Growth Series and contains reports, original research, case series, and review articles from internationally renowned experts, scientists, and clinicians. The 58th volume and the entire Craniofacial Growth Series are made available to the public through the University of Michigan Deep Blue Repository https://deepblue.lib.umich.edu/handle/2027.42/146667.

As in previous years, the *Symposium* honored the late Dr. Robert Moyers, Professor Emeritus of Dentistry and Fellow Emeritus and Founding Director of the Center for Human Growth and Development at the University of Michigan.

We thank Michelle Jones of the Office of Continuing Dental Education for coordinating and managing the *Presymposium* and the *Symposium*. We also thank Dawn Bielawski, PhD for her invaluable work as Copy Editor and Katelyn Lee and Vanessa Mitchell for verifying all citations and references.

We acknowledge Dr. Nan Hatch, the Chair of the Department of Orthodontics and Pediatric Dentistry for her support of the meeting and this publication.

Finally, we thank the speakers and participants of the *Symposium* and the *Presymposium* and appreciate their attendance and support throughout the 49 years of history of the meeting.

Greg Huang, Editor
Jae Hyun Park, Co-Editor
Hera Kim-Berman, Co-Editor and University Liaison
February, 2023

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KEY FACTORS WHEN USING TSADs TO CORRECT DEEP OVERBITE IN CLASS II PATIENTS

Jae Hyun Park

ABSTRACT

Class II patients with a severe, deep overbite present some of the most challenging conditions clinicians face. Appropriate biomechanics such as intrusion of the anterior teeth, extrusion of the posterior teeth, or a combination thereof should be used to avoid negative side effects that have been common with conventional treatment in the past. During diagnosis and treatment planning, the vertical position of the maxillary anterior teeth should be considered for an esthetic incisor display; then, an adequate maxillary occlusal plane angle must be obtained for both esthetic and functional reasons. This paper presents some important considerations such as mandibular growth rotation, sagittal correction of Class II patients, and the use of TSADs to correct deep overbite in Class II patients. Then it examines factors that affect the long-term stability of anterior deep overbite treatment.

KEY WORDS: Class II patients, Deep Overbite, TSADs, Mandibular Growth Rotation, Distalization

INTRODUCTION

Anterior deep overbite is an excessive vertical overlapping of the mandibular anterior teeth by the maxillary anterior teeth. The condition represents one of two categories: skeletal or dental. Skeletal deep overbite can be caused by a clockwise rotation of the maxilla, a counterclockwise rotation of the mandible, or the combination thereof, while dental deep overbite can be the result of infraeruption of the molars or supraeruption of the incisors, or a combination thereof [1]. Either way, correction of the condition is very difficult to maintain after treatment because of the tendency for relapse [2].

Alhammadi et al. reported that the prevalence of deep overbite decreases from mixed to permanent dentition because of both occlusal stabilization involving full eruption of premolars and second molars, and a more pronounced mandibular growth [3]. They also found a reduction in the instance of Class II as well as an increase in Class III during the transitional dentition.

The etiologies of anterior deep overbite include tooth morphology (length of the anterior crowns), skeletal pattern (frequently found with Class II malocclusion), condylar growth pattern (counterclockwise growth of the condyle), muscular differences (short-faced individuals generally exhibit increased masticatory muscle mass and thus have greater molar bite force), and lateral tongue thrust habits (habits will cause an infraocclusion of posterior teeth which leads to a deep bite) [1, 4, 5].

When deep overbite is diagnosed during an extraoral exam, it is common for the patients to exhibit short, square faces, short upper lips, and prominent chins with a deep mentolabial fold. Also, they often have a toothless appearance (due to their decreased lower anterior facial height), and their maxillary incisors may be hidden behind their upper lips when they speak or smile, especially in the case of Class II Division 2 [1, 4, 6].

During an intraoral examination, deep overbite patients are usually found to have anterior crowding, constricted mandibular intercanine widths, widened transverse maxillae, supraeruption of mandibular incisors, and excessive curve of Spee in their mandibular arches. Radiographic examinations typically show a decreased lower anterior facial height, reduced gonial angle, low mandibular plane angle, prominent pogonion, and increased posterior ramus height [1, 4, 7].

Treatment options depend on the patient's stage of development. Since dentoskeletal responses to bite opening efforts will be minimal-to-none with adults, it is necessary to determine whether it would be better to correct their deep overbite with dentoalveolar compensation or orthognathic surgery [8].

Because anterior deep overbite often occurs in Class II patients and is related to their growth pattern, we will examine the basic considerations in Class II treatment using temporary skeletal anchorage devices (TSADs).

PRIMARY INDICATORS FOR MANDIBULAR GROWTH ROTATION IN CLASS II PATIENTS

Facial growth and development are important considerations in orthodontic treatment planning. Many investigators have concluded that mandibular morphology can be used to predict the direction of facial growth, while some have identified the ratio of posterior facial height to anterior facial height as a key indicator [9-11]. This ratio can be determined by comparing mandibular inclination to facial height (the Jarabak quotient) [11].

Skieller and Bjork conducted one of the most widely recognized classic studies on the key parameters determining the direction and degree of mandibular growth rotation [9]. They analyzed 44 morphologic variables with a multivariate statistical method to identify the variables that showed the highest predictive value on mandibular rotation (alone or in combination). They identified four relevant parameters: mandibular inclination, intermolar angle, the shape of the lower border of the mandible, and inclination of the symphysis (Figure 1) [9, 10]. When these parameters were combined, the prognostic estimate was at its highest (86%). Of these four parameters, mandibular inclination was the most important in predicting the direction and amount of mandibular rotation. In their report, the mandibular inclination was represented by Index I. The ratio between the posterior and anterior facial height ranged from 57% to 70%. Index I was the most significant indicator of rotation because it explained 62% of mandibular growth rotation when used alone and as much as 86% when combined with the three other values [9].

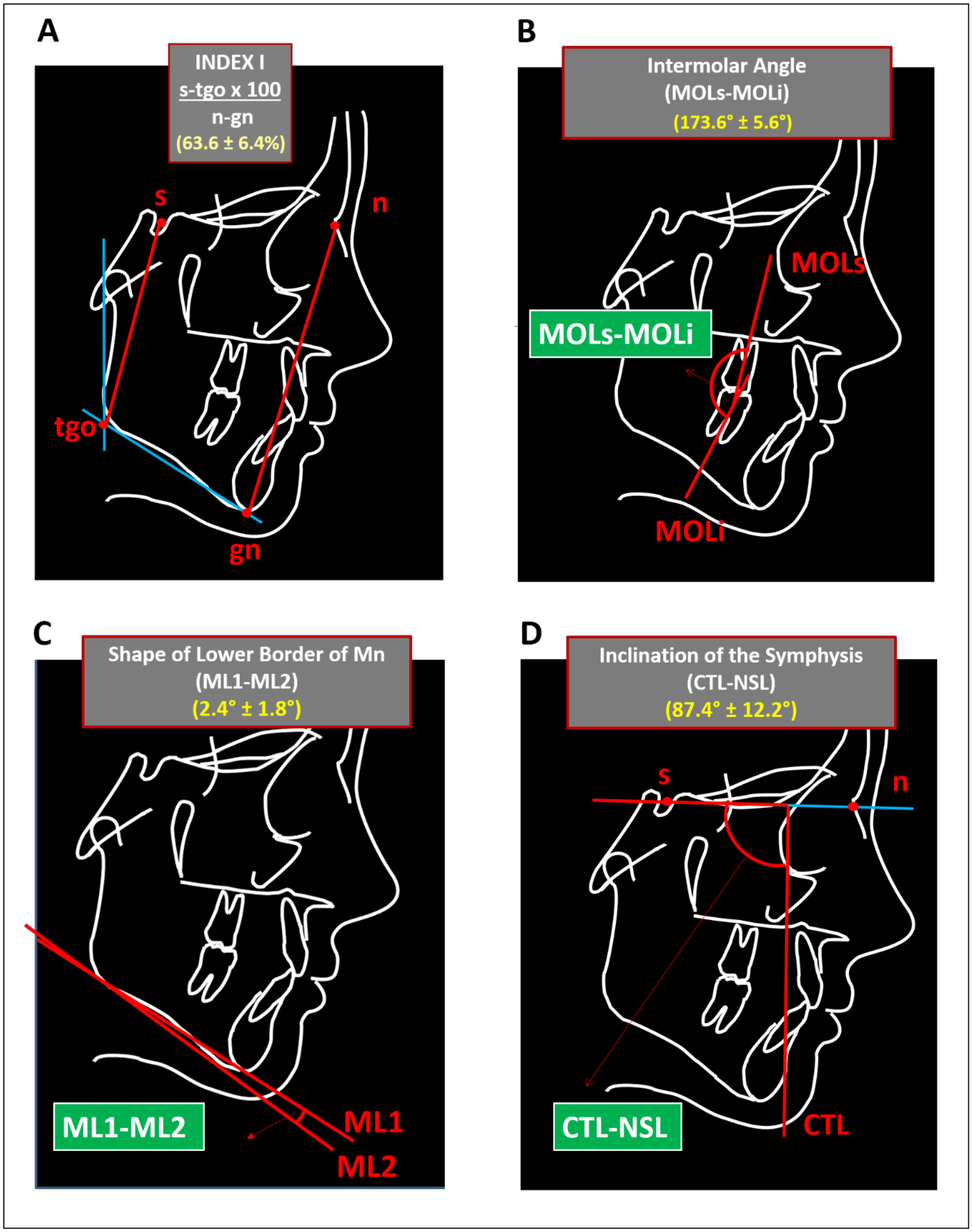


Figure 1. A) Mandibular inclination: Index I, B) intermolar angle, C) shape of the lower border of the mandible, D) inclination of the symphysis.

Siriwat and Jarabak categorized facial morphology based on three distinct patterns as defined by the facial height ratio, or the Jarabak Quotient [11]. They reported these patterns were commonly associated with rotational growth changes that tend to accentuate the pattern characteristics with growth, so these static evaluations were used in terms of growth, as shown in Figure 2 [10]. When the ratio is between 59% and 63%, the mandible can be expected to follow a neutral growth pattern of downward and forward movement, which is the most prevalent. If the ratio is less than 59%, mandibular growth may be hyperdivergent, while greater than 63% may be hypodivergent. Thus, both the mandibular inclination as represented by Index I and the facial height ratio (Jarabak quotient) are expressions of the ratio of posterior facial height to anterior facial height, which is the most important parameter for predicting the direction of rotation in orthodontic treatment planning. As with Index I, this parameter is also the ratio of posterior facial height to anterior facial height and is measured by the equation (S-Go/N-Me)/100.

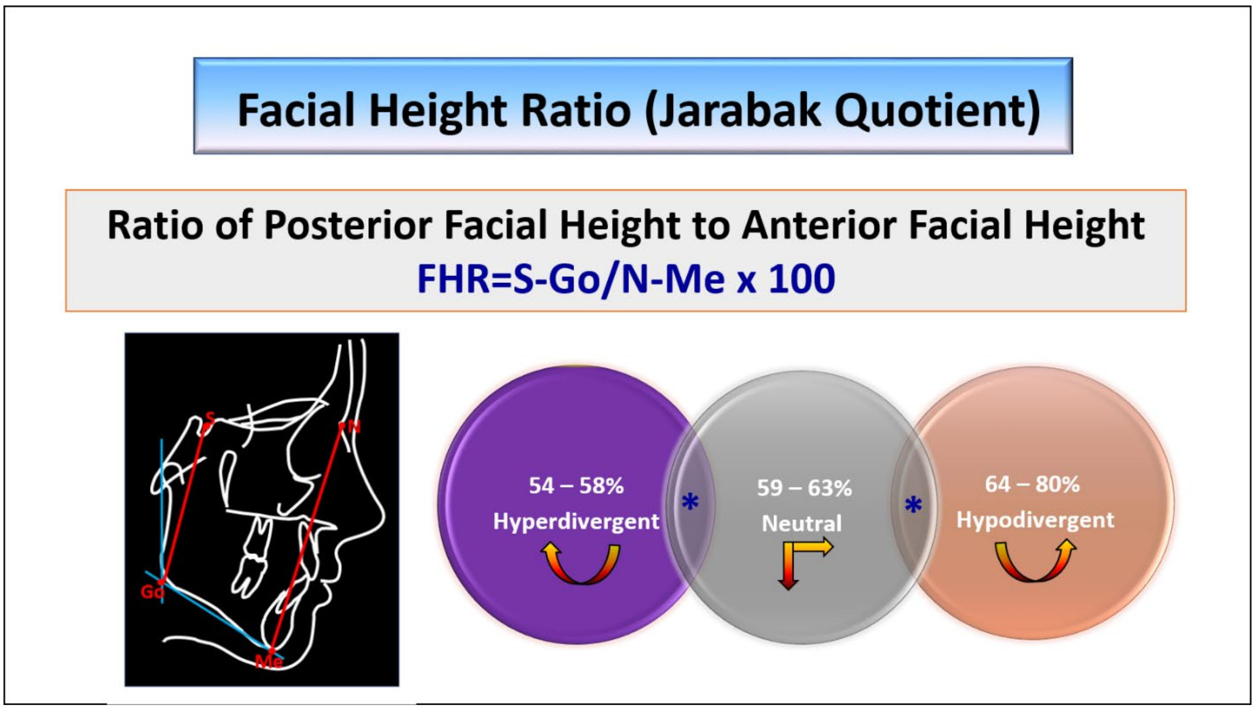


Figure 2. Facial height ratio (Jarabak quotient).

In my book, *Temporary Anchorage Devices in Clinical Orthodontics*, Buschang and Tadlock [12] listed seven indicators of growth patterns in skeletal Class II malocclusion that can be used to predict whether patients with skeletal Class II malocclusions will have favorable or unfavorable growth patterns (Figure 3), with one of these being the ratio of posterior to anterior facial height [10].

Factors	Favorable	Unfavorable
Mandibular plane angulation	Small	Large
Gonial angulation	Small	Large
Post./Ant. facial height ratio	Large	Small
Antegonial notching	Little/none	Increased
Condylar growth direction	Anterior/slightly posterior	Posterior
Symphyseal morphology	Short and broad w/chin	Long and narrow
Condylar summit to FH	Below	Above

Figure 3. Seven determining factors of favorable or unfavorable growth patterns.

SIDE EFFECTS OF CONVENTIONAL CLASS II CORRECTORS

Since the beginning of contemporary orthodontics, various removable and fixed appliances have been used to treat Class II patients. When the goal was to treat maxillary dentoalveolar protrusion, molar distalizing appliances were used, but mandibular enhancing appliances were adopted when the goal was to correct mandibular skeletal retrusion.

As early as the 1950s removable appliances such as headgear were being used to hold or distalize maxillary molars, while various removable appliances such as Bionator and Twin Block have been used in mandibular skeletal retrusion cases. Unfortunately, these removable appliances were often not very effective due to a lack of patient compliance. To overcome this shortcoming, various fixed appliances were developed that did not require compliance. Over the years, many fixed maxillary molar distalizing appliances have been used, including pendulum appliances, Jones jigs, and distal jets. Also, there have been several fixed mandibular enhancing appliances such as Forsus, Herbst, and MARA.

While these appliances successfully corrected overjet and molar occlusal relationships, they often caused anterior anchorage loss, leading to incisor proclination and increased overjet, forward movement of distalized molars during the anterior retraction phase, and distal tipping and extrusion of molars (Figure 4) [10]. Consequently, there was an increase in the mandibular plane angle and a corresponding increase in the lower anterior facial height. Also, because orthodontists were widely using Class II elastics for dentoalveolar correction of Class II patients, lingual tipping and extrusion of the maxillary incisors, labial tipping and intrusion of the mandibular incisors, and mesialization and extrusion of the mandibular molars occurred [13], similar to the negative effects associated with the other fixed maxillary distalizing appliances. In terms of the long-term effectiveness, both the maxillary molar distalizing fixed appliances and Class II elastics are similar to all other functional appliances.

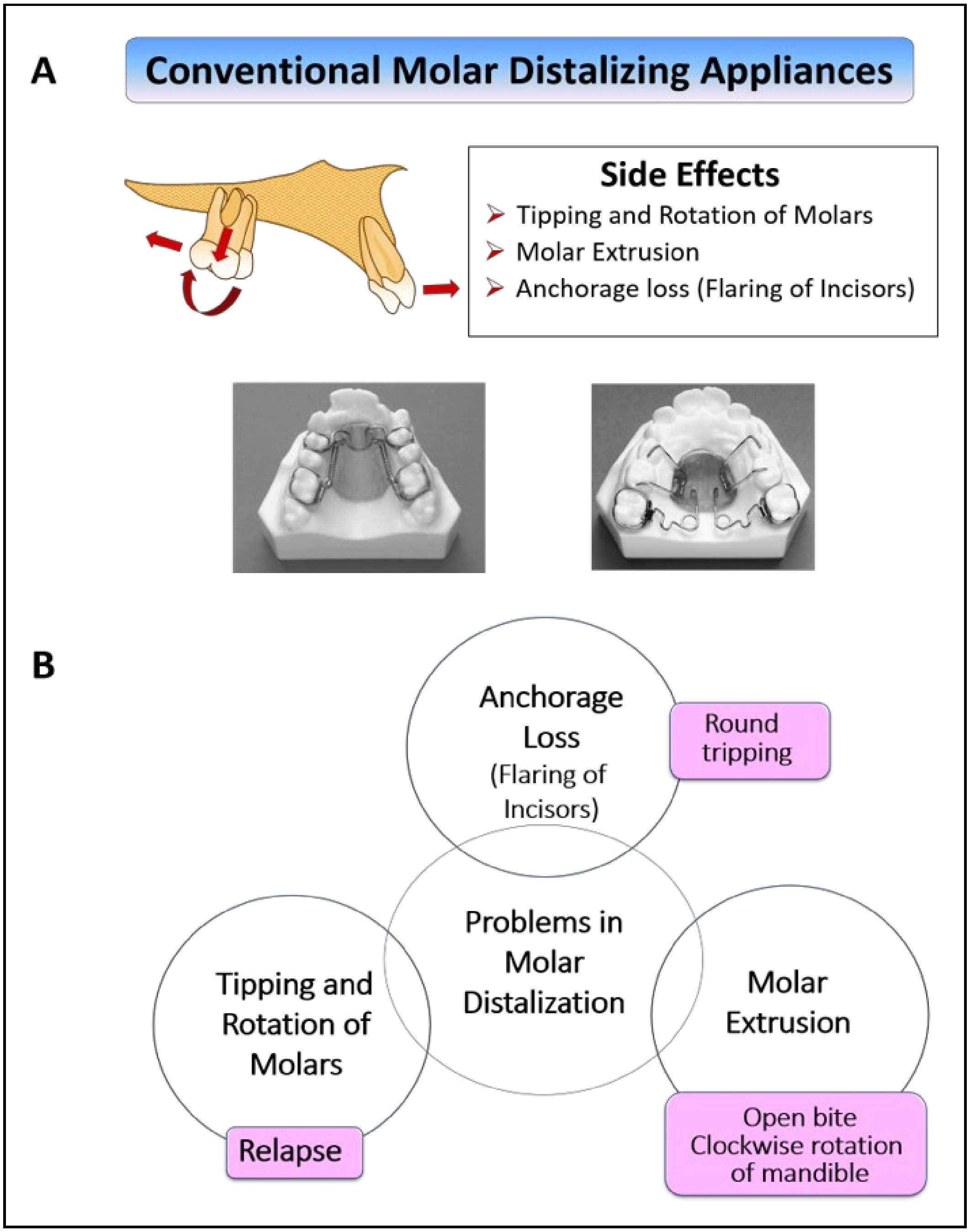


Figure 4. A) Side effects of conventional molar distalizing appliances, B) side effects of conventional molar distalizing appliances.

USING TSADs TO TREAT CLASS II PATIENTS

An effective way to overcome some of the negative effects mentioned is to use TSADs or miniscrews in the palate combined with molar distalizers [14]. The TSADs create a stable anchorage which means there is no proclination of the incisors and no posterior anchorage loss during the retraction phase when the same TSADs are reused as absolute retraction anchorage.

Unfortunately, TSADs in the palate still do not resolve distal tipping and extrusion of the maxillary molars, which causes an unfavorable clockwise rotation of the mandible. For this reason, some clinicians have started placing TSADs in different anatomical sites in the maxilla in combination with traditional types of fixed molar distalizers or simultaneously with only fixed appliances for retraction of the posterior segments or entire arches in nonextraction Class II patients. They have achieved intrusive retraction of the molars with TSADs in the buccal interradicular bone [15], zygomatic buttresses [16, 17], thereby eliminating the extrusive side effects on the maxillary molars.

This process only works when the force vector is carefully aligned with the center of resistance (CR) and proper biomechanical principles are applied, but even then, there are still some limitations with these techniques. For instance, the buccal interradicular approach can potentially cause root perforations because of the limited space between the roots (only 3 mm of distalization is possible without distal repositioning of the TSADs) [15]. Additionally, the stability of the TSADs is likely to be compromised when the cortex is thin due to the low density of the trabecular bone in the maxillary buccal area.

The zygomatic buttress or zygomatic crest approach allows for molars to be distalized beyond 3 mm without repositioning the TSADs, but there is still a risk of sinus perforations, even more so than with the buccal interradicular approach, and it involves surgical procedures. Also, the zygomatic approach makes TSADs more prone to cause soft-tissue irritation, so cone-beam computed tomography imaging is necessary to place them properly. In most cases, there is still a chance for molar tipping and extrusion of the maxillary molars.

USING MODIFIED C-PALATIAL PLATES FOR NONEXTRACTION TREATMENT OF CLASS II PATIENTS

Dr. Yoon-Ah Kook and our research team introduced a new palatal anchorage device called the modified palatal anchorage plate (MPAP) in 2014. It is an alternative to the previously reported types of palatal anchorage molar distalization appliances [18]. It has been found to effectively distalize the posterior teeth in adults and adolescents alike. A finite element analysis suggested that distalization with palatal plates provides bodily molar movement without tipping or extrusion and a simultaneous orthopedic effect similar to headgear. Dr. Kook and I collaborated to publish a case study on modified C-palatal plates (MCPPs) that showed better results compared to those of the original MPAP (Figure 5) [10, 19].

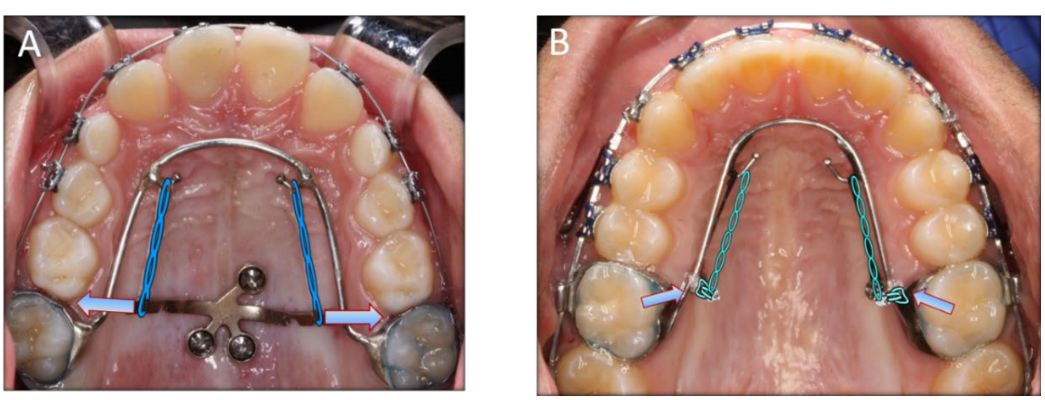


Figure 5. A) Total arch distalization with a modified C-palatal plate. B) If the palatal plate fails, it still can work for distalization by adding two palatal slope TADs. It also can control the vertical dimension by intruding the maxillary molars.

With MCPPs, it is now possible to effectively retract the maxillary dentoalveolar complex while efficiently directing the vertical force vector (Figure 6)[10, 20] to pass through the CR of the maxilla. In this way, it is possible to achieve translational tooth movement parallel to the occlusal plane while distalizing the maxillary dentition, so orthodontists can now actively prevent molar tipping and extrusion that used to cause unfavorable clockwise rotation of the mandible with other types of maxillary molar distalization strategies in the past [21, 22]. The application of MCPPs has produced a significant amount of total arch distalization without undesirable effects on the transverse dimensions or changes to the oropharynx airway space [23].

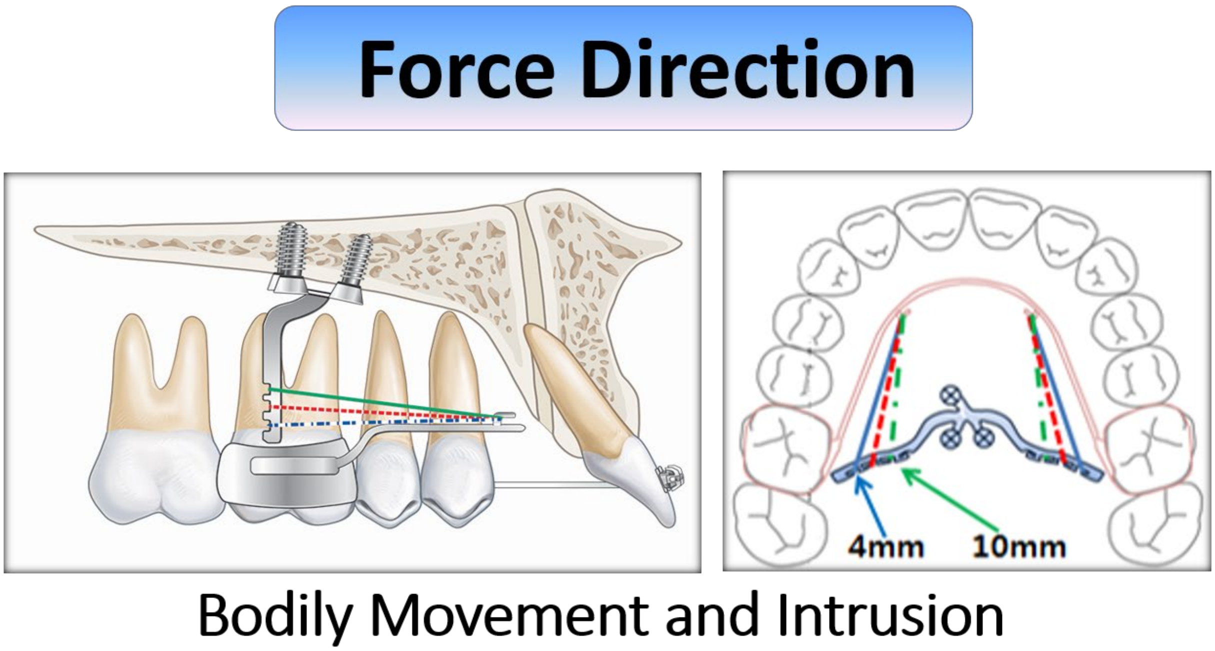


Figure 6. MCPP creates three different force systems; by applying the forces to the 10-mm notches, these forces pass through the center of resistance of the maxilla, allowing bodily movement of teeth during intrusive retraction.

From our study on the retention phase that was recently published in the *American Journal of Orthodontics & Dentofacial Orthopedics* [24], patients treated with palatal plates showed minimal dental, skeletal, and soft-tissue changes at 3 years posttreatment, resulting in just a 12% relapse of distalization and 35% of intrusion from the initial changes achieved on the maxillary first molar. Thus, palatal plates are a viable treatment option for maxillary total arch distalization (Figure 7) [10, 24].

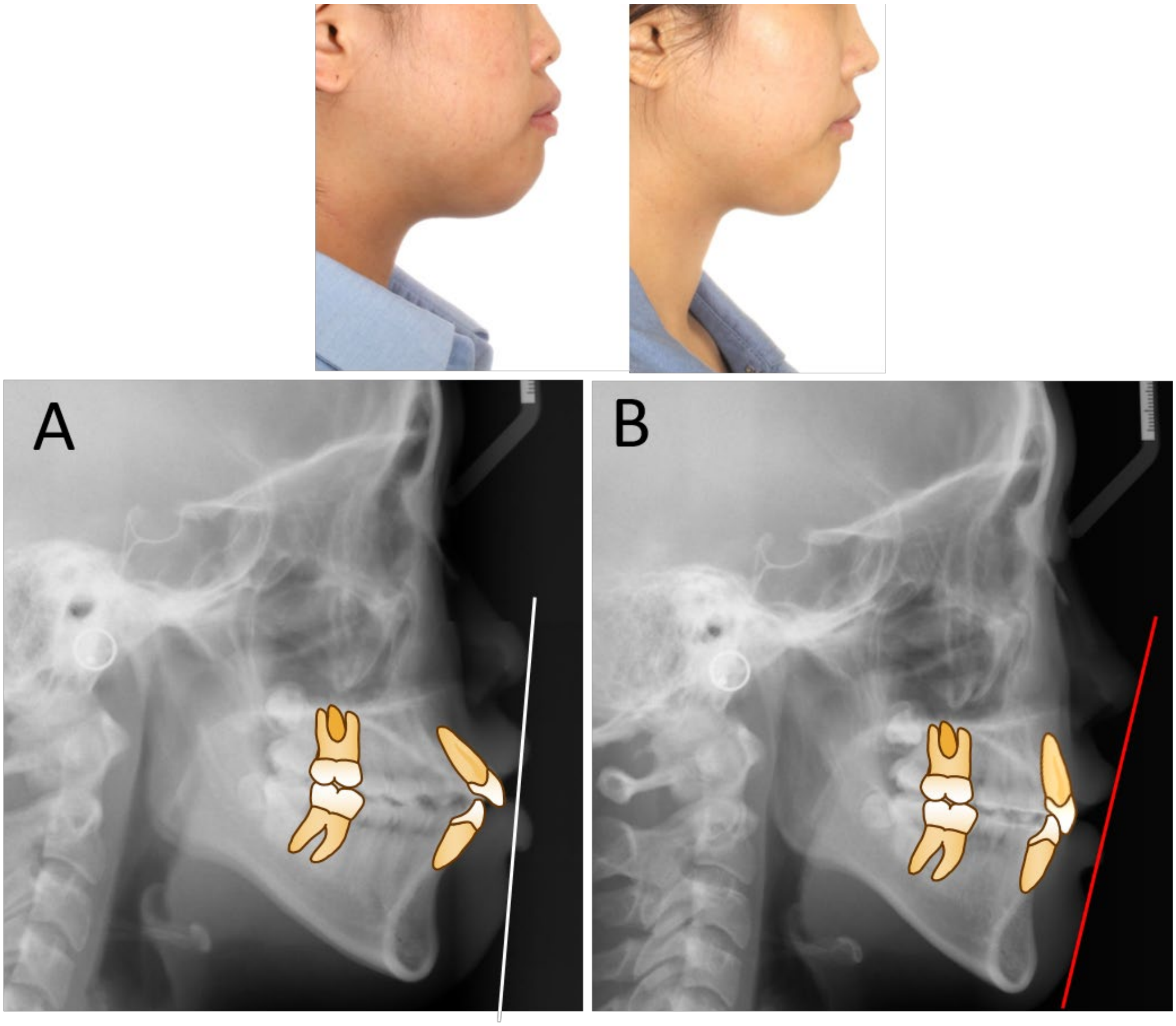


Figure 7. Total arch distalization using an MCPP.

In adolescent patients, the amount of distalization of the maxillary first molars was 20% greater when the second molar was unerupted than after the second molar had erupted [25]. However, the presence of a third molar follicle did not affect the distalization. If the maxillary second molar should tip back due to distal movement of the maxillary first molar, a marginal ridge discrepancy could be created. A second molar mesial marginal ridge elevation could be a clinical sign of tipping. To prevent such distal tipping during distalization, the second molars should be included in the distalization, and sufficiently stiff wires should be used [26].

With traditional types of molar distalizers, third molars can be impacted when there is distal tipping of the second molars, and their presence can also impede the distal movement of the first and second molars. For this reason, the third molars are usually extracted whenever possible prior to molar distalization treatment. In a recent study, we concluded that maxillary total arch distalization caused the unerupted third molars to move backward and upward [25]. In contrast, they moved downward and forward in the control group, but the difference in the posttreatment volume of maxillary tuberosity was insignificant. We also found that unerupted third molars tend to move buccally, allowing easier access for future extraction. Considering the favorable eruption of the maxillary third molars in the long term, these findings suggest that their extraction before molar distalization in Class II adolescents could be optional [27].

CONSIDERATIONS OF GROWTH WHEN TREATING CLASS II DIVISION 2 PATIENTS WITH TSADs

Treating adult patients with a severe deep overbite is clearly one of the most challenging orthodontic treatments, even though its prevalence is relatively low compared to other malocclusions. The stage of a patient's development is a significant factor when considering treatment options for Class II Division 2 patients. For some growing patients, extrusion of the posterior teeth can be an option to increase the vertical dimension and to correct the deep overbite of their anterior teeth. Still, it is not indicated for patients with normal incisor display or increased lower anterior facial height.

Fortunately, many studies have reported the successful use of TSADs as absolute anchorage to intrude the maxillary incisors without extrusion of the maxillary molars or an undesirable clockwise rotation of the mandible [28-34]. Treatment of deep overbite needs to include intrusion of the anterior teeth, extrusion of the posterior teeth, or a combination thereof [35-38]. Extrusion of the posterior teeth can be effective for some growing patients since it results in increased lower anterior facial height due to a clockwise rotation of the mandible [39]. However, clinicians recognize that deep overbite correction by extrusion of posterior teeth is difficult to accomplish and is less stable when performed on adult patients [40, 41].

Numerous factors such as the patient's smile line, incisor display, occlusal plane angle, vertical dimension, lip competence, and stability can affect the choice of treatment mechanics [42-44]. Proper vertical correction is critical when treating these patients, and various treatment techniques and biomechanical approaches have been suggested. For instance, intrusion arches have been widely used to correct deep overbites in adults [35-37], but these mechanics frequently cause labial tipping of the anterior teeth and extrusion of the anchorage posterior teeth. Moreover, the later distal movement of labially tipped anterior teeth might also cause uncontrolled root resorption [45]. Furthermore, this technique does not guarantee stability, especially in adult patients and those with average-to-low mandibular plane angles [35-37]. Therefore, the absolute intrusion of the anterior teeth is recommended in some deep overbite cases [40, 46].

When establishing treatment plans for patients with Class II Division 2 malocclusions, the vertical and sagittal position of the maxillary incisors should be determined first, and the treatment goals should be set accordingly. The inclination of the maxillary occlusal plane also significantly influences masticatory function and facial and smile esthetics [44]. If a patient has a flat maxillary occlusal plane, for example, they will show a reverse smile arc, which is likely to have a negative effect on their facial esthetics [42]. Therefore, to achieve proper function and esthetics through orthodontic treatment, it is important to determine the vertical position of the maxillary anterior and posterior teeth correctly, and this should be decided strategically according to the visual treatment objective (VTO) [43, 47].

The first step of the VTO is to determine the anteroposterior and vertical positions of the maxillary incisors. The anteroposterior position of the maxillary incisor is determined according to the lip prominence, whereas the vertical position is set where the maxillary incisor tip is exposed by 3 mm below the superior stomion in the resting position. Then, the vertical position of the maxillary posterior teeth should be positioned such that the inclination of the maxillary occlusal plane to the true vertical line is about 100°

to optimize the function and esthetics. The vertical position of the posterior teeth will be determined in consideration of changes in overjet, overbite, and the anteroposterior position of the chin point (Figure 8) [38, 48].

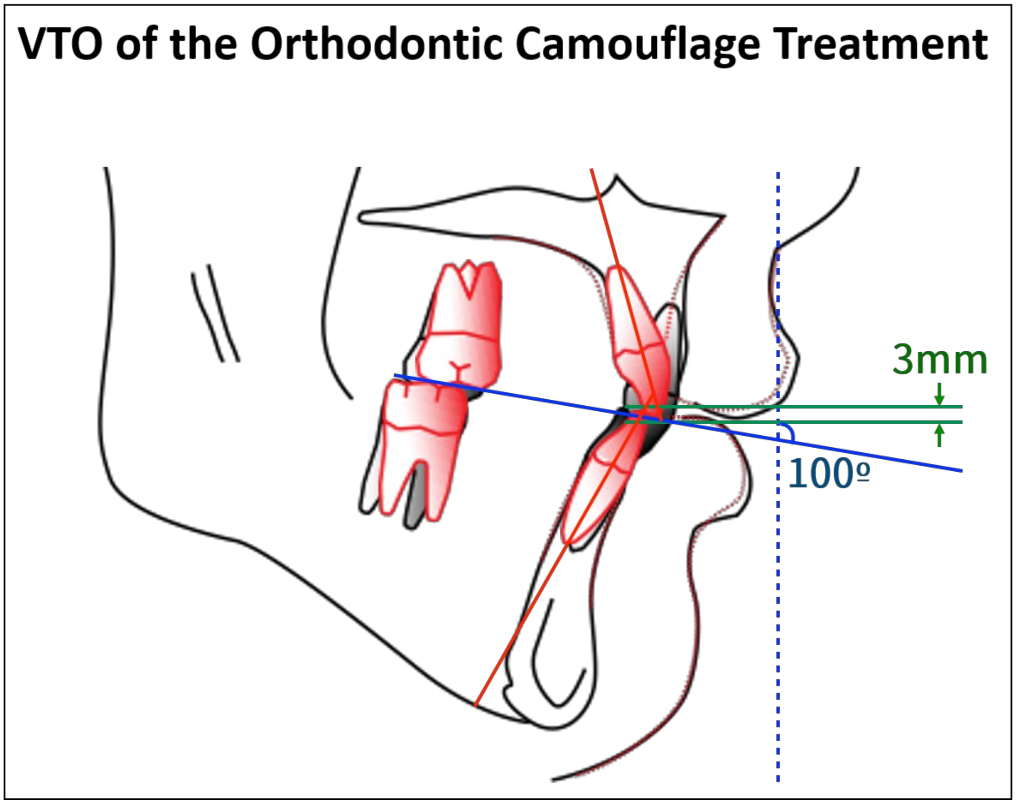


Figure 8. Result of VTO of the orthodontic camouflage treatment. Black, pretreatment; red, expected result after orthodontic treatment involving anterior and posterior teeth intrusion using TSADs after maxillary first premolar extractions.

The use of TSADs placed beneath the anterior nasal spine (ANS) has helped us and others to overcome these side effects. Many successful cases have been reported where labial crown tipping and the palatal root torque of the maxillary anterior teeth have produced an effective intrusive force [28, 30]. Meanwhile, Kim et al. reported on the treatment of a Class II Division 2 patient with severe deep bite and retroclined maxillary incisors in which they placed TSADs in the maxillary buccal alveolar bone and achieved reliable and effective treatment modality for torque control and intrusion of maxillary anterior teeth [49].

For the treatment of Class II Division 2 patients with severe deep overbite, intrusion and palatal root movement of the maxillary incisors are essential. For intrusion of the maxillary incisors, some studies have proposed that TSADs be installed underneath ANS [28, 30] or between the maxillary lateral incisor and canine roots [46]. However, if TSADs are placed in the position where mainly intrusive vertical forces are applied, the vector of the orthodontic force tends to cause labial tipping of the crown rather than palatal root movement on the maxillary incisors [50]. Therefore, in the treatment of Class II Division 2 patients with a severe deep overbite, a distointrusive force vector is required to prevent proclination of the maxillary incisors and to induce pure intrusion with palatal root movement (Figure 9) [38].

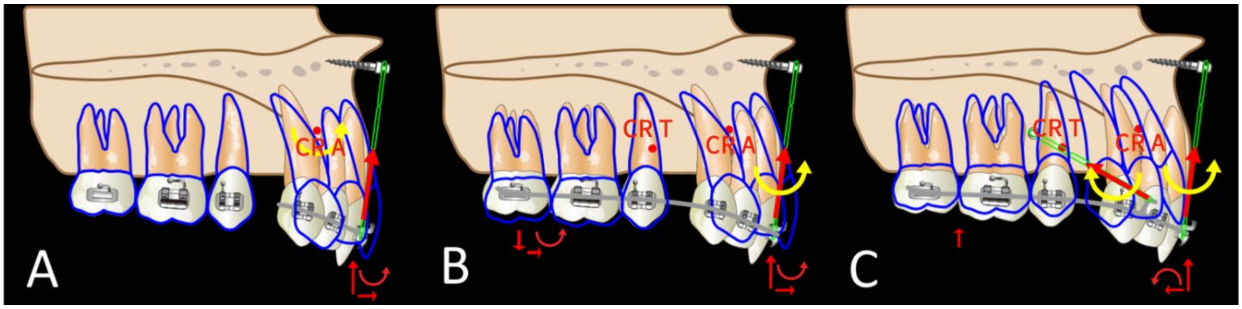


Figure 9. Treatment mechanics for intrusion: A) A single TSAD installed underneath the ANS for the intrusion of maxillary anterior segments tends to produce labial proclination and round tipping of the anterior teeth, as the intrusion force passes through the anterior point of the center of resistance (Center of resistance A; CR A); B) When the entire dentition is connected with a continuous archwire, counterclockwise rotation of the full maxillary dentition occurs on the rotation axis (Center of resistance T; CR T), which leads to extrusion of the maxillary molars; C) To prevent labial proclination and induce a genuine intrusion of the anterior teeth, a distointrusive force must pass through the center of resistance of the full dentition (CR T).

As in Figure 9 (B and C), since a continuous archwire connected the anterior and posterior segments of the dentition, the target segment was the whole arch, and the center of resistance (CR) would be localized around the premolars [49]. Therefore, the distointrusive force of the TSADs installed between the second premolars and first molars, passing close to the imaginary center of resistance of the maxillary dentition, leads to the intrusion of the maxillary molars as well as the incisors [38, 49].

When a large amount of intrusion is required, obtaining an optimized incisor display is essential, as the biomechanics necessary to accomplish this tend to produce a flat smile arc [42-44, 51]. In the treatment of a patient who shows a gummy smile or steep inclination of their maxillary occlusal plane, massive intrusion of maxillary incisors can be an effective treatment option from the esthetic point of view, but the intrusion of maxillary incisors must be carefully controlled in patients with a normal or flat maxillary occlusal plane inclination. Sarver stated that maxillary intrusion arches or maxillary archwires with an accentuated curve could result in a flattening of the smile arc [51]. Sabri also reported that if the maxillary incisors are overintruded to correct an overbite or if the maxillary occlusal plane is canted upward anteriorly, the smile arc may be flattened [44]. Therefore, when treating Class II Division 2 malocclusion patients, we should create an appropriate smile arc not only by the intrusion of the maxillary incisors but also by the intrusion of the maxillary molars.

FACTORS THAT AFFECT THE LONG-TERM STABILITY OF ANTERIOR DEEP OVERBITE TREATMENT

One of the most important considerations when treating Class II Division 2 malocclusion is the long-term stability of the overbite, and therefore the treatment mechanics should be selected on this basis. Deep overbite correction by extrusion of the posterior teeth can be an effective option for some growing patients. However, it is known that the stability is questionable when adult patients with average-to-low mandibular plane angles are treated due to masseteric muscle force, so the intrusion of the anterior teeth should accompany extrusion of the posterior teeth [35-38]. Furthermore, maintaining the original mandibular plane angle can be beneficial for long-term stability in the treatment of adult Class II Division 2 patients [41].

It has been reported that the pretreatment severity of an anterior deep bite correlates with the long-term stability of the treatment results [52]. Late adolescents might experience some overbite reduction and seem to be slightly more stable after deep bite treatment. Patients with more divergent facial patterns exhibit less deep overbite relapse. In other words, high-angle subjects tend to have less relapse in overbite than low-angle and normal-angle subjects [53-55]. Riedel reported that a large interincisal angle at the end of treatment was associated with a greater chance of relapse of deep overbite [56]. A proper interincisal angle and effective incisal stops must be produced to achieve the best stability possible [40].

Previous studies have reported that absolute intrusion and lingual root torque of the anterior teeth increased the possibility of unwanted root resorption [57-60]. Parker and Harris suggested that incisor intrusion, together with an increase in lingual root torque, were the strongest predictors of external apical root resorption [59]. However, Aras and Tuncer reported interesting data suggesting that the rates of root resorption were higher when incisor intrusion was achieved with TSADs placed at the CR of the maxillary anterior teeth between the maxillary lateral incisor and canine as compared with anterior intrusion accompanied with root torque control [61]. The results of their study suggested that when the intrusion mechanics used to move incisor roots toward the palatal cancellous bone avoided any contact with labial cortical bone, there was less root resorption.

No significant differences were seen in the amount of correction or relapse when comparing extraction versus nonextraction treatment results from different Angle malocclusion groups [52]. Likewise, there were no significant differences between treatment techniques using sectional versus continuous wires [62,63]. Finally, intruding maxillary incisors to avoid excessive contact with the lower lip lessens relapse in patients with Class II Division 2 malocclusion [64].

Unfortunately, the studies we reviewed did not completely detail the types and regimens they used when reporting their retention results. Furthermore, there was an inconsistency in how the various studies defined postretention, and they did not include important information about patient compliance/noncompliance. There was no clear pattern at the time of follow-up nor any indication of whether retainers were still in place or used at the posttreatment follow-up [52]. This lack of credible scientific evidence makes it extremely challenging to assess the retention results.

CONCLUSIONS

Many methods have been used to treat deep overbite. When treating adult Class II Division 2 patients, properly placed TSADs can enable a remarkable amount of intrusion of the maxillary anterior teeth and help establish proper inclination of the occlusal plane. Using this treatment modality with the proper biomechanics will minimize root resorption. It can be helpful in achieving improvement of facial esthetics and a pleasing smile arc with long-term stability when treating Class II deep overbite patients.

REFERENCES

1. Varshini GN, Padmavati R, Kannan MS. Deep bite-an overview. Eur J Mol Clin Med 2020; 7(8):1767-1771.
2. Fattahi H, Pakshir H, Afzali Baghdadabadi N, Shahian Jahromi S. Skeletal and dentoalveolar features in patients with deep overbite malocclusion. J Dent (Tehran) 2014; 11(6):629-638.

3. Alhammadi MS, Halboub E, Fayed MS, Labib A, El-Saaidi C. Global distribution of malocclusion traits: A systematic review. Dental Press J Orthod 2018; 23(6):40 e41-40 e10.
4. Wessberg GA, Fish LC, Epker BN. The short face patient: surgical-orthodontic treatment options. J Clin Orthod 1982; 16(10):668-685.
5. Pepicelli A, Woods M, Briggs C. The mandibular muscles and their importance in orthodontics: a contemporary review. Am J Orthod Dentofacial Orthop 2005; 128(6):774-780.
6. Burstone CJ. Lip posture and its significance in treatment planning. Am J Orthod 1967; 53(4):262-284.
7. Bell WH, Jacobs JD, Legan HL. Treatment of Class II deep bite by orthodontic and surgical means. Am J Orthod 1984; 85(1):1-20.
8. Pamukcu H, Ozsoy OP. A comparison of treatment results of adult deep-bite cases treated with lingual and labial fixed appliances. Angle Orthod 2021; 91(5):590-596.
9. Skieller V, Bjork A, Linde-Hansen T. Prediction of mandibular growth rotation evaluated from a longitudinal implant sample. Am J Orthod 1984; 86(5):359-370.
10. Park JH, Kim J. Class II Correction. PCSO Bulletin 2020; 92(2):58-64.
11. Siriwat PP, Jarabak JR. Malocclusion and facial morphology is there a relationship? An epidemiologic study. Angle Orthod 1985; 55(2):127-138.
12. Buschang PH, Tadlock L. Treating skeletal Class II hyperdivergent patients—a structured decision-making process. In: Park JH, ed. Temporary Anchorage Devices in Clinical Orthodontics. Hoboken, NJ: Wiley; 2020:Ch 14.
13. Janson G, Sathler R, Fernandes TM, Branco NC, Freitas MR. Correction of Class II malocclusion with Class II elastics: a systematic review. Am J Orthod Dentofacial Orthop 2013; 143(3):383-392.
14. Fudalej P, Antoszewska J. Are orthodontic distalizers reinforced with the temporary skeletal anchorage devices effective? Am J Orthod Dentofacial Orthop 2011; 139(6):722-729.
15. Oh YH, Park HS, Kwon TG. Treatment effects of microimplant-aided sliding mechanics on distal retraction of posterior teeth. Am J Orthod Dentofacial Orthop 2011; 139(4):470-481.
16. Sugawara J, Kanzaki R, Takahashi I, Nagasaka H, Nanda R. Distal movement of maxillary molars in nongrowing patients with the skeletal anchorage system. Am J Orthod Dentofacial Orthop 2006; 129(6):723-733.
17. Chang C, Roberts WE. Insights to extra-radicular bone screw application for challenging malocclusions. In: Park JH, ed. Temporary Anchorage Devices in Clinical Orthodontics. Hoboken, NJ: Wiley; 2020:433-444.
18. Kook YA, Bayome M, Trang VT, Kim HJ, Park JH, Kim KB, Behrents RG. Treatment effects of a modified palatal anchorage plate for distalization evaluated with cone-beam computed tomography. Am J Orthod Dentofacial Orthop 2014; 146(1):47-54.
19. Kook YA, Park JH, Bayome M, Jung CY, Kim Y, Kim SH. Application of palatal plate for nonextraction treatment in an adolescent boy with severe overjet. Am J Orthod Dentofacial Orthop 2017; 152(6):859-869.
20. Park JH. Die modifizierte C-Gaumenplatte (MCPP). Kieferorthopadie Nachrichten 2019; 17:12-15.
21. Kook YA, Park JH, Bayome M, Sa'aed NL. Correction of severe bimaxillary protrusion with first premolar extractions and total arch distalization with palatal anchorage plates. Am J Orthod Dentofacial Orthop 2015; 148(2):310-320.
22. Lee SK, Abbas NH, Bayome M, Baik UB, Kook YA, Hong M, Park JH. A comparison of treatment effects of total arch distalization using modified C-palatal plate vs buccal miniscrews. Angle Orthod 2018; 88(1):45-51.
23. Park JH, Kim S, Lee YJ, Bayome M, Kook YA, Hong M, Kim Y. Three-dimensional evaluation of maxillary dentoalveolar changes and airway space after distalization in adults. Angle Orthod 2018; 88(2):187-194.

24. Shoaib AM, Park JH, Bayome M, Abbas NH, Alfaifi M, Kook YA. Treatment stability after total maxillary arch distalization with modified C-palatal plates in adults. Am J Orthod Dentofacial Orthop 2019; 156(6):832-839.
25. Lee YJ, Kook YA, Park JH, Park J, Bayome M, Vaid NR, Kim Y. Short-term cone-beam computed tomography evaluation of maxillary third molar changes after total arch distalization in adolescents. Am J Orthod Dentofacial Orthop 2019; 155(2):191-197.
26. Park YC, Kim JK, Lee JS. Biomechanical considerations with temporary anchorage devices. In: Graber LW, Vanarsdall RL Jr, Vig KWL, eds. Orthodontics: Current Principles and Techniques. Philadelphia, PA: Elsevier; 2012, 381-419.
27. Kang H, Lee NK, Kim J, Park JH, Kim Y, Kook YA. Factors associated with the maxillary third molar position after total arch distalization using a modified C-palatal plate in adolescents. Orthod Craniofac Res 2021; 24 Suppl 1:31-38.
28. Creekmore TD, Eklund MK. The possibility of skeletal anchorage. J Clin Orthod 1983; 17(4):266-269.
29. Kanomi R. Mini-implant for orthodontic anchorage. J Clin Orthod 1997; 31(11):763-767.
30. Ohnishi H, Yagi T, Yasuda Y, Takada K. A mini-implant for orthodontic anchorage in a deep overbite case. Angle Orthod 2005; 75(3):444-452.
31. Deguchi T, Murakami T, Kuroda S, Yabuuchi T, Kamioka H, Takano-Yamamoto T. Comparison of the intrusion effects on the maxillary incisors between implant anchorage and J-hook headgear. Am J Orthod Dentofacial Orthop 2008; 133(5):654-660.
32. Upadhyay M, Yadav S, Patil S. Mini-implant anchorage for en-masse retraction of maxillary anterior teeth: a clinical cephalometric study. Am J Orthod Dentofacial Orthop 2008; 134(6):803-810.
33. Polat-Ozsoy O, Arman-Ozcirpici A, Veziroglu F. Miniscrews for upper incisor intrusion. Eur J Orthod 2009; 31(4):412-416.
34. Saxena R, Kumar PS, Upadhyay M, Naik V. A clinical evaluation of orthodontic mini-implants as intraoral anchorage for the intrusion of maxillary anterior teeth. World J Orthod 2010; 11(4):346-351.
35. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977; 72(1):1-22.
36. Nanda R. Correction of deep overbite in adults. Dent Clin North Am 1997; 41(1):67-87.
37. Burstone CJ. Biomechanics of deep overbite correction. Semin Orthod 2001; 7(1):26-33.
38. Lee GH, Park JH, Moon D, Lee SM. Use of temporary skeletal anchorage devices for intrusive root movement of maxillary incisors in adult patients with Class II Division 2 malocclusion. Am J Orthod Dentofacial Orthop Clin Companion 2022; 2(2):152-166.
39. Bayram M. Combined orthodontic-orthopedic treatment of an adolescent Class II Division 2 patient with extreme deepbite using the Forsus Fatigue Resistant Device. Am J Orthod Dentofacial Orthop 2017; 152(3):389-401.
40. Houston WJ. Incisor edge-centroid relationships and overbite depth. Eur J Orthod 1989; 11(2):139-143.
41. McDowell EH, Baker IM. The skeletodental adaptations in deep bite correction. Am J Orthod Dentofacial Orthop 1991; 100(4):370-375.
42. Zachrisson BU. Esthetic factors involved in anterior tooth display and the smile: Vertical dimension. J Clin Orthod 1998; 32:432-445.
43. Lindauer SJ, Lewis SM, Shroff B. Overbite correction and smile esthetics. Semin Orthod 2005; 11(2): 62-66.
44. Sabri R. The eight components of a balanced smile. J Clin Orthod 2005; 39(3):155-167; quiz 154.
45. Jayade V, Annigeri S, Jayade C, Thawani P. Biomechanics of torque from twisted rectangular archwires. A finite element investigation. Angle Orthod 2007; 77(2):214-220.
46. Upadhyay M, Nagaraj K, Yadav S, Saxena R. Mini-implants for en masse intrusion of maxillary anterior teeth in a severe Class II division 2 malocclusion. J Orthod 2008; 35(2):79-89.

47. Tai K, Park JH. Improvement of facial profile by nonextraction orthodontic treatment with temporary skeletal anchorage devices and visual treatment objectives. Am J Orthod Dentofacial Orthop 2018; 154(5):708-717.
48. Lee GH, Park JH, Moon DN, Lee SM. Protocols for orthodontic treatment of patients with temporomandibular joint disorders. Am J Orthod Dentofacial Orthop 2021; 159(3):373-388.
49. Kim SJ, Kim JW, Choi TH, Lee KJ. Combined use of miniscrews and continuous arch for intrusive root movement of incisors in Class II division 2 with gummy smile. Angle Orthod 2014; 84(5):910-918.
50. Park HK, Sung EH, Cho YS, Mo SS, Chun YS, Lee KJ. 3-D FEA on the intrusion of mandibular anterior segment using orthodontic miniscrews. Korean J Orthod 2011; 41(6):384-398.
51. Sarver DM. The importance of incisor positioning in the esthetic smile: the smile arc. Am J Orthod Dentofacial Orthop 2001; 120(2):98-111.
52. Huang GJ, Bates SB, Ehlert AA, Whiting DP, Chen SS, Bollen AM. Stability of deep-bite correction: A systematic review. J World Fed Orthod 2012; 1(3):e89-e86.
53. Pollard D, Akyalcin S, Wiltshire WA, Rody WJ, Jr. Relapse of orthodontically corrected deepbites in accordance with growth pattern. Am J Orthod Dentofacial Orthop 2012; 141(4):477-483.
54. Bock N, Ruf S. Post-treatment occlusal changes in Class II division 2 subjects treated with the Herbst appliance. Eur J Orthod 2008; 30(6):606-613.
55. Harris EF, Vaden JL. Posttreatment stability in adult and adolescent orthodontic patients: a cast analysis. Int J Adult Orthodon Orthognath Surg 1994; 9(1):19-29.
56. Riedel RA. A review of the retention problem. Angle Orthod 1960; 30:179-199.
57. Harris DA, Jones AS, Darendeliler MA. Physical properties of root cementum: part 8. Volumetric analysis of root resorption craters after application of controlled intrusive light and heavy orthodontic forces: a microcomputed tomography scan study. Am J Orthod Dentofacial Orthop 2006; 130(5):639-647.
58. Han G, Huang S, Von den Hoff JW, Zeng X, Kuijpers-Jagtman AM. Root resorption after orthodontic intrusion and extrusion: an intraindividual study. Angle Orthod 2005; 75(6):912-918.
59. Parker RJ, Harris EF. Directions of orthodontic tooth movements associated with external apical root resorption of the maxillary central incisor. Am J Orthod Dentofacial Orthop 1998; 114(6):677-683.
60. Kaley J, Phillips C. Factors related to root resorption in edgewise practice. Angle Orthod 1991; 61(2):125-132.
61. Aras I, Tuncer AV. Comparison of anterior and posterior mini-implant-assisted maxillary incisor intrusion: Root resorption and treatment efficiency. Angle Orthod 2016; 86(5):746-752.
62. Preston CB, Maggard MB, Lampasso J, Chalabi O. Long-term effectiveness of the continuous and the sectional archwire techniques in leveling the curve of Spee. Am J Orthod Dentofacial Orthop 2008; 133(4):550-555.
63. Dake ML, Sinclair PM. A comparison of the Ricketts and Tweed-type arch leveling techniques. Am J Orthod Dentofacial Orthop 1989; 95(1):72-78.
64. Lapatki BG, Baustert D, Schulte-Monting J, Frucht S, Jonas IE. Lip-to-incisor relationship and postorthodontic long-term stability of cover-bite treatment. Angle Orthod 2006; 76(6):942-949.

CLINICAL MANAGEMENT OF DEEP BITE: PERSPECTIVE OF THE CLINICAL ORTHODONTIST

Steven J. Lindauer

ABSTRACT

Deep overbite is a common characteristic that accompanies many presentations of malocclusion treated by orthodontists throughout the world. Despite its high prevalence, deep bite alone is not usually the primary motivator for patients to seek orthodontic care. Orthodontists know, however, that correction of deep overbite is necessary during the course of orthodontic treatment in order to create the space needed to align maxillary and mandibular teeth and position the arches into a functional and esthetic Class I relationship necessary to achieve ideal overjet. Patients, parents, and orthodontists themselves expect high-quality outcomes, and achieving those results often requires mechanics dedicated to improving overbite. Excessive overbite can be corrected by the true intrusion of maxillary or mandibular incisors, relative intrusion, or by opening the bite to allow eruption of posterior teeth. While more than one mechanism may be used to contribute to overbite correction during treatment, orthodontists should intentionally plan the means of deep bite correction for each patient individually, based on relevant diagnostic information and their knowledge of the effects of the mechanics they choose to employ. Utilizing the so-called “side-effects” of deep bite correction mechanics advantageously can improve treatment efficiency, shorten the duration of treatment, and lead to better patient outcomes.

KEY WORDS: Overbite, Intrusion, Mechanics, Esthetics

INTRODUCTION

Deep overbite is a common malocclusion trait, with prevalence reported at about 22% worldwide, and over 30% in the United States, in the permanent dentition [1, 2]. Nearly all orthodontic treatment plans include achieving normal overbite as one of the primary objectives. Reducing deep overbite is usually attained early in the course of orthodontic treatment with fixed appliances and may take between 3.5 and 5.5 months or longer during an average overall treatment time that extends over 23 to 29 months [3-5].

The website of the American Association of Orthodontists (AAO) explains to potential patients that there are important reasons why a deep bite should be fixed [6]. One reason given is that overerupted lower incisors tend to wear down more quickly. In severe cases, a patient may be occluding on palatal tissue, resulting in painful sores and ulcers that could impair mastication. For those instances, overbite correction might serve as a preventive measure to avoid future harm. Previous studies have reported some associations between deep bite and attrition [7-9] as well as periodontal damage [10, 11]; however, the cause-and-effect relationships and whether orthodontic treatment can prevent or reverse these conditions remain unclear [10]. Lastly, the AAO website explains that deep bite correction is often needed to provide space to align crowded, malaligned teeth. In their classic paper on stability of orthodontic treatment results, Bishara et al. stated that “overbite correction is mechanically desirable to obtain the bracket clearance needed to complete the retraction of maxillary anterior teeth” [12]. Orthodontists know that normalization of the overbite is required to achieve ideal overjet with well-aligned maxillary and mandibular arches in a Class I relationship.

PATIENT PERCEPTIONS OF DEEP BITE

Common orthodontic treatment objectives include aligning the teeth and achieving ideal overbite and overjet with the maxillary and mandibular arches in a Class I (canine) relationship. While the oral health benefits of treatment in terms of caries prevention [13, 14], avoiding future periodontal disease [15, 16], and preserving temporomandibular joint function [17-19] have been shown to be minimal or non-existent on a population basis [20], it is widely accepted that there are social advantages derived from having an attractive smile. Both general dentists and orthodontic specialists rated the psychosocial improvements from orthodontic treatment more highly than the oral health benefits [21]. Several studies have demonstrated that individuals derive social benefits from improved smile esthetics [22-24]. A negative impact of malocclusion on oral health-related quality of life has been demonstrated along with significant improvements in those measures resulting from orthodontic treatment [25-27].

Patient motivation to seek orthodontic treatment is consistent with the perceived benefits. A study in the United Kingdom reported that the prime motivating factor for adolescents seeking orthodontic treatment was improved esthetics, but, for their parents, it was preventing future problems [28]. However, a study performed in the US around the same time found that over 90% of both adolescents and their parents rated improved esthetics as the most important factor [29]. A more recent report, conducted by observing online chat discussions, confirmed earlier findings of esthetic improvement as the most frequent motivator, followed by functional concerns and then psychosocial reasons [30]. Interestingly, the findings also reported esthetic motivation as the most negative factor, with the sentiment expressed that people should be able to accept themselves as they are without undergoing cosmetic alterations.

If esthetic improvement is the prime motivator, it is unlikely that deep bite is an important factor influencing patients to seek orthodontic treatment. Studies evaluating esthetic smile characteristics have not found increased overbite to be a characteristic that experts and lay raters consider as one of the most important unpleasing factors. In a comprehensive study examining thresholds of tolerance for 54 different smile characteristics by 243 laypeople, Kerr et al. reported that study participants were “…tolerant of a deeper bite which enables the creation of a congruent smile arc” [31]. Of all the characteristics examined, overbite had the most variability and was the least reliable to assess, with tolerances reported up to 5.7 mm. In another study specifically evaluating the esthetic tolerance for overbite variations compared to other malocclusion characteristics, only about half of the lay evaluators were able to detect deep bite compared to almost universal recognition of other occlusal deviations, including protrusion (98%), crowding (97%), spacing (96%), openbite (94%), and crossbite (78%) [32].

Even though the appearance of deep bite is not considered as particularly objectionable to patients, as previously pointed out by Bishara et al. [12], overbite correction is essential for achieving other goals of orthodontic treatment, including tooth alignment with ideal overjet. Although it might be assumed that patients seek treatment merely to achieve well-aligned teeth, previous studies confirm that other occlusal relationships, as well as placement of the teeth in proper positions in all three dimensions relative to the soft tissues, are important for achieving patient satisfaction [31, 32]. Despite research showing that patients, parents, and orthodontists desire shorter treatment times [33], it was also found that parents and orthodontists generally would agree to extending treatment duration to achieve better occlusal outcomes [34]. In most cases, parents recommended extending treatment times more often and for longer periods than orthodontists, by up to 7 months on average. Parents were less willing to terminate treatment early if it meant accepting outcomes they did not consider to be ideal [34]. The outcomes expected by patients, parents, and orthodontists, including good intercuspation with normal incisor overjet, would almost always require deep overbite to be corrected as part of the overall orthodontic treatment plan.

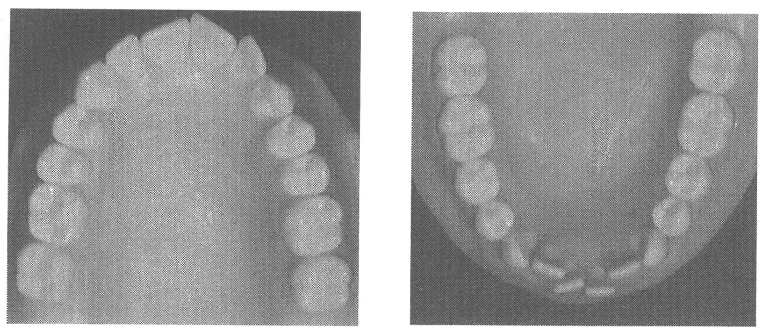
METHODS FOR CORRECTING DEEP BITE

Conceptually, there are three basic ways to correct deep overbite: true intrusion of anterior teeth, flaring of anterior teeth (relative intrusion), and erupting posterior teeth (which also rotates the mandible open). True intrusion can be achieved in the maxillary or mandibular incisors by applying intrusively directed forces by archwires, such as intrusion arches [3, 35-37], or by elastics extended from temporary anchorage devices inserted into the alveolar bone [38]. This results in true vertical movement of the centers of resistance of the incisors to correct overbite. Flaring of anterior teeth occurs by inserting a leveling archwire or clear aligners on crowded teeth or using curved archwires to correct the overbite [39]. This may or may not also result in vertical movement of the centers of resistance of the incisors, but the primary mechanism of overbite correction during flaring is due to vertical intrusive movement of the incisal edges. Lastly, bite planes or bite turbos can be used to cause posterior disclusion, thereby allowing for eruption of posterior teeth, reducing anterior overbite, and rotating the mandibular plane open [3, 37, 39]. Of course, the method chosen should be different for various patients, depending on the initial presentation and specific goals of treatment [37]. Intruding maxillary incisors will result in less incisor show at rest and during smiling, which may or may not be desirable depending on the original patient characteristics [37]. Flaring of incisors may be contraindicated in patients with thin gingival biotype or for stability concerns [40]. Lastly, increasing vertical facial height may be unfavorable in patients with vertical facial excess and may also be unstable long-term.

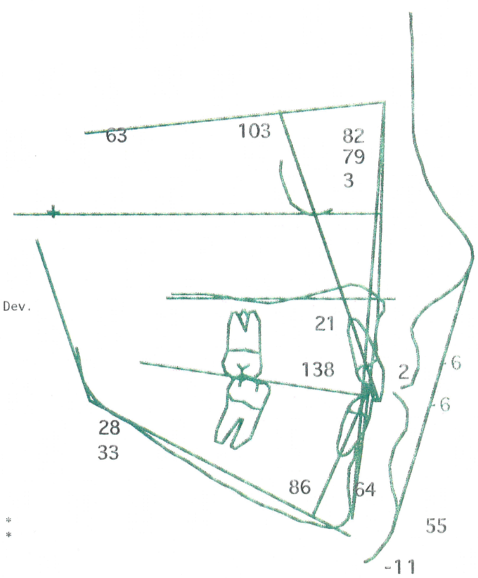
It is likely that all three of these mechanisms: true intrusion, relative intrusion, and posterior tooth eruption, occur during the course of most orthodontic treatment when patients start with excessive overbite. A retrospective study involving 31 adult patients treated with and without four premolar extractions examined differences in overbite correction contributions between maxillary and mandibular incisors due to intrusion and flaring [41]. Pre- and post-treatment lateral cephalometric superimpositions were used to evaluate tooth movements and rule out mandibular plane inclination changes. Mean overbite reduction was about 2 mm on average. There were no significant differences in changes recorded between extraction and non-extraction cases. Mandibular incisor movement contributed a significantly greater amount toward overbite correction than maxillary incisor movement. Despite there having been no specific mechanics directed toward achieving true incisor intrusion in the cases analyzed, true intrusion of both maxillary and mandibular incisors contributed more to overbite correction than did flaring. Overall, the greatest contribution to overbite correction in these adult patients was true intrusion of the mandibular incisors. This finding was in contrast to a previous study conducted in growing 11 – 16-year-olds, in which the mandibular incisors erupted by 0.2 mm to 0.9 mm on average during treatment involving deep overbite correction [42]. The differences in results between these two studies may be attributable to substantial mandibular growth having occurred in the sample consisting of young adolescents.

The degree of deep bite present in a patient at the start of treatment likely has an influence on whether an extraction or non-extraction treatment plan will be chosen by an orthodontic provider. Since extractions are expected to result in space to relieve crowding and allow retraction of the incisors, clinicians anticipate that incisor uprighting will cause increased overbite during the course of treatment, and this is calculated into the overall treatment plan. A study was designed to determine the influence of lip profile, incisor inclination, and amount of overbite present on the extraction/non-extraction decision-making process [43]. Records of an adolescent Class I patient with a borderline amount of crowding were altered to show flat/average/full lips (facial photos), upright/average/flared incisors (cephalometric radiograph and measures), and deep/average/open bite (study models) (Figure 1).

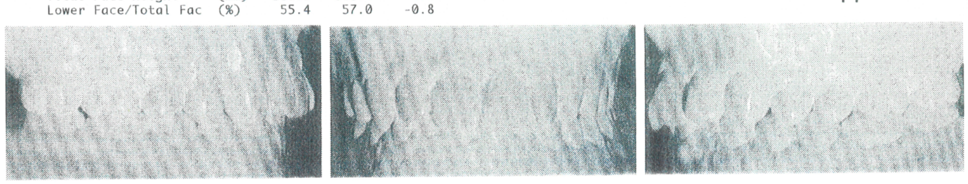
1A



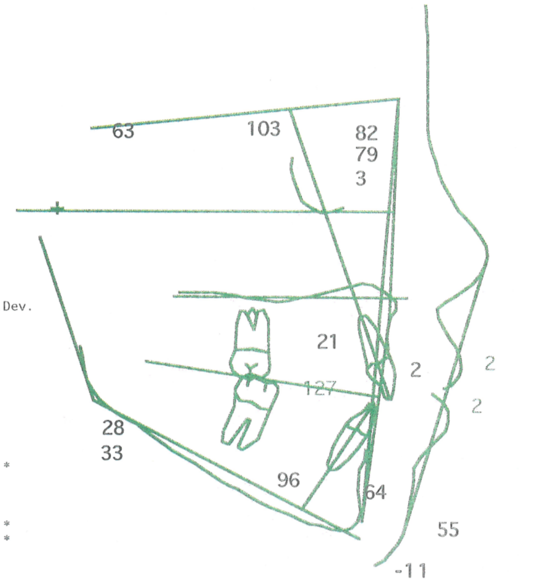
1B



		Tr	Norm	Clin.Dev.	
SNA	(dg)	81.9	81.0	0.3	
SNB	(dg)	79.4	79.0	0.1	
ANB	(dg)	2.5	2.0	0.3	
FH-MPA	(dg)	27.6	25.0	0.4	
SN - MPA	(dg)	33.3	32.0	0.3	
Mx 1 - SN	(dg)	102.7	103.0	-0.0	
Mx 1 - NA Angle	(dg)	20.9	18.0	0.5	
Mx 1 - NA	(mm)	1.9	3.0	-0.4	
IMPA	(dg)	86.0	91.0	-0.8	
Interincisor Angle	(dg)	138.0	135.0	0.5	
Y - Axis	(dg)	62.6	66.0	-1.0	
Facial Contour Angle	(dg)	-10.7	-11.0	0.1	
A-B (OP) - WITS	(mm)	0.2	-1.0	0.6	
E Plane - Upper Lip	(mm)	-5.8	-2.0	-1.9	*
E Plane - Lower Lip	(mm)	-5.5	-2.0	-1.8	*
Lower Ant Fac Height	(mm)	63.8	62.0	0.5	
Total Face Height	(mm)	115.2	111.4	0.6	
Lower Face/Total Fac	(%)	55.4	57.0	-0.8	



1C



		Tr	Norm	Clin.Dev.	
SNA	(dg)	81.9	81.0	0.3	
SNB	(dg)	79.4	79.0	0.1	
ANB	(dg)	2.5	2.0	0.3	
FH-MPA	(dg)	27.6	25.0	0.4	
SN - MPA	(dg)	33.3	32.0	0.3	
Mx 1 - SN	(dg)	103.3	103.0	0.1	
Mx 1 - NA Angle	(dg)	21.4	18.0	0.6	
Mx 1 - NA	(mm)	1.9	3.0	-0.4	
IMPA	(dg)	96.1	91.0	0.9	
Interincisor Angle	(dg)	127.3	135.0	-1.3	*
Y - Axis	(dg)	62.6	66.0	-1.0	
Facial Contour Angle	(dg)	-10.7	-11.0	0.1	
A-B (OP) - WITS	(mm)	0.2	-1.0	0.6	
E Plane - Upper Lip	(mm)	1.9	-2.0	2.0	*
E Plane - Lower Lip	(mm)	1.9	-2.0	1.9	*
Lower Ant Fac Height	(mm)	63.8	62.0	0.5	
Total Face Height	(mm)	115.2	111.4	0.6	
Lower Face/Total Fac	(%)	55.4	57.0	-0.8	

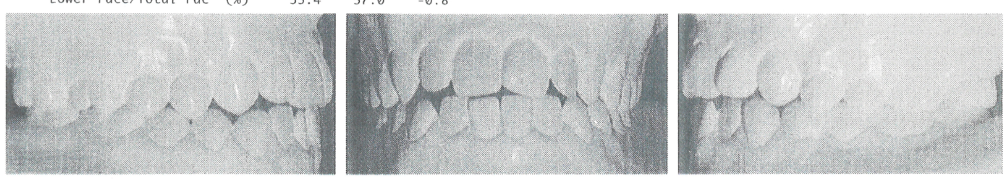


Figure 1. Digitally altered records distributed to orthodontic evaluators as a part of a study evaluating the influence of lip fullness, incisor inclination, and amount of overbite on the extraction/non-extraction decision. A) Maxillary and mandibular arch crowding was the same for every case presented. B) Case example with the highest proportion of non-extraction recommendations. In this case, the lips were flat, the incisors were upright, and the overbite was deep. C) Case example with the highest proportion of extraction recommendations. In this case, the lips were full, the incisors were flared, and the bite was open.

All combinations (27 variations) were sent to 128 orthodontists around the US, and, for each case, they were asked to decide if they would treat the case with or without extractions. The response rate was 77%. The amount of intra-arch crowding was the same for each case (Figure 1A). Of all combinations, the case with the lowest number of extraction recommendations was the one with flat lips, upright incisors, and deep bite (2:96, extraction:non-extraction decision; Figure 1B). The case with the highest number of extraction recommendations was the one with full lips, flared incisors, and openbite (7:90, extraction:non-extraction decision; Figure 1C). Lip profile, incisor inclination, and overbite all significantly affected extraction/non-extraction decision-making ($P<0.05$), with lip profile being the most influential.

PLANNING AND TREATMENT FOR DEEP BITE CORRECTION

The decision of whether to treat a patient with deep bite using intrusion mechanics (true intrusion), flaring of anterior teeth (relative intrusion), or inserting a bite plate or bite turbos (posterior extrusion) depends on initial presentation and provider preference. For each patient, diagnostic information including skeletal, dental, and facial characteristics, and specific contributing factors such as growth status, periodontal conditions, and other considerations should be taken into account. Additionally, the duration of treatment might be different depending on which method of overbite correction is chosen.

Overbite correction by true intrusion

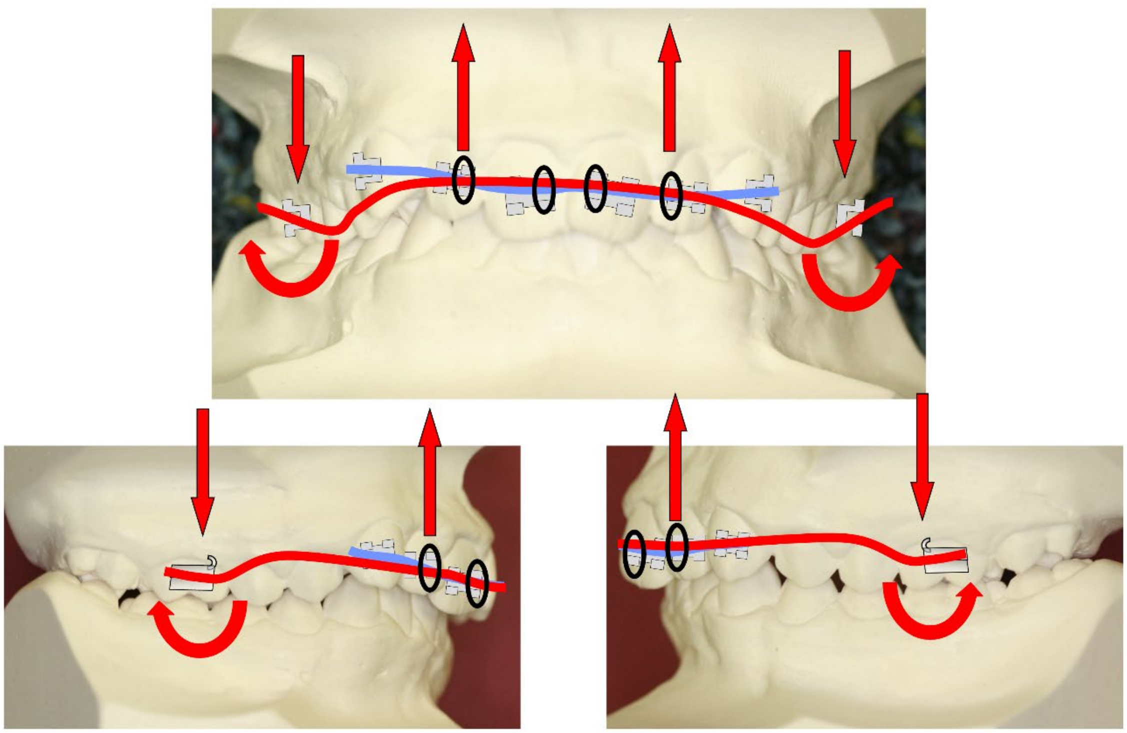
Deep bite can be corrected by intrusion of either maxillary or mandibular incisors or both. However, in this paper, only intrusion of maxillary incisors will be discussed. General indications for choosing to intrude maxillary incisors to correct deep bite include excessive, or at least adequate, initial incisor show at rest and during smiling, and having a Class II tendency is also favorable, though not required. Classically, an intrusively activated rectangular wire (intrusion arch) is placed into auxiliary tubes on the maxillary first molars and tied anteriorly to a rigid segment of wire, including the anterior teeth to be intruded (anterior segment). The intrusive force is derived by curving or bending the wire to create a tip-back couple at the molar tube, and because the wire is not inserted into the anterior brackets, no couple is expressed anteriorly. Two equal and oppositely directed forces are created: intrusive anteriorly and extrusive posteriorly. More information regarding the appliances and force systems can be found in previous publications [35, 36, 44, 45]. Generally, increasing the size of any segment will increase its anchorage so, if less molar tip-back is desired, the molar can be connected to more posterior teeth. If less intrusion is desired, the anterior segment can be extended posteriorly to include the canines.

Some of the advantages of using an intrusion arch to reduce deep overbite include: 1) Maxillary molar tipback is helpful for Class II correction, 2) Tipback couple at the molar is helpful for increasing posterior anchorage for retraction [46], and 3) Since the extrusive side effect is concentrated on the maxillary molar, vertical side effects can be more easily controlled in vertically sensitive patients. Potential disadvantages of intrusion arch mechanics include: 1) Intrusion occurs slowly, so overbite correction may take longer than other methods, 2) Incisor display will decrease with intrusion, 3) Incisor root resorption is a potential side effect; hence force levels should be low, about 60 g for a four tooth segment, and this may reduce risks of molar extrusion and incisor root resorption [44].

Figure 2A shows the initial intraoral photos of a 12.5-year-old male who presented with Class II malocclusion and deep bite. He was full step Class II on the right and end on Class II on the left. The treatment plan included using a transpalatal arch (TPA) to rotate the maxillary first molars mesiobuccally to improve molar positioning, followed by an anterior intrusion arch from the maxillary first molars to an anterior segment from canine to canine. A canine-to-canine segment was chosen to maximize molar tipback during anterior intrusion to help correct the Class II molar relationship during a period of active mandibular growth. The intended force system is depicted in Figure 2B on the starting models. Figure 2C shows the intraoral photographs at the end of the overbite correction phase, which took 9 months. At this point, the deep overbite was corrected, and the buccal occlusion was Class I without bonding the maxillary premolars or the lower arch. Mandibular growth was partially responsible for the favorable response. Figure 2D shows the final occlusion and the post-treatment panoramic radiograph demonstrating parallel root positioning.

2A

2B



2C

2D

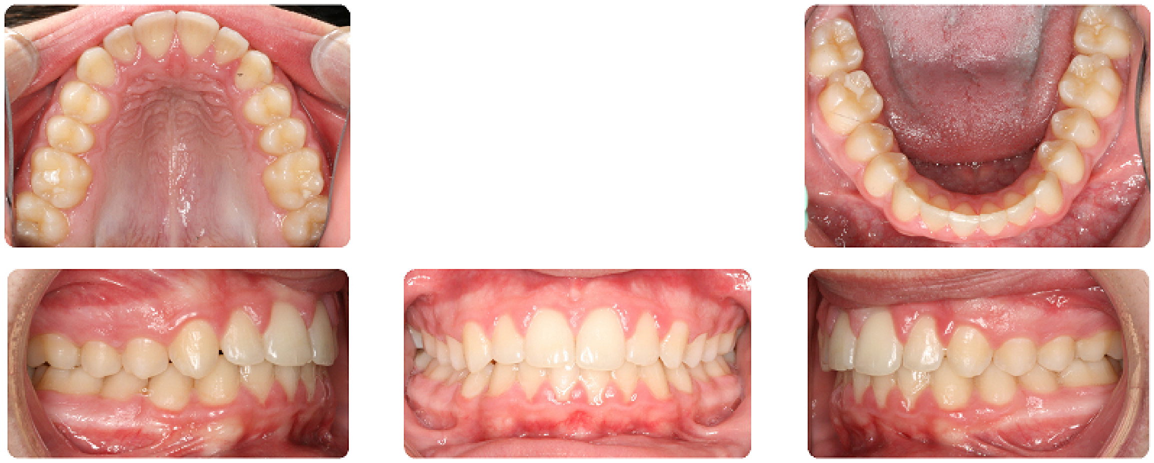


Figure 2. Case presentation of a 12.5-year-old male patient with deep bite treated with intrusion mechanics. A) Initial intraoral photos showing deep overbite, full step Class II on the right and end on Class II on the left. B) Mechanics of the force system generated by an intrusion arch showing tip-back couples at the maxillary molars, intrusive force anteriorly, and extrusive force posteriorly. C) Intraoral photos at the end of intrusion (9 months) show reduction of overbite and correction of the buccal occlusion to Class I without bonding the premolars or the lower arch. D) Final occlusion and panoramic radiograph showing root parallelism.

Overbite correction by relative intrusion

Deep bite correction by relative intrusion happens whenever crowding is resolved by aligning the teeth without creating additional space. When the anterior teeth flare, additional arch circumference is created [47], and the incisal edges relatively intrude to reduce overbite. Since excessive flaring of the anterior teeth is considered to be inherently unstable [48], relative intrusion to reduce overbite should generally be used as the primary source of overbite correction only in cases with minimal initial crowding and in which the initial deep bite is not severe. Figure 3 shows the frontal view of a patient with about 40% overbite at the start of treatment and after alignment of the teeth. Flaring of the anterior teeth resulted in reduction of the deep bite without any additional mechanics specifically directed toward overbite correction.

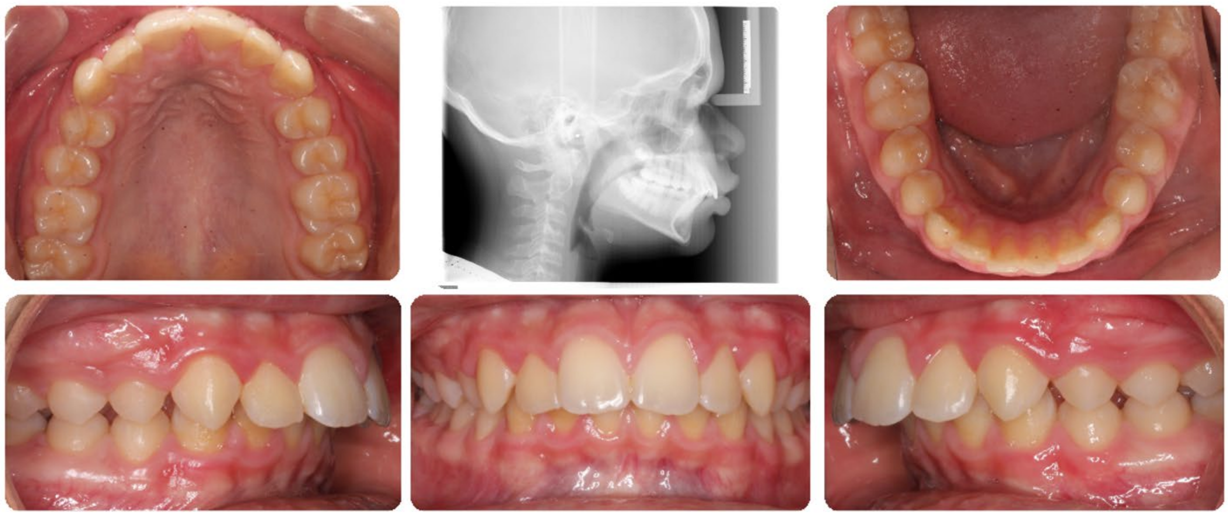
Figure 3. Overbite correction by relative intrusion. Pretreatment photo (above) shows minor crowding and 40% overbite. After alignment (below), the overbite is corrected.

Overbite correction by posterior tooth eruption

For patients in whom posterior tooth eruption is indicated for the correction of a deep bite, a bite plate or anterior bite turbos can be used to prop the bite open, thereby allowing for eruption of the posterior teeth. Indications for posterior tooth eruption include: 1) Patients with short lower anterior facial height and a flat mandibular plane, 2) Competent lips, and 3) Usually growing patients, since some adult patients may be less tolerant of facial height changes. The greatest advantage of this method is that bite opening is immediate, allowing the mandibular arch to be bonded and aligned as soon as the bite is opened and, therefore, expediting treatment efficiently. A possible disadvantage is that the long-term stability of the facial height increase is uncertain.

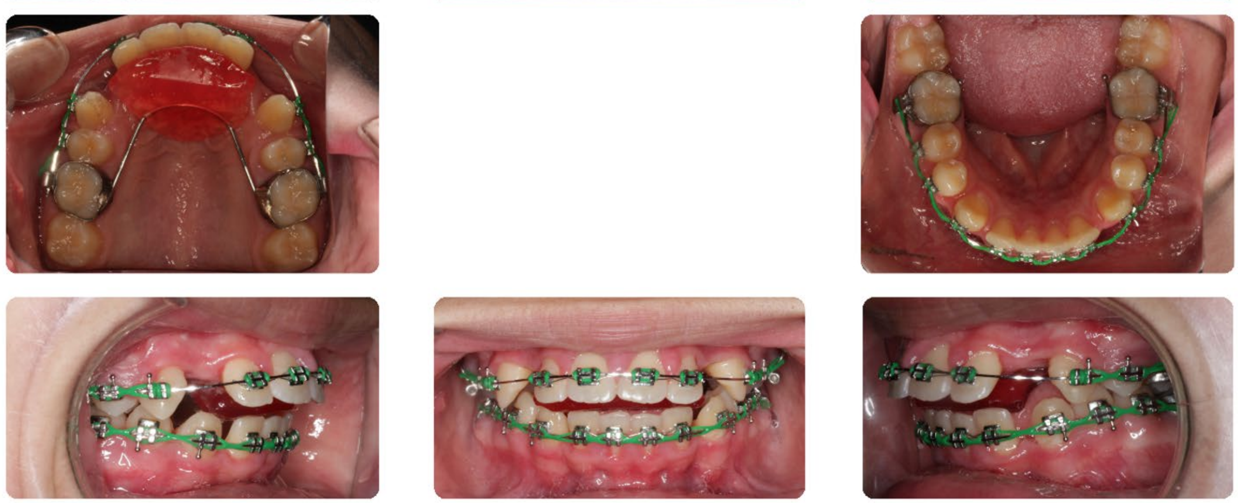
Pretreatment photos and the cephalometric radiograph of a 13-year-old male with a Class II deep bite malocclusion are shown in Figure 4A. After 3 months of alignment, maxillary first premolars were extracted, and a fixed anterior bite plate was inserted to disclude the posterior teeth (Figure 4B). The bite plate was used as anchorage for 7 months to retract the maxillary canines while the posterior teeth erupted into occlusion (Figure 4C). Then, the bite plate was removed, and the anterior teeth were retracted (Figure 4D). Final photos and the cephalometric radiograph are shown in Figure 4E.

4A



4B

4C



4D

4E

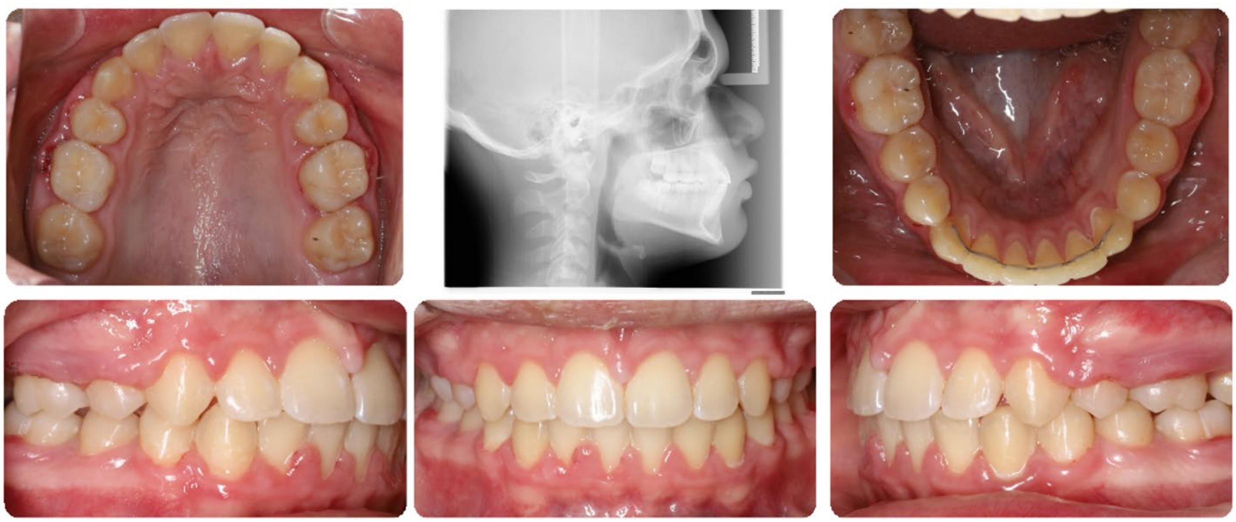
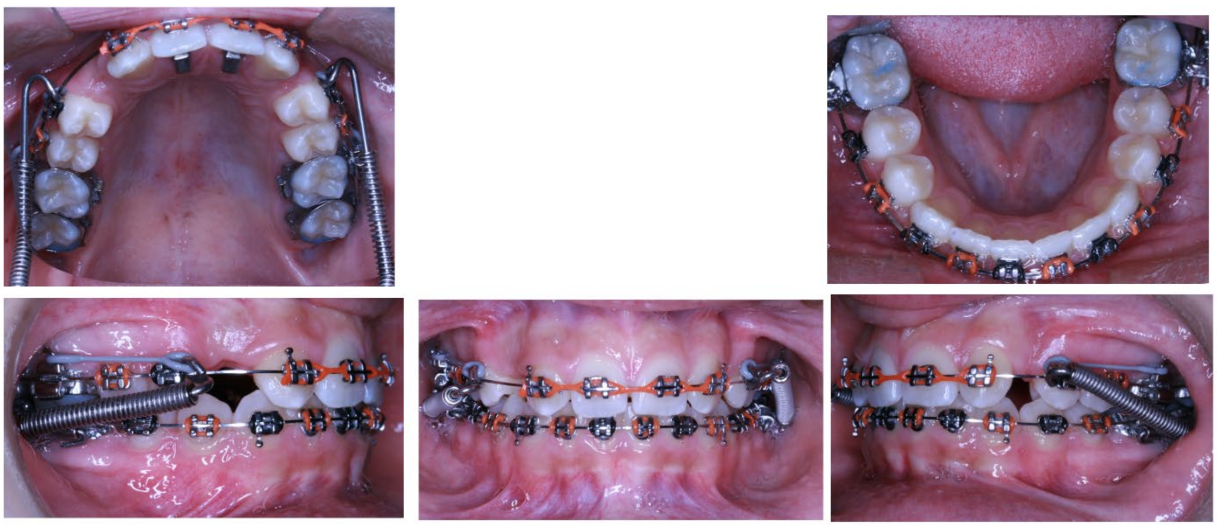


Figure 4. Deep bite correction by posterior tooth eruption enabled by an anterior bite plate. A) Pretreatment intraoral photographs and lateral cephalometric image. The plan was to open the bite using a bite plate and correct the Class II relationship by extracting maxillary premolars and retracting anterior teeth. B) A fixed bite plate was placed, discluding the posterior teeth. C) The bite plate was used as anchorage to retract the canines and, during those 7 months, the posterior teeth erupted into occlusion. D) The bite plate was removed to retract the incisors. E) Final occlusion and cephalometric radiograph.

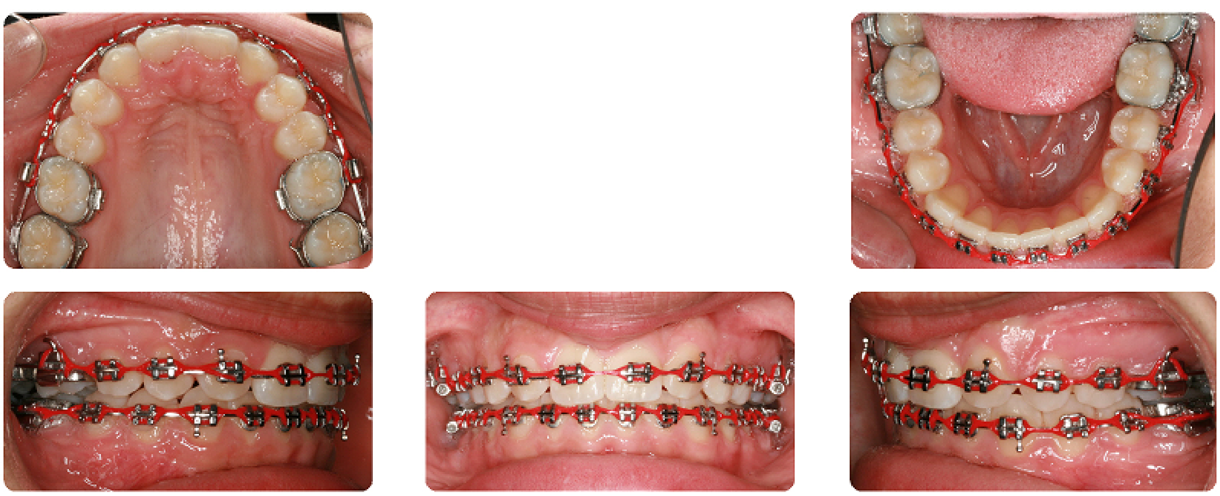
Figure 5A shows the pretreatment photos and cephalometric radiograph of a 12-year-old female with a Class I molar relationship and deep bite. She was congenitally missing both maxillary lateral incisors. The treatment plan was to extract maxillary primary canines and protract the posterior segments into a Class II relationship, using the permanent canines to substitute for the missing lateral incisors. The bite was opened using stainless steel bite turbos bonded on the lingual surfaces of the maxillary central incisors, and a Forsus appliance (3M Orthodontics, USA) was configured to protract the posterior teeth using a K module (3M Orthodontics, USA) (Figure 5B). Once the posterior segments were protracted, the bite turbos and Forsus appliance were removed (Figure 5C). Final photographs and a cephalometric radiograph are shown in Figure 5D.

5A

5B



5C



5D

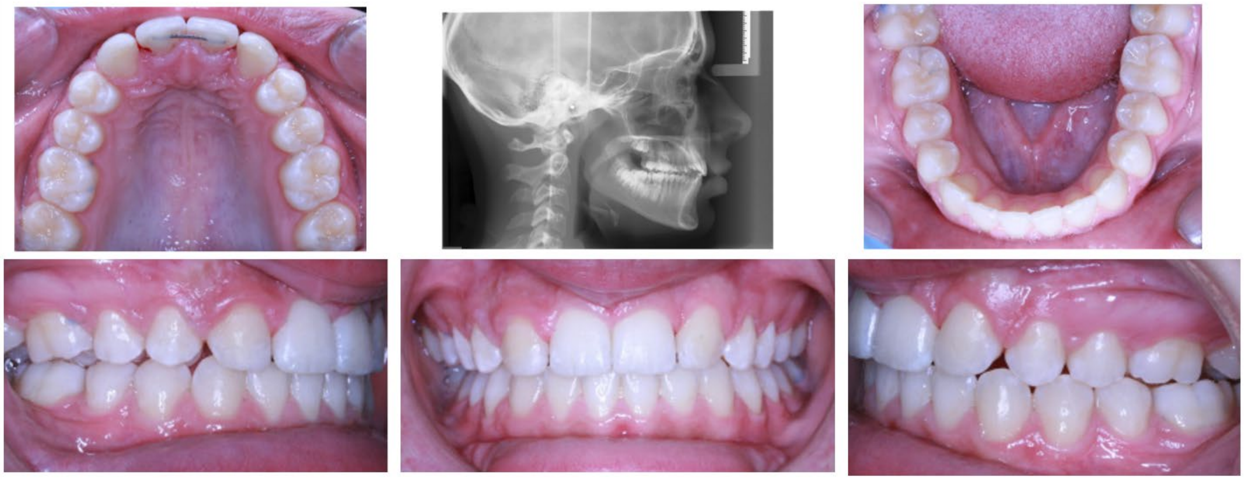


Figure 5. Deep bite correction by posterior tooth eruption enabled by anterior bite turbos. A) Pretreatment intraoral photographs and cephalometric radiograph. The patient had a Class I molar relationship but was missing permanent maxillary lateral incisors. The plan was to extract primary canines and protract the maxillary posterior teeth to enable canine substitution for the missing lateral incisors. B) Anterior bite turbos bonded on the lingual surfaces of the maxillary central incisors opened the bite and a Forsus appliance was configured to protract the maxillary posterior teeth. A K-module connecting the appliance to the maxillary molar enabled protraction. C) After the posterior teeth were protracted, the bite turbos were removed. D) Final photos and cephalometric radiograph.

DIFFERENCES IN OUTCOMES BETWEEN DEEP BITE CORRECTION METHODS

Depending on which method is chosen to achieve deep bite correction, different outcomes would logically be expected. For example, intrusion of maxillary incisors should result in decreased incisor display and minimal vertical facial changes. However, posterior tooth eruption would be anticipated to increase vertical facial height without appreciable changes in incisor show at rest or during smiling. A prospective, non-randomized clinical trial was undertaken to investigate whether these expected differences actually occurred during the overbite correction phase and also whether they were maintained at the conclusion of orthodontic treatment. Details of the selection criteria, methods used, and outcomes have been reported previously [3, 37].

Changes observed in 18 patients who had deep bite corrected by maxillary incisor intrusion were compared to those seen in 14 patients who wore fixed anterior bite plates. Initial overbite was a little over 4 mm on average and was successfully resolved to under 2 mm with both methods. Time to achieve deep bite correction was significantly faster in the bite plate group (3.7 vs 5.3 mos; $P<0.05$). However, total treatment duration was not different between groups. The upper incisor incisal edge significantly intruded in both groups during overbite correction, but this effect was greater in the intrusion arch group by 1 mm. The difference was also maintained through to the end of active treatment. However, at the end of treatment, there was no difference in incisor tooth display at rest between the groups. Therefore, the data confirmed that the mechanism of overbite correction was different between the methods when measured cephalometrically, but incisor display differences could not be distinguished clinically.

Similarly, the changes in mandibular plane inclination between groups were not statistically significant or clinically relevant. This may have been because most of the patients involved were growing adolescents and due to the changes in overbite required being only about 2 mm on average. It is likely that the small differences observed in this clinical trial would be more important when more substantial deep bite correction is needed for individual patients. Therefore, careful consideration of the theoretical differences between incisor intrusion and posterior eruption methods of deep bite correction for patients with substantial amounts of overbite are still recommended.

Ackerman et al. previously observed that 33% of patients experienced flattening of the smile arc during orthodontic treatment [49]. In the clinical trial comparing incisor intrusion to bite plate correction of deep bite, 29% of intrusion arch patients and 38% of bite plate patients had flattening of the smile arc during treatment, consistent with the findings of Ackerman et al. Bracket positioning is probably more important than the choice of deep overbite correction method for creating and maintaining a pleasing smile arc appearance [50-52]. When trying to produce a consonant smile arc, use of reverse curve and accentuated curve wires for overbite correction should be avoided, since the force systems applied result in geometries that are unfavorable for esthetic outcomes (Figure 6) [53].

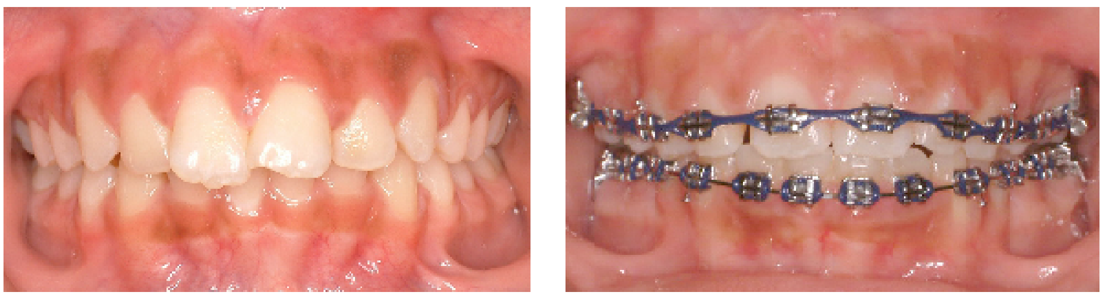


Figure 6. Reverse curve wires for overbite correction should be avoided due to force systems that are unfavorable for achieving esthetic smile outcomes. Before treatment (left) and after reverse curve wires were used to reduce overbite (right). Note the reverse smile arc created in the maxillary anterior teeth.

USING INTRUSION MECHANICS CREATIVELY

Deep overbite correction using an intrusion arch results in molar tip-back, intrusive force anteriorly, and extrusive force posteriorly. It is possible to dissect these mechanical components and use them advantageously to resolve unique problems in individual patients depending on the initial characteristics of their malocclusion. For example, the anterior intrusive force can be applied asymmetrically to correct a cant of the anterior occlusal plane during overbite correction. In contrast, if a patient presents with a Class II subdivision malocclusion, the tip-back couple can be used on the Class II side to help correct molar position, while a transpalatal arch (TPA) activated asymmetrically can be used to cancel out the unwanted tip-back on the Class I molar.

Intrusion to correct a maxillary anterior occlusal plane cant

The pretreatment intraoral frontal photograph of a patient with a maxillary anterior occlusal plane cant (down on the patient's left) is shown in Figure 7A. The treatment plan included intruding the maxillary incisors to reduce overbite. By applying the intrusive force left of the midline (white arrows), rather than symmetrically, it will cause intrusion of the incisors and rotate the occlusal plane up on the left (yellow arrows) simultaneously (Figure 7B). Extrusive force is present at the molars (white arrows), while both molars experience a tip-back couple (not shown). In this case, a light continuous wire was present in the brackets and the intrusion arch was a heavier rectangular stainless-steel wire. Figure 7C shows the final intraoral frontal view.

7A

7B

7C

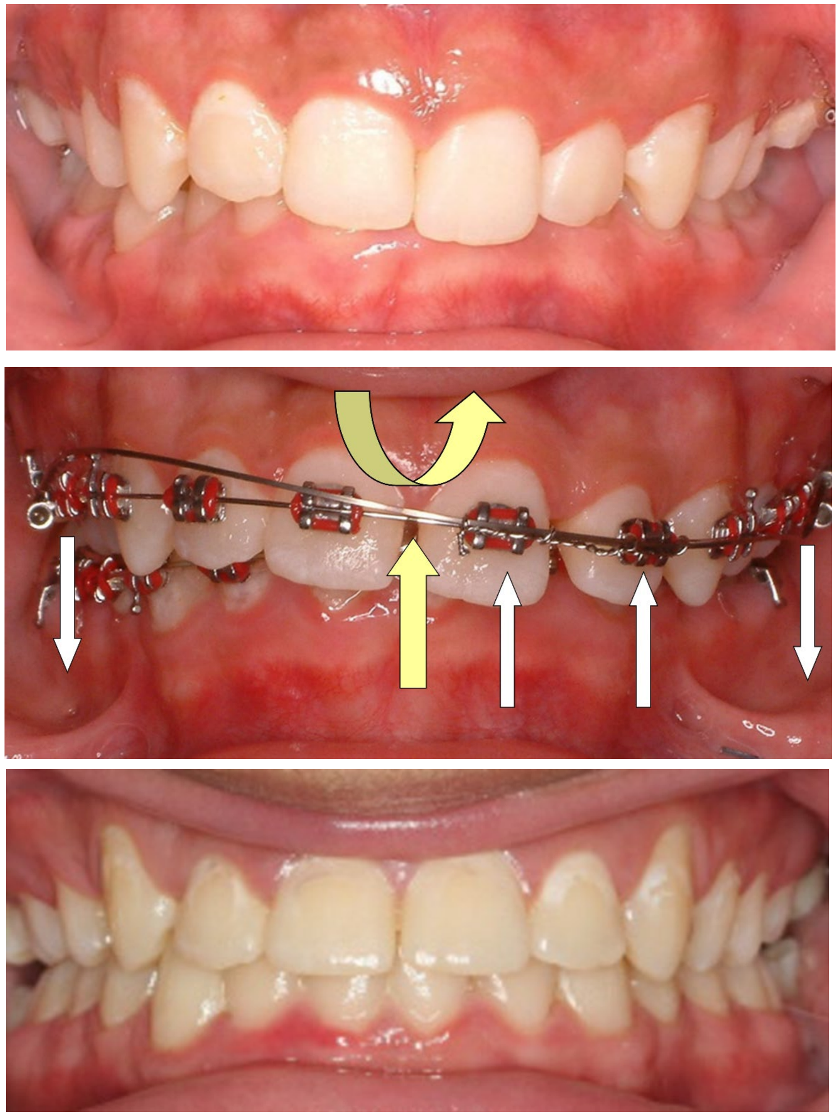


Figure 7. Anterior intrusion arch applied asymmetrically to correct a cant of the anterior occlusal plane. A) Pretreatment intraoral photograph showing maxillary anterior occlusal plane canted down on the left. B) The intrusion arch was tied to the maxillary left central and lateral incisors. White arrows show the vertical forces applied by the intrusion arch. Yellow arrows show that the anterior occlusal plane will intrude and rotate up on the left (counterclockwise). C) Final occlusion.

Using tip-back to correct asymmetric Class II

An asymmetrically activated transpalatal arch (TPA) can be used to augment the tip-back couple exerted on one of the first molars when an intrusion arch is used to correct deep bite. Tip-back would be increased on the Class II side to enhance Class II correction, and decreased on the Class I side to prevent tip-back. Second order TPA activations are always equal and opposite, with tip-back on one side balanced by tip-forward on the other side (Figure 8).

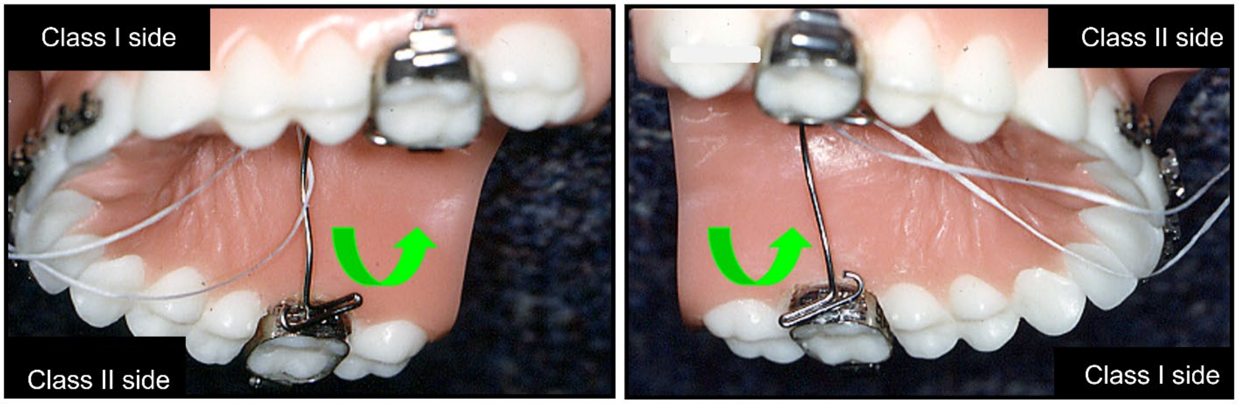


Figure 8. Second order activation of a transpalatal arch (TPA). The arch is activated to tip back the Class II molar (left photo) and tip forward the Class I molar (right photo). Second order activations of a TPA are always equal and opposite.

In a Class II subdivision patient with deep bite, an intrusion arch may be used to reduce overbite, resulting in tip-back of molars bilaterally (Figure 9A). Simultaneously, a TPA with second order activation can be added to increase tip-back on the Class II side and reduce or even cancel out tip-back on the Class I side (Figure 9B). Together, the two appliances result in anterior intrusion, posterior extrusion, and tip-back of the Class II molar with no tip-back of the Class I molar (Figures 9C and 9D).

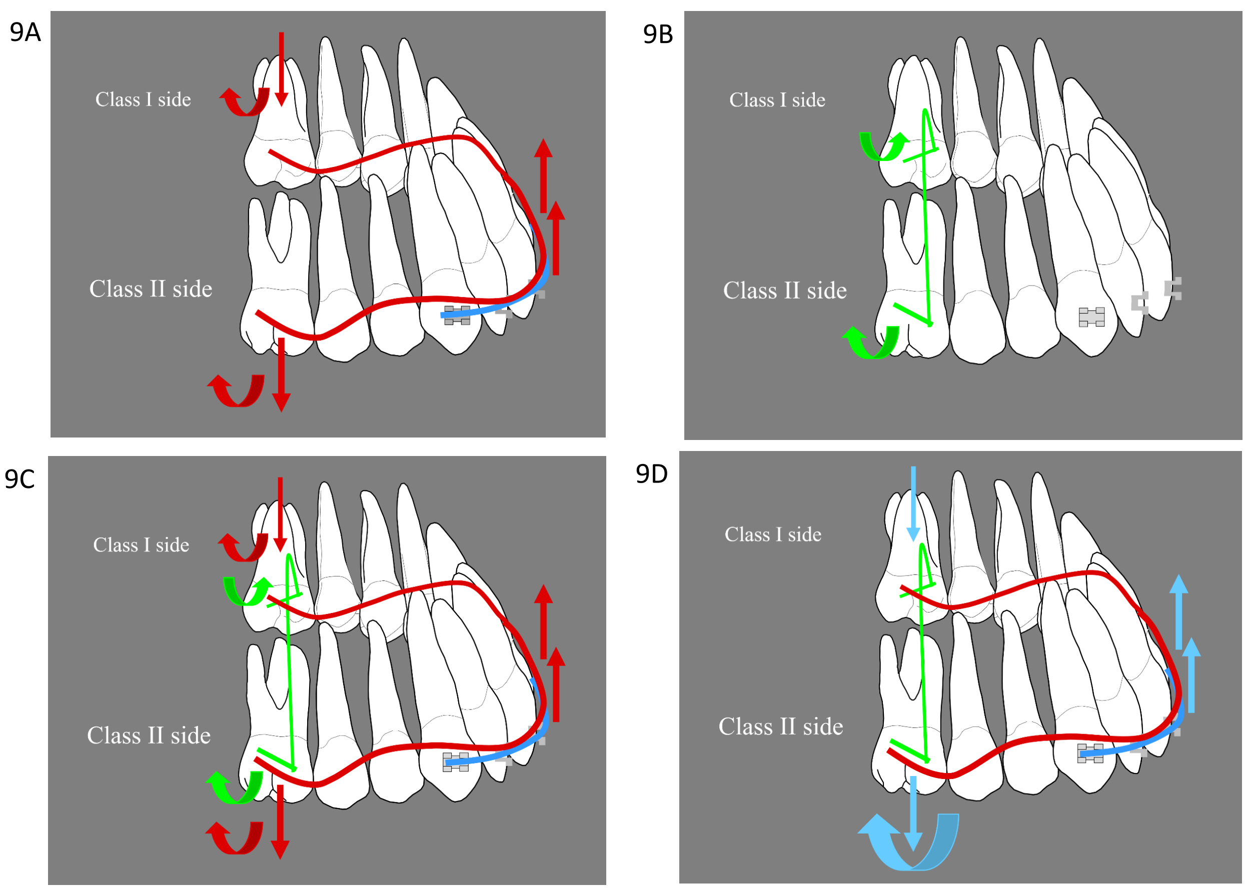


Figure 9. Mechanics from combining an anterior intrusion arch and asymmetrically activated TPA. A) Force system from an activated intrusion arch. B) Force system from a second order activation of a TPA. C) Combined force systems from an intrusion arch (red) and asymmetric TPA (green). D) Net force system from the two appliances together (blue).

Initial intraoral photos and study models of an adolescent female before treatment are shown in Figure 10A. She had about 50% overbite. The maxillary midline is left of the face and the mandibular midline is on with the facial midline. The right side is Class II end on, and the left side is Class I. The treatment plan included using an intrusion arch to reduce overbite, along with an asymmetrically activated TPA to enhance tip-back of the Class II molar and reduce tip-back of the Class I molar. Alignment of the maxillary canine to canine segment was initiated to consolidate those teeth prior to inserting the intrusion arch (Figure 10B). Use of a canine-to-canine anterior segment, rather than just including the incisors, was done intentionally to increase anterior anchorage during intrusion. This would slow intrusion of the anterior segment to maximize the tip-back effect observed on the molars.

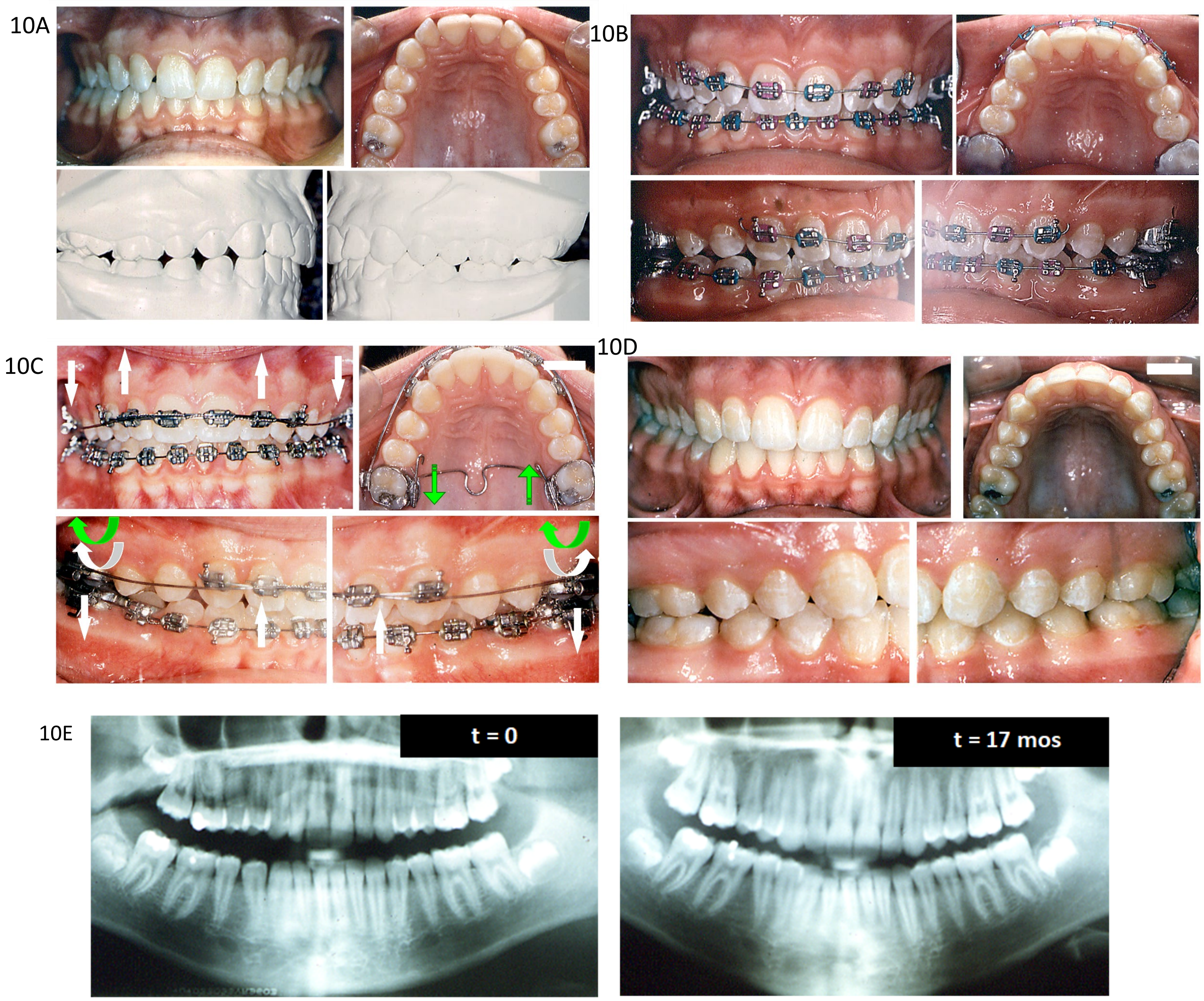


Figure 10. Using combined mechanics from anterior intrusion with an asymmetric TPA to treat a Class II subdivision malocclusion with asymmetric mechanics in the maxillary arch. A) Initial photographs showing a patient with 50% overbite, Class II end on occlusion on the right and Class I on the left. The maxillary midline is left of the facial midline. B) Initial alignment of the anterior segment canine to canine. Maxillary premolars are not bonded. C) Maxillary intrusion arch is used to correct overbite and tip-back the molars (white arrows). The TPA is used to enhance tip-back on the Class II side and reduce tip-back on the Class I side (green arrows). Note that the right premolars have drifted distally into a Class I relationship. D) Final intraoral photographs. E) Start (above) and finish (below) panoramic radiographs showing that maxillary molar root positions have been corrected after tip-back mechanics.

After consolidating the anterior canine to canine segment, the intrusion arch was used for overbite correction (white arrows) and to apply a tip-back couple (white curved arrows) to maxillary molars (Figure 10C). The TPA was used to enhance tip-back on the right molar and reduce tip-back on the left molar (green arrows). Tip-back of the right molar is evident by the spacing seen in the right maxillary segment, while the left side has no spacing and remains intercuspated in a Class I relationship. Tip-back of the right molar enabled drifting of the unbonded right premolars into a Class I relationship (Figure 10C). After achieving a Class I molar occlusion bilaterally, the remaining teeth (premolars and second molars) were bonded and the TPA was removed, leaving the intrusion arch in place to maintain overbite correction. The intrusion arch was removed once full-sized wires were in place and final detailing was accomplished. Total treatment time was 17 months (Figure 10D). Pre- and post-treatment panoramic radiographs show that the maxillary molar inclinations were normalized during the detailing and finishing stage (Figure 10E).

CONCLUSIONS

Deep bite correction is a necessary component of orthodontic treatment and is usually accomplished in the early stages before the maxillary and mandibular arches can achieve good intercuspation in a Class I relationship with ideal overjet. Reduction of excess overbite can be accomplished by true or relative incisor intrusion, or by posterior tooth eruption enabled by insertion of an anterior bite plate or anterior bite turbos. Using anterior intrusion mechanics may provide other benefits by using the side effects of intrusion advantageously to correct Class II relationships or reinforce posterior anchorage during space closure. Posterior tooth eruption strategies, however, may be a more efficient and faster means of correcting deep overbite, allowing orthodontists to proceed earlier to other planned treatment procedures. The choice of mechanism should depend on specific patient characteristics and the individualized goals of treatment determined by the treating provider along with the patient.

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REFERENCES

1. Alhammadi MS, Halboub E, Fayed MS, Labib A, El-Saaidi C. Global distribution of malocclusion traits: a systematic review. Dental Press J Orthod 2018; 23(6):40 e41-40 e10.
2. Proffit WR, Fields HW, Jr., Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the NHANES III survey. Int J Adult Orthodon Orthognath Surg 1998; 13(2):97-106.
3. Hamdan AM, Lewis SM, Kelleher KE, Elhady SN, Lindauer SJ. Does overbite reduction affect smile esthetics? Angle Orthod 2019; 89(6):847-854.
4. Fink DF, Smith RJ. The duration of orthodontic treatment. Am J Orthod Dentofacial Orthop 1992; 102(1):45-51.
5. Beckwith FR, Ackerman RJ, Jr., Cobb CM, Tira DE. An evaluation of factors affecting duration of orthodontic treatment. Am J Orthod Dentofacial Orthop 1999; 115(4):439-447.
6. American Association of Orthodontists. Why does a deep bite need to be fixed? In: Orthodontics: what’s trending. What is a deep bite? 2019. https://www.aaoinfo.org/blog/what-is-a-deep-bite. Accessed May 29, 2022.
7. Ritchard A, Welsh AH, Donnelly C. The association between occlusion and attrition. Aust Orthod J 1992; 12(3):138-142.
8. Oltramari-Navarro PV, Janson G, de Oliveira RB, Quaglio CL, Castanha Henriques JF, de Carvalho Sales-Peres SH, McNamara JA, Jr. Tooth-wear patterns in adolescents with normal occlusion and Class II Division 2 malocclusion. Am J Orthod Dentofacial Orthop 2010; 137(6):730 e731-735; discussion 730-731.
9. Khayat N, Winocur E, Kedem R, Winocur Arias O, Zaghal A, Shpack N. The Prevalence of temporomandibular disorders and dental attrition levels in patients with posterior crossbite and/or deep bite: a preliminary prospective study. Pain Res Manag 2021; 2021:8827895.
10. Zimmer B, Seifi-Shirvandeh N. Changes in gingival recession related to orthodontic treatment of traumatic deep bites in adults. J Orofac Orthop 2007; 68(3):232-244.
11. Sulistiawati S, Awalia H, Zakyah AD, Putri JA. The effect of deep bite on periodontal status of anterior teeth. J Indones Dent Assoc 2020;3(2):71-75.
12. Bishara SE, Chadha JM, Potter RB. Stability of intercanine width, overbite, and overjet correction. Am J Orthod 1973; 63(6):588-595.
13. Helm S, Petersen PE. Causal relation between malocclusion and caries. Acta Odontol Scand 1989; 47(4):217-221.
14. Proffit WR, Fields HW, Sarver DM. Contemporary Orthodontics, 4th Edition. St. Louis: Mosby/Elsevier 2007, p. 18.
15. Polson AM, Subtelny JD, Meitner SW, Polson AP, Sommers EW, Iker HP, Reed BE. Long-term periodontal status after orthodontic treatment. Am J Orthod Dentofacial Orthop 1988; 93(1):51-58.
16. Bollen AM, Cunha-Cruz J, Bakko DW, Huang GJ, Hujoel PP. The effects of orthodontic therapy on periodontal health: a systematic review of controlled evidence. J Am Dent Assoc 2008; 139(4):413-422.
17. Luther F. TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. Br Dent J 2007; 202(1):E2; discussion 38-39.
18. Luther F. TMD and occlusion part II. Damned if we don't? Functional occlusal problems: TMD epidemiology in a wider context. Br Dent J 2007; 202(1):E3; discussion 38-39.
19. Mohlin B, Axelsson S, Paulin G, Pietila T, Bondemark L, Brattstrom V, Hansen K, Holm AK. TMD in relation to malocclusion and orthodontic treatment. Angle Orthod 2007; 77(3):542-548.

20. Macey R, Thiruvenkatachari B, O'Brien K, Batista K. Do malocclusion and orthodontic treatment impact oral health? A systematic review and meta-analysis. Am J Orthod Dentofacial Orthop 2020; 157(6):738-744 e710.
21. Hunt O, Hepper P, Johnston C, Stevenson M, Burden D. Professional perceptions of the benefits of orthodontic treatment. Eur J Orthod 2001; 23(3):315-323.
22. Henson ST, Lindauer SJ, Gardner WG, Shroff B, Tufekci E, Best AM. Influence of dental esthetics on social perceptions of adolescents judged by peers. Am J Orthod Dentofacial Orthop 2011; 140(3):389-395.
23. Pithon MM, Nascimento CC, Barbosa GC, Coqueiro Rda S. Do dental esthetics have any influence on finding a job? Am J Orthod Dentofacial Orthop 2014; 146(4):423-429.
24. AlQuraini N, Shah R, Cunningham SJ. Perceptions of outcomes of orthodontic treatment in adolescent patients: a qualitative study. Eur J Orthod 2019; 41(3):294-300.
25. Liu Z, McGrath C, Hagg U. The impact of malocclusion/orthodontic treatment need on the quality of life. A systematic review. Angle Orthod 2009; 79(3):585-591.
26. Andiappan M, Gao W, Bernabe E, Kandala NB, Donaldson AN. Malocclusion, orthodontic treatment, and the Oral Health Impact Profile (OHIP-14): Systematic review and meta-analysis. Angle Orthod 2015; 85(3):493-500.
27. Javidi H, Vettore M, Benson PE. Does orthodontic treatment before the age of 18 years improve oral health-related quality of life? A systematic review and meta-analysis. Am J Orthod Dentofacial Orthop 2017; 151(4):644-655.
28. Prabakaran R, Seymour S, Moles DR, Cunningham SJ. Motivation for orthodontic treatment investigated with Q-methodology: patients' and parents' perspectives. Am J Orthod Dentofacial Orthop 2012; 142(2):213-220.
29. Daniels AS, Seacat JD, Inglehart MR: Orthodontic treatment motivation and cooperation: a cross-sectional analysis of adolescent patients' and parents' responses. Am J Orthod Dentofacial Orthop 2009, 136(6):780-787.
30. Pittman JW, Bennett ME, Koroluk LD, Robinson SG, Phillips CL. Characterizing the orthodontic patient's purchase decision: A novel approach using netnography. Am J Orthod Dentofacial Orthop 2017; 151(6):1065-1072.
31. Ker AJ, Chan R, Fields HW, Beck M, Rosenstiel S. Esthetics and smile characteristics from the layperson's perspective: a computer-based survey study. J Am Dent Assoc 2008; 139(10):1318-1327.
32. Boonchuay N, Thongudomporn U, Leethanakul C, Lindauer SJ, Youravong N. Overbite recognition and factors affecting esthetic tolerance among laypeople. Angle Orthod 2022; Manuscript Accepted October 1, 2022.
33. Uribe F, Padala S, Allareddy V, Nanda R: Patients', parents', and orthodontists' perceptions of the need for and costs of additional procedures to reduce treatment time. Am J Orthod Dentofacial Orthop 2014; 145(4 Suppl):S65-73.
34. Lindsey DH, Shroff B, Carrico CK, Dodd J, Lindauer SJ. Orthodontists' and parents' perception of finished occlusion and willingness to extend treatment time. Am J Orthod Dentofacial Orthop 2020; 158(6):799-806.
35. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977; 72(1):1-22.
36. Lindauer SJ, Isaacson RJ. One-couple orthodontic appliance systems. Semin Orthod 1995; 1(1):12-24.
37. Lindauer SJ, Lewis SM, Shroff B. Overbite correction and smile esthetics. Semin Orthod 2005; 11(2): 62-66.

38. Sosly R, Mohammed H, Rizk MZ, Jamous E, Qaisi AG, Bearn DR. Effectiveness of miniscrew-supported maxillary incisor intrusion in deep-bite correction: a systematic review and meta-analysis. Angle Orthod 2020; 90(2):291-304.
39. Al-Zoubi EM, Al-Nimri KS. A comparative study between the effect of reverse curve of Spee archwires and anterior bite turbos in the treatment of deep overbite cases. Angle Orthod 2022; 92(1):36-44.
40. Melsen B, Allais D. Factors of importance for the development of dehiscences during labial movement of mandibular incisors: a retrospective study of adult orthodontic patients. Am J Orthod Dentofacial Orthop 2005; 127(5):552-561.
41. Arroyo-Pratt M, Lindauer SJ, Rebellato J, Isaacson RJ. Components of clinical overbite correction. J Dent Res 2000; 79(1_suppl) abstract 134:160.
42. Parker CD, Nanda RS, Currier GF. Skeletal and dental changes associated with the treatment of deep bite malocclusion. Am J Orthod Dentofacial Orthop 1995; 107(4):382-393.
43. Sheats RD, Supak LA, Rebellato J, Lindauer SJ, Isaacson RJ. Borderline extraction decisions: Influence of lips, incisor inclination, and overbite. J Dent Res 1997; 76(1_suppl) abstract 894:125.
44. Burstone CJ. Biomechanics of deep overbite correction. Semin Orthod. 2001; 7(1): 26-33.
45. Shroff B, Yang I-H, Jenkins TS. Deep overbite correction: Biomechanics and clinical implications. Semin Orthod. 2020; 26(3): 126-133.
46. Kuhlberg AJ, Burstone CJ. T-loop position and anchorage control. Am J Orthod Dentofacial Orthop 1997; 112(1):12-18.
47. Germane N, Lindauer SJ, Rubenstein LK, Revere JH, Jr., Isaacson RJ. Increase in arch perimeter due to orthodontic expansion. Am J Orthod Dentofacial Orthop 1991; 100(5):421-427.
48. Little RM. Stability and relapse of dental arch alignment. Br J Orthod 1990; 17(3):235-241.
49. Ackerman JL, Ackerman MB, Brensinger CM, Landis JR. A morphometric analysis of the posed smile. Clin Orthod Res 1998; 1(1):2-11.
50. Sarver DM, Ackerman MB. Dynamic smile visualization and quantification: part 1. Evolution of the concept and dynamic records for smile capture. Am J Orthod Dentofacial Orthop 2003; 124(1):4-12.
51. Zachrisson BU. Esthetic factors involved in anterior tooth display and the smile: Vertical dimension. J Clin Orthod 1998; 32:432-445.
52. Pitts TR. Bracket positioning for smile arc protection. J Clin Orthod 2017; 51(3):142-156.
53. Ackerman MB, Ackerman JL. Smile analysis and design in the digital era. J Clin Orthod 2002; 36(4):221-236.

ESTHETIC AND FUNCTIONAL CONSIDERATIONS WHEN TREATING ANTERIOR DEEP BITE

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ABSTRACT

Deep bite is an excessive overlap of incisors and should be treated in cases with gingival impingement or with long incisor guidance that can lead to incisor wear. It is multifactorial in nature and can be associated with a decreased mandibular angle, incisor extrusion, and/or posterior underdevelopment. Posterior tooth wear or erosion can also lead to deep bite due to loss of occlusal vertical dimension, mandibular counterclockwise rotation, and bite deepening. Many treatment approaches are available, which may include incisor intrusion, molar and premolar extrusion or posterior prosthodontic build ups. Esthetic and functional aspects will determine which treatment approach to use and should be considered in treatment planning, most specifically upper incisor display at rest and occlusal vertical dimension. To illustrate and discuss different treatment approaches, a series of cases are presented, pointing out important diagnostic criteria to define how different varieties of deep bite can be addressed.

KEY WORDS: Deep Bite, Tooth Intrusion, Tooth Extrusion, Orthodontic Anchorage Technique, Dental Esthetics

INTRODUCTION

Deep bite can be defined as an overlap greater than 5mm, or greater than one third of the crown, and include mild, moderate, or severe levels. According to Moyers, defining what truly is considered an excessive overlap is difficult [1]. Wide variations of bite depths may be found, but with no harm to the teeth or surrounding tissues. The importance of an adequate overbite is to establish anterior guidance, which is the disocclusion of the posterior teeth during protrusive mandibular excursion. This protects the posterior teeth from protrusive and lateral stress, decreases jaw muscle activity, and decreases the force on the anterior teeth. Anterior guidance basically depends on three factors - interincisor angle, amount of overbite, and the inclination of the anterior articular eminence [2]. Therefore, different amounts of incisor overlap can result in anterior guidance depending on the variation of the other two factors, but a pathological deep bite can happen in two situations: gingival impingement or incisor wear.

Gingival impingement will generally be associated with an overjet and consequently, over eruption of lower incisors. Bite marks can easily be seen on the palate. On the other hand, incisor wear due to deep bite can happen in long anterior excursion with no posterior disocclusion or parafunction. These are the situations in which deep bites must be corrected (Figure 1).

Figure 1. Clinical situations in which deep bites can lead to tissue damage: gingival impingement and incisor wear. Cases with increased overjet (A) and extruded lower incisors (B) can have bite marks in the palate (C). Incisor wear will happen in cases with a long anterior guidance, with extruded (D) and upright (E) upper and lower incisors, with protrusive movement that leads to incisor wear (F).

The frequency of deep bite as measured in static occlusion ranges from 11% to 25% in the permanent dentition and 15% to 37% in the mixed dentition, with no differences in regions of the world [3]. It is more prevalent in the mixed dentition and is a normal trait during the development of occlusion [4].

Various dental and skeletal factors are associated with the presence of a deep bite. In a retrospective study, 125 non-treated deep bite patients were evaluated for the most common dento-skeletal components of this malocclusion. The contributing skeletal factor was a reduced gonial angle and the dental factors were a deep curve of Spee, the overeruption of lower or upper incisors, and undereruption of posterior teeth [5]. Regarding these structural factors, in general, deep bite treatments involve intrusion of anterior teeth or extrusion of posterior teeth, and consequently a clockwise rotation of the mandible or a combination of both [6]. Incisor intrusion can be performed by specific mechanics, such as the Burstone intrusion arches [7], arches with built-in curve of Spee, or skeletal anchorage. Molar extrusion can be achieved with bite plates by spontaneous eruption of posterior teeth [8], or bite turbos with fixed appliances [9, 10], and most recently, with aligners [11].

But, before getting carried away with the enthusiasm of performing such movements and solving deep bites, it should be remembered that intruding anterior teeth will affect esthetics and extruding posterior teeth will affect function, most specifically occlusal vertical dimension. These two aspects will be discussed and their impact in treatment planning will be demonstrated and illustrated with clinical cases.

SMILE ESTHETICS AND DEEP BITE CORRECTION

The concept of smile esthetics was a consequence of prosthodontists wishing to reproduce a natural smile with full dentures. The classic publication which described esthetic requirements for midlines, tooth proportions, vertical allocation of incisors and gradation of posterior teeth was published in the 1950s. But it was only because of the interdisciplinary demands of adult treatment and of orthognathic surgery that smile esthetics was included in the orthodontic scenario in the 1970s [12]. Cephalometric methods were described to establish the vertical allocation of the upper incisors [13, 14], later photographic methods of upper incisor display [15, 16] and the smile line were diagnosed with specific phonemes and the importance of speech and smile dynamics were also included [17, 18]. All of these diagnostic methods are now a fundamental part of contemporary orthodontics.

Incisor intrusion

Various approaches can be used to intrude upper incisors. The most common are intrusion archwires [7], arches with curve of Spee, and the use of skeletal anchorage. Much theory surrounds the intention of true versus relative intrusion, in which true intrusion is a bodily movement and relative intrusion is a change in incisor inclination. Independent of what intrusion mechanism will be used, the most important diagnostic factor, which allows upper incisors to be intruded, without jeopardizing smile and speech esthetics, is the amount of incisor display at rest. Normal upper incisor display at rest varies from 2 to 4.5mm, when measured from the incisor border to the upper lip [15-18]. Reducing this measurement will lead to reduced or lack of exposure of these teeth at smiling, but also during speech, which is considered a sign of aging. So, in other words, patients with deep bites can only have upper incisors intruded if there is excessive upper incisor display at rest.

The clinical situation presented in Figures 2 through 6 describes a case in which upper incisor intrusion is indicated for deep bite correction. The patient presented with a hyperdivergent facial pattern, lip incompetence, a broad and gummy smile, and a Class II tendency. A clear excessive smile curvature and increased upper incisor display at rest were present, which are fundamental characteristics to indicate upper incisor intrusion.

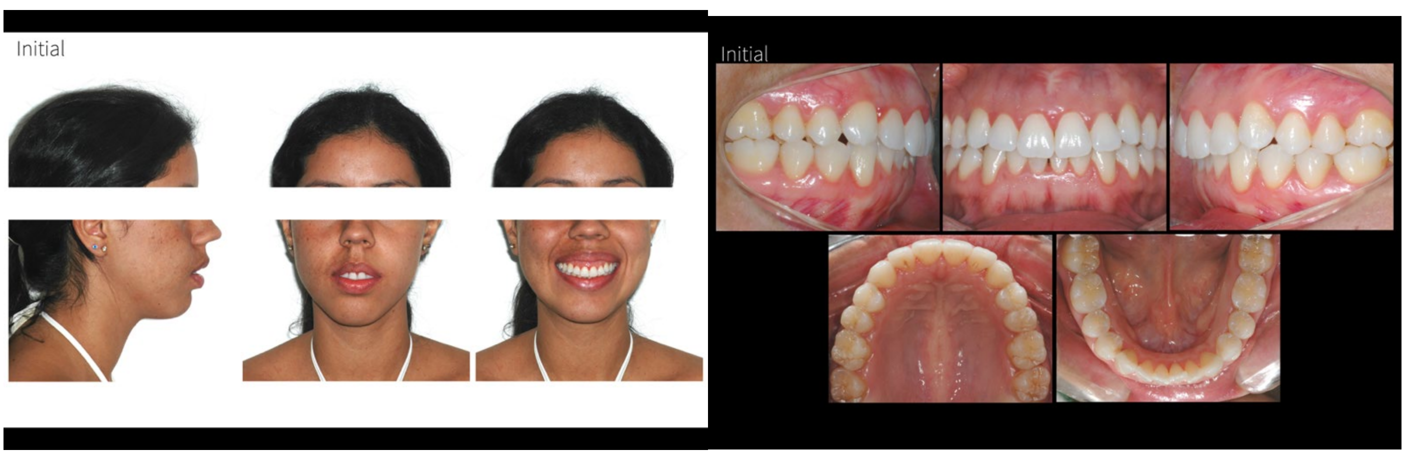

Figure 2. Initial facial (A-C) and intra oral (D-H) photographs of a young female adult patient presenting a hyperdivergent facial profile, with an increased lower facial third, protrusive lips, lack of passive lip seal and a broad smile with excessive curvature and anterior gingival exposure. She also exhibited a Class II tendency, overexpanded arches, deep bite, and reverse curve of Spee in the upper arch due to overerupted upper incisors.

Upper incisor display at rest can be evaluated on cephalometric radiographs or in clinical photographs. The distance from the lower border of the upper lip to the border of the upper incisor should measure from 2 to 4.5mm [13]. Any value greater than this allows upper incisor intrusion. This patient presented with 10mm of upper incisor exposure and an excessive smile curvature (Figure 3), therefore deep bite correction could be performed by intruding upper incisors.

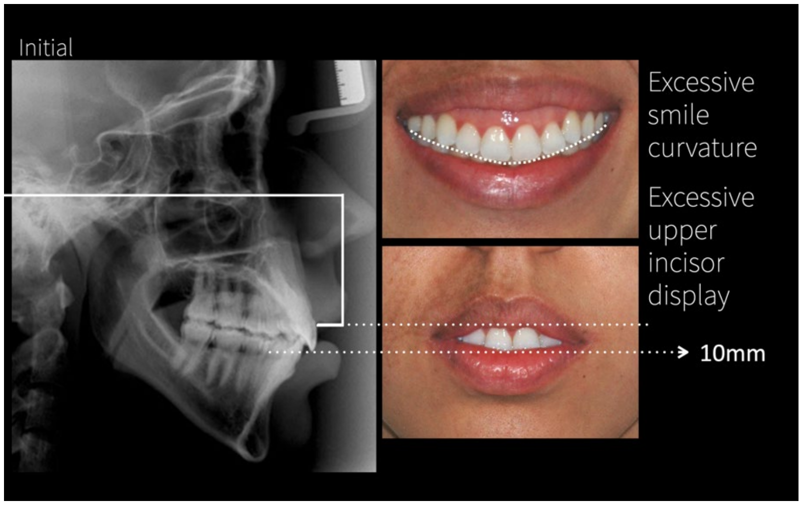

Figure 3. Cephalometric and photographic evaluation of upper incisor display at rest. The distance from the lower border of the upper lip to the upper incisal edge measured 10mm, demonstrating excessive incisor display at rest (A). The same feature was observed in a close-up photograph with interlabial gap at rest (B). The smile close-up shows an excessive smile curvature, which is advantageous in cases that need upper incisor intrusion, which generally flattens the smile curve (C).

Due to bimaxillary protrusion, a four first premolar extraction treatment was performed, which began with an upper intrusion archwire, associated with indirect skeletal anchorage on upper molars to avoid extrusion and lengthening of the lower facial third. Upper arch retraction was also performed with a distal and intrusive direction with the aid of mini-implants (Figure 4).

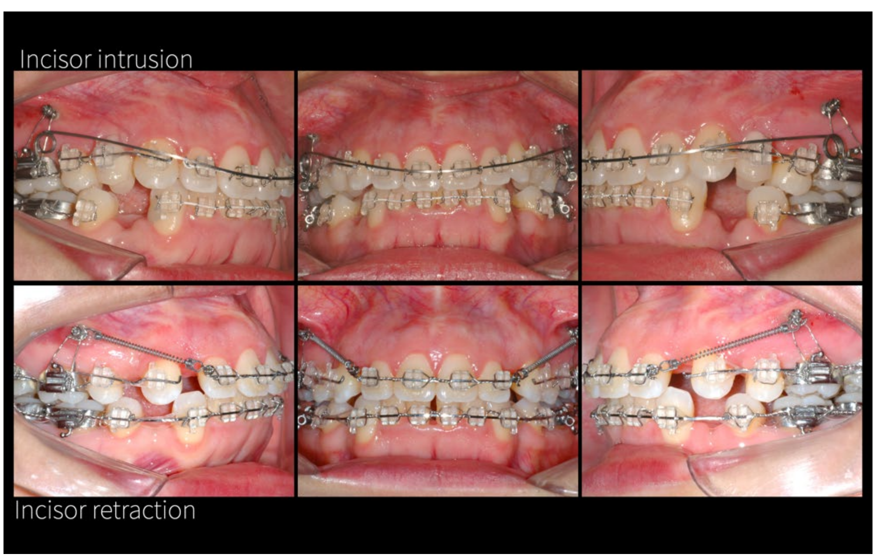

Figure 4. Intrusion (A-C) and retraction (D-F) mechanics used for upper incisor intrusion and space closure. Indirect skeletal anchorage was used to avoid molar extrusion as well as retract upper incisor with an intrusive force vector.

Final results (Figure 5) presented a harmonious facial profile and a significant improvement of smile esthetics. This was due to the incisor retraction associated with intrusion (Figure 6), resulting in a more esthetic smile line and adequate lip seal. A Class I occlusion was established, with sufficient reduction of the deep bite.

Figure 5. Final facial (A-C) and intra oral (D-H) photographs presenting a harmonious facial profile, with passive lip seal and good smile line. A Class I malocclusion was established with upright upper and lower incisors and an adequate overbite.

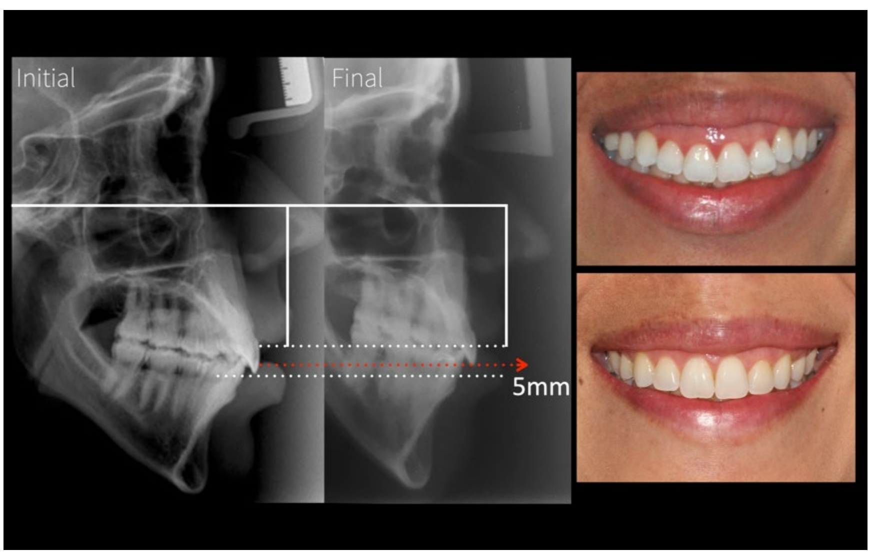

Figure 6. Initial (A) and final (B) cephalometric radiographs depicting the amount of incisor intrusion (5mm). Initial (C) and final (D) smile close ups shows the improvement of smile esthetics after incisor intrusion, reducing excessive smile curvature, as well as gingival display.

Conversely, in patients with minimum upper incisor display, deep bite correction cannot be performed by intruding upper incisors. In this situation, the deep bite should generally be addressed by intruding lower incisors. Figures 7 through 11 illustrate this clinical condition, a hypodivergent patient with good lip seal, a flat smile line, a Class I occlusion with multiple diastemas, and a deep bite (Figure 7).

Figure 7. Initial facial (A-C) and intra oral (D-H) photographs of a young female adult patient presenting a hypodivergent facial profile, good lip seal, and a shallow smile with reduced dental exposure. She had a Class I malocclusion with generalized spacing and a deep bite.

Upper incisor display at rest was only 1 mm, therefore, her treatment plan included a reverse curve of Spee in the lower arch (which would lead to lower incisor intrusion) followed by upper incisor extrusion, with the intention of improving smile esthetics (Figure 8). What seemed to be an easy case, was not, since an extra amount of lower incisor intrusion was needed to allow upper incisor extrusion, and at the same time correct the deep bite.

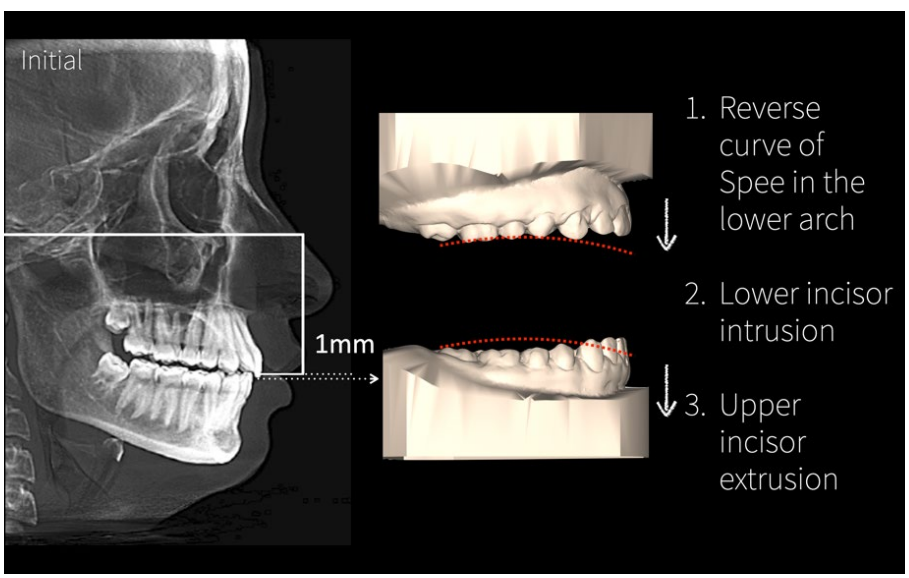

Figure 8. Cephalometric evaluation for upper incisor display at rest with 1mm (A). Due to the reduced incisor exposure (B), the treatment plan included a reverse curve of Spee in the lower arch, so that lower incisors would be intruded (C). Overcorrection of the deep bite was planned, so that upper incisors could be extruded to improve smile esthetics (arrows).

A reverse curve of Spee in the lower archwire proclined the lower incisors, leading to relative intrusion of these teeth. Lower premolars were extruded, and some bite opening was achieved. This created enough vertical space for upper incisor extrusion and improvement of incisor display (Figure 9). No facial changes were expected, but an improved smile line was achieved. An adequate final occlusion was reached and gingivoplasty was performed to correct tooth proportions (Figure 10).

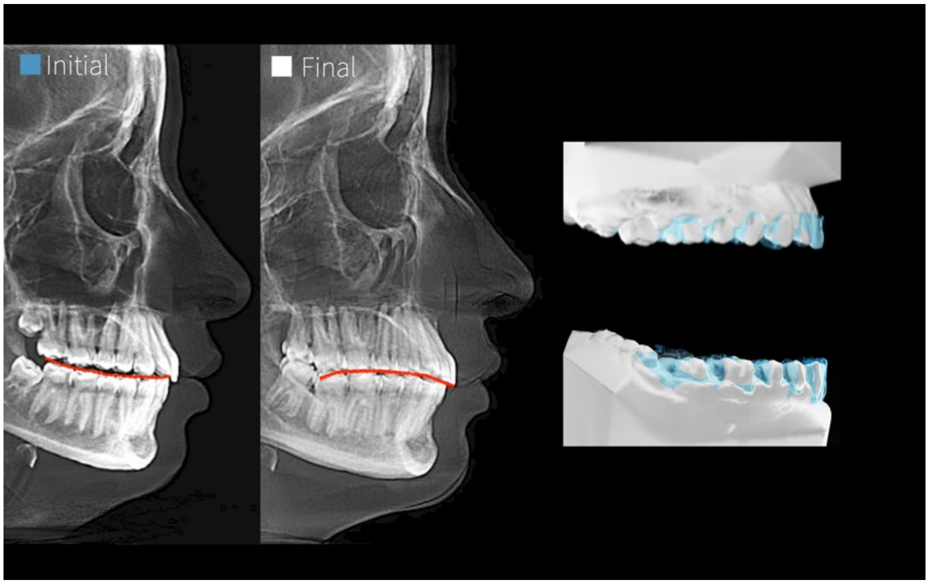

Figure 9. Initial (A) and final (B) cephalometric radiographs showing the changes in the occlusal plane created by reverse curve of Spee in the lower arch. (C) Superimposition of initial (white) and final (blue) digital models illustrates the amount of lower incisor proclination and relative intrusion, lower premolar extrusion and upper incisor extrusion.

Figure 10. Final facial (A-C) and intra oral (D-H) photographs presenting a harmonious facial profile, with passive lip seal and good smile line. A Class I malocclusion was maintained, with spaces closed and overbite corrected.

Figure 11. Final esthetic result, with a consonant smile line (A, B) and adequate upper incisor exposure at rest (C).

Incisor intrusion and posterior extrusion

Most deep bite cases will need a combination of anterior and posterior vertical tooth movements, since a deep curve of Spee is frequently present in the lower arch. When continuous wires are used, to level deep curves of Spee, there is a combination of premolar extrusion and incisor intrusion [19].

In cases where the vertical allocation of incisors does not permit enough intrusion to correct the deep bite, extrusion of the posterior segment will also be necessary, such as in the case presented in Figures 12 through 17. This young patient presented with a concave facial profile, consonant smile line, Class I malocclusion, deep bite, and retroclined upper and lower incisors.

Figure 12. Initial facial (A-C) and intra oral (D-F) photographs of a young female adult patient presenting a normodivergent and concave facial profile, with good lip seal, and a consonant smile with adequate dental exposure. A Class I malocclusion with significantly retroclined upper and lower incisors and a deep bite was present.

Upper incisor display at rest was 6mm, which allowed approximately 2mm of upper incisor intrusion. Care was taken not to flatten the smile line with incisor intrusion, since the patient presented with a consonant smile line, as can be seen in Figure 13.

Figure 13. Cephalometric evaluation for upper incisor display at rest (6mm), with consonant smile line.

Therefore, it was initially planned to level the upper arch, including second molars. The step between first and second molars opens the bite approximately 3 mm (20), which allows the placement of the lower arch appliance. The curve of Spee was levelled by continuous archwires and adequate torquing was performed to procline upper and lower incisors. This resulted in enough relative incisor intrusion in both arches to obtain an adequate final occlusal and esthetic result (Figures 14 and 15).

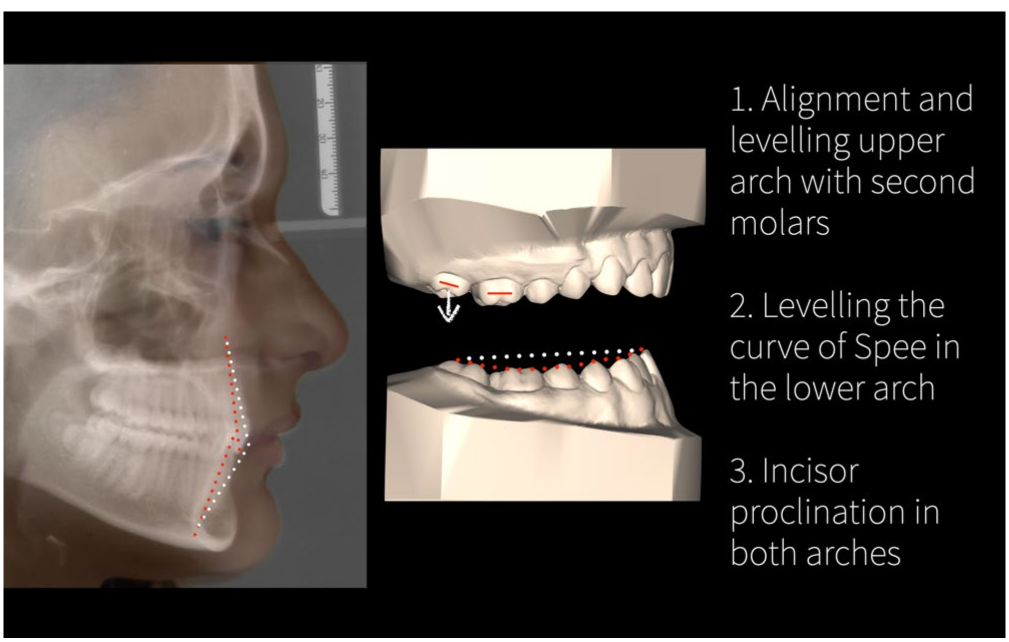

Figure 14. Overbite correction was planned by extrusion of upper second molars, followed by levelling of the curve of Spee in the lower arch and relative incisor intrusion by proclination in both arches.

Figure 15. Final facial (A-C) and intra oral (D-F) photographs presenting a pleasant facial profile, with passive lip seal and good smile line. A Class I malocclusion was achieved, with adequate incisor inclination and corrected overbite.

When comparing initial and final digital model superimpositions (Figure 16), extrusion of the upper second molar can be noticed, which caused bite opening and therefore space to place the lower brackets. Lower posterior teeth were also extruded, and upper and lower incisors were proclined, which promoted relative intrusion of these teeth. The deep bite was corrected and a significant proclination of upper and lower incisor was achieved. Upper incisor proclination was sufficient to perform relative intrusion of upper incisors without flattening the smile line and maintaining adequate upper incisor display at rest (Figure 17).

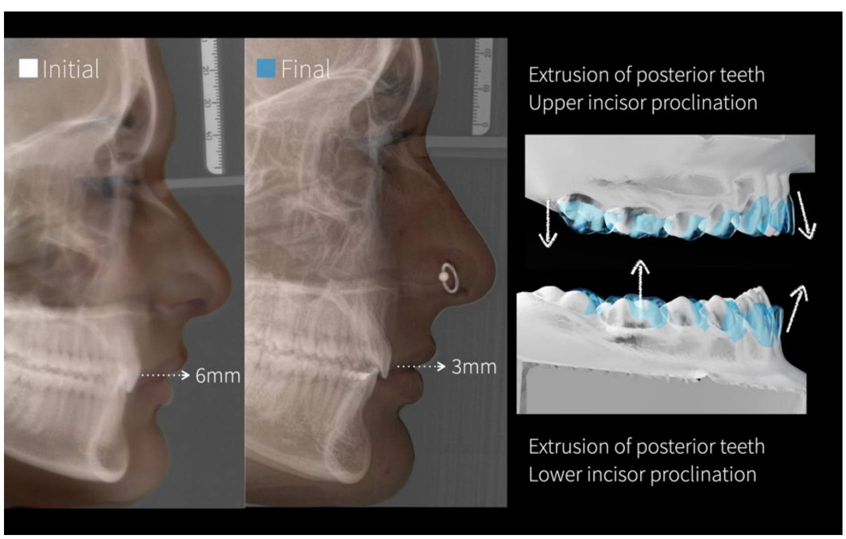

Figure 16. Initial (A) and final (B) cephalometric radiographs showing the changes in upper incisor display at rest (3mm). Superimposition (C) of initial (white) and final (blue) digital models illustrates the amount of extrusion of posterior teeth in both arches, especially the upper second molars, and proclination of upper and lower incisors. Posterior extrusion and incisor proclination were the main mechanisms used for overbite correction.

Figure 17. Final smile esthetic result with good upper incisor inclination (A), consonant smile line (B) and upper incisor exposure at rest greater than 2mm (C).

Many other combinations of treatment possibilities can be devised based on diagnosis. Due to the multifactorial nature of malocclusions, different combinations of features can be the origin of deep bites. Therefore, customized treatment planning is necessary, always considering smile esthetics, especially the vertical allocation of anterior teeth. The case shown in Figures 18 through 24 demonstrates a clinical situation in which a deep bite presented good position of upper incisors, and all the deep bite correction was achieved by moving the upper posterior teeth and lower incisors. This young man presented with a straight facial profile and good gingival exposure when smiling. He was a Class II division 2, with a small overjet and a deep bite (Figure 18). Despite the gingival exposure, upper incisor display was good, and upper incisor intrusion was not desired (Figure 19).

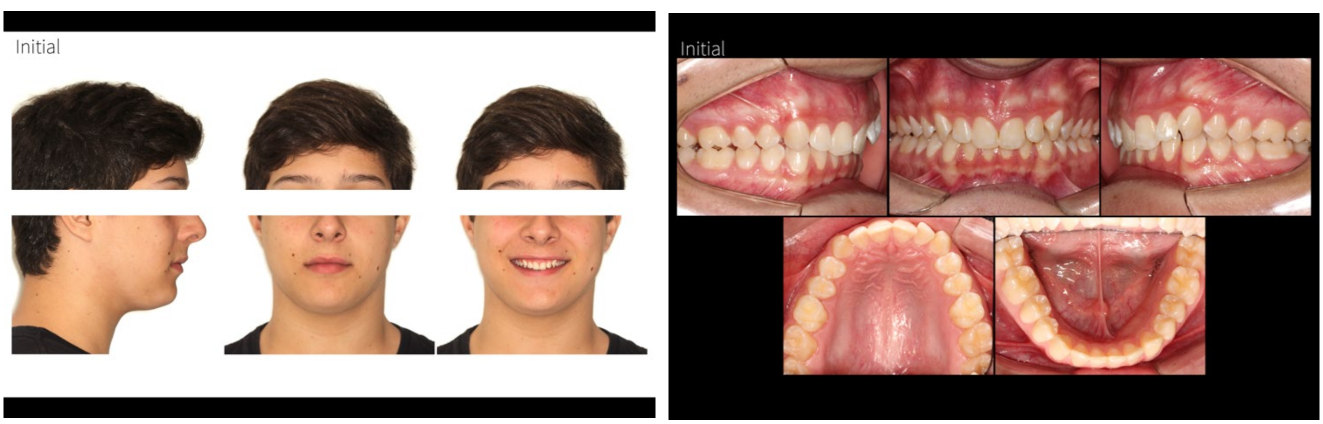

Figure 18. Initial facial (A-C) and intra oral (D-H) photographs of a young adult male patient presenting a normodivergent and straight facial profile, with good lip seal, and a consonant smile with minor gingival exposure. He had a Class II, division 2 malocclusion with normal overjet and a deep bite.

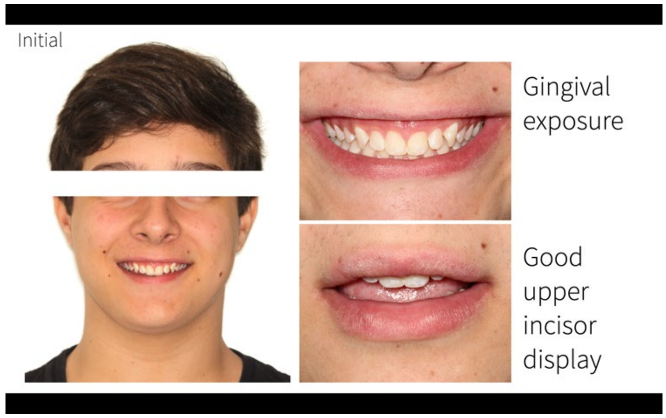

Figure 19. Esthetic analysis with smile (A), smile close up (B) and upper incisor exposure (C) photographs, showing a consonant smile line, with gingival exposure and good upper incisor display at rest.

Since the patient had good upper incisor inclination and vertical position, these teeth should be minimally changed. For Class II correction, distalization of the upper posterior teeth was needed without changing upper incisor position. Finally, to correct the deep bite, extrusion of upper posterior teeth during leveling, extrusion of lower posterior teeth, and lower incisor intrusion and proclination were required (Figure 20).

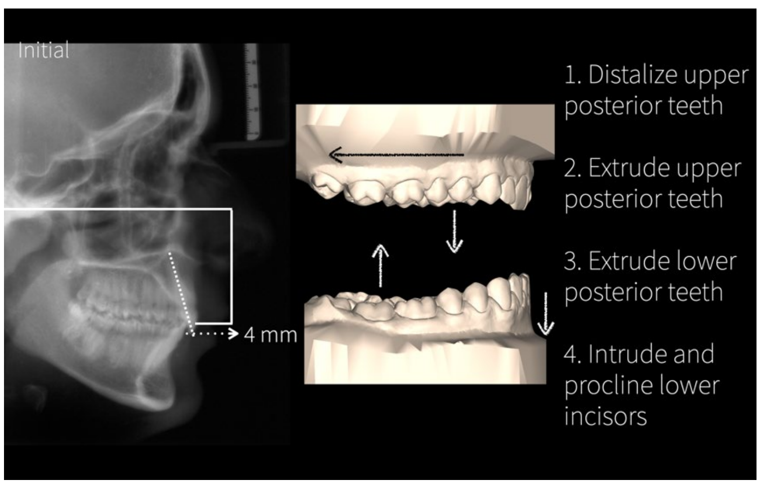

Figure 20. Cephalometric evaluation for upper incisor display at rest with 4mm and good upper incisor inclination (A). Due to the adequate incisor exposure and inclination, the treatment plan included distalization of the upper arch for Class II correction, extrusion of upper and lower posterior teeth, and intrusion of lower anterior teeth for overbite correction (B).

A first phase of treatment was performed with a Carriere® Motion 3D™ appliance to achieve Class II correction (Figure 21). When comparing digital models at the beginning of treatment and after the Carriere appliance (Figure 22), we can notice the distalization of upper posterior teeth as well as extrusion. The lower arch moved mesially promoting relative intrusion of lower incisors. The patient was then treated with aligners to an adequate final occlusion, with a harmonious facial result and a pleasing smile line, despite the gingival exposure (Figure 23). Upper incisor display at rest was maintained and levelling of the upper arch happened by posterior extrusion, which helped correct the deep bite associated with lower incisor position (Figure 24).

Figure 21. Intraoral photographs of Carriere appliance in use with Class II elastics (A-C) and occlusal result (D-F) after 9 months of use with complete sagittal correction. Improvement of upper arch levelling is observed due to extrusion of the upper posterior segment.

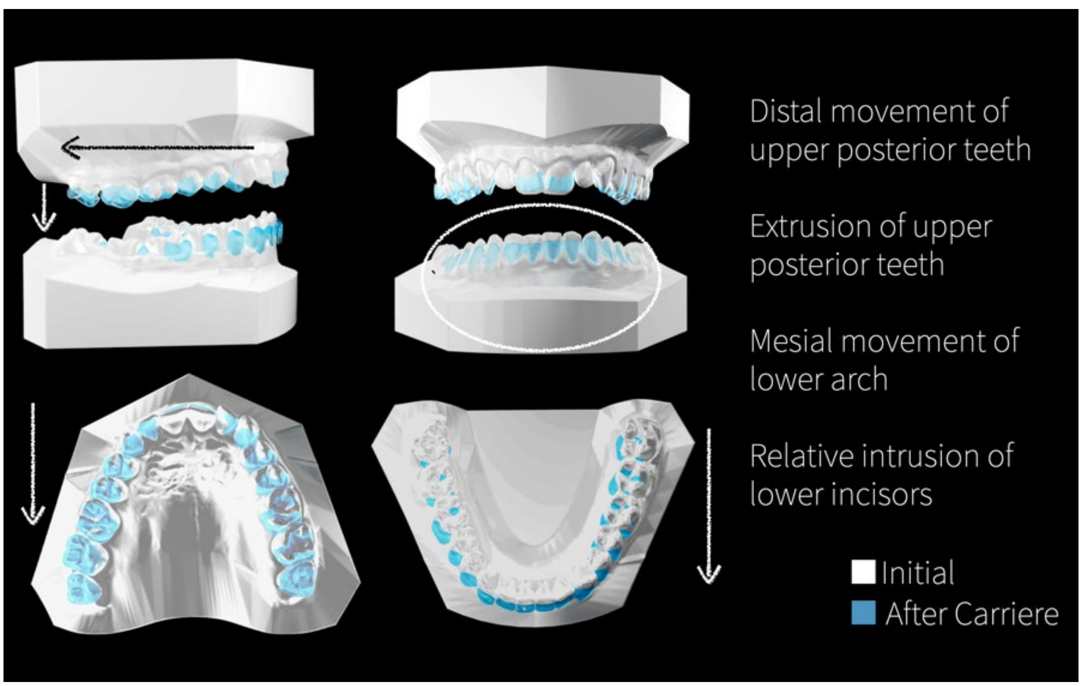

Figure 22. Superimposition of initial (white) and after Carriere appliance (blue) digital models illustrates the amount of distal movement of the upper arch, extrusion of the upper posterior teeth, mesial movement of the lower arch and relative intrusion of the lower incisors.

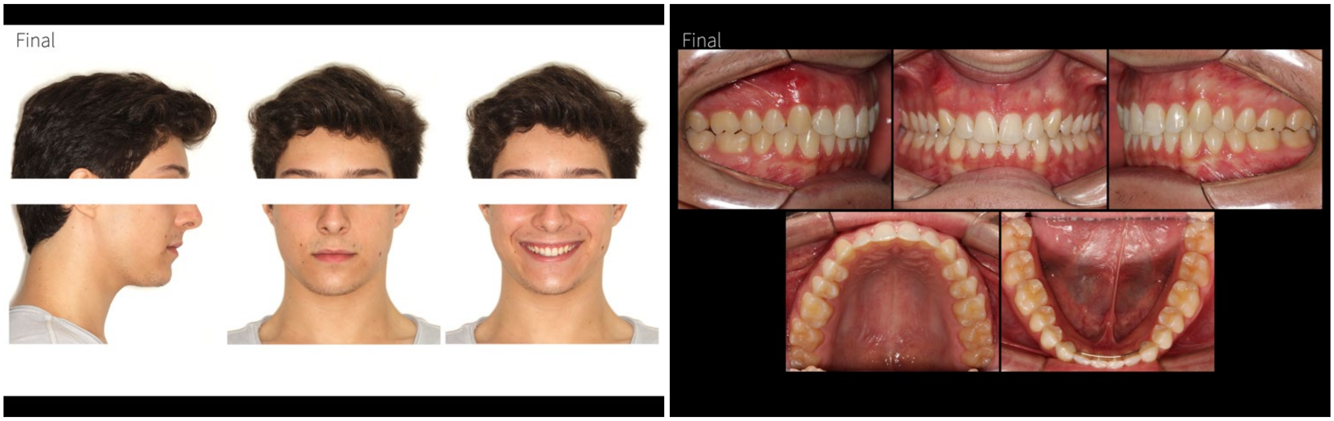

Figure 23. Final facial (A-C) and intraoral (D-H) photographs presenting a good facial profile, with passive lip seal, adequate smile line and gingival exposure. A Class I malocclusion was achieved, with proper incisor inclination and corrected overbite.

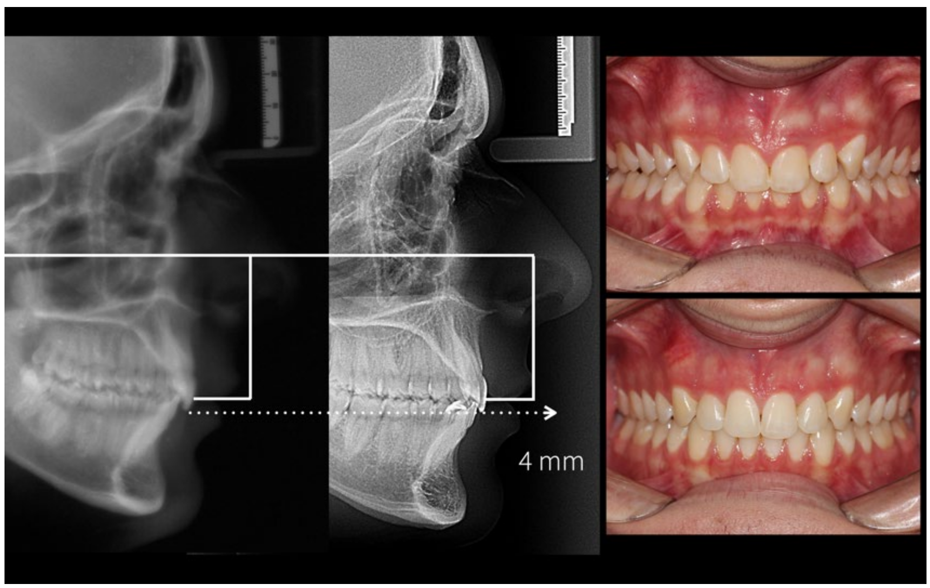

Figure 24. Initial (A) and final (B) cephalometric radiographs showing no significant changes in upper incisor display at rest (4mm). Overbite correction was obtained by extrusion of posterior teeth and relative intrusion of lower incisors, when levelling the curve of Spee (C-D).

What these two previous cases have in common is that part of the overbite correction was obtained by levelling the curve of Spee. When levelling the curve of Spee, space is necessary and lower incisor proclination is unavoidable, no matter what technique you use [19, 21]. Bite raising mechanisms can also be useful in treating overbites, but there is no defined protocol in the literature on where to place bite raising devices [10]. Some orthodontists believe that high angle cases would benefit from posterior bite raising to avoid extrusion of posterior teeth, but this seems paradoxical, since deep bite correction is dependent on extrusion of the midsegments of the lower arch [21] On the other hand, low angle cases should have anterior bite raisers to enhance posterior extrusion. Bite raisers placed in the anterior region would be preferable, mostly due to the deprograming effect that it has in the musculature, but the orthodontic literature is inconclusive at this time.

Posterior underdevelopment

Another contributing factor for deep bites is the vertical underdevelopment of the posterior region. These patients have a short anterior facial height which leads to inverted and pouched lips, as if there was excessive soft tissue in the anterior region [22]. The patient in Figures 25 through 29 was in the final phase of mixed dentition, when a deep bite is a relatively normal physiologic trait. Nevertheless, the reduced anterior facial height associated with reduced incisor display at rest, as well as in smiling, could be improved to a more harmonious facial appearance (Figures 25 and 26).

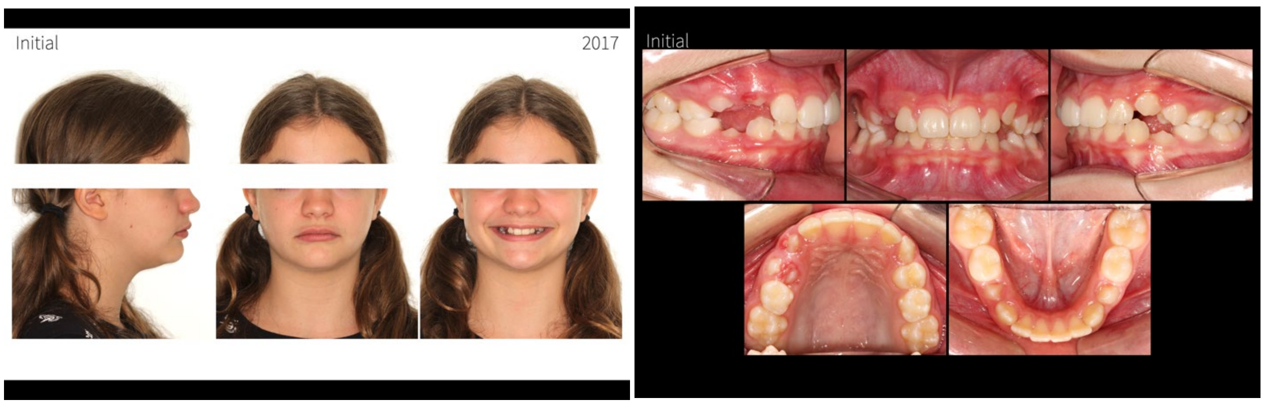

Figure 25. Initial facial (A-C) and intra oral (D-H) photographs of a young girl presenting with a hypodivergent and straight facial profile, excessive lip seal, and a consonant smile with minor dental exposure. She had a Class I occlusion in the mixed dentition with a deep bite.

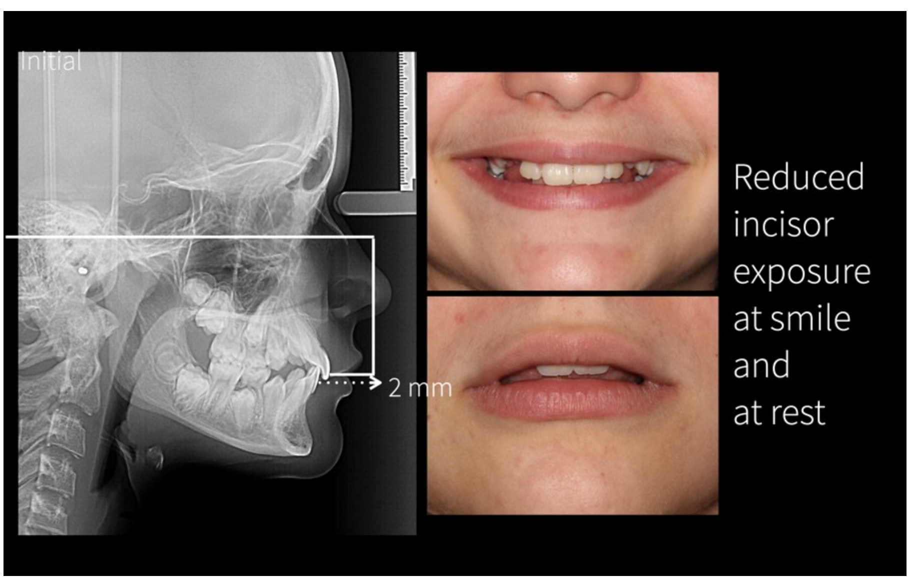

Figure 26. Esthetic analysis with cephalometric radiograph (A), smile close up (B) and upper incisor exposure photographs (C), showing a consonant smile line, with minimal upper incisor display at rest.

With the intention of promoting posterior disocclusion and spontaneous extrusion of posterior teeth, a removable bite plate was installed. Facial differences can be noticed immediately, with improvement of the lower anterior facial height (Figure 27). This bite plate was used throughout the final phase of mixed dentition for 12 months. A second bite plate was needed one year later and used until full eruption of permanent second molars (Figure 28). Final results show a good facial profile, adequate lip seal, and improved upper incisor exposure. The shape of the lips at rest also improved considerably, as well as the increase in lower facial height.

Figure 27. (A-C) Facial photographs immediately after bite plate placement. An increase in anterior facial height is seen due to posterior disocclusion.

Figure 28. Intraoral photographs immediately after bite plate was installed (A-C), 12 months later (D-F), after a second bite raise when treatment was discontinued (G-I), and 2 years follow up with no treatment (J-L). Note the improvement of the deep bite due to posterior vertical alveolar growth.

Figure 29. Facial photographs before bite plate was installed (A,D,G), 24 months later, when bite plate use was discontinued (B,E,H), and 24 months later with no treatment (C,F,I). Note the increase in lower facial third and the improvement of the lip seal, as well as the dental exposure.

Despite the successful results of this treatment, some questions arise when analyzing these final results. Was the increase in vertical alveolar growth caused using the bite plate? To the best of our knowledge there are no studies on this subject, but it has been observed that patients with anterior rotation growth pattern tend to worsen [23]. When is the ideal time to begin treatment? Some evidence has shown there is no difference in early versus late treatment for deep bite [24], but overbite was the only outcome evaluated, rather than incisor display and lower anterior facial height. And finally, what is the limit for alveolar growth by bite opening? That is another factor still to be answered in future clinical research.

Stability of vertical tooth movement

Even though successful results were obtained in the previously presented cases, how will they behave in the long term? Regarding stability of overbite treatment, not much is known. The systematic review published by Huang and collaborators [25] is the best compilation of information on the subject. The variation of deep bite relapse is very large, ranging from 0 to 82%, but in what areas relapse occurs is not known. Is it in the anterior region or the posterior region? When we extrude posterior teeth, we change occlusal vertical dimension, a concept also described by prosthetic dentistry which can be very useful in deep bite correction. This will be illustrated in the next section.

Occlusal vertical dimension and deep bite correction

Occlusal Vertical Dimension (OVD) is the vertical relationship between maxilla and mandible when the teeth are occluded in maximum intercuspation. Interocclusal distance is the difference between rest vertical dimension and OVD, also known as freeway space. Rest vertical dimension is the distance from nose to chin when the mandible is in rest position [26]. These concepts are extremely useful in cases with loss of occlusal vertical dimension with deep bites.

Loss of occlusal vertical dimension

Loss of occlusal vertical dimension occurs when there is vertical reduction of occlusal tooth surface, leading to a counterclockwise rotation of the mandible and bite closure. It can basically happen in two situations by carious and non-carious tooth destruction [27].

Carious tooth destruction

Clinical situations with severe carious destruction of posterior teeth can be particularly challenging, as demonstrated in Figures 30 through 34. A 60-year-old woman who had been treated orthodontically in the past for a Class II with premolar extractions was having difficulty restoring her posterior teeth. The patient presented with a Class II malocclusion, severe deep bite, a large overjet, a severe hyperdivergent facial pattern with chin prosthesis, and good upper incisor display. The deep bite was associated with an increased curve of Spee.

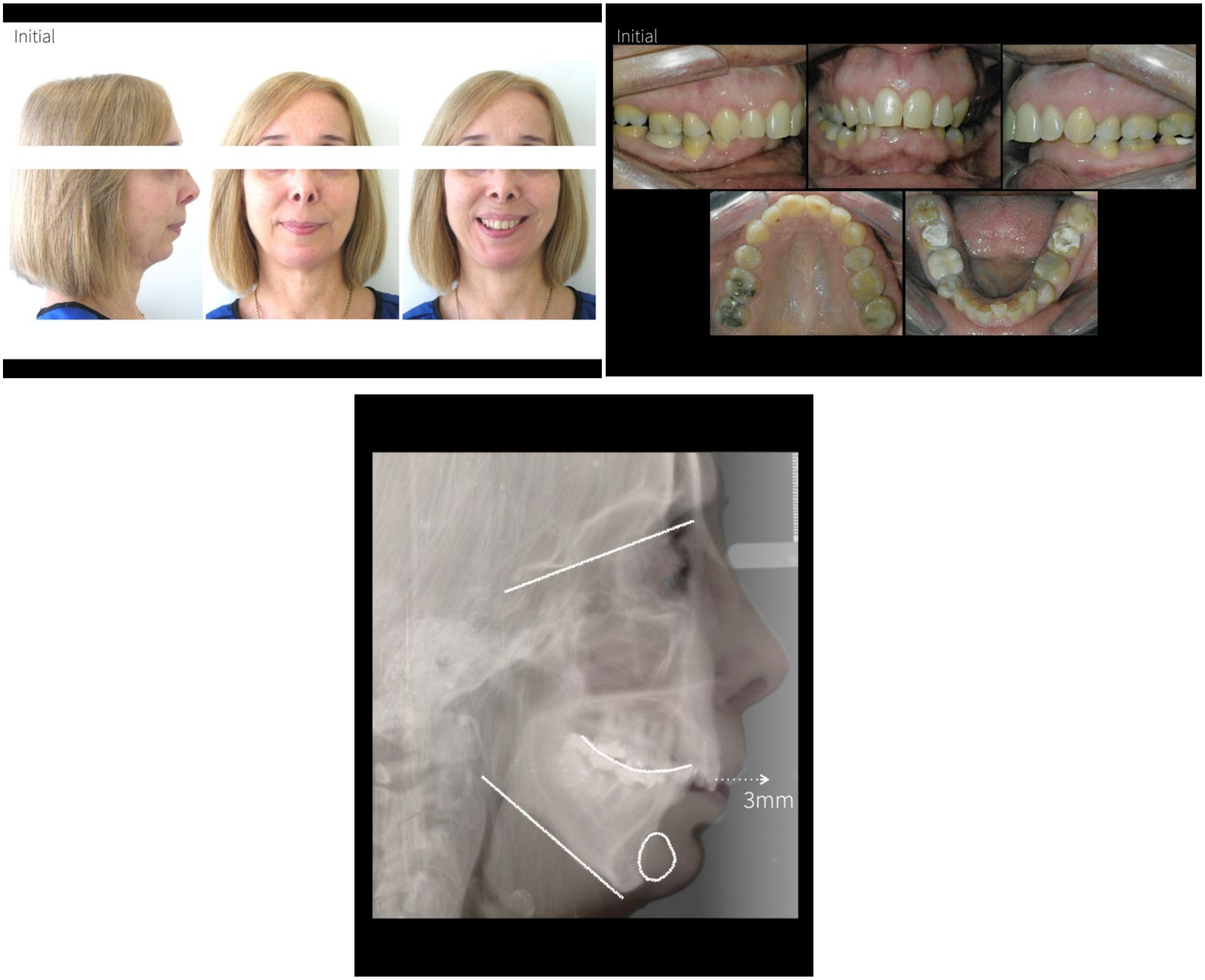

Figure 30. Initial facial (A-C) and intra oral (D-H) photographs of a 60-year-old woman presenting with a hyperdivergent facial profile, adequate lip seal, and a consonant smile with adequate dental exposure. She had a Class II, division 1 malocclusion with a large overjet and a deep bite. Cephalometric radiograph (I) shows a high mandibular plane angle, a chin prosthesis, a very deep curve of Spee, and upper incisor exposure at rest of 3mm.

During the consultation, she complained of a persistent lesion that had not healed since the extraction of her lower third molars three months ago. Due to treatment for osteoporosis with sodium risendronate, there was a suspicion of osteonecrosis. It was confirmed by the oral pathologist and treated with hydrogen peroxide rinses and medication until complete healing occurred.

Osteonecrosis is a medication-related condition of the jaws that can occur with Intravenous or oral bisphosphonates or Receptor activator of nuclear factor-kB (RANK) ligand inhibitor (denosumab), which are generally used for osteoporosis or bone cancer. Treatment is usually conservative with hydrogen peroxide rinses to debride dead oral cells and a suspension of osteoporosis medication. Orthodontic treatment has a small risk of triggering osteonecrosis, but tooth movement may be slower in patients taking antiresorptive drugs. Most importantly, dentoalveolar surgery at any level is considered a major factor for developing osteonecrosis and should be avoided [28, 29].

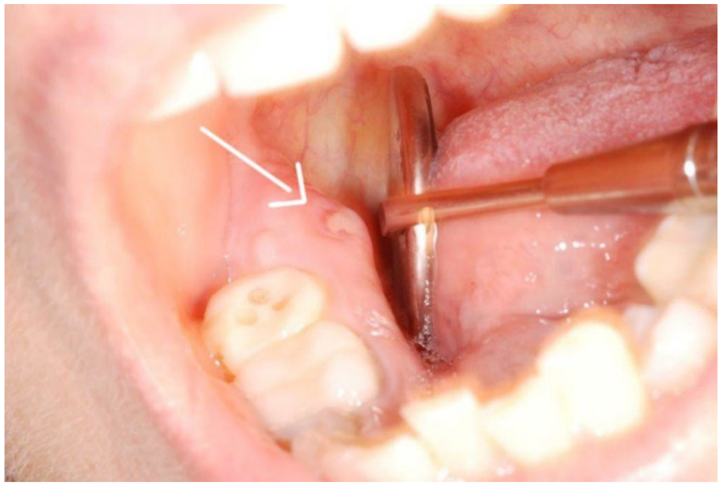

Figure 31. Persistent lesion in the lower right retromolar area that occurred after third molar removal with lack of healing.

Therefore, dentoalveolar surgery was absolutely contraindicated in this patient, despite her facial characteristics. Since no invasive procedure could be used, her treatment began with 5 mm of prosthetic raising of the vertical occlusal dimension on both sides (Figure 32), followed by regular alignment and levelling with fixed orthodontic appliances. Obviously, no major facial improvement was expected, but an adequate occlusion was achieved. Comparing initial and final records, the prosthetic increase in OVD led to a clockwise rotation of the mandible. Lower incisors were significantly proclined due to the levelling of the curve of Spee, which led to relative intrusion of lower incisors. But what do we know about the long-term effects of such treatments?

Figure 32. Posterior occlusal bite raising to restore occlusal caries and gain occlusal vertical dimension (A, C). A significant improvement in deep bite can be observed (B).

Figure 33. Final facial (A-C) and intraoral (D-H) photographs presenting a hyperdivergent and retrognathic facial profile, with passive lip seal and consonant smile line with significant gingival exposure. A Class I malocclusion was achieved, with adequate incisor inclination and corrected overbite.

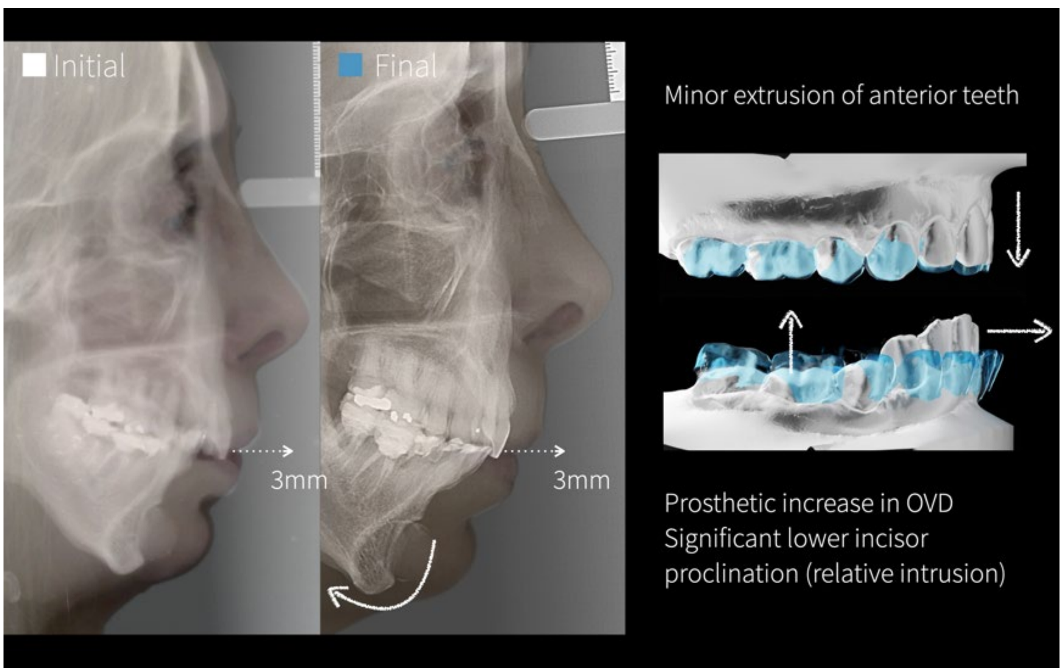

Figure 34. Initial (A) and final (B) cephalometric radiographs showing no changes in upper incisor display at rest (3mm). Superimposition (C) of initial (white) and final (blue) digital models illustrates the amount of posterior prosthetic build up in the lower arch, significant lower incisor proclination, and therefore relative intrusion and minor changes in the upper arch. Posterior build ups and incisor proclination were the main mechanisms for overbite correction.

There is very little evidence evaluating long term results of prosthetic build ups. The best piece of available evidence using modern diagnostic methods was a preliminary study published in 2019 that evaluated 6 patients with CBCTs treated with bite raising who were followed for 2 years. The results demonstrated that the increased OVD did not relapse to baseline and that relapse was mostly in the occlusal dental height rather than in the alveolar process heights [30].

Non-carious tooth destruction

Non-carious tooth destruction can happen in four different ways: abrasion, attrition, erosion and abfraction. Attrition and erosion are the two processes that can reduce the occlusal surface of posterior teeth and therefore cause loss of occlusal vertical dimension and consequently bite closure [27].

Perimolysis is the consequence of gastric acid flowing into the mouth from the dorsum of the tongue, onto the palatal surfaces of the upper teeth, the occlusal surfaces of both arches, and the buccal surface of lower teeth (Figure 35). Treatment demands a multidisciplinary approach with immediate referral to a gastroenterologist and dental treatment can only be started after gastric reflux has ceased [31], as shown in the case in Figures 36 through 41.

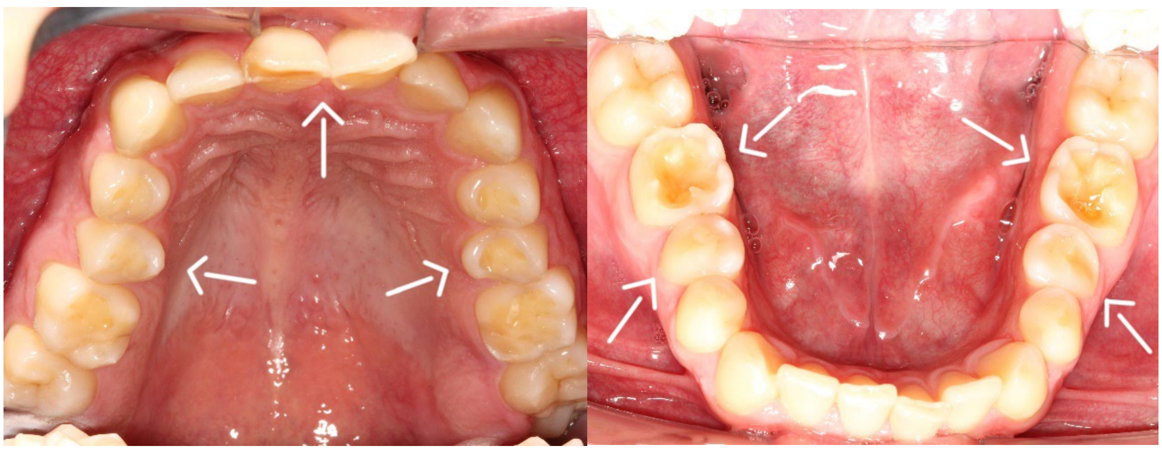

Figure 35. Arrows show erosion areas on the path of regurgitated hydrochloric acid over the dorsum of the tongue that characterize perimolysis: palatal surfaces of the maxillary teeth (A), occlusal surfaces of both arches (A-B) and buccal surfaces of posterior mandibular teeth (B).

This patient presented with a Class II malocclusion and deep bite due to significant occlusal erosion of his teeth. Erosion lesions were present on the lingual and occlusal surfaces of upper teeth and on the occlusal and buccal surfaces of lower teeth, which characterizes perimolysis. After referral to the gastroenterologist, treatment began with temporary composite restorations of the posterior teeth. This promoted bite raising, which allowed bracket placement in the lower arch, but worsened the sagittal relationship due to the counterclockwise mandibular rotation (Figure 37).

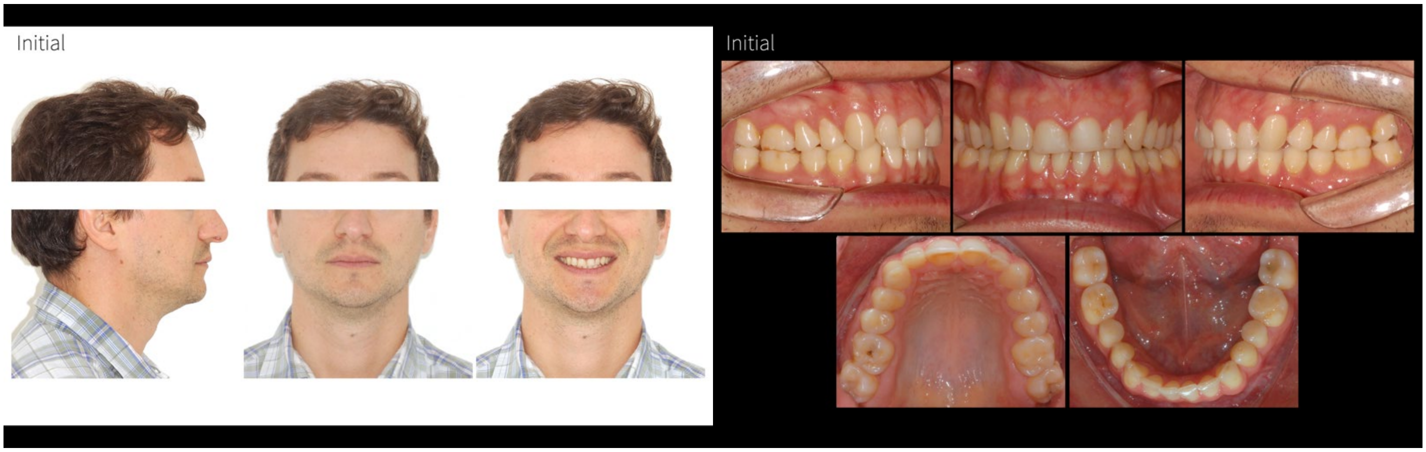

Figure 36. Initial facial (A-C) and intra oral (D-H) photographs of a young male adult presenting a normodivergent and straight facial profile, with adequate lip seal, and a consonant smile with adequate dental exposure. He had a Class II, division 2 malocclusion with a small overjet and a deep bite. Occlusal anterior and posterior wear can be seen, characterizing perimolysis due to silent gastric reflux.

Figure 37. Posterior occlusal bite raising to restore occlusal wear and gain occlusal vertical dimension. A significant improvement in deep bite can be observed (B), as well as worsening of the sagittal relationship due to clockwise mandibular rotation (A,C).

The orthodontic treatment plan included levelling the upper gingival margins by upper incisor intrusion, despite the poor upper incisor display and short upper incisor crowns (Figure 38). This created vertical occlusal anterior dimension for upper incisor restorations and improvement of the proportions of clinical crowns, which also improved upper incisor display with minimal invasiveness. A straight facial profile was maintained, with good lip seal and a significantly improved smile line. This was due to both the posterior bite raising and upper incisor intrusion. Posterior restorations helped in gaining anterior vertical dimension and lower incisor intrusion (Figure 39).

Figure 38. Esthetic analysis with cephalometric radiograph, smile close up and upper incisor exposure photographs, showing a flat smile line, with minor upper incisor display at rest (1mm), but small upper incisor crowns due to occlusal wear (A). Therefore, upper incisor intrusion was needed to gain vertical anterior dimension for incisor restoration and improvement of dental proportions (B).

Figure 39. Final facial (A-C) and intraoral (D-H) photographs presenting a normodivergent and straight facial profile, with passive lip seal and consonant smile line with adequate dental exposure. A Class I malocclusion was achieved, with adequate overbite, as well as improvement of tooth proportions.

In this case, overbite correction was obtained by recovering occlusal vertical dimension by restoring the eroded occlusal surfaces of posterior teeth. This led to bite opening with mandibular clockwise rotation. Upper and lower incisor intrusion provided vertical dimension to restore eroded incisors and improve smile esthetics, as well as upper incisor display at rest from 1 to 4mm (Figures 40 and 41).

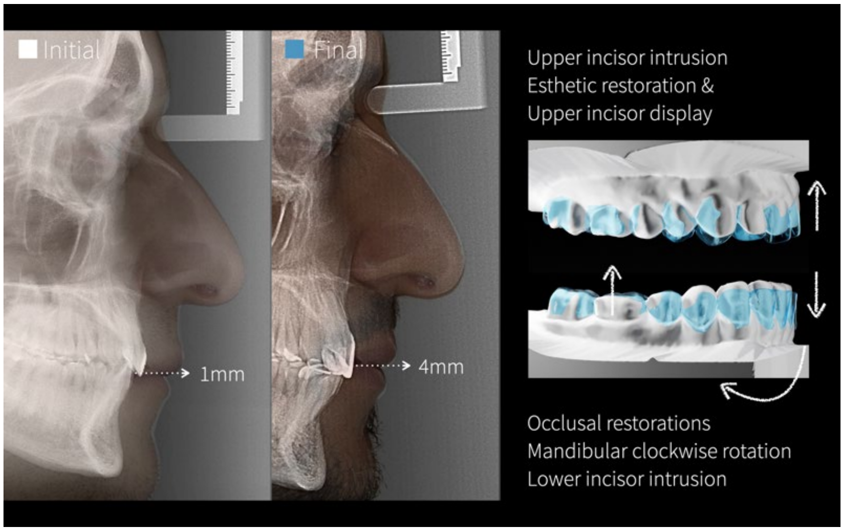

Figure 40. Initial (A) and final (B) cephalometric radiographs showing improvement in upper incisor display at rest from 1 to 4mm. Superimposition (C) of initial (white) and final (blue) digital models illustrates the amount of posterior prosthetic build ups in both arches, which led to a clockwise rotation of the mandible and bite opening. Upper and lower incisor intrusion was needed to create vertical space for restorations and improvement of incisor proportions.

Figure 41. Initial (A, B) and final (C, D) incisor display and smile esthetics.

CONCLUSIONS

Orthodontic tooth movement for overbite correction includes incisor intrusion, molar and premolar extrusion, or a combination of both. When anterior teeth are intruded, smile esthetics will be impacted, most especially upper incisor display at rest. When posterior teeth are extruded, function will be affected, i.e., occlusal vertical dimension. The vertical position of upper incisors will be a paramount diagnostic factor to determine if they can be intruded. If not, other ways of correcting the deep bite should be considered, such as extruding posterior teeth and opening the bite by clockwise rotation of the mandible.

In cases with posterior underdevelopment, posterior eruption is indicated by using bite raisers. Nevertheless, not much is known about this treatment modality in growing patients and how much dento-alveolar vertical increase is really obtained. On the other hand, patients with loss of occlusal vertical dimension due to occlusal wear or erosion should be restored before orthodontic treatment. Despite prosthetic increase in OVD being traditionally performed in dentistry, not much is known regarding muscular adaptability and stability [32]. Even though the level of evidence is low, patients must be treated to the best standard of care. The changes we cause by intruding or extruding molars are not yet clear, and need to be investigated, especially regarding muscular adaptability and stability, for more predictable and long-lasting results.

REFERENCES

1. Moyers RE. Handbook of Orthodontics for the Student and General Practitioner, 3d ed. Chicago: Year Book Medical Publishers; 1973.
2. Broderson SP. Anterior guidance--the key to successful occlusal treatment. J Prosthet Dent 1978; 39(4):396-400.
3. Alhammadi MS, Halboub E, Fayed MS, Labib A, El-Saaidi C. Global distribution of malocclusion traits: a systematic review. Dental Press J Orthod 2006; 23(6):40.e1-40.e10.
4. Bergersen EO. A longitudinal study of anterior vertical overbite from eight to twenty years of age. Angle Orthod 1988; 58(3):237-256.
5. El-Dawlatly MM, Fayed MMS, Mostafa YA. Deep overbite malocclusion: analysis of the underlying components. Am J Orthod Dentofacial Orthop 2012;142(4):473-480.
6. Engel G, Cornforth G, Damerell JM, Gordon J, Levy P, McAlpine J et al. Treatment of deep-bite cases. Am J Orthod 1980; 77(1):1-13.
7. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977; 72(1):1-22.
8. Korn M, Melsen B. Early treatment with a maxillary lip bumper-bite plateau combination. Angle Orthod 2008; 78(5):838-846.
9. Kravitz N, Jorgensen G, Frey S, Cope J. Resin bite turbos. J Clin Orthod 2018; 52(9):456-461.
10. Singh G, Gupta H, Rathi A, Bisht D, Goyal V, Singh RK, Dhawan S. The use of bite raisers in orthodontic treatment - a review of literature. Acta Sci Dent Sci 2021; 5:219-228.
11. Greco M, Rombolà A. Precision bite ramps and aligners: an elective choice for deep bite treatment. J Orthod 2022; 49(2):213-220.
12. Lombardi RE. The principles of visual perception and their clinical application to denture esthetics. J Prosthet Dent 1973; 29(4):358-382.
13. Arnett GW, Jelic JS, Kim J, Cummings DR, Beress A, Worley JR M et al. Soft tissue cephalometric analysis: diagnosis and treatment planning of dentofacial deformity. Am J Orthod Dentofacial Orthop 1999; 116(3):239-253.
14. Câmara CA, Martins RP. Functional Aesthetic Occlusal Plane (FAOP). Dental Press J Orthod 2016; 21(4):114-125.
15. Kokich VG. Esthetics: the orthodontic-periodontic restorative connection. Semin Orthod 1996; 2(1):21-30.
16. Zachrisson BU. Facial esthetics: guide to tooth positioning and maxillary incisor display. World J Orthod 2007; 8(3):308-314.
17. Ackerman MB, Ackerman JL. Smile analysis and design in the digital era. J Clin Orthod 2002; 36(4):221-236.

18. Sarver DM, Ackerman MB. Dynamic smile visualization and quantification: part 1. Evolution of the concept and dynamic records for smile capture. Am J Orthod Dentofacial Orthop 2003; 124(1):4-12.
19. Bernstein RL, Preston CB, Lampasso J. Leveling the curve of Spee with a continuous archwire technique: a long term cephalometric study. Am J Orthod Dentofacial Orthop 2007; 131(3):363-371.
20. Andreasen GF. The importance of banding the second permanent molars. Am J Orthod Dentofacial Orthop 1986; 90(6):501-502.
21. Al-Zoubi EM, Al-Nimri KS. A comparative study between the effect of reverse curve of Spee archwires and anterior bite turbos in the treatment of deep overbite cases: A randomized clinical trial. Angle Orthod 2022; 92(1):36-44.
22. Nielsen IL. Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment. Angle Orthod 1991; 61(4):247-260.
23. Björk A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. J Dent Res 1963; 42(1)Pt 2:400-411.
24. Baccetti T, Franchi L, Giuntini V, Masucci C, Vangelisti A, Defraia E. Early vs late orthodontic treatment of deepbite: a prospective clinical trial in growing subjects. Am J Orthod Dentofacial Orthop 2012; 142(1):75-82.
25. Huang GJ, Bates SB, Ehlert AA, Whiting DP, Chen SSH, Bollen AM. Stability of deep-bite correction: a systematic review. J World Fed Orthod 2012; 1(3):e89-e86.
26. Fayz F, Eslami A. Determination of occlusal vertical dimension: a literature review. J Prosthet Dent. 1988; 59(3):321-323.
27. Hanif A, Rashid H, Nasim M. Tooth surface loss revisited: classification, etiology, and management. J Res Dent 2015; 3:37-43.
28. Ruggiero SL, Dodson TB, Fantasia J, Goodday R, Aghaloo T, Mehrotra B, O'Ryan F. American Association of Oral and Maxillofacial Surgeons position paper on medication-related osteonecrosis of the jaw – 2014 update. J Oral Maxillofac Surg 2014; 72(10):1938-1956.
29. Woolley J, Akintola O, Yates J, Calasans-Maia MD, Calasans-Maia JA, Kocherhina I, Sacco R. The risk of osteonecrosis of the jaw and adverse outcomes in patients using antiresorptive drugs undergoing orthodontic treatment: a systematic review. Heliyon 2021; 7(1):e05914.
30. Liu C, Huang D, Zhou L, Liu G, Wang Y, Jiang T. Two-year observation of the occlusal vertical dimension after bite raising via cone-beam computerized tomography: A preliminary study. Sci Rep 2019; 9(1):3509.
31. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci 1996; 104(2 Pt 2):151-155.
32. Goldstein G, Goodacre C, MacGregor K. Occlusal vertical dimension: best evidence consensus statement. J Prosthodont 2021; 30(S1):12–19.

DEEP BITE TREATMENT WITH CLEAR ALIGNER TECHNIQUE

Sandra Khong Tai

ABSTRACT

Diagnosis is key to the treatment of any malocclusion. The biomechanics of deep bite correction would depend on the etiology of the deep bite. These would include correcting maxillary and mandibular incisor inclination where the incisors are retroclined, intruding maxillary incisors if they are hypererupted and levelling the curve of Spee with mandibular incisor intrusion or a combination of mandibular incisor intrusion and premolar extrusion. Innovations specifically designed for the treatment of deep bite malocclusions include pressure areas, optimized deep bite attachment, precision bite ramps and the power ridge feature. Adequate anchorage is required for levelling the curve of Spee in the lower arch and this means that attachments are required on the first and second premolars. If posterior extrusion has been requested, these attachments may be active in order to facilitate premolar extrusion. Staging the sequence of maxillary and mandibular incisor tooth movements to first procline, then intrude, and finally to retract is key to achieving clinical predictability in deep bite treatment. A review of the literature shows a difference between the amount of intrusion programmed into the digital treatment plan and the actual amount of intrusion achieved. Therefore, over-treatment of the deep bite malocclusion is recommended when designing the tooth movements in the digital treatment plan. The more severe the deep bite in the initial malocclusion, and the older the patient, the more over-treatment is recommended for the digital treatment plan. Finally, two clinical cases, one teen and one adult patient, are presented as case reports for deep bite treatment.

KEY WORDS: Aligners, Deep Bite, Intrusion, Invisalign, Staging

INTRODUCTION

The etiology of a deep bite malocclusion is multifactorial [1]. One key question in the diagnosis is whether the deep bite is skeletal or dental in origin. Oftentimes, a deep bite malocclusion is a combination of various factors both skeletal and dental. Skeletal patterns associated with deep bite malocclusion are brachyfacial, short face height patients. Dental factors associated with deep bite malocclusion are retroclined maxillary and mandibular incisors, hypererupted maxillary incisors, and a steep curve of Spee in the lower arch (Figure 1).

Therefore, the biomechanics of deep bite correction would depend on the etiology of the deep bite malocclusion [2]. These would include correcting maxillary and mandibular incisor inclination where the incisors are retroclined, intruding maxillary incisors if they are hypererupted and levelling the curve of Spee with mandibular incisor intrusion or a combination of mandibular incisor intrusion and premolar extrusion. Maxillary incisor intrusion would have the advantage of correcting an unesthetic smile with excessive gingival display. If the etiology of the deep bite has a skeletal component, then it may be desirable to increase the lower face height in brachyfacial patients with posterior extrusion. A very severe deep bite would require a combination of both anterior intrusion and posterior extrusion to resolve the malocclusion.

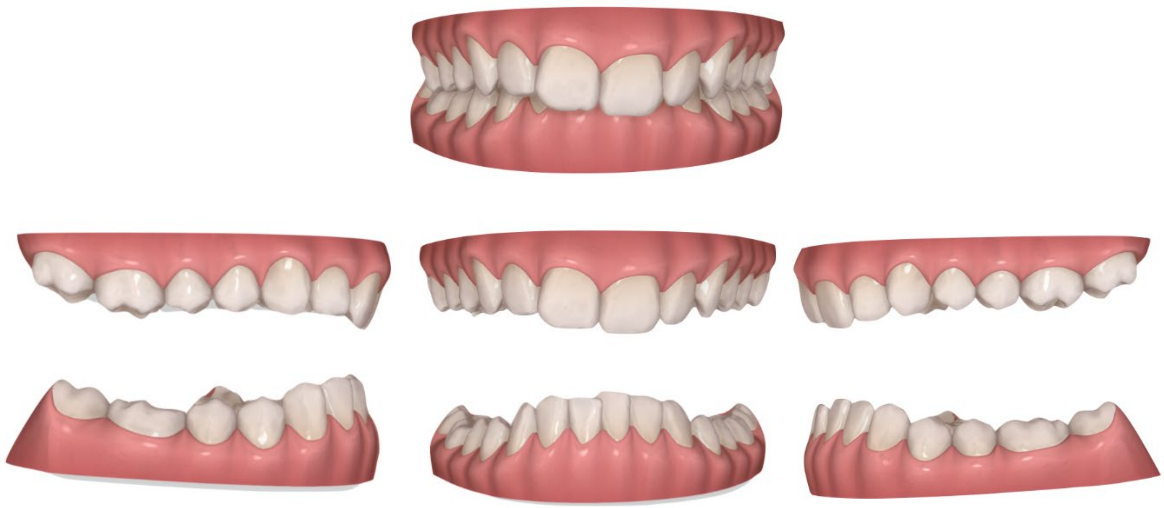

Figure 1. Dental factors associated with deep bite malocclusion: retroclined maxillary and mandibular incisors, hypererupted maxillary incisors, steep curve of Spee in lower arch.

Figure 2. Innovations for deep bite treatment. A) Pressure areas that redirect the intrusive force along the long axis of the tooth, B) Optimized deep bite attachments, C) Optimized support attachments on the maxillary lateral incisors to support intrusion of the maxillary central incisors, D) Precision bite ramps. (Reproduced with permission from Align Technology Inc).

CLEAR ALIGNER INNOVATIONS FOR DEEP BITE TREATMENT

The Invisalign® system by Align Technology [3] has several innovations specifically designed for the treatment of deep bite malocclusions [4] (Figure 2). These are:

Pressure areas

Pressure areas are concavities on the lingual surface of the maxillary and mandibular incisors and canines. They function to redirect the intrusive force along the long axis of the tooth for more efficient intrusion, where 0.5 mm or more of intrusion has been programmed into the digital software plan.

Optimized deep bite attachments

Optimized deep bite attachments are usually placed on first or second premolars, or both. Optimization refers to an activation built into the aligners, so if posterior extrusion is requested, when the aligners are fabricated, they exert an active force on the active surface of the attachments for extrusion and occlusal clearance is provided for the tooth to extrude into. In this case, the optimized deep bite attachments would be 'active'. These attachments may also be passive if no posterior extrusion was required. In that case, they would function as anchorage attachments to facilitate lower anterior intrusion.

Precision bite ramps

Precision bite ramps are protuberances built into the aligner on the palatal surface of upper incisors. They are dynamic, and their positions on the aligner change to maintain occlusion with the mandibular incisors as they align. Precision bite ramps function to disclude the posterior teeth as well, so the posterior teeth would be free to extrude if posterior extrusion was requested.

Conventional bite ramps

Conventional bite ramps may also be requested on the maxillary canines. There may be several scenarios where conventional bite ramps on the maxillary canines may be requested. If there is excessive overjet, the mandibular incisors may be not be occluding on the palatal surface of the maxillary incisors. In this case, bite ramps may be requested for the maxillary canines to assist in discluding the posterior occlusion to assist in deep bite correction. Since both the pressure areas and precision bite ramps occupy the palatal surface of the maxillary incisors, a clinician may choose to prioritize pressure areas for maxillary incisor intrusion, instead of precision bite ramps. In this scenario, conventional bite ramps may also be placed on the maxillary canines. Finally, there may be some patients with excellent buccal interdigitation prior to treatment and conventional bite ramps may be placed on the maxillary canines as a preventive measure to disclude the posterior occlusion where a patient may clench down on the aligners and inadvertently exert a posterior intrusive force creating a posterior open bite.

Optimized support attachments

These attachments may be placed on maxillary and mandibular lateral incisors as anchorage to support the intrusion of central incisors or canines. They are automatically placed by the software when there is 0.5 mm or more of intrusion programmed on either the adjacent canine or central incisor.

Power ridge feature

The power ridge feature is not specifically one of the innovations for deep bite treatment. It is designed for lingual root torque of maxillary and mandibular incisors and are useful for correcting incisor inclination where the incisors are retroclined.

A BRIEF REVIEW OF THE LITERATURE

A study by Khosravi et al. in 2017 [5], found that the Invisalign® appliance was effective for correcting deep overbites. The primary mechanism of action was proclination of the mandibular incisors, intrusion of the maxillary incisors and extrusion of mandibular molars. There was a 1.5 mm median opening of the overbite in deep bite patients. On average mandibular first and second molars extruded 0.5 mm. However, this study was carried out on patients before the innovations for deep bite correction was launched, meaning these patients were treated without precision bite ramps, pressure areas or optimized deep bite attachments.

Since intrusion is one of the primary mechanisms of deep bite correction, it is important to consider the clinical predictability of intrusion designed into the digital software plan. In a study by Grunheid et al. on non-extraction cases [6], it was found that anterior teeth were positioned more occlusally than predicted, and maxillary incisors had more lingual crown torque than predicted. Even though these differences were not large enough to be clinically relevant, the authors recommended building in the necessary compensations into the virtual treatment plan. Another study by Haouili [7] found a marked improvement in overall accuracy of tooth movement with the Invisalign® system since 2009. They stated that incisor intrusion remained a challenge with an overall accuracy of 35%. A study by Kravitz [8] found an accuracy in intrusion of 41.3%. Krieger [9] compared intra-oral scans with the digital ClinCheck plan set-up and found a difference of 0.71 mm in overbite correction. Blundell's findings were that 39.2% of intrusion was expressed and the deeper the initial overbite, the more challenging it would be to achieve the overbite reduction [10]. When evaluating studies on deep bite correction with aligners, it would be helpful to assess when the study was done and what features for deep bite correction were incorporated into the aligner design.

A more recent study published by Henick et al. [11] looked at the effect of Invisalign G5 (Align Technology Inc) with virtual bite ramps for skeletal deep bite correction in adult patients and found that although fixed appliance treatment had more apparent skeletal change, both fixed appliances and clear aligners were effective in opening deep bites at the dentoalveolar and skeletal levels.

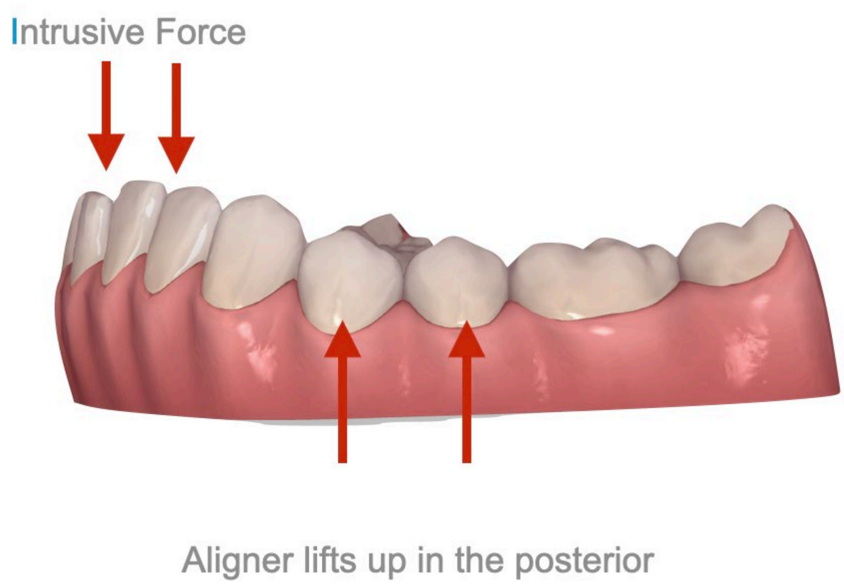

Figure 3. Intrusive force anteriorly with reciprocal extrusive force posteriorly.

ANCHORAGE DESIGN FOR LEVELLING THE CURVE OF SPEE

When intruding mandibular incisors to level the curve of Spee, an intrusive force is directed in the anterior segment of the arch. Concurrently, there will be a reciprocal extrusive force in the posterior segment. Therefore, the aligner will tend to lift up in the posterior (Figure 3). There needs to be sufficient posterior retention or anchorage, to prevent the lifting up of the aligner. Attachments are required for the first and second premolars and sometimes first molars as well for anchorage. This prevents the aligner from lifting up and provides the anchorage needed for the intrusive force to be directed in the anterior segment for incisor intrusion. The preferred attachment design would be the optimized deep bite attachments due to the ability for extrusive forces to be incorporated where posterior extrusion is desired. For other aligner systems, where optimized deep bite attachments are not available, a horizontal rectangular attachment, bevelled on the gingival may be placed (Figure 4).

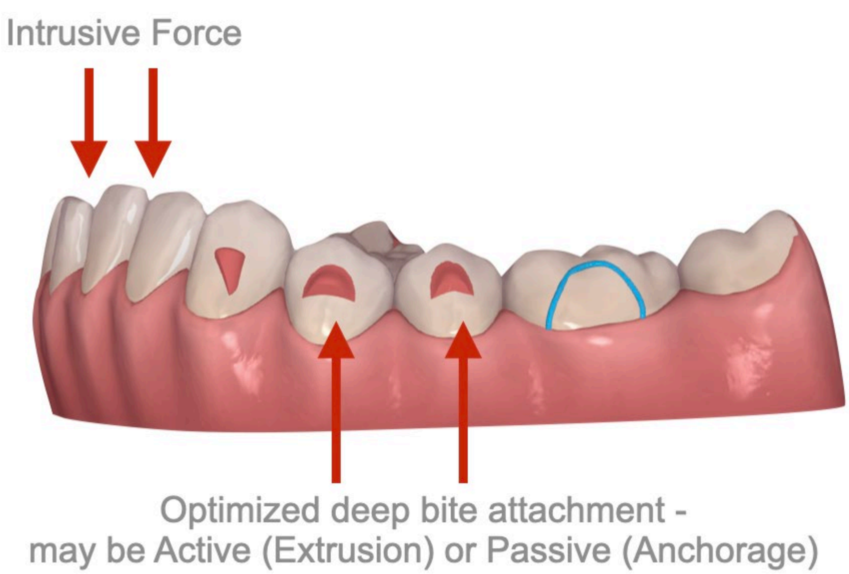

Figure 4. Optimized deep bite attachments on premolars.

No attachments are required on the lower incisors as attachments may interfere with the intrusion. More importantly, attachments will negate a more recent innovation by Align Technology known as SmartForce® aligner activations that are unique to the Invisalign® system [12]. With SmartForce® aligner activations, the shape of the aligner itself is adjusted for activation that is calibrated individually to provide sufficient and balanced intrusion for the anterior teeth. SmartForce® aligner activations apply to both maxillary and mandibular incisor intrusion (Figure 5).

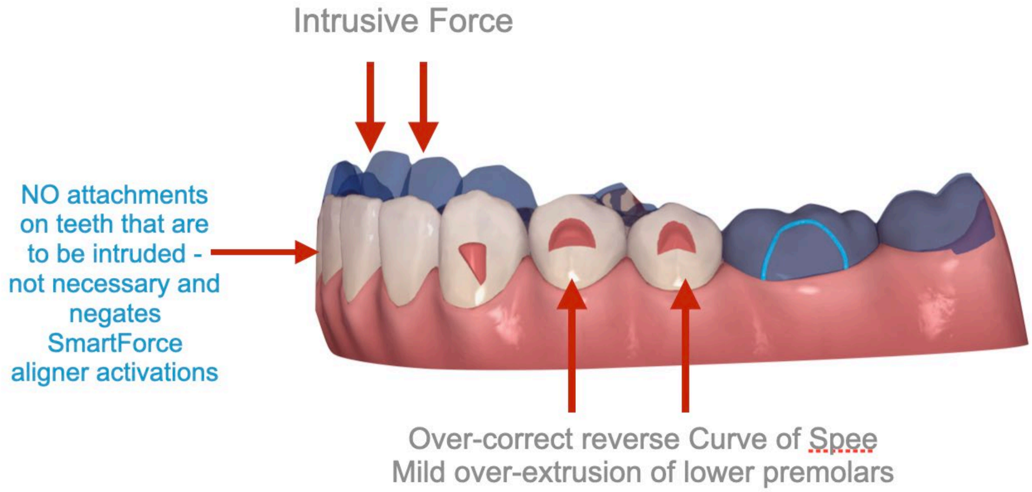

Figure 5. Superimposition showing anterior intrusion, posterior intrusion.

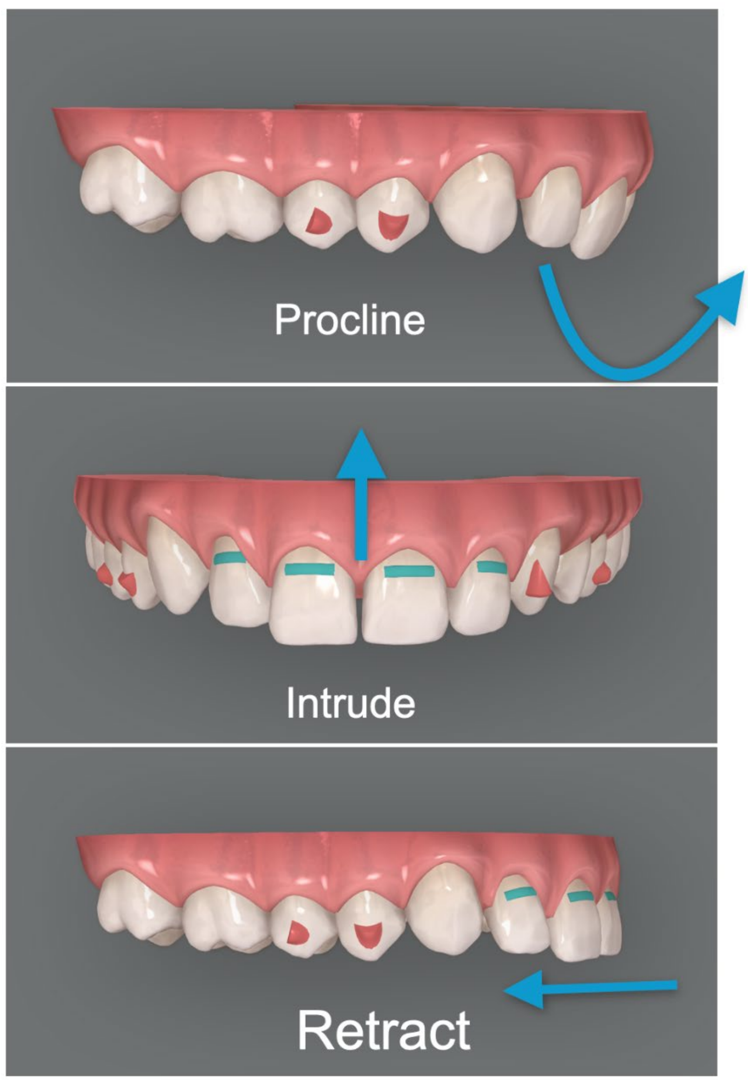

Figure 6. Staging incisor movements to procline, intrude, retract.

STAGING TOOTH MOVEMENTS FOR DEEP BITE TREATMENT

One important consideration in deep bite treatment is the sequence of tooth movements. Aligner systems are designed for simultaneous tooth movement. If a tooth requires lingual root torque, intrusion and rotation, every aligner would have all of these tooth movements built in. However, sometimes tooth movements in more than one place of space are difficult to execute. For example, in the case of retroclined, hypererupted incisors, it would be more clinically predictable to separate out the tooth movements and stage them to first procline, then intrude, finally to retract [13] (Figure 6). Retroclined maxillary incisors should first be proclined or mandibular incisors uprighted in the lower arch to correct the incisor inclination. Once the inclination is corrected, the incisors may then be intruded along the long axis of the tooth and any remaining spacing or overjet, may then be closed or the overjet decreased through incisor retraction.

During the proclination stage, the power ridge feature may be used for lingual root torque in combination with precision bite ramps. Once proclination is completed, there may now be excess overjet with the mandibular incisors now occluding behind the bite ramps. The precision bite ramps may be removed to allow pressure areas to be placed on the lingual of the incisors to assist with incisor intrusion. Conventional bite ramps may be placed on the maxillary canines to continue to disclude the posterior occlusion.

Box 1. Digital software plan design checklist.

Digital software plan design checklist

- Bite ramps
- Power ridges
- Anchorage attachments needed on first, second premolars and first molars
  - Optimized deep bite attachments – largest possible – first choice
  - Horizontal rectangular horizontal beveled in gingival – second choice
  - If premolar rotations present, vertical rectangular attachment – third choice
- Avoid attachments on lower incisors as they interfere with intrusion

Staging tooth movements in the software plan

- Procline, Intrude, Retract for both upper and lower incisors - software defaults are simultaneous staging, separating out such complex movements increase the clinical predictability of the programmed deep bite correction
- Program **anterior** (incisor) **intrusion**, as well as **posterior** (premolar) **extrusion** for severe deep bite cases

In setting up the virtual treatment plan in the software, it is imperative to have a checklist of the features for deep bite treatment designed into the digital treatment plan. Adequate anchorage considerations for levelling and intrusion, together with proper attachment design and staging patterns will lead to clinically predictable correction of a deep bite malocclusion (Box 1).

Since the literature shows that only a proportion of the intrusion programmed into the digital software plan is expressed clinically, it is recommended to set up the final occlusion in the digital software plan to over-treat the overbite, as well as the lingual root torque required for both upper and lower incisor inclination. The amount of over-treatment would vary depending on the age of the patient. For teens and younger, the final occlusion could be set up to ideal. For older adults, depending on the severity of the deep bite in the initial malocclusion, it is recommended to over-treat the final occlusion to 0 mm overbite or even to an anterior open bite.

CASE REPORT #1: TEEN CLASS II DEEP BITE MALOCCLUSION

Diagnosis and treatment plan

A teen female patient aged 15 years and 6 months presented with a mild Class II malocclusion with deep bite and minor crowding. Both upper and lower dental arches are squarish in arch form, and upper and lower incisors are retroclined. There is a steep curve of Spee in the lower arch. Cephalometric analysis revealed a Class I skeletal pattern (Figures 7-11).

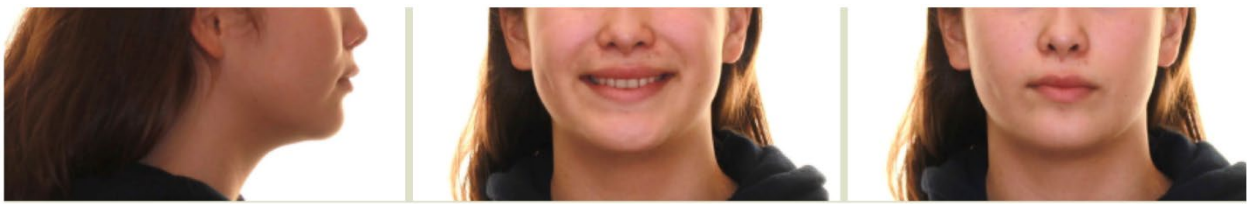

Figure 7. Pre-treatment facial photographs. A) Profile, B) Smiling, C) Relaxed lips.

Figure 8. Pre-treatment anterior and buccal intra-oral views. A) Pre-treatment right buccal intra-oral view, B) Pre-treatment anterior intra-oral view, C) Pre-treatment left buccal intra-oral view.

Figure 9. Pre-treatment upper (A) and lower (B) occlusal views.

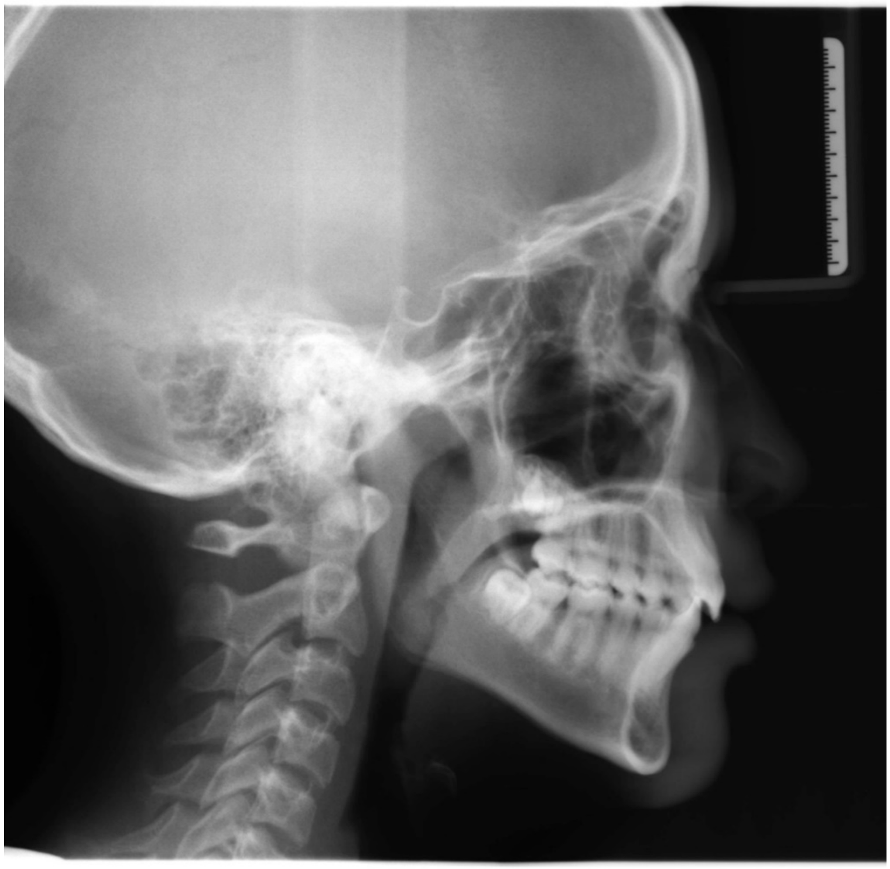

Figure 10. Pre-treatment cephalometric radiograph.

Figure 11. Pre-treatment panoramic radiograph.

Digital software plan

The digital software plan integrated a number of features for deep bite treatment. The power ridge feature was placed on maxillary and mandibular incisors. Precision bite ramps were placed on the lingual surfaces of the maxillary incisors. Although optimized deep bite attachments were not used in this case, both first and second premolars had attachments that also functioned for anchorage purposes to enable mandibular incisor intrusion to level the curve of Spee in the lower arch. Precision cut hooks for Class II elastics were placed on the maxillary canines and mandibular molars. A simulation jump was added in the software plan to simulate the effect of Class II elastic wear (Figures 12-15).

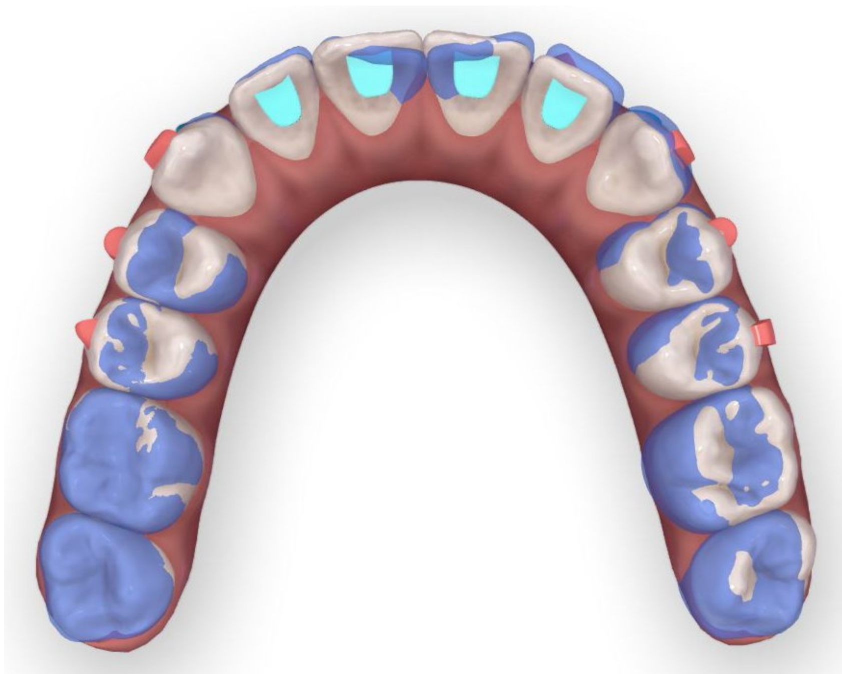

Figure 12. Precision bite ramps.

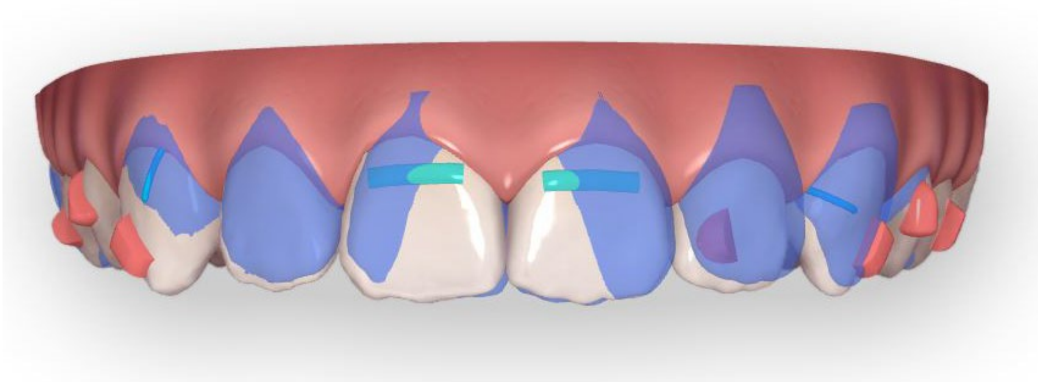

Figure 13. Power ridge feature on maxillary incisors.

Figure 14. Levelling curve of Spee with anterior intrusion.

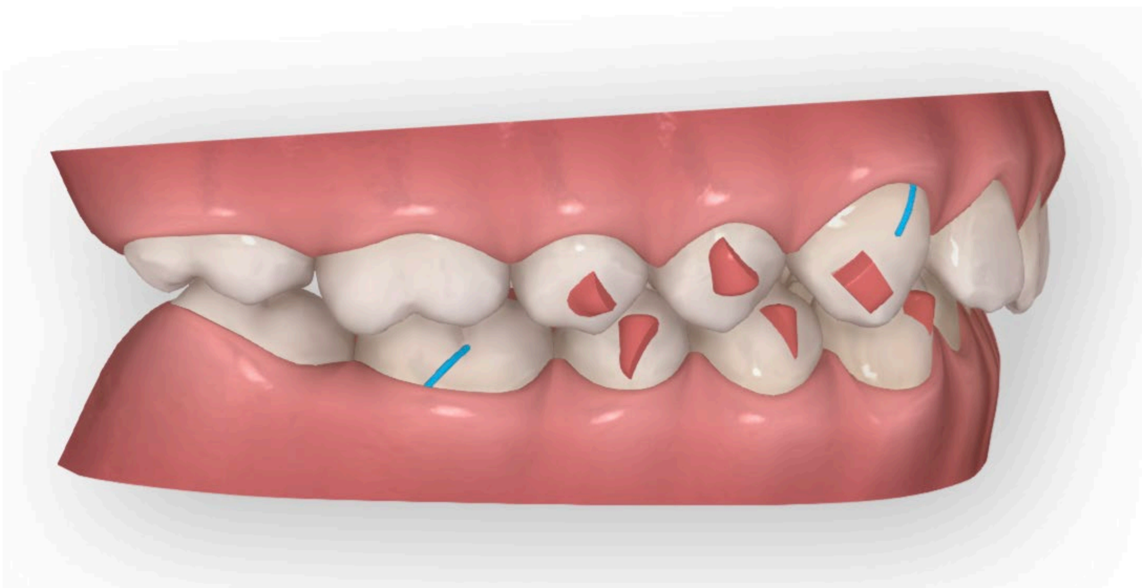

Figure 15. Precision cut hooks for Class II elastics.

Treatment summary

There were only 16 aligners in the treatment plan. Although it is the norm to change aligners every 7 days, this patient was advised to change the aligners every 14 days to allow the Class II elastics time to work, in order to correct the occlusion from Class II to Class I. ¼ inch, 4 oz elastics were worn full time. Treatment completed in 7 months without requiring additional aligners. The deep bite was corrected. Maxillary and mandibular incisor inclination were also corrected, and the curve of Spee was levelled in the lower arch. Both dental arches were aligned into an ovoid arch form. A comparison may be made between the pre and post treatment cephalometric radiographs. The patient was retained with Vivera® retainers from Align Technology Inc with bite ramps on the lingual of the maxillary incisors (Figures 16-19).

Figure 16. Post-treatment facial photographs. A) Profile, B) Smiling, C) Relaxed lips.

Figure 17. Post-treatment anterior and buccal intra-oral views. A) Post-treatment right buccal intra-oral view, B) Post-treatment anterior intra-oral view, C) Post-treatment left buccal intra-oral view.

Figure 18. Post-treatment upper and lower occlusal views. A) Post-treatment upper occlusal view, B) Post-treatment lower occlusal view.

Figure 19. Post-treatment cephalometric radiograph.

CASE REPORT #2: ADULT CLASS II DEEP BITE MALOCCLUSION

Diagnosis and treatment plan

A 27-year-old female presented with a Class II subdivision malocclusion with 100% deep bite. Maxillary and mandibular incisors were retroclined with incisor rotations. There was moderate lower arch crowding with the lower left first premolar erupted lingually. A steep curve of Spee was present. The buccal occlusion was Class I on the right and half cusp Class II on the left. Adding to the complexity of the case, the maxillary left second molar had erupted into buccal crossbite. Cephalometric analysis showed a Class I brachyfacial skeletal pattern with upright maxillary and mandibular incisors (Figures 20-24).

Figure 20. Pre-treatment facial photographs. A) Profile, B) Smiling, C) Relaxed lips.

Figure 21. Pre-treatment anterior and buccal intra-oral views. A) Pre-treatment right buccal intra-oral view, B) Pre-treatment anterior intra-oral view, C) Pre-treatment left buccal intra-oral view.

Figure 22. Pre-treatment upper and lower occlusal views. A) Pre-treatment upper occlusal view, B) Pre-treatment lower occlusal view.

Figure 23. Pre-treatment panoramic radiograph.

Figure 24. Pre-treatment cephalometric radiograph.

Digital software plan

The digital software plan incorporated features for deep bite correction such as precision bite ramps on the maxillary incisors. Power ridge features were placed on the maxillary incisors. Optimized deep bite attachments were placed on several premolars with root control and rotation attachments on the remaining premolars. The moderate crowding in the lower arch was resolved through a combination of interproximal reduction and expansion.

Class II correction was achieved through maxillary molar distalization on the maxillary left quadrant. This was supported by Class II elastics facilitated by precision cut hooks on maxillary canines and button cutouts on the mandibular first molars. To assist with the buccal crossbite correction of the maxillary right second molar, button cutouts were placed on the lingual surface of the maxillary right second molar and the buccal surface of the mandibular right second molar. The maxillary incisors were staged according to the procline, intrude, retract protocol (Figures 25-28).

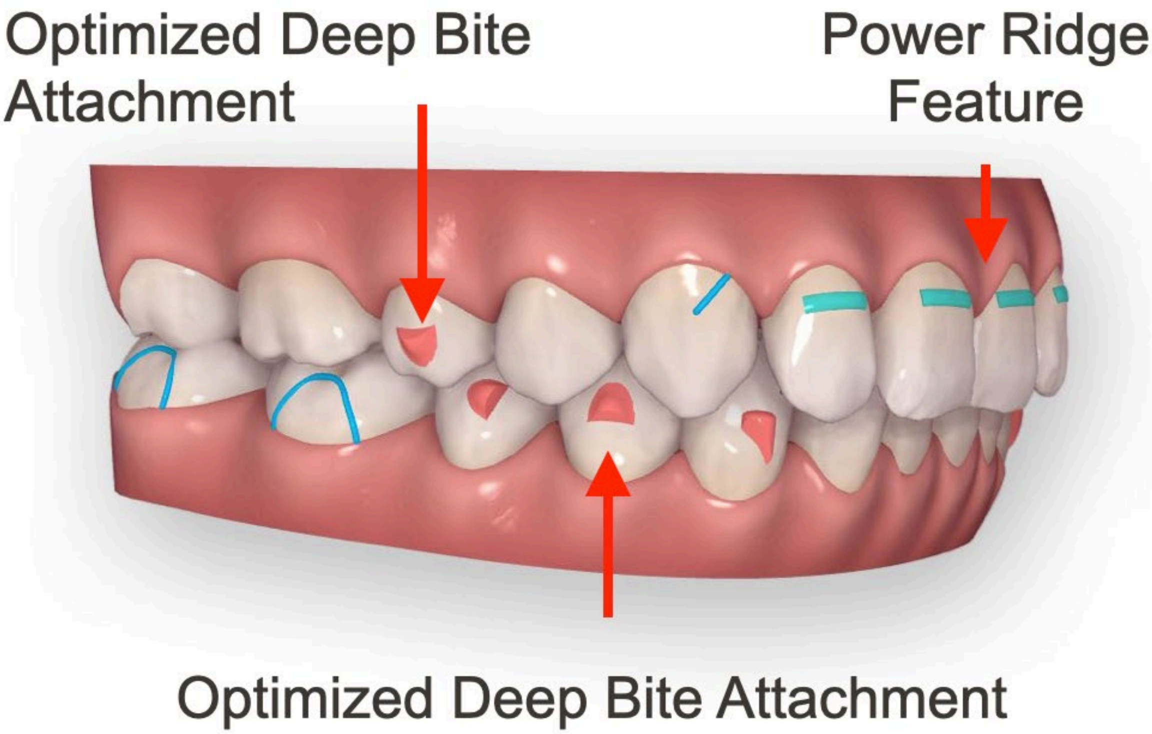

Figure 25. Optimized deep bite attachments, power ridge feature and precision cuts for Class II elastics.

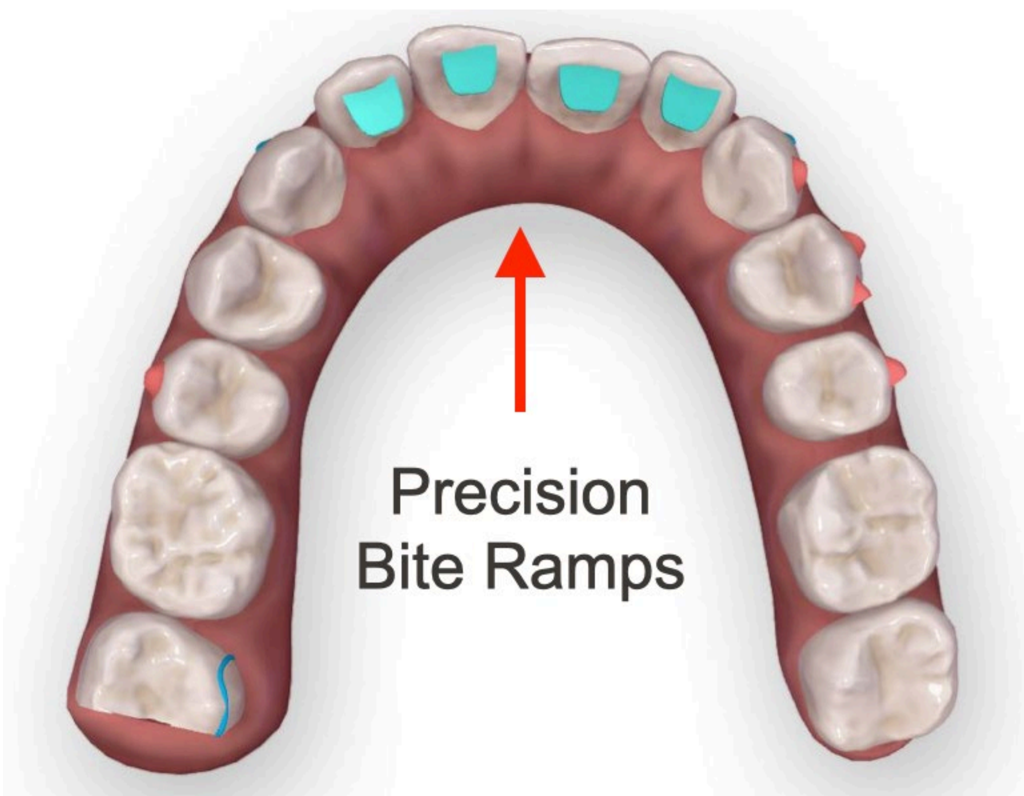

Figure 26. Precision bite ramps.

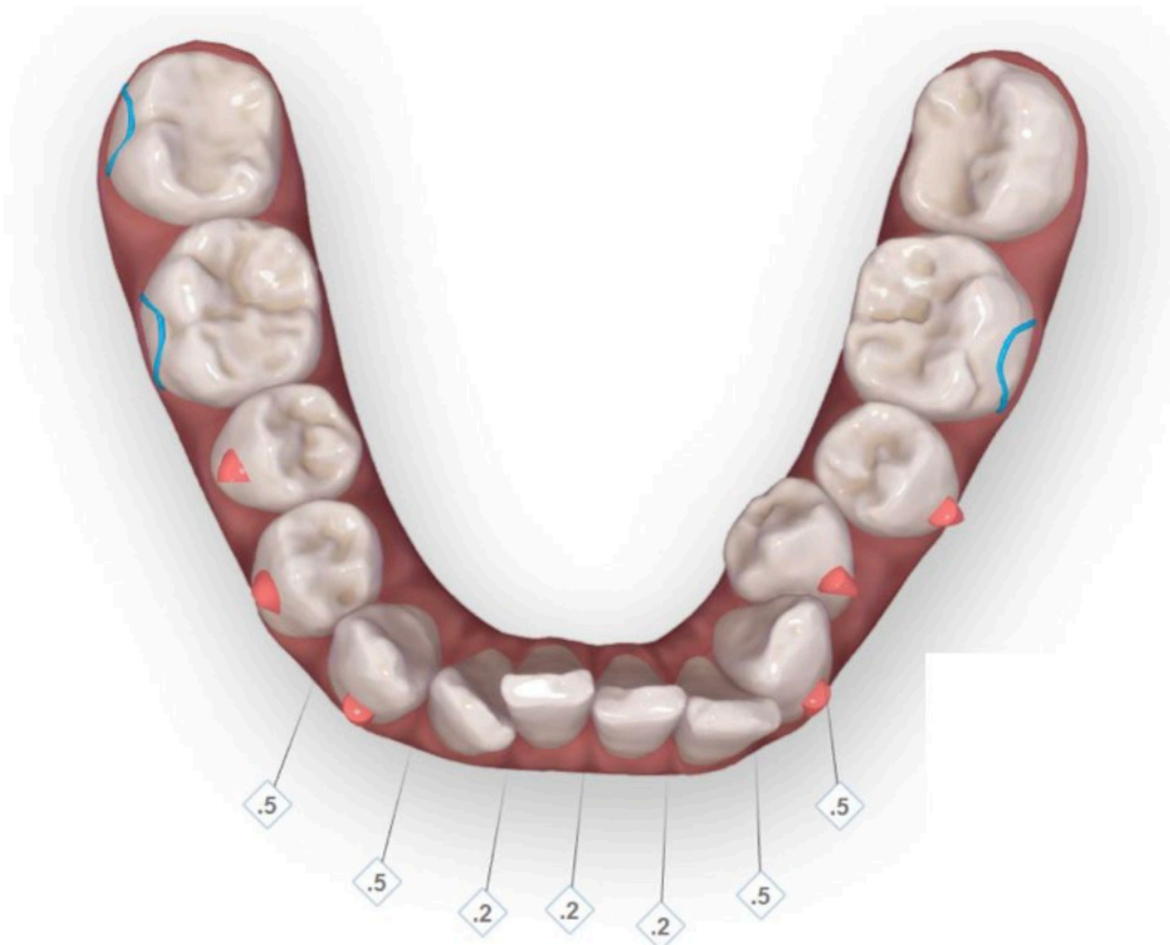

Figure 27. Lower arch interproximal reduction.

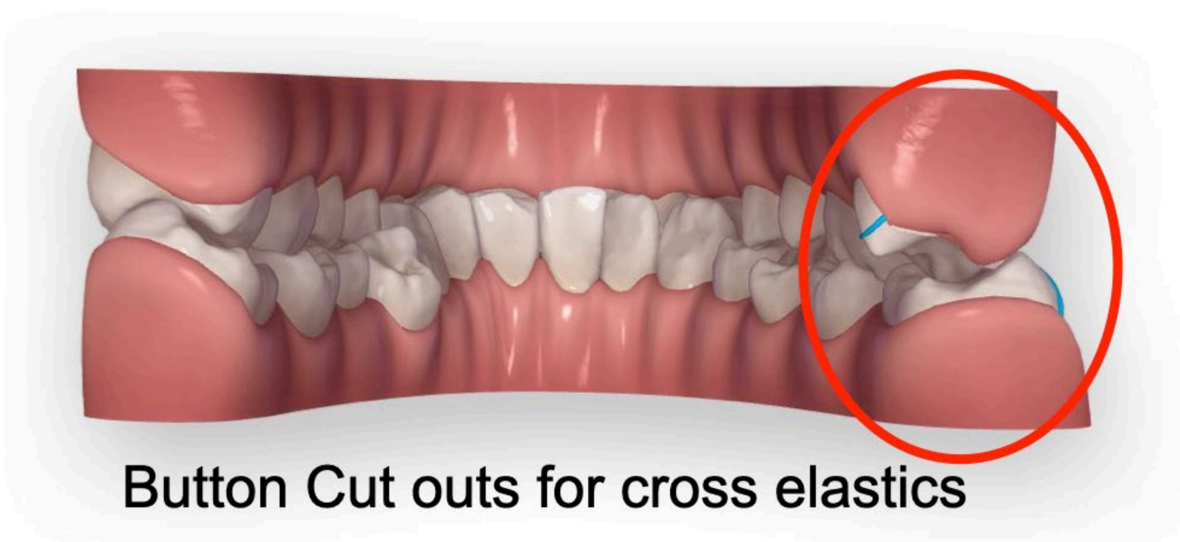

Figure 28. Button cutouts for cross elastics for the second molars on the right side.

Treatment summary

The initial series comprised 37 aligners. At the end of the first series of aligners, the deep bite had corrected 50%. There was still a residual curve of Spee in the lower arch. Maxillary and mandibular incisors were still fairly upright. The left buccal occlusion was a mild Class II. The crossbite of the maxillary right second molar had been successfully corrected.

Additional aligners were made. In the additional aligner stage, more maxillary molar distalization was programmed in the upper left quadrant, supported by Class II elastics. There was more levelling of the curve of Spee in the lower arch. The power ridge feature was again placed on the maxillary incisors for further lingual root torque. There were 31 aligners. Treatment completed in 27 months. The deep bite was successfully corrected. Maxillary and mandibular dental arches were aligned. And the buccal occlusion was corrected to Class I. The patient wore Vivera® clear retainers from Align Technology Inc with bite ramps (Figures 29-32).

Figure 29. Post-treatment facial photographs. A) Profile, B) Smiling, C) Relaxed lips.

Figure 30. Post-treatment anterior and buccal views. A) Post-treatment right buccal intra-oral view, B) Post-treatment anterior intra-oral view, C) Post-treatment left buccal intra-oral view.

Figure 31. Post-treatment occlusal views. A) Upper occlusal view, B) Lower occlusal view.

Figure 32. Post-treatment cephalometric radiograph.

CONCLUSIONS

In conclusion, deep bite malocclusion may be successfully treated with clear aligners. Proper diagnosis of the etiology of the deep bite malocclusion is critical in deciding what type of tooth movements should be programmed into the digital software plan. A checklist of the desired features such as pressure areas, optimized deep bite attachments, precision bite ramps, optimized support attachments, and power ridge feature should be developed. In reviewing the tooth movements in the digital software plan, proper staging protocols such as procline, intrude, and retract should be followed. Finally, based on the current evidence on predictability of tooth movements with aligners, over-treatment should be built into the final projected occlusion.

REFERENCES

1. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977; 72(1):1-22.
2. Nanda R. Esthetics and biomechanics in orthodontics. St. Louis, Missouri: Elsevier; 2015.
3. Tuncay OC. The Invisalign system: QP United Kingdom; 2006.
4. Align Technology. 2013. https://www.aligntech.com/documents/Invisalign_G5_Brochure.pdf
5. Khosravi R, Cohanim B, Hujoel P, Daher S, Neal M, Liu W, Huang G. Management of overbite with the Invisalign appliance. Am J Orthod Dentofacial Orthop 2017; 151(4):691-699 e692.
6. Grunheid T, Loh C, Larson BE. How accurate is Invisalign in nonextraction cases? Are predicted tooth positions achieved? Angle Orthod 2017; 87(6):809-815.

7. Haouili N, Kravitz ND, Vaid NR, Ferguson DJ, Makki L. Has Invisalign improved? A prospective follow-up study on the efficacy of tooth movement with Invisalign. Am J Orthod Dentofacial Orthop 2020; 158(3):420-425.
8. Kravitz ND, Kusnoto B, BeGole E, Obrez A, Agran B. How well does Invisalign work? A prospective clinical study evaluating the efficacy of tooth movement with Invisalign. Am J Orthod Dentofacial Orthop 2009; 135(1):27-35.
9. Krieger E, Seiferth J, Marinello I, Jung BA, Wriedt S, Jacobs C, Wehrbein H. Invisalign(R) treatment in the anterior region: were the predicted tooth movements achieved? J Orofac Orthop 2012; 73(5):365-376.
10. Blundell HLD, Weir TD, Kerr BD, Freer ED. Predictability of overbite control with the Invisalign appliance. Am J Orthod Dentofacial Orthop 2021; 160(5):725-731.
11. Henick D, Dayan W, Dunford R, Warunek S, Al-Jewair T. Effects of Invisalign (G5) with virtual bite ramps for skeletal deep overbite malocclusion correction in adults. Angle Orthod 2021; 91(2):164-170.
12. Align Technology 2020. https://www.invisalign.com/provider/G8
13. Tai S: Clear aligner technique. Hanover Park, IL: Quintessence Publishing; 2018.

COMPARISON OF OUTCOMES IN DEEP BITE CASES TREATED WITH BRACES, TEMPORARY ANCHORAGE DEVICES (TADS), AND INVISALIGN

Toru Deguchi

ABSTRACT

Deep bite cases are some of the most difficult malocclusions to correct with orthodontic treatment. There are basically three ways to correct deep bites: 1) absolute intrusion of incisors, 2) relative intrusion of incisors, and 3) extrusion of molars. The treatment method used should be selected according to the patient's condition such as high angle or low angle deep bite, the patient's requests and the clinician's preferences. In this presentation, I introduce 3 different types of orthodontic treatment: 1) fixed appliances, 2) miniscrews, and 3) Invisalign therapy for treating deep bite cases. I compare the treatment outcomes and discuss the difference in the mechanics and limitations of each treatment option. With fixed appliances, the use of reverse curve of Spee (RCOS), utility arches, and the Burstone segmented archwire techniques will be discussed. With miniscrews, both maxillary and mandibular miniscrews are used to intrude incisors, and for Invisalign cases, the key factors for successful intrusion of the incisors will be indicated. In addition, some deep bite clinical cases treated by these treatment methods will be presented along with a discussion of the pros and cons for each option.

KEY WORDS: Deep Bite, Fixed Appliance, Miniscrew, Invisalign

INTRODUCTION

Approximately 15-20% of the population has deep bite (overbite ≥ 5mm) in the United States [1]. Some of the suggested problems with severe deep bite include esthetic and functional problems. During mixed dentition or at early permanent stage, deep bite with impinging mandibular incisors may cause damage to the palatal tissue. In the long-term, some have suggested that there may be a correlation between the presence of deep bite and occurrence of temporal mandibular disorder (TMD) [2]. According to the severity and type of deep bite, esthetics also could be a concern such as overexposure of maxillary incisors during smile, concave type of profile due to decreased mandibular plane angle or increased overjet in high angle deep bite cases. Thus, orthodontists should know what type of appliances or biomechanics is required to treat specific type of deep bite cases.

Besides orthognathic surgery, incisor intrusion and/or molar extrusion would be the typical treatment goal to treat deep bite. The type and the amount of tooth movement is determined by dental and skeletal severity of the deep bite. In case of low angle deep bite, in general, molar extrusion is allowed by using treatments such as reverse curve of Spee wires [3], bite plate appliances [4], and elastics [5]. On the other hand, in high angle cases, intrusion wires [6], head gears [7], and temporary anchorage devices (TADs) [8] are commonly used for the correction of deep bite. However, in any of these appliances, there will be always a reciprocal force resulting in the opposite teeth (anterior teeth vs posterior teeth). One of the keys for successful treatment is to figure out how to control the reciprocal force.

Before the TADs were introduced, intrusion archwire was one of the main methods to treat deep bite cases. However, one of the disadvantages on using this intrusion wire is that there is a tendency to extrude molars or flare the incisors. To cancel these reciprocal forces, 3-piece segmented techniques, transpalatal arch (TPA), and headgear were used [6, 7]. After the introduction of TADs, the mechanics seemed to be less simple, however, surgical procedure is required and miniscrew failure or root proximity became a concern with the use of TADs [9]. In recent years, aligners have been used to correct deep bite from esthetic reasons, especially in adult patients. Although, it is suggested that molar intrusion could be successful, but intrusion of incisors was suggested to be one of the limitations with aligner therapy [10].

Therefore, in this presentation, I would like to introduce the pros and cons of different appliances and techniques regarding the treatment of deep bite from an evidence-based point of view by introducing past articles and some of our data.

DEEP BITE CORRECTION WITH CONVENTIONAL EDGEWISE THERAPY

There are three main methods for treating patients with deep bite using braces: 1) Reverse curve of Spee (RCOS) [3,11], 2) Utility Arch [12, 13], and 3) Burstone segmented arch wire [3, 14].

I-I Correction of deep bites with RCOS

One of the most common methods for correcting deep bite is by leveling the curve of Spee in the mandibular arch. In general, RCOS is added to a rectangular stainless steel (SS) or beta titanium (TMA) wire (.016x022 or .017x.025). The result of the tooth movement is intrusion of the incisors with labial crown tipping and extrusion of premolar and molar with distal tipping. In this way, the amount of deep bite is decreased by intrusion of the mandibular incisors. However, there is also labial crown tipping, which is an undesirable movement. A past study compared the efficacy of deep bite correction between continuous arch wire (n=25, mean age 23.3) and the segmented arch Burstone technique (n=25, mean age 25.6) [15]. With the continuous wire, the mandibular incisors intruded by about 1.0mm with 5.7° of labial flaring and 1.3mm of molar extrusion. The molar extrusion was significantly greater than with the Burstone technique. Thus, continuous wire should be avoided in high angle cases, or when the mandibular incisors are already flared.

One of the easiest ways to avoid incisor flaring is to chinch back or tie back the wire. With the Alexander technique, they recommend that the wire be cinched back when applying the RCOS [16]. A study that analyzed the effect of continuous RCOS wire (n=31, mean age 12.6) with cinch back or tie back basically resulted in 1.3mm of premolar extrusion (considering the amount of growth was 1.5mm, so the total was 2.8mm), 0.5mm of intrusion of the incisors (considering that the amount of growth was 1.4mm, so the total was 0.9mm), and a 0.9mm extrusion of the mandibular molars (considering the amount of growth was 1.4mm, so the total was 2.3mm). The mean increase of mandibular incisor inclination was 0.8° and distal tipping of molars resulted in about 6°. This indicates that with cinch or tie back mechanics, incisor labial flaring can be mostly prevented and treatment will result in extrusion of premolars and distal tipping of the molars. The only consideration with cinch or tie back mechanics may be extrusion of molars, especially in adult patients due to substantial distal tipping of molars. In this study, there was only about 1mm of extrusion because of growth.

I-II Correction of deep bites with the use of utility arches (U-arches)

The use of the U-arch technique was introduced by Ricketts, who suggested that extrusion of premolars by RCOS would increase the lower facial height that may negatively affect the stability [17]. A past study that analyzed the outcome of U-arches (n=30, mean age 10.4) resulted in 1.3mm of intrusion with 5.3° of labial flaring [12]. This study also used a U-arches in the maxillary arch that resulted in -1.1mm of intrusion and 1.7° of labial flaring. In addition, the maxillary molars extruded by 2.9mm, however, since these cases still had future mandibular (vertical) growth, no significant clockwise rotation of the mandible was observed. In another study that compared the effects of mandibular incisors intrusion between the U-arch and TAD supported intrusion, the U-arch group (n=13, mean age=16.3) resulted in 1.5mm of intrusion of the mandibular incisors and 8° of labial flaring [13]. Mandibular molars also tipped 9° with 0.5mm of extrusion. Since they also used U-arches in the maxilla, the incisors flared 8.9°, and the molars tipped 4.3° distally. Thus, with the use of U-arches, there was still substantial incisor flaring and molar tipping.

I-III Correction of deep bite with the use of Burstone segmented archwires

The third method for correcting deep bite by a conventional edgewise method is the Burstone segmental arch wire. Burstone segmental arch wires consist of three parts: 1) a posterior anchorage unit, 2) an anterior segment, and 3) an intrusive arch spring [15]. A sectional wire of at least .018x.018 SS, progressing to .018x.025 SS is used as a buccal stabilizing segment. In general, the intrusive arch is made with a .018x.022 or .025 SS with a helix placed mesial of the auxiliary tube. When the intrusion arch is tied to the level of the incisors, an intrusive force, ideally about 25-50 gm, is delivered. In addition, a TPA is used in the maxilla, and a lingual arch (LA) is used to control the anchorage and torque. A past study that compared the effects of overbite correction using a continuous arch versus a segmented arch technique (n=25, mean age 25.6) indicated that the Burstone segmented arch wire technique resulted in an average of about -1.7mm of mandibular incisor intrusion and an increased labial inclination of 4° [3]. Mandibular molars extruded by about 0.6mm. Maxillary incisors also intruded by about -1.5mm with no flaring, and molars were hardly extruded at all. They concluded that the Burstone segmented technique is superior to the conventional continuous arch wire techniques if arch leveling by incisor intrusion is indicated. Moreover, since minimum extrusion of molars was observed, this method should be ideal for high angle cases.

In conclusion, with conventional intrusion arch wire techniques: 1) maxillary incisors can be intruded by approximately 1.5mm, 2) mandibular incisors can be intruded by approximately 2.0mm, and 3) molars can be extruded by approximately 1.5mm. However, with the use of sectional archwire mechanics, extrusion can be minimized.

DEEP BITE CORRECTION WITH TEMPORARY ANCHORAGE DEVICES (TADs)

II-I Maxillary incisor intrusion with miniscrews

TADs have been utilized in recent decades as one the major ways to effectively move teeth and control anchorage. With deep bite, TADs are mainly used to intrude incisors [18, 19]. One study that compared the intrusion effect of maxillary incisors between intrusive arch (n=15, mean age 22.6) and miniscrew supported intrusion (n=15, mean age 19.5) resulted in a mean intrusion of about 2.6mm with miniscrews and 2.3mm with intrusive arches [18]. There was significantly more labial tipping of incisors with the intrusive arch (7.7°) compared to miniscrews (2.3°). No significant movement in the molars was observed.

We published a study that compared the intrusion effects of maxillary incisors with miniscrew versus J-hook headgear in maxillary first premolar extraction cases [19]. One of the reasons that we were interested in analyzing the effect of maxillary incisor intrusion with J-hook headgear was probability of less anchorage loss and side effects in the posterior teeth compared to other wire intrusion mechanics. The results in the miniscrew groups (n=8, mean age 21.5) was an average of 3.5mm with 6° of incisor retraction, whereas in the J-hook group (n=10, mean age 20.7) there was -1.5mm of intrusion with 8.5° of incisor retraction. There was significantly more intrusion effect with miniscrews than with J-hook headgear. However, miniscrews tend to flare the incisors more than when J-hook headgear is used.

II-II Correction of excessive gingival display (gummy smile) with miniscrews

Since miniscrews seem to successfully intrude maxillary incisors without reciprocal force to the molars, it is possible to successfully correct excessive gingival exposure (gummy smile) that is related to extruded maxillary incisors. One of the important aspects for effectively intruding incisors is the location of the miniscrews. Some authors prefer placing them between the centrals and laterals [20, 21] whereas others place them between laterals and canines [22]. From my experience, since placing miniscrews between the lateral and canine creates a line of force close to the center of the resistance (CR) of six anterior teeth, there will be less labial flaring compared to miniscrews that are placed between the central and lateral. However, intrusion force (vertical vector) will be less, and more force will be required to effectively intrude the four incisors.

Figure 1. Pretreatment photos 17Y 5M.

Case One

This was a gummy smile case treated with miniscrews in the maxillary anterior region between the central and lateral incisors (Figure 1). From the facial photograph, this patient (17 years and 5 months, female) had a convex profile, hyper mentalis, acute nasolabial angle, decreased mentolabial angle, and excessive gingival display. From an oral photograph, Class I molar and Class II canine relationships with increased overjet were observed. Cephalometric analysis (Figure 2) resulted in Skeletal 1 (ANB 4.1°), high angle (SN/MP 39.2°), and increased axial inclination of the mandibular incisors (L1/MP 96.3°). This case was diagnosed as Skeletal 1, Angle Class I, high angle with increased overjet and excessive gingival display. The treatment plan called for extraction of the maxillary first premolars and mandibular second premolars and the use of 4 miniscrews, 2 in the maxillary anterior nasal spine region (1.3mm diameter x 5mm, Absoanchor Dentos, Daegu, Korea) and another 2 (1.3mm diameter x 6mm, Absoanchor Dentos, Daegu, Korea) between the maxillary second premolars and first molars as an anchorage device.

Figure 2. Pretreatment radiographs.

After 6 months of treatment, there was noticeable incisor intrusion during canine retraction (Figure 3). T-loops were used to effectively intrude the incisors, but the intrusion could have been done with a ligature wire or power chain. After 10 months, the overbite almost had an edge-to-edge relationship, and after 12 months, the incisors had an edge-to-edge relationship so up-down elastics were used from the hook of the maxillary incisors to the hook of mandibular incisors to maximize the intrusion of maxillary incisors.

Figure 3. Progress photos.

Figure 4. Post-treatment photos at 19Y 9M.

A post-treatment facial photograph (Figure 4) shows improved gummy smile and a convex profile. However, there was still a slight strain in the mentalis. The total treatment time was 28 months. From the intra-oral photos (Figure 4), Class I canine and decreased overjet can be seen. Post-treatment cephalometric analysis showed a slight increase of the mandibular plane angle (SN/MP +0.3°), increases in the axial inclination of the maxillary incisors (SN/U1 5.2°), uprighted mandibular incisors (L1/Mp 8.2°), and -4.8mm of intrusion of the maxillary incisors (Figure 5). However, substantial root resorption was observed in the maxillary incisors from the panoramic radiographs.

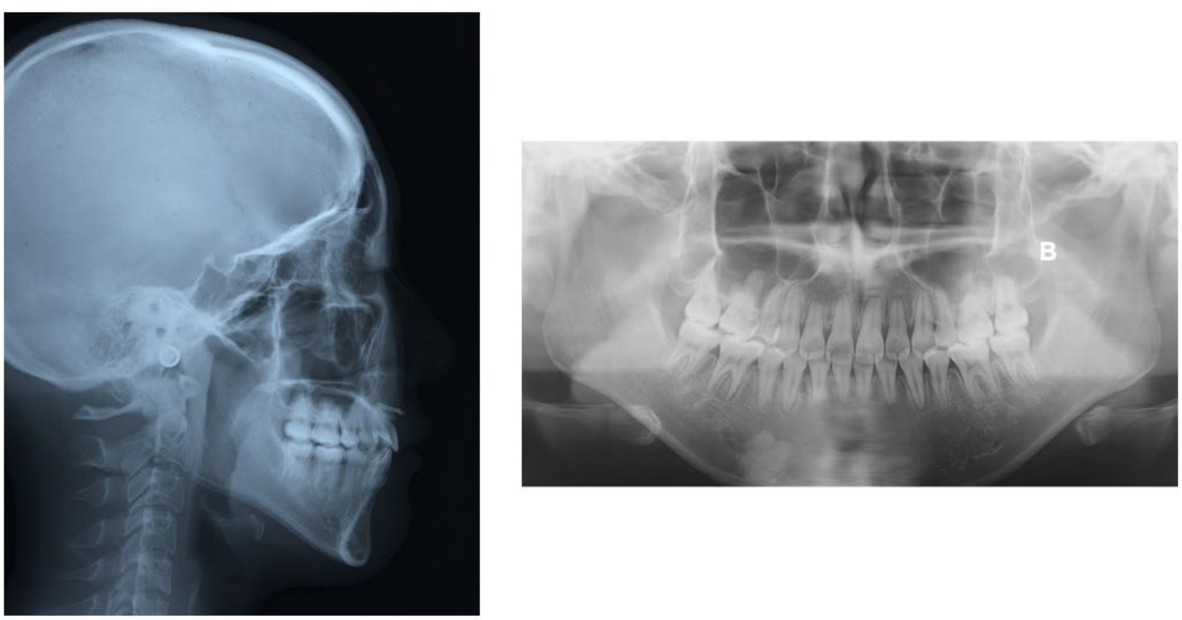

Figure 5. Post-treatment radiographs.

Overall superimposition of pre (17Y 5M) to post (19Y 6M) treatment images indicated an improved soft tissue profile due to flatting of the lips (Figure 6). By maxillary superimposition, a significant amount of incisor intrusion with bodily retraction was observed. From mandibular superimposition, controlled tipping of the incisors can be observed. Note that there was hardly any change in the vertical. These photos (Figure 7 A, B) show substantial improvement of the gummy smile after about 15 months of intrusion. Also, from CBCT images (Figure 7 C, D), significant change in the morphology in the anterior region of the maxilla after intrusion and retraction is observed. However, as indicated earlier, a large amount of incisor intrusion can cause severe root resorption.

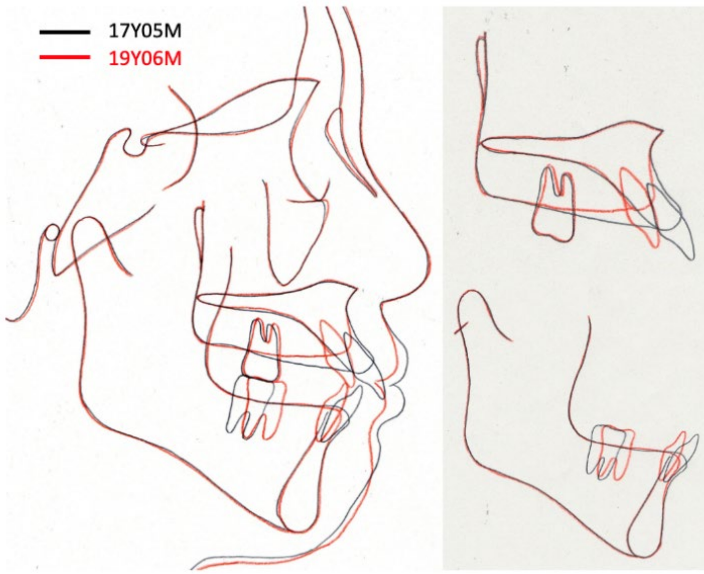

Figure 6. Superimposition of pre- and post-treatment.

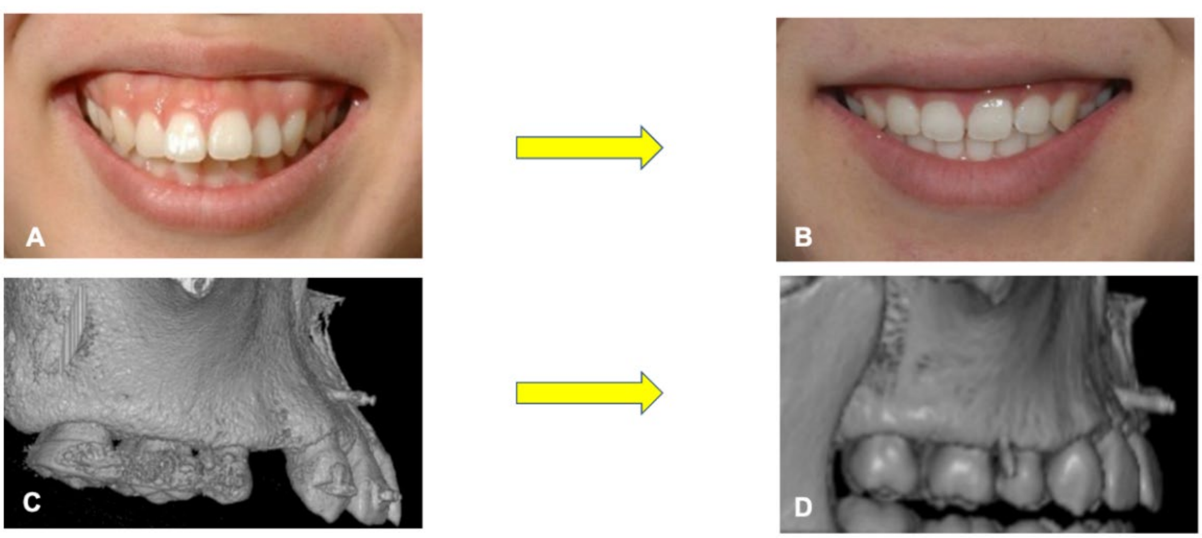

Figure 7. A) Pre-treatment, B) post-treatment intraoral photos and C) pre-treatment and D) post-treatment CBCT images.

Case Two

A second gummy smile case was treated by using miniscrews between the lateral incisor and the canine (Figure 8). Facial photographs show a bi-maxillary profile, acute nasolabial angle, decreased mentolabial angle, and some excessive gingival display. Intra-oral photographs indicate Class I molar and Class II canine relationship with moderate crowding on both arches. Cephalometric analysis revealed Skeletal 2 (ANB 6.2°), a short mandible, and bi-maxillary protrusion (Figure 9). The case was diagnosed as a Skeletal 2 Angle Class I, bi-maxillary case. We noticed some excessive gingival display, but the patient was not concerned about it at the time. The treatment plan was to extract the maxillary and mandibular first premolars and use two miniscrews in the posterior for absolute anchorage.

Figure 8. Pre-treatment photos at 28Y 3M.

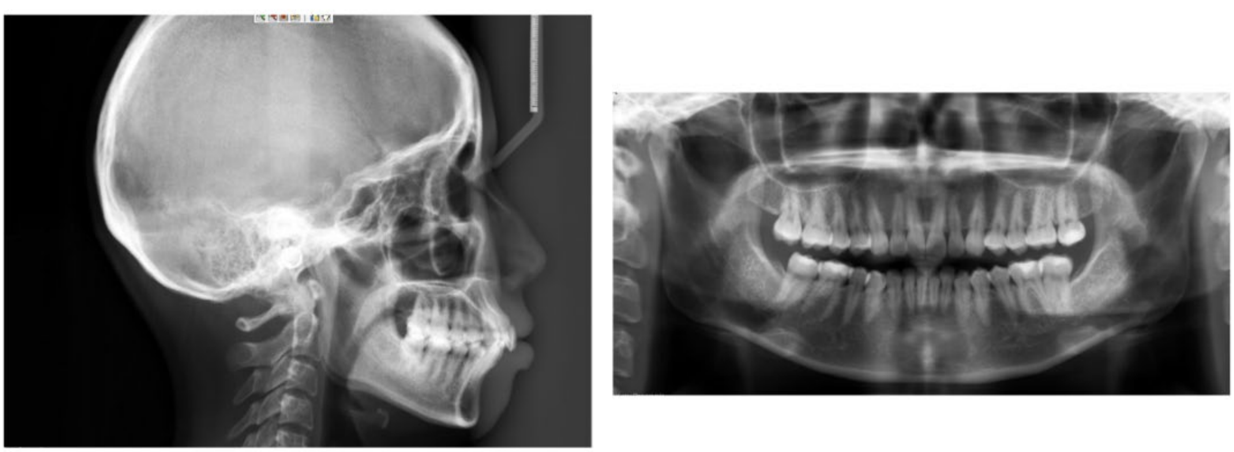

Figure 9. Pretreatment radiographs.

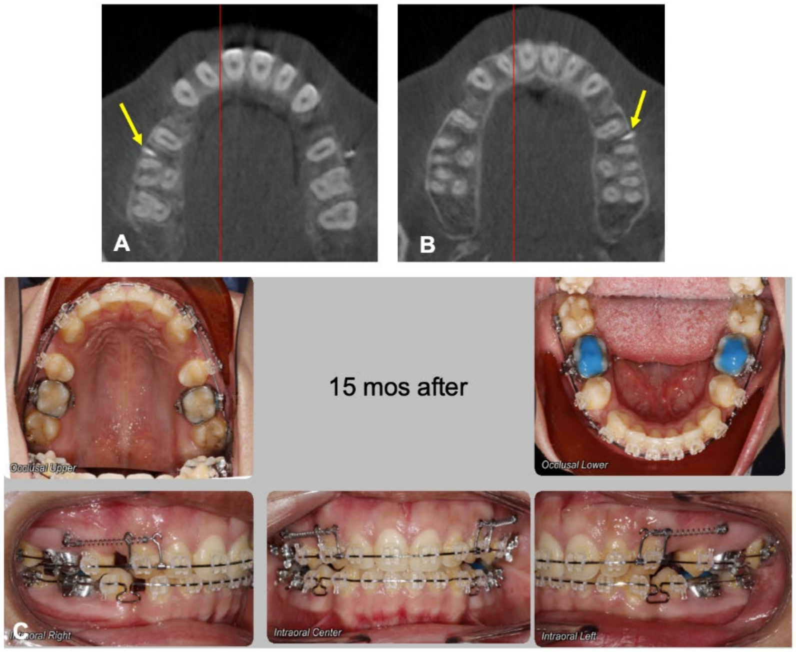

Figure 10. CBCT images of the location of miniscrews (yellow arrows) in A) right and B) left side between maxillary second premolar and first molar. C) Intraoral photos after 15 months of treatment.

Eight months after the start of treatment, we placed miniscrews (1.3mm diameter x 5mm, Absoanchor Dentos, Daegu, Korea) between the maxillary second premolar and first molars (Figure 10 A, B) and immediately started en masse retraction after the miniscrews were placed. Fifteen months after the start of treatment (7 months after retraction), the mandibular incisors were almost retracted (Figure 10 C), but a month later, the patient expressed concern over the appearance of the gummy smile (Figure 11 A). After taking a panoramic radiograph (Figure 11 B), we noticed there was more space between the lateral and canine, so we decided to place the miniscrews in those areas (Figure 12 A), and were able to intrude the incisors from them with the use of a T-loop to control vertical and torque while retraction (Figure 12 B).

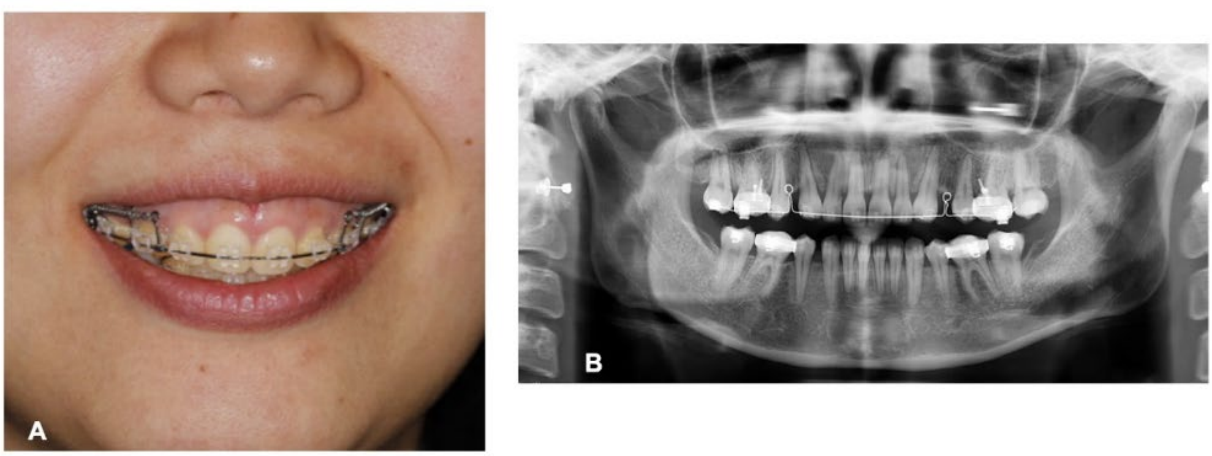

Figure 11. A) Intraoral photo showing a gummy smile during treatment, and B) a panoramic radiograph at the same time.

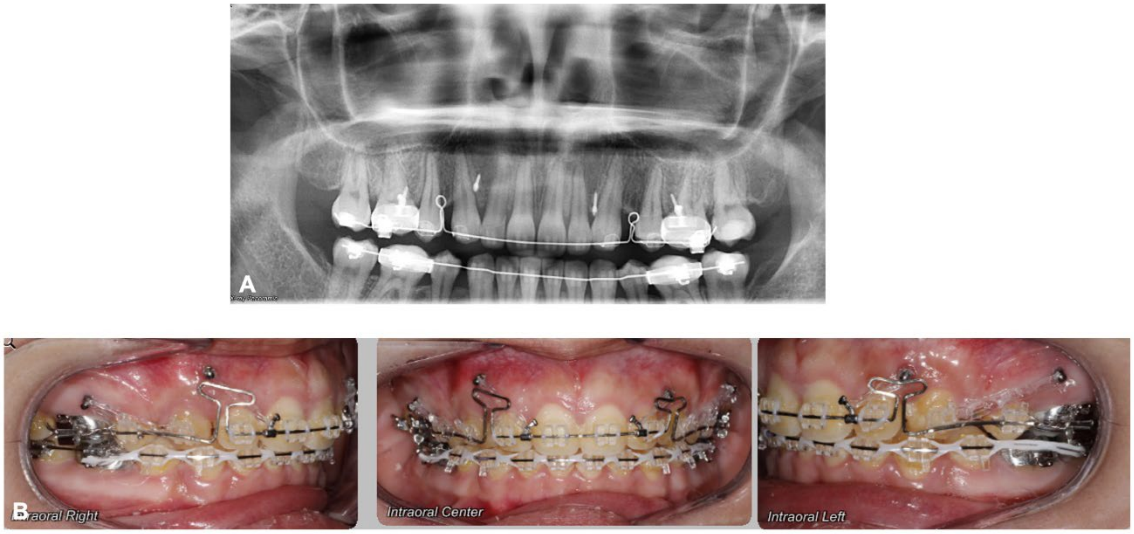

Figure 12. A) Panoramic radiograph after placement of miniscrews between the lateral and canine. B) Intraoral photos during intrusion and retraction of incisors.

Post-treatment facial photographs (Figure 13) indicated improved gummy smile and a flattened profile. Intra-oral photos show Class I molar and canine relationship with ideal overjet and overbite. The total treatment duration was 26 months. Cephalometric analysis indicated an increased mandibular plane angle (SN/Mp +3.5°), retracted maxillary incisors (U1/FH -6.9°) and mandibular incisors (L1/Mp -3.9°), and intrusion of the maxillary incisors by -2.9mm (Figure 14). However, there was some problem with root paralleling in the maxillary right first premolar, canine, and left lateral with some root resorption on the maxillary incisors, as seen in the panoramic radiograph.

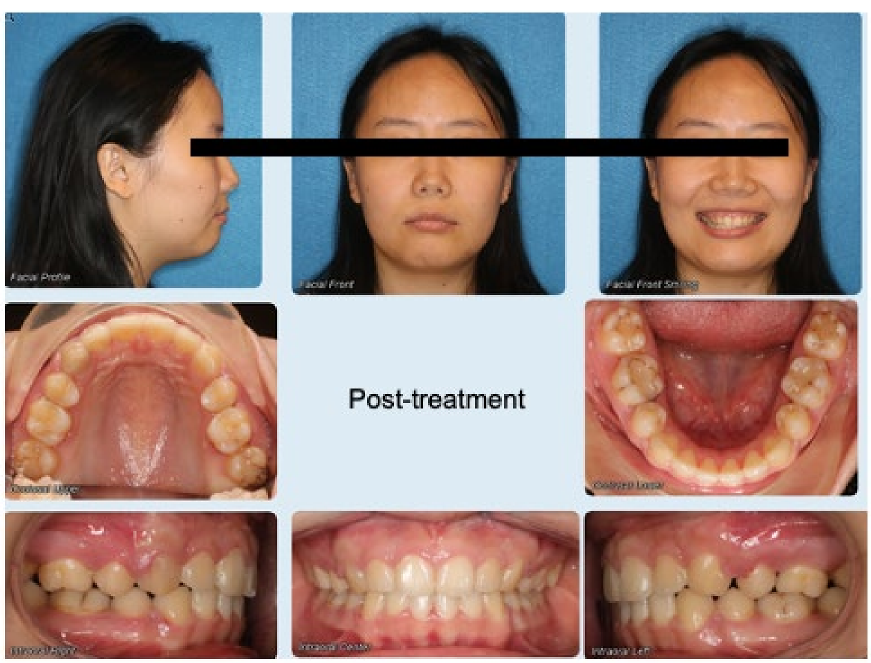

Figure 13. Post-treatment photos at 30 Y 5M.

Figure 14. Post-treatment radiographs.

Overall superimposition (Figure 15) shows retraction of upper and lower lips and slight clockwise rotation of the mandible. Maxillary superimposition indicates approximately 3.0mm of incisor intrusion with a slight loss of anchorage of the molars due to the extrusion. From the mandibular superimposition, some controlled tipping of the incisors and a slight loss of anchorage was observed in the molars.

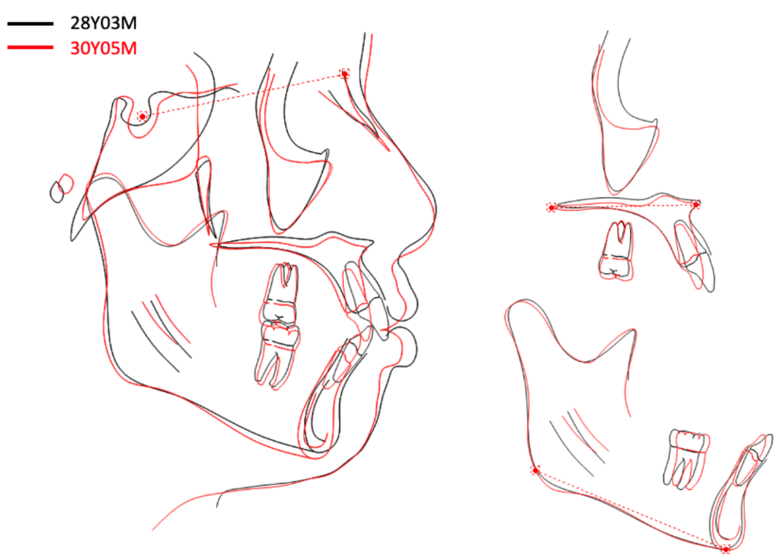

Figure 15. Superimposition of pre- and post-treatment.

As these two cases show, placing miniscrews between the central and lateral incisors may result in more effective incisor intrusion than placing them between the lateral and canine. However, with more intrusion, there is a greater risk of root resorption. This could be one of the disadvantages of using miniscrews.

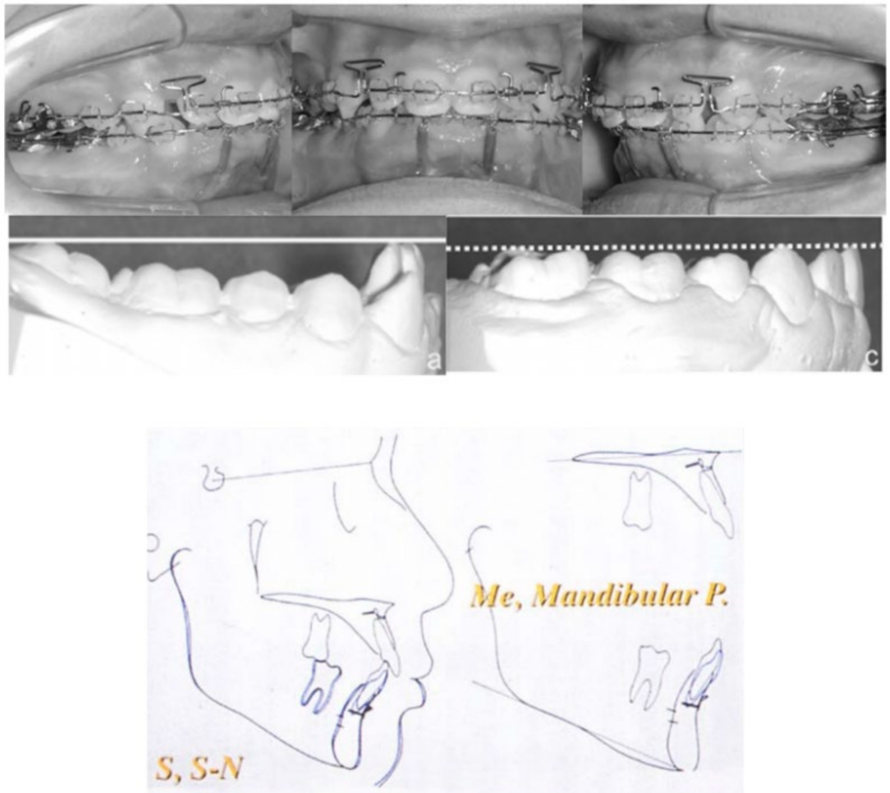

Figure 16. Effect of mandibular incisor intrusion by miniscrews.

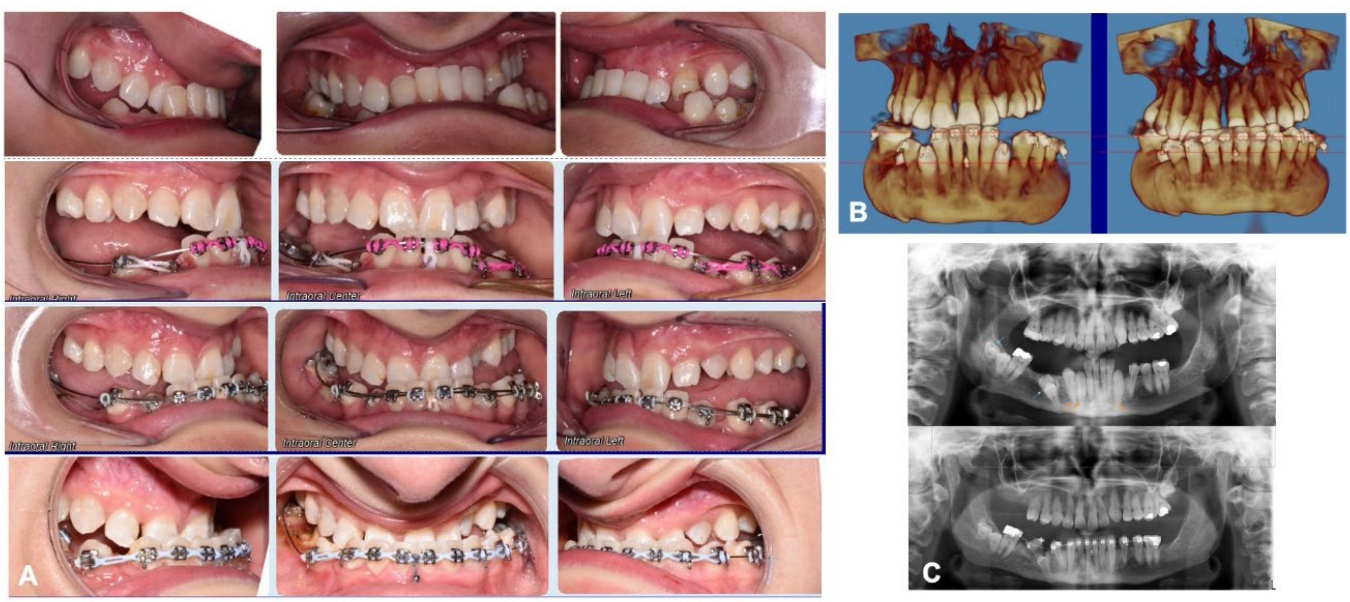

Figure 17. A) Intraoral photographs, B) CBCT, and C) panoramic radiographs of mandibular incisor intrusion of Cleidocranial Dysplasia.

II-III Intrusion of mandibular incisors with miniscrews

Not only in the maxilla, miniscrews are often used to intrude the mandibular incisors. As a matter of fact, the first reported use of miniscrew was placement in the mandibular symphysis area to intrude incisors by Kanomi, who placed three miniscrews and intruded the mandibular incisors by approximately 6mm [23]. In addition, we also used 2 miniscrews to intrude the mandibular incisors by about 4mm to flatten the curve of Spee (Figure 16). Recently, we used just one miniscrew to intrude the mandibular incisors by about 5mm in a cleidocranial dysplasia case. Effective mandibular incisor intrusion can be seen in the intraoral photos (Figure 17 A), CBCT (Figure 17 B), and panoramic radiographs (Figure 17 C). Thus, effective intrusion of mandibular incisors is also possible with miniscrews.

In conclusion, by using miniscrews as anchorage: 1) the maxillary incisors can be intruded about 3.0mm, 2) the mandibular incisors can also be intruded about 3.0mm, and 3) reciprocal tooth movement such as molar extrusion should be minimal compared to other intrusion mechanics.

DEEP BITE CORRECTION WITH INVISALIGN THERAPY

Invisalign has been one of the optional treatment methods used in orthodontic treatment. However, there has been some concern about the limitations of Invisalign treatment as we have reported in the past [24]. Prediction of tooth movement is also a concern with Invisalign treatment. One aspect that is difficult to predict is vertical incisor movement. There is less than a 50% likelihood that the predicted intrusion can be achieved for central incisors [10, 25]. A study analyzed the effects of Invisalign on correcting overbite including deep bite and open bite cases [26]. In the deep bite cases, they used 46 non-extraction cases (mean age 40) in which there was more than 4mm of overbite and virtual bite ramps were used with posterior elastics. Treatment results showed an average of 1.5mm of median opening, proclination of the mandibular incisors (2.5°), 0.5mm of maxillary incisor intrusion, and mandibular molars that were extruded by about 0.5mm. They concluded that Invisalign improves deep bite primarily by proclination of the mandibular incisors. More on this later, but this observation is very important and is one of the keys to correcting deep bite treatment with Invisalign therapy.

Another study recently analyzed the effect of Invisalign therapy (G5) in skeletal deep bite cases [27]. They compared Invisalign (n=24, mean age 37) with fixed appliances (n=24, mean age 27) for overbites of 4.5mm in Invisalign and 4.6mm in fixed appliance groups. In the fixed appliance groups, .018 slots with reverse curve of Spee were used in the mandible along with Class II elastics. There were significant differences between the groups in ANB (Invisalign: -0.2° vs Fixed: -1.0°), ANS-Menton (Invisalign: +0.6mm vs Fixed: +1.9mm), U1/SN (Invisalign: +0.6° vs Fixed: +4.1°), L1/MP (Invisalign: -1.3mm vs Fixed: -2.0mm), IMPA (Invisalign: +0.3° vs Fixed: +3.5°), and overbite (Invisalign: -1.3mm vs Fixed: -2.0mm). In this study, there was a greater increase in vertical and intrusion of mandibular incisors with fixed appliances compared to Invisalign. They concluded that both systems were effective in opening the bite, but the fixed appliances produced more apparent skeletal changes.

In our recent study, we also compared the treatment effects of deep bite cases (overbite with more than 5mm and 60%) between Invisalign (n=25, mean age 23 years old) and fixed appliances (n=25, mean age 23 years old) [28]. When comparing the results of another study [27], we did not observe any change in the ANB of our fixed appliance group, but more change in the vertical (N-Me due to extrusion of the mandibular molars (Table 1). In the Invisalign group of this study, there was more uprighting of the maxillary incisors, some flaring of the mandibular incisors with no change in the vertical, and more overbite correction due to mandibular incisor intrusion (2.0mm).

Table 1. Comparison of cephalometric variables between Fixed and Invisalign groups study by Henick et al., and Fujiyama et al.

Appliance	Fixed		Invisalign	
Article	Henick	Fujiyama	Henick	Fujiyama
ANB(°)	-1.0*	-0.3	-0.2	0
U1/SN(°)	4.1*	-0.3	0.6	-3.2
L1/Mp(°)	3.5*	4.8	0.3	3.2
N-Me (ANS-Me)(mm)	1.9	3.4*	0.6	-0.4
U1/NF(mm)	-1.1	-0.5	-0.9	-1.1
U6/NF (mm)	0.6*	0.4	0.4	0.7
L1/Mp(mm)	-2.0*	-1.9	-1.3	-1.4
L6/Mp(mm)	0.8	2.5*	0.6	0.8
OB	-2.0*	-3.8	-1.3	-3.6

*Statistical difference compared with Invisalign group

Figure 18. Intraoral photos of initial phase of deep bite treatment with fixed appliances, A) Pre-treatment, B) 2-3 months after, C) 4 months after.

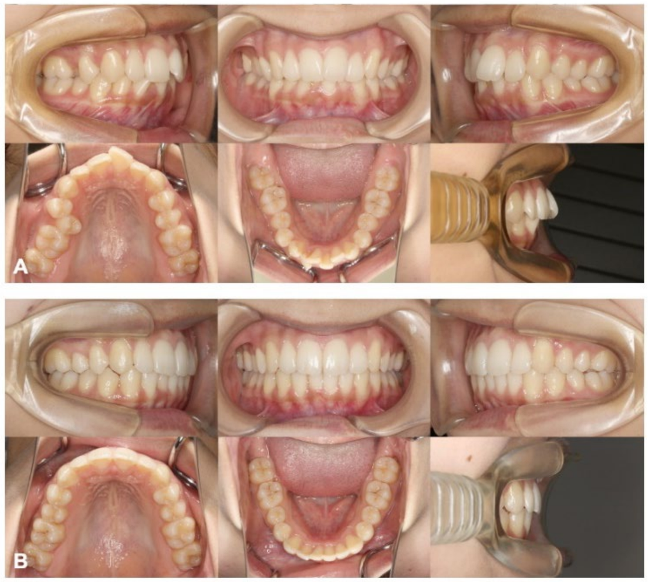

Figure 19. A) Pre-treatment and B) post-treatment intraoral photos.

One reason for the conflicted results between the past study and this one is the fixed appliance group in which we placed bite plate type Nance appliances and used RCOS NiTi wires in the mandible with the combination of posterior up-down elastics (Figure 18). This produced a substantial amount of extrusion of the molars. For Invisalign cases, I will report on some typical cases that were used in our study.

Case One

The first Invisalign case is a patient (20Y 7M) with Class II molars on one side, overbite of 5.9mm, with ANB of 7.6° and FMA of 20.6° (Figure 19 A). After 18 months of treatment, Class I molar and canine was achieved, with an FMA of 21.2 (+0.6)°, and reduced overbite of 2.3 (-3.6) mm due to 2.4mm of mandibular incisor intrusion and 1.5mm of maxillary intrusion (Figure 19 B). Twenty-five trays were planned initially with an additional 15 trays for refinement, for a total of 40 trays. Superimposition shows that deep bite was basically corrected due to maxillary and mandibular incisor intrusion (Figure 20).

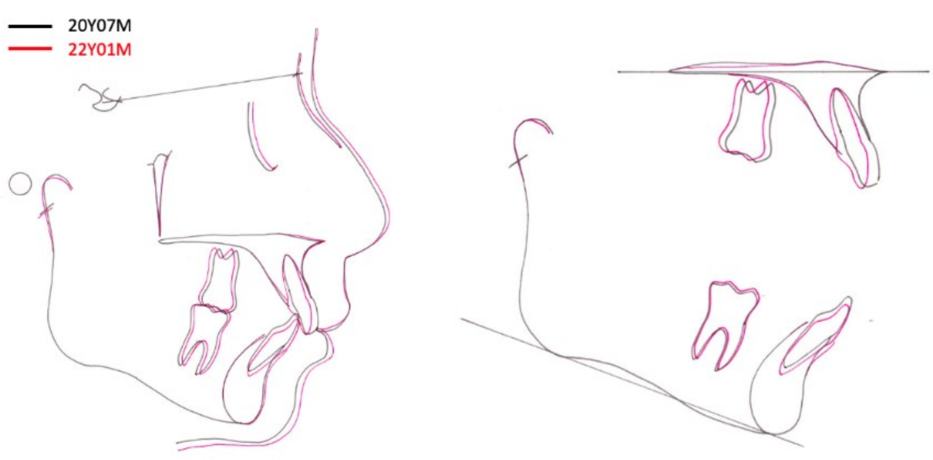

Figure 20. Superimposition of pre-treatment and post-treatment images.

Case Two

The second case is a patient (37Y 7M) with Class I molar relationship, uprighted maxillary incisors, overbite of 6.0mm, ANB of 5.8° and FMA of 28.1° (Figure 21 A). After 21 months of treatment, no change of vertical, with intrusion of maxillary (-1.1mm) and mandibular (-1.8mm) incisors resulted in reduction of 3.5mm of overbite (Figure 21 B). There were 17 initially planned trays with additional 18 trays for refinement, for a total of 35 trays. Superimposition shows that deep bite was basically corrected by maxillary and mandibular incisor intrusion with labial flaring (Figure 22).

Figure 21. A) Pre-treatment and B) post-treatment intraoral photos.

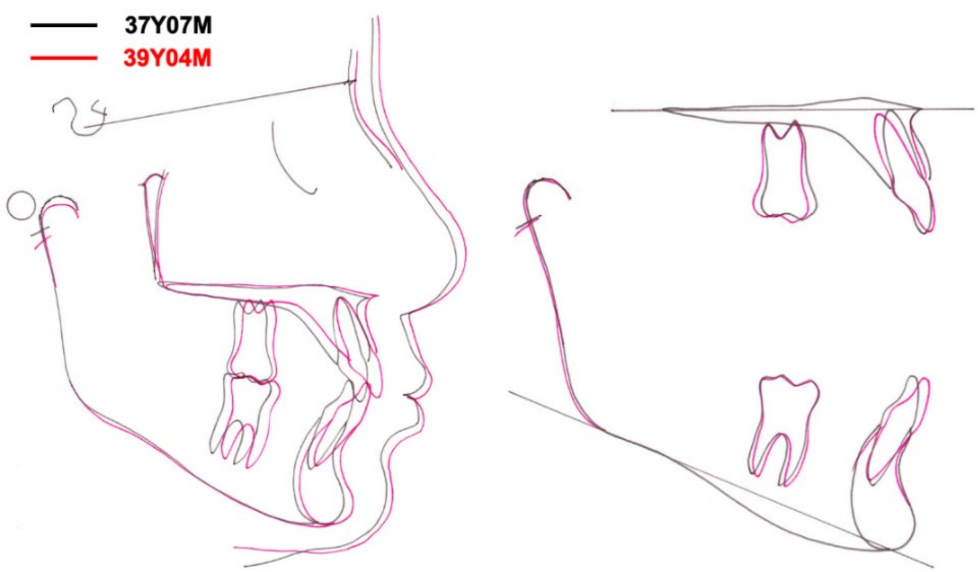

Figure 22. Superimposition of pre-treatment and post-treatment images.

Figure 23. A) Pre-treatment and B) post-treatment intraoral photos.

Case Three

The last case is a patient (19Y 4M) with Class I molar with spaced maxillary arch, overbite of 5.2mm, ANB of 3.8° and FMA of 23.4° (Figure 23 A). After 24 months of treatment, there was no change of vertical, with intrusion of maxillary (-1.0mm) and mandibular (-1.8mm) incisors that resulted in reduction of 2.5mm overbite (Figure 23 B). Twenty trays were initially planned with an additional 10 trays for refinement, for a total of 30 trays. However, there was a substantial amount of relapse after two years of retention resulting in about 50% deep bite (Figure 24 B) compared with immediately after debond (Figure 24 A). Thus, since the main correction of deep bite with Invisalign relies on incisor intrusion, relapse is a concern in these deep bite cases.

Figure 24. A) Intraoral photos of post-treatment and B) two years of retention.

Consider the following aspects when Clincheck planning for deep bite cases with Invisalign: 1) expand mandibular canine width by about 1.5-2.0mm, 2) space is required for absolute incisor intrusion, 3) try to avoid unnecessary IPR in the mandible, 4) maxillary molars need to be distalized (sequential distalization), 5) after distalizing the second premolar, start using Class II elastics, 6) use precision bite ramps if necessary, 7) always coordinate maxillary and mandibular arch forms. These are not all the factors; there are additional important features depending on the nature of the deep bite.

In conclusion, with Invisalign, 1) mandibular incisors can be intruded by about 2.0mm, 2) maxillary incisors can be intruded by about 1.0mm, and 3) expect only a minimum amount of molar extrusion with Invisalign therapy.

CONCLUSIONS

In conclusion, with the use of only wires, the correction of deep bite would be a combination of molar extrusion and incisor intrusion. The use of 3-piece segmented technique, intraoral appliance, and headgear could prevent reciprocal force during incisor intrusion in high angle deep bite cases. With the use of TADs (miniscrews), the main correction is by intrusion of incisors. The location and the number of miniscrews used in the anterior region could change the amount of flaring of incisors. Even with successful intrusion of incisors, substantial amount of root resorption may be seen especially with miniscrews. Aligner therapy could be an option to treat low angle deep bite cases with adequate ClinCheck prediction and the use of attachments.

REFERENCES

1. Brunelle JA, Bhat M, Lipton JA. Prevalence and distribution of selected occlusal characteristics in the US population, 1988–1991. J Dent Res 1996; 75:706–13.
2. Costa MD, Froes Júnior GRT, Santos CN. Evaluation of occlusal factors in patients with temporomandibular joint disorder. Dental Press J Orthod 2012;17(6):61-8.
3. Weiland FJ, Bantleon HP, Droschl H. Evaluation of continuous arch and segmented arch leveling techniques in adult patients—a clinical study. Am J Orthod Dentofacial Orthop 1996; 110(6):647-652.
4. Lindauer SJ, Lewis SM, Shroff B. Overbite correction and smile aesthetics. Semin Orthod 2005;11:62–66.
5. Valarelli FP, Carniel R, Cotrin-Silva PP, Patel MP, Cançado RH, Freitas KMS, de Freitas MR. Treatment of a Class II malocclusion with deep overbite in an adult patient using intermaxillary elastics and Spee curve controlling with reverse and accentuated archwires. Contemp Clin Dent 2017; 8(4):672-678.
6. Shroff B, Yoon WM, Lindauer SJ, Burstone CJ. Simultaneous intrusion and retraction using a three-piece base arch. Angle Orthod 1997; 67(6):455-61.
7. van Steenbergen E, Burstone CJ, Prahl-Andersen B, Aartman IH. The role of a high pull headgear in counteracting side effects from intrusion of the maxillary anterior segment. Angle Orthod 2004; 74(4):480-6.
8. Sosly R, Mohammed H, Rizk MZ, Jamous E, Qaisi AG, Bearn DR. Effectiveness of miniscrew-supported maxillary incisor intrusion in deep-bite correction: A systematic review and meta-analysis. Angle Orthod 2020; 90(2):291-304.

9. Watanabe H, Deguchi T, Hasegawa M, Ito M, Kim S, Takano-Yamamoto T. Orthodontic miniscrew failure rate and root proximity, insertion angle, bone contact length, and bone density. Orthod Craniofac Res 2013; 16(1):44-55.
10. Kravitz ND, Kusnoto B, BeGole E, Obrez A, Agran B. How well does Invisalign work? A prospective clinical study evaluating the efficacy of tooth movement with Invisalign. Am J Orthod Dentofacial Orthop 2009; 135(1):27-35.
11. Bernstein RL, Preston CB, Lampasso J. Leveling the curve of Spee with a continuous archwire technique: a long term cephalometric study. Am J Orthod Dentofacial Orthop 2007; 131(3):363-371.
12. Dake ML, Sinclair PM. A comparison of the Ricketts and Tweed-type arch leveling techniques. Am J Orthod Dentofacial Orthop 1989; 95(1):72-78.
13. Aydoğdu E, Özsoy ÖP. Effects of mandibular incisor intrusion obtained using a conventional utility arch vs bone anchorage. Angle Orthod 2011; 81(5):767-775.
14. Kinzel J, Aberschek P, Mischak I, Droschl H. Study of the extent of torque, protrusion and intrusion of the incisors in the context of Class II, division 2 treatment in adults. J Orofac Orthop 2002; 63(4):283-299.
15. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977; 72(1):1-22.
16. Alexander RG. The principles of the Alexander Discipline. Semin Orthod 2001; 7(2):62-66.
17. Ricketts RM. Bioprogressive therapy as an answer to orthodontic needs. Part I. Am J Orthod 1969; 70(3);241-268.
18. El Namrawy MM, Sharaby FE, Bushnak M. Intrusive Arch versus Miniscrew-Supported Intrusion for Deep Bite Correction. Open Access Maced J Med Sci 2019; 7(11):1841-1846.
19. Deguchi T, Murakami T, Kuroda S, Yabuuchi T, Kamioka H, Takano-Yamamoto T. Comparison of the intrusion effects on the maxillary incisors between implant anchorage and J-hook headgear. Am J Orthod Dentofacial Orthop 2008; 133(5):654-660.
20. Jain RK, Kumar SP, Manjula WS. Comparison of intrusion effects on maxillary incisors among mini implant anchorage, j-hook headgear and utility arch. J Clin Diagn Res 2014; 8(7):ZC21-24.
21. Kumar P, Datana S, Londhe SM, Kadu A. Rate of intrusion of maxillary incisors in Class II Div 1 malocclusion using skeletal anchorage device and Connecticut intrusion arch. Med J Armed Forces India 2017; 73(1):65-73.
22. Senisik NE, Turkkahraman H. Treatment effects of intrusion arches and mini-implant systems in deepbite patients. Am J Orthod Dentofacial Orthop 2012; 141(6):723-733.
23. Kanomi R. Mini-implant for orthodontic anchorage. J Clin Orthod 1997; 31(11):763-767.
24. Gu J, Tang JS, Skulski B, Fields HW, Jr., Beck FM, Firestone AR, Kim DG, Deguchi T. Evaluation of Invisalign treatment effectiveness and efficiency compared with conventional fixed appliances using the Peer Assessment Rating index. Am J Orthod Dentofacial Orthop 2017; 151(2):259-266.
25. Krieger E, Seiferth J, Marinello I, Jung BA, Wriedt S, Jacobs C, Wehrbein H. Invisalign® treatment in the anterior region: were the predicted tooth movements achieved? J Orofac Orthop 2012; 73(5):365-376.
26. Khosravi R, Cohanim B, Hujoel P, Daher S, Neal M, Liu W, Huang G. Management of overbite with the Invisalign appliance. Am J Orthod Dentofacial Orthop 2017; 151(4):691-699 e692.
27. Henick D, Dayan W, Dunford R, Warunek S, Al-Jewair T. Effects of Invisalign (G5) with virtual bite ramps for skeletal deep overbite malocclusion correction in adults. Angle Orthod 2021; 91(2):164-170.
28. Fujiyama K, Kera Y, Yujin S, Tanikawa C, Yamashiro T, Guo X, Ni A, Deguchi T. Comparison of clinical outcomes between Invisalign and conventional fixed appliance therapies in adult patients with severe deep overbite treated with nonextraction. Am J Orthod Dentofacial Orthop 2022; 161(4):542-547.

AN ARTIFICIAL INTELLIGENCE APPROACH TO DIAGNOSE ERUPTION DISORDERS?

Cristina Grippaudo, Rosalia Leonardi, Silvia Allegrini, Sylvia A. Frazier-Bowers

ABSTRACT

The management of eruption disorders provides a unique example of how the interplay between the clinical characterization and the use of biological markers (i.e., mutational analysis) can comprise a more sophisticated diagnostic rubric. As a result, the application of artificial intelligence software may be quite useful in diagnosing and treatment planning for malocclusions involving eruption disorders. In this overview, we explore advances made in the science of artificial intelligence and its applicability in dentistry and orthodontics, specifically. We juxtapose the fruitful possibilities with the inevitable challenges faced in definitively diagnosing eruption disorders such as ankylosis, impaction, primary failure of eruption, and severe delayed eruption. Our current rubric for diagnosis and management of eruption disorders remains a useful evidence-based approach to provide the best care for orthodontic problems involving an eruption defect. But the promise of an artificial intelligence application may improve our diagnostic acumen in the not-so-distant future.

KEY WORDS: Eruption, Primary Failure of Eruption, AI, Artificial Intelligence, Diagnostic Rubric

INTRODUCTION

One of the most consequential clinical situations in orthodontics is navigating anomalies in dental eruption (i.e., timing, sequence, and spatial capacity). We certainly cannot treat teeth orthodontically if they have not emerged into the dental arch. The resultant challenge extends from diagnosis to management and retention. To arrive at the most accurate diagnosis, the clinician must gather complete anamnestic data, including the position and development of the tooth or teeth with eruptive problems and the corresponding bony substrate. This systematic approach necessarily allows for distinguishing acquired problems of eruption from (e.g., obstruction of the eruptive path) from those that are genetic in origin. A logical first step - for either genetic or acquired eruption problems – is completing the clinical and radiographic examination. However, even prior to this step, it is critical that the medical and dental history assessment asks the question, “Is there a family history of eruption problems?” If there is compelling evidence of a family history, then a genetic origin must be considered. If you are unsure but suspect a genetic origin, tests for gene variants in the *PTH1R* gene should be performed. Finally, this systematic sequence must derive the proper therapeutic plan which considers timing and expected outcomes of the treatment course. Hence, the paradigm - proper diagnosis equals proper treatment.

A systematic approach that uses a diagnostic rubric undoubtedly offers effective management of malocclusion due to eruption failure (Figure 1). But the notion of deploying artificial intelligence (AI) to diagnose and treat eruption disorders offers an even more attractive possibility because it may speed up the accuracy and comparison of knowledge [1]. The clinical spectrum of eruption disorders is vast – ranging from rare genetic disorders to the more frequently observed impacted teeth. Specifically, maxillary canines are notably the most frequently impacted teeth, and therefore the most studied among eruption disorders. Therefore, while many aspects of impacted canine diagnosis and treatment have been described and vetted, other dental eruption problems, including ankylosis, primary failure of eruption (PFE), and severe delayed eruption, are more rare, less diagnosed, and therapeutically trickier. It raises the question of whether an AI approach might be the best solution for increasing excellence in patient care and provider confidence.

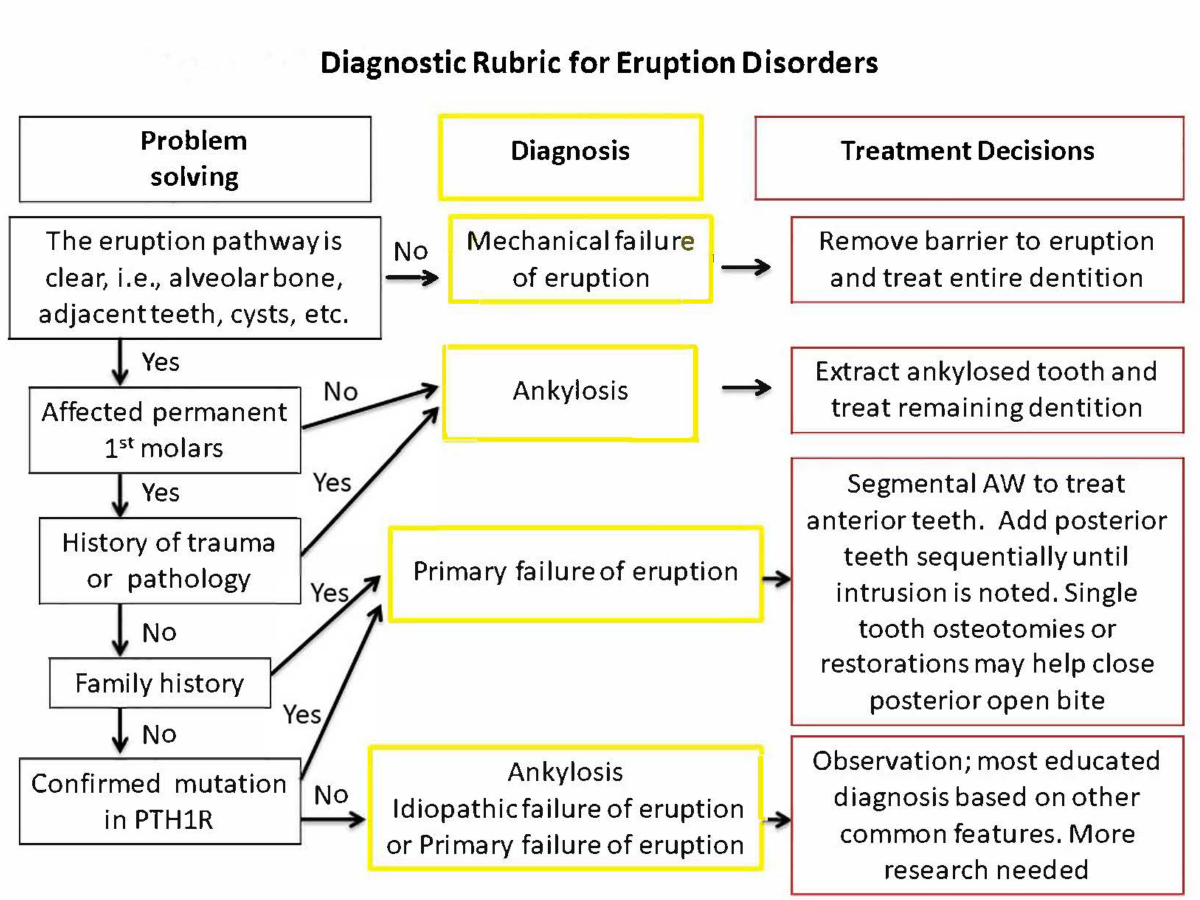

Figure 1. A diagnostic and treatment rubric can be used as an aid for the systematic decision-making process for eruption disorders. While there are limitations in the specificity, this tool is a necessary part of management of eruption disorders and may serve as a precursor for the development of a future AI approach.

AI IN DENTISTRY AND ORTHODONTICS

The advancement of technologies via AI available to medicine and dentistry has the potential to improve diagnostic and therapeutic processes. Before we explore the possibilities of AI and management of eruption disorders, we must frame this possibility in the context of what AI is, and in some cases – what AI is not. According to the Cambridge Dictionary, AI is defined as the study of how to produce machines that feature some of the qualities that the human mind has, such as the ability to understand language, recognize pictures, solve problems, and learn. Medicine has already taken up the challenge of introducing AI to support assessments for diagnosis, therapy, prognosis, and patient monitoring [2]. The introduction of computer science into the medical field was a decisive step that led to radical changes in clinical practice. In the early days, computers represented an irreplaceable means of managing archives, but their field of action now extends to operational procedures mainly because of AI [3]. Changing the way of communicating with technological devices represented a pivotal breakthrough in computer science. Now computers can be taught to learn according to patterns that can be traced back to human decisions. On one hand, there are vast improvements in training computers to think like humans. On the other hand, there was a sudden increase in the amount of available information, due to the broad diffusion of the internet and of internet-based media such as social networks, cloud computing, and big-data platforms.

The orthodontic specialty would benefit from AI as we continue to witness an inflection point transforming the way we practice orthodontics toward being completely digitally based. With the advent of more archived digital data, we can expect advances in orthodontics to be based on the processing and the correct interpretation of this vast information [4]. This unprecedented availability of information will undoubtedly impact the logic underlying decision-making processes; computerized systems designed to advise have gradually changed into systems providing ad-hoc information in decision-based (intelligent) tasks [5, 6]. The prospect of implementing an AI based diagnostic and treatment planning system in orthodontics that produces a responsible and reliable decision comparable to that of a human mind is tempting [7]. But the question of the extent to which we can expect the AI-derived decision to be equivalent to that of the orthodontic practitioner remains elusive. Irrespective of our aspirational goals surrounding personalized and precision orthodontics, it is useful to understand what components lead to the AI result. AI uses Machine Learning (ML) algorithms to process the input data and evaluate it, giving back a value that can then be interpreted by the AI, which processes the decision-making response [8, 9]. Realistically, we should always think about this as an intelligent system that effectively supports the clinician – somewhat creating a synergy – to implement their treatment options. ML, like human learning, ensures that the machine acquires experience from processing information and uses it in calculating the data entered subsequently. Depending on the type of data evaluated, there are different ML methods: supervised learning, unsupervised learning, semi-supervised learning, reinforcement learning, and self-learning [10].

The models used by ML for learning include artificial neural networks (ANNs), decision trees, support vector machines (SVMs), regression analysis, Bayesian networks, genetic algorithms (GA), and fuzzy logic. The methods of ML used most frequently are supervised learning and unsupervised learning. In the first case, which is used for classification, the machine compares the entered data with other data provided with a well-specified meaning. In the second case, there is no known reference for the input data, but the machine learns to organize the dataset by analyzing its structure and by making associations according to the recorded characteristics. This method is used to solve clustering or categorization problems.

In orthodontics, the classification systems have gradually acquired greater complexity due to the introduction of many elements aiming for an increasingly individualized diagnosis, which makes expected treatment outcomes more predictable. An example can be found in the classification of diagnostic data for the determination of the facial appearance using automatic systems. In one study of the 3D facial norms projects, genetic data and 3D images of the face were associated to identify the normal values of the facial proportions [11]. As improvements in facial recognition evolve through deep learning algorithms, we witness how these systems learn by recognizing a variety of shapes of known objects and the identification of the objects' individual characteristics. This forms the basis of automatic recognition and definition of anatomical parts. The AI method that resembles human reasoning, the expert system (ES) - based on a setting of pre-established rules (knowledge base), and on the use of the interference engine - this system deduces added information [12].

AI systems also use artificial neural networks (ANNs) to mimic the connections of human neurons, forming a network capable of sifting stimuli to produce a reaction based on a specific threshold. The discovery of neural networks and their application in many medical fields also represents a valuable method to give a logical explanation to some complex phenomena, such as language learning or reactivity to emotional stimuli [13, 14]. We have witnessed this in orthodontics as early as 1997 when Moss and colleagues described the effect of the functional matrix on the skeletal microunit [15]. If we flash forward some 25 years, Liu et al. [9], posited that the four major AI-driven tasks in dentistry are classification, regression, detection, and segmentation. We can also apply these four AI-driven tasks to orthodontic treatment. Classification tasks serve to diagnose skeletal type and predict bone age. Regression tasks can yield clinical decision-making, such as whether or not to extract teeth. Automatic identification of landmarks belongs to detection tasks, and the acquisition of tooth segmentation belongs to segmentation tasks.

To provide an idea of how AI is now a reality in the world of orthodontics, including dental eruption problems, we will show some examples of application in the fields of diagnosis and treatment planning, personalized treatment, clinical trial research, and smart electronic health records. The use of AI in orthodontic diagnosis has evolved, thanks to the digitization of diagnostic records. Orthodontic diagnosis is based on the observation and measurement of the dental arches and soft tissues of the face and on the cephalometric analysis. Today, we can make use of virtual models acquired with scanners, 3D photographs, and 2 dimensional (2D) and 3D digital radiographs. Using dedicated software to run the analyses of the models of dental arches as well as of the images of the face allows us to perform measurements with high precision and calculation speed [16]. This is an advantage, but it doesn't change traditional diagnostic procedures very much. The possibility of matching 3D radiographs offers a huge advancement - allowing the clinician to view all the structures of the patient's craniofacial region [17].

The use of algorithms with human-like ability to identify structures was developed to facilitate cephalometry [18]. For instance, in the study of 3D cone beam computed tomography (CBCT) radiographs, the clinician may need to isolate specific anatomic structures, using segmentation methods. The steps to complete this include identification of the structures to be segmented in the various layers that make up the 3D image, and using programs to reconstruct them, highlighting them in their entirety. Through deep learning methods, researchers have also developed the possibility of proceeding with automatized segmentation, which is precise and less time-consuming [19, 20, 21, 22, 23]. We posit that this same technique can be applied to the identification (location and prognosis) of impacted teeth. Impacted maxillary canines can be precisely located and segmented to understand their spatial relationships and plan therapeutic interventions [24, 25]. By segmenting and moving the dental elements, we can simulate an orthodontic treatment outcome by obtaining a virtual set up and corresponding predicted movement of crowns and roots leading to programming of treatment that is more effective [26].

Our studies have highlighted that the diagnosis of dental eruption problems is associated with specific genetic variants. Genetic diagnosis is particularly amenable to AI-based applications, but limitations remain. According to Dias and Torkamani, mimicking human intelligence is the inspiration for AI algorithms, but AI applications in clinical genomics tend to target tasks that are impractical to perform using human intelligence and are error prone when addressed with standard statistical approaches [27]. Many of the techniques employing AI have been adapted to address the various steps involved in clinical genomic analysis, including variant calling, genome annotation, variant classification, and phenotype-to-genotype correspondence. Eventually, they can also be applied for genotype-to-phenotype predictions.

By consulting the genetic databases, we can determine the variant's classification and additional information toward understanding the etiopathogenetic mechanisms. For instance, there are methods for the classification of nonsynonymous variants by using ML algorithms [28]. In this way, the effect of the variants in the proteins production is predictable, and it is therefore possible to evaluate its pathogenicity.

Moreover, AI technologies make it possible to evaluate the effect of non-coding variations on the splicing disruption [29]. We can then use electronic health records (EHR) to discern the phenotype-genotype correlation in rare disease cases. Dental eruption problems that are often genetic in origin can benefit from this system, which offers a potential advantage over the simple flow chart (Figure 1).

CONCLUSIONS

AI will soon become a necessary part of our everyday functions, and this is certainly true for healthcare, including dentistry and orthodontics. Technological advances have made it possible to exchange data and information rapidly with the backdrop of a society that is constantly connected (i.e., social media, email, electronic patient records, etc.). Our role as orthodontists must be to confront and understand AI technology and therefore derive all the benefits that may result. The prudent clinical orthodontist must preserve their primary role in the conduct of orthodontic treatment while mitigating the misuse of AI-based applications that may lead to DIY orthodontics or non-specialty led treatment.

It is likely that the human experience with AI will reflect the challenge to keep pace with the stratification and interpretation of information, hence the full implementation of AI-based diagnostics may be still out in the future, and the potential benefits may be correspondingly slow to come to fruition. The challenge for AI-assisted orthodontics will be the successful transfer of the clinician's way of thinking to the machine. Specifically, the framework that includes orthodontic biomechanics must also consider the patient in their biological and psychophysical entirety to implement therapies that increasingly respond to the P4 principle [30]. Furthermore, the prohibitive costs of technological development suggests that the demand for clinical decision supported systems (CDSS) in dentistry will not be immediate. Indeed, some orthodontic clinician practices have not yet become fully digital to avail of the AI-based diagnostics and therapeutics. Nonetheless, it is realistic for the orthodontic profession to work toward the eventual integration of the clinician's experience and knowledge with the potential of the AI application.

The combination of a complex and rare presentation of some eruption disorders necessitates that an AI-based diagnostic may be difficult to achieve. This reality is further confounded by the limited number of cases available with which to train the machine (i.e., ML) and therefore create meaningful AI-based clinical tools. At minimum, it is reasonable to expect with continued research in the field that we will have the capacity to collect sufficient data toward AI applications for differential diagnosis.

REFERENCES

1. Bichu YM, Hansa I, Bichu AY, Premjani P, Flores-Mir C, Vaid NR. Applications of artificial intelligence and machine learning in orthodontics: a scoping review. Prog Orthod 2021; 22(1):18.
2. Combi C. Editorial from the new Editor-in-Chief: artificial intelligence in medicine and the forthcoming challenges. Artif Intell Med 2017; 76:37-39.
3. Leonardi RM. 3D imaging advancements and new technologies in clinical and scientific dental and orthodontic fields. J Clin Med 2022; 11(8):2200.
4. Allareddy V, Rengasamy Venugopalan S, Nalliah RP, Caplin JL, Lee MK, Allareddy V. Orthodontics in the era of big data analytics. Orthod Craniofac Res 2019; 22 Suppl 1:8-13.
5. Patel VL, Shortliffe EH, Stefanelli M, Szolovits P, Berthold MR, Bellazzi R, Abu-Hanna A. The coming of age of artificial intelligence in medicine. Artif Intell Med 2009; 46(1):5-17.
6. Obermeyer Z, Emanuel EJ. Predicting the future - big data, machine learning, and clinical medicine. N Engl J Med 2016; 375(13):1216-1219.

7. Gili T, Di Carlo G, Capuani S, Auconi P, Caldarelli G, Polimeni A. Complexity and data mining in dental research: a network medicine perspective on interceptive orthodontics. Orthod Craniofac Res 2021; 24 Suppl 2:16-25.
8. Asiri SN, Tadlock LP, Schneiderman E, Buschang, PH. Applications of artificial intelligence and machine learning in orthodontics. APOS Trends in Orthodontics 2020; 10(1):17-24.
9. Liu J, Chen Y, Li S, Zhao Z, Wu Z. Machine learning in orthodontics: challenges and perspectives. Adv Clin Exp Med 2021; 30(10):1065-1074.
10. Sidey-Gibbons JAM, Sidey-Gibbons CJ. Machine learning in medicine: a practical introduction. BMC Med Res Methodol 2019; 19(1):64.
11. Weinberg SM, Raffensperger ZD, Kesterke MJ, Heike CL, Cunningham ML, Hecht JT et al. The 3D Facial Norms Database: Part 1. A web-based craniofacial anthropometric and image repository for the clinical and research community. Cleft Palate Craniofac J 2016; 53(6):e185-e197.
12. Xie X, Wang L, Wang A. Artificial neural network modeling for deciding if extractions are necessary prior to orthodontic treatment. Angle Orthod 2010; 80(2):262-266.
13. LeCun Y, Bengio Y, Hinton G. Deep learning. Nature 2015; 521(7553):436-444.
14. Dayhoff JE. Neural network architectures: an introduction. New York, N.Y.: Van Nostrand Reinhold; 1990.
15. Moss ML. The functional matrix hypothesis revisited. 2. The role of an osseous connected cellular network. Am J Orthod Dentofac Orthop 1997; 112(2):221-226.
16. Lee RJ, Pi S, Park J, Nelson G, Hatcher D, Oberoi S. Three-dimensional evaluation of root position at the reset appointment without radiographs: a proof-of-concept study. Prog Orthod 2018; 19(1):15.
17. Mangano C, Luongo F, Migliario M, Mortellaro C, Mangano FG. Combining intraoral scans, cone beam computed tomography and face scans: the virtual patient. J Craniofac Surg 2018; 29(8), 2241–2246.
18. Kunz F, Stellzig-Eisenhauer A, Zeman F, Boldt J. Artificial intelligence in orthodontics: evaluation of a fully automated cephalometric analysis using a customized convolutional neural network. J Orofac Orthop 2020; 81(1):52-68.
19. Leonardi R, Giordano D, Maiorana F. An evaluation of cellular neural networks for the automatic identification of cephalometric landmarks on digital images. J Biomed Biotechnol 2009; 2009:717102.
20. Leonardi R, Giordano D, Maiorana F, Spampinato C. Automatic cephalometric analysis. Angle Orthod 2008; 78(1):145-151.
21. Hwang HW, Park JH, Moon JH, Yu Y, Kim H, Her SB et al. Automated identification of cephalometric landmarks: part 2- might it be better than human? Angle Orthod 2020; 90(1):69-76.
22. Park JH, Hwang HW, Moon JH, Yu Y, Kim H, Her SB et al. Automated identification of cephalometric landmarks: part 1-comparisons between the latest deep-learning methods YOLOV3 and SSD. Angle Orthod 2019; 89(6):903-909.
23. Spampinato C, Palazzo S, Giordano D, Aldinucci M, Leonardi R. Deep learning for automated skeletal bone age assessment in X-ray images. Med Image Anal 2017; 36:41-51.
24. Laurenziello M, Montaruli G, Gallo C, Tepedino M, Guida L, Perillo L et al. Determinants of maxillary canine impaction: retrospective clinical and radiographic study. J Clin Exp Dent 2017; 9(11):e1304-e1309.
25. Chen S, Wang L, Li G, Wu TH, Diachina S, Tejera B et al. Machine learning in orthodontics: introducing a 3D auto-segmentation and auto-landmark finder of CBCT images to assess maxillary constriction in unilateral impacted canine patients. Angle Orthod 2020; 90(1):77–84.
26. Kim J, Chun YS, Kim M. Accuracy of bracket positions with a CAD/CAM indirect bonding system in posterior teeth with different cusp heights. Am J Orthod Dentofacial Orthop 2018; 153(2):298-307.
27. Dias R, Torkamani A. Artificial intelligence in clinical and genomic diagnostics. Genome Med 2019; 11(1):70.

28. Tang H, Thomas PD. Tools for predicting the functional impact of nonsynonymous genetic variation. Genetics 2016; 203(2):635–647.
29. Chatterjee S, Ahituv N. Gene regulatory elements, major drivers of human disease. Annu Rev Genomics Hum Genet 2017; 18:45–63.
30. Moore JH, Boland MR, Camara PG, Chervitz H, Gonzalez G, Himes BE et al. Preparing next-generation scientists for biomedical big data: artificial intelligence approaches. Per Med 2019; 16(3):247-257.

USE OF CONE BEAM COMPUTED TOMOGRAPHY AND INTRAORAL SCANS TO ASSESS LATERAL OPEN BITES IN PATIENTS WITH OSTEOGENESIS IMPERFECTA

Jean-Marc Retrouvey & Juliana Marulanda

ABSTRACT

Osteogenesis imperfecta is a rare genetic disease of varying severity that mainly affects the production of type I collagen. It may result in bone fragility, multiple long bone fractures, cervical compression fractures, dentinogenesis imperfecta and severe malocclusions. These malocclusions are atypical and involve Class III intermaxillary relation and are often associated with impacted, missing, or deformed teeth. Either anterior or posterior multiple crossbites are present, and a large percentage of these malocclusions involve the presence of lateral open bites, creating an absence of posterior occlusal contacts. Orthodontic treatment is very challenging due to dentinogenesis imperfecta, long term use of bisphosphonates, unpredictable tooth movement, and great difficulty in performing orthognathic surgery. The objective of this chapter is to describe the typical malocclusion found in patients with moderate-to-severe (type III and IV) OI. A case report of a patient with Type IV OI with a large bilateral posterior open bite is presented. Future potential treatment approaches are also described.

KEY WORDS: Osteogenesis Imperfecta, Craniofacial, Open Bite, Malocclusion

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INTRODUCTION

Osteogenesis imperfecta (OI) represents a group of genetic connective tissue disorders characterized by low bone mass and bone fragility. The majority (85-90%) of OI cases are caused by dominant pathogenic variants in *COL1A1* or *COL1A2*, which in turn, affect the formation of collagen type I [1]. The remaining cases of OI are caused by pathogenic variants in non-collagenous genes that encode proteins involved in collagen assembly, crosslinking, trafficking, and modification. Furthermore, mutations in genes that encode for transcription factors and signaling molecules related to osteoblast function and extracellular matrix mineralization can also cause OI [2]. In 1979, Dr. Sillence classified OI into distinct types: type I with a mild non-deforming phenotype, type II being perinatally lethal, type III the most severe surviving type and type IV with moderate phenotype, intermediate between type I and type III [2, 3]. Currently, more than 19 types of OI have been described [4].

The etiology of the most common types of OI is a dominantly inherited point mutation in a glycine residue on the alpha chains of COL1A1 and COL1A2 that severely affects the formation of the collagen triple-helix, directly affecting bones and other tissues rich in this protein [1, 5]. These genetic disorders result in shorter than normal patients, multiple fractures of long bones, cervical compression fractures, hearing loss, blue sclera, pulmonary dysfunction and Dentinogenesis imperfecta (DI) [6, 7].

Bisphosphonates are the therapy of choice to alleviate the symptoms of OI and reduce the chance of long bone and vertebral compression fractures [8].

Craniofacial and dental manifestations are varied but the most frequent characteristics are platybasia, a Class III malocclusion caused by a retrognathic and retropositioned maxilla, impacted and missing teeth as well as DI [9-11]. Lateral open bites are a frequent finding. Their nature and clinical appearance are variable as posterior teeth are frequently misaligned in the vertical dimension. Lateral open bites, an uncommon finding in the general population, affect the OI population to varying degrees [12]. Correction of this condition poses unique challenges as complex treatment approaches are required. The objectives of this pilot project were to measure lateral open bites in a group of OI type III and IV patients as a baseline for the creation of therapeutic approaches.

Osteogenesis imperfecta

Osteogenesis imperfecta (OI), or brittle bone disease, was first described by Lobstein in 1835 [13]. This disorder of genetic origin is caused mainly by mutations in the *COL1A1* and *COL1A2* genes which encode for collagen type I α1 and α2 chains. A glycine substitution in the triple helix of collagen is the main factor involved in OI [1].

OI type I is the least severe and is usually caused by mutations that result in *COL1A1* haploinsufficiency, or smaller amounts of normally formed collagen type I in bone and expressing tissues. Affected patients present with frequent fractures of long bones, blue sclera, and about 25% of OI type I patients will present with DI of various degrees of severity [1]. The most common malocclusion presented is a moderately severe Class III malocclusion caused by a hypoplastic and retropositioned maxilla [14]. Type II is usually lethal at birth or during the perinatal period due to the extreme severity of the syndrome [5].

Type III is the most severe type of survivable OI and is characterized by a short stature and lack of ambulatory movement. Patients with type III are usually wheelchair bound and present a significant amount of long bone fractures, compression fractures of the spine, severe scoliosis, and respiratory malfunction. At the craniofacial level, they present DI with severe class III malocclusions associated with impacted and missing teeth [10, 11]. The malocclusion is atypical as it is characterized by significant cross bites but also by lateral open bites of unknown origin. These patients often present with sleep apnea either of central or obstructive origin, and their nasal septum is deviated in most of the OI type III patients [9]. The cranial base also presents with significant anomalies, which may preclude to severe craniocervical issues that can be life threatening, such as basilar invagination and platybasia [9].

Type IV OI is considered of moderate severity, characterized by ambulatory and taller patients when compared to OI III. However, OI type IV is somewhat difficult to distinguish from type III at the craniofacial and craniocervical level since the face and skull have been shown to be independently affected. Thus, the craniofacial involvement does not correlate with the postcranial phenotype and OI type [9].

OI type V is much less common and is caused by a mutation in the gene *IFMIT5*. The manifestations are comparable to OI type IV but without blue sclera or DI. The affected patients may present hyperplastic callus formation. Their craniofacial phenotypes are highly variable but are associated with multiple missing teeth and a Class III malocclusion with or without an associated lateral open bite [15, 16].

Other non-collagenous forms of OI are mainly associated with an autosomal recessive mode of inheritance [17]. Mutations in *SERPINF1* (OI type VI) cause defects in bone mineralization, mutations in

CRTAP (OI type VII), *LEPRE1* (OI type VIII) and *PP1B* (OI type IX) cause collagen 3-hydroxylation defects, and mutations in *SERPINH1* (OI type X) and *FKBP10* (OI type XI) result in collagen folding defects. Pathogenic variants in *BMP1* (OI type XII) result in collagen processing defects. More recently, mutations in *SP7*, *WNT1* have been associated with osteoblast differentiation defects that also cause OI [7, 17]. Their skeletal involvement ranges in severity, often from mild to moderate. The craniofacial phenotype will not be discussed in this chapter because they are much rarer types of OI and very few reports have been produced in the literature describing them.

THERAPEUTIC APPROACH

The therapeutic approach involves the use of different intravenous (IV) bisphosphonates beginning at birth and continuing until late adolescence. IV bisphosphonate infusions have significantly increased the quality of life of the patients by increasing bone density, thus reducing the number of long bone and vertebral compression fractures. However, bisphosphonates present significant side effects and their influence on craniofacial development of OI patients has not been well studied. Very few comparison studies have been published as few non-bisphosphonate cohorts have been studied for this aspect of the disease.

The fact that several teeth are missing or poorly shaped and positioned in the more severe OI types is a contributing factor and presents significant challenges for the clinician. The use of IV bisphosphonates makes the therapy riskier and many practitioners shy away from treating these patients due to the complexity and unpredictability of the correction and the inherent risk of treating patients with IV bisphosphonates. It is worth noting that the half life of IV phosphonate is exceptionally long and their use has been associated with osteonecrosis of the jaw (ONJ). However, patients affected by ONJ are usually immunocompromised, require head and neck irradiation, are diagnosed with a local malignancy, and are under chemotherapy or glucocorticoid treatment [18]. High-dose IV bisphosphonates have been identified as a risk factor for ONJ in patients with cancer. Conversely, low-dose bisphosphonates used in patients with osteoporosis or other metabolic bone diseases like OI have not been causally linked to the development of ONJ [18, 19]. It appears then, that the use of IV bisphosphonates poses no significant risk of ONJ in patients with OI and is warranted due to the positive effect they have on the quality of life of patients with OI [8].

Additional antiresorptive drugs are currently being tested in children with OI. Denosumab consists of an antibody against RANKL that inhibits osteoclast differentiation and activity. Positive treatment effects such as a decrease in bone metabolism markers and an increase in bone mineral density have been reported [20].

Anabolic drugs are also under evaluation, as they promote the formation and function of osteoblasts instead of inhibiting the action of osteoclasts. For instance, anti-sclerostin antibody therapy has been tested in Phase 2 clinical trials with promising results in adults with moderate OI [21]. However, all these therapeutic approaches are in their initial stages and will not be discussed in this paper, as their effects on the craniofacial complex development is still unknown.

Thanks to the support of the National Institutes of Health (NIH), the Brittle Bone Disorders Consortium, and the Osteogenesis Imperfecta Foundation, a database of 75 patients affected with moderate-to-severe OI (type III and IV) was created to study their craniofacial phenotypes. The database consisted of extraoral and intraoral photographs, intraoral scans, and cone beam computed tomography

(CBCT) images. Findings of this NIH-sponsored research were published but the summary indicated that patients presenting with OI types III and IV have significant craniofacial anomalies. Abnormal cranial vault development results in wider intertemporal distance and a triangular face. A study using artificial intelligence to quantify the differences in facial anomalies between types I, III and IV showed that the phenotype was expressed in the same manner, but the severity of the deviation was related to the OI type [22]. The differences were variable and usually type IIIs were much more affected than type IVs, which were also more affected than type I. Frontal bossing has been reported but not quantified. Significant atypical malocclusions are present and have been described in a qualitative manner. Using CBCT and intraoral scans allowed for a quantitative assessment of the craniofacial deformities and malocclusions. One consistent finding in patients with moderate-to-severe OI at the occlusal level was the presence of severe lateral open bite affecting mastication and quality of life of these patients.

CBCT has allowed for visualization of the craniofacial structure in three dimensions of space to allow for a more precise assessment of the craniofacial anomalies and malocclusion. The CBCT analysis done by an oral radiologist usually starts from the cranial base and progresses to the bony structures and the airway. An important but often overlooked aspect of OI is the presence of platybasia, basilar impression, and basilar invagination. These conditions can lead to central sleep apnea (CSA) and may be lethal. It is therefore very important for the orthodontist obtaining a CBCT image of patients with OI to send the image to an oral radiologist for a final diagnosis of the overall craniofacial condition of the patient, not just the malocclusion [23].

In severe cases, CBCT reveals a deformed cranial vault with an enlarged inter-temporalis distance and a triangular appearance in the transverse plane. The maxilla is hypoplastic and retropositioned while the mandible is usually of normal size with a normally developed basal bone. The dental alveolar processes are underdeveloped due to a significant number of missing and impacted teeth. For instance, a study published by our group suggests that each OI patient has on average 2.4 missing teeth, with premolars being the most commonly missing teeth and the upper second premolars the most commonly unerupted teeth [11]. This lack of vertical development is particularly apparent in the posterior segments resulting in lateral open bites. It is important to determine whether the etiology of the lateral open bite is of dental or skeletal origin. In some cases, a combination of dental and skeletal etiologies may be present.

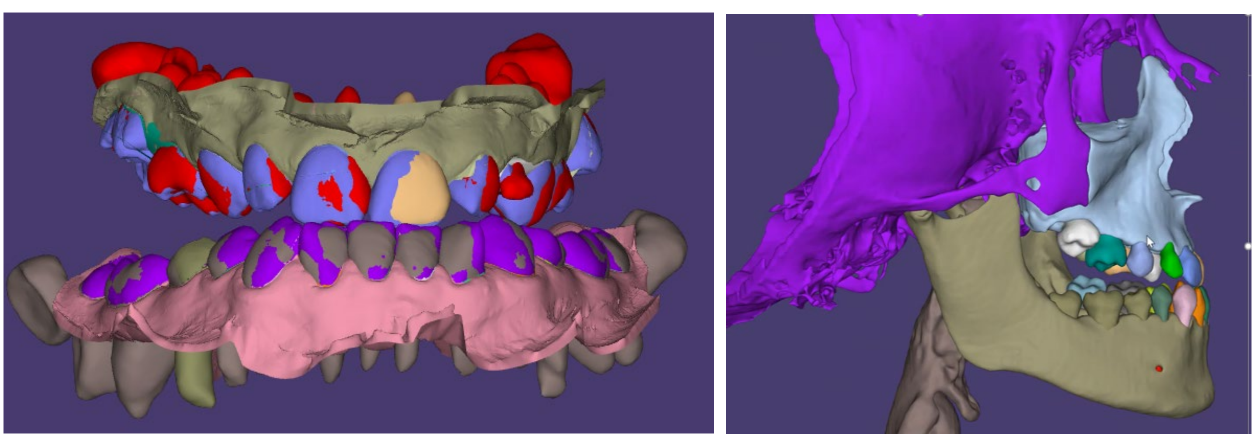

Figure 1. Intraoral scan rendering in STL file allowing a detailed assessment of the dental anatomy and malocclusion.

The stereolithography (STL) files obtained from the intraoral scans allow for a more precise assessment of the dental anatomy and of the malocclusion at the occlusal level (Figure 1). The possibility of fusing these STL files to the CBCT DICOM data allows for a perfect orientation of the STL files derived from the intraoral scans. Using both CBCT data and the STL files in this manner allows for a complete assessment of the cranial base anomalies. Assessment of the malocclusion using the cranial base and occlusal plane as reference planes is the best approach to fully quantify the malocclusion at the individual dental and skeletal levels.

Using neural networks and artificial intelligence, it is now possible to precisely segment all structures of the craniofacial complex including the dentition, maxilla, mandible, temporomandibular joints, and all craniofacial bones. Being able to segment these bony and dental structures and import them into simulation software allows for virtual and individualized correction of the malocclusion [24]. Valuable 3D data of the needed movements are easily obtained and referenced to specific planes to quantify the full correction of these challenging malocclusions at the dental and skeletal levels. All movements are first measured using Euclidean coordinates in 3D (Figure 2). Eventually, volumetric assessments and displacements, as well as statistical shape analysis, will be used to further improve the accuracy of the correction of these malocclusions in a very predictable manner.

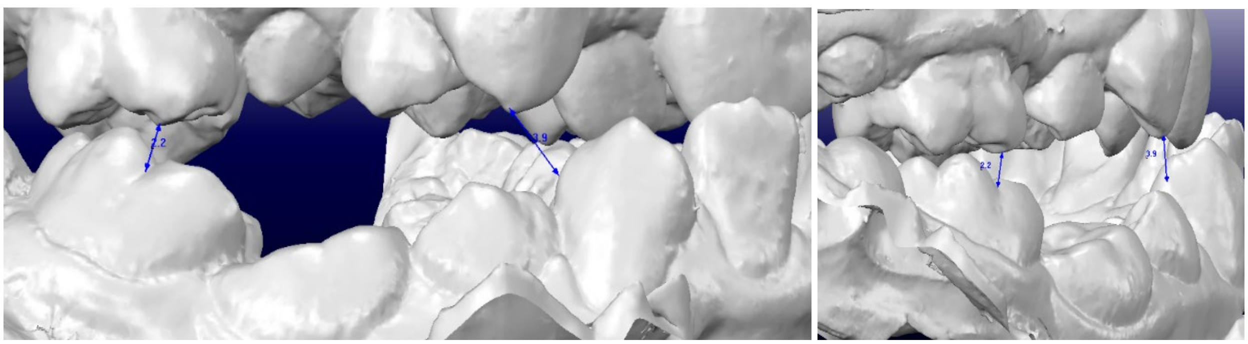

Figure 2. Euclidian distances measured with rendering of intraoral scans (Ortho-CAD software).

A literature review did not allow us to determine a precise quantitative way to assess the lateral open bite dimensions as very few reports have been published on this condition except for lateral open bites involving primary failure of eruption [25].

Several studies have reported severe posterior lateral open bites in the moderate-to-severe OI type III and IV malocclusions [12, 26]. These lateral open bites result in masticatory deficiencies and may result in increased tooth fractures of the anterior teeth due to the extra pressure these teeth must absorb during mastication (Figure 3).

Patients with OI present with significantly severe malocclusions and their manifestations are much more complex than the average malocclusions encountered in the general population. Class III malocclusions affect less than 3% of all Caucasians and up to 20% of the Japanese population but are rarely associated with missing, impacted, or poorly shaped teeth. Lateral open bites associated with posterior or anterior crossbites that are frequently found in the OI population make the malocclusion a unique challenge. Therefore, most of the malocclusions involve the whole craniofacial complex and ideally require complex craniofacial surgeries, which are unfortunately contraindicated.

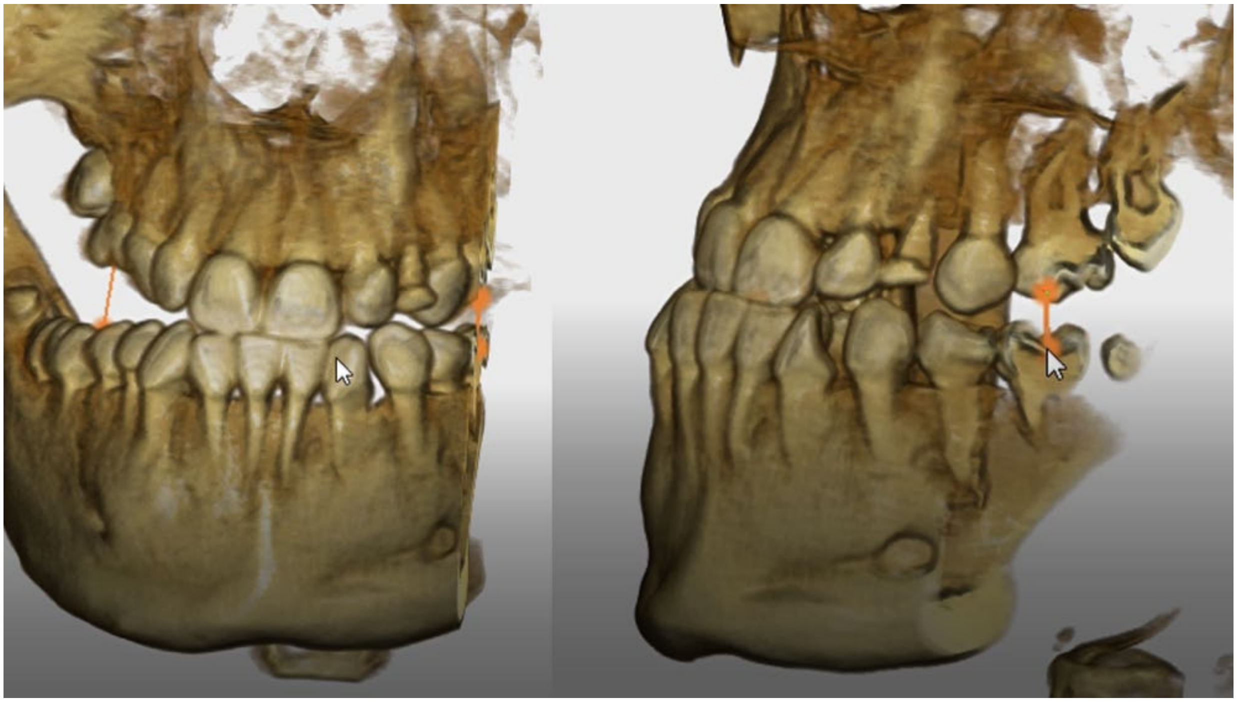

Figure 3. Example of a lateral open bite in a OI type IV subject (Drishti Software)

The goal of the orthodontic treatment should be to intercept and improve, but not necessarily to fully correct, these malocclusions, so the most appropriate objectives for many of these patients would be to improve masticatory function, optimize aesthetics, and prepare the dentition for prosthodontic treatments whenever possible due to the presence of DI.

Due to limited knowledge and experience demonstrated by clinicians in addressing these unique malocclusions, a pilot study addressing some of their orthodontic needs was designed. The PAR index, a universally accepted index of malocclusion [27], was used to evaluate whether this index could be reduced significantly to a more manageable level. Also, there was an assessment of whether the teeth would move in a satisfactory manner, given that the alveolar bone was of poor quality, plus the fact that these patients are treated with IV bisphosphonates. The modality of choice was Invisalign appliances, which apply force to teeth using preprogrammed plastic aligners, usually in 0.2 to 0.3 mm increments. Before pursuing this study, patients without significant DI and those interested in the Invisalign therapy were selected. The presence of a lateral open bite was mandatory.

CASE REPORT

A 14-year-old male patient presented to our clinic with a diagnosis of OI type IV and a request to improve his malocclusion. He presented with a severe lateral open bite with only one central upper incisor and one lower central incisor in occlusal contact. The lateral open bite was 8.5 mm in the molar area (Figure 4).

Figure 4. Lateral and frontal intraoral pictures in occlusion in an OI type IV patient before Invisalign treatment.

Facially, he presented with a typical but very moderate OI type IV phenotype without severe frontal bossing or a triangular face. His profile was slightly concave due to a retrognathic maxilla and a normally projected mandible. His face height was normal, and his smile line indicated that the upper incisors were positioned too vertically and the amount of incisor showing at rest was 0-1 mm during an unstrained smile.

As the patient exhibited some mild form of DI, especially visible on the panoramic x-ray, it was decided and in accordance with the wishes of his mother, to use the Invisalign technique. Attachments were planned as well as buttons for elastics, but they could be easily ground out with a high-speed handpiece, unlike the limitations with metal brackets which would have required debonding with a significant shearing force. The Invisalign appliance had a proven potential to move the teeth in their correct anteroposterior position, but the effect of elastics on such a large lateral open bite was unknown. The plan was to create overjet and overbite and promote the extrusion of the upper molars and use them as distractors to bring down the dentoalveolar bone to correct the open bite or at least minimize the conditions.

Intraoral scans plus a full set of records were obtained and a detailed prescription was sent to Invisalign to overcorrect the malocclusion. The virtual simulation (Clinchek-Invisalign) was accepted, the attachments and buttons were bonded to the teeth, and the patient was advised to wear light (30z-5-16 Dentsply) intermaxillary elastics and to wear the aligners 24 hours a day except when eating.

The objectives of treatment were to correct the end-to-end anterior occlusion and to correct the lateral open bites as much as possible without correcting the posterior crossbites. Unfortunately, correcting the transverse maxillary deficiency as initially planned would require some form of surgical intervention, but the parents rejected it. We explained to them that placing the upper and lower dentition in contact even in a cross-bite situation would still improve the occlusion dramatically and that an occlusal rehabilitation using composite resins or onlays would be necessary in the future to get a more stable occlusion. We also advised the patient that long-term retention with light nighttime wear of elastics would be necessary.

It took three refinements (corrective virtual simulation obtained from new intraoral scans) of the Invisalign procedures to get to a satisfactory result. The open bites were closed, occlusal contacts were established, and positive overbite and overjet were obtained. The patient was referred to the prosthodontist to modify the occlusal surfaces to provide a complete dental occlusion (Figure 5).

Figure 5. Lateral and frontal intraoral pictures in occlusion in a patient with OI type IV after 18 months of Invisalign treatment.

The result was deemed satisfactory even though the posterior crossbites were not corrected. The patient was very happy with the masticatory function that he had gained and with the improved aesthetics due to the positive overbite and overjet. The goal of improving function was achieved and the patient reported having a much-improved quality of life (Figures 6 and 7).

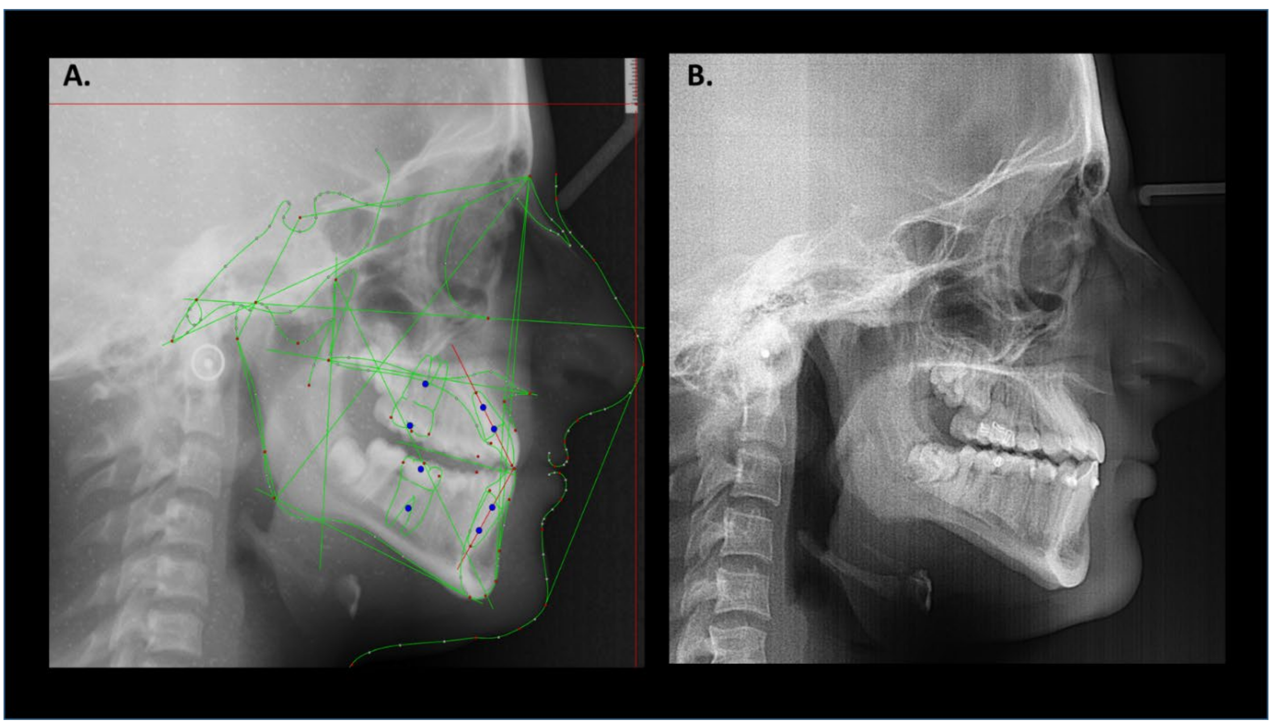

Figure 6. Cephalometric radiographs before (A) and after (B) completion of Invisalign treatment.

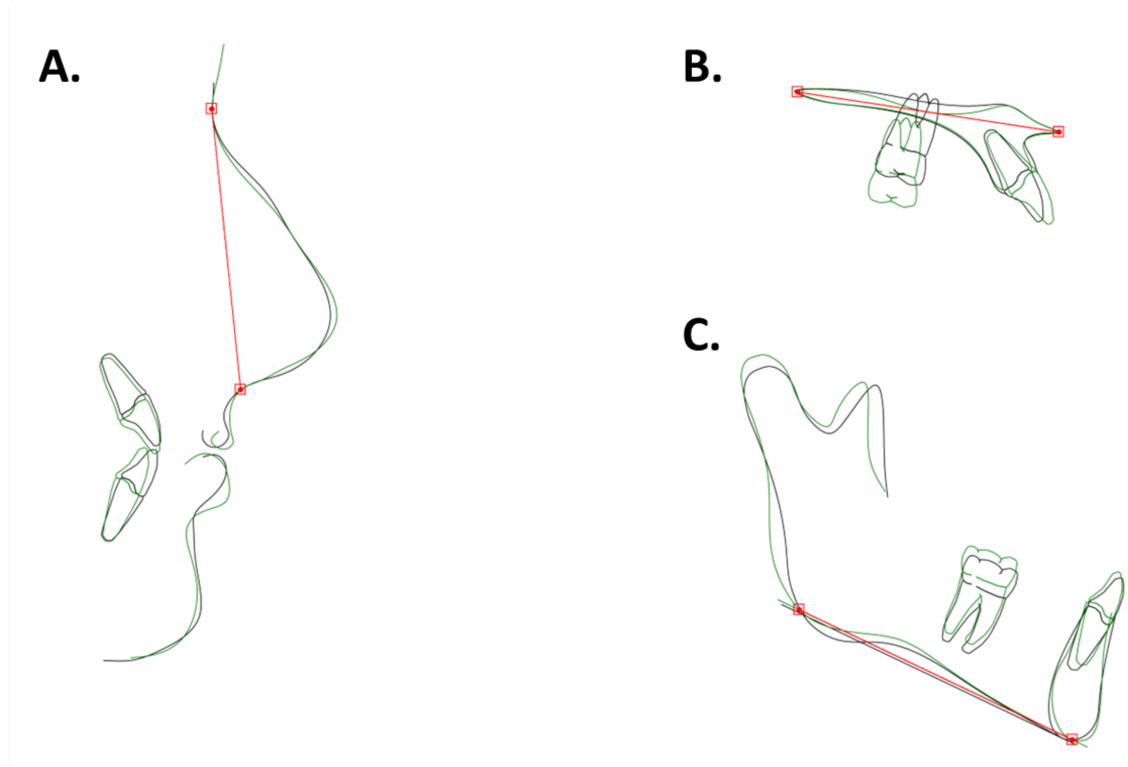

Figure 7. Cephalometric superimposition over SN line of before (black) and after (green) Invisalign treatment showing the incisor (A, B, and C) and molar (B and C) movements.

Prospective study

Based on the results of a few treated cases, a prospective study investigating the potential correction of lateral open bites and Class III malocclusions in subjects with moderate OI was initiated. These malocclusions had to be of moderate amplitude with a maximum value for a PAR index of 40. This value was based on our observations published in 2013 about the average PAR index of 32 in the OI population [12].

Future studies and assessments

Digital technologies and accessible open-source software have allowed for a seamless integration of intraoral scan and CBCT data as a first step. A second step is the availability of AI-based auto segmentations of structures and a fusion of all 3D data into a single record of correctly oriented STL files. The third step was to then use open-source or open format software to simulate treatment, growth patterns and tooth movements in a virtual environment. These simulations then allowed for a fully customized and individualized approach to treatment by combining growth prediction, orthodontic, and orthognathic interventions into a single file system (Figure 8).

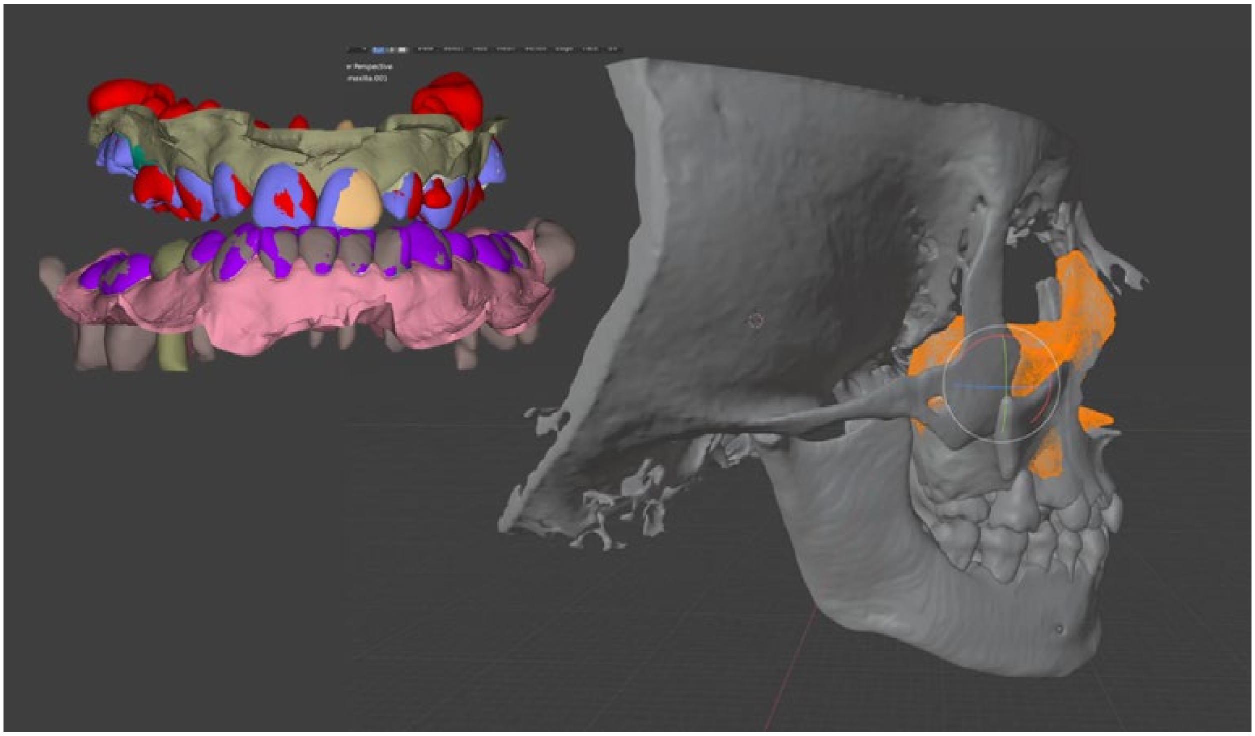

Figure 8. Integration of Cone Beam and STL data.

Integration of CBCT and STL data

A 15-year-old female with OI type IV presented for potential treatment of a moderate Class III malocclusion with a bilateral posterior open bite and missing teeth. CBCT, intraoral scan, and extraoral and intraoral photographs were obtained (Figure 9). The dataset was then sent for fusion and segmentation (Diagnocat™). The cranial vault, maxilla, airway, upper teeth, lower teeth, mandibular canal, and mandible were segmented into STL files (Figure 10). Drishti software was also used to better assess the dentoalveolar levels present around the teeth to evaluate the possibility of tooth movement.

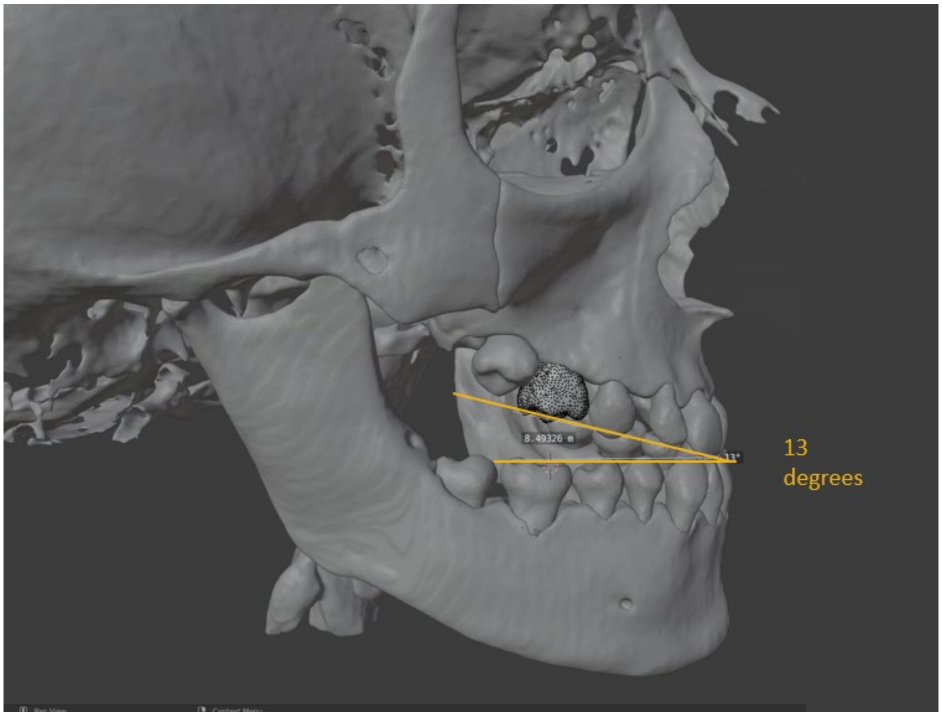

Figure 9. OI type IV patient with severe open bite.

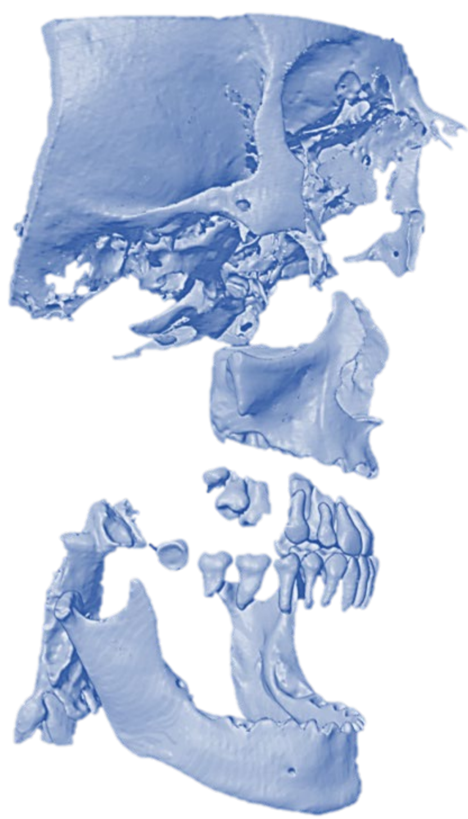

Figure 10. Segmentation of CBCT data with Deep Learning algorithms.

The segmented data was then transferred to an open-source software (Blender) where simulations were performed to determine the most appropriate course of treatment for the malocclusion presented. An orthodontic approach was first used to move the dentition into its corrected position (Figure 11) and then an orthognathic approach was tested and the amount of movement necessary measured (Figure 12). This technique allows us to fully correct a malocclusion and obtain precise measurements of the bony segments or of the dental components. The movements are also confined to the dentoalveolar processes by software constraints to avoid root exposure and periodontal problems. The objectives of improving facial aesthetics and masticatory functions while respecting the dentoalveolar processes and remaining within the limitations of a surgical approach can be tested with multiple treatment approaches until one is selected and carried out on the patient.

Figure 11 shows the dentition that has been reformatted before and after repositioning to calculate the amount of movement necessary to correct the malocclusion. Both datasets are superimposed to get an appreciation of the biomechanics needed to be applied at the crown and root level to maintain the teeth into the dentoalveolar process while correcting or improving the torque values, alignment, and mesial distal angulation of the roots to get the best possible aesthetic and functional result.

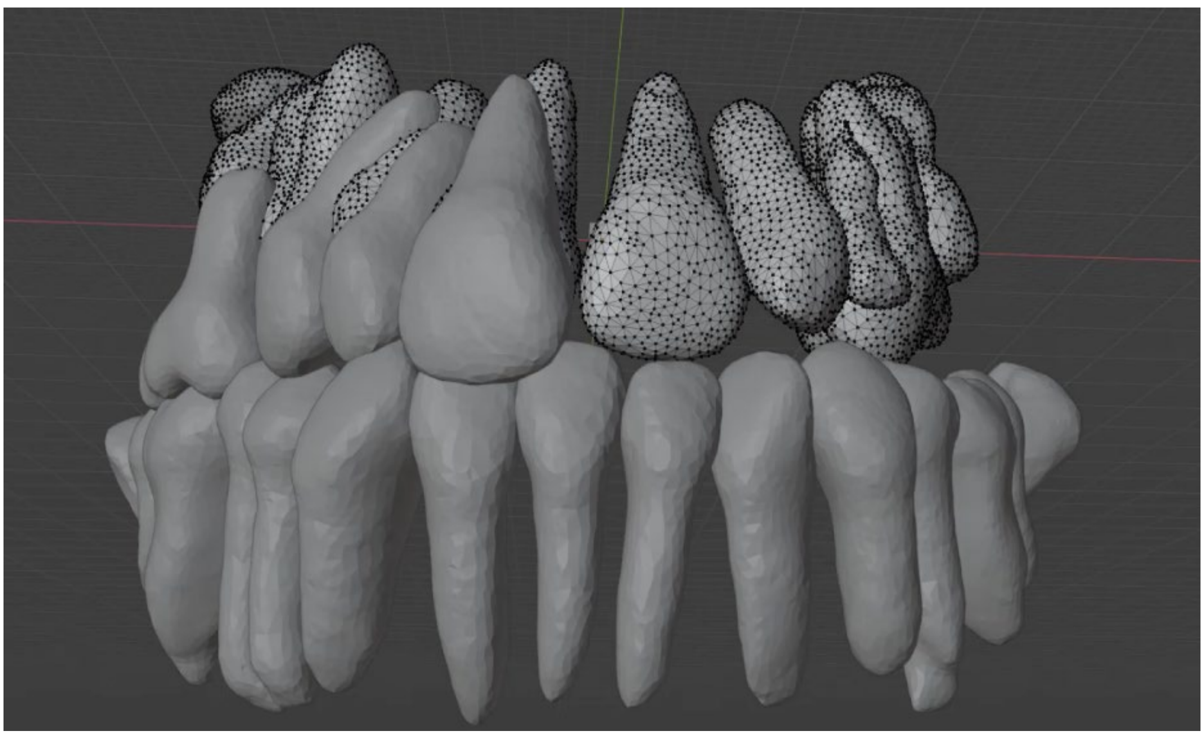

Figure 11. Simulation of dentoalveolar approach. Solid teeth are the teeth repositioned in correct occlusion.

Figure 12 shows a surgical approach why the maxilla is detached from the cranial base and brought down in one piece to recreate an acceptable occlusal plane. In this case the occlusal plane created by the lower dentition was deemed acceptable and used to move the maxillary complex into an acceptable position. The maxillary complex showed an amount of displacement of over 10 millimeters. This large movement indicates that a combination of an orthodontic approach paired with an orthognathic approach may be the best treatment option.

Figure 12. Surgical repositioning of the maxilla. The orange represents the preoperative position. Notice the over 9mm of down grafting necessary.

Simulation software allows practitioners to individualize the treatment approach and to analyze the feasibility of multiple treatment approaches with the surgeons, orthodontist, and entire treatment team. The simulations also allow for the creation of biomechanical systems and appliances to fully customize the treatment to match each patient's needs.

As many OI patients are affected by OSA, a study of the airway and an assessment of the position of the hyoid bone may prove to be useful diagnostic procedures. CBCT should also be used to look for platybasia, basilar impression, and invagination as part of the diagnosis.

3D CBCT diagnostic records allow the orthodontist and the entire team to assess the feasibility of treatment as well as the probability of success to assure that the prognosis for a good resolution of the malocclusion is as high as possible. Also, the profession needs a lot of education regarding the most appropriate approach to address these challenging malocclusions. Determining what to expect from an intervention at the onset of treatment and how the patient care will progress can significantly improve the way the care is delivered in the future. More research is necessary on the biology of tooth movement in rare diseases, but it has been possible to move teeth in OI patients even when they are under IV bisphosphonate treatment.

Practitioners interested in treating patients with OI should understand that slowing the tooth movement process and reducing the forces placed on the dentition is of great importance. Understanding the limitations and not aiming for a perfect result but settling for a significant improvement that will give the patient the best possible function and aesthetics is a valid treatment goal. Orthognathic surgeons experienced in OI management can provide guidance on what should be attempted.

For instance, a hypoplastic maxilla formed from poor bone creates a severely limiting factor to our therapeutic approach. However, using distraction osteogenesis and interceptive orthodontics at a younger age could reduce the interventions needed to reposition the maxilla once the patient reaches adulthood. This is a very exciting treatment option that needs to be developed for the orthodontic treatment of the OI population.

CONCLUSIONS

Our experience shows that it is possible to significantly improve the quality of life of patients with OI by addressing their specific functional and aesthetic needs and identifying what is feasible on a case-by-case basis. Some cases are too severe, but many can be improved. Thus, early intervention in patients with moderate OI is a potential method to decrease the severity of developing handicapping malocclusions and can address antiresorptive treatments (bisphosphonates) in the lack of development of the craniofacial complex. This is just one example of valuable research that can result in improved care of special needs patients, thus adding knowledge in the field of orthodontics.

REFERENCES

1. Rauch F, Lalic L, Roughley P, Glorieux FH. Genotype-phenotype correlations in nonlethal osteogenesis imperfecta caused by mutations in the helical domain of collagen type I. Eur J Hum Genet 2010; 18(6):642-647.
2. Rossi V, Lee B, Marom R. Osteogenesis imperfecta: advancements in genetics and treatment. Curr Opin Pediatr 2019; 31(6):708-715.
3. Van Dijk FS, Sillence DO. Osteogenesis imperfecta: clinical diagnosis, nomenclature and severity assessment. Am J Med Genet A 2014; 164A(6):1470-1481.
4. Sturznickel J, Jahn-Rickert K, Zustin J, Hennig F, Delsmann MM, Schoner K, Rehder H, Kreczy A, Schinke T, Amling M et al. Compound heterozygous frameshift mutations in MESD cause a lethal syndrome suggestive of osteogenesis imperfecta Type XX. J Bone Miner Res 2021; 36(6):1077-1087.
5. Rauch F, Glorieux FH: Osteogenesis imperfecta. Lancet 2004; 363(9418):1377-1385.
6. Mortier GR, Cohn DH, Cormier-Daire V, Hall C, Krakow D, Mundlos S, Nishimura G, Robertson S, Sangiorgi L, Savarirayan R et al. Nosology and classification of genetic skeletal disorders: 2019 revision. Am J Med Genet A 2019; 179(12):2393-2419.
7. Marini JC, Cabral WA, Barnes AM. Null mutations in LEPRE1 and CRTAP cause severe recessive osteogenesis imperfecta. Cell Tissue Res 2010; 339(1):59-70.
8. Trejo P, Rauch F. Osteogenesis imperfecta in children and adolescents-new developments in diagnosis and treatment. Osteoporos Int 2016; 27(12):3427-3437.
9. Reznikov N, Dagdeviren D, Tamimi F, Glorieux F, Rauch F, Retrouvey JM. Cone-beam computed tomography of osteogenesis imperfecta Types III and IV: three-dimensional evaluation of craniofacial features and upper airways. JBMR Plus 2019; 3(6):e10124.
10. Taqi D, Moussa H, Schwinghamer T, Ducret M, Dagdeviren D, Retrouvey JM, Rauch F, Tamimi F, Members of the BBDC. Osteogenesis imperfecta tooth level phenotype analysis: Cross-sectional study. Bone 2021, 147:115917.

11. Taqi D, Moussa H, Schwinghamer T, Vieira AR, Dagdeviren D, Retrouvey JM, Rauch F, Tamimi F, Members of the BBDC. Missing and unerupted teeth in osteogenesis imperfecta. Bone 2021, 150:116011.
12. Rizkallah J, Schwartz S, Rauch F, Glorieux F, Vu DD, Muller K, Retrouvey JM. Evaluation of the severity of malocclusions in children affected by osteogenesis imperfecta with the peer assessment rating and discrepancy indexes. Am J Orthod Dentofacial Orthop 2013; 143(3):336-341.
13. Ottley CM. Osteopsathyrosis (Lobstein's disease): a critical review. Arch Dis Child 1932; 7(39):137-48.
14. Jabbour Z, Al-Khateeb A, Eimar H, Retrouvey JM, Rizkallah J, Glorieux FH, Rauch F, Tamimi F. Genotype and malocclusion in patients with osteogenesis imperfecta. Orthod Craniofac Res 2018; 21(2):71-77.
15. Dagdeviren D, Tamimi F, Lee B, Sutton R, Rauch F, Retrouvey JM. Dental and craniofacial characteristics caused by the p.Ser40Leu mutation in IFITM5. Am J Med Genet A 2019; 179(1):65-70.
16. Retrouvey JM, Taqi D, Tamimi F, Dagdeviren D, Glorieux FH, Lee B, Hazboun R, Krakow D, Sutton VR, Members of the BBDC. Oro-dental and cranio-facial characteristics of osteogenesis imperfecta type V. Eur J Med Genet 2019; 62(12):103606.
17. Marini JC, Reich A, Smith SM. Osteogenesis imperfecta due to mutations in non-collagenous genes: lessons in the biology of bone formation. Curr Opin Pediatr 2014; 26(4):500-507.
18. Campisi G, Fedele S, Colella G, Lo Casto A, Fusco V. Canadian consensus practice guidelines for bisphosphonate associated osteonecrosis of the jaw. J Rheumatol 2009; 36(2):451-453.
19. Hennedige AA, Jayasinghe J, Khajeh J, Macfarlane TV. Systematic review on the incidence of bisphosphonate related osteonecrosis of the jaw in children diagnosed with osteogenesis imperfecta. J Oral Maxillofac Res 2013; 4(4):e1.
20. Tauer JT, Robinson ME, Rauch F. Osteogenesis imperfecta: new perspectives from clinical and translational research. JBMR Plus 2019; 3(8):e10174.
21. Glorieux FH, Devogelaer JP, Durigova M, Goemaere S, Hemsley S, Jakob F, Junker U, Ruckle J, Seefried L, Winkle PJ. BPS804 Anti-sclerostin antibody in adults with moderate osteogenesis imperfecta: results of a randomized phase 2a trial. J Bone Miner Res 2017; 32(7):1496-1504.
22. Rousseau M, Vargas J, Rauch F, Marulanda J, Retrouvey JM. Facial morphology analysis in osteogenesis imperfecta types I, III and IV using computer vision. Orthod Craniofac Res 2021; 24(2):92-9.
23. Rousseau M, Retrouvey JM, Members of the Brittle Bone Disease Consortium. Osteogenesis imperfecta: potential therapeutic approaches. PeerJ 2018; 6:e5464.
24. Chetty M, Roberts TS, Stephen L, Beighton P. Craniofacial manifestations in osteogenesis imperfecta type III in South Africa. BDJ Open 2017; 3:17021.
25. Frazier-Bowers SA, Koehler KE, Ackerman JL, Proffit WR. Primary failure of eruption: further characterization of a rare eruption disorder. Am J Orthod Dentofacial Orthop 2007; 131(5):578 e571-511.
26. O'Connell AC, Marini JC. Evaluation of oral problems in an osteogenesis imperfecta population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 87(2):189-196.
27. Richmond S, Shaw WC, O'Brien KD, Buchanan IB, Jones R, Stephens CD, Roberts CT, Andrews M. The development of the PAR Index (Peer Assessment Rating): reliability and validity. Eur J Orthod. 1992; 14(2):125-39.

THE ROLE OF PRIMARY CILIA AND CILIA-RELATED GENES IN BONE REMODELING

Jessica Kang, Lucy Eun Hwan Kim, Mina Oh, Hyeran Helen Jeon

ABSTRACT

The skeleton undergoes continuous bone remodeling to adapt to its local environment, such as mechanical forces. Mechanotransduction is an essential component of bone homeostasis in which bone cells sense and convert mechanical stimuli into biochemical responses. Although the exact mechanisms remain elusive, the primary cilium has been suggested as a potential mechanosensory and chemosensory organelle in bone remodeling. This chapter discusses the various intraflagellar transport (IFT) proteins necessary for ciliogenesis and functional primary cilia in osteoblasts, osteocytes, and chondrocytes. Findings indicate that deletion of selective IFT proteins leads to impaired ciliogenesis or dysfunctional primary cilia with possible bony defects, which illustrates the importance of these proteins in ultimately maintaining healthy bone. However, further research efforts are needed to fully understand the complex mechanisms of the mechanosensory features of primary cilia in bone homeostasis.

KEY WORDS: Primary Cilia, Intraflagellar Transport (IFT) Proteins, Bone Remodeling

INTRODUCTION

Mechanical force-induced bone remodeling is a dynamic process that is required to maintain the structure and function of the skeleton [1]. Bone is sensitive to external environments and adapts to meet the physical demands of mechanical loading. Bone remodeling depends on mechanotransduction, where mechanical energy is converted into biomechanical responses [2]. During mechanical loading, fluid flows through a 3-dimensional network of interconnected lacunae and canaliculi, which helps osteocytes contact neighboring osteocytes, osteoblasts, and osteoclasts [3]. This load-induced fluid flow stimulates osteogenic and osteoclastic responses. However, the exact mechanism of how these bone cells translate these changes in fluid flow into biochemical signals remains largely unknown [4].

Different bone cells work in a coordinated manner to maintain a balance between bone formation and bone resorption [5]. Osteoblasts are specialized bone-forming cells derived from mesenchymal stem cells [6]. They secrete bone matrix and produce growth factors, hormones, and enzymes [7]. As osteoblasts mature, they become encapsulated in newly deposited bone and become osteocytes [8]. Osteocytes are the most abundant cell type and the primary mechanosensory cells of the bone. They regulate bone homeostasis by controlling osteoblasts and osteoclasts [9]. Chondrocytes are responsible for cartilage formation and play an important role during endochondral ossification [10].

Primary cilia are microtubule-based organelles that are present on nearly all mammalian cells [11]. They are considered the mechanosensor and chemosensor organelle in the bone [12]. The load-induced fluid flow causes a deflection of the primary cilia, eliciting many downstream signaling pathways (Figure 1) [13]. In addition, primary cilia are dependent on intraflagellar transport (IFT) proteins for their formation and function [14]. This chapter reviews the role of primary cilia and related genes in bone cells including osteoblasts, osteocytes, and chondrocytes during bone remodeling.

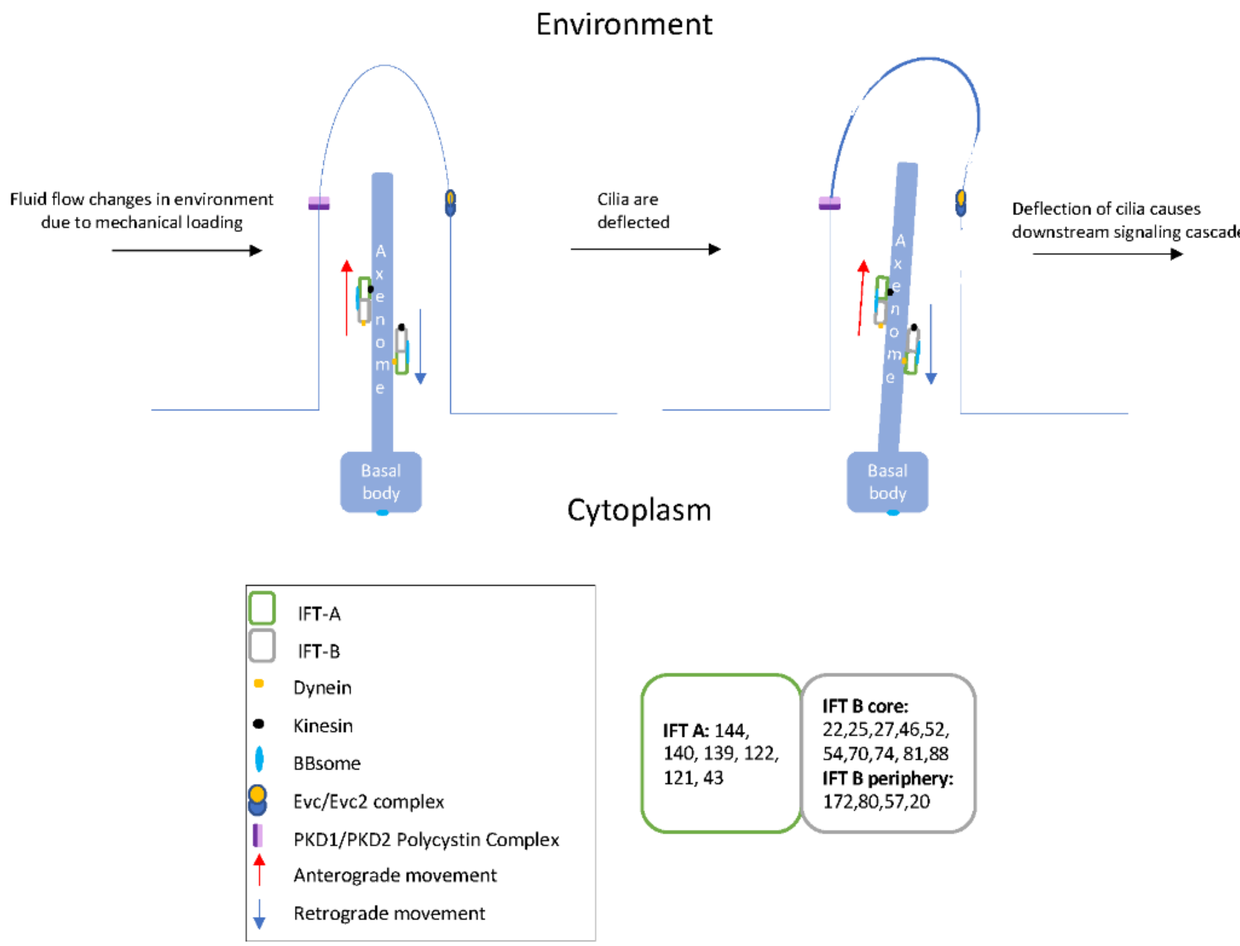

Figure 1. Primary cilia on bone cells. They sense environmental load-induced changes to fluid flow, which deflect the cilia and, in turn, activate downstream signaling pathways that elicit bone remodeling changes.

PRIMARY CILIA

Primary cilia are the hair-like organelles that sense changes in extracellular signals in the surrounding environment and act through various transduction pathways, including hedgehog, transforming growth factor β, platelet derived growth factor, and fibroblast growth factor [15, 16]. The interactions between cilia and these pathways are essential for the formation of bone and cartilage [15]. For instance, cilia are the mechanosensory machinery in bone, responding to increased mechanical forces to stimulate osteogenesis [15].

Ciliary genes: IFT system and related genes

Cilia do not make their own proteins. Instead, proteins are made in the cytosol and transported in and out of the cilia by the bidirectional transport system called IFT [16]. The IFT system is a multi-protein complex made of IFT complex B (IFT-B) proteins, IFT complex A (IFT-A) proteins, BBSome, IFT motors, and associated cargo [17, 18]. IFT-B is composed of fourteen IFT proteins consisting of IFT20, IFT22, IFT25, IFT27, IFT46, IFT52, IFT54, IFT57, IFT70, IFT74/IFT72, IFT80, IFT81, IFT88, and IFT172. IFT-A is made up of six proteins including IFT144, IFT140, IFT139, IFT122, IFT121, and IFT43 [15]. Kinesin-II and cytoplasmic dynein-2 are IFT motors. IFT-B is driven by kinesin II proteins which allow transport from the base of the cilia to the tip while IFT-A is driven by dynein proteins allowing for transport from the cilia axoneme to the cell body. The BBSome forms part of the cilia basal body and plays a role in the proper formation of not only the cilia but also the cilia transportation components. However, the exact mechanism is not fully known [19, 20].

IFT-B complex proteins

IFT20, the smallest of the IFT-B complex proteins, is necessary for cilia formation and can be found in the basal body of cilia as well as the golgi complex [21]. IFT20 moves between the golgi complex and cilia, and it has been suggested to transport proteins from the golgi complex to the cilia. Follit et al. found that when IFT20 is deleted, cilia assembly is disrupted [21]. Similarly, IFT20 deletion in mice kidney cells resulted in a lack of primary cilia formation [22]. In addition, Noda et al. demonstrated that IFT20 regulates collagen movement intracellularly and is important for skull development [23].

IFT22 is not crucial for cilia formation [24]. IFT22 acts independently of ciliary formation and the IFT complex. IFT22 does assist in the recruitment of the BBSome into the cilia. Further, Xue et al. found that in IFT22 mutants, the cilia were still functional but BBsome recruitment was altered and that IFT22 aided in BBSome binding to the cilia and subsequent entry into the cilia [24].

IFT25 is not required for cilia formation in somatic cells. Liu et al. found that IFT25 homozygous knockout male mice (*Stra8-iCre+*.IFT25$^{f/f}$) had no observable abnormalities besides sterility. Thus, they deduced that IFT25 plays a role in sperm formation but not cilia formation in somatic cells [25].

IFT27 is important for transport of the BBSome complex out of the cilia. Inside the cilia, IFT27 separates from IFT-B complex and activates ARL6 in addition to aiding in BBSome coat assembly and transport of the BBSome out of the cilia [26].

IFT46 is crucial for vertebral cilia formation. Lee et al. found that IFT46 homozygous KO mice did not survive to birth while IFT46 heterozygous KO survived. Severe abnormalities including heart issues as well as the failure of notochord closure during development, resulted in embryo death of homozygous KO mice [27].

IFT52 mutations in humans lead to a skeletal deformity known as short rib polydactyly syndrome. IFT52 plays a key role in IFT-B complex formation and function. Zhang et al. showed that human fetuses with IFT52 mutant cells synthesize fewer IFT-B complex proteins, such as IFT74, IFT81, IFT88 and ARL13B [28]. Thus, IFT52 affects other IFT-B complex proteins because of its role of aiding in the attachment of other IFT-B complex proteins to the cilia basal body and stabilizing the entire IFT-B complex.

IFT54 is necessary for anterograde transfer in the IFT-B complex through its interactions with kinesin and dynein [29]. Zhu et al. noted that the deletion of IFT54 using a *Chlamydomonas* model led to disrupted anterograde transport and subsequent build-up of IFT proteins in the cilia. The authors postulated that the deletion of IFT54 interrupts normal binding to motor proteins, thereby inhibiting the proper motor protein movement [29].

IFT57 is required for cilia maintenance and control of IFT-particle-kinesin II dissociation in vertebral photoreceptor cells [30]. Krock et al. found that without IFT57, IFT20 is unable to bind IFT and either IFT57, IFT20, or both proteins are required to mediate kinesin 2 function [30].

IFT70, known as DYF-1, is a core component of cilia and is required for proper cilia formation. Takei et al. found that double-knockout IFT70A and IFT70B cells lacked cilia formation even though IFT70 is associated peripherally with the IFT-B complex via the IFT52–IFT88 dimer. Even in the absence of IFT70, other IFT-B subunits were still assembled at the ciliary base, suggesting IFT70 is essential for cilia formation but nonessential for the assembly of the residual IFT-B subunits [31].

IFT74 and IFT81 work together to bind tubulin. Bhogaraju et al. found that IFT81 increases the specificity for tubulin, while IFT74 increases the affinity for tubulin. Only cells with both normal IFT74/81 protein complexes were able to bind tubulin with a high affinity [32]. In addition, IFT81 mutations are associated with short rib polydactyly syndrome. Duran et al. reported the short rib polydactyly syndrome with IFT81 mutations. They found that the IFT-B complex is unstable in the presence of IFT81 mutations and IFT81 mutations did not affect the production levels of a kinesin motor protein even though the IFTB complex was disrupted. Further, the growth was disrupted by the IFT81 mutations due to disrupted hedgehog signaling pathways and IFT81 mutations led to altered chondrocytes in the growth plate [33].

IFT80 is required for the cilia formation, maintenance, and function. IFT80 mutations cause Jeune asphyxiating thoracic dystrophy and short rib polydactyly type III. IFT80 mutations disrupt the balance between canonical and non-canonical Hh-Gαi-RhoA pathways. Yuan et al. examined the *OSX-Cre;IFT80* $^{f/f}$ transgenic mice and found that IFT80 deletion in osteoblasts is associated with growth retardation and osteopenia and that IFT80 is necessary for differentiation of osteoblast cells [34].

IFT88 is important for cilia formation and its mutations can lead to ciliopathies, such as craniofacial disorders in mice. Tian et al. generated the *Wnt1-Cre;Ift88* $^{f/f}$, which is IFT88 deletion in neural crest cells. They found that homozygous IFT88 mice died at birth due to severe defects, such as tongue agenesis and cleft lip/palate. In addition, palatal mesenchymal cells lost cilia formation. The number of cilia and cilia length decreased significantly in mice without IFT88, indicating that cilia were absent or significantly disrupted in these cells [35]. Another study found that IFT88 knockout mice presented a decreased number of osteoblasts/osteocytes and reduced bone density [36].

IFT172, the largest protein in the IFT-B complex, is important for the anterograde transport of dynein-2 and/or the IFT trains around the ciliary tip [37]. IFT172 mutations in humans are associated with skeletal ciliopathies, such as short rib thoracic dysplasia with or without polydactyly. Roberts et al. surgically manipulated mice embryos to generate IFT72 KD. They found that the mutant cells had no cilia compared to WT but acetylated alpha tubulin levels were not significantly different between WT and IFT172 KD mice. Therefore, IFT72 is crucial for proper cilia function and mutations have a wide range of defects and disruptions in cell pathways [38].

IFT-A complex proteins

IFT144 is important for IFT-A complex formation. Ashe et al. found that the transgenic mice with a hypomorphic missense mutation in the IFT144 gene presented disrupted IFT-A complex formation and had skeletal abnormalities, including shortened limbs, ribs, polydactyl, and craniofacial abnormalities with disrupted hedgehog signaling pathways [39].

IFT140 mutations in humans has been associated with skeletal ciliopathies. Tao et al. generated the transgenic Osx-Cre.IFT140$^{f/f}$ mice, deleting IFT140 in the early stages of osteoblast formation. The transgenic mice showed bone defects including decreased bone length, decreased bone density, and decreased bone mineral apposition rate. Thus, IFT140 is crucial for bone development and growth plate formation [40].

IFT139 is not necessary for cilia formation, but it is critical for proper IFT-A complex trafficking to occur in addition to IFT-B trafficking and G protein-coupled receptors (GPCRs) function in cilia function. Hirano et al. generated IFT139 KO cells *in vitro* and found that IFT-A, IFT-B, and GPCRs accumulated at the cilia tip, and had disrupted traffic to the cilia base [41].

IFT122 connects core and peripheral IFT-A complex proteins and forms a complex with IFT140 and IFT144. Takahara et al. generated IFT122 KO mice by introducing a missense mutation in the initiation codon of IFT122. When compared to other IFT-A complex proteins, the IFT122 deletion had the largest cilia defects. In addition, IFT122 knockout cells had no cilia present on cell lines [42].

IFT121 interacts with the IFT43 complex and mutations in both are associated with short rib polydactyly syndrome in humans. Duran et al. collected human cells from patients diagnosed with IFT43 and IFT121 mutations. IFT43 mutated fibroblasts showed no cilia but the IFT-A complex stability was not affected. In addition, growth plate abnormalities were found in tissues with the IFT43 mutation. Mutations in IFT121 led to decreased IFT43 levels and skeletal abnormalities presented in a similar way as the IFT43 mutation. Again, the IFT121 mutation did not affect the IFT-A complex stability but in contrast to IFT43 mutations, cilia were not completely absent in IFT121 mutant cells. Mutations in both IFT43 and IFT121 in humans presented short rib polydactyly syndrome and only IFT43 mutation caused a complete absence of cilia [43].

IFT motors

Kinesin and dynein are motor proteins important for the proper formation and function of primary cilia. Kinesins depend on ATP to transport proteins along microtubules in an anterograde direction. Common to all kinesins is a core motor domain, while different kinesins have different amino acid sequences in the periphery that allow them to transport a variety of objects towards the plus end of the microtubule. Heterotrimeric kinesin-2 is the main anterograde motor component of IFT. Disruption of kinesin-2 causes a lack of cilia in animal models, such as *C. elegans* and mice [16]. Dyneins are ATP-dependent enzymes that move proteins towards the minus end of the microtubule, in a retrograde manner towards the basal body. Mammalian cells have 2 dyneins that work together to transport particles. Cytoplasmic dynein 1 moves particles retrograde and functions separately from dynein 2. Dynein 2 is part of the IFT complex and moves particles in motile and sensory cilia [37]. Dynein 2 works in conjunction with kinesin 2 to transport particles along IFT [44]. Dynein 2 is carried anterograde to the cilia tip, and once there, the IFT complex remodels and continues retrograde movement back to base.

Others

Polycystin-1 (PKD1) and Polycystin-2 (PKD2) are associated with renal primary cilia, osteoblast, and osteocytes. This complex acts as a mechanosensor, responding to changes in force and stress in the kidney environment [45]. Their function in bone development is still not fully known. Xiao et al. found that loss of PKD1 in mice disrupted osteoblast differentiation and led to disrupted bone development and osteopenia [46]. In addition, mutations in PKD1 and PKD2 led to disruptions in chondrogenesis as well [45].

Evc is associated with primary cilia and is located in the cilia basal body. It is necessary for Indian hedgehog (Ihh) signaling to occur at cartilage growth plates [47]. Caparrós-Martín et al. showed that Evc2$^{-/-}$ and Evc$^{-/-}$; Evc2$^{-/-}$ mice had comparable skeletal defects due to impaired Ihh signaling. Skeletal abnormalities in transgenic mice included shortened limbs, ribs, and defects in the cranial base, demonstrating an important role of the EVC/EVC2 in regulating Ihh signaling [48].

PRIMARY CILIA AND OSTEOBLASTS

Osteoblasts are specialized bone-forming cells, and their primary function is to produce a collagen-rich matrix to lay down new bone [49]. Osteoblasts secret growth factors and hormones, including bone morphogenetic proteins, transforming growth factor β, and osteocalcin [7].

The primary cilia in osteoblasts act as mechanosensors and are responsible for the osteogenic changes in response to dynamic fluid flow [1]. Malone et al. found that when MC3T3-E1 osteoblasts were exposed to oscillatory fluid flow, a three-fold increase in osteopontin (OPN) expression along with increased prostaglandin E2 (PGE2) release and cyclooxygenase 2 (COX2) mRNA levels, which are the constituents of the bone matrix, were observed [1, 4]. To determine whether these changes were dependent on primary cilia, bone cells were treated with chloral hydrate or siRNA to delete the primary cilia. Chloral hydrate and siRNA treatment caused a 90% and 50% reduction in the fraction of cells with primary cilia as scored by acetylated alpha tubulin immunofluorescence stain and western blot. The treated cells did not respond with the increased expression levels of OPN, PGE2, and COX2 as a response to fluid flow [1]. In addition, they examined the OPG/RANKL ratio in MLO-Y4 osteocyte-like cells as it controls osteoclast formation. In untreated cells, fluid flow increased the OPG/RANKL mRNA ratio by 1.6-fold, whereas the cells treated with chloral hydrate or siRNA did not cause significant changes in the OPG/RANKL mRNA ratio. Considering all, primary cilia are essential for both osteogenic and bone resorptive responses to dynamic fluid flow. Furthermore, Delaine-Smith et al. demonstrated that primary cilia are required in MLO-A5 murine osteoblasts for the secretion of PGE2 and calcium deposition in response to fluid flow [4].

In vivo studies support the various roles of primary cilia in osteoblasts during bone formation. Moore et al. demonstrated the necessity of primary cilia in periosteal osteochondroprogenitors (OCP) as the mechanosensors and regulators of osteogenic response [50]. Tamoxifen-induced primary cilium knockout in periosteal OCPs displayed an absence of increased expression levels of osteogenic markers (COX2, OPN, RUNX2) that were seen in OCP with intact primary cilia [50]. In addition, differentiation into osteoblasts was upregulated under mechanical stress only in the presence of primary cilium [50]. Chen et al. found that mechanical loading enhanced bone marrow-derived cells to commit to the osteogenic lineage, leading to increased bone formation [51].

IFT20 regulates cell polarity and alignment, cilia formation, and bone mass during bone development [52]. Lim et al. reported that the deletion of IFT20 in osteoblastic lineage cells led to disrupted osteoblast polarity and decreased cilia formation and length in osteoblasts and osteocytes [52]. In addition, IFT20 is involved in regulating collagen biosynthesis during bone formation [53]. Yamaguchi et al. examined the IFT20:Wnt1-Cre and IFT20:Ocn-Cre mice and observed an increased hydroxylysine-aldehyde cross-linking and decreased lysine-aldehyde cross-linking in the IFT20:Ocn-Cre mice [11]. Disruption of IFT20 in both mouse strains led to craniofacial abnormalities [53], indicating the important role of IFT20 in collagen biosynthesis.

IFT80 is an essential regulator of osteoblast differentiation and cilia formation [54]. Yang and Wang discovered that IFT80 was highly expressed in preosteoblasts, mature osteoblasts, bone tissue, and during osteoblast differentiation [54]. Silencing of IFT80 in murine mesenchymal progenitor line and bone marrow-derived stromal cells resulted in impaired cilia formation and inhibited osteoblastic differentiation via significantly decreased expression of osteoblast marker genes including RUNX2, OCN, bone sialoprotein (BSP), and alkaline phosphatase (ALP) [54]. Furthermore, the expression level of Gli2, a transcriptional factor involved in the hedgehog signaling pathway, was largely reduced [54]. Rix et al. generated a murine IFT80 gene-trap line (IFT80$^{gt/gt}$) and discovered that these mice were hypomorphic compared to the true null mice [55]. The surviving 2% of gene-trap mice exhibited similar phenotypes as Jeune asphyxiating thoracic dystrophy and short rib polydactyly type III [13]. Chiniparadaz et al. focused on the role of IFT80 in fracture healing and found IFT80 deletion in osteoblasts led to reduced cilia formation and delayed fracture healing [56].

Haycraft et al. deleted IFT88 in mature osteoblasts to assess its role in bone formation. Decreased osteoblast number and bone density were observed in IFT88-deleted mice of a mixed genetic background while an inbred C57BL/6J genetic background displayed increased bone volume/total volume ratio and trabecular thickness [17, 36]. Additional research is necessary to uncover whether these differences are due to the different genetic backgrounds. Moreover, whether IFT88 deletion disrupted cilia formation and signaling pathways was not discussed and requires further investigation.

IFT140 is implicated in patients with severe skeletal disorders [40]. Zhang et al. found that IFT140 levels in bone were highly expressed during development [57]. IFT140 levels in osteoblasts fluctuated in accordance with the crucial stages of endochondral ossification [57]. Mouse femurs from osteoporosis models, either from aged or ovariectomized group, exhibited significantly decreased IFT140 mRNA levels compared to control group [57]. Tao et al. reported that the preosteoblast-specific IFT140 deletion led to smaller body size and weight, decreased bone formation, and delayed growth compared to control mice [40]. In addition, the experimental Osx-Cre:IFT140$^{f/f}$ mice displayed decreased expression levels of osteoblastic markers like OCN, OPN, BSP, and ALP in comparison to the control mice [40].

Mutations in IFT144 is associated with human ciliopathies including Sensenbrenner and Jeune syndromes [39]. Ashe et al. assessed the mouse mutant with a hypomorphic missense mutation in the IFT144 gene [39]. These transgenic mice showed enhanced hedgehog signaling despite impaired ciliogenesis and a reduced ability to respond to upstream activation of signaling. IFT144^{twt} embryos exhibited severe craniofacial defects including the loss of frontal bone, cleft palate, and open bite [39].

Kif3a is an IFT motor protein essential for ciliogenesis in osteoblasts and osteocytes [58]. Temiyasathit et al. demonstrated that skeletally mature *Colα1(I) 2.3-Cre;Kif3a*$^{fl/fl}$ mice presented the defective cilia and decreased bone formation in response to mechanical ulnar loading [58]. Leucht et al. focused on bone healing around intra-osseous implants in the tibia of mice with conditional Kif3a knockout [59].

Conditional deletion of Kif3a in pre-osteoblasts resulted in dysfunctional, truncated primary cilium as well as decreased response to mechanical loading and failure to differentiate into mature osteoblasts [59]. Qiu et al. showed that selective deletion of Kif3a in osteoblasts resulted in decreased length and numbers of primary cilia [60]. Furthermore, they found Kif3a knockout mice developed osteopenia with reduced femoral bone mineral density, trabecular bone volume, and cortical thickness [60]. These studies collectively suggest the role of primary cilia as mechanosensors and their possible function in regulating bone physiology.

PRIMARY CILIA AND OSTEOCYTES

Osteocytes comprise 95% of bone cells and are the main mechanosensory cell in the bone [61]. Osteocytes communicate with other osteocytes and osteoblasts via direct contact with other cells at gap junctions and through indirect paracrine signaling pathways [2, 62]. In addition, osteocytes also produce macromolecules, such as sclerostin, FGF23, and RANKL in response to mechanical force stimuli, and the proteins reach target cells via fluid flow produced by the mechanical force changes [62]. Cilia in osteocytes detect changes in the bone, allowing for bone homeostasis [61]. During mechanical loading, fluid flows through the lacunae and between the channels connecting lacunae, deflecting cilia, and stimulating osteogenesis [1]. The exact location and orientation of cilia in osteocytes *in vivo* are not fully understood and the difficulty of cilia visualization *in vivo* due to its small size, makes the identification more challenging.

Xiao et al. found that the MLO-4 osteocyte-like cells present the cilia-like structures using acetylated alpha tubulin immunofluorescence stain, which is specific for the microtubules of the primary cilia [63]. The immunofluorescence stain demonstrated that the cilia were extending from the osteocyte cell surface [1]. During mechanical loading, osteocytes produce various signals, such as NO, ATP, PGE2, and $Ca2^+$ thereby stimulating downstream bone remodeling effects [2]. Upon treatment with chloral hydrate to remove cilia, cells free from chloral hydrate produced over triple the amount of PGE2 when exposed to flow compared to the cells treated with chloral hydrate, which had no difference in PGE2 production when exposed to flow. Similarly, when the untreated cells were compared to cells treated with chloral hydrate in no flow versus flow conditions, the untreated cells produced over 1.5 times higher OPG/RANKL ratio during flow conditions while the chloral hydrate treated cells had no significant change in OPG/RANKL ratio between flow and no flow conditions. These findings indicate that primary cilia in osteoblasts/osteocytes were found to deviate during fluid flow *in vitro* and change the expression of osteopontin, PGE2, COX2 and a ratio of OPG/RANKL [1].

The prevalence of primary cilia on osteocytes has conflicting findings, possibly due to difficulties visualizing cilia *in vivo* and different methods of identifying cilia. Only 4% of mice osteocytes contain cilia by transmission electron microscopy (TEM) while 94% of rat osteocytes contain cilia using an immunohistochemistry stain [64-66]. Several studies also reported varying lengths of cilia *in vivo*, ranging from 1.62µm up to 7µm in length [67].

Lim et al. reported that the deletion of IFT20 in osteoblastic lineage cells led to decreased cilia formation and length in osteocytes and unorganized osteocyte alignment [52]. Only 30% of osteocytes in experimental $IFT20^{f/f}$; Col1cre-ERT mice had cilia while 90% of WT mice osteocytes contained cilia. The cilia length on experimental mice osteocytes was also decreased, measuring approximately 0.7µm compared to the normal value of 2.3µm in WT [52]. In addition, in 3-month-old experimental $IFT20^{f/f}$; Col1cre-ERT mice, osteocyte alignment was affected even after bone remodeling had occurred in those older mice. The authors postulate that this disruption in osteocyte alignment can impact collagen fibril organization as well.

The IFT80 deletion in osteocytes and its impact during orthodontic bone remodeling has been investigated by Jeon et al (manuscript under revision). Transgenic mice were bred using IFT80$^{f/f}$ mice and DMP1-cre mice. Dentin matrix protein 1 (Dmp1) was chosen due to its specificity for osteocyte formation and maturation. While IFT80 expression decreased in IFT80 knockout mice, there was no difference in bony response, such as orthodontic tooth movement (OTM) distance and bone mineral density in transgenic mice compared to WT mice during OTM. This is a novel look into the role of IFT80 deletion on osteocytes *in vivo* during OTM.

Xiao et al. found that MLO-4 osteocyte-like cells expressed PKD1 and PKD2 and suspected that PKD1 and PKD2 may play an important role in bone development [63]. They studied how PKD1 missense mutation in mice embryos affected skeletal development and compared it to WT mice. The missense mutated mice were obtained from a mutant mice regional research center and contained arginine in place of methionine at the first transmembrane domain, resulting in an inactivating mutation [63]. The mice with the PKD1 missense mutation had disrupted long bone and calvaria formation, indicating impaired intramembranous and endochondral formation [63]. Thus, polycystin expression in osteocytes is likely to be important for osteocyte-mediated bone formation. In another study, Xiao et al. investigated polycystin complex and its effect on primary cilia on osteocytes [68]. PKD1 is a fluid sensor in renal cells, but its role in cilia on other cell types is not well known. They found that homozygous *Pkd1*$^{m1Bei/m1Bei}$ were embryonically lethal and thus conditional PKD1 knockout mice, *Pkd1*$^{flox/cko}$, were bred using *Pkd1*$^{flox/flox}$ mice and *Dmp1*-Cre;*Pkd1*$^{m1Bei/+}$mice. MicroCT analysis found that conditional deletion of PKD1 led to a decrease of 48% in trabecular volume and 17% decrease in cortical bone thickness compared to the WT control mice. In response to ulnar loading, multiple genes including Runx2-II, Runx2, Oc, Osteopontin, Bsp, RANKL, MMP13, Dmp1, and Phex were significantly decreased in the conditional knockout mice compared to the control group [68]. Based on these findings, PDK1 is important for proper mechanosensing in osteocytes and PDK1 disruption leads to impaired mechanotransduction signaling in osteocytes and decreased bone formation.

PRIMARY CILIA AND CHONDROCYTES

Chondrocytes are unique cell types present in the articular cartilage and are responsible for cartilage formation and function. Mesenchymal cell condensation induces chondrogenesis and the differentiated chondrocytes secrete components that aggregate to form the extracellular matrix [69]. Previous research supports the presence of cilia in chondrocytes as a sensory organelle involved in mechanotransduction during compressive and tensile forces in bone and cartilage.

IFT46 is responsible for ciliogenesis and craniofacial cartilage development [70]. Knockdown of IFT46 induced a significant reduction in the cilia number and length, which led to a decrease in ciliogenesis and severe impairment of cartilage formation, including the branchial stream, hyoid stream, and non-neural crest. IFT46 is preferentially expressed in hypertrophic chondrocytes in the growth plates during the early phase [71]. IFT46 knockdown by siRNA promoted the skeletogenesis-related genes, such as Col12a1, Msx1, Fgfr1, Bgn, Hsp47, and Mmp10 while there was no impact on the chondrogenesis-related gene expressions, such as Col2a1, Col10a1, Sox9.

IFT80 is an important regulator of chondrocyte differentiation during hedgehog (Hh) and Wingless (Wnt) signaling pathways [72]. High expression of IFT80 in all chondrocytes resides in the growth plate. IFT80 mRNA level in bone marrow-derived stromal cells (BMSCs) showed a gradual increase during the chondrogenic events and was maintained until the chondrocyte maturation took place. These high expressions of IFT80 mRNA and protein levels indicate the significant role of IFT80 in chondrocyte differentiation. Furthermore, silencing IFT80 expression in BMSCs blocks cilia assembly and interferes with differentiation in chondrocytes with diminished Hh signaling pathway activity and enhanced Wnt/β-catenin signaling activity [72].

IFT88, also known as Tg737 or Polaris, localizes to the basal bodies of primary cilia [73]. Wann et al. studied the role of IFT88 in chondrocyte cilia in response to the compressive strain *in vitro* [74]. The study compared Ca^{2+} signaling, extracellular matrix synthesis, and ATP release after loading in WT and $IFT88^{orpk}$ mutant chondrocytes, which lacked cilia. Both cell types produced ATP after loading, but $IFT88^{orpk}$ mutant chondrocytes did not show a Ca^{2+} response to ATP, demonstrating the important role of primary cilia in chondrocyte mechanotransduction. McGlashan et al. studied the link between long bone growth and primary cilia in chondrocytes [75]. *Orpk* mice carry a hypomorphic mutation in the Tg737 gene that results in the absence of polaris, a protein crucial for cilia formation. *Orpk* mice marked disrupted articular cartilage and showed limited growth in appositional and endochondral aspects with significantly reduced length and width in growth plates. These findings suggest that failure of functional primary ciliogenesis impacts chondrocyte differentiation and leads to retarded chondrocyte hypertrophy within the *orpk* growth plate [75]. In addition, Chang et al. examined the transgenic Col2aCre;$Ift88^{fl/fl}$ mice in which primary cilia were deleted in chondrocytes. IFT88 transgenic mice showed upregulation of Hh signaling and thick cartilage due to increased cell density and decreased apoptosis during remodeling. In addition, mutant cartilage presented the increased expression of osteoarthritis markers, supporting the role of primary cilia for the development and maintenance of articular cartilage [76].

The Ellis-van Creveld syndrome (EVC) protein is found at the base of primary cilia in chondrocytes and is important for chondrocyte differentiation. Mice that lacked *Evc* ($Evc^{-/-}$) presented the chondrocyte cilia but displayed EVC-like characteristics, such as impaired growth, narrow rib cage, supernumerary digits, and orofacial malformations along with progressive chondrocyte maturation in the growth plate. Expression of Indian hedgehog (IHH) was within normal limit in the growth plates of $Evc^{-/-}$ mice, but IHH transduction was hindered as the IHH downstream genes, such as Ptch1 and Gli1 were noticeably reduced, supporting that EVC is an intracellular component of the Hh signal transduction pathway [77].

In the postnatal growth plate, chondrocytes align themselves in columns parallel to the long axis of bone. Lack of *Kif3a* in chondrocytes (*Kif3a*;Col2α1-Cre) resulted in cilia loss and disorganization in the growth plate and changes in chondrocytes columnar orientation. *Kif3a*;Col2α1-Cre mice showed postnatal dwarfism, because decreased cell proliferation and enhanced hypertrophic differentiation resulted in loss of the growth plate prematurely. These finding indicate that IFT proteins and primary cilia play crucial roles during the development of the postnatal growth plate, maintaining the columnar arrangement through the process of chondrocytes rotation [78].

The roles of cilia-related genes in osteoblasts, osteocytes and chondrocytes are summarized in Table 1.

Table 1. Summary of the Role of Cilia-related Genes in Osteoblasts, Osteocytes and Chondrocytes.

Ciliary Genes	Osteoblasts	References
IFT20	Deletion of IFT20 in osteoblastic lineage cells resulted in disrupted osteoblast polarity and loss of primary cilia formation. Conditional deletions of IFT20 in osteoblast precursors and differentiated osteoblasts (IFT20f/f;Col1-CreERT mice and IFT20f/f;OSX-Cre mice) showed increased hydroxylysine-aldehyde cross-linking and decreased lysine-aldehyde crosslinking, indicating its importance in collagen biosynthesis and subsequent bone formation.	52, 53
IFT80	Silencing of IFT80 in mesenchymal progenitor line and bone marrow derived stromal cells resulted in defective cilia formation and inhibited osteoblastic differentiation through decreased expression of osteoblast marker genes.	54
	IFT80 deletion in mice (IFT80$^{gt/gt}$) displayed similar phenotypes as Jeune asphyxiating thoracic dystrophy and short rib polydactyly type III.	55
	IFT80 deletion in osteoblasts (OSX^{cretTA}IFT80$^{f/f}$) exhibited reduced cilia formation and delayed fracture healing.	56
IFT88	Deletion of IFT88 in mature osteoblasts resulted in decreased number of osteoblasts and bone density in mice of a mixed genetic background while mice with inbred C57BL/6J background showed increased bone volume/total volume ratio and trabecular thickness.	17, 36
IFT140	IFT140 levels were highly expressed in bone during development with expression levels varying during different stages of endochondral ossification.	57
	Deletion of IFT140 in pre-osteoblastic cells (IFT140 cKO) resulted in hypomorphic mice, decreased bone formation, and delayed growth along with decreased expression levels of osteoblastic markers.	40
IFT144	IFT144 mutations are associated with human ciliopathies. Enhanced hedgehog signaling in addition to defective cilia formation was observed in mice with IFT144 mutation (Ift144^{twt} mice).	39
Kif3a	Deletion of Kif3a resulted in defective cilia formation and bone formation in response to ulnar loading in Colα1(I) 2.3-Cre;Kif3a$^{fl/fl}$ mice.	58
	Conditional deletion of Kif3a in mice (Col1Cre;Kif3a$^{fl/fl}$) tibia exhibited defective primary cilia in addition to decreased response to mechanical loading and failure of pre-osteoblasts to differentiate into mature osteoblasts around intra-osseous implants.	59

	Osteopenia, reduced femoral bone mineral density, trabecular bone volume and cortical thickness were observed in Kif3a knockout mice (Kif3a$^{Oc\text{-}cKO}$).	60

Ciliary Genes	Osteocytes	References
IFT20	Deletion of IFT20 (IFT20f/f; Col1cre-ERT) in osteoblastic lineage mice cells led to decreased cilia formation and length in osteocytes and unorganized osteocyte alignment.	52
PKD1	Mice with a missense mutation of PKD1 (*Pkd1*^{m1Bei}) disrupted intramembranous and endochondral bone formation in transgenic mice.	63
	Conditional knockout of PKD1 (*Pkd1*$^{flox/cko}$) in mice led to decreased trabecular volume and cortical bone thickness compared to wildtype and decrease gene expression of osteocyte downstream signals.	68

Ciliary Genes	Chondrocytes	References
IFT46	IFT46 is responsible for ciliogenesis and craniofacial cartilage development. Knockdown of IFT46 induced a significant reduction in the cilia number and length, which caused ciliogenesis and severe impairment of cartilage formation.	70
	IFT46 knockdown by siRNA promoted the skeletogenesis-related genes while there was no impact on the chondrogenesis-related gene expressions.	71
IFT80	IFT80 is an important regulator of chondrocyte differentiation during hedgehog (Hh) and Wingless (Wnt) signaling pathways.	72
IFT88	IFT88 has an important role in downstream signaling in chondrocyte during compressive loading. IFT88orpk mutant chondrocytes did not show a Ca2+ response to ATP, demonstrating the important role of primary cilia in chondrocyte mechanotransduction.	74
	In Tg737*orpk* (orpk) mice, disruption of IFT88 impacts primary cilia in chondrocyte and is associated with impeded growth of the long bones.	75
	Col2aCre;Ift88$^{fl/fl}$ mice had thickened cartilage due to increased cell density and decreased apoptosis during cartilage remodeling with the increased expression of osteoarthritis markers.	76
EVC	Evc−/− mice had the cilia in chondrocytes but displayed EVC-like characteristics with progressive chondrocyte maturation in the growth plate and hindered bone collar formation.	77

Kif3a	Kif3a;Col2α1-Cre mice results in loss of cilia along the growth plate, characterized by the disoriented growth plate and modifications to chondrocytes columnar orientation. Kif3a;Col2α1-Cre mice shows postnatal dwarfism due to loss of the growth plate prematurely.	78

POSSIBLE ROLE OF PRIMARY CILIA IN ORTHODONTIC TOOTH MOVEMENT

OTM is a highly coordinated process where mechanical force application causes the alveolar bone remodeling through bone resorption on the compression side and bone apposition on the tension side, mediated by the periodontal ligament (PDL). Among several theories explaining this remodeling process, the fluid flow theory has a possible link to the role of primary cilia [79]. This theory proposes that the PDL continues to act as a hydrostatic system where the PDL space fluids could potentially trigger sensory systems and induce a cellular response. Since the dental socket wall possesses fenestrations that enable the expression of compressed fluid from the PDL to the marrow spaces, not only the PDL cells can detect the fluid flow dynamics but osteocytes and cells in the marrow spaces can also mediate a cellular response. This observation suggests primary cilium's involvement in fluid flow and cellular mechanotransduction during OTM. Disruption of cilia assembly in murine osteoblast inhibits expression of COX2, osteopontin, and PGE2 and reduces mechanosensing response to fluid flow [14].

A Ca^{2+} channel complex comprises the transmembrane proteins including polycystin-1 (PC1) and polycystin-2 (PC2) and is responsible for mediating the effect of cilia oscillation [80]. Shalish et al. reported that PC1 mutation in the craniofacial region led to impaired tooth movement and reduction in osteoclast activity [81]. The 7-week-old transgenic PC1/Wnt1-cre mice that lack PC1 in the craniofacial region and WT mice were subject to 20g orthodontic force for four days using a NiTi coil spring placed between the incisors and the left 1st molar. In WT mice, the molar significantly moved mesially with increased number of osteoclasts. On the other hand, the PC1/Wnt1-cre mutant mice showed no molar movement, palatally tipped incisor, and slightly wide PDL in the tension area. TRAP staining could not detect osteoclasts on the compression side in transgenic mice. These differences possibly resulted from the absence of signal from PDL due to PC1 deficiency, suggesting the critical role of PC1 in OTM as a mechanical sensor [81].

Silencing of prime ciliary proteins, such as ITF80, IFT88, EVC, kif3a, and PC in osteoblasts resulted in disruption in cilia formation *in vivo*. In absence of ciliogenesis, loss of mechanosensory transduction led to impaired osteoblast function and differentiation, decreased osteoid formation, as well as inhibition of bone mineralization under mechanical loading [14, 54, 60]. These results may provide insight into how primary cilia may serve an essential role in bone apposition mediated by osteoblast from the tension during OTM.

CONCLUSIONS

This review explored the potential roles of primary cilia and IFT proteins in osteoblasts, osteocytes, and chondrocytes during bone remodeling. Current studies support the function of primary cilia as a mechanosensory and chemosensory organelle that responds to load-induced fluid flow in bone remodeling. Moreover, the possible implication of primary cilia in orthodontic tooth movement was discussed. However, future studies are still needed to fully comprehend the mechanisms behind the mechanotransduction of primary cilia and its role in regulating skeletal homeostasis. Potential therapies that target diseases with ciliopathies or disorders caused by dysfunctional responses to loading can be assessed in the future.

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REFERENCES

1. Malone AM, Anderson CT, Tummala P, Kwon RY, Johnston TR, Stearns T, Jacobs CR. Primary cilia mediate mechanosensing in bone cells by a calcium-independent mechanism. Proc Natl Acad Sci USA 2007; 104(33):13325-13330.
2. Temiyasathit S, Jacobs CR. Osteocyte primary cilium and its role in bone mechanotransduction. Ann N Y Acad Sci 2010; 1192:422-428.
3. Tan SD, de Vries TJ, Kuijpers-Jagtman AM, Semeins CM, Everts V, Klein-Nulend J. Osteocytes subjected to fluid flow inhibit osteoclast formation and bone resorption. Bone 2007; 41(5):745-751.
4. Delaine-Smith RM, Sittichokechaiwut A, Reilly GC. Primary cilia respond to fluid shear stress and mediate flow-induced calcium deposition in osteoblasts. FASEB J 2014; 28(1):430-439.
5. Wittkowske C, Reilly GC, Lacroix D, Perrault CM. In Vitro Bone Cell Models: Impact of Fluid Shear Stress on Bone Formation. Front Bioeng Biotechnol 2016; 4:87.
6. Mohamed AM. An overview of bone cells and their regulating factors of differentiation. Malays J Med Sci 2008; 15(1):4-12.
7. Ottewell PD. The role of osteoblasts in bone metastasis. J Bone Oncol 2016; 5(3):124-127.
8. Bellido T. Osteocyte-driven bone remodeling. Calcif Tissue Int 2014; 94(1):25-34.
9. Creecy A, Damrath JG, Wallace JM. Control of Bone Matrix Properties by Osteocytes. Front Endocrinol (Lausanne) 2020; 11:578477.
10. Akkiraju H, Nohe A. Role of Chondrocytes in Cartilage Formation, Progression of Osteoarthritis and Cartilage Regeneration. J Dev Biol 2015; 3(4):177-192.
11. Spasic M, Jacobs CR. Primary cilia: Cell and molecular mechanosensors directing whole tissue function. Semin Cell Dev Biol 2017; 71:42-52.
12. Anderson CT, Castillo AB, Brugmann SA, Helms JA, Jacobs CR, Stearns T. Primary cilia: cellular sensors for the skeleton. Anat Rec (Hoboken) 2008; 291(9):1074-1078.
13. Wheway G, Nazlamova L, Hancock JT. Signaling through the Primary Cilium. Front Cell Dev Biol 2018; 6:8.
14. Yuan X, Yang S. Primary Cilia and Intraflagellar Transport Proteins in Bone and Cartilage. J Dent Res 2016; 95(12):1341-1349.
15. Chinipardaz Z, Liu M, Graves DT, Yang S. Role of Primary Cilia in Bone and Cartilage. J Dent Res 2022; 101(3):253-260.
16. Verhey KJ, Dishinger J, Kee HL. Kinesin motors and primary cilia. Biochem Soc Trans 2011; 39(5):1120-1125.
17. Yuan X, Serra RA, Yang S. Function and regulation of primary cilia and intraflagellar transport proteins in the skeleton. Ann NY Acad Sci 2015; 1335:78-99.
18. Klink BU, Gatsogiannis C, Hofnagel O, Wittinghofer A, Raunser S: Structure of the human BBSome core complex. Elife 2020; 9.
19. Nachury MV, Seeley ES, Jin H. Trafficking to the ciliary membrane: how to get across the periciliary diffusion barrier? Annu Rev Cell Dev Biol 2010; 26:59-87.

20. Wingfield JL, Lechtreck KF, Lorentzen E. Trafficking of ciliary membrane proteins by the intraflagellar transport/BBSome machinery. Essays Biochem 2018; 62(6):753-763.
21. Follit JA, Tuft RA, Fogarty KE, Pazour GJ. The intraflagellar transport protein IFT20 is associated with the Golgi complex and is required for cilia assembly. Mol Biol Cell 2006; 17(9):3781-3792.
22. Jonassen JA, San Agustin J, Follit JA, Pazour GJ. Deletion of IFT20 in the mouse kidney causes misorientation of the mitotic spindle and cystic kidney disease. J Cell Biol 2008; 183(3):377-384.
23. Noda K, Kitami M, Kitami K, Kaku M, Komatsu Y. Canonical and noncanonical intraflagellar transport regulates craniofacial skeletal development. Proc Natl Acad Sci USA 2016; 113(19):E2589-2597.
24. Xue B, Liu YX, Dong B, Wingfield JL, Wu M, Sun J, Lechtreck KF, Fan ZC. Intraflagellar transport protein RABL5/IFT22 recruits the BBSome to the basal body through the GTPase ARL6/BBS3. Proc Natl Acad Sci USA 2020; 117(5):2496-2505.
25. Liu H, Li W, Zhang Y, Zhang Z, Shang X, Zhang L, Zhang S, Li Y, Somoza AV, Delpi B et al. IFT25, an intraflagellar transporter protein dispensable for ciliogenesis in somatic cells, is essential for sperm flagella formation. Biol Reprod 2017; 96(5):993-1006.
26. Liew GM, Ye F, Nager AR, Murphy JP, Lee JS, Aguiar M, Breslow DK, Gygi SP, Nachury MV. The intraflagellar transport protein IFT27 promotes BBSome exit from cilia through the GTPase ARL6/BBS3. Dev Cell 2014; 31(3):265-278.
27. Lee MS, Hwang KS, Oh HW, Kim, JA, Kim HT, Cho HS, Lee JJ, Yeong Ko J, Choi JH, Jeong YM et al. IFT46 plays an essential role in cilia development. Dev Biol 2015; 400(2):248-257.
28. Zhang W, Taylor SP, Nevarez L, Lachman RS, Nickerson DA, Bamshad M, University of Washington Center for Mendelian Genomics C, Krakow D, Cohn DH. IFT52 mutations destabilize anterograde complex assembly, disrupt ciliogenesis and result in short rib polydactyly syndrome. Hum Mol Genet 2016; 25(18):4012-4020.
29. Zhu X, Wang J, Li S, Lechtreck K, Pan J. IFT54 directly interacts with kinesin-II and IFT dynein to regulate anterograde intraflagellar transport. EMBO J 2021; 40(5):e105781.
30. Krock BL, Perkins BD. The intraflagellar transport protein IFT57 is required for cilia maintenance and regulates IFT-particle-kinesin-II dissociation in vertebrate photoreceptors. J Cell Sci 2008; 121(11):1907-1915.
31. Takei R, Katoh Y, Nakayama K. Robust interaction of IFT70 with IFT52-IFT88 in the IFT-B complex is required for ciliogenesis. Biol Open 2018; 7(5).
32. Bhogaraju S, Cajanek L, Fort C, Blisnick T, Weber K, Taschner M, Mizuno N, Lamla S, Bastin P, Nigg EA et al. Molecular basis of tubulin transport within the cilium by IFT74 and IFT81. Science 2013; 341(6149):1009-1012.
33. Duran I, Taylor SP, Zhang W, Martin J, Forlenza KN, Spiro RP, Nickerson DA, Bamshad M, Cohn DH, Krakow D. Destabilization of the IFT-B cilia core complex due to mutations in IFT81 causes a Spectrum of Short-Rib Polydactyly Syndrome. Sci Rep 2016; 6:34232.
34. Yuan X, Cao J, He X, Serra R, Qu J, Cao X, Yang S. Ciliary IFT80 balances canonical versus non-canonical hedgehog signalling for osteoblast differentiation. Nat Commun 2016; 7:11024.
35. Tian H, Feng J, Li J, Ho TV, Yuan Y, Liu Y, Brindopke F, Figueiredo JC, Magee W, Sanchez-Lara PA et al. Intraflagellar transport 88 (IFT88) is crucial for craniofacial development in mice and is a candidate gene for human cleft lip and palate. Hum Mol Genet 2017; 26(5):860-872.
36. Session S. Ift88 function in the murine skeleton. In: ASBMR 2010 Annual Meeting: 2010; Toronto; 2010.
37. Roberts AJ. Emerging mechanisms of dynein transport in the cytoplasm versus the cilium. Biochem Soc Trans 2018; 46(4):967-982.
38. Pruski M, Hu L, Yang C, Wang Y, Zhang JB, Zhang L, Huang Y, Rajnicek AM, St Clair D, McCaig CD et al. Roles for IFT172 and Primary Cilia in Cell Migration, Cell Division, and Neocortex Development. Front Cell Dev Biol 2019; 7:287.

39. Ashe A, Butterfield NC, Town L, Courtney AD, Cooper AN, Ferguson C, Barry R, Olsson F, Liem KF, Jr., Parton RG et al. Mutations in mouse Ift144 model the craniofacial, limb and rib defects in skeletal ciliopathies. Hum Mol Genet 2012; 21(8):1808-1823.
40. Tao D, Xue H, Zhang C, Li G, Sun Y. The Role of IFT140 in Osteogenesis of Adult Mice Long Bone. J Histochem Cytochem 2019; 67(8):601-611.
41. Hirano T, Katoh Y, Nakayama K. Intraflagellar transport-A complex mediates ciliary entry and retrograde trafficking of ciliary G protein-coupled receptors. Mol Biol Cell 2017; 28(3):429-439.
42. Takahara M, Katoh Y, Nakamura K, Hirano T, Sugawa M, Tsurumi Y, Nakayama K: Ciliopathy-associated mutations of IFT122 impair ciliary protein trafficking but not ciliogenesis. Hum Mol Genet 2018; 27(3):516-528.
43. Duran I, Taylor SP, Zhang W, Martin J, Qureshi F, Jacques SM, Wallerstein R, Lachman RS, Nickerson DA, Bamshad M et al. Mutations in IFT-A satellite core component genes IFT43 and IFT121 produce short rib polydactyly syndrome with distinctive campomelia. Cilia 2017; 6:7.
44. Prevo B, Scholey JM, Peterman EJG. Intraflagellar transport: mechanisms of motor action, cooperation, and cargo delivery. FEBS J 2017; 284(18):2905-2931.
45. Katsianou MA, Skondra FG, Gargalionis AN, Piperi C, Basdra EK. The role of transient receptor potential polycystin channels in bone diseases. Ann Transl Med 2018; 6(12):246.
46. Xiao ZS, Quarles LD. Role of the polycytin-primary cilia complex in bone development and mechanosensing. Ann NY Acad Sci 2010; 1192:410-421.
47. Blair HJ, Tompson S, Liu YN, Campbell J, MacArthur K, Ponting CP, Ruiz-Perez VL, Goodship JA. Evc2 is a positive modulator of Hedgehog signalling that interacts with Evc at the cilia membrane and is also found in the nucleus. BMC Biol 2011; 9:14.
48. Caparros-Martin JA, Valencia M, Reytor E, Pacheco M, Fernandez M, Perez-Aytes A, Gean E, Lapunzina P, Peters H, Goodship JA et al. The ciliary Evc/Evc2 complex interacts with Smo and controls Hedgehog pathway activity in chondrocytes by regulating Sufu/Gli3 dissociation and Gli3 trafficking in primary cilia. Hum Mol Genet 2013; 22(1):124-139.
49. Raggatt LJ, Partridge NC. Cellular and molecular mechanisms of bone remodeling. J Biol Chem 2010; 285(33):25103-25108.
50. Moore ER, Zhu YX, Ryu HS, Jacobs CR. Periosteal progenitors contribute to load-induced bone formation in adult mice and require primary cilia to sense mechanical stimulation. Stem Cell Res Ther 2018; 9(1):190.
51. Chen JC, Hoey DA, Chua M, Bellon R, Jacobs CR. Mechanical signals promote osteogenic fate through a primary cilia-mediated mechanism. FASEB J 2016; 30(4):1504-1511.
52. Lim J, Li X, Yuan X, Yang S, Han L, Yang S. Primary cilia control cell alignment and patterning in bone development via ceramide-PKCzeta-beta-catenin signaling. Commun Biol 2020; 3(1):45.
53. Yamaguchi H, Terajima M, Kitami M, Wang J, He L, Saeki M, Yamauchi M, Komatsu Y. IFT20 is critical for collagen biosynthesis in craniofacial bone formation. Biochem Biophys Res Commun 2020; 533(4):739-744.
54. Yang S, Wang C. The intraflagellar transport protein IFT80 is required for cilia formation and osteogenesis. Bone 2012; 51(3):407-417.
55. Rix S, Calmont A, Scambler PJ, Beales PL. An Ift80 mouse model of short rib polydactyly syndromes shows defects in hedgehog signalling without loss or malformation of cilia. Hum Mol Genet 2011; 20(7):1306-1314.
56. Chinipardaz Z, Liu M, Graves D, Yang S. Diabetes impairs fracture healing through disruption of cilia formation in osteoblasts. Bone 2021; 153:116176.
57. Zhang C, Zhang S, Sun Y. Expression of IFT140 During Bone Development. J Histochem Cytochem 2019; 67(10):723-734.

58. Temiyasathit S, Tang WJ, Leucht P, Anderson CT, Monica SD, Castillo AB, Helms JA, Stearns T, Jacobs CR. Mechanosensing by the primary cilium: deletion of Kif3A reduces bone formation due to loading. PLoS One 2012; 7(3):e33368.
59. Leucht P, Monica SD, Temiyasathit S, Lenton K, Manu A, Longaker MT, Jacobs CR, Spilker RL, Guo H, Brunski JB et al. Primary cilia act as mechanosensors during bone healing around an implant. Med Eng Phys 2013; 35(3):392-402.
60. Qiu N, Xiao Z, Cao L, Buechel MM, David V, Roan E, Quarles LD. Disruption of Kif3a in osteoblasts results in defective bone formation and osteopenia. J Cell Sci 2012; 125(Pt 8):1945-1957.
61. Rochefort GY, Pallu S, Benhamou CL. Osteocyte: the unrecognized side of bone tissue. Osteoporos Int 2010; 21(9):1457-1469.
62. Schaffler MB, Cheung WY, Majeska R, Kennedy O. Osteocytes: master orchestrators of bone. Calcif Tissue Int 2014; 94(1):5-24.
63. Xiao Z, Zhang S, Mahlios J, Zhou G, Magenheimer BS, Guo D, Dallas SL, Maser R, Calvet JP, Bonewald L et al. Cilia-like structures and polycystin-1 in osteoblasts/osteocytes and associated abnormalities in skeletogenesis and Runx2 expression. J Biol Chem 2006; 281(41):30884-30895.
64. Coughlin TR, Voisin M, Schaffler MB, Niebur GL, McNamara LM. Primary cilia exist in a small fraction of cells in trabecular bone and marrow. Calcif Tissue Int 2015; 96(1):65-72.
65. Tonna EA, Lampen NM. Electron microscopy of aging skeletal cells. I. Centrioles and solitary cilia. J Gerontol 1972; 27(3):316-324.
66. Uzbekov RE, Maurel DB, Aveline PC, Pallu S, Benhamou CL, Rochefort GY. Centrosome fine ultrastructure of the osteocyte mechanosensitive primary cilium. Microsc Microanal 2012; 18(6):1430-1441.
67. Qin L, Liu W, Cao H, Xiao G. Molecular mechanosensors in osteocytes. Bone Res 2020; 8:23.
68. Xiao Z, Dallas M, Qiu N, Nicolella D, Cao L, Johnson M, Bonewald L, Quarles LD. Conditional deletion of Pkd1 in osteocytes disrupts skeletal mechanosensing in mice. FASEB J 2011; 25(7):2418-2432.
69. Kronenberg HM. Developmental regulation of the growth plate. Nature 2003; 423(6937):332-336.
70. Park I, Lee HK, Kim C, Ismail T, Kim YK, Park JW, Kwon OS, Kang BS, Lee DS, Park TJ et al. IFT46 plays crucial roles in craniofacial and cilia development. Biochem Biophys Res Commun 2016; 477(3):419-425.
71. Gouttenoire J, Valcourt U, Bougault C, Aubert-Foucher E, Arnaud E, Giraud L, Mallein-Gerin F. Knockdown of the intraflagellar transport protein IFT46 stimulates selective gene expression in mouse chondrocytes and affects early development in zebrafish. J Biol Chem 2007; 282(42):30960-30973.
72. Wang C, Yuan X, Yang S. IFT80 is essential for chondrocyte differentiation by regulating Hedgehog and Wnt signaling pathways. Exp Cell Res 2013; 319(5):623-632.
73. Haycraft CJ, Swoboda P, Taulman PD, Thomas JH, Yoder BK. The C. elegans homolog of the murine cystic kidney disease gene Tg737 functions in a ciliogenic pathway and is disrupted in osm-5 mutant worms. Development 2001; 128(9):1493-1505.
74. Wann AK, Zuo N, Haycraft CJ, Jensen CG, Poole CA, McGlashan SR, Knight MM. Primary cilia mediate mechanotransduction through control of ATP-induced Ca2+ signaling in compressed chondrocytes. FASEB J 2012; 26(4):1663-1671.
75. McGlashan SR, Haycraft CJ, Jensen CG, Yoder BK, Poole CA. Articular cartilage and growth plate defects are associated with chondrocyte cytoskeletal abnormalities in Tg737orpk mice lacking the primary cilia protein polaris. Matrix Biol 2007; 26(4):234-246.
76. Chang CF, Ramaswamy G, Serra R. Depletion of primary cilia in articular chondrocytes results in reduced Gli3 repressor to activator ratio, increased Hedgehog signaling, and symptoms of early osteoarthritis. Osteoarthritis Cartilage 2012; 20(2):152-161.

77. Ruiz-Perez VL, Blair HJ, Rodriguez-Andres ME, Blanco MJ, Wilson A, Liu YN, Miles C, Peters H, Goodship JA. Evc is a positive mediator of Ihh-regulated bone growth that localises at the base of chondrocyte cilia. Development 2007; 134(16):2903-2912.
78. Song B, Haycraft CJ, Seo HS, Yoder BK, Serra R. Development of the post-natal growth plate requires intraflagellar transport proteins. Dev Biol 2007; 305(1):202-216.
79. Bien SM. Fluid dynamic mechanisms which regulate tooth movement. Adv Oral Biol 1966; 2:173-201.
80. Nauli SM, Alenghat FJ, Luo Y, Williams E, Vassilev P, Li X, Elia AE, Lu W, Brown EM, Quinn SJ et al. Polycystins 1 and 2 mediate mechanosensation in the primary cilium of kidney cells. Nat Genet 2003; 33(2):129-137.
81. Shalish M, Will LA, Fukai N, Hou B, Olsen BR. Role of polycystin-1 in bone remodeling: orthodontic tooth movement study in mutant mice. Angle Orthod 2014; 84(5):885-890.

EMERGENCE OF MACHINE LEARNING TOOLS IN ORTHODONTIC PRACTICE AND RESEARCH

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ABSTRACT

After the emergence of artificial intelligence (AI) in the 1950s, the technology went through periods of optimism ("AI spring"), promising life-changing applications followed by periods of disappointment ("AI winter"), where the promises seemed unattainable. During the past decade, fast paced breakthroughs in machine learning (ML) reached the point where AI actually started to have a direct and increasing impact on the quality of everyday life. It seemed as though we were in a long and promising "spring" in which not a single sector was spared, including the field of orthodontics. This chapter offers a quick overview of the roles that machine learning is currently playing in assisting in orthodontics and orthodontic research. ML is able to process images more quickly and with greater consistency via automated clinical decision support systems than what can be accomplished by humans when performing basic steps such as landmark identification and segmentation.

KEY WORDS: Automated Tools, Segmentation, Landmark Identification, Deep Learning, Machine Learning

INTRODUCTION

After many breakthroughs in computing power and AI research over the past decade, the development of machine learning tools has had a direct and increasing impact on everyday life. Currently, most social networks offer personalized recommendations to consumers through AI algorithms [1, 2]. In agriculture, ML applications in various areas have shown outstanding results, from detecting weeds and diseases, to offering predictions regarding livestock production and quality of crops [3]. Automobile companies get closer each day to a fully self-driving car [4]. In research, Google DeepMind recently made AlphaFold, a solution to a 50-year-old grand challenge in biology, available for free to the whole world [5]. DeepMind has already been used to predict protein structures of SARS-CoV-2 [6]. The list of examples is endless, not a single field is spared by the emergence of ML tools, including orthodontics.

Accurate segmentation, registration, multimodality fusion, and quantification of multi-facial structures, including the maxilla, mandible, mandibular nerve canal, dental crowns, roots, and their root canals are vital to determine safety margins in the bones, adjacent teeth, and gingival margins in dental, oral, and craniofacial (DOC) care. The current tools such as ITK-SNAP [7] and 3D Slicer [8] are set up for individual and manual scan registration. It is a time-consuming and challenging task, due to the low signal/noise ratio, particularly in full face cone-beam computed tomography (CBCT) scans. Developing AI to perform fast and robust registration, segmentation, and landmark identification on a large set of CBCT or intraoral scans (IOS) could also allow experts to reach high stability and agreement on orthodontic decision-making procedures and treatment effect evaluation.

Recent ML models have been found to perform at or above the accuracy levels achieved by humans in landmark identification, skeletal classification, bone age prediction, and tooth segmentation. Commercial companies have recently marketed AI-based segmentation and registration tools for CBCT and IOS scans, but they are expensive and the precision of their algorithms is not yet generalizable and they do not provide quantitative decision support tools. Our lab worked on improving this generalizability and developing free, open-source tools to perform segmentation and landmark identification tasks by using large databases. This goal was made possible thanks to a collaboration of 15 clinician centers from all over the world: University of Michigan, University of Pacific, University of Minnesota, University of Medellin (Colombia), University Científica del Sur (Peru), University of Firenze and Genoa (Italy), State University of Sao Paulo, Federal University of Ceara, Federal University of Goias and Federal University of Rio de Janeiro, Hospital for Rehabilitation of Craniofacial Anomalies in Bauru (Brazil), and two private practices in the US and in Egypt. Through common effort and collaboration, we have developed four machine learning tools ready to be used by clinicians on 3D Slicer and our web-based system, the Data Storage for Computation and Integration (DSCI). FiboSeg and AMASSS were developed to automatically segment IOS and CBCT, respectively, while Automatic Landmark Identification (ALI) in CBCT ALI-CBCT and in IOS (ALI-IOS) specialize in landmark identification on both supports.

MACHINE LEARNING

An AI is the simulation of human intelligence processes by machines. Simply, it can be considered as a function that intelligently processes the input to give output as if computer systems were smart. Machine Learning is a subcategory of artificial intelligence, that refers to the process by which computers learn from a given dataset to generalize and make accurate predictions when exposed to new data. In ML the function is a mathematical equation made of adjustable parameters represented by what we call a neural network, or artificial neural network (ANN) [9]. It requires an input layer and an output layer separated by one or more hidden layers. We speak about deep learning when a neural network consists of more than three layers. Today's deepest neural Network is GPT-3 from Open AI [10]. It is a model with over 175 billion machine learning parameters specialized to produce human-like text. This number might seem large, but it stays rather small compared to over 100 trillion synapses (up to 1,000 trillion, by some estimates) connecting around 100 billion neurons in the human brain [11].

For an AI, learning means adjusting the parameters of the neural network to return an expected output from a given input. The machine learning field is made of 4 main branches used to perform different tasks [12]. They differ in the strategy used to modify the network parameters. The most common are supervised and unsupervised learning followed by semi-supervised learning and reinforcement learning.

Supervised learning

As the name suggests, supervised learning involves learning in the presence of a supervisor. For machine learning algorithms, it means providing feedback on the performances during the training process. To do so, the given dataset is made of pre-labeled data. The parameters of the neural network are slightly updated at each training iteration to reduce the error between the network prediction and the labeled output. It is used for tasks such as image recognition, medical diagnosis, statistical arbitrage, predictive analysis, and data extraction. A well-known and widespread example of machine learning applications is facial recognition, which can be used to unlock smartphones [13].

Unsupervised learning

Unsupervised learning, in contrast to supervised learning, is a type of algorithm that learns patterns from unlabeled data. The machine is fed raw data and is forced to build a compact internal representation of its world. It is mainly developed to do clustering, anomaly detection, market segmentation, or dimensionality reduction. Financial organizations utilize the technique to spot fraudulent transactions [14]. It is also widely used for more efficient marketing and targeting campaigns by sorting customers' information.

Semi-supervised learning

Semi-supervised machine learning combines supervised and unsupervised learning. It uses a large amount of unlabeled data mixed with some labeled data. This strategy provides the benefits of both previous learning strategies while avoiding the challenges of finding a large amount of labeled data. It is used to perform speech recognition, web content classification, and text document classification.

Reinforcement learning

Reinforcement learning is a special and complex subfield of ML. The AI is trained with a system of reward and punishment. The developers create a virtual environment that gives rewards when the ML algorithm performs desired behaviors and punishment for negative behaviors. In this context, AI evolves to maximize the reward. It is a very promising field of ML used to train robots and computers to interact with the real world. A good example is the Google DeepMind Challenge Match where AlphaGo [15], an AI trained to play Go, won 4 of the 5 games against the top human Go player Lee Sedol in 2016. More recently, the robot company Boston Dynamics released a video of their robots' ability to evolve and dance with outstanding smoothness in a challenging real-life environment [16].

ORTHODONTIC TOOLS

The machine learning tools we developed belong to the supervised learning branch. We used large datasets pre-labeled by expert clinicians. We specifically focused on developing the following tools to perform important tasks which are segmentation and landmark identification on cone beam computed tomography (CBCT) and intraoral optical scan IOS data.

AMASSS - CBCT (Automatic multi-anatomical skull structure segmentation in CBCT)

The segmentation of medical and dental images is a fundamental step in automated clinical decision support systems. It supports the entire clinical workflow from diagnosis, therapy planning, intervention, and follow-up. With medical and dental images being acquired at multiple scales and/or with multiple imaging modalities, automated image analysis techniques are required to integrate patient data across scales of observation. Anatomic image segmentation, registration, and multimodality fusion using current open-source tools, such as ITK-SNAP and 3D Slicer are time-consuming and challenging for clinicians and researchers due to the low signal/noise ratio. Particularly in the large field of view CBCT images that are commonly used for orthodontics and oral maxillofacial surgery clinical applications. To perform a manual full-face segmentation, 7 hours of work on average is required by experienced clinicians, including approximately 1.5h for the mandible, 2h for the maxilla and 2h for the cranial base (CB), 1h for the cervical vertebra (CV), and 0.5h for the skin. The accurate and robust automatic anatomical segmentation of medical imaging data is a challenging problem due to the rich variety of anatomical structures and the difference in scan acquisition protocols from one center to another. Patients with facial bone defects may pose additional challenges for automatic segmentation. For this reason, in our study, we also included gold standard (ground truth) segmentations of CBCT images from patients with craniofacial large bone defects such as cleft lip and palate (CLP). Being able to accurately segment those deformities in the maxilla is challenging but necessary to generate 3D models for the diagnosis and treatment planning of patients with craniomaxillofacial anomalies (Figure 1). To answer this request, our lab developed a tool to automatically and accurately process a full-face segmentation in about 5 minutes using the state-of-the-art UNEt TRansformers (UNETR), a neural network of the Medical Open Network for Artificial Intelligence (MONAI) framework. We trained and tested our models using 618 de-identified CBCT volumetric images of the head acquired with several parameters from different centers for a generalized clinical application. Our results showed high accuracy and robustness. This tool can be used to segment the mandible, the maxilla, the cranial base, the cervical vertebra, and the airways in large CT as well as the crown, root, and mandible canal in a small field of view.

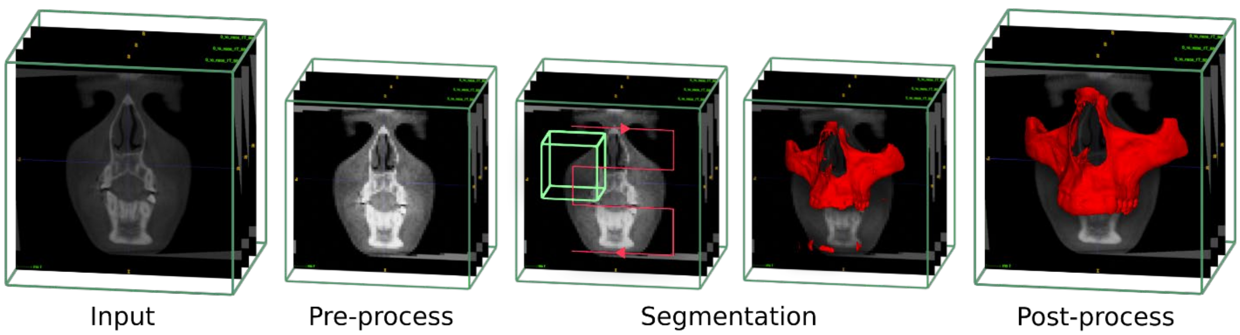

Figure 1. Visualization of the automatic maxilla segmentation steps. Re-sample and contrast adjustment of the input image, segmentation with the sliding window using UNETR, and finally, re-sampling of the cleaned-up segmentation to the input size.

ALI - CBCT (Automatic Landmark Identification in CBCT)

Robust and accurate solutions for anatomical landmark detection is a fundamental task to perform image-to-image registration, structure tracking, and simulations. We developed a new approach that reformulates landmark detection as a classification problem through a virtual agent placed in a 3D CBCT scan. This agent is trained to navigate in a multi-scale volumetric space to reach the estimated landmark position. The landmark detection task is set up as a behavior classification problem for an artificial agent that navigates through the voxel grid of the image at different spatial resolutions (the agent's environments). The detection starts at a low-resolution image with a global context and continues at the higher-resolution image, thereby capturing increased levels of detail. The image features are used as indicators to guide the landmark search. To adapt the feature extraction, we train different neural networks at each resolution. After the feature extraction, the network takes the image features as input and decides in which direction the agent should move to get closer to its target landmark position. The agent's movement decision relies on a combination of Densely Connected Convolutional Networks (DCCN) and fully connected layers. To predict the landmark positions in a CBCT, we rescale it to the resolutions used during training (here 1mm and 0.3mm voxel size). For each landmark to predict, an agent is generated with its corresponding network.

The landmark prediction is then made in 3 steps (Figure 2):

- **Step 1:** The prediction begins at the low-resolution level. The agent is placed in the middle of the scan to optimize the search time. Once the agent reaches a confident zone, it goes to the high-resolution layer.
- **Step 2:** The agent starts moving in the high-resolution from the confident zone. A preliminary estimation is set where the agent stops moving.
- **Step 3:** Now, a verification step is applied. This step consists of searching again in the high resolution scan starting from 6 positions (in each direction) in a small radius from the predicted point in **Step 2**. The result is an average of the 6 predicted positions.

The stopping criteria is active at prediction time and is implemented using a visitation map. If the agent tries to reach a previously visited voxel, it stops. The third step increased the prediction accuracy and compensated for a portion of the error caused by the discrete aspect of the space.

We evaluated our approach on 60 CBCT scans from teenagers to older patients. For each CBCT, 31 ground truth landmark positions were identified by clinicians. Our method achieved accuracy with an average of 1.54±0.87 mm error on the 31 landmark positions with rare failures. It takes an average of 4.2s computation time for the algorithm to identify each landmark on one large 3D CBCT scan. After a successful proof of concept, we trained new agents to reach a total of 119 landmarks that could be automatically identified using a 3D slicer module or the DSCI.

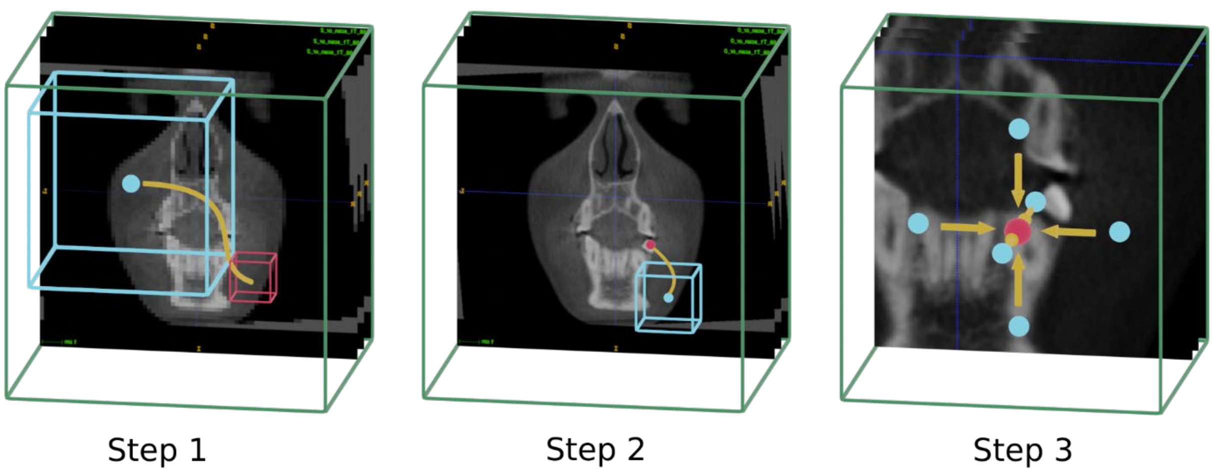

Figure 2. Visualization of the agent (blue) in the multi-scale environment (green) searching for the target (red).

FiboSeg - IOS (Fully automated segmentation of upper and lower jaws from 3D IOS.)

Developments in dentistry led to an improved application of 3D technologies such as IOS scanners which are used to design ceramic crowns, veneers, inlays, and occlusal guards, as well as to assist with implants. IOS is being used more often for automated diagnoses such as caries detection, analysis of risk factors of tooth movement, and treatment planning. These 3D surface models require shape analysis techniques for analyzing and understanding the geometry and they achieve state-of-the-art performance for tasks such as segmentation, classification, and retrieval. In this paper, we present a novel method for 3D surface segmentation based on a multi-view approach. Fast and accurate segmentation of the IOS remains a challenge due to various geometric shapes of teeth, complex tooth arrangements, different dental model qualities, and varying degrees of crowding problems. Our target application is the multi-class segmentation following the Universal Numbering System proposed by the American Dental Association (ADA), the dental notation system used in the US.

The multi-view approach consists of generating 2D images of the 3D surface from different viewpoints. The generated images serve as a training set for a neural network. We used Pytorch3D (open-source, https://pytorch.org/) to generate images of the surface on the fly during training and a one-to-one mapping that relates faces in the 3D model and pixels in the generated images (Figure 3). This is useful in inference time when we have to put the resulting labels from the images back into the 3D model.

This new method for automatic multi-class segmentation of 3D surfaces has proven to be accurate and effective, as well as easy to integrate into existing processing pipelines; there is no sub-sampling of the surface as it is required by the competing approaches such as MeshSegNet (open-source code available at https://github.com/Tai-Hsien/MeshSegNet) and PointConv (open-source code available at https://github.com/DylanWusee/pointconv). A great advantage of this method is the ability to predict the universal IDs of the crowns in the upper and lower jaws. The results reported by the competing approaches focused on upper models only or use a classification model to identify upper/lower jaws. Our approach is fully automated and labels both upper and lower crowns with a single model. The model learns to identify features specific to each jaw. We are aware that this model can still be improved and we can safely expect better results for segmentation of wisdom teeth as our sample size increases.

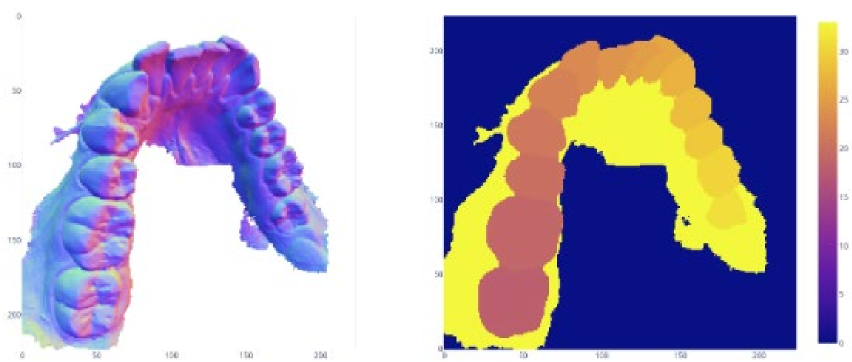

Figure 3. Rendered 2D view of the IOS. Left: surface normal encoded in RGB components, (additional surface properties may be rendered or extracted from the surface using the face-ID maps). Right: segmentation of the dental crowns rendered with a color map.

ALI - IOS (Automatic Landmark Identification in IOS)

Proper placement of the dental crowns is crucial to treatment planning, tooth movement, fabrication of dental restorations, monitoring and maintaining periodontal health, and attaining stable treatment outcomes and occlusal function. IOS are 3D surface models of the upper and lower dentition used to accurately evaluate the clinical crown position without exposing the patient to radiation. Given that, intraoral scanning and digitization of tooth geometries is a fundamental step in dental digital workflow, the accuracy of measurements in IOS is a must. Experts need to segment each tooth and annotate the corresponding anatomical landmarks to plan and assess crowns' position and/or movement for restorative, orthodontics movements, and/or implant dentistry. Manually performing these tasks is time-consuming and prone to inconsistency. There is a clinical need to develop fully automatic methods instead of manual operation. Facing this challenge, we developed a new algorithm for Automatic Landmark Identification on IntraOral Scans (ALLIOS), which combines image processing, image segmentation, and machine learning approaches to automatically and accurately identify commonly used landmarks on IntraOral Scans. More than 150 digital dental models were pre-processed by clinicians to manually annotate the landmarks on each dental crown.

The AI was then trained using the pre-labeled data. A virtual camera moved around each crown of the dental model taking pictures at various locations. The network (U-Net) was then trained to estimate the landmark position in each image. The final predicted landmark position was an average of the location on each picture (Figure 4). Our results showed an average distance error of 0.29 ± 0.23 mm between the prediction and the clinician's landmarks.

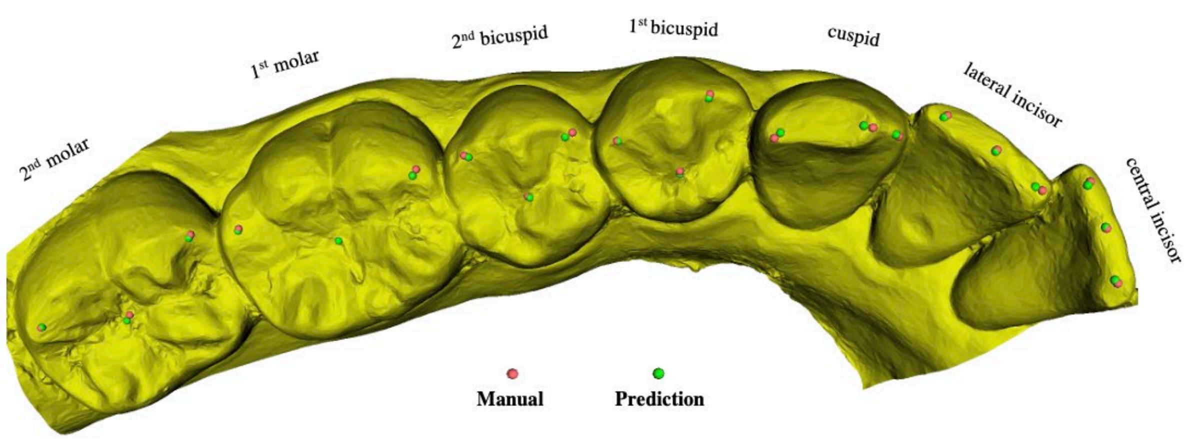

Figure 4. Comparison between manual landmarks and predicted landmarks on different teeth. The red spheres represent the clinician's landmarks (manual) and the green spheres were predicted by ALLIOS.

CONCLUSIONS

Machine learning tools such as ALI-CBCT and FiboSeg allow fast segmentation with high accuracy and robustness on CBCT images and IOS with heterogeneous acquisition parameters. We imagine that in future projects, other structures could be added to the segmentation and a larger dataset will allow better generalization of the application for our tool to work with other types of scans such as magnetic resonance imaging.

The landmarks detected by ALI-CBCT and ALI-IOS will be used to initialize two registration methods, voxel or surface-based registration, and registration of IOS and CBCT scans. They may also increase the efficiency and accuracy of quantitative assessment in clinical practice without the need for human interactions/annotations.

Given the high robustness and time performance, the developed tools are being implemented in an open-source web-based clinical decision support system, the Data Storage for Computation and Integration (DSCI) [17], and in user-friendly 3D Slicer modules (Figure 5). The computer-aided diagnostic tools will aid in therapy planning, and provide a step towards the implementation of dentistry decision support systems, as machine learning techniques are becoming important to automatically analyze dental images.

Machine learning tools are still in their early implementation in orthodontics, and current research on ML raises important questions regarding interpretability and dataset sample reliability. Therefore, better collaboration between orthodontic experts and ML engineers is urged to achieve a positive symbiosis between AI and clinician experts.

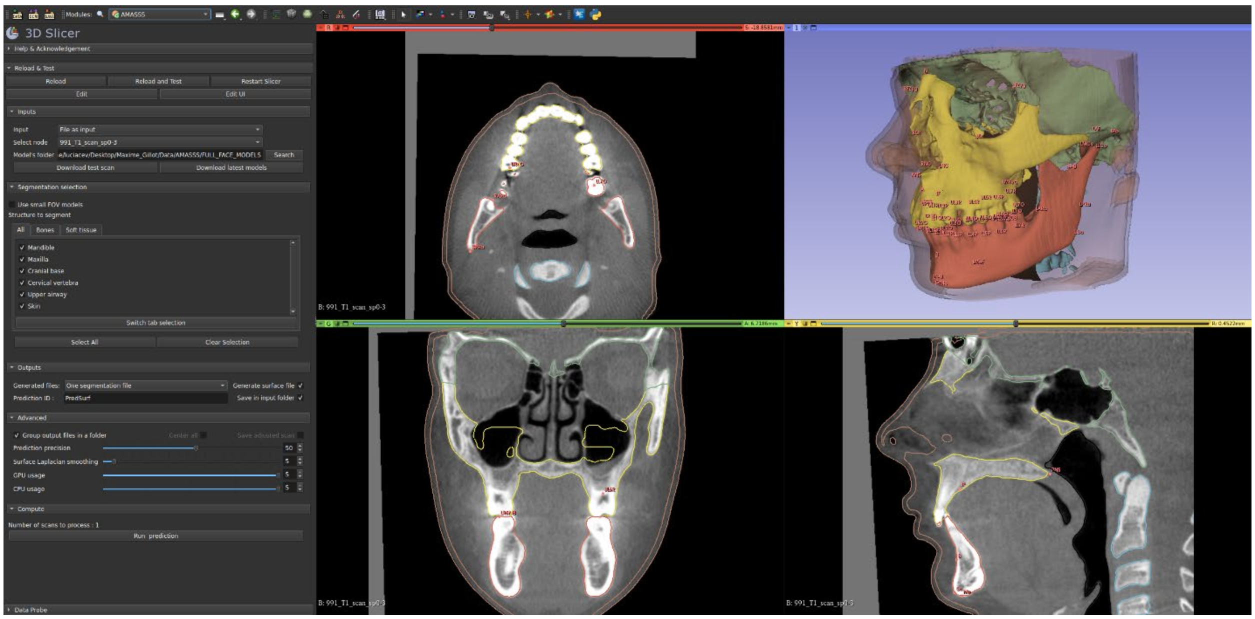

Figure 5. 3D Slicer AMASSS module on the left panel, with a visualization of ALI-CBCT/AMASSS result on the right.

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REFERENCES

1. Chaudhry, S., Dhawan, S. (2020). AI-Based Recommendation System for Social Networking. In: Pant, M., Sharma, T., Verma, O., Singla, R., Sikander, A. (eds) Soft Computing: Theories and Applications. Advances in Intelligent Systems and Computing, vol 1053. Springer, Singapore. https://doi.org/10.1007/978-981-15-0751-9_58
2. Hoyer, WD, Kroschke, M, Schmitt, B, Kraume, K, & Shankar, V. Transforming the customer experience through new technologies. Journal of Interactive Marketing 2020, 51(1), 57–71.
3. Liakos KG, Busato P, Moshou D, Pearson S, Bochtis D. Machine learning in agriculture: a review. Sensors (Basel) 2018; 18(8):2674.

4. Bachute MR, and Subhedar JM. Autonomous driving architectures: insights of machine learning and deep learning algorithms. Machine Learning With Applications 2021; 6100164.
5. Jumper J et al. AlphaFold 2. London, England and Seoul, South Korea: Fourteenth Critical Assessment of Techniques for Protein Structure Prediction; 2020.
6. Heo L, Feig M. Modeling of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) proteins by machine learning and physics-based refinement. bioRxiv [Preprint] 2020; Mar 28:2020.03.25.008904.
7. Yushkevich PA, Yang G, Gerig G: ITK-SNAP: An interactive tool for semi-automatic segmentation of multi-modality biomedical images. Annu Int Conf IEEE Eng Med Biol Soc 2016, 2016:3342-3345.
8. Pieper S, Halle M, Kikinis R. 3D Slicer. Arlington, Virginia: 2004 2nd IEEE International Symposium on Biomedical Imaging: Nano to Macro (IEEE Cat No. 04EX821); 2004; 1:632-635.
9. Yegnanarayana B. Artificial neural networks. India: Prentice-Hall of India Pvt. Ltd; 2009.
10. Dale R. GPT-3: What's it good for? Natural Language Engineering 2021; 27,113-118.
11. Zhang J. Basic neural units of the brain: neurons, synapses and action potential. arXiv [Preprint] 2019;1906.01703.
12. Ayodele TO. Types of machine learning algorithms. In: New Advances in Machine Learning. London, England: IntechOpen; 2010.
13. Song I, Kim H, Barom Jeon P. Deep learning for real-time robust facial expression recognition on a smartphone. Las Vegas, Nevada: 2014 IEEE International Conference on Consumer Electronics (ICCE). IEEE 2014, 564-567.
14. Monamo P, Marivate V, Twala B, "Unsupervised learning for robust Bitcoin fraud detection," 2016 Information Security for South Africa (ISSA), Johannesburg, South Africa, 2016, 129-134.
15. Chen, J. (2016). The Evolution of Computing: AlphaGo. Computing in Science & Engineering. 18(4): 4-7.
16. Boston Dynamics, accessed Feb 03 2023, https://www.youtube.com/watch?v=fn3KWM1kuAw.
17. Brosset S, Dumont M, Cevidanes L, Soroushmehr R, Bianchi J, Gurgel M, Deleat-Besson R, Le C, Ruellas A, Yatabe M et al. Web infrastructure for data management, storage and computation. Proc SPIE Int Soc Opt Eng 2021 Feb;11600:116001N.

3D PRINTING APPLICATIONS FOR THE ORTHODONTIC OFFICE

Tarek Elshebiny, Luciane Macedo de Menezes, Juan Martin Palomo

ABSTRACT

The practice of orthodontics has been revolutionized to adapt to the digital world. Recent developments in three dimensional (3D) digital models, digital intraoral scanners, and 3D printing are at the forefront of these technological advances. 3D printing technology is now being used by orthodontists for multiple applications. The purpose of this chapter is to describe how clinicians can use 3D printers in their practices for different applications such as retainers, aligners, indirect bonding trays, and study models.

KEY WORDS: 3D Printing, 3D Virtual Planning, In-house Aligners

INTRODUCTION

3D printing, also referred to as additive manufacturing or rapid prototyping, is performed by layers being sequentially deposited to build an object. 3D printing is known by many experts to be the next industrial revolution and is expected to one day supersede many current techniques in fabrication and manufacturing. 3D printing has exponentially advanced since the advent of 3D printing in 1983 by Charles W. Hull [1]. In 1986, Hull co-founded the 3D Systems company (Rock Hill, South Carolina, US) to advertise the first machine for rapid prototyping. This was the first 3D printing company in the world. By 1987, the first stereolithography printer was commercialized. With that, the company also commercialized the "Standard Tessellation Language" or "Standard Triangular Language" (STL) file format that permits computer-aided design (CAD) software data to be translated for 3D printers. Tessellation is a term used for breaking the geometry of a surface into a series of small triangles, or other polygons. The STL file format describes the surface of an object as a triangular mesh, that is, as a representation of a 3D surface in triangular facets. Without the STL file, 3D printing would not be possible.

TYPES OF 3D PRINTING TECHNOLOGIES

Stereolithography

The first form of 3D printing was done by a stereolithography apparatus (SLA) [2]. The SLA consists of a bath of photosensitive resin, model building platform, and ultraviolet (UV) laser for curing the resin (Figure 1). The fabrication method is with the photopolymer resin being struck by the pattern of UV light which solidifies and attaches to what came before it. After a layer of resin is cured, the part would then drop down slightly below the surface in a bath of liquid resin, which in turn keeps each printed layer the same distance from the light source. This process repeats, so that a new thin layer of liquid resin is cured and added to the final build.

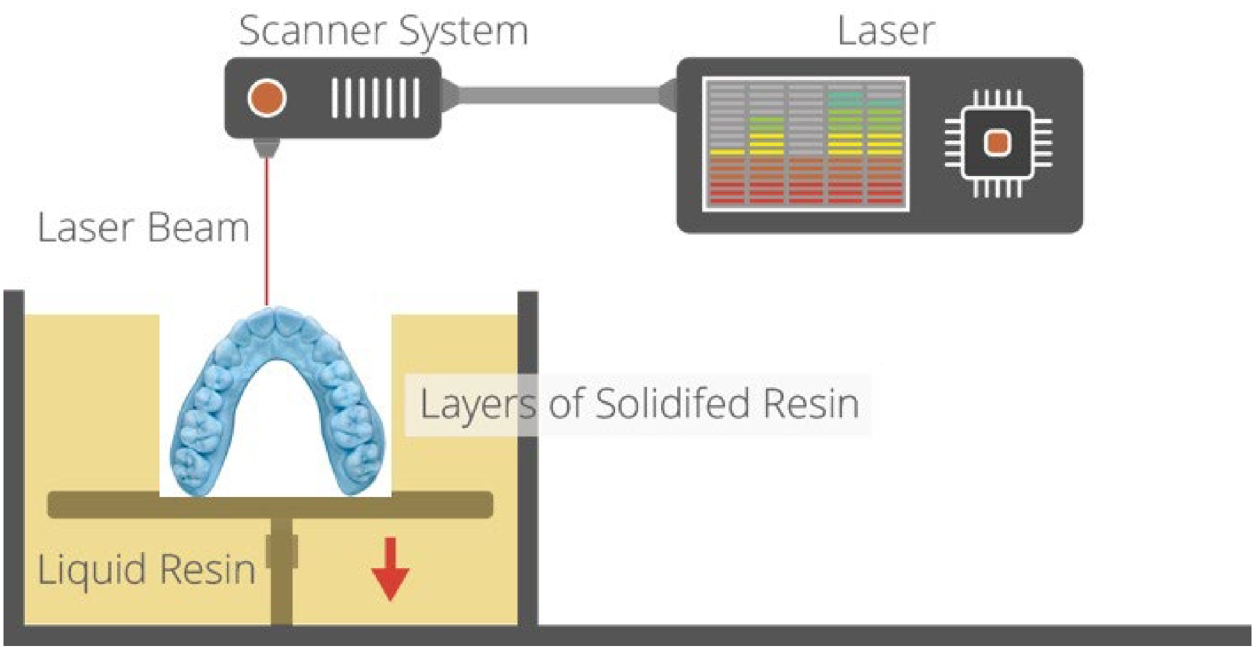

Figure 1. Stereolithography (SLA) technology with a laser for curing the resin at a single point each time.

Fused Deposition Modeling

In 1989, Scott Crump invented Fused Deposition Modeling (FDM) and commercialized it in 1990 with printers from Stratasys company (Rehovot, Israel). FDM is the 3D printing method that most people using the technology are familiar with today. The FDM method uses STL files from 3D models created in a modeling program and the printer's interfacing software converts the file and slices the model into sections as well as determining how the layers are printed. After this is done, the model is sent to the 3D printer, and the build material is extruded through a heated nozzle layer by layer until the part is complete. This type of 3D printing is the most commonly used today and is usually referred to as "desktop 3D printers".

Polyjet printing technology

Objet Geometries company (Rehovot, Israel) founded polyjet printing technology. Polyjet printing uses jet heads that spray or jet the resin into the desired areas. As the jet heads make subsequent passes, each sprayed layer is cured using a UV light source. A key element of the inkjet-based 3D printing process is the print head that sprays layers of photosensitive polymer, which precisely represent the cross-sectional profile of the model on the build platform.

Digital Light Processing

In 2012, B9Creations company (Rapid City, South Dakota, US) developed a 3D printer that used Digital Light Processing (DLP) technology (Figure 2). This introduced the era of laser- and DLP-based SLA printing techniques. DLP is the newest and has the best finish quality compared with the previous three printing styles mentioned above [3]. DLP-based 3D printing technology comes from the image projection technology developed by Texas Instruments (Dallas, Texas, US) in the 1980s [4]. This method uses a set of chipsets based on optical micro-electromechanical technology to process working light sources to photosensitive materials. The main functional part is a digital micro mirror device which consists of a group of micron-sized, controllable mirrors. The mirrors rotate to control the path of light and then project it onto the photosensitive resin [5]. The projection of an image that is composed of small square pixels called “voxels” passes through an optical lens and cures the photo-curable polymer resin [6]. DLP printers can produce very intricate designs, are usually faster than their counterparts and are cost effective. This method of 3D printing uses a process called photopolymerization, meaning it will build an object by using a light source to change a liquid resin to a solid. It uses the STL file as a map to direct the light and create the object. While some printers use a light source such as a laser to cure a single point (SLA method), DLP uses a light board to cure an entire layer at once [7]. Studies have shown that DLP 3D printing can produce dental models with high reproducibility and a high degree of agreement when compared to models fabricated using alginate and stone. Investigators have determined that 3D printing can produce clinically acceptable models which are considered to be viable options for clinical applications [8].

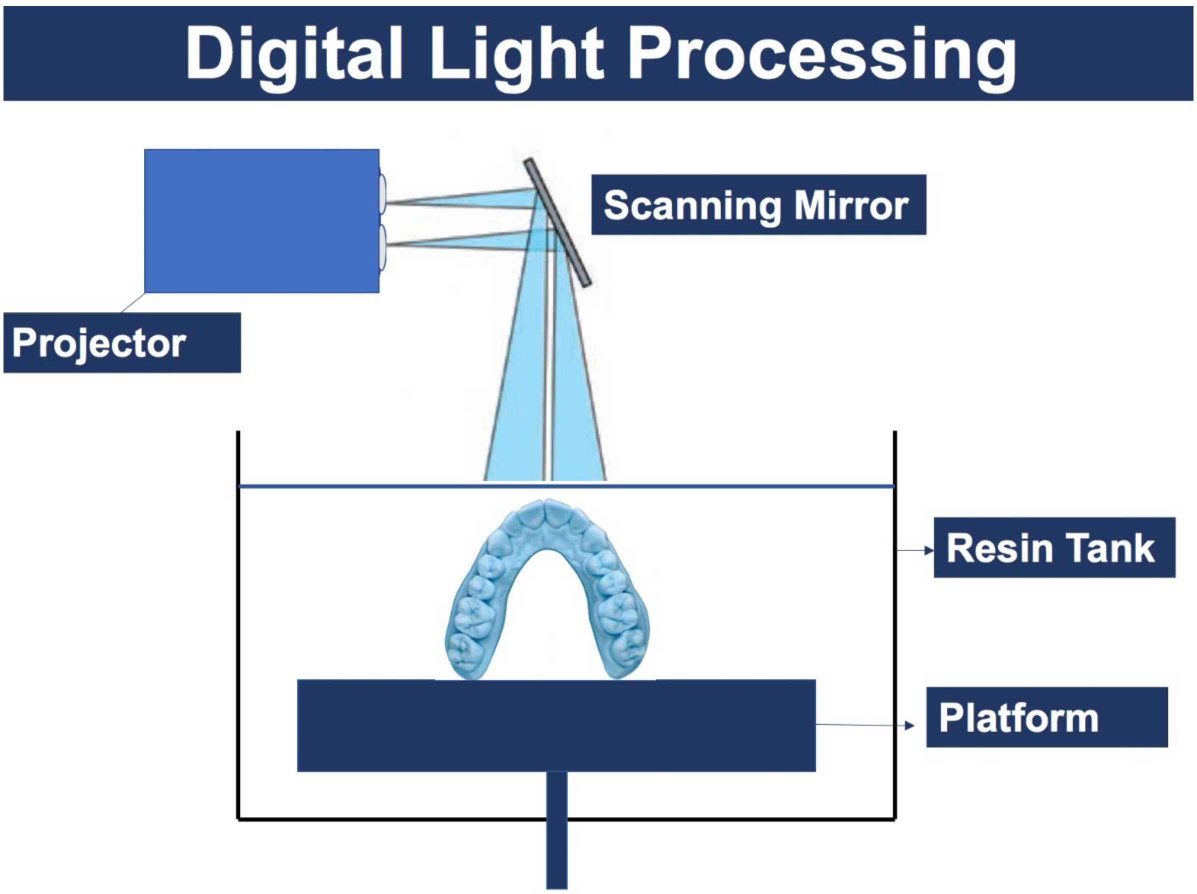

Figure 2. Digital Light Processing (DLP) technology using a light board to cure an entire resin layer at once.

PRINTING LAYER THICKNESS

The layer thickness, or print layer height, in turn influences how many layers it will take to complete an object as well as the duration of the print job. 3D printers have settings for how thick each layer should be. The print layer height ranges from 20-100 µm, and the smaller the print layer height, the more layers are required to print an object, thus making the print job longer in duration. While one may think a smaller print height and more layers allows for a higher degree of detail, and this is true, there are other considerations that need to be considered when evaluating the accuracy of the build. Besides the longer print job duration, with each layer that is printed there is a possibility of an error in the curing, which can either cause a distortion in the model or a complete failure in the printing.

In 2018, Favero et al. investigated how close similar builds at varying print heights were to the original STL files [9]. Using a single master STL file of a dental model, the investigators printed models at print layer heights of 25, 50 and 100 µm using 4 different commercially available printers. All the models were then rescanned using an optical scanner and the respective STL was then superimposed onto the master STL to determine the amount of deviation. The results of the study showed statistically significant deviations at each layer height with the models printed, with 25 µm layer height having the greatest amount of deviation, while the models printed at 100 µm layer height had the least deviation. The investigators also found differences in accuracy between each printer. While finding these differences, the authors concluded that all printed models fell within clinically acceptable limits. They advised, “The print layer height and printer model can affect the accuracy of a 3D printed orthodontic model, but the impact should be considered with respect to the clinical tolerances associated with the envisioned application.” In 2021, Ko et al. generally confirmed these results with a similar study comparing the STL from the scan of 3D printed models of varying print layer heights to the initial STL and the printed model [10]. They likewise found that the smallest print height, 20 µm in this study, produced the least accurate model. Ko and colleagues concluded most models were within clinical acceptable limits and that the choice of layer height should be determined by the application, desired print time, and preferred efficiency of each print job.

In 2019, Loflin et al. aimed to compare 3D printed models of varying print layer heights using an objective assessment method developed by the American Board of Orthodontics (ABO) [11]. They scanned 12 sets of stone models that had previously been submitted and passed the ABO clinical examination and then printed each set at 25, 50, 100 µm print layer heights and subjected each to the same ABO grading system as the original stone models. They found that print layer height did not have a statistically significant effect on the grading of these models in any category, or the score. The printed models were all positively correlated with the stone models for the total score with the strongest correlation found with models printed at the 100 µm print layer height. The authors determined that all layer heights may be acceptable for ABO objective assessment, diagnosis, and treatment planning. They also recommended 100 µm height models due to the decreased printing time.

3D PRINTING USES IN ORTHODONTICS

With more advanced technology becoming available, printing has become easier. It has been predicted that 3D printing will become the future of orthodontics and play a major role in many offices for various applications [12]. Currently, 3D printing plays more of a major role in the orthodontic specialty than it does in the general dentistry sector. From 2015 through 2022, 3D printing has exponentially grown in orthodontics. This has changed how many orthodontists structure their offices and perform tooth movement. In orthodontics, 3D printing is commonly performed using DLP, SLA, or material jetting technology. Although these are the most popular ones used, there are other ways applications are 3D printed.

A common use for 3D printing in orthodontics is to help create retainers following active orthodontic treatment. It is imperative to hold the teeth to prevent relapse. Many authors have agreed that long-term immovability is closely connected to the type of removable retention appliance [13-18]. The Essix clear retainer was created by Dr. John Sheridan in 1993. It was coined the first true clear retainer. Its name came from an acronym for S-Six or Sheridan's Simple System for Stabilizing the Social Six [19]. The traditional way of fabricating a clear retainer is to use an alginate impression and stone model. The model is used to create a vacuum formed retainer with thermoplastic sheets. With the new CAD/CAM software, this alginate impression and stone cast step can be eliminated. Now, retainers can be fabricated from 3D printed models [20]. These retainers are just as reliable as conventional vacuum formed aligners.

Another very popular application for 3D printing in orthodontics is removable clear aligner therapy. Clear aligner therapy has increased tremendously over the past 10 years. Moving teeth without brackets and wires is a concept that started in the 1940s. In 1945, an orthodontist named Harold Kesling announced his "tooth positioning appliance". The device was invented to be used as a finishing appliance after comprehensive orthodontic treatment with fixed appliances. This device could achieve minor root parallelism in extraction cases, establish proper axial inclination and interdigitation, and even de-rotate teeth. The device was intended to be delivered as soon as the patient was de-banded. The patient had to wear the appliance for four hours during the day and while they slept. According to Kesling, the device, if worn properly, would complete its results in a month's time [21]. Even though Kesling was the first to create such a device, two students from Stanford University would get the credit for inventing removable clear aligner therapy. In 1997, Zia Chishti and Kelsey Worth invented the world's first complete clear aligner system. They called it Invisalign, and it conceptualized a series of clear plastic aligners that could move teeth of adults discreetly. The company that blossomed out of this creation was called Align Technology (Tempe, Arizona, US). Align started producing and selling Invisalign branded aligners in 1999. When Invisalign launched their campaign for 3D aligner therapy, this brought attention to 3D printing and the advantages of the technology in orthodontics. They were the first company to initiate the use of CAD/CAM software for tooth movement [18]. Invisalign® was the first system [22, 23] and Align Technology is currently the largest provider of custom-made clear aligners for orthodontic tooth movement [18]. Invisalign uses biocompatible polyurethane thermoformed sheets and uses 3D System's SLA 3D printers for model fabrication [24].

Additionally, indirect bonding technique has benefited from 3D printing technology. The indirect bonding technique was first developed by Silverman and Cohen to heighten patient comfort and reduce chair time [25, 26]. The original method for indirect bonding was performed by using cement for attaching brackets to the stone model, sealant as a clinical adhesive and thermoplastic trays for the transfer of the brackets from the model to the patient. In 1979, the "custom composite base technique" was invented, which is still the most widely accepted technique used today [26, 27]. Over the years, the same technique has been modified to increase bond strength and to decrease bond failures. One modification was mixing the universal and catalyst resins outside of the mouth and directly applying it to the bracket. This enhanced bond strength and made it comparable to direct bonding [28, 29]. The next hallmark enhancement came in 1990, when light-cured resins were used for indirect bonding [25]. However, as technology skyrocketed and CAD entered the realm of orthodontics, this caused an incredible enhancement to the indirect bonding technique. Several companies started offering 3D CAD/CAM created techniques for the manufacturing of indirect bonding trays. Companies such as OraMetrix (Richardson, Texas, US) and Ormco (Brea, California, US) developed technologies such as SureSmile [30] and Insignia [31, 32] and fabricated these types of 3D printed bonding trays. The fabrication method first uses an intraoral scanner to scan the teeth to create a 3D image. That image is then used for a digital set-up and the brackets are placed in the ideal region on each tooth (Figure 3). Finally, the customized transfer trays for indirect bonding are created and a customized archwire is assembled [33]. One of the main reasons that indirect bonding trays have become popular is because CAD eliminates the time needed to manually place the bracket on each tooth [33]. It also reduces chair time up to 30 minutes in comparison with the conventional way of bonding brackets [33].

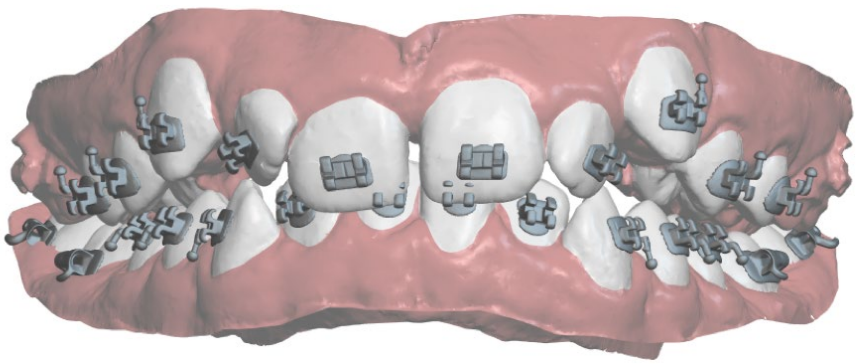

Figure 3. Digital set-up with the brackets placed on each tooth.

There are several other applications that 3D printing can be used for in orthodontics. Some orthodontists have integrated this technology to fabricate orthodontic expanders. Others use it to make functional appliances. Metal appliances can be 3D printed using selective laser sintering (SLS). This is a tremendous advantage because it reduces time in the lab to make nearly impossible appliances. It also eliminates the need to place separators in patients' mouths in preparation for banding. The 3D printed metal "bands" overlay the teeth, removing the need for interproximal space between teeth. The most common 3D printed appliance is the Hyrax rapid palatal expander. Even though these printed appliances cost more to fabricate, it can save chair time, reduce patient's discomfort, and decrease the number of appointments for the patient [34]. Mouth guards and occlusal splints can also be made with 3D printing technology. Temporary anchorage device (TAD) templates or guides, when 3D printed, can assist with the accuracy of TAD placement.

A recent application for 3D printing is the manufacturing of the orthodontic bracket. During the last 100 years, orthodontic brackets have advanced from basic tooth attachments to exceedingly precise fixed appliances with pre-adjusted prescriptions [35-37]. Now, brackets can be tailored and personalized to specifically fit each patient's malocclusion and needs. This reduces the time spent bending wires to make precise movements of teeth. The first 3D printed bracket was created by a company named Light-Force Orthodontics (Burlington, Massachusetts, US). The company was founded in 2015 by Alfred Griffin III. He hypothesized that 3D printing technology would be able to fabricate patient specific brackets. This dream came to fruition when the first bracket was developed and manufactured using polycrystalline alumina in the form of a twin bracket with idealized geometries that was customized and patient specific.

COMMERCIALLY AVAILABLE 3D PRINTERS USED IN ORTHODONTICS

3D printing has infiltrated the orthodontic field by storm. More and more orthodontic applications are being fabricated using this technology. In the past, 3D printers were large, bulky, and very expensive. Today, printers are dramatically reduced in size and pricing. Now, there are 3D printers that are used in orthodontic offices and some that are used strictly in orthodontic laboratories.

Numerous companies have developed their own printers. EnvisionTEC (Gladbeck, Germany) introduced its 3D printer in 2002. It was founded by Al Siblani. This company produced the first digital light processing (DLP) 3D printer, which is the most popular method for printing in dentistry. A popular in-house 3D printer is the Formlabs (Somerville, Massachusetts, US) printer. It was founded in 2011 by three MIT Media Lab students. This was the very first desktop-sized printer that was also very affordable and easy to use. This 3D printer used the stereolithography technology whereby liquid resin is cured into a solid material by the application of laser light. Another 3D printer that is used in orthodontics is the SprintRay (Los Angeles, California, US). It was founded in 2014 at University of Southern California, Marshall School of Business by Hossein Bassir, Jing Zhang, and Amir Mansouri. It uses the DLP technology. Anycubic (Shenzhen, China) 3D printers were founded in 2015 by two childhood friends, James Ouyang and Lu Ouyang. This is now one of the most popular 3D printer brands worldwide. Like the SprintRay, Anycubic printers use DLP technology to fabricate prints. Another popular brand used in orthodontics is the EPAX 3D (Morrisville, North Carolina, US) printer. Founded in 2016 by William Dai, it uses DLP technology and is also a desktop printer. Additionally, a 3D printer by the name of Juell 3D (Ardmore, Oklahoma, US) was founded in the same year. Like many 3D printers used in orthodontics, it uses DLP technology. This printer is one of the fastest on the market today, capable of producing orthodontic models in under 18 minutes.

COMMERCIALLY AVAILABLE SOFTWARE PACKAGES USED IN ORTHODONTICS FOR IN-HOUSE ALIGNERS

The goal of orthodontic treatment is to improve the malocclusion and to achieve as near perfect occlusion as possible. Orthodontic setup, which was first introduced in 1946 [21], plays an important role in achieving predicted tooth movement. 3D virtual setup has become more popular in orthodontics with the introduction of intraoral scanners and diagnostic software programs. 3D virtual setup can simulate the orthodontic treatment by segmenting each tooth as a separate object and moving each individual tooth to its planned position. Virtual setup has been used to present different treatment options and can be useful when considering extractions, interproximal reduction (IPR), anchorage management, and pre-surgical orthodontics [20]. Virtual tooth movement had a great impact in orthodontics by making clear aligner therapy increasingly available, with digital treatment tools facilitating comprehensive orthodontics for adults and teens, rather than just for minor tooth movement [21]. Align Technology, which introduced Invisalign for orthodontic use, utilizes Clincheck software program, which presents stages of virtual tooth movements to achieve the desired tooth movements.

Recently, various 3D diagnostic software programs have offered a virtual tooth movement feature, which allows orthodontists to produce clear aligners in-house via integration with 3D intraoral scanners and 3D printers. There are several aligner planning software programs, which include, but are not limited to, Archform, uLab, SureSmile®, Ortho Insight 3D®, and OrthoAnalyzer. Archform (San Jose, California, US) was founded in 2017 and is a teeth-aligner software designed to import 3D scanned models, create and purchase fabricated aligners, or use the design for in-office 3D printing. The software detects open intra-oral scans and caps them, uses machine-learning for accurate tooth segmentation, and offers numbered aligners and automated attachments. Amir Abolfathi and Charlie Wen founded uLab Systems (Memphis, Tennessee, US) in 2015. It is an orthodontic aligner movement planning software. Using this software, STL files can automatically be labeled, trimmed, and exported to a 3D printer. The software is compatible with the leading printers and thermoformers. This allows orthodontists to fabricate same day aligners and start treatment effectively and efficiently. SureSmile® technology was developed by OraMetrix (Richardson, Texas, US), a company that was founded in 1998 by Dr. Rohit Sachdeva and Friedrich Riemeier. SureSmile® and OraMetrix introduced Elemetrix in 2015. Elemetrix was a newly designed digital treatment planning platform that expanded the available orthodontic treatment options, which included clear aligners. This paved the way for advances in aligner design incorporating control, accuracy, and clinical efficiency. In 2018, Dentsply Sirona (Charlotte, North Carolina, US) acquired OraMetrix. This led to the rebranding of Elemetrix to the name of SureSmile® Ortho and innovations were added to their aligner system, including cut-outs that could use orthodontic auxiliaries to correct mild Class II and Class III occlusion cases. Furthermore, automated pontics and variable trim lines were added to allow for more flexibility and convenience for patients. SureSmile® Ortho also allows for do-it yourself (DIY) aligner staging packages for the orthodontist to create a staged model sequence.

Other commercial software companies include Ortho Insight 3D® created by Motion View Software LLC (Hixson, Tennessee, US) in 2012. The original company designed orthodontic software and was the first to integrate cephalometric tracing predictions with the morphing of digital photographs [38]. Today, the company offers the Ortho Insight 3D®, Facial Insight 3D, and Ortho Share 3D software. Furthermore, it produces a 3D scanner and printer to use in conjunction with this software. Ortho Analyzer® is a product of a Danish company called 3Shape (Copenhagen, Denmark). The company manufactures 3D scanners and CAD/CAM software solutions. 3Shape was founded by Tais Clausen and Nikolaj Deichmann, two graduate students. In 2009, the company introduced its first orthodontic software allowing labs and practices to take full advantage of digital technology. They introduced the TRIOS intraoral scanner in 2011 and became one of the premiere leaders in CAD/CAM for laboratories [39]. 3Shape offers a variety of software solutions such as the Clear Aligner Studio for fabrication of in-house aligners along with Ortho Analyzer software [40].

One study compared the final outcomes of the same amount of tooth movement among four different virtual setup software programs [41]. This was a retrospective study of 32 patients who underwent Invisalign treatment. Table tooth movements were obtained from ClinCheck® Pro software and imported with patients' initial STL files to three different software programs (SureSmile® Aligner, Ortho insight 3D®, and Ortho Analyzer™). After virtually moving teeth based on the numbers from table tooth movements, final STL files were exported from all four software programs. ClinCheck® Pro final STL files were used as references, while final STL files from the other software programs were used as targets. Superimpositions were performed between references and target STL files using Geomagic® Control X™ software (3D Systems - Rock Hill, South Carolina, US) and color-coded maps were obtained to illustrate the differences. It was shown that the differences between absolute averages, averages of positive and negative values for both upper and lower models were significant among all software programs (ClinCheck® Pro, SureSmile® Aligner, Ortho insight 3D® and Ortho Analyzer™), for both upper and lower STL files, the smallest difference was found to be between ClinCheck® Pro and SureSmile® Aligner with median of (0.03, 0.31, -0.19) mm for upper STL files (Abs Avg., +Avg. and -Avg.) and median of (0.02, 0.29, -0.17) mm for lower STL files (Abs Avg., +Avg. and -Avg.), respectively. On the other hand, the biggest difference was found between ClinCheck® Pro and Ortho Analyzer™ with median of (0.05, 0.46, -0.45) mm for upper STL files (Abs Avg., +Avg. and -Avg.) and median of (0.06, 0.48, -0.40) mm for lower STL files (Abs Avg., +Avg. and -Avg.). As a follow up study [42], the same group compared the differences in tooth movements when implementing the same virtual setup on four different software packages: ClinCheck® Pro, Ortho Analyzer®, SureSmile® and Ortho Insight 3D µm. Data from 25 adult patients treated with Invisalign® at Case Western Reserve University's Department of Orthodontics were retrospectively collected. Initial STL files were obtained and imported into three software packages. The teeth were moved to replicate the virtual setup from ClinCheck® Pro. Final outcomes were exported from each software package. ClinCheck® Pro STL files were used as the reference while STL files produced by the other software packages were used as targets. Best fit superimpositions were performed using Geomagic® Control X. Based on the results, tooth position was adjusted in the three software packages until the virtual setups from ClinCheck® Pro were replicated. Once confirmed, the tables containing the tooth movements were compared. The number of aligners and number of attachments automatically generated from each of the software packages were also evaluated. It was shown that extrusion/intrusion and translation buccal/lingual were significantly different among the software packages. ClinCheck® Pro and SureSmile®, SureSmile® and Ortho Insight 3D® ($p \leq 0.014$), SureSmile® and Ortho Analyzer®, and Ortho Insight 3D® and Ortho Analyzer® ($p \leq 0.000$) generated a significantly different number of maxillary aligners. The results varied slightly for mandibular aligners with only ClinCheck® Pro and Ortho Insight 3D® ($p \leq 0.000$), SureSmile® and Ortho Insight 3D®, and Ortho Insight 3D® and Ortho Analyzer® ($p \leq 0.000$) exhibiting a significant difference. ClinCheck® Pro and SureSmile® differed significantly in the number of attachments produced.

EXAMPLES AND ILLUSTRATIONS OF COMMERCIALLY AVAILABLE SOFTWARE SYSTEMS

In this section, the steps involved in fabrication of in-house aligners using two different software programs (Ortho Analyzer™ and SureSmile®) and 3D virtual setups (Ortho Analyzer™ and Ortho Insight 3D) are outlined.

OrthoAnalyzer virtual setup

The first step using Clear Aligner Studio in OrthoAnalyzer software is to prepare the model using the following steps:

1. Alignment of 3D Scan with cone beam computed tomography (CBCT) image, if present. The two files are aligned by placing three points around the first molars and one of the incisors.
2. Setting up both occlusal and sagittal planes to orient the models and define references for different measurements.
3. Segmentation of upper and lower scans, which allows separation of each individual tooth into a separate object to enable tooth movement in 3D directions. Segmentation is performed by virtually drawing lines between mesial and distal surfaces. The software automatically outlines the borders of each individual tooth, defines the gingival margins and the rotation center of each tooth based on the user defined mesial-distal segmentation (Figure 4). Using the CBCT scan to define the teeth axes is highly recommended at this step.

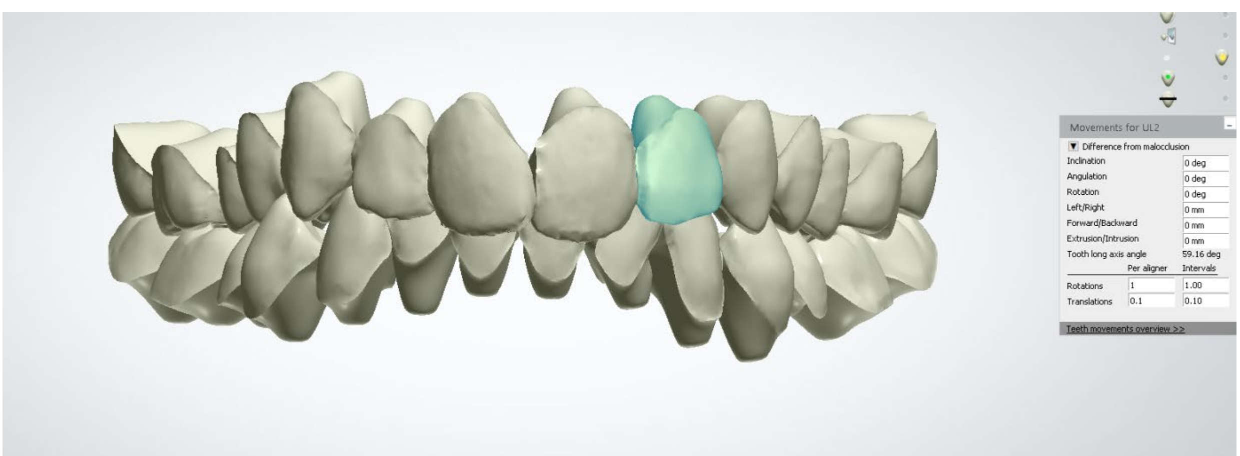

Figure 4. Separation of each tooth allows movement in all 3D directions. The upper left lateral incisor is highlighted with its table of displacements using the OrthoAnalyzer software program.

Once the segmentation step is complete, the software allows the user to move the segmented teeth in 3D to modify inclinations in labial and lingual directions, tip in mesial and distal directions, rotations, bodily movements mesiodistally and buccolingually, and finally, extrusion and intrusion movements. For symmetric movements, the user can select contralateral teeth at the same time to make sure the movements are symmetric bilaterally. Once tooth movement is completed and moved to the desired position, the setup can be viewed with the original position of teeth selected, which recreates a shadow of the selected tooth in its original position. The user can divide the setup to sub setups based on the number of aligners needed for each case. The final step is exporting the sub setups in STL format to be ready for in-house 3D printing.

Automatic model preparation and teeth segmentation software program (SureSmile software)

The SureSmile software for virtual tooth movements is cloud based, which makes it easily accessible to the user. Once the patient profile is created in the software, the user can choose from the options of DIY aligner staging or full-service aligner staging. Once the patient is scanned for treatment, scans are imported to the software as STL files. SureSmile offers a paid service for the model preparation and teeth segmentation. Once this is completed, the user has access to a prepared model with segmented teeth to be able to start the virtual setup for the in-house aligner option.

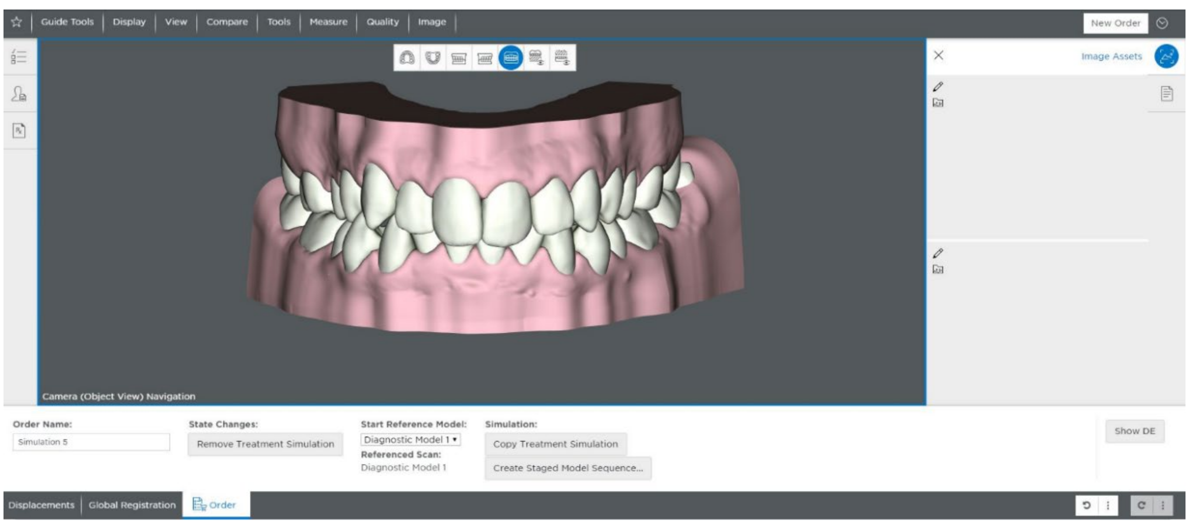

Figure 5. A prepared model for treatment simulation with the SureSmile software program.

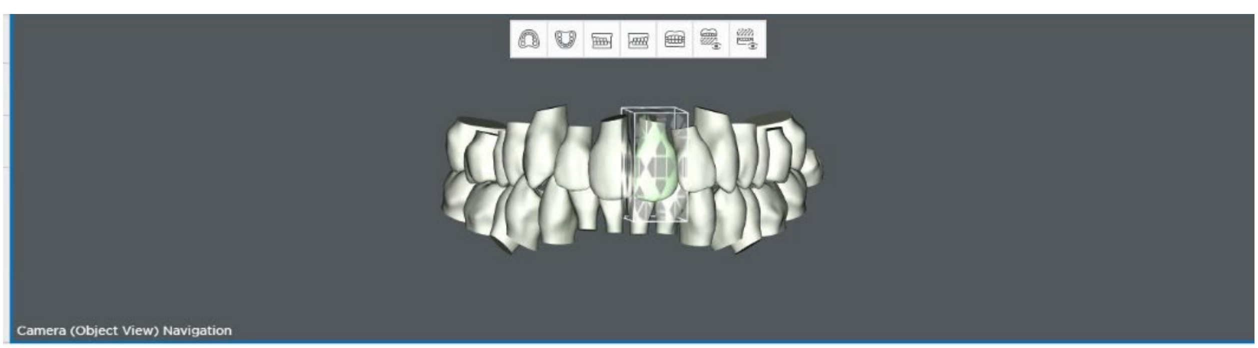

Figure 6. Prepared model with segmented teeth without the gingiva.

SureSmile virtual setup

The software displays the prepared model with segmented teeth. The user can select the option to display only the segmented teeth without the gingiva and the option to select one arch at a time. The software provides a table of displacements, which represents in numbers the amount and direction of movement for each tooth separately. For virtual tooth movement, the user can use the arrows in the displacement table or the virtual box, which appears on the selected tooth with different arrows for different 3D tooth movement. Distal and mesial intersections tabs can be utilized to avoid any overlap with neighboring teeth. Once the setup is completed, the final tooth movements can be viewed with a reference of the original teeth positions. Once the user approves the setup, the software automatically calculates the stages of aligners and automatically places attachments on selected teeth for specific tooth movement. The final step is exporting the staged models in STL format to be ready for in-house 3D printing (Figures 5-9).

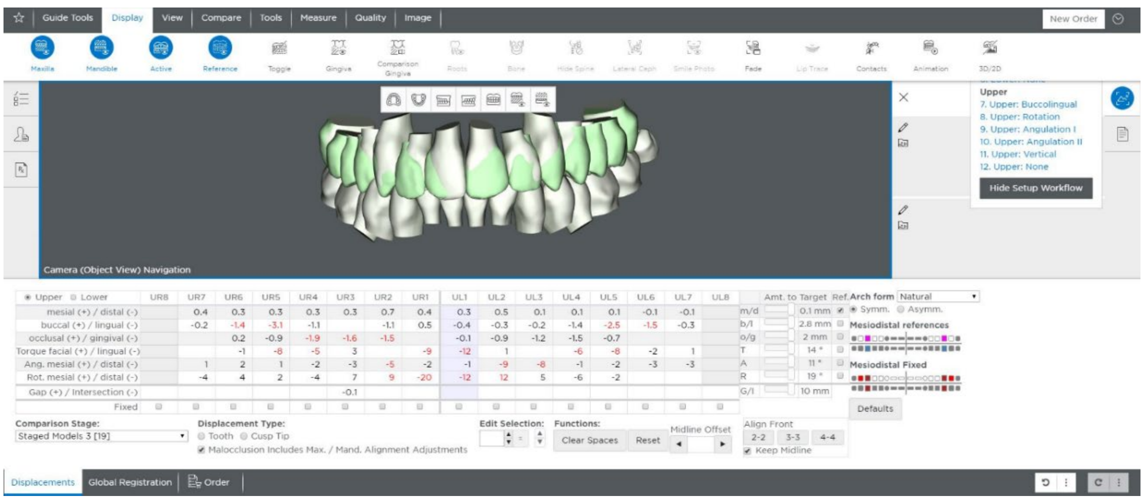

Figure 7. Virtual setup with the table of displacements. The numbers represent the amount and direction of movement for each tooth.

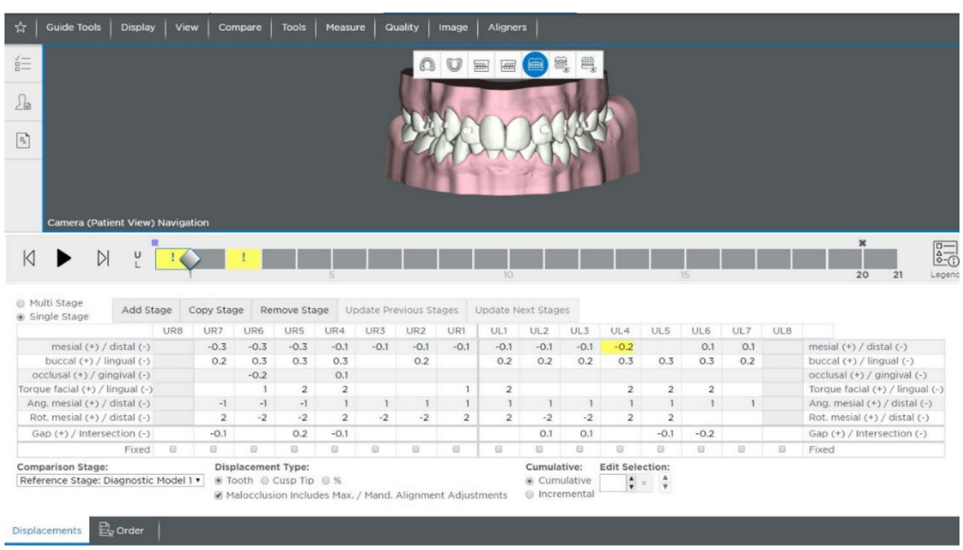

Figure 8. When the setup is completed and approved, the software automatically calculates the stages of aligners and places attachments on selected teeth for specific tooth movement.

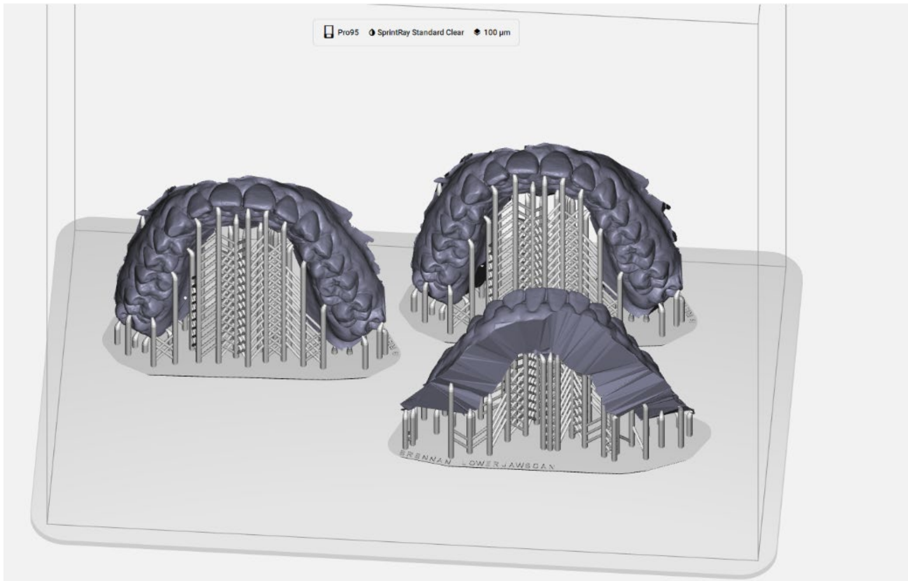

Figure 9. The staged virtual models in STL files are exported and printed.

Steps for 3D virtual indirect bonding using OrthoAnalyzer program

1. Segmentation of the scan file is performed by determining the mesial and distal marginal ridges of the posterior teeth and the mesial and distal incisal edges of the anterior teeth, the software automatically outlines the tooth structures and defines the gingival margins.
2. Digital placement of the brackets: Select the bracket icon to align the axis points along the clinical crown lines.
3. Choose the brackets design from the brackets library. The selected brackets populate on the model. A red dot indicates a bracket that can be moved to the desired position. Confirm the brackets position and save the file.

Figures 10 through 12 demonstrate the same concept of 3D virtual indirect bonding using Ortho Insight 3D software program.

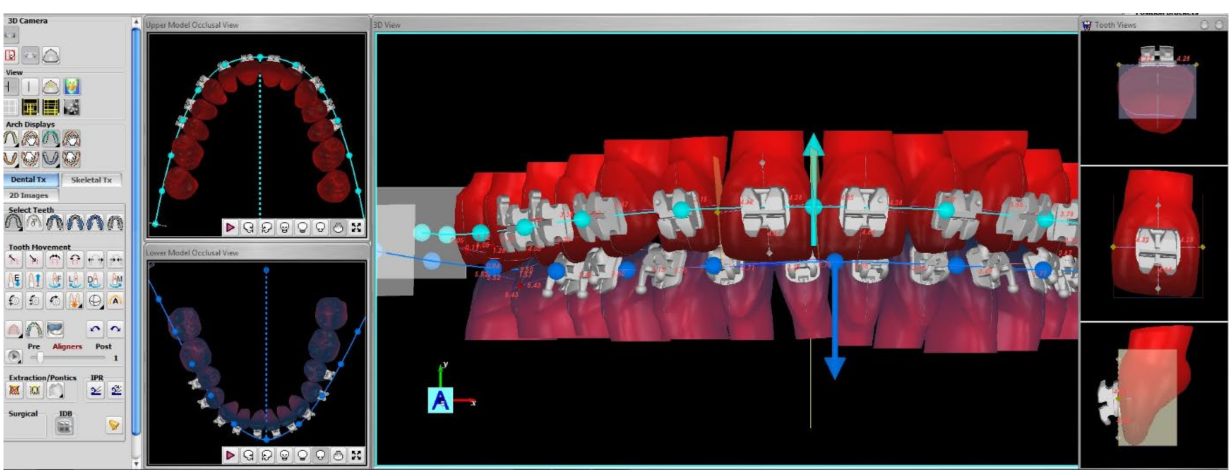

Figure 10. Digital bracket positioning using Ortho Insight 3D software program.

Figure 11. Brackets digitally placed (Ortho Insight 3D software program).

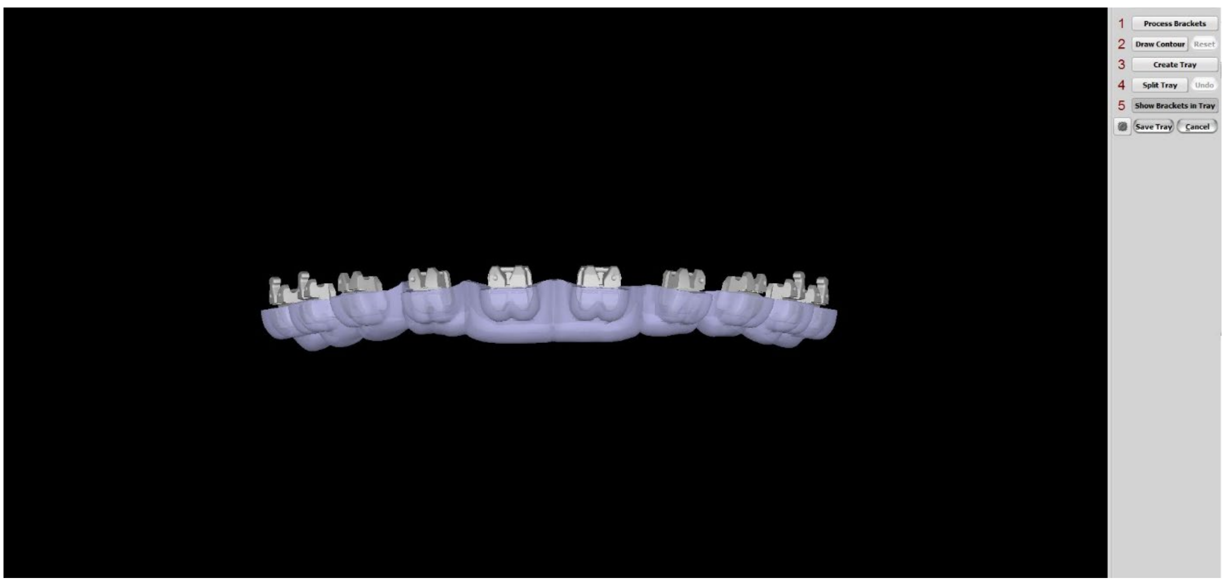

Figure 12. Virtual Tray with brackets in position (Ortho Insight 3D software program).

CONCLUSIONS

Recent advances in digital technology have increased its application in dentistry. 3D printing equipment and products are now part of an orthodontic office. Printed models, aligners, or indirect bonding trays are some of the possible daily uses of these technologies. With more potential applications on the horizon, research of new products and technologies to optimize their use for clinical orthodontics is needed.

REFERENCES

1. Pechter, D. History of 3D Printing – Who Invented the 3D Printer? All3DP. 2018. https://all3dp.com/2/history-of-3d-printing-who-invented-the-3d-printer/.
2. Gonzalez, CM. Infographic: The History of 3D Printing, The American Society of Mechanical Engineers. 2020. https://www.asme.org/topics-resources/content/infographic-the-history-of-3d-printing.
3. Sherman SL, Kadioglu O, Currier GF, Kierl JP, Li J. Accuracy of digital light processing printing of 3-dimensional dental models. Am J Orthod Dentofacial Orthop 2020; 157(3):422-428.
4. Lu Y, Mapili G, Suhali G, Chen S, Roy K. A digital micro-mirror device-based system for the microfabrication of complex, spatially patterned tissue engineering scaffolds. J Biomed Mater Res A. 2006; 77(2):396-405.
5. Zhang J, Hu Q, Wang S, Tao J, Gou M. Digital light processing based 3D printing for medical applications. Int J Bioprint 2020; 6(1):242.
6. Hribar KC, Soman P, Warner J, Chung P, Chen S. Light-assisted direct-write of 3D functional biomaterials. Lab Chip 2014; 14(2):268-275.
7. Snikhovska K. The different types of 3D printing technology. 2020. https://penandplastic.com/3d-printer-types/
8. Brown GB, Currier GF, Kadioglu O, Kierl JP. Accuracy of 3-dimensional printed dental models reconstructed from digital intraoral impressions. Am J Orthod Dentofacial Orthop 2018; 154(5):733-739.
9. Favero CS, English JD, Cozad BE, Wirthlin JO, Short MM, Kasper FK. Effect of print layer height and printer type on the accuracy of 3-dimensional printed orthodontic models. Am J Orthod Dentofacial Orthop 2017; 152(4):557-565.
10. Ko J, Bloomstein RD, Briss D, Holland JN, Morsy HM, Kasper FK, Huang W. Effect of build angle and layer height on the accuracy of 3-dimensional printed dental models. Am J Orthod Dentofacial Orthop 2021; 160(3):451-458.e452.
11. Loflin WA, English JD, Borders C, Harris LM, Moon A, Holland JN, Kasper FK. Effect of print layer height on the assessment of 3D-printed models. Am J Orthod Dentofacial Orthop 2019; 156(2):283-289.
12. Groth C, Kravitz ND, Jones PE, Graham JW, Redmond WR. Three-dimensional printing technology. J Clin Orthod 2014; 48(8):475-485.
13. Rinchuse DJ, Miles PG, Sheridan JJ. Orthodontic retention and stability: a clinical perspective. J Clin Orthod 2007; 41(3):125-132.
14. Sheridan JJ, LeDoux W, McMinn R. Essix retainers: fabrication and supervision for permanent retention. J Clin Orthod 1993; 27(1):37-45.
15. Laboda M. The effect of Essix appliances on anterior open-bite, Thesis, Louisiana State University Department of Orthodontics, 1995.
16. Hilliard K, Sheridan JJ. Adjusting Essix appliances at chairside. J Clin Orthod 2000; 34(4):236–238.
17. Nasef A, El-Beialy A, Eid F, Mostafa Y. Accuracy of orthodontic 3D printed retainers versus thermoformed retainers. Open Journal of Medical Imaging 2017; 7(4):169-179.
18. Kuroda T, Motohashi N, Tominaga R, Iwata K. Three-dimensional dental cast analyzing system using laser scanning. Am J Orthod Dentofacial Orthop. 1996; 110(4):365-369.
19. Galan-Lopez L, Barcia-Gonzalez J, Plasencia E. A systematic review of the accuracy and efficiency of dental movements with Invisalign®(R). Korean J Orthod 2019; 49(3):140-149.

20. Allison Williams, Sompop Bencharit, Il-Hyung Yang, Spiro Christos Stilianoudakis, Caroline K. Carrico, Eser Tüfekçi. Effect of print angulation on the accuracy and precision of 3D-printed orthodontic retainer. Am J Orthod Dentofacial Orthop 2022; 161(1):133-139.
21. Kesling HD. Coordinating the predetermined pattern and tooth positioner with conventional treatment. Am J Orthod Oral Surg 1946; 32(5):285-293.
22. Ojima K, Kau CH. A perspective in accelerated orthodontics with aligner treatment. Seminars in Orthodontics 2017; 23(1):76-82.
23. Boyd RL, Miller R, Vlaskalic V. The Invisalign® system in adult orthodontics: mild crowding and space closure cases. J Clin Orthod 2000; 34(4):203-212.
24. Haria R. How 3D Printing Has Changed Dentistry, A Billion Dollar Opportunity. 2017. https://3dprintingindustry.com/news/3d-printing-impact-on-dentistry-121284/
25. Pamukçu H, Özsoy ÖP. Indirect Bonding Revisited. Turk J Orthod 2016; 29(3):80-86.
26. Silverman E, Cohen M, Gianelly AA, Dietz VS. A universal direct bonding system for both metal and plastic brackets. Am J Orthod 1972; 62(3):236–244.
27. Thomas RG. Indirect bonding: simplicity in action. J Clin Orthod 1979; 13(2):93-106.
28. Miles PG. A comparison of retention rates of brackets with thermally-cured and light-cured custom bases in indirect bonding procedures. Aust Orthod J 2000; 16(2):115-117.
29. Read MJ, O'Brien KD. A clinical trial of an indirect bonding technique with a visible light-cured adhesive. Am J Orthod Dentofacial Orthop 1990; 98(3):259-262.
30. Sachdeva R, Frugé JF, Frugé AM, Ingraham R, Petty WD, Bielik KL, et al. SureSmile: a report of clinical findings. J Clin Orthod 2005; 39(5):297–314.
31. Gracco A, Tracey S. The insignia system of customized orthodontics. J Clin Orthod 2011; 45(8):442-451.
32. Kalange JT. Ideal appliance placement with APC brackets and indirect bonding. J Clin Orthod 1999; 33(9):516-526.
33. Guenthner TA, Larson BE. Indirect Bonding: A technique for precision and efficiency. Semin Orthod 2007; 13(1):58–63.
34. Shannon T, Groth C. Be your own manufacturer: 3D printing intraoral appliances. Semin Orthod 2021; 27(3):184-188.
35. Vaden L. A century of the edgewise appliance. APOS Trends Orthod, 2015; 5:239-249.
36. Andrews LF. The straight-wire appliance. Br J Orthod 1979, 6(3):125-143.
37. Feldman A. Meet the Latest Unicorn: Formlabs, A 3-D Printing Startup Founded by Three Young MIT Grads" Forbes. 2018. https://www.forbes.com/sites/amyfeldman/2018/08/01/3-d-printing-startup-formlabs-venture-capital-unicorn-1-billion-valuation-financing-jeff-immelt-forbes-30-under-30-alumni-max-lobovsky/?sh=2e58341e7471
38. Lund B. You Should Know: Motion View Software, LLC. In. Orthotown. 2013. https://www.orthotown.com/magazine/article/4374/you-should-know-motion-view-software-llc
39. 3shape. Our Story. 2020. https://www.3shape.com/en/our-story.
40. Gierie, WV. Clear aligner therapy: an overview. J Clin Orthod 2018; 52(12):665-674.
41. Eleliwi M, Elshebiny T, Stefanovic N, Palomo JM. Comparing virtual setup software programs for clear aligner treatment. Thesis Project. Case Western Reserve University; 2020.
42. Dhingra A, Palomo JM, Stefanovic N, Elshebiny T. Comparing 3D tooth movement when implementing the same virtual setup on different software packages. J Clin Med. 2022; 11(18):5351.

MANDIBULAR ASYMMETRY: DIAGNOSIS, PREVALENCE, AND ORTHOPEDIC TREATMENT

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ABSTRACT

Facial symmetry plays a major role in esthetically pleasing facial features by balancing size and proportions of craniofacial structures and the smile. Asymmetric features can affect social and psychological aspects of quality of life, which influence the decision to seek orthodontic and/or orthognathic surgical treatment. In orthodontic patients, the main region responsible for facial asymmetry is the lower third of the face, involving mandibular asymmetry in almost 75% of cases. The complexity of the asymmetric conditions of the mandible must be well defined in the diagnostic steps, in terms of positional and morphological asymmetries. Knowing the appropriate diagnostic methods, morphological features and malocclusion type with mandibular asymmetry are crucial aspects of follow-up in orthodontic patients. Management of positional and morphological asymmetries of the mandible can prevent asymmetry deterioration, minimize the risk of developing facial asymmetry, and solve asymmetric disharmonies in the orthodontic treatment. This chapter presents a discussion on the diagnosis of mandibular asymmetry, the influence of different skeletal anterior-posterior patterns on the prevalence of mandibular asymmetry, and mandibular asymmetry behavior in patients with unilateral posterior crossbite before and after treatment.

KEY WORDS: Asymmetry, Mandible, Diagnosis, Angle´s Malocclusion Classification, Posterior Crossbite

INTRODUCTION

Craniofacial embryogenesis is a complex event in the human development of single and double anatomical structures of the face which results in a proportional symmetry of all components. Perfect symmetry of the human face is considered an ideal concept, and subclinical asymmetry is found in most patients with normal craniofacial development and esthetically pleasing faces [1]. Facial symmetry is defined as a balance in size, shape, and position of bilateral anatomical regions on opposite sides, and coinciding position of individual anatomical parts in relation to the midsagittal plane (MSP) [1]. The absence of such factors and an imbalance between the right and left sides favors facial asymmetry, as well as facial disharmony [1]. Reports on the prevalence of facial asymmetry show a range of 12% to 33.9% in patients presenting for orthodontic treatment [2, 3]. This demands that orthodontists and oral surgeons broaden their knowledge to provide better clinical management.

Obvious facial asymmetries occur mainly in syndromes of the first and second branchial arches [4-6] and in patients with a history of facial trauma [5, 7], tumors, joint ankylosis and infections [5]. In non-syndromic patients with no known etiology, the asymmetric growth of the lower third of the face has shown that the most affected bone is the mandible, reaching rates of 73.7% of asymmetric cases [2]. In terms of the prevalence of mandibular asymmetry in different anterior-posterior patterns, some studies have also shown a higher frequency of patients with Class III malocclusions than in those with Classes I or II [8, 9]. The excessive growth of the mandible in Class III patients is seen as a risk factor for unbalanced development on both sides of the mandible. In view of this predominance in the lower third, an accurate understanding of the location in mandibular regions and amplitude of mandibular asymmetry is essential for purposes of diagnosing facial deformities, as well as for surgical planning [10, 11]. In addition, an understanding of the occlusal consequences of mandibular asymmetry is crucial for orthodontic planning at the different stages of human life, childhood, adolescence, and adulthood.

Asymmetries of the lower third of the face lead to both an unesthetic appearance and establishing asymmetrical malocclusions. Mandibular asymmetry can develop as a result of positional asymmetry and/or skeletal morphological asymmetry [12]. The most common malocclusion related to positional mandibular asymmetry is unilateral posterior crossbite. Unilateral crossbite can occur from the early stages of life through to adulthood, which means from deciduous to permanent dentitions [12]. A prevalence rate of up to 22% of this type of malocclusion has been shown in the population [13], and early intervention has been recommended in order to prevent the skeletal asymmetry from deteriorating.

This chapter discusses mandibular asymmetry from different orthodontic perspectives: 1) three-dimensional (3D) diagnosis of mandibular asymmetry, 2) prevalence of mandibular asymmetry in different skeletal anterior-posterior patterns, and 3) mandibular asymmetry in patients with unilateral posterior crossbite before and after treatment.

MANDIBULAR ASYMMETRY DIAGNOSIS

To diagnose mandibular asymmetry, the head must be held in an appropriate position during the clinical examination, and a 3D image analysis is required. It is recommended that the head be held with the Frankfurt horizontal plane perpendicular to the midsagittal plane (MSP). Several methods of quantifying the reference plane in 3D images have been developed [14-18]. In the clinical examination, the glabella and subnasal landmarks are the references for the direction of the MSP by means of the soft tissues. In skeletal image analysis, the MSP is determined through the alignment of the glabella (G), crista galli (CG), and basio (Ba) perpendicular to the components of the 3D Frankfurt, such as the bilateral porion (Po) and the bilateral infraorbitale (Or) [19].

Skeletal mandibular asymmetry is commonly diagnosed by the horizontal deviation of the chin, by identifying cephalometric points, such as the Pogonion (Pog), Menton (Me), and Gnathion (Gn) in the central region of the mandible and measuring the distance between these points and the MSP, and classifying it as mild (<2 mm), moderate (2-4 mm), or severe (>4 mm) [1, 20-22]. Diagnosis of mandibular asymmetry in image analysis is mainly performed using cone-beam computed tomography (CBCT) images [23,24] or anteroposterior cephalometric radiography [23]. Due to the complexity of the mandible morphology, as well as the different planes of space involved in asymmetries, a 3D analysis is preferable [23]. Figure 1 shows CBCT models of different degrees of severity in horizontal mandibular asymmetries.

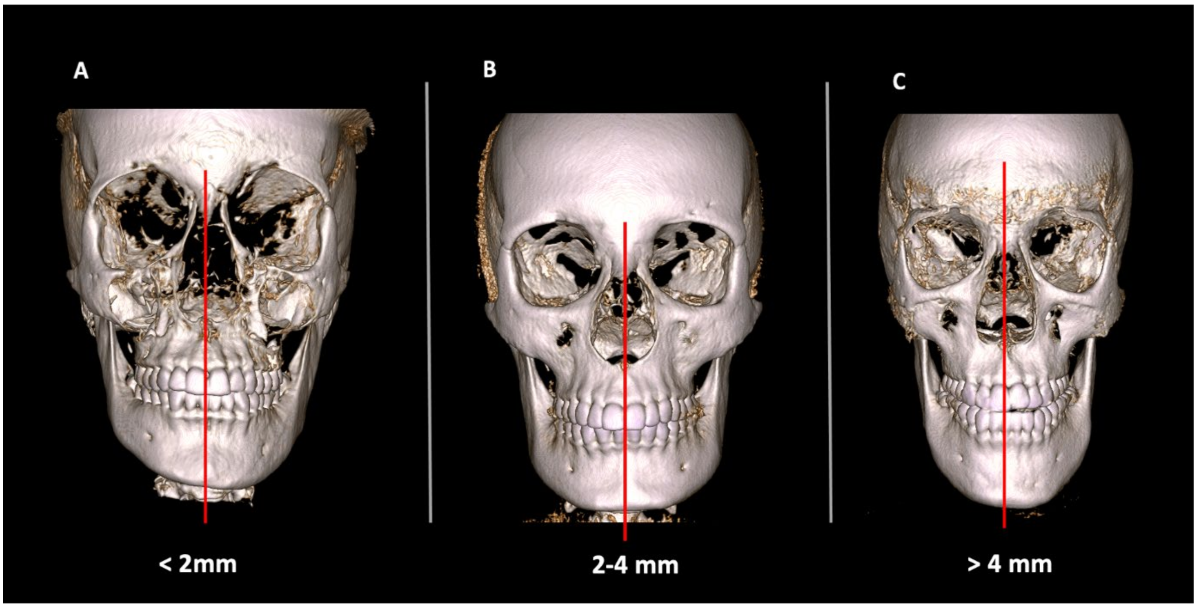

Figure 1. CBCT rendered models of A) relative, B) mild, and C) severe horizontal mandibular asymmetries.

Another approach to diagnosing mandibular asymmetry involves an analysis of its vertical dimensions, measured by the asymmetry index applied in panoramic radiographs for ramus and/or condyle height asymmetries [25]. There is vertical asymmetry when the index values are greater than 3%, which means the proportional difference between both sides. This method has also been applied to the 3D images, such as CBCT scans, with greater accuracy [26]. A proposed classification of mandibular asymmetry in CBCT images [27] referred to location, whether in the ramus, body, and/or chin, noting that the more posterior, the greater the vertical asymmetry. Transverse asymmetry is usually greater in the body of the mandible. In addition, the difference in ramus height and the difference in body length are some of the factors which can cause chin deviation. It is important to note that mandibular asymmetry can be underestimated when evaluated without considering the asymmetry index, as a prevalence rate 2.5 times higher can be found when this analysis is performed [28]. The severity grade of vertical asymmetry is defined by cut-off values to differentiate between different grades (> 3%, > 6%, and > 10%) [28]. Figure 2 shows different cases of asymmetry index.

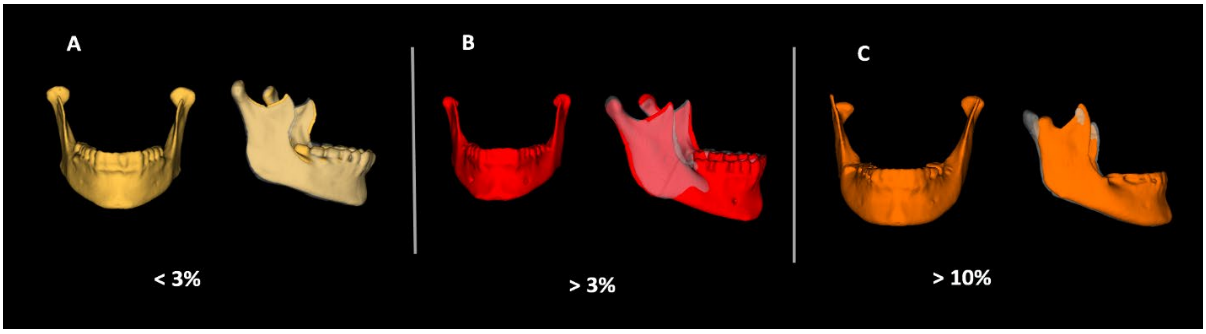

Figure 2. 3D mandible models with anterior and lateral views and respective asymmetry index values. A) Vertical symmetry, B) Vertical asymmetry > 3%, and C) Vertical asymmetry > 10%. The lateral view shows superimposed mirrored models in semitransparency. Note the marked vertical asymmetry of the models in C.

CBCT imaging diagnosis is optimized by different 3D analysis techniques of bilateral craniofacial structures [24, 29], combined with the methodology of segmentation and overlapping of anatomical structures [30]. 3D superimposition of the original models and mirroring are key methods in the analysis of craniofacial and mandibular asymmetries [24,29], which make quantitative and qualitative analyses of specific issues of the mandibular hemiarches possible, as seen in Figure 2. Additionally, the plane can be used to mirror the healthy side of the mandible in cases of asymmetric deformities [14]. However, standardization of the reference region for registration is essential in the analysis of asymmetry, thereby allowing for the access of regional and positional asymmetries.

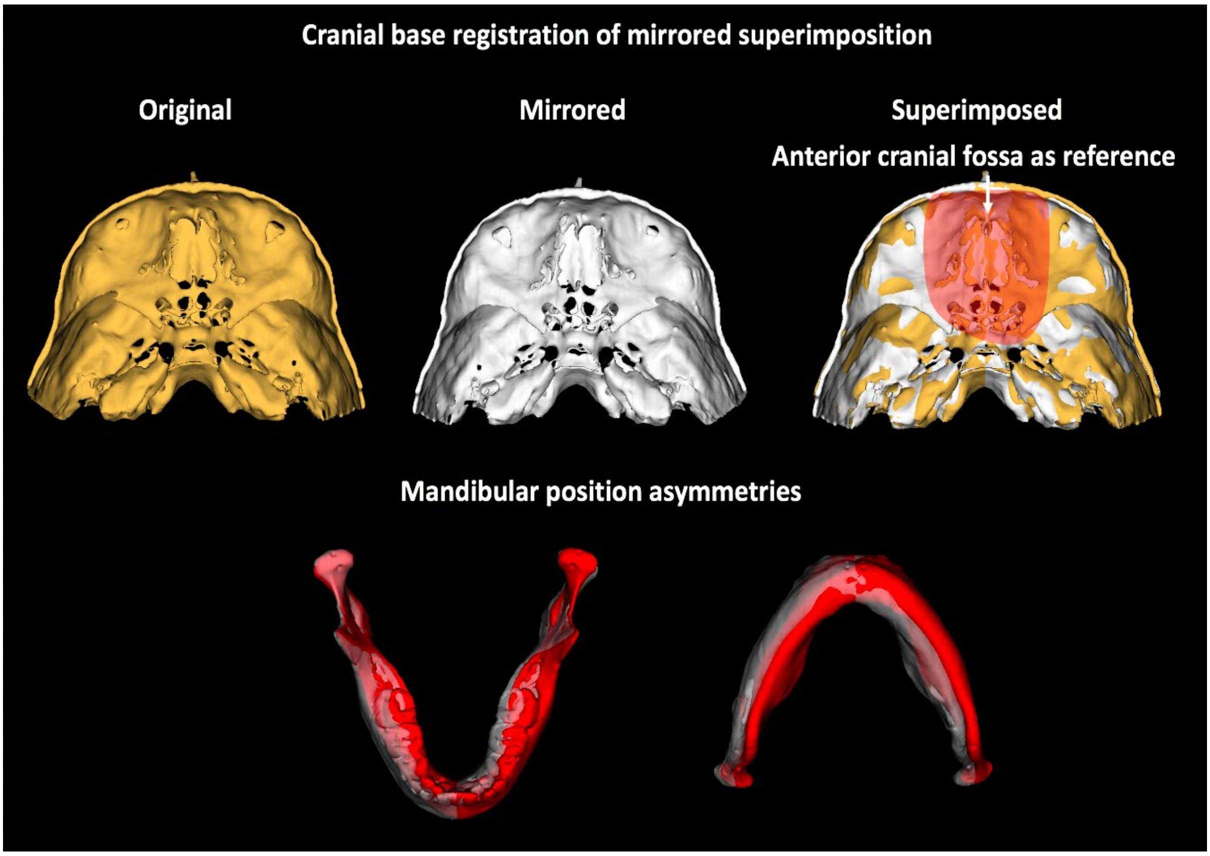

Figure 3. 3D models with upper view of original cranial base (yellow) and mirrored model (white) superimposed with anterior cranial fossa as reference (delimited in light red) to detect the mandibular position asymmetry, as seen with mirrored mandible model in semitransparency on original mandible model (red). Note the lateral position of the original model to the left side.

Positional asymmetry diagnosis using mirroring methodology consists of mirroring the tomographic scans and registering the superimposition of the original and mirrored images with the anterior cranial fossa as reference and then providing information on the mandibular asymmetry position in terms of the face [29, 31] (Figure 3). This mirroring method functions best when applied in cases of mandibular asymmetry, but with a symmetrical skull base. The superimposition applied to the skull base is, therefore, intended to investigate positional mandibular asymmetry [12]. The regional asymmetry diagnosis differs in methodology from the positional diagnosis as it uses another region to register the superimposed images. The morphological asymmetry of the mandible uses the mandibular base as a reference for registration. In this method, the differences in the vertical dimensions of the ramus and condyle between both sides of the mandible, and the horizontal mandibular discrepancies are detected [12, 29, 31] (Figure 4). Both overlay methods are accurate, reliable, and reproducible. In addition, the images can be seen three-dimensionally, and also separately in the sagittal, axial, and coronal views [29].

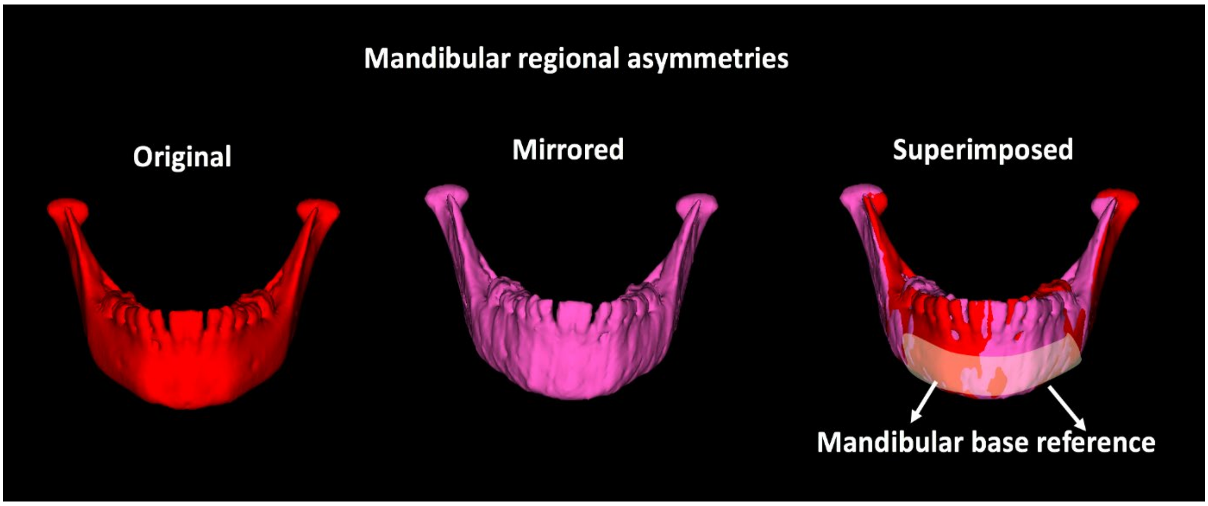

Figure 4. 3D models with anterior view of original mandible (red) and mirrored model (pink) superimposed with mandibular base as reference (delimited in light pink) to detect mandibular regional asymmetries. Note the more lateral inclination of the left condyle through the red color.

PREVALENCE OF MANDIBULAR ASYMMETRY AND SKELETAL ANTERIOR-POSTERIOR PATTERNS

The mandibular asymmetry features in different skeletal anterior-posterior patterns are fundamental items of information for orthodontic and orthognathic surgical treatment planning. In fact, few studies present information on this topic, and there is still a need for more epidemiological studies using populational-based samples to provide stronger evidence, as well as studies with comparison data. A systematic review recently aimed to identify the prevalence in Classes I, II, and III skeletal malocclusions. The results of the five studies which were included for quality analysis, presented valuable information for each pattern [9]. The prevalence of mandibular asymmetry in skeletal malocclusions can be identified separately in different directions, such as horizontal and vertical dimensions. In terms of horizontal asymmetry of the mandible, the chin deviation to the MSP has the highest prevalence in Class III patients (22.9% - 78%), while Class II has the lowest (10% - 25.5%). Class I showed an intermediate prevalence, with rates of 17.7% to 55.6%. Interestingly, there was a considerable prevalence of vertical asymmetry in Classes I, II, and III malocclusions, with rates of 69.0%, 71.7%, and 80.4%, respectively [9]. Even though vertical asymmetry showed high frequency in all skeletal anterior-posterior patterns, Class III malocclusion was the most prevalent, especially in an asymmetry index over 10% [9].

The different prevalence rates in each skeletal anterior-posterior pattern open up a discussion about the impact of mandibular asymmetries associated to different malocclusions. Horizontal deviation of the mandible seems to be more related to greater growth potential of the mandible. In other words, the excessive and/or normal growth of the mandible could influence the higher prevalence rates of mandibular asymmetry in Classes I and III patients than in patients with lower growth potential, such as the Class II skeletal pattern. Class III skeletal malocclusions could, thus, be considered a risk factor for the disharmonious development of the mandible in terms of symmetry [9, 20]. On the other hand, the results of the asymmetry index found differences in ramus height, with expressive prevalence in all anterior-posterior patterns [28] and indicate that vertical asymmetries could be a common feature in all skeletal anterior-posterior patterns or even a common characteristic of the human face. This information should be treated with caution, since only one study includes this methodological approach. Further studies with large population-based samples could help to clarify this issue.

Another perspective concerning vertical asymmetries of the mandible has to do with the fact that even though high prevalence rates are a common feature in all skeletal anterior-posterior patterns, their clinical impact on facial asymmetry should be better understood. The upper region of the face, such as the maxilla, zygoma, and glenoid fossa of the temporal bone, can camouflage asymmetric morphological aspects of the mandible [32]. There is still a lack of information on analysis of the facial asymmetry which distinguishes the upper, middle, and lower third features for each antero-posterior skeletal pattern. Further studies investigating the contribution of the different vertical facial thirds could provide information on how the different regions can interact to mask or promote mandibular asymmetries.

MANDIBULAR ASYMMETRY AND POSTERIOR CROSSBITE

Posterior crossbite, a negative transverse discrepancy between maxilla and mandible, can be bilateral or unilateral [33]. Posterior crossbite commonly occurs as early as deciduous and mixed dentitions, with prevalence rates ranging from 7.5% to 22% [13, 34]. The prevalence of functional crossbite in deciduous dentition is around 8.4% and decreases to 7.2% in mixed dentition [34]. The unilateral is the most common type with a frequency of around 67% to 97% of cases, with a functional shift of the mandible toward the crossbite side [34, 35].

Unilateral posterior crossbite is an asymmetric malocclusion, which can have any combination of dental, skeletal, and neuromuscular functional components, but its most frequent cause is a reduction in the width of the maxillary dental arch [36]. Dental etiology involves incorrect tooth positioning in conjunction with adequate palatal width, such as an excessive lingual inclination of posterior upper teeth [33]. Skeletal etiology involves a narrowed maxilla or, less often, a larger transverse dimension of the mandible [33, 36]. Transverse maxillary deficiency can be induced by non-nutritive sucking habits, certain swallowing habits, or obstruction of the upper airways caused by adenoid tissues or nasal allergies [36]. The major common non-nutritive sucking habits are using a pacifier and/or finger sucking [37]. The tongue, retained in a low position by pacifier or finger, could be prevented from applying pressure against the palate needed for transverse maxillary arch growth [38].

Transverse maxillary deficiency can lead to incorrect mandible function in occlusal contact, and consequently to functional posterior unilateral crossbite [34]. The mandible may have to move to the lateral side to allow molars and premolars/deciduous molars a more comfortable occlusal position. A mandibular shift, known as functional crossbite, then occurs [34]. This functional shift is diagnosed when there is a difference between the lower midline dental position in maximum intercuspation and in the centric relationship position. This condition clearly reflects a horizontal deviation of the mandibular position (Figure 5).

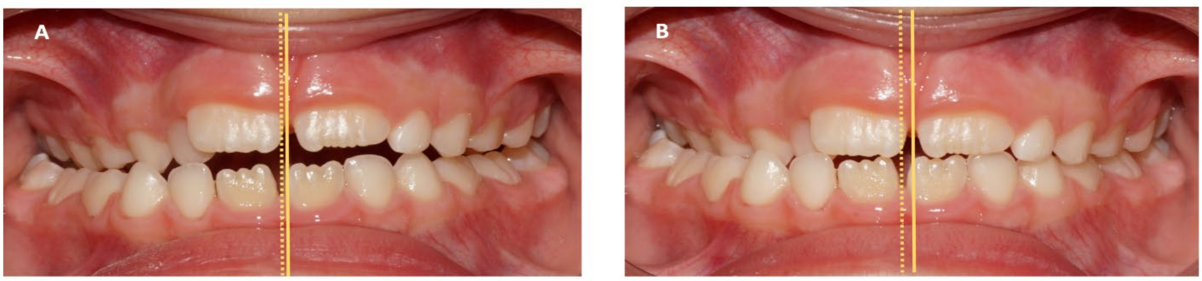

Figure 5. Intraoral photographs of the mandibular functional shift of an 8-year-old patient with unilateral posterior crossbite. A) Centric relationship and B) Maximum intercuspation. Note the widened distance of the upper and lower midlines in B.

Unilateral posterior crossbite and characteristics at different ages

The characteristics of unilateral posterior crossbite can vary according to etiology and dentition (deciduous, mixed, permanent) and age group (children, adolescents, adults) and consequently lead to different implications of craniofacial growth and development. It should be mentioned that there can be a multifactorial etiology, involving dental, and/or skeletal, and/or functional etiologies. Although the mandibular position is rotated in horizontal plane (yaw rotation), minor skeletal mandibular asymmetries are present at early ages [12]. As a patient with functional mandibular shift continues to grow, a definitive lateral mandibular position is established as a response of skeletal growth to the yaw rotation. Thus, the differences between maximum intercuspation and the centric relationship position seen in functional shift disappears, and the skeletal growth stimulates a stable but asymmetric mandibular position, which suggests that condyles and glenoid fossa in older patients could have undergone an adaptive repositioning [39, 40].

Comparisons of morphological characteristics between different age groups when there is unilateral posterior crossbite are scarce. Evangelista et al. recently added new information about this topic in a cross-sectional design study using mirrored overlapped models in three different age groups. Their image analysis distinguished the contributory components of the morphological asymmetries and the positional asymmetry by comparing children, adolescents, and adults. The results showed that morphological asymmetries, described as regional asymmetries, were greater in adult patients and located in latero-medial condylar width (0.7 mm), total ramus height (2.0 mm) and mandibular length (1.5 mm). An interesting perspective is that the differences detected were small in clinical terms, but the adult patients may not have sought treatment earlier because their asymmetries could have been milder when they were younger.

In terms of asymmetry direction in patients with unilateral posterior crossbite, it should be highlighted that horizontal asymmetry, assessed by yaw rotation of the mandible, seems to be a stable position, with an average 2.0^{o} of yaw rotation. In agreement with these values, horizontal chin deviation also showed similar magnitude between children, adolescents, and adults. The temporomandibular joint in the functional unilateral posterior crossbite in children presents the condyles in an asymmetric position in the articular fossa. Recent studies have suggested that, during craniofacial growth, remodeling of the temporomandibular joints (condyles and fossas) tends to adapt to the mandibular deviation. Such adaptive remodeling could explain why adolescents or adults did not present greater mandibular rotation or lateral deviation [12, 41], as seen in Figure 6. In terms of the vertical dimension, the differences in total ramus height between both sides showed that children present smaller differences (0.9 mm) than adults (2.0 mm). Those results imply that a compensation of vertical dimensions could be a mechanism to respond to the horizontal position in children.

The hypothesis that the mandibular position in patients with unilateral posterior crossbite could be further deviated in adult patients has not been confirmed. Children, adolescents, and adults presented a similar mandibular yaw rotation in relation to cranial base. This result indicates that there must be natural adaptive compensation of unilateral crossbite during growth and development, but such compensation does not seem to progress throughout the entire growth period. Moreover, mandibular morphological asymmetry is an adaptation associated with postural adjustments characterized by the crossbite side of the mandible being shorter than the non-crossbite side, thereby indicating a non-crossbite side dominance [12, 41].

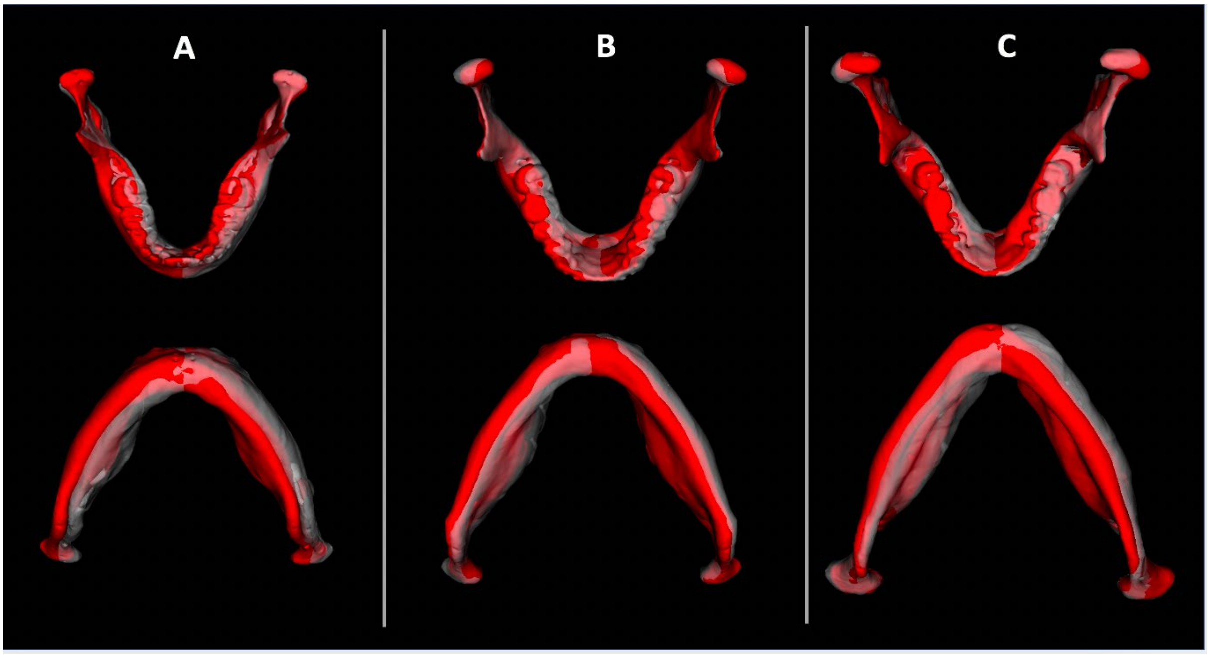

Figure 6. 3D superimposition registered in cranial base to detect the mandibular position with original (red) and mirrored (semitransparent) models in A) children, B) adolescents, and C) adult patients with unilateral posterior crossbite. Note the similar mandibular rotation in patients through the semitransparency.

Mandibular asymmetry after unilateral crossbite treatment with rapid maxillary expansion

The treatment of unilateral posterior crossbite depends on the diagnosis of the main etiology and affected tissues, or in other words, the dentoalveolar or skeletal origins. Genetic and environmental factors, such as persistent oral habits and oral breathing must be considered in the treatment of skeletal etiology of unilateral posterior crossbite, because of the presence of narrower maxilla or transverse maxillary deficiency [42, 43]. The occlusal features vary widely depending on facial type, sagittal skeletal pattern, and prolonged persistence of etiological factors. In cases of transverse maxillary deficiency, rapid maxillary expansion (RME) is a well-known orthopedic treatment approach and is the first choice in the orthodontic management of unilateral posterior crossbite. RME is also considered effective in promoting oral and generalized health [44], increasing the transversal maxillary distances [45]. The positive effects of RME on mandibular asymmetry have been reported in children with functional unilateral posterior crossbite, which mean correction of the mandibular shift [35].

The effects of RME on positional and regional asymmetries of the mandible after one year have shown how beneficial this orthopedic procedure is in patients with unilateral posterior crossbite considering mandibular asymmetry [32]. The mandibular asymmetry assessed before treatment showed statistically significant differences between the right and left sides, which meant greater mandibular horizontal rotation and lateral hemimandibular asymmetry in children with unilateral posterior crossbite when compared to children without crossbite, but there was no difference in the position of the glenoid fossa. After RME, the effects on mandibular asymmetry could be seen clearly in mandibular rotation, which reduced from 1.84° to 0.54° in means. Another significant effect could be seen in the lateral hemimandibular asymmetry angle, which decreased the difference between the two sides from 2.67° to 0.95°. In such circumstances, RME is recommended for children with unilateral posterior crossbite, to guide the correct development of morphology and position the mandible. Figure 7 shows a child patient with unilateral posterior crossbite before and after one year of RME.

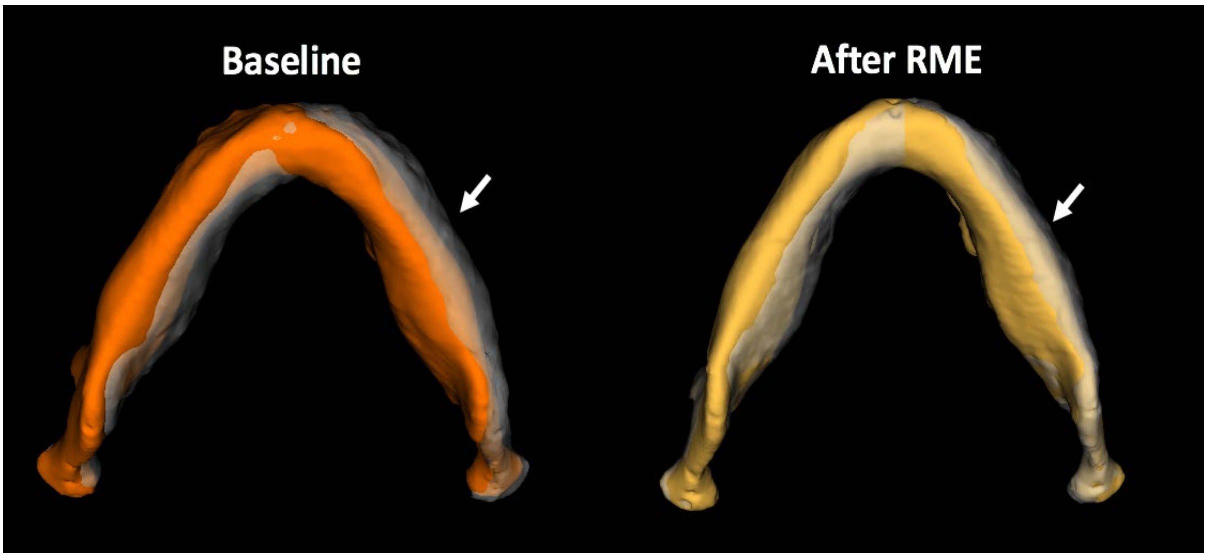

Figure 7. 3D superimposition registered in cranial base to detect the mandibular position with original orange (Baseline) and yellow (After) RME and mirrored (semitransparent) models of a 7-year-old patient with unilateral posterior crossbite. The white arrows show the reduction in mandibular rotation through the semitransparency after RME treatment.

CONCLUSIONS

Diagnosis of mandibular asymmetry plays a substantial role in orthodontic and orthognathic surgical treatment. A correct diagnosis, considering horizontal and vertical dimensions and grades of severity, guides appropriate treatment planning in terms of the skeletal anterior-posterior pattern, especially in Class III skeletal malocclusions and the early treatment of unilateral posterior crossbite. Knowing the factors that contribute to mandibular asymmetry could change treatment planning, prognosis, and early interventions to bring about symmetric development of the mandible.

REFERENCES

1. Peck S, Peck L, Kataja M. Skeletal asymmetry in esthetically pleasing faces. Angle Orthod 1991; 61(1):43-48.
2. Severt TR, Proffit WR. The prevalence of facial asymmetry in the dentofacial deformities population at the University of North Carolina. Int J Adult Orthodon Orthognath Surg 1997; 12(3):171-176.
3. Sheats RD, McGorray SP, Musmar Q, Wheeler TT, King GJ. Prevalence of orthodontic asymmetries. Semin Orthod 1998; 4(3):138-145.
4. Cohen MM, Jr. Asymmetry: molecular, biologic, embryopathic, and clinical perspectives. Am J Med Genet 2001; 101(4):292-314.
5. Cohen MM, Jr. Perspectives on craniofacial asymmetry. III. Common and/or well-known causes of asymmetry. Int J Oral Maxillofac Surg 1995; 24(2):127-133.
6. Johnson JM, Moonis G, Green GE, Carmody R, Burbank HN. Syndromes of the first and second branchial arches, part 2: syndromes. AJNR Am J Neuroradiol 2011; 32(2):230-237.
7. Pirttiniemi P, Peltomaki T, Muller L, Luder HU. Abnormal mandibular growth and the condylar cartilage. Eur J Orthod 2009; 31(1):1-11.
8. Qu GL, Xu J, Zhou Q. Evaluation of the spatial position and correlation of mandibular ramus in skeletal Class III patients with mandibular asymmetry. J Craniofac Surg 2019; 30(5):e439-e442.
9. Evangelista K, Teodoro AB, Bianchi J, Cevidanes LHS, de Oliveira Ruellas AC, Silva MAG, Valladares-Neto J. Prevalence of mandibular asymmetry in different skeletal sagittal patterns. Angle Orthod 2022; 92(1):118-126.

10. O'Grady KF, Antonyshyn OM. Facial asymmetry: three-dimensional analysis using laser surface scanning. Plast Reconstr Surg 1999; 104(4):928-937.
11. Thiesen G, Gribel BF, Freitas MPM, Oliver DR, Kim KB. Mandibular asymmetries and associated factors in orthodontic and orthognathic surgery patients. Angle Orthod 2018; 88(5):545-551.
12. Evangelista K, Valladares-Neto J, Garcia Silva MA, Soares Cevidanes LH, de Oliveira Ruellas AC. Three-dimensional assessment of mandibular asymmetry in skeletal Class I and unilateral crossbite malocclusion in 3 different age groups. Am J Orthod Dentofacial Orthop 2020; 158(2):209-220.
13. Krooks L, Pirttiniemi P, Kanavakis G, Lahdesmaki R. Prevalence of malocclusion traits and orthodontic treatment in a Finnish adult population. Acta Odontol Scand 2016; 74(5):362-367.
14. De Momi E, Chapuis J, Pappas I, Ferrigno G, Hallermann W, Schramm A, Caversaccio M. Automatic extraction of the mid-facial plane for cranio-maxillofacial surgery planning. Int J Oral Maxillofac Surg 2006; 35(7):636-642.
15. Gellrich NC, Schramm A, Hammer B, Rojas S, Cufi D, Lagreze W, Schmelzeisen R. Computer-assisted secondary reconstruction of unilateral posttraumatic orbital deformity. Plast Reconstr Surg 2002; 110(6):1417-1429.
16. Hassfeld S, Muhling J. Computer assisted oral and maxillofacial surgery--a review and an assessment of technology. Int J Oral Maxillofac Surg 2001; 30(1):2-13.
17. Junck L, Moen JG, Hutchins GD, Brown MB, Kuhl DE. Correlation methods for the centering, rotation, and alignment of functional brain images. J Nucl Med 1990; 31(7):1220-1226.
18. Liu Y, Collins RT, Rothfus WE. Robust midsagittal plane extraction from normal and pathological 3-D neuroradiology images. IEEE Trans Med Imaging 2001; 20(3):175-192.
19. Ruellas AC, Tonello C, Gomes LR, Yatabe MS, Macron L, Lopinto J, Goncalves JR, Garib Carreira DG, Alonso N, Souki BQ et al. Common 3-dimensional coordinate system for assessment of directional changes. Am J Orthod Dentofacial Orthop 2016; 149(5):645-656.
20. Thiesen G, Gribel BF, Kim KB, Pereira KCR, Freitas MPM. Prevalence and associated factors of mandibular asymmetry in an adult population. J Craniofac Surg 2017; 28(3):e199-e203.
21. Masuoka N, Momoi Y, Ariji Y, Nawa H, Muramatsu A, Goto S, Ariji E. Can cephalometric indices and subjective evaluation be consistent for facial asymmetry? Angle Orthod 2005; 75(4):651-655.
22. Good S, Edler R, Wertheim D, Greenhill D. A computerized photographic assessment of the relationship between skeletal discrepancy and mandibular outline asymmetry. Eur J Orthod 2006; 28(2):97-102.
23. Yousefi F, Rafiei E, Mahdian M, Mollabashi V, Saboonchi SS, Hosseini SM. Comparison efficiency of posteroanterior cephalometry and cone-beam computed tomography in detecting craniofacial asymmetry: a systematic review. Contemp Clin Dent 2019; 10(2):358-371.
24. AlHadidi A, Cevidanes LH, Mol A, Ludlow J, Styner M. Comparison of two methods for quantitative assessment of mandibular asymmetry using cone beam computed tomography image volumes. Dentomaxillofac Radiol 2011; 40(6):351-357.
25. Habets LL, Bezuur JN, Naeiji M, Hansson TL. The Orthopantomogram, an aid in diagnosis of temporomandibular joint problems. II. The vertical symmetry. J Oral Rehabil 1988; 15(5):465-471.
26. Halicioglu K, Celikoglu M, Yavuz I, Sekerci AE, Buyuk SK. An evaluation of condylar and ramal vertical asymmetry in adolescents with unilateral and bilateral posterior crossbite using cone beam computed tomography (CBCT). Aust Orthod J 2014; 30(1):11-18.
27. Leung MY, Leung YY. Three-dimensional evaluation of mandibular asymmetry: a new classification and three-dimensional cephalometric analysis. Int J Oral Maxillofac Surg 2018; 47(8):1043-1051.
28. Mendoza LV, Bellot-Arcis C, Montiel-Company JM, Garcia-Sanz V, Almerich-Silla JM, Paredes-Gallardo V. Linear and volumetric mandibular asymmetries in adult patients with different skeletal classes and vertical patterns: a cone-beam computed tomography study. Sci Rep 2018; 8(1):12319.

29. Cevidanes LH, Alhadidi A, Paniagua B, Styner M, Ludlow J, Mol A, Turvey T, Proffit WR, Rossouw PE. Three-dimensional quantification of mandibular asymmetry through cone-beam computerized tomography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011; 111(6):757-770.
30. Grauer D, Cevidanes LS, Proffit WR. Working with DICOM craniofacial images. Am J Orthod Dentofacial Orthop 2009; 136(3):460-470.
31. Cevidanes LH, Styner MA, Proffit WR. Image analysis and superimposition of 3-dimensional cone-beam computed tomography models. Am J Orthod Dentofacial Orthop 2006; 129(5):611-618.
32. Evangelista K, Ferrari-Piloni C, Barros LAN, Avelino MAG, Helena Soares Cevidanes L, Ruellas ACO, Valladares-Neto J, Silva MAG. Three-dimensional assessment of craniofacial asymmetry in children with transverse maxillary deficiency after rapid maxillary expansion: A prospective study. Orthod Craniofac Res 2020; 23(3):300-312.
33. Proffit WR, Fields Jr H, Larsno B, Sarver DM. Contemporary orthodontics, 6th ed. St. Louis, MO: Mosby; 2019:744p.
34. Caroccia F, Moscagiuri F, Falconio L, Festa F, D'Attilio M. Early orthodontic treatments of unilateral posterior crossbite: a systematic review. J Clin Med 2020; 10(1).
35. Pinto AS, Buschang PH, Throckmorton GS, Chen P. Morphological and positional asymmetries of young children with functional unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 2001; 120(5):513-520.
36. Andrade Ada S, Gameiro GH, Derossi M, Gaviao MB. Posterior crossbite and functional changes. A systematic review. Angle Orthod 2009; 79(2):380-386.
37. Petrén S, Bondemark L, Söderfeldt B. A systematic review concerning early orthodontic treatment of unilateral posterior crossbite. Angle Orthod 2003; 73(5):588-596.
38. Volk J, Kadivec M, Mušič MM, Ovsenik M. Three-dimensional ultrasound diagnostics of tongue posture in children with unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 2010; 138(5):608-612.
39. Langberg BJ, Arai K, Miner RM. Transverse skeletal and dental asymmetry in adults with unilateral lingual posterior crossbite. Am J Orthod Dentofacial Orthop 2005; 127(1):6-15; discussion 15-16.
40. O’Byrn BL, Sadowsky C, Schneider B, BeGole EA. An evaluation of mandibular asymmetry in adults with unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 1995; 107(4):394-400.
41. Talapaneni AK, Nuvvula S. The association between posterior unilateral crossbite and craniomandibular asymmetry: a systematic review. J Orthod 2012; 39(4):279-291.
42. Melsen B, Stensgaard K, Pedersen J. Sucking habits and their influence on swallowing pattern and prevalence of malocclusion. Eur J Orthod 1979; 1(4):271-280.
43. Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. Acta Otolaryngol Suppl 1970; 265:1-132.
44. McNamara JA Jr, Lione R, Franchi L, Angelieri F, Cevidanes LH, Darendeliler MA, Cozza P. The role of rapid maxillary expansion in the promotion of oral and general health. Prog Orthod 2015; 16:33.
45. Kanomi R, Deguchi T, Kakuno E, Takano-Yamamoto T, Roberts WE. CBCT of skeletal changes following rapid maxillary expansion to increase arch-length with a development-dependent bonded or banded appliance. Angle Orthod 2013; 83(5):851-857.

POST-TREATMENT EVALUATION OF OPEN BITE TREATMENT IN THE GROWING PATIENT

Veronica Giuntini, Roberta Lione, James A. McNamara Jr., Paola Cozza, Lorenzo Franchi

ABSTRACT

The aim of this chapter was to evaluate open bite treatment in the growing patient characterized by anterior open bite, with or without persisting digit sucking habits. In the first clinical situation, treatment consisted of controlling the sucking habit, allowing the involved teeth to erupt into a normal position. The use of a quad-helix appliance that incorporated a tongue crib (Q-H/C) led to successful outcomes in 93% of the patients, with a mean closure of the anterior open bite of about 2 mm in comparison to untreated controls 4 years post-treatment. The Q-H/C protocol also produced a clinically significant decrease of intermaxillary divergence (-2.2°), mainly due to a downward rotation of the palatal plane (1.8°). This favorable outcome contributed significantly to the overall correction of the anterior open bite with improvement in vertical skeletal relationships.

In patients with skeletal open bite but without associated sucking habits, an alternative treatment protocol was used. This protocol consisted of rapid maxillary expansion (RME) combined with a removable mandibular bite-block (RMBB) that was effective in correcting negative overbite (+1.8 mm) in 100% of the patients. In addition, the vertical eruption of the maxillary and mandibular molars was less than that in untreated open bite subjects (-3.3 mm). In comparison to controls, an improvement in the vertical skeletal dimension was observed, as indicated by a reduction in FMA of -2.8°. Thus, the results of this clinical study indicated that early treatment with RME and RMBB remained stable 4 years after the completion of treatment.

KEY WORDS: Open Bite, Sucking Habits, Quad-Helix with Crib, Rapid Maxillary Expansion, Posterior Bite Blocks

INTRODUCTION

Anterior open bite is a malocclusion characterized by a deficiency in the normal vertical overlap between antagonist incisal edges when the posterior teeth are in occlusion. The prevalence of anterior open bite ranges from 1.5% to 16.5% among different ages and ethnic groups [1-4]. In younger children, anterior open bite can be caused by a single factor or a combination of factors such as sucking habits, enlarged tonsils or adenoids, tongue position, constricted maxilla, and skeletal open bite growth pattern.

In non-nutritive sucking patients, whose malocclusion is primarily dentoalveolar in nature, treatment consists of both removing the etiologic factor and controlling the habit, allowing the teeth to erupt into a normal position. A palatal crib often is used to promote normal development of the anterior segment [5-7].

On the contrary, in skeletal open bite malocclusion, patients typically display backward and downward rotation of the mandible, increased vertical growth of posterior dentoalveolar structures, increased lower anterior facial height, and a narrow maxillary arch. Several authors have emphasized that a skeletal open bite should be treated in the mixed dentition to take advantage of active growth by expanding the maxillary arch and preventing further vertical growth of the upper and lower posterior dentoalveolar regions [5, 8-9].

Unfortunately, the most recent systematic reviews on this topic [5-9] did not report encouraging findings. As a matter of fact, they agreed that no reliable or definitive conclusions or recommendations can be derived concerning the efficacy of the different treatment protocols for the correction of dento-skeletal open bite in the growing patient, either in the short or in the long term.

When considering open bite in the mixed dentition from a diagnostic point of view, it is important to distinguish between at least 2 forms: dentoalveolar anterior open bite with persisting digit sucking habits and dentoskeletal open bite without sucking habits (i.e., patients with vertical skeletal imbalance). These two forms of anterior open bite in the growing patient require different treatment approaches.

ANTERIOR OPEN BITE WITH PERSISTENT DIGIT SUCKING HABITS

Sucking habits frequently are involved in the development or the maintenance of anterior open bite during growth. Digital sucking, e.g., thumb, finger, is a significant risk factor for the establishment of an anterior open bite in subjects with increased vertical skeletal relationships. The same subjects often show increased flaring of the upper incisors as well as lingual inclination of the lower incisors. In addition, a prolonged sucking habit can lead to transverse discrepancies concurrent with the vertical problems. The correction of maxillary constriction is a main target for treatment in open-bite patients [6, 7].

In 2005, in a joint investigation between the University of Florence and the University of Rome "Tor Vergata", Cozza et al. [10] looked at possible risk factors for the development of anterior open bite in the presence of thumb-sucking. They analyzed a sample of 1710 subjects with the mean age of nine years who were in the intermediate or late mixed dentition. These subjects had been observed from 1990 to 2005 at the orthodontic departments of the University of Florence and the University of Rome "Tor Vergata" as part of a multicenter clinical investigation on the prevalence of dentoskeletal open bite. For each subject, the following data were collected: presence or absence of persistent thumb-sucking habits beyond two and half years of age and the presence or absence of hyper-divergency, with hyperdivergence defined as a mandibular plane angle (FMA) larger than 25°. In addition, the presence or absence of anterior open bite was noted, as determined by the presence of a negative overbite with the lack of vertical overlap of the anterior dentition (lateral incisor to lateral incisor).

When the 1710 subjects were analyzed, 303 (18% of the total sample) presented with an anterior open bite, with the remainder (1407 subjects) having a positive vertical overlap of the anterior dentition. The prevalence of thumb-sucking habits in the absence of hyper-divergency was then determined. No statistically significant difference was found in the prevalence rate of thumb-sucking habits in the absence of hyper-divergency between the group with anterior open bite (27%) and the group without anterior open bite (20%). Similarly, when looking at aspects of hyper-divergence in absence of thumb-sucking of the two groups, the prevalence rates in the two groups were almost identical (about 25%). Only when the concurrent presence of sucking habits and facial hyper-divergence was considered, the prevalence rate of this condition was significantly greater in the group with anterior open bite with respect to the group without anterior open bite (36.3% and 9.1%, respectively). Therefore, only the simultaneous presence of thumb-sucking and hyper-divergency can be considered an increased risk for anterior open bite.

Treatment Alternatives

How can anterior open bite be treated successfully? What kind of therapy is best indicated? Can treatment be accomplished with fixed or removable appliances? And, is treatment of anterior open bite stable in the long term?

Quad-helix with tongue crib

The use of a crib has been advocated to stop sucking habits by acting as a digit-inhibiting appliance. A proposed treatment protocol aimed to eliminate the thumb-sucking habit and to correct both the anterior open bite and the maxillary transverse deficiency in growing subjects is a quad-helix (Q-H) appliance with the addition of a tongue crib (Figure 1) [11].

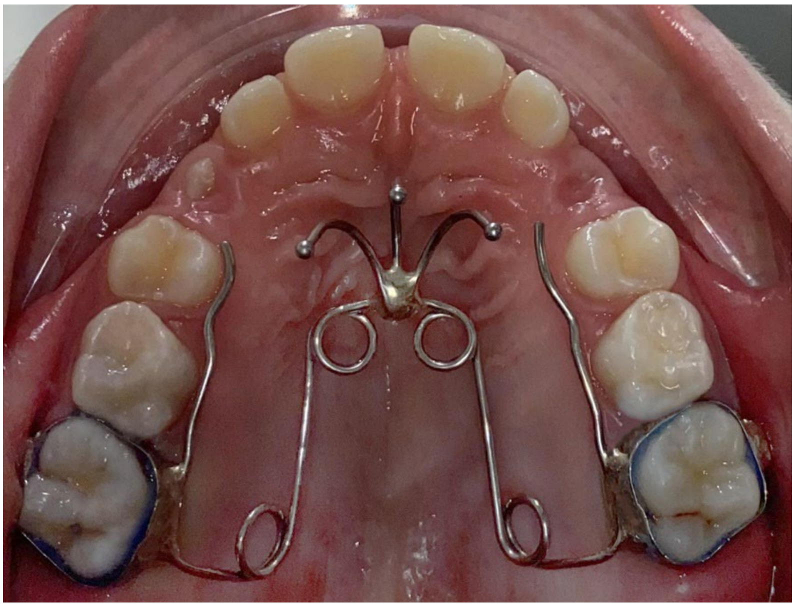

Figure 1. Intraoral view of Q-H/C appliance in place.

In terms of Q-H/C treatment protocol, the Q-H/C appliance is cemented on the second deciduous molars or on the first permanent molars of the upper arch (Figure 1). The lingual arms of the appliance extend mesially to the deciduous canines or even to the permanent incisors. Spurs intended for thumb-sucking prevention are formed from three or four short pieces of .036” stainless steel wire that are soldered to the anterior part of the palatal wire of the Q-H and inclined lingually to avoid impingement on the sublingual mucosa. The Q-H/C is activated transversally at about half of each molar on both sides of the maxilla. The appliance is worn for an average period of 18 months and is reactivated once or twice during treatment to achieve overcorrection of the transverse relationships.

The results of a previous longitudinal short-term study by our research group showed the clinical effectiveness of the Q-H/C in correcting the dental open bite in 90% of patients [12]. In this study 23 subjects treated with the Q-H/C appliance were compared with a control group of 23 untreated subjects with similar vertical relationships. Lateral cephalograms were analyzed before treatment (T1) and immediately after treatment (T2).

The average increase in overbite during Q-H/C therapy (3.6 mm more than the control group) overcorrected the amount of anterior open bite at T2. Both the maxillary and mandibular incisors had significantly greater lingual inclinations (approximately 4.0°) associated with greater extrusion (1.4 and 1.0 mm, respectively) in the Q-H/C group than in the control group. In addition, the treated group showed a greater downward rotation (1.2°) of the palatal plane than did the control group. This change was associated with a slight increase in upper anterior facial height (0.7 mm) and a clinically significant reduction in the palatal plane to mandibular plane angle (-1.7°) in the Q-H/C group with respect to the controls. The upper and lower lips showed significant tendencies toward retraction relative to the E-plane in the treated group (2.6 and 2.9 mm, respectively) compared with the controls.

Figure 2. A and B: Open bite bionator.

Open bite bionator vs quad-helix with tongue crib

In 2007, Cozza et al. [13] showed the results of the comparison of a removable appliance, the Open bite bionator (OBB; Figure 2A), versus a fixed appliance, the Q-H/C appliance in growing subjects who presented with thumb-sucking habits and with dento-skeletal open bite. The inclusion criteria of the study were: persistent digit sucking, dentoalveolar open bite with a negative overbite, facial hyper-divergency, fully erupted permanent first molars and incisors and the availability of pre- and post-treatment lateral cephalograms.

In terms of Q-H/C treatment protocol, the Q-H/C was cemented with bands on the second deciduous molars or on the first permanent molars in the upper arch. The OBB had posterior acrylic bite blocks to prevent extrusion of the posterior teeth (Figure 2B). The acrylic portion of the lower lingual part extended into the maxillary incisor region in the form of a lingual shield that would close off the anterior space without touching the maxillary teeth. This portion of the appliance was intended to stop the tongue from getting into the open bite. The mean age at the time of the first and second observation and mean observation intervals were basically identical. The duration of treatment was 2.5 years. The changes from the T1 to T2 included 1 year post treatment effects without any retention.

In terms of vertical skeletal measurement, the results of this study found no significant differences in the effects of the Q-H/C appliance versus OBB. The differences were all within 1 mm or degree. On the contrary, in terms of vertical dentoalveolar measurements, it is important to emphasize that the Q-H/C appliance showed a significantly greater correction of the overbite (+1.9 mm). The maxillary incisors presented with significantly greater extrusion (+1.5 mm); this extrusion of the upper incisors facilitated the closure of the open bite.

Thus, the comparison of two treatment protocols for open bite malocclusions, one year after the end of treatment showed that the Q-H/C is more effective than the OBB for the improvement of overbite in anterior open bite growing patients. Better outcomes of the Q-H/C appliance are also associated with the extrusion of the maxillary incisors. The findings indicate that a compliance-free appliance such as the Q-H/C can induce more favorable changes in the vertical plane than a removable appliance such as the OBB.

Figure 3. Removable upper plate with crib.

Quad-helix appliance with crib vs removable plate with crib

Another appliance that has been proposed for the early treatment of anterior open bite in patients with persisting sucking habits is the removable upper plate with a crib (RP/C) (Figure 3) [14]. The removable plate consisted of a modified Schwarz plate with Adams clasps on the maxillary first molars and ball clasps between the second and first deciduous molars. Additional components were a midline jackscrew, a crib consisting of loops modeled with 0.036” stainless steel wire, and a labial wire (0.036” stainless steel) with loops distal to the maxillary canines. The jackscrew was activated once per week for slow expansion. The appliance was worn at least 16 hours for day for an average period of 16 months. Compliance was good in all patients.

In 2008, Giuntini et al. [14] compared the effects of the Q-H/C appliance and a removable plate with a crib (RP/C) in patients with dentoskeletal open bite. The Q-H/C sample and the RP/C sample consisted of 20 subjects. Lateral cephalograms were analyzed before treatment (T1) and after active treatment (T2). The mean duration of treatment was 1.5 years in both groups.

The patients had the following features: thumb-sucking habit before treatment (T1), dentoskeletal open bite at T1 (negative overbite and MPA ≥25°), no permanent teeth extracted before or during treatment, consecutive lateral cephalograms of good quality taken at T1 and post-treatment (T2), and prepubertal skeletal maturity at both T1 and T2 (CS1 or CS2 according to the cervical vertebral maturation method) [15].

The results of this study showed no significant differences between the 2 appliance groups for any examined cephalometric variable at T1, apart from the position of Pogonion relative to Nasion perpendicular, which was more protruded in the Q-H/C group, and the nasolabial angle, which was smaller in the Q-H/C group when compared with the RP/C group. There were no significant differences between the 2 groups for any measures in the sagittal plane of the maxilla and the mandible from T1 to T2 except for Point A to Nasion perpendicular, which demonstrated significant decreases in the Q-H/C group when compared with the RP/C group (-1.1 mm).

The changes in the sagittal intermaxillary relationships were similar in the Q-H/C and in the control groups. The Q-H/C group had greater downward rotation of the palatal plane when compared with the RP/C group (1.5°). This change was associated with a significant reduction in the palatal plane-mandibular plane angle (-1.2°) in the Q-H/C group with respect to the RP/C group. The conclusions that can be drawn from this study were that both the Q-H/C and the RP/C appliances induced favorable dental effects. However, a compliance-free appliance, such as the Q-H/C, produced more favorable vertical skeletal changes.

Post-treatment Stability of Open Bite Treatment

Another challenging question is if the early correction of anterior open bite in patients with persisting sucking habits will remain stable in the post-treatment period. In a study of Mucedero et al. in 2013 [16], investigators evaluated the post-treatment stability of Q-H/C appliance treatment in subjects who presented with thumb-sucking habits and anterior dentoskeletal open bite. Both active treatment and post-treatment effects were analyzed in treated patients. The results of this treatment protocol were compared with the growth changes in an untreated control group (CG) with the same dentoskeletal disharmony during a follow-up period of at least 5 years.

Twenty-eight subjects (11 boys, 17 girls; mean age, 8.2 +/- 1.3 years) were treated consecutively with Q-H/C appliance. The patients were re-evaluated at the end of active treatment with the Q-H/C (mean age, 9.7 +/- 1.6 years) and at least 5 years after the completion of treatment (mean age, 14.6 +/- 1.9 years). A CG of 20 untreated subjects with the same dentoskeletal disharmony was used for the statistical comparison.

The inclusion criteria were thumb-sucking habit before treatment, negative overbite, constricted maxillary arch, full eruption of first permanent molars and permanent incisors. All subjects were at a prepubertal stage of skeletal maturity according to the cervical vertebral maturation method (CS 1 or CS 2) at T1 and postpubertal skeletal maturity at T3 (CS 4-6). All patients were re-evaluated at T3 at least 5 years after the completion of treatment with the Q-H/C. During the T2-T3 interval, all patients received fixed appliances with no auxiliaries, including vertical or sagittal elastics.

During the T1-T2 interval, in comparison to CG, the treated group exhibited a significant increase in overbite of about 2 mm, as well as a significant downward rotation of the palatal plane of about 2° that led to a significant reduction of about 2° in the palatal plane to mandibular plane angle. These dentoskeletal changes are not only statistically significant but more importantly they are clinically relevant. No significant changes in the mandibular plane angle, however, could be recorded. No significant differences in post-treatment changes (T2-T3) were found between the Q-H/C and control groups (Tables for T1-T2 and T2-T3 changes the are available in the original article) [16].

In the overall post-treatment period, from T1 to T3 (Table 1), the use of the Q-H/C and fixed appliances led to successful outcomes in about 93% of the patients. The intermaxillary skeletal relationships showed a significant reduction in the ANB angle of -1.3° in the Q-H/C group compared with the CG. Vertical skeletal variables maintained a significant improvement in the Q-H/C group vs the controls (Frankfort horizontal to palatal plane, 1.8°; palatal plane to mandibular plane, -2.2°). This favorable outcome contributed significantly to the overall correction of the anterior open bite of about 5 mm (2.1 mm with respect to the controls) and a significant decrease was found for overjet in the Q-H/C group vs the controls (-1.5 mm).

Table 1. Comparison of overall post-treatment changes (T1-T3).

	Q-H/C group T1 (n=28)		*Q-H/C group T3 (n=28)*			
Cephalometric measurement	*Mean*	*SD*	*Mean*	*SD*	*Difference (T1-T3)*	*Significance*
Maxillary skeletal						
SNA (°)	-0.6	2.3	1.5	3.5	-2.1	NS
Point A-nasion perp (mm)	-0.2	2.5	1.7	3.8	-1.9	NS
Mandibular skeletal						
SNB (°)	1.3	2.3	2.1	3.5	-0.8	NS
Pg-nasion perp (mm)	3.8	4.6	4.6	7.0	-0.8	NS
Co-Gn (mm)	14.2	6.1	15.8	4.4	-1.6	NS
Maxillary/mandibular						
ANB (°)	-1.9	1.9	-0.6	1.7	-1.3	*
Wits (mm)	0.6	3.6	2.3	3.7	-1.7	NS
Vertical skeletal						
FH-PP (°)	1.1	2.9	-0.7	1.7	1.8	*
MPA (°)	-3.2	2.7	-2.8	3.7	-0.4	NS
PP-mandibular plane (°)	-4.3	3.0	-2.1	3.3	-2.2	*
ANS-Me (mm)	6.3	3.3	7.7	3.1	-1.4	NS
Co-Go (mm)	7.1	5.1	8.5	3.9	-1.4	NS
Gonial angle (°)	-6.0	4.2	-4.5	3.6	-1.5	NS
Interdental						
Overjet (mm)	-0.6	2.7	0.9	2.0	-1.5	*
Overbite (mm)	4.9	2.0	2.8	1.7	2.1	†
Interincisal angle (°)	4.1	12.7	-0.2	8.4	4.3	NS
Molar relationship (mm)	1.3	2.6	0.8	2.1	0.5	NS
Maxillary dentoalveolar						
U1-Point A vert (mm)	1.7	2.5	2.3	2.0	-0.6	NS
U1-FH (°)	-1.0	8.4	0.7	6.3	-1.7	NS
Mandibular dentoalveolar						
L1-Point A Pg (mm)	1.0	2.3	0.9	1.8	0.1	NS
L1-MPA(°)	0.8	6.1	2.4	4.9	-1.6	NS

NS, Not significant; perp, perpendicular; Pg, pogonion; FH, Frankfort horizontal; PP, palatal plane; U1, maxillary central incisor; vert, vertical; L1, mandibular central incisor. *$P<0.05$; †$P<0.01$.

In summary, when analyzing the post-treatment changes, the Q-H/C appliance and fixed appliances led to successful outcomes in 93% of the patients and a mean closure of the anterior open bite of about 2 mm with respect to untreated controls. The Q-H/C protocol also produced a clinically significant decrease of intermaxillary divergence (-2.2°) mainly due to a downward rotation of the palatal plane (1.8°). This favorable outcome contributed significantly to the overall correction of the anterior open bite with an improvement in the vertical skeletal relationships.

Dentoskeletal Open Bite Without Sucking Habits

What are the treatment options during the mixed dentition for an open bite growing patient with dentoskeletal open bite without sucking habits? These patients have a vertical skeletal imbalance rather than a dentoalveolar malocclusion.

In skeletal open bite malocclusion, patients display backward and downward rotation of the mandible, increased vertical growth of posterior dentoalveolar structures, increased lower anterior facial height, and a narrow maxillary arch. Several authors emphasized that a skeletal open bite should be treated in the mixed dentition to take advantage of active growth by expanding the maxillary arch and preventing further vertical growth of the upper and lower posterior dentoalveolar regions [5,8-9].

Mucedero et al, in 2018 [17], evaluated the post-treatment stability of Rapid Maxillary Expansion (RME) and Removable Mandibular Bite-Block (RMBB) therapy in growing subjects with dentoskeletal open bite (no sucking habits) when compared to a control group with untreated open bite. The treated group (TG) comprised 16 subjects (14 girls, 2 boys) with a mean age of 8.1 ± 1.1 years who were treated consecutively at the Department of Orthodontics of the University of Rome, ''Tor Vergata.''

The inclusion criteria included no sucking habits, overbite < 0 mm, posterior transverse interarch discrepancy ≥ 3 mm, Frankfort horizontal to mandibular plane angle greater than 26°, full eruption of first permanent molars and of maxillary and mandibular incisors (to prevent the ''pseudo-open bite'' due to undererupted permanent incisors), and no permanent teeth extracted before or during treatment. Each patient had to have three consecutive lateral cephalograms of good quality with adequate landmark visualization.

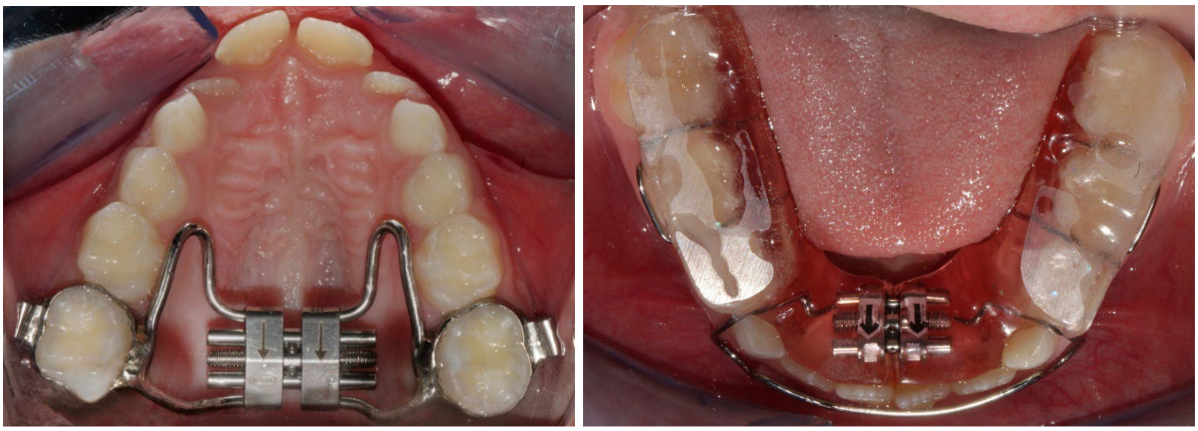

Figure 4. A) Rapid maxillary expander, B) Posterior bite block appliance.

The cephalograms were taken before treatment (T1), at the end of the active treatment with RME and BB (T2), and at a follow-up observation at least 4 years after the completion of treatment (T3). The treatment protocol included RME with soldered to bands on the second deciduous molars or on the first permanent molars (Figure 4A) The expander was left in place for at least 8 months as a passive retainer. No removable appliance was applied after RME removal. The RMBB appliance consisted of a lower Schwartz plate with 5-mm-thick posterior occlusal resin splints. The RMBB was prescribed for 12 months to control the vertical dimension (Figure 4B). The patients were instructed to wear the RMBB 24 hours a day.

During the active phase of treatment from T1 to T2, control of excessive vertical growth in the dentoalveolar segments provided by acrylic coverage of the posterior arch induced an anterior rotation of the mandible (FMA -2.8° in TG vs CG). In particular, the reduced increase in the vertical skeletal dimension was associated with a smaller extrusion of both the maxillary and mandibular first molars. Although the changes of these values were not statistically significant, the sum of reduced upper and lower molar extrusion was both statistically and clinically significant (-3.3 mm), positively affecting the mandibular vertical position.

After the completion of the active phase of treatment with fixed appliances, no statistically significant differences were observed between the TG and CG during the post-treatment T2–T3 interval. (Tables for T1-T2 and T2-T3 changes the are available in the original article) [17].

Table 2. Descriptive Statistics and Statistical Comparisons (Independent-Samples t-Tests) of the T1–T3 Changes in the Treated Group vs the Control Group.

	Treated Group		*Control Group*				*95% Cl of the Difference*	
Variable	*Mean*	*SD*	*Mean*	*SD*	*Difference*	*P Value*	*Lower*	*Upper*
Maxillary skeletal								
Age, y	5.4	1.5	5.0	1.4	0.4	.394	-0.6	1.5
SNA (°)	0.1	1.2	-0.1	1.8	0.2	.682	-0.9	1.3
SNB (°)	1.8	1.6	0.9	1.6	0.9	.127	-0.3	2.0
ANB (°)	-1.7	2.0	-1.0	1.4	-0.7	.279	-1.9	0.6
Wits (mm)	-0.6	3.3	1.0	3.0	-1.6	.157	-3.9	0.7
FMA (°)	-0.5	1.6	-2.3	1.7	**-2.8**	**.000***	-4.0	-1.6
PP- MP (°)	-2.2	2.5	-2.5	1.7	0.3	.709	-1.3	1.8
ANS-Me (mm)	5.1	4.8	6.3	2.8	-1.2	.366	-4.1	1.6
Co-Go (mm)	7.0	2.4	8.6	2.9	-1.6	.109	-3.5	0.4
Overbite (mm)	3.9	1.5	2.1	1.4	**1.8**	**.002***	0.8	2.9
U6 to PP (mm)	2.7	1.6	4.6	1.9	**-1.9**	**.004***	-3.2	-0.6
L6 to MP (mm)	2.6	1.1	3.9	1.5	**-1.3**	**.009***	-2.3	-0.3
Sum, mm[a]	5.2	2.5	8.5	2.8	**-3.3**	**.002***	-5.1	-1.3

y years, PP, palatal plane; MP mandibular plane; U6, maxillary first molar; L6, mandibular first molar; Sum indicates maxillary first molar to palatal plane + mandibular first molar to mandibular plane; SD Standard deviation; *P<0.05.

In the follow-up observation after at least 4 years (T3), all 16 subjects (100%) in the treated group (TG) showed a corrected overbite with a significantly greater increase in overbite of 1.8 mm in the TG compared with the untreated subjects. The comparison of the overall post-treatment changes (T1–T3; Table 2) revealed a significantly greater decrease of the vertical skeletal relationships in treated subjects when compared with the control group (Frankfort horizontal to mandibular plane angle: -2.8°). The TG exhibited a significantly smaller extrusion of both maxillary (maxillary first molar to palatal plane: -1.9 mm) and mandibular first molars (mandibular first molar to mandibular plane: -1.3 mm) compared with the CG. At T2, the prevalence rate of success for recovery of positive overbite was 63% (10 subjects) in the TG, while in the CG, spontaneous correction was not observed in any subject.

The treatment protocol with RME and RMBB was effective in the correction of negative overbite in growing children. The TG exhibited reduced extrusion of maxillary and mandibular molars and, consequently, a significant improvement in the vertical skeletal dimension when compared with untreated open bite subjects. The effects of early treatment with RME and RMBB were stable at a post-treatment follow-up.

Recently, Lione et al. [18] evaluated the mandibular modifications in anterior open bite (OB) growing subjects treated with Rapid Maxillary Expansion and bite block (RME/BB) or Quad Helix with crib (Q-H/C) when compared with a Control Group (CG) by using Geometric Morphometric Method (GMM) [19] and conventional cephalometrics.

The OB group comprised 34 subjects (26 girls, 8 boys) with dentoskeletal OB and a mean age of 8.0 ± 1.0 years. OB group was divided in two subgroups according to the presence or absence of prolonged sucking habits: RME/RMBB group comprised 17 subjects while Q-H/C group included 17 subjects. In the RME/BB group 12 patients, while in the QH/C 11 subjects performed a second phase with fixed conventional appliance to finish the occlusion. No active biomechanics or vertical elastics to extrude the incisors were applied during fixed appliance therapy. No intraoral Class II elastics or bite ramps on posterior teeth or other anterior extrusive/posterior intrusive mechanics were used.

The two subgroups were compared with a CG of 17 subjects (13 girls, 4 boys) matched for sex, age, vertical pattern, and observation periods. Two consecutives lateral cephalograms were available: the first one was taken before treatment (T1), and the second one was acquired at a follow-up observation at least 4 years after the completion of treatment (T2).

Cephalometric analysis was performed according to the method of Franchi et al. [20] to assess ramus inclination to cranial base and mandibular plane. Specifically, the stable basicranial line (SBL), through the most superior point of the anterior wall of sella turcica at the junction with the tuberculum sellae (point T), was drawn tangent to the lamina cribrosa of the ethmoid bone. RME/RMBB group showed a significantly greater decrease of the Condax^MP (Condylar Axis to the mandibular plane) angle and increase in the Condax^SBL (Condylar Axis to SBL) angle when compared to CG and QH/C.

To study the mandibular structures, the GMM was applied. For the evaluation of the shape of the mandible, 2 continuous curves with 31 points, 6 of them being fixed cephalometric landmarks were drawn. In morphological mandibular comparison between RME/RMBB (blue) and QH/C (yellow) the mandibular shape changes were located along the mandibular ramus the mandibular ramus increased in height in RME/RMBB group with tendency to a forward bending of the ramus and condyle (Figure 5).

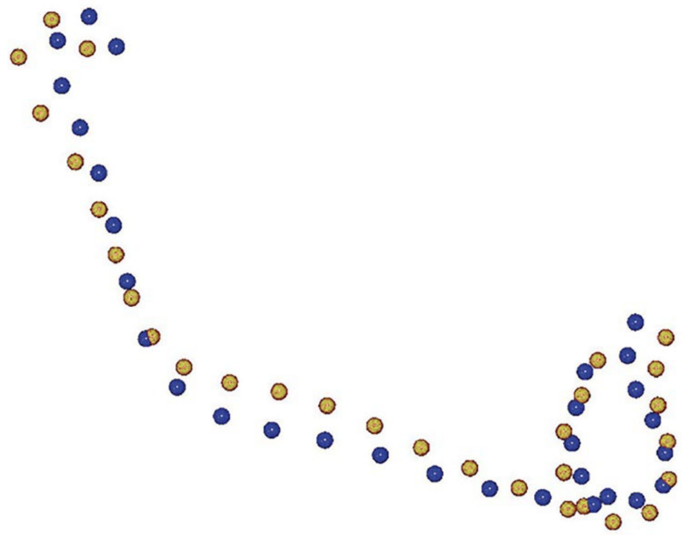

Figure 5. Morphological mandibular comparison between RME/BB (blue) and QH/C (yellow) (from Lione et al. [18]).

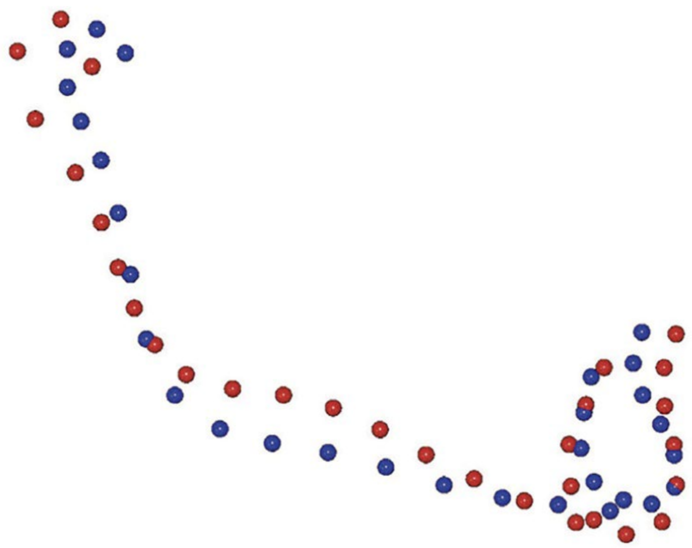

Figure 6. Morphological mandibular comparison between RME/BB (blue) and CG (red) (from Lione et al. [18]).

In morphological mandibular comparison between RME/RMBB (blue) and CG (red), similar mandibular shape differences were found (Figure 6). No statically significant morphologic mandibular changes were found when comparing Q-H/C (yellow) and CG (red) groups (Figure 7). RME/RMBB patients showed significant changes in the vertical orientation of the mandibular ramus with a tendency to a forward bending when compared with Q-H/C and CG subjects. Elongation of the mandibular ramus can contribute to open bite correction leading to reduced steepness of the mandibular and occlusal planes. The RME/RMBB subjects showed significant changes in the shape of the mandibular ramus with a counterclockwise rotation tendency when compared with Q-H/C and CG subjects.

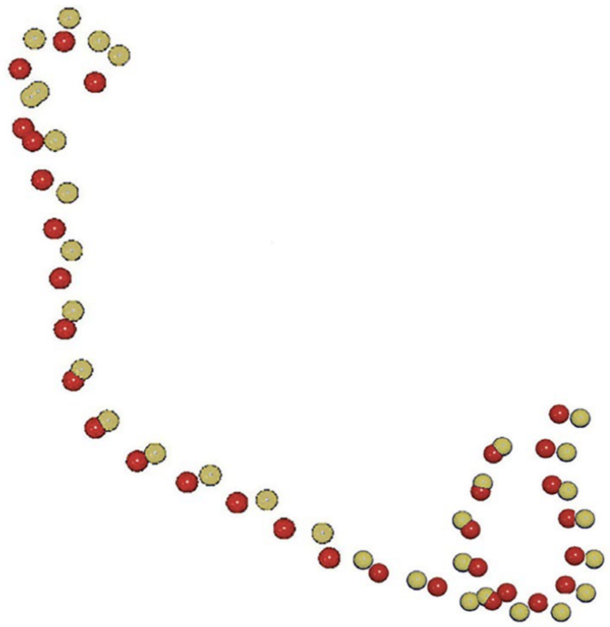

Figure 7. Morphological mandibular comparison between QH/C (yellow) and CG (red) (from Lione et al. [18]).

CONCLUSIONS

In patients with sucking habits, anterior open bite, and skeletal open bite tendency, favorable post-treatment changes in overbite and intermaxillary divergence (downward rotation of the palatal plane) was observed with the Q-H/C appliance protocol. In patients without sucking habits, anterior open bite, and skeletal open bite tendency the RME/RMBB showed favorable post-treatment changes in overbite and facial divergence. RME/RMBB may have contributed to an elongation of the mandibular ramus and a forward bending of the ramus and the condylar process that may have resulted in open bite correction.

REFERENCES

1. Kelly JE, Sanchez M, Van Kirk LE. An assessment of the occlusion of the teeth of children 6-11 years, United States. Vital Health Stat 11 1973; (130):1-60.
2. Proffit WR, Fields HW, Jr., Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the NHANES III survey. Int J Adult Orthodon Orthognath Surg 1998; 13(2):97-106.
3. Ng CS, Wong WK, Hagg U. Orthodontic treatment of anterior open bite. Int J Paediatr Dent 2008; 18(2):78-83.
4. Avrella MT, Zimmermann DR, Andriani JSP, Santos PS, Barasuol JC. Prevalence of anterior open bite in children and adolescents: a systematic review and meta-analysis. Eur Arch Paediatr Dent 2022; 23(3):355-364.
5. Lentini-Oliveira DA, Carvalho FR, Rodrigues CG, Ye Q, Prado LB, Prado GF, Hu R. Orthodontic and orthopaedic treatment for anterior open bite in children. Cochrane Database Syst Rev 2014(9):CD005515.
6. Borrie FR, Bearn DR, Innes NP, Iheozor-Ejiofor Z. Interventions for the cessation of non-nutritive sucking habits in children. Cochrane Database Syst Rev 2015(3):CD008694.

7. Feres MF, Abreu LG, Insabralde NM, de Almeida MR, Flores-Mir C. Effectiveness of open bite correction when managing deleterious oral habits in growing children and adolescents: a systematic review and meta-analysis. Eur J Orthod 2017; 39(1):31-42.
8. Feres MF, Abreu LG, Insabralde NM, Almeida MR, Flores-Mir C. Effectiveness of the open bite treatment in growing children and adolescents: a systematic review. Eur J Orthod 2016; 38(3):237-250.
9. Pisani L, Bonaccorso L, Fastuca R, Spena R, Lombardo L, Caprioglio A. Systematic review for orthodontic and orthopedic treatments for anterior open bite in the mixed dentition. Prog Orthod 2016; 17(1):28.
10. Cozza P, Baccetti T, Franchi L, Mucedero M, Polimeni A. Sucking habits and facial hyperdivergency as risk factors for anterior open bite in the mixed dentition. Am J Orthod Dentofacial Orthop 2005; 128(4):517-519.
11. Cozza P, Giancotti A, Rosignoli L. Use of a modified quad helix in early interceptive treatment. J Clin Orthod 2000; 34(8):473-476.
12. Cozza P, Baccetti T, Franchi L, McNamara JA, Jr. Treatment effects of a modified quad-helix in patients with dentoskeletal open bites. Am J Orthod Dentofacial Orthop 2006; 129(6):734-739.
13. Cozza P, Baccetti T, Franchi L, Mucedero M. Comparison of 2 early treatment protocols for open-bite malocclusions. Am J Orthod Dentofacial Orthop 2007; 132(6):743-747.
14. Giuntini V, Franchi L, Baccetti T, Mucedero M, Cozza P. Dentoskeletal changes associated with fixed and removable appliances with a crib in open-bite patients in the mixed dentition. Am J Orthod Dentofacial Orthop 2008; 133(1):77-80.
15. McNamara JA, Jr., Franchi L. The cervical vertebral maturation method: a user's guide. Angle Orthod 2018; 88(2):133-143.
16. Mucedero M, Franchi L, Giuntini V, Vangelisti A, McNamara JA, Jr., Cozza P. Stability of quad-helix/crib therapy in dentoskeletal open bite: a long-term controlled study. Am J Orthod Dentofacial Orthop 2013; 143(5):695-703.
17. Mucedero M, Fusaroli D, Franchi L, Pavoni C, Cozza P, Lione R. Long-term evaluation of rapid maxillary expansion and bite-block therapy in open bite growing subjects: A controlled clinical study. Angle Orthod 2018; 88(5):523-529.
18. Lione R, Fusaroli D, Mucedero M, Paoloni V, Pavoni C, Cozza P. Changes in mandibular shape after early treatment in subjects with open bite: a geometric morphometric analysis. Eur J Orthod 2020 Jan 15:cjz104. doi: 10.1093/ejo/cjz104. Epub ahead of print.
19. Huanca Ghislanzoni L, Lione R, Cozza P, Franchi L. Measuring 3D shape in orthodontics through geometric morphometrics. Prog Orthod 2017; 18(1):38.
20. Franchi L, Baccetti T, Tollaro I. Predictive variables for the outcome of early functional treatment of class III malocclusion. Am J Orthod Dentofacial Orthop 1997; 112(1):80-86.

SKELETAL AND DENTAL EFFECTS WITH EXPANDER WITH DIFFERENTIAL OPENING AND FAN-TYPE EXPANDER: RESULTS FROM A RANDOMIZED CONTROLLED TRIAL

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ABSTRACT

Rapid maxillary expansion (RME) is one of the most common therapy options to treat maxillary constriction and posterior crossbite. The study of the outcomes of different expander designs is highly important to guide clinicians during treatment planning. This chapter aims to present the main findings of a randomized controlled trial comparing two treatment options for RME in growing patients: the expander with differential opening (EDO) and the fan-type expander (FE). Dentoskeletal outcomes of the EDO and the FE were studied by means of digital dental models and cone-beam computed tomography (CBCT). The EDO and the FE produced skeletal changes in the maxilla. The EDO produced greater transverse skeletal expansion compared to the FE. Vertical and anteroposterior effects were similar. In both expander designs, greater transverse increase was observed in the anterior region of the maxillary dental arch compared to the posterior region. Greater changes were observed in the molar region in the EDO and in the canine region for the FE, resulting in distinct arch shapes after expansion. From a clinical point of view, both expanders are indicated to correct a greater dental arch constriction in the canine region compared to the molar region, but the EDO should be preferred when the posterior crossbite extends to molars.

KEY WORDS: Palatal Expansion Technique, Orthodontic Appliance Design, Three-dimensional Imaging

INTRODUCTION

Rapid maxillary expansion (RME) comprises the orthopedic procedure commonly indicated to treat maxillary constriction and posterior crossbite by means of separating and expanding the midpalatal suture [1]. The first report of this therapy is credited to Angell in 1860 [2], becoming more used in orthodontics after publications from Haas [1, 3, 4]. Since then, different appliances for RME have been presented in the literature [1, 5-8]. The study of the outcomes of different expanders' designs is highly important to help clinicians make appropriate treatment modality decisions in their daily clinical practice.

The most popular expander design has a screw positioned in the center of the palate [1, 5, 6]. These conventional expanders, including the Haas type expander, the Hyrax expander, and the bonded expander, promote a parallel opening of the screw and the maxillary dental arch. The fan-type expander (FE) has one screw positioned anteriorly on the palate (Figure 1) [7]. The FE has a hinge in the posterior region that concentrates the expansion force in the canine region and reduces it in the molar region. Previous comparison studies between the FE and the Hyrax expander showed similar expansion in the canine region and smaller skeletal and intermolar expansion in the FE group [9, 10]. Another design is an expander with differential opening (EDO), which has two palatal screws located in the anterior and the posterior regions (Figure 2) [8]. The different amount of activation of these two screws allows for independent expansion in the molar and canine regions. Compared to the traditional Hyrax expander with the screw positioned in the center, the EDO promotes greater changes in the anterior region of the maxilla with similar changes in the posterior dental arch width [11].

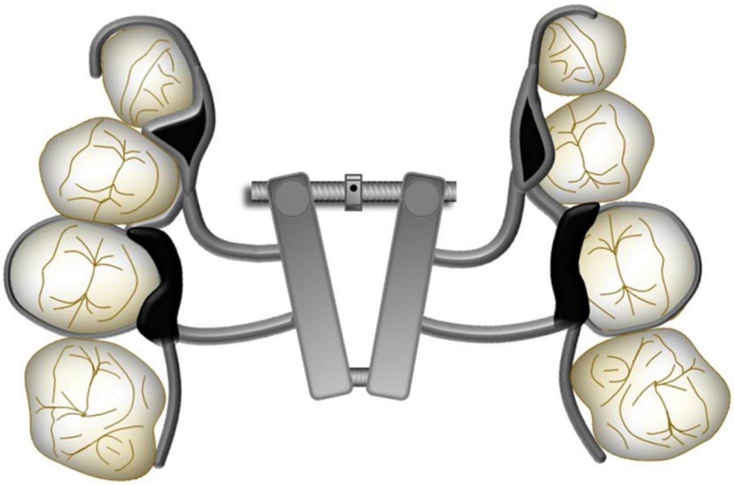

Figure 1. The fan-type expander after activation. The expander has a posterior hinge, and the screw is positioned in the anterior region of the palate.

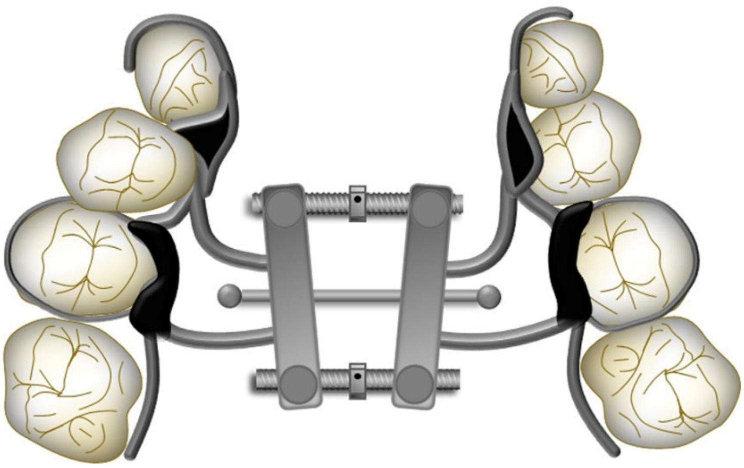

Figure 2. The expander with differential opening after activation. Note that the expander has two screws. The greater activation of the anterior screw compared to the posterior results in a trapezoidal shape opening of the expander.

Currently, there are no studies that compare the treatment effects of the EDO and the FE. Are the dentoskeletal changes produced by these two expanders similar? To better answer this question, a randomized controlled trial (RCT) to compare the outcomes of these two expanders in the mixed dentition was conducted (Trial Registration: clinicaltrials.gov, NCT03705871) [12, 13].

The sample consisted of 48 subjects from 7 to 11 years of age presenting with posterior crossbite. Patients were enrolled and randomly allocated into two groups: 24 patients were treated with the EDO (13 females, 11 males and mean initial age of 7.6 years ±0.92); and 24 patients were treated with the FE (14 females, 10 males and mean initial age of 7.8 years ±0.96). In both groups, bands were placed in the deciduous second molars, clasps were bonded in the deciduous canines, and wire extensions were added along the gingival margins of permanent first molars (Figures 3 and 4). In the FE group (Figure 3), the expander screw was activated two-quarter turns in the morning and two-quarter turns in the evening for 10 consecutive days (0.8 mm per day), resulting in an expansion of 8 mm. In the EDO group (Figure 4), the anterior and posterior screws were activated two-quarter turns in the morning and two-quarter turns in the evening for the first 6 days (0.8 mm in each screw per day), resulting in an initial parallel opening of the expander. After this first phase, only the anterior screw was further activated with the same protocol for 4 extra days, resulting in a greater expansion in the anterior screw (8 mm) compared to the posterior one (4.8 mm). The retention period with the appliance comprised 6 months. Digital dental models and cone-beam computed tomography (CBCT) were acquired before and after expansion to compare the dentoskeletal outcomes of the EDO and the FE. The post-treatment CBCT was taken from the first to the sixth month after active phase of the expansion. Post-expansion digital dental models were taken after retention. In the present chapter, the main findings of this RCT will be presented [12, 13].

Figure 3. A 7-year-old female patient treated with the fan-type expander. A) Pre-treatment records showing a skeletal Class I patient in the mixed dentition with bilateral posterior crossbite. B) Delivery of the fan-type expander. C) end of the active phase of the expansion (note the increase in the interincisal diastema width). D) Six months post expansion, after expander removal. After debonding, a partial fixed appliance was delivery to close the diastemas and align the maxillary incisors. E) Patient in the permanent dentition, 2 years after expansion. A-E refer to rows.

A
B
C
D
E

Figure 4. A 10-year-old female patient treated with the expander with differential opening. A) Pre-treatment records showing a skeletal Class I patient in the mixed dentition with posterior crossbite on the left side. B) Delivery of the expander with differential opening. C) End of the active phase of the expansion. Observe the opening of a diastema between the maxillary central incisors as well as the trapezoidal shape of the expander after the different activation of the anterior and posterior screws. D) Six months post-expansion, after expander debonding. A spontaneous correction of the maxillary incisor alignment was observed after the increase in the arch width and arch perimeter as well as the improvement in the maxillary arch shape. E) Patient in the permanent dentition, 2 years after expansion. A-E refer to rows.

CHANGES IN THE NASOMAXILLARY COMPLEX

The CBCT scans were used to assess the skeletal outcomes of the RME with EDO and FE in the nasomaxillary complex [12]. Pre and post expansion CBCT scans were superimposed on the cranial base. Lateral, vertical, and anteroposterior displacements of the maxilla were quantitatively assessed [12]. The results of the CBCT analyses showed that there was a difference in skeletal changes in the nasomaxillary complex following expansion with EDO and the FE (Figure 5). The EDO produced greater changes compared to the FE at the level of the orbit, nasal cavity, zygomatic bone, and palatine foramen (Figure 6). The skeletal expansion at the level of the palatine foramen was increased in patients treated with the EDO (2 mm) compared to those treated with the FE (1 mm) [12]. Considering the activation of 8 mm in both expanders, the skeletal effect of RME on the posterior region of the palate and at the zygomatic bone corresponded to 26% and 32% for the EDO, and 11% and 20% for the FE, respectively. The greater skeletal effect of the EDO compared to the FE may be explained by the activation of the second screw in the EDO.

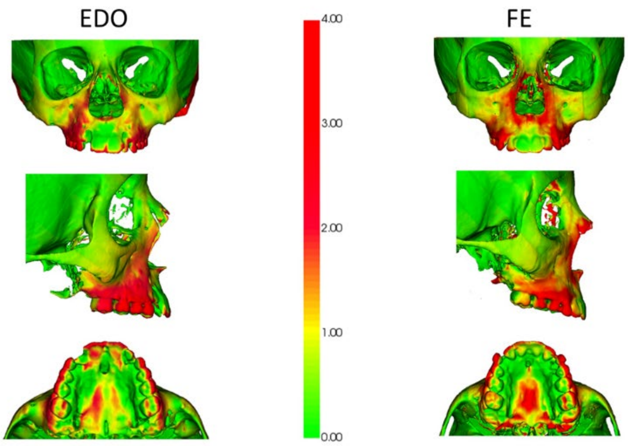

Figure 5. Color map images from a patient treated with the expander with differential opening, EDO, and the fan-type expander, FE (range, 0 mm, no displacements, to 4 mm).

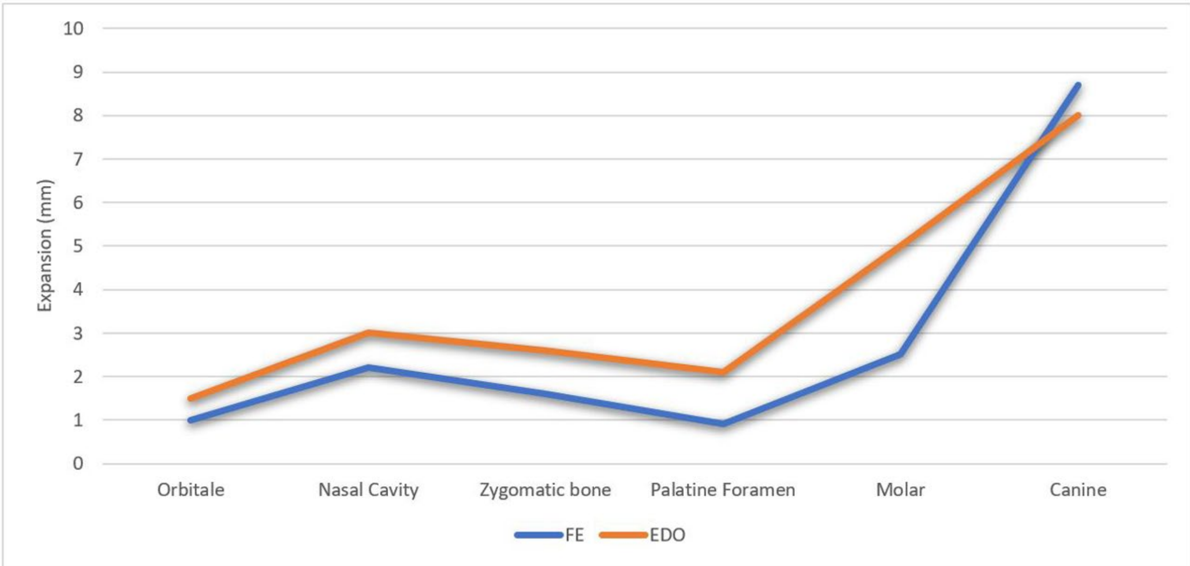

Figure 6. Amount of expansion, in millimeters, after rapid maxillary expansion with the expander with differential opening (EDO; orange color line) and the fan-type expander (FE; blue color line) at the level of canines, molars, palatine foramen, zygomatic bone, nasal cavity and orbitale.

The expansion at the level of the nasal cavity was 3 mm in the EDO and 2 mm in the FE group [12]. The impact of the RME on the nasal cavity has been studied by Haas [1]. In 1959, Krebs showed a transverse increase in the nasal cavity close to 2 mm after conventional expansion [14]. The effects of the RME in the nasal cavity occur because of the lateral movement of the nasal walls and the lowering of the palatine processes [1]. These changes in the nasal area may benefit patients with obstructive breathing disorders, and further studies are being developed to investigate the functional impact of the RME.

A slight downward and forward maxillary displacement after conventional RME was previously reported in the literature [1, 15]. As the midpalatal sutures separated and expanded, the right and left maxillary segments displaced and rotated, moving the nasomaxillary complex. The maxillary vertical and anteroposterior displacements were mild and similar after expansion with the EDO and FE [12]. A slight forward and downward movement of the nasomaxillary complex was observed with both expander designs [12].

MAXILLARY AND MANDIBULAR DENTAL ARCH CHANGES

After RME with the EDO and the FE, several changes were observed in the maxillary dental arch [13]. However, changes in the mandibular dental arch were very mild [13].

Interincisal diastema

The clinical evidence of maxillary disjunction and midpalatal suture separation following RME procedure is the opening of a temporary diastema between the central incisors [1]. In this study, changes in the interincisal diastema were observed immediately after the active phase of the RME with EDO and FE. The diastema was clinically assessed using an odontometric caliper (Precision Equipment Co., Boston, Massachusetts, USA) [13]. All patients showed a temporary increase in the distance between the mesial surface of right and left maxillary central incisors following expansion (Figure 3, Row C and Figure 4, Row C). The change was similar in patients treated with the EDO and the FE [13]. This result was expected because the amount of activation performed in the anterior screw of the EDO and in the screw of the FE was standardized to 8 mm. The interincisal diastema width increased approximately 3.5 mm in both groups when measured immediately after the active phase of the expansion. The increase in the interincisal diastema width after differential and fan expansions corresponded to approximately 50% of the screw activation.

Maxillary arch width

Maxillary and mandibular dental arch dimensions including arch widths and arch perimeter were measured in pre and post expansion digital dental models, using OrthoAnalyzer 3D software (3-Shape A/S, Copenhagen, Denmark) [13]. Maxillary arch widths were measured at the level of the cusp tips of the deciduous canines, deciduous first and second molars, and permanent first molars. The FE showed a slightly greater increase in the intercanine distance compared to the EDO (1 mm) [13]. This finding may be explained by the greater canine buccal inclination also observed in the patients treated with the FE (Figure 7) [12].

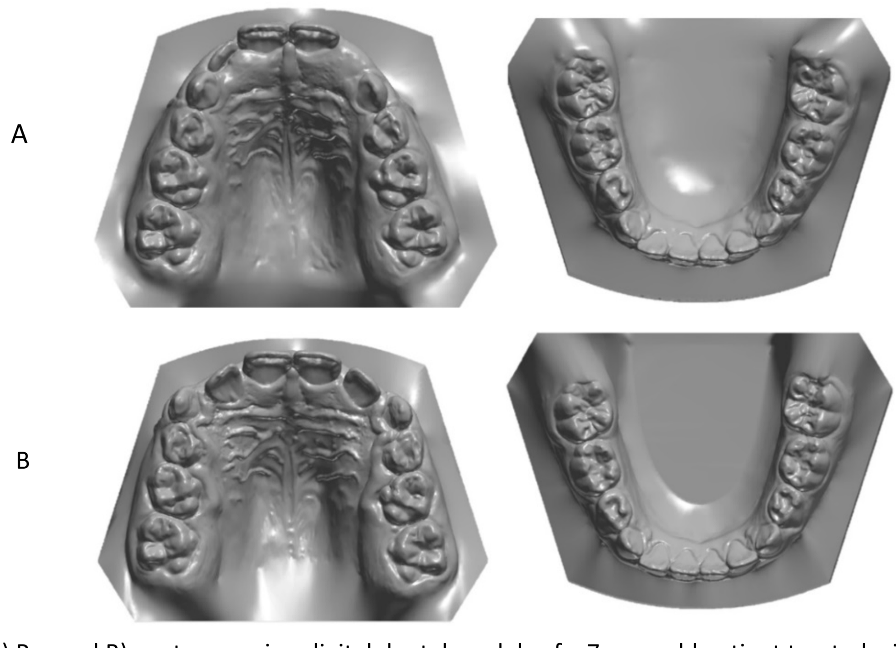

Figure 7. A) Pre and B) post expansion digital dental models of a 7-year-old patient treated with the fan-type expander. Observe the increase in the maxillary dental arch perimeter and arch widths.

Conversely, patients treated with the EDO (Figure 8) showed greater increase in the distances between deciduous second molars (mean difference of 1.4 mm) and permanent first molars (mean difference of 2.7 mm) [13]. The activation of the second screw of the EDO, positioned in the posterior region of the palate, and the presence of a posterior hinge in the FE explain this clinically relevant difference between the two expander designs. The amount of expansion was 1.5 and 3.5-fold greater in the canines than in the first molars for the EDO and FE, respectively [13]. Figures 7 and 8 illustrated the treatment changes in the maxillary dental arch after RME with the FE and EDO, respectively.

A

B

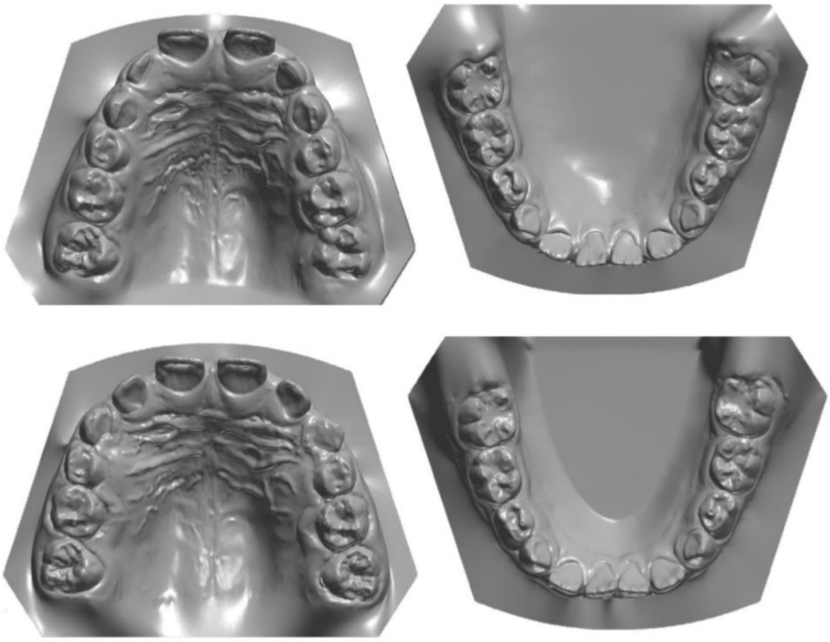

Figure 8. A) Pre and B) post expansion digital dental models of a 7-year-old patient treated with the expander with differential opening. Observe the increase in the maxillary dental arch perimeter and arch widths.

Maxillary arch perimeter

The dental arch perimeter corresponded to the sum of the four segments from mesial aspect of the right first permanent molar to the mesial aspect of the contralateral tooth. After RME, an increase in the arch perimeter is expected as a result of the increase in the maxillary arch width. Interestingly, a similar increase in the maxillary arch perimeter was observed in both expanders' designs used in the study despite the previously mentioned differences for the arch widths [13]. The fan (Figures 3 and 7) and differential (Figures 4 and 8) expansions promoted an increase of approximately 5 mm in the maxillary arch perimeter. This finding is relevant and reinforces the clinical indication of both EDO and FE as an interceptive approach to treat mild to moderate crowding in the maxillary arch during mixed dentition.

Maxillary arch shape

Digital dental models were also used to assess the changes in the maxillary and mandibular dental arch shape [13]. For this analysis, the Stratovan Checkpoint (Stratovan Corporation, Davis, California, USA) and the MorphoJ software (Klingenberg Lab, Manchester, UK) were used to create the mean arch shape for each expander at the two time points and perform the superimposition [13]. Both the EDO and the FE changed the maxillary arch shape after RME, as shown in Figures 7 and 8 [13]. Changes in the maxillary arch shape resulted from the increases in the arch widths after expansion. In addition, the post expansion maxillary arch shape was different when comparing the two expanders' designs, as shown in Figure 9. The maxillary dental arch was wider in the molar region after RME with the EDO. Conversely, arch shape was wider in the canine region after expansion with the FE.

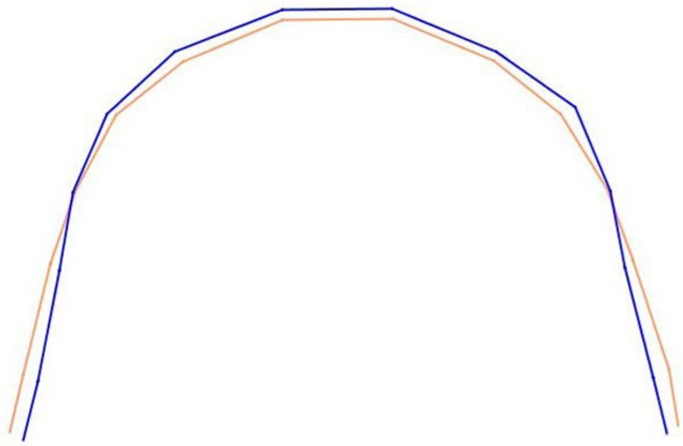

Figure 9. Maxillary mean arch shape superimposition after rapid maxillary expansion with the expander with differential opening (orange color line) and the fan-type expander (blue color line). Note the different arch shape after expansion. Changes were greater in the canine region for the fan expander and in the molar region for the expander with differential opening.

Mandibular dental arch changes

Changes in the mandibular arch were measured in pre and post expansion digital dental models (Figures 7 and 8). The mandibular dental arch showed negligible spontaneous changes after RME with EDO and FE [13]. Dentoalveolar expansion in the mandible after maxillary expansion may occur as a result of changes in the balance between facial muscles (tongue and buccinator), as well as in the cusp-fossa relationship, resulting in up righting of the mandibular posterior teeth [1, 16]. No difference between the two expanders was observed in the mandibular arch, except for the slightly greater expansion at the level of permanent first molars observed in the EDO (0.9 mm) compared to the FE (0.1 mm) [13]. The greater expansion in the posterior region of the maxillary dental arch observed in patients treated with the EDO probably explains this finding in the mandible. The difference between the EDO and the FE in the mandible was very mild and not sufficient to result in a divergent mandibular arch shape between both groups after RME [13].

DENTAL INCLINATION

Buccal inclination of the anchorage teeth is expected after RME procedure [1]. In our study, the assessment of the dental inclination was performed using the CBCT images acquired before and after expansion [12]. Angular measurements were performed at the level of the deciduous canines and permanent first molars. In the molar region, the buccal inclination was very mild after RME with the EDO (2°) and the FE (1°) [12]. This finding is understandable because the expansion in the posterior region of the dental arch was small with both expanders. However, the increased molar buccal inclination observed in patients treated with the EDO may be explained by the greater posterior expansion observed with this expander [12, 13]. Conversely, the canine buccal inclination was 3° greater in patients treated with the FE (8°) compared to those treated with the EDO (5°) [12]. This result may explain the slightly greater increase in the intercanine distance (1 mm) observed in patients treated with FE [12, 13]. A greater expansion in the canine region compared to the molar region was achieved with both expander designs and, consequently, a greater buccal inclination of the anchorage teeth in this region was observed as well.

CORRECTION OF POSTERIOR CROSSBITE AND PATIENT DISCOMFORT

The posterior crossbite was corrected in all 24 patients treated with the EDO and in 18 out of 24 patients treated with the FE [13]. The explanation is that the presence of the posterior hinge in the FE limited the expansion in the posterior region and, consequently, the crossbite was not fully corrected in the molars in 6 patients treated with this expander.

Some discomfort during the activation of the screw is expected in the first days of therapy. In our study, approximately half of patients (26 out of 48) reported some pain or discomfort during the active phase of the expansion [12, 13]. From the 26 patients who reported some discomfort, 57% were from the EDO group (15 out of 24 patients treated with the EDO) and 42% were from the FE group (11 out of 24 patients treated with the FE). The presence of the second screw is probably responsible for this difference between expanders. Further studies could bring more information about the patients' perception and the impact of the RME with EDO and FE in the patient´s quality of life.

CONCLUSIONS

There were similarities and differences with the dental arch and the nasomaxillary complex following RME in patients treated with the EDO and the FE.

- EDO produced greater transverse expansion in the nasomaxillary complex when compared with the FE.
- The increase in the maxillary dental arch transverse width was greater in the anterior region compared to the posterior region in both groups.
- There were greater changes in the molar region with the EDO and a greater change in the canine region with the FE, resulting in distinct arch shapes after expansion with the different expander designs.
- From a clinical point of view, both expanders' designs could be indicated to correct a greater dental arch constriction in the canine region compared to the molar region. However, the EDO should be preferred in patients with posterior crossbite which includes permanent molars.

ACKNOWLEDGEMENTS

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REFERENCES

1. Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. Angle Orthod 1961; 31(2):73-90.
2. Angell EH. Treatment of irregularities of the permanent or adult teeth. Dental Cosmos 1 1860.
3. Haas AJ. The treatment of maxillary deficiency by opening the midpalatal suture. Angle Orthod 1965; 35:200-217.
4. Haas AJ. Palatal expansion: just the beginning of dentofacial orthopedics. Am J Orthod 1970; 57(3):219-255.
5. Biederman W. A hygienic appliance for rapid expansion. J Pract Orthod 1968; 2(2):67-70.
6. McNamara JA, Brudon WL, Kokich VG. Orthodontics and dentofacial orthopedics. Ann Arbor, MI: Needham Press; 2001.
7. Levrini L, Filippi V. A fan-shaped maxillary expander. J Clin Orthod 1999; 33(11):642-643.
8. Garib D, Garcia L, Pereira V, Lauris R, Yen S. A rapid maxillary expander with differential opening. J Clin Orthod 2014; 48(7):430-435.
9. Corekci B, Goyenc YB. Dentofacial changes from fan-type rapid maxillary expansion vs traditional rapid maxillary expansion in early mixed dentition. Angle Orthod 2013; 83(5):842-850.
10. Doruk C, Bicakci AA, Basciftci FA, Agar U, Babacan H. A comparison of the effects of rapid maxillary expansion and fan-type rapid maxillary expansion on dentofacial structures. Angle Orthod 2004; 74(2):184-194.
11. de Medeiros Alves AC, Janson G, Mcnamara Jr JA, Lauris JRP, Garib DG. Maxillary expander with differential opening vs Hyrax expander: A randomized clinical trial. Am J Orthod Dentofacial Orthop 2020; 157(1):7-18.
12. Massaro C, Garib D, Cevidanes L, Janson G, Yatabe M, Lauris JRP, Ruellas AC. Maxillary dentoskeletal outcomes of the expander with differential opening and the fan-type expander: a randomized controlled trial. Clin Oral Investig 2021; 25(9):5247-5256.
13. Massaro C JG, Miranda F, Aliaga-Del Castillo A, Pugliese F, Lauris JRP, Garib D. Dental arch changes comparison between expander with differential opening and fan-type expander: a randomized controlled trial. Eur J Orthod 2021; 43(3):265-273.
14. Krebs A. Expansion of the midpalatal suture, studied by means of metallic implants. Acta Odontol Scand 1959; 17(4):491-501.
15. Bazargani F, Feldmann I, Bondemark L. Three-dimensional analysis of effects of rapid maxillary expansion on facial sutures and bones. Angle Orthod 2013; 83(6):1074-1082.
16. Alves ACM, Maranhao OBV, Janson G, Garib DG. Mandibular dental arch short and long-term spontaneous dentoalveolar changes after slow or rapid maxillary expansion: a systematic review. Dental Press J Orthod 2017; 22(3):55-63.

THREE-DIMENSIONAL ANALYSIS OF MAXILLARY IMPACTED CANINE MOVEMENT USING TEMPORARY ANCHORAGE DEVICES (TADs)

Marco Migliorati

ABSTRACT

Impaction is defined as the failure of tooth eruption at its appropriate site in the dental arch, within its normal period of growth. The etiology can include local and systemic causes and impacted canines may remain asymptomatic for a long time; however, some of them may increase the risk of infection, cystic follicular lesions, and external root resorption of adjacent teeth. Cone beam computed tomography (CBCT) has become the gold standard for diagnosis and treatment planning when canine impaction occurs. CBCT analysis reduces the radiation dose compared to computerized tomography (CT) and provides coronal, sagittal, and axial views that allow the clinician to assess the three-dimensional (3D) location of the canine and estimate the degree of difficulty. Different therapeutic approaches have been reported with or without the use of temporary anchorage devices, including the canine first approach. Advantages of this approach rely on the possibility to minimize the risk of root resorption on adjacent teeth during treatment, decrease discomfort for the patient, and reduce treatment time as anchorage preparation is not needed. This chapter will analyze different anchorage approaches,, evaluating the movement of the impacted canines, root length changes, and anchorage loss. Techniques used to treat palatally displaced canines were the transpalatal arch (TPA), direct anchorage using only one miniscrew, and a TPA reinforced with miniscrew. The analyses were obtained by comparing CBCT superimpositions before treatment and 3 months later.

KEY WORDS: Impacted Canines, Temporary Anchorage Devices, 3D Analysis

INTRODUCTION

The objective of this chapter is to analyze the results of the use of a transpalatal bar connected to temporary anchorage devices (TADs) to treat palatally impacted canines. Diagnoses and treatment plans will be systematically presented for different approaches and anchorage systems that are currently available. 3D analyses report the movement and mechanics during correction of canine position and evaluate possible side effects on other teeth and the anchorage unit.

An impacted canine is defined as, a tooth retained in the maxillary or mandibular arch beyond the date of eruption, surrounded by its pericoronal sac and has no contact with the oral cavity" [1]. Other definitions have been proposed among clinicians, and considering the clinical aspect and the pathologic terms, impacted teeth can be defined as occurring when a tooth remains embedded in the oral mucosa or bone past its normal eruption period and is fully developed [2-4]. Other authors also associate this definition with the risk to adjacent teeth lesions - for example, extensive root damage.

Maxillary canine impaction has been widely studied in literature, both from an interceptive approach to prevent the impaction, and from a prognostic approach that can inform us about the duration of treatment, severity, and risks related to the treatment. Upper canines are commonly impacted teeth, second only to third molars [5].

The literature reports a prevalence of approximately 2% canine impaction in the population, and it is twice as common in females as in males. Lower canine impaction has a lower incidence and presents generally different aspects and prognoses. While the upper canines generally remain in their side of impaction, lower canines can often move in a horizontal plane and cross the midline, becoming a transposed impacted canine, with no chance of orthodontic correction.

Nearly 8% of impacted maxillary canines present as bilateral, aapproximately one-third of impacted maxillary canines are labially displaced, and the remainder are palatally displaced. Etiology of the impactions has been studied by several authors who describe it as multifactorial, with some characteristics that can help predict the risk of impaction. Jacoby's research found that 85% of palatally impacted canines had sufficient space for eruption, whereas only 17% of labially impacted canines had sufficient space. So, arch length discrepancy appears to be much more common for labially impacted canines [6-9].

Several etiologic factors for canine impactions have been proposed, including localized, systemic, or genetic. If we study impacted canines, we should remember the normal path they take during eruption. Broadbent conducted several studies in 1941 using a sample of 5000 children from the Bolton study. He was the first to describe the Ugly Ducking Stage, or the so-called Broadbent phenomenon, when the upper lateral incisors flare distally. This stage may persist for 3 or 4 years until the cuspids erupt. Broadbent, describing this stage, wrote that by the 7th year, the crown of permanent cuspids have been completed, but they have not yet moved far from their site of origin. Only "with sufficient increase in dimension in the size of the maxilla the cuspids move downward, forward and laterally away from the root ends of laterals" [10].

It is possible to describe different factors and many of them have been described earlier by Moyers [11]. In 1963, Moyers described the potential factors associated with canine impaction [11]. He summarized the etiology for impaction as being due to either: primary causes, (a) rate of root resorption of deciduous teeth, (b) trauma to the primary tooth bud, (c) disturbance in tooth eruption sequence, (d) availability of space in the arch, (e) rotation of tooth buds, (f) premature root closure, (g) canine eruption into the cleft area in cleft-palate individuals; or secondary causes, (a) abnormal muscle pressure, (b) febrile disease, (c) endocrine disturbances, and (d) vitamin D deficiency.

In 1976, Bishara et al. wrote an interesting paper on the management of impacted canines. In that paper they underlined the importance of diagnosis, in particular by means of x-ray and clinical evaluation. At that time, the images available were periapical film, panorex, and occlusal film [12]. They also described different treatment methods, and among these they described the use of the lower arch as anchorage unit, applying the force to the lower canine by the means of interarch elastics.

In 2004, Kokich et al. published an article describing the option of the "canine first approach", where the canine is allowed to erupt spontaneously after exposure. Kokich suggested that approach could have several advantages, among these "reducing time in orthodontic appliances". In fact, no anchorage preparation would be necessary and this allowed a spontaneous eruption with little to no risk to adjacent teeth lesions. He also described the surgical procedure: a key factor was that the teeth should be carefully evaluated during the procedure, including adequate bone removal over the crown of the impacted canine. All bone over the crown should be removed down to the cementoenamel junction. If not, slow or no movement could occur [13]. Later, he and his coauthors described the periodontal results of this approach, and they found that no differences in crestal bone height or probing attachment level were found around the previously impacted canines when compared with the control canines, even though a loss of probing attachment level was found at the distolingual aspect of the lateral incisor on the impacted side. They also evaluated roots length changes of lateral incisors and canines, and the roots of the previously impacted canine and adjacent lateral incisor were significantly shorter than those of the control canine and lateral incisor. The previously impacted canine was an average of 1.08 mm shorter (P = .025) than the control canine; the adjacent lateral incisor was an average of 1.87 mm shorter (P = .01) than the contralateral control lateral incisor [14]. It seems reasonable that approaching an impacted maxillary canine with this technique should be preferable, as Kokich stated that the advantages are many and the importance of reducing risk linked to particular circumstances is noteworthy, such as ankylosis or invasive cervical root resorption (ICRR).

Becker et al. in 2010 analyzed the failure in treatment of these teeth and summarized them in three groups: 1) Patient-dependent factors, 2) Orthodontist-dependent factors, 3) Surgeon-dependent factors. In the first group, we can include factors like abnormal morphology of the impacted tooth, age, resorption of the root of an adjacent tooth, and poor compliance with hygiene or appointments. In the second group of reasons that belong to the orthodontist, we include errors starting from the diagnostic stage (inclusion site mistaken) to the biomechanics applied. The latter is very important, since it can affect both the success of the canine correction and the side effects that occur to adjacent teeth. It is important to maintain 3D control of the canine, in in order to prevent or minimize iatrogenic damage. The third group includes all those issues related to the surgical procedure: diagnosis, inappropriate exposure, damage to other teeth or to the canine, and damage to the soft tissue [15].

Becker et al. reported ICRR as a possible cause of failure [16]. They described ICRR as “a rare, insidious, and aggressive form of external root resorption, also known as extracanal invasive resorption. Typically, it begins at a focal point on the surface of the root below the epithelial attachment in the clinical cervical area and progresses into dentin coronally, apically, and circumpulpally, halting only at the predentin layer surrounding the pulp. Typically, it does not involve the pulp” [16]. They reported that ICRR can be one of the most likely causes of canine impaction treatment failure, and generally it is overlooked. In this context, CBCT analysis can be considered for early diagnosis and to overcome all the consequences of possible ICRR.

Summarizing the advantages of the “canine first” approach, we can emphasize the following points: Reducing root damage of adjacent teeth; no time needed to create an anchorage unit with fixed appliances; safer treatment option in case of canine traction failure (i.e., ICRR, ankylosis); controlled biomechanics/force vectors planned on CBCT. The combined use of intraoral scans (STL files) and CBCT (DICOM files) can be helpful to plan specific appliances with optimal vectors of force to successfully treat impactions.

In the treatment planning, the orthodontist generally addresses issues like anchorage unit, through which vector of orthodontic forces will be established. The amount of force is generally between 50 and 70 grams and has an extrusive direction. Depending on the original canine position, other vectors may include distal, horizontal, or buccal. The extrusive force can be applied by the means of metallic coils, elastic chains or wire, or even B-titanium or stainless steel cantilevers.

Traditional anchorage units include multi-bracket fixed appliance, transpalatal arch or bar, and other maxillary fixed appliances. Anchorage in the lower arch has been described as well [12]. When a multi-bracket appliance is used, a preliminary stage of alignment and leveling is necessary until a sufficiently rigid arch wire can be placed. This could lead to an undesirable elongation of treatment duration and expose adjacent teeth to possible root resorption.

All these approaches have teeth as the anchor units, with significant undesirable movements if the anchor unit is not sufficient to provide the necessary resistance during treatment. The need to find a more efficient and reliable anchorage system was thus necessary considering the importance of anchorage and the problems that the traditional systems exhibit. Dental implants, introduced in the early 1950s thanks to the research of orthopedic surgeon P.I. Brånemark [17-19], have become one alternative to traditional anchorage.

The modern dental implant is a surgical component that interfaces with the bone of the jaw to support a dental prosthesis. After placement, dental implants become a unique functional unit with the bone through a biologic process called osseointegration. This is also referred to as secondary stability. Osseointegration is defined as the formation of a direct interface between an implant and bone, without intervening soft tissue. In other words, it is the intimate connection between the cells (like osteoblasts) and the endoessous implant. Douglas, in 1987, proposed dental implants as anchorage in orthodontic treatment [20], and other authors in the early 90s studied the possibility to use a dental, osseointegrated implant, as an anchorage unit [21-23].

Along with the increased use of dental implants in orthodontics, there has been an increased interest in finding implants with different sizes and shapes to achieve anchorage without traditional dental implant fixtures. The success of dental implants or other kinds of osseo-integrated devices depends on the ability to provide anchorage until the end of orthodontic treatment. In the literature, success rates of osseointegrated devices are fully documented with excellent long-term stability [24, 25]. Higuchi and Slack used osseointegrated implants in seven patients to move teeth during orthodontic treatment. In the above and in another study, implants remained stable throughout the treatment and procedures yielded good results [21].

In spite of their successful use, dental implants' size and cost, as well as the need for two extensive surgical procedures, have limited their use in orthodontics. Changes in implant design were desirable, as osseointegrated implants had several limitations, including postoperative pain, anatomical placement limits, and a design that is not ideal for orthodontic application (use of elastics, wires, or coils). For these reasons, modifications to the dental implant configuration were made with the aim to create a specific skeletal anchorage different from a dental implant. In some cases, the design was completely changed, creating a flat implant (disk like): the Onplant [26].

Successively, mini-implant anchorage systems have been developed and effectively used as an alternative to headgear and all other anchorage devices, dental implants included. However, mini-implants showed some limitations: the size of these devices impacts the available sites to be used, like the retromolar region, an edentulous area, or the midpalatal suture. Furthermore, they require a precise 2 stage protocol and a sufficient healing period lasting at least 2 months [27-30]. Therefore, specific systems were developed, with the miniscrews in use today typically being between 1.2 and 2mm in diameter and between 7 and 15mm in length [31, 32].

Miniscrews can be inserted and removed easily, are more affordable, and can be inserted at various maxillary and mandibular sites. TA knowledge of maxillary and mandibular bone characteristics is important when considering the use of miniscrews. Many terms have been used for these devices:

- miniscrew,
- mini-implant,
- micro-screw,
- temporary anchorage devices (TAD),
- orthodontic mini-implant (OMI),
- miniscrew implants (MSI).

Apart from the name being used, these fixtures are variations of surgical screws used for rigid fixation in maxillofacial surgery, and they solve many of the problems of the endosseus implants. The benefits include great flexibility regarding the placement sites, easy surgery (for placement and removal), low cost, and moreover, the possibility to be immediately loaded. Miniscrews, in fact, do not require osseointegration to be used as a good extradental, intraoral anchorage system. The success of miniscrews, in terms of allowing adequate support during orthodontic treatment, is related to the concept of primary stability. This is defined as a mechanical interface between the screw and the bone. Once the primary stability is lost, the miniscrew will be also. Key parameters to sustain stability have been studied as well as the concept of bone adaptation or bone relaxation. Tension, considered as the force keeping the miniscrew stable between bone and screw, is not stable over time, but changes due to the phenomenon of bone relaxation. Bone relaxation is the reduction of the tension at the trabecular level of the bone. This leads to a reduction in stability in the early stages after miniscrew placement [33, 34].

Immediate loading and a correct insertion torque management reduces the risk of failures. In this perspective, TADs could be significantly useful to manage palatally displaced canines, either for direct or indirect anchorage. Direct anchorage includes all those approaches where the force is directly connected to the miniscrews - elastics, coil, or cantilever. Indirect anchorage usually means connecting anchorage teeth to the miniscrew, and then connecting a force between the anchorage teeth and the teeth to be moved.

PATIENTS AND METHODS

A study of the 3D movements of impacted canines using different anchorage units was conducted. The anchorage units evaluated were a traditional transpalatal bar (TPA), a miniscrew reinforced TPA, and direct anchorage to temporary anchorage devices (TADs). The first part of the study was conducted on a group of patients who had the following inclusion and exclusion criteria:

Inclusion criteria: presence of one or two impacted maxillary canines requiring surgical exposure and orthodontic treatment.
Exclusion Criteria: permanent teeth extraction-based treatment, current or previous orthodontic treatment in the last 12 months, current systemic disease, current antibiotic, or anti-inflammatory therapy that could possibly compromise the results.

The study was designed as a randomized clinical trial conducted with two parallel groups with an allocation ratio of 1:1. The trial was registered at www.clinicaltrials.gov with registration number NCT01717417. As reported in the article published in 2021, two interventions were compared. The first group of patients received a TPA as anchorage unit for canine traction while the second group received a TAD as anchorage unit [35].

In the TAD group, one or two 8 mm long miniscrews (Orthoeasy, Forestadent, Pforzheim, Germany) were used as anchorage. The anchorage was indirect, using the molars or transplatal bar anchored to the mini-screw. In both groups, the approach to solve the impaction was “canine first”: no anchorage preparation was performed besides the TPA or the miniscrew. The biomechanics approach included the use of a Beta-titanium cantilever applying a force of 50–60g measured with a pen gauge; biomechanics varied among the patients including extrusive and distalizing vectors. The miniscrew insertion sites varied depending on the impacted canine position, as well as the cantilever in the TPA group. The day of the surgical exposure of the canine was coincident with the beginning of traction. Close surgical intervention technique was performed for all included patients. Local anesthesia was performed before miniscrew insertion, and no pre-drilling was performed. All the miniscrews were inserted directly thorough the mucosa.

All patients received two CBCTs, one before treatment and one 3 months after the treatment started, as reported in the experimental protocol approved by the ethics committee, San Martino Hospital, Genova, Italy, and in the patient's consent form.

The image assessment went through several steps in order to evaluate the speed, direction, and amount of movement between the impacted canines anchored on a TPA and the impacted canines indirectly anchored on a miniscrew. The linear displacement of the canine was divided by the observation period in weeks or months to obtain the rate of tooth movement. The first step included a standardization of the CBCT volumes and data. Due to variations in the CBCT image acquisition protocol, the "Downsize" tool in Slicer was utilized to standardize the image resolution and avoid any heterogeneity of the imaging data. All scans were reformatted to a 0.5 mm^3 voxel from the original scans of 0.4 mm^3 voxel size using SlicerCMF version 4.0 (https://sites.google.com/a/umich.edu/dentistry-image-computing/) to standardize the scan resolution and decrease the computational power and time for image registration. The second step included the creation of a virtual model converting the Digital Imaging and Communications in Medicine (DICOM) file into a Guy's Image Processing Lab (GIPL) format. Three dimensional models from T0 and T1 were created using ITK-SNAP open-source software. This process, called segmentation, required outlining the shape of the dental arches visible in the slices and setting up a threshold of the tissue density to select the anatomical structures of interest.

The next step was registration. This step defined stable reference structures following the maxillary regional registration methods validated by Ruellas et al. [36]. In this particular case, we used the maxillary bone as a reference for superimposition due to the short observation period between T0 and T1. The registration method compared voxel by voxel gray level CBCT images, and calculated the rotation and translation parameters between the 2 time point images. The process is fully automated, even though a preliminary manual alignment of the two CBCTs was performed using using the CMF registration module in SlicerCMF (https://sites.google.com/a/umich.edu/dentistry-image-computing/). This step was fundamental to establish that the two volume images were placed in the same coordinate system, thereby allowing measurements and comparison. As reported in Migliorati et al. [35], the next step was the Overlay of the 3D models and quantitative evaluation. This was performed using VAM software (VAM v. 3.7.6, Canfield 113 Scientific Inc., Fairfield, NJ), overlaying the registered 3D models. In this way, it was possible to evaluate the displacement of each canine and to measure the amount of movement. In particular, we evaluated the distances between the tip of the cusp of the canine from T0 and T1, and the distances between the apex of the root of the canine from T0 and T1. All distances were calculated in mm (Figure 1).

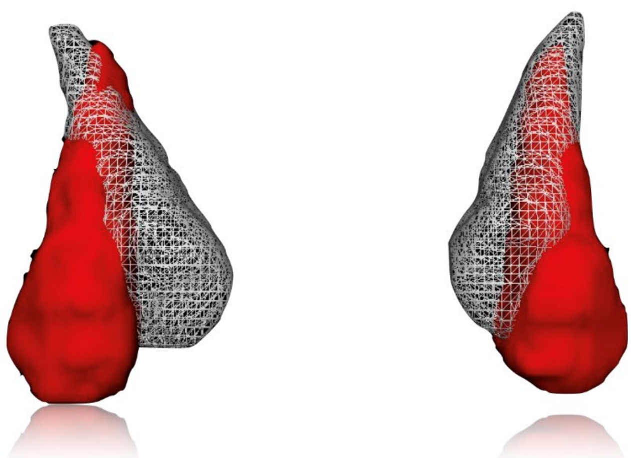

Figure 1. Results of CBCT superimposition of palatally displaced canine before and after second CBCT.

Error of the method was evaluated for each measurement taken by the same operator 1 month after the first evaluation. Intraclass correlation coefficient (ICC) was calculated both for the apex and canine tip distances. ICC values were 0.87 and 0.88 for canine tip and canine root apex, respectively.

Statistical analysis

Descriptive statistics are expressed as median and inter-quartile ranges. The data were tested for normality using the Shapiro-Wilk test. The nonparametric Spearman's rank correlation test was used to evaluate the dependence among the measured characteristics. The nonparametric Mann-Whitney U test was used to evaluate differences between groups. Differences with a p-value less than 0.01 were selected as significant and data were acquired and analyzed using R v3.4.4 software environment [37].

In the first study, tooth anchorage versus TADs anchorage was compared, and a total of 22 patients (12 female, 10 male, mean age: 13.4 years, SD 2.4) undergoing orthodontic treatment for impacted maxillary canines (both labial and palatal) were recruited. No differences were observed between groups for apex displacement, tip displacement, or observation timespan, and no correlations were found between apex displacement and observation timespan, or patients age. No correlations were found between tip displacement and observation timespan, or patient age. An apex root movement of 0.4–0.8mm per month was found, while average canine tip movement ranged between 1.1 mm and 2.0 mm per month. No miniscrew failures were observed.

These values are very similar to those reported in the literature when a canine is moved with multi-bracket appliances during space closure. In fact, one article reported cuspid movement in extraction cases to be between 1.0-2.4mm/month [38]. These similar values of tooth movement indicate that properly calibrated forces during traction allow significant dental movement even when a tooth is impacted. The total amount of canine movement between the two groups was similar and not statistically different; a total apex movement of 4.4mm and 5.1mm were found for the TAD and TPA groups, respectively. Larger differences were found with respect to total cusp movement, but again, the statistical difference was not significant. Many aspects could affect the total amount of movement, and one of these is the bone quality of each patient.

Successively, we evaluated the first molar movement, by superimposing the T0 and T1 patient's CBCT, and we found approximately 40% anchorage loss in the sagittal dimension with dental anchorage (Figure 2). These side-effects are consistent with other results reported in literature when dental anchorage is used to retract canines during space closure treatment. Anchorage loss with miniscrews resulted in clinically insignificant loss, even though 15% anchorage loss in the sagittal dimension was found for TAD group patients. These preliminary findings alllowed us to better understand the efficiency of these two anchorage units during treatment and highlighted the rate of movement of impacted canines.

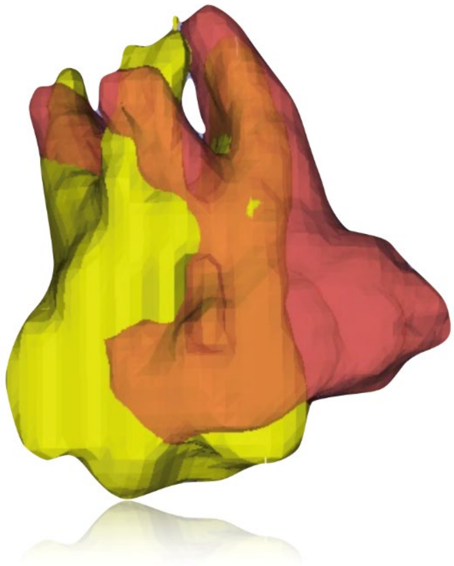

Figure 2. First molar anchorage loss evaluation by superimposition of two consecutive CBCTs using as reference structure the maxilla. The mean anchorage loss for teeth anchorage unit is approximately 40%.

For the second comparison, two other groups were compared - a full-digital miniscrew anchored TPA (mTPA) (Figure 3) versus direct anchorage on a single miniscrew. The experimental flowchart was similar to the previous comparison and allowed the superimposition of two consecutive CBCTs of the patient's group. Moreover, in this part of the analysis, it was also possible to evaluate root length changes and movement of adjacent teeth, such as the first premolars and lateral incisors.

The overall dental movement measured was similar to the previous analysis, with apex root movement of 0.9mm per month, while average canine tip movement was 1.5 mm per month, with no differences between groups with respect to the anchorage unit. Root lengths didn't change, as expected, confirming no effect on adjacent teeth during the traction period, and all miniscrews remained stable during the treatment.

Figure 3. mTPA. Trans-palatal bar minscrew reinforced. The structure was 3D printed using laser-melting technique and after printing the structure was polished.

Digital planning

All the mTPA structures were digitally planned. The first stage of the digital planning is the insertion site of the minsicrews. Thanks to dedicated software, it is possible to superimpose an intraoral scan (STL file) with a DICOM file from the patient's CBCT. Once superimposed, miniscrews can be uploaded in the 3D image, and the clinician can carefully place the anchorage device, maintaining adequate distances from the roots and from the canine. Generally, the most suitable position is between the root of the first molar and the second premolar, even though the palatal median suture can also be used.

Once the position is planned, the STL file can be used to create the digital TPA that can be 3D printed with the laser melting technique or laser-sintering technique. Once the raw piece comes to the office or the technician, it must be treated and polished (Figure 3). Meanwhile, the technician can create a surgical guide for miniscrew insertion. These kind of guides can be either full 3D printed or created by thermoforming 2.5mm thick PETG discs. The thermoformed sheet has to be cropped in the middle to leave space for the screws. Afterwards, screws analogues are inserted, and the metal sleeves placed on the analogue's head, together with the blade used to insert the miniscrews. Resin fixes the metal sleeves to complete the guide (Figure 4).

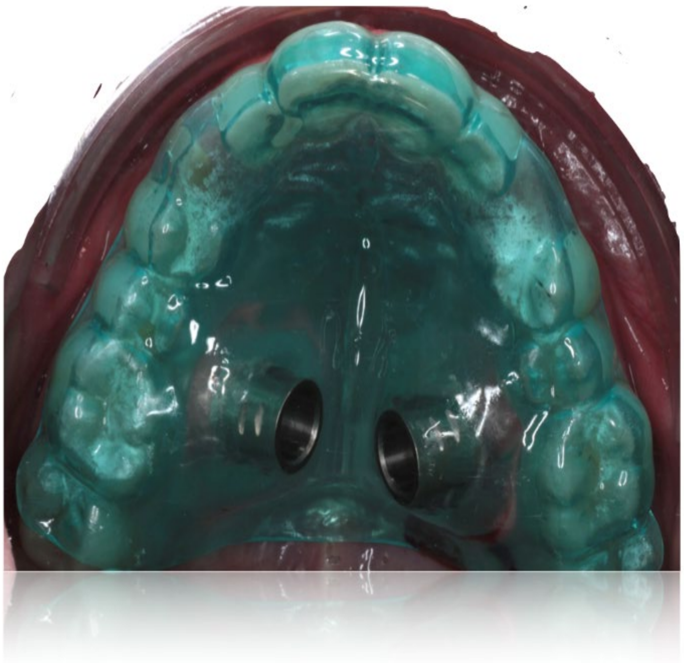

Figure 4. Surgical guide obtained by thermoforming 2.5mm thick PETG discs. Intraoral view before miniscrew insertion.

This workflow allows the clinician to receive both the surgical guide and the device to be placed in the patient mouth, the so-called "one visit protocol". The workflow precision has been investigated in respect to the miniscrew position, from the planned position to the achieved position in the patients' mouth. By using superimposition techniques, it was possible to estimate the miniscrew position error. The first lack of accuracy was between the planned position and the ones evaluated on the 3D printed, even though the angular discrepancy was under 2°. The angular differences between the planned position and intraoroal position were higher, but didn't exceed 6.8°. These values are clinically not significant and indicate a good reliability of the workflow with respect to the digital planning.

CONCLUSIONS

Palatally impacted canines remain a challenging situation to be treated by orthodontists, since anatomical, biomechanical, and patient difficulties must be addressed. As Kokich described, a “canine first” approach can be preferable, reducing the risk of undesirable side-effects. The rate of movement of impacted canines was found to be approximately 1-1.5mm per month, and these values are similar to normal dental movement of non-impacted canines. The anchorage unit needs to be carefully evaluated, planned and managed: a miniscrew anchored unit could reduce the risk of anchorage-loss and can be helpful during overall orthodontic treatment. The digital approach to planning the anchorage appliance and surgical guides are noteworthy developments for clinicians in daily practice.

REFERENCES

1. Delsol L, Orti V, Chouvin M, Canal P. Canines et incisives maxillaires incluses: diagnostic et thérapeutique. Odontologie. 2006. doi:10.1016/S1283-0860(06)44146-4.
2. Andreasen JO, Petersen JK, Laskin DM. Textbook and Color Atlas of Tooth Impactions. Copenhagen, Denmark: Munksgaard; 1997:126-166.
3. Becker A. Orthodontic treatment of impacted teeth. 2nd ed. Florida: CRC Press; 2007.
4. Lindauer SJ, Rubenstein LK, Hang WM, Andersen WC, Isaacson RJ. Canine impaction identified early with panoramic radiographs. J Am Dent Assoc 1992;123(3):91-97.
5. Litsas G, Acar A. A review of early displaced maxillary canines: etiology, diagnosis and interceptive treatment. Open Dent J 2011; 5:39–47.
6. Bishara SE. Impacted maxillary canines: A review. Am J Orthod Dentofacial Orthop 1992; 101(2):159-171.
7. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Eur J Orthod 1988;10(4):283–295.
8. Mitchell L. An Introduction to Orthodontics, 3rd edition. Eur J Orthod 2007; 29(5):542.
9. Jacoby H. The etiology of maxillary canine impactions. Am J Orthod 1983; 84(2):125-132.
10. Broadbent BH. Ontogenic development of occlusion. Angle Orthod 1941; 11:223-241.
11. Moyers, R. E. Handbook of Orthodontics, 2nd ed. Chicago: Year Book Medical Publishers, Inc.; 1963:83-88.
12. Bishara SE, Kommer DD, McNeil MH, Montagano LN, Oesterle LJ, Youngquist HW. Management of impacted canines. Am J Orthod 1976, 69(4):371-387.
13. Kokich VG. Surgical and orthodontic management of impacted maxillary canines. Am J Orthod Dentofacial Orthop 2004; 126(3):278-83.
14. Schmidt AD, Kokich VG. Periodontal response to early uncovering, autonomous eruption, and orthodontic alignment of palatally impacted maxillary canines. Am J Orthod Dentofacial Orthop 2007; 131(4):449-455.
15. Becker A, Chaushu G, Chaushu S. Analysis of failure in the treatment of impacted maxillary canines. Am J Orthod Dentofacial Orthop 2010; 137(6):743-754.
16. Becker A, Abramovitz I, Chaushu S. Failure of treatment of impacted canines associated with invasive cervical root resorption. Angle Orthod 2013; 83(5):870-876.
17. Branemark PI, Adell R, Breine U, Hansson BO, Lindstrom J, Ohlsson A. Intra-osseous anchorage of dental prostheses. I. Experimental studies. Scand J Plast Reconstr Surg 1969; 3(2):81.
18. Albrektsson T, Branemark PI, Hansson HA, Lindstrom J. Osseointegrated titanium implants. Requirements for ensuring a long-lasting, direct bone-to-implant anchorage in man. Acta Orthop Scand 1981; 52(2):155-170.
19. Branemark PI. Osseointegration and its experimental background. J Prosthet Dent 1983; 50(3):399-410.

20. Douglass JB, Killiany DM. Dental implants used as orthodontic anchorage. J Oral Implantol 1987; 13(1):28-38.
21. Higuchi KW, Slack JM. The use of titanium fixtures for intraoral anchorage to facilitate orthodontic tooth movement. Int J Oral Maxillofac Implants 1991; 6(3):338-344.
22. Stean H. Clinical case report: an improved technique for using dental implants as orthodontic anchorage. J Oral Implantol 1993; 19(4):336-340.
23. Huang LH, Shotwell JL, Wang HL. Dental implants for orthodontic anchorage. Am J Orthod Dentofacial Orthop 2005; 127(6), 713-722.
24. Adell R, Lekholm U, Rockler B, Branemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg 1981; 10(6):387-416.
25. Clementini M, Morlupi A, Canullo L, Agrestini C, Barlattani A. Success rate of dental implants inserted in horizontal and vertical guided bone regenerated areas: a systematic review. Int J Oral Maxillofac Surg 2012; 41(7):847-852.
26. Block MS, Hoffman DR. A new device for absolute anchorage for orthodontics. Am J Orthod Dentofacial Orthop 1995; 107(3):251-258.
27. Wehrbein H, Feifel H, Diedrich P. Palatal implant anchorage reinforcement of posterior teeth: prospective study. Am J Orthod Dentofacial Orthop 1999; 116(6):678–686.
28. Wehrbein H, Merz BR, Diedrich P, Glatzmaier J. The use of palatal implants for orthodontic anchorage. Design and clinical application of the orthosystem. Clin Oral Implants Res 1996; 7(4):410-416.
29. Costa A, Raffainl M, Melsen B. Miniscrews as orthodontic anchorage: a preliminary report. Int J Adult Orthodon Orthognath Surg 1998; 13(3):201-209.
30. Kanomi R. Mini-implant for orthodontic anchorage. J Clin Orthod 1997; 31(11):763-767.
31. Wehrbein H, Glatzmaier J, Yildirim M. Orthodontic anchorage capacity of short titanium screw implants in the maxilla. An experimental study in the dog. Clin Oral Implants Res 1997; 8(2):131-141.
32. Ohmae M, Saito S, Morohashi T, Seki K, Qu H, Kanomi R, Yamasaki KI, Okano T, Yamada S, Shibasaki Y. A clinical and histological evaluation of titanium mini-implants as anchors for orthodontic intrusion in the beagle dog. Am J Orthod Dentofacial Orthop 2001; 119(5):489-497.
33. Migliorati M, Drago S, Gallo F, Amorfini L, Dalessandri D, Calzolari C, Benedicenti S, Silvestrini-Biavati A. Immediate versus delayed loading: comparison of primary stability loss after miniscrew placement in orthodontic patients-a single-centre blinded randomized clinical trial. Eur J Orthod 2016; 38(6):652-659.
34. Migliorati M, Drago S, Amorfini L, Nucera R, Silvestrini-Biavati A. Maximum insertion torque loss after miniscrew placement in orthodontic patients: A randomized controlled trial. Orthod Craniofac Res 2021; 24(3):386-395.
35. Migliorati M, Cevidanes L, Sinfonico G, Drago S, Dalessandri D, Isola G, Biavati AS. Three dimensional movement analysis of maxillary impacted canine using TADs: a pilot study. Head Face Med 2021; 17(1):1.
36. Ruellas AC, Huanca Ghislanzoni LT, Gomes MR, Danesi C, Lione R, Nguyen T, McNamara JA, Jr., Cozza P, Franchi L, Cevidanes LH. Comparison and reproducibility of 2 regions of reference for maxillary regional registration with cone-beam computed tomography. Am J Orthod Dentofacial Orthop 2016; 149(4):533-542.
37. R Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna. https://www.R-project.org. Accessed Mar 2020.
38. Iwasaki LR, Liu Y, Liu H, Nickel JC. Speed of human tooth movement in growers and non-growers: Selection of applied stress matters. Orthod Craniofac Res 2017; 20(Suppl 1):63-67.

VERTICAL CHALLENGES WHEN TREATING PATIENTS WITH BILATERAL CLEFT LIP AND PALATE

Marilia Yatabe

ABSTRACT

Achieving long-term goals of the oral rehabilitation of patients with bilateral cleft lip and palate is challenging for healthcare providers. Primary interventions used during infancy are designed to establish function and esthetics early. Yet, these primary interventions may negatively influence the maxillary growth and development throughout the years, resulting in the need for maxillary surgical advancement to improve the negative sagittal discrepancy. This chapter reviews the surgical and non-surgical approaches to improving the position of the premaxilla in patients with bilateral cleft lip and palate.

KEY WORDS: Bilateral Cleft Lip and Palate, BCLP, Premaxilla

INTRODUCTION

BILATERAL CLEFT LIP AND PALATE (BCLP)

Cleft of the lip and palate is the most common craniofacial anomaly in humans, with a prevalence of 1:2,000 births worldwide and 1:600 in the US [1, 2]. It is characterized by the lack of fusion of the maxillary and frontonasal processes, leading to the different magnitude and severity of defect: unilateral or bilateral, complete or incomplete, cleft lip, alveolus, and/or palate [3]. The most severe type of cleft is the complete bilateral cleft lip and palate (BCLP), accounting for 10% of the cases [4]. In these cases, the lack of fusion results in a 3-piece segmented maxilla (Figure 1). The lateral segments of the maxilla and the premaxilla represent the most challenging portion of the oral rehabilitation.

Figure 1. Intra-oral occlusal picture of the maxilla of a baby with unrepaired bilateral cleft lip and palate.

The craniofacial morphology is characterized by a prominent premaxilla, a retrognathic maxilla, reduced posterior maxillary height, and a small, retruded mandible [5, 6]. The premaxilla develops from the sixth week of intrauterine life and reaches severe proportions within 4 weeks. It has been proposed that multiple factors lead to a prominent premaxilla, including the septo-premaxillary ligament, unrestrained anterior nasal septal growth, the abnormal direction of alveolar growth, lack of bony and soft tissue continuity, disruption of the balance between circum-oral musculature and tongue, and underdevelopment of the maxillary segments [7-11]. After birth, the prominence may increase from the overgrowth of the vomero-premaxillary suture [12] and/or the tongue activity against the flexible and relatively unsupported premaxillary bone [4, 7, 12]. The anterior nasal spine adapts to the anterior border of the nasal septum, the alveolar portion is lingually rotated, and the incisors assume a more vertical or retroclined inclination [11] (Figure 2).

Figure 2. Intra-oral frontal view of a premaxilla excessively extruded.

The protruded premaxilla may lead to psychological problems, functional problems, absence of proper anterior occlusion, lateral mobility of the premaxillary segment, labial or palatal fistula, and speech and oral hygiene problems [10]. In addition, the nostrils are stretched, and the tip of the nose is broad. The columella appears to be shortened or nonexistent, and the prolabium often seems to be joined directly to the tip of the nose [13] (Figure 3).

The standard oral rehabilitation for patients with BCLP starts very early in life with primary plastic surgeries – typically at 3 months of age – to repair the cleft lip and improve facial esthetics and function as it helps with sealing during breast or bottle feeding. It is followed by the palatal surgery at 12 months of age to close the oral-nasal communication involving the embryologic secondary palate and anatomic repair of the musculature within the soft palate, which is important for normal production of speech [14].

Figure 3. Extra-oral view of a premaxilla excessively protruded and deviated to the left before the primary surgeries. Note the short columella length.

The mid-term and long-term unintended consequences of these primary surgeries are a topic of interest for most researchers in the field, as they may compromise the maxillary development and/or growth throughout adulthood. Previous research reported that these surgical procedures could affect the position of the premaxilla as early as 4 years of age, with a negative overjet increasing significantly [6, 7]. The premaxillary position can vary significantly. While some researchers reported that a severe protrusion of the premaxilla at 6 years of age could reduce to almost normal at 16 years of age [7], others have suggested that the premaxilla becomes retrusive after 12 years of age [15].

In adult patients who have not had surgery, the combination of an extremely prominent premaxilla and a smaller mandible resulted in a great facial convexity and vertical growth pattern, resulting in an obtuse gonial angle and a long anterior lower face height, reduced posterior facial height, and a tendency toward retroclination of incisor teeth in both jaws [5].

The importance of the vomero-premaxillary suture

The premaxilla develops from the frontonasal process and comprises three parts: the alveolar part with the facial process, which encompasses two pairs of incisors; the palatine process; and the *processus stenonianus* that fuses with the nasal septum and the vomer through the vomero-premaxillary suture (Figure 4) [16]. Under normal conditions, the premaxilla would be controlled by the forward growth of midline structures and the lateral processes [15], excessive cartilage apposition at the vomero-premaxillary junction [6, 12], and the alveolar bone apposition associated with the development and eruption of the central incisors [11]. Its forward position could be associated with the overgrowth of the premaxillary-vomerine suture and/or could be an illusion created by the underdevelopment segments of the palatal shelves and mandibular retrognathian [6]. Vertical and sagittal excess of the premaxilla is induced by an overgrowth at the premaxillary-vomerine suture [6, 17]. In addition, the "septo-premaxillary ligament" composed of fibers from the nasal septum to the premaxilla may be an important factor related to the anterior displacement of the premaxilla, as it appears to be the means for the transmission of septal growth force to the maxilla [18].

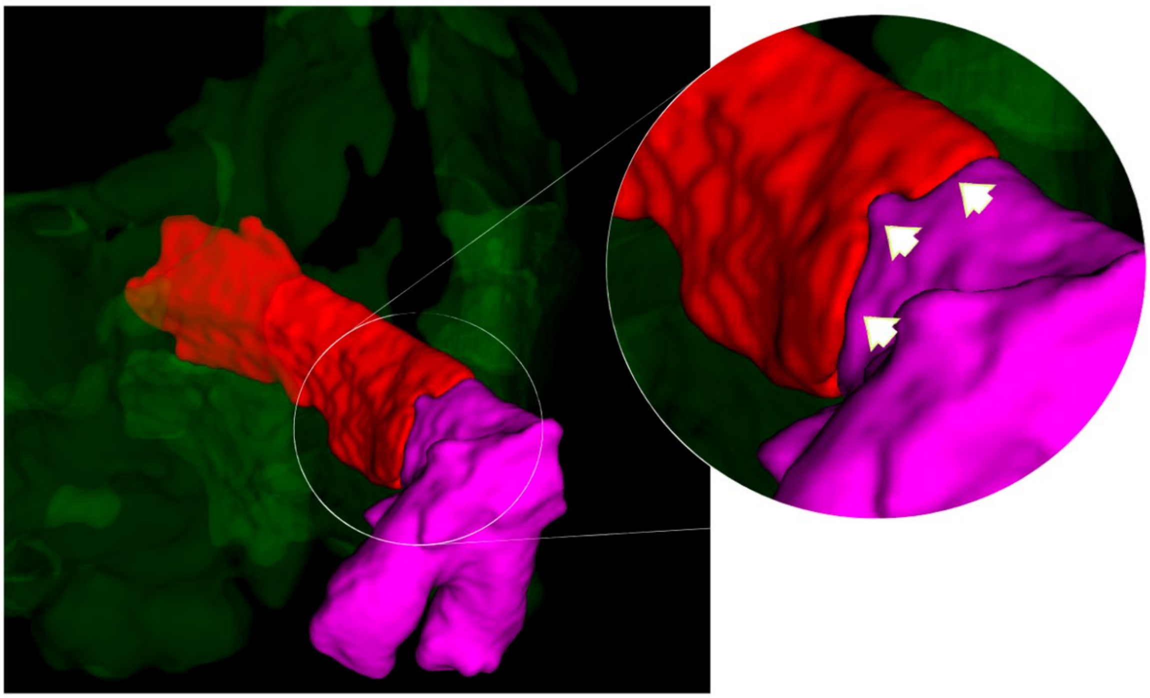

Figure 4. Illustration from the premaxilla in pink, the vomer in red, and the vomero-premaxillary suture indicated by white arrows.

The bilateral alveolar cleft causes the premaxilla to be mobile from birth and only apically fixed to the vomer bone [19]. The additional deposition of cartilage in the premaxillary-vomerine junction could contribute to the forward position of the premaxilla as well as to its considerable mobility [20]. Functional and cosmetic disorders result. The cleft *orbicularis* muscle lacks sphincter function and therefore does not restrain the protruding premaxilla. Sometimes the whole segment is rotated [19].

Potential therapies

Patients with BCLP have more difficulty achieving good results due to a significant lack of bone and soft tissue in the cleft area, severe protrusion of the premaxilla, maxillary segments atresia, excessive premaxilla mobility, and large bucconasal fistulas [21]. Regarding the premaxilla, its vertical excess does not seem to improve during growth, leading to an unesthetic appearance and a psychosocial problem. Also, performing an alveolar bone graft surgery can be challenging when the maxillary segments exhibit different vertical levels [22]. There are many controversies over the treatment of BCLP patients, including the need for preoperative orthopedic treatment, timing of palatal closure, timing of bone grafting, and possible osteotomy of the premaxilla [23]. On the other hand, opponents of this procedure advocate that injuring the premaxilla–vomerine suture may retard the growth of the maxilla and vomeral growth, resulting in a hypoplastic maxilla, an obtuse nasolabial angle, a short columella, and poor speech results, or combinations of these [23-25].

Figure 5. Extra-oral and intra-oral pictures of a patient with bilateral cleft lip and palate that had the premaxilla excised during infancy. Note the extremely deficient midface in the extra-oral photos, severe sagittal discrepancy, and the collapsed lateral segments of the maxilla.

1. SURGICAL INTERVENTIONS

1a. *Resection of the premaxilla* – an invasive and radical procedure performed in cases with extreme premaxillary projection to improve outcomes of the primary lip surgery, especially in underdeveloped countries where families do not have a good health support system to follow up with patients. It leads to deleterious long-term effects, such as severe concave profile, lack of support for upper lip and nose, disturbance in archform, and impediments to masticatory function and speech (Figure 5) [21, 26].

1b. *Surgical repositioning* - Advocates of surgical repositioning claim that regardless of the premaxilla projection, patients with BCLP are potential candidates for orthognathic surgery, and therefore, it is not advantageous to wait for the maxilla to grow completely or to expect that orthodontic treatment to significantly improve the problem [21]. The bone compression theory explains that when an osteotomy site is compressed by the application of external force, it takes advantage of bone resorption with osteolysis and bone remodeling [22, 24].

Defendants of surgical repositioning of the premaxilla have suggested that if the vertical overbite is more than +4 mm or less than −2 mm, an osteotomy of the premaxilla is justified. This applies to every negative sagittal relationship of the premaxilla and the reverse torque position. If the premaxilla is rotated [23], it does not result in a retrusive maxillary position at adolescence (14-16y) [4]. Considering that the premaxillary–vomerine junction is a growth center for the maxilla, nose, and alveolar arches, injury should be avoided during surgical positioning of the premaxilla. Therefore, the literature suggests that an osteotomy anterior to the premaxillary–vomerine suture is a safe procedure; although, removing this area was associated with midfacial growth arrest [23]. However, these procedures should be done only in selective cases, since this procedure may cause necrosis of the segment and damage to the tooth buds or teeth as a result of traumatized blood supply [25].

INFANCY

The surgical reposition of the premaxilla may be performed to allow primary lip closure, avoid excessive wound tension, and set back the premaxilla in a more favorable position [4, 15, 27]. However, it is known to have a significant negative impact on maxillary growth and profile development in the long term, and therefore it is not often used today [10, 19].

CHILDHOOD

During childhood, before 8 years of age, repositioning of the premaxilla may be performed to avoid psychosocial and traumatic troubles related to the premaxilla protrusion [4, 23]. The main challenge at this stage is to achieve stable fixation of the osteotomized segment. Some techniques and devices have been recommended for fixation, such as miniplate, interdental wiring, arch bar, orthodontic brackets, and occlusal splints. After using these fixation methods, clinicians may be confronted with adequate stabilization and noticeable movements of the premaxilla [4].

Considering that approximately 96% of cranial growth and 65% of facial structures have been completed between 5-10 years of age [23], the literature suggests the end of this period would be an advantageous time for a combination of early alveolar bone graft with premaxillary osteotomy. It would lead to a positive result in optimal positioning and fixation of the premaxilla, residual bone height, the ability to guide the permanent teeth through the bone graft, and closure of residual oronasal communications [23, 28]. Even though there is a trend towards maxillary growth retardation in both vertical and sagittal dimensions after early secondary closure, this is partially compensated by orthodontic and dental treatment [23, 25].

A late secondary osteotomy has similar goals, but the surgery is postponed in order to minimize the maxillary growth impact [4, 10, 17, 25]. Similar to what was previously described, delayed osteotomy of the premaxilla associated with the secondary alveolar bone graft allows for vertical improvement of the premaxilla in relation to the occlusal plane, closure of large fistulas, increases successful alveolar bone graft, facilitates the improvement of overjet and overbite, and allows for spontaneous eruption and migration of permanent teeth through the graft [4, 10, 17, 21, 23, 28]. Most times, orthodontic treatment is indicated to expand and align the maxillary segments prior to premaxillary osteotomy with an alveolar bone graft. Additionally, the extraction of primary or supernumerary teeth in the cleft site/flap region should be completed 6-8 weeks prior to surgery to facilitate wound healing. Occlusal splints may be constructed and delivered during surgery to stabilize the premaxilla, especially if there is a potential trauma from the anterior occlusion that may hinder the post-operative stability and lead to non-union of the sites (Figure 6) [10].

Figure 6. Intra-oral frontal and occlusal view of the maxilla before and after the surgical reposition of the premaxilla. Before the surgical reposition, the premaxilla was severely rotated, blocking the right segment of the maxilla. Once its position improved, the orthodontist expanded the maxilla and improved its archform.

ADULTHOOD

Ideally, the oral rehabilitation of patients with BCLP will be completed by adulthood. But in special circumstances or underdeveloped countries, it is possible to perform surgical repositioning later in adulthood, and the objective is to achieve both functional and esthetic results [4, 28].

Surgical interventions in summary:

Indications [10]:

- Vertical overdevelopment of the premaxilla
- Lateral displacement of the premaxilla
- Severely protrusive premaxilla
- Discrepant gingival level of the premaxilla and the lateral maxillary segments

Contra-indications [10]:

- When no orthodontic treatment is available
- Blood supply risk from previous scarring
- Inadequate dental eruption, which can compromise proper fixation/stability of the splint
- Non-compliant patient/parent – splint must be in place for 5-6 weeks post-op

Benefits [4, 10, 15, 17, 19, 28]:

- Combine with alveolar bone graft surgery – it supports teeth and dental implants, stabilizes the alveolar ridge, provides bony support and favorable periodontal health to teeth adjacent to the alveolar cleft, facilitates eruption of permanent teeth, closes residual fistulas, and supports the alar base of the nose
- Nasal closure can be performed more accurately
- Better surgical access
- Better position of the premaxilla, avoids extensive orthodontic treatment
- Dental arch can be aligned and stabilized
- Better inclination (torque) of the incisors
- Decreased relapse

Complications [4, 19]:

- Dehiscence of the wound
- Recurrent fistulas
- Resorption of grafted bone
- Instability of the premaxilla
- Necrosis or loss of the premaxilla
- Secondary growth impairment

2. NON-SURGICAL INTERVENTION

One of the theories that may explain the effects of the orthopedic approach is the sutural contraction osteogenesis [22, 24]. According to this theory, the application of external compression force may induce bone formation, remodeling, or both inside a craniofacial suture. And similar to the surgical intervention, it can also be performed at different stages of life:

INFANCY

Nasoalveolar molding is a procedure performed within the first months of life because the structures are more pliable, and therefore the movements occur faster [15]. The purpose is the correction of nasal cartilage deformity, stretching the nasal mucosal lining, and achieving non-surgical elongation of the columella [17, 29]. The intra-oral device can be adjusted weekly to facilitate the alignment and approximation of the alveolar segments while simultaneously achieving correction of the nasal cartilage and soft tissue deformity (Figure 7). It is maintained with surgical tape applied to the cheeks and cleft lip segments.

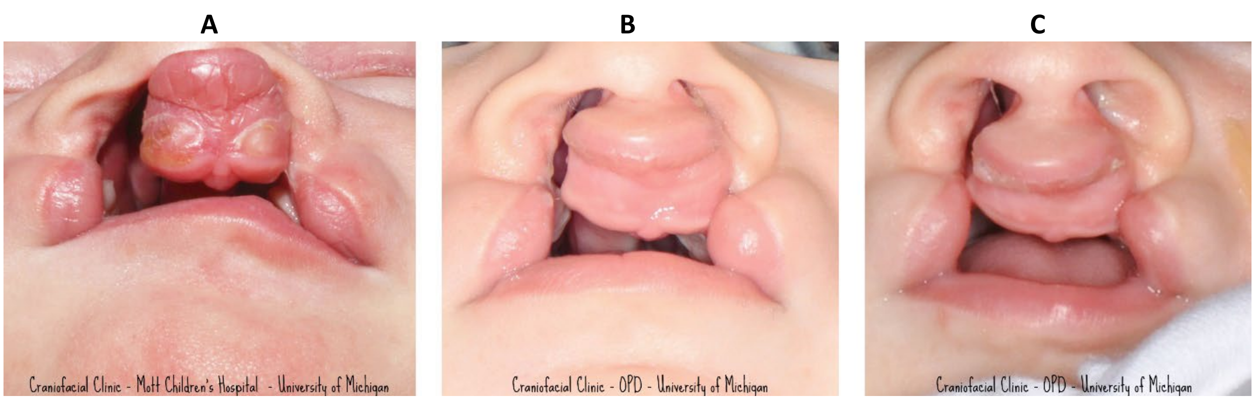

Figure 7. Extra-oral frontal photos of a baby's bilateral cleft lip and palate: A) initial, B) 9 weeks, and C) 12 weeks of nasoalveolar molding to improve the premaxillary position, nasal symmetry, and columella elongation.

Advocates of this therapy claim that there are no complications and there is minimal effect on maxillary growth [19]. The short-term outcomes have been extensively published, and the main advantage is in relation to approximating and lengthening the soft tissue, with the following other advantages [29]:

- achievement of surgical soft tissue repair under minimal tension
- optimal conditions to minimize scar formation.
- reduction in the number and complexity of revision surgeries
- long-term retention of nasal symmetry
- avoid surgery for columella elongation

The disadvantages are related to the increased burden of care for parents due to the weekly visits necessary and the long-term effect on the development and potential restriction of the maxillary growth [30].

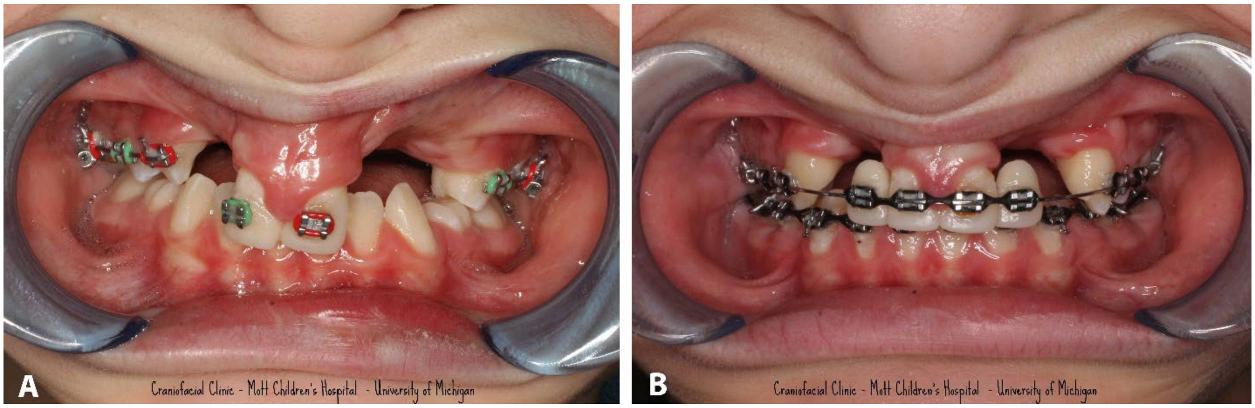

Figure 8. Intra-oral frontal view of a vertically displaced premaxilla before (A) and after (B) non-surgical reposition of the premaxilla anchored on teeth with partial fixed appliances (courtesy of Dr. Owinga, personal communication).

CHILDHOOD

Once the incisors are occluding, the maxillary anterior teeth and the premaxilla tend to come forward by accompanying the mandibular growth [15]. Successful orthopedic treatment to reposition the premaxilla can be obtained with different methods, including modified plates with bonded brackets and tubes to the acrylic, full fixed appliances, temporary skeletal anchorage device, intra-oral appliances, or even headgear [31]. The desired outcomes include a normalized occlusion plane (Figure 8), dental intrusion of the maxillary incisors, no significant nose deviation, and an orthopedic intrusion of the premaxilla [22, 24, 31, 32].

However, special consideration is needed regarding the incisors. Prior to the alveolar bone graft, the incisors adjacent to the cleft may present with limited and thin alveolar bone support [33] and significant angulation towards the opposite side of the cleft (Figure 9).

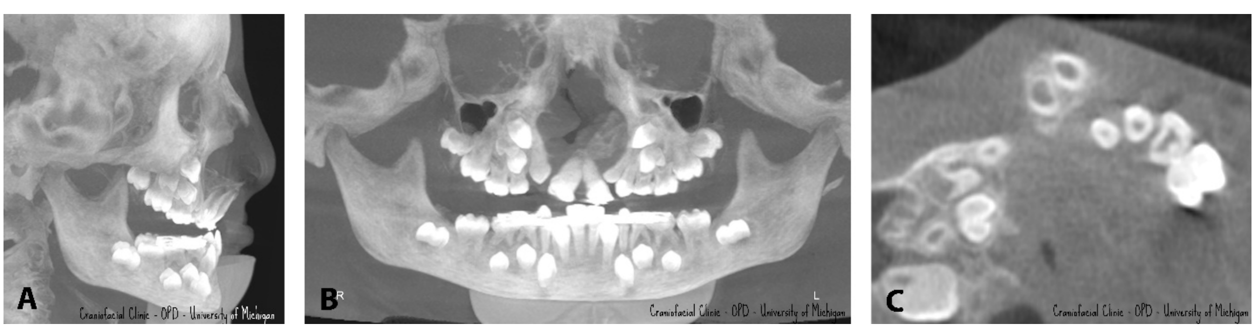

Figure 9. A) Reconstructed lateral cephalogram, B) panoramic, and C) axial view. Note the severity of the lingual inclination of the incisors in the premaxilla in the lateral ceph; the mesial root inclination of the maxillary permanent central incisors, with limited visualization of bone support in the panoramic; and the thin alveolar bone in surrounding the root of the incisors in the axial view.

Therefore, the bonding technique prior to the alveolar bone graft must be adapted to the current position of the teeth to avoid uprighting those teeth – the bracket must be placed as parallel to the horizontal plane as possible, and a passive wire must be bent to avoid any unwanted dental movement. The applied force must also be very light to avoid dental tipping and increased premaxilla movement.

ADULTHOOD

During adulthood, the chances of orthopedically moving the premaxilla are very limited. At this stage, it is preferred to level and align the teeth to the best of our ability while respecting the periodontal boundaries. Afterward, the patient can continue care with orthognathic surgery and rehabilitate with a prosthesis.

CONCLUSIONS

The challenge of treating patients with BCLP is a continuing and important topic to healthcare providers. Even though there is no consensus on how to best treat these patients, it is important to respect the limits and knowledge of each professional.

The literature consensus is that non-surgical repositioning of the premaxilla should be performed with caution. When performed at an early age, it should also focus on improving nasal symmetry and lengthening the columella. If performed later, anchored in teeth, it should respect the periodontal boundaries and should avoid dental tipping. Surgical repositioning of the premaxilla should be performed between 8-11 years of age, associated with the secondary alveolar bone graft, because, at this stage, over 90% of the midface growth is complete and facilitates the eruption of the permanent dentition through the bone graft.

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REFERENCES

1. Salari N, Darvishi N, Heydari M, Bokaee S, Darvishi F, Mohammadi M. Global prevalence of cleft palate, cleft lip and cleft palate and lip: a comprehensive systematic review and meta-analysis. J Stomatol Oral Maxillofac Surg 2022; 123:110-120.
2. Mai CT, Isenburg JL, Canfield MA, Meyer RE, Correa A, Alverson CJ, Lupo PJ, Riehle-Colarusso T, Cho SJ, Aggarwal D et al. National population-based estimates for major birth defects, 2010-2014. Birth Defects Res 2019; 111(18):1420-1435.
3. Chadha A, Cobb ARM. Development of cleft lip and palate. In: Carachi R, Doss SHE, eds. Clinical Embryology: An Atlas of Congenital Malformations. Cham, Switzerland: Springer Link; 2019:111-118.
4. Drilleaud A, Corre P, Perrin JP, Loin J, Durand T, Salagnac JM, Delaire J, Mercier JM, Bertin H. Surgical repositioning of the premaxilla: incidence, indications and growth study about a 189 bilateral cleft lip +/- palate population. Cleft Palate Craniofac J 2022; 0(0). doi:10.1177/10556656211056807

5. da Silva Filho OG, Carvalho Lauris RC, Capelozza Filho L, Semb G. Craniofacial morphology in adult patients with unoperated complete bilateral cleft lip and palate. Cleft Palate Craniofac J 1998; 35(2):111-119.
6. Pruzansky S. The growth of the premaxillary-vomerine complex in complete bilateral cleft lip and palate. Tandlaegebladet 1971; 75(12):1157-1169.
7. Trotman CA, Ross RB. Craniofacial growth in bilateral cleft lip and palate: ages six years to adulthood. Cleft Palate Craniofac J 1993; 30(3):261-273.
8. Latham RA. Development and structure of the premaxillary deformity in bilateral cleft lip and palate. Br J Plast Surg 1973; 26(1):1-11.
9. Latham RA, Deaton TG, Calabrese CT. A question of the role of the vomer in the growth of the premaxillary segment. Cleft Palate J 1975; 12:351-355.
10. Aburezq H, Daskalogiannakis J, Forrest C. Management of the prominent premaxilla in bilateral cleft lip and palate. Cleft Palate Craniofac J 2006; 43(1):92-95.
11. King BF, 3rd, Workman CH, 3rd, Latham RA. An anatomical study of the columella and the protruding premaxillae in a bilateral cleft lip and palate infant. Cleft Palate J 1979; 16(3):223-229.
12. Friede H, Morgan P. Growth of the vomero-premaxillary suture in children with bilateral cleft lip and palate. A histological and roentgencephalometric study. Scand J Plast Reconstr Surg 1976; 10(1):45-55.
13. McComb H. Primary repair of the bilateral cleft lip nose: a 15-year review and a new treatment plan. Plast Reconstr Surg 1990; 86(5):882-889; discussion 890-883.
14. Gungor A, Ruiz R, Costello BJ. Cleft Lip and Palate: Comprehensive treatment and technique. In: Operative Otolaryngology: Head and Neck Surgery. edn. Edited by Meyer EN; 2008: 765-790
15. Vargervik K. Growth characteristics of the premaxilla and orthodontic treatment principles in bilateral cleft lip and palate. Cleft Palate J 1983; 20(4):289-302.
16. Shepherd WM, McCarthy MD. Observations on the appearance and ossification of the premaxilla and maxilla in the human embryo. Anat Rec 1955; 121(1):13-28.
17. Heidbuchel KL, Kuijpers-Jagtman AM, Freihofer HP. An orthodontic and cephalometric study on the results of the combined surgical-orthodontic approach of the protruded premaxilla in bilateral clefts. J Craniomaxillofac Surg 1993; 21(2):60-66.
18. Latham RA. Maxillary development and growth: the septo-premaxillary ligament. J Anat 1970; 107(Pt 3):471-478.
19. Bittermann GK, de Ruiter AP, Janssen NG, Bittermann AJ, van der Molen AM, van Es RJ, Rosenberg AJ, Koole R. Management of the premaxilla in the treatment of bilateral cleft of lip and palate: what can the literature tell us? Clin Oral Investig 2016; 20(2):207-217.
20. Murthy J, Manisha D. Premaxillary complex morphology in bilateral cleft and hypothesis on laterality of deviated premaxilla. Indian J Plast Surg 2016; 49(3):336-339.
21. Carlini JL, Biron C, Gomes KU, Da Silva RM. Surgical repositioning of the premaxilla with bone graft in 50 bilateral cleft lip and palate patients. J Oral Maxillofac Surg 2009; 67(4):760-766.
22. Meazzini MC, Cohen N, Autelitano L, Radojicic J. Non-surgical treatment of vertical excess of the premaxilla in growing bilateral cleft lip and palate patients. Int J Oral Maxillofac Surg 2021; 50(12):1576-1582.
23. Geraedts CT, Borstlap WA, Groenewoud JM, Borstlap-Engels VM, Stoelinga PJ. Long-term evaluation of bilateral cleft lip and palate patients after early secondary closure and premaxilla repositioning. Int J Oral Maxillofac Surg 2007; 36(9):788-796.
24. Liou EJ, Chen PK, Huang CS, Chen YR. Orthopedic intrusion of premaxilla with distraction devices before alveolar bone grafting in patients with bilateral cleft lip and palate. Plast Reconstr Surg 2004; 113(3):818-826.

25. Chauhan JS, Sharma S, Bandi N. Secondary correction of premaxilla in bilateral cleft lip and palate with lag-screw fixation. Br J Oral Maxillofac Surg 2020; 58(9):1123-1127.
26. Motohashi N, Pruzansky S. Long-term effects of premaxillary excision in patients with complete bilateral cleft lips and palates. Cleft Palate J 1981; 18(3):177-187.
27. Friede H, Pruzansky S. Longitudinal study of growth in bilateral cleft lip and palate, from infancy to adolescence. Plast Reconstr Surg 1972; 49(4):392-403.
28. Bittermann GKP, van Es RJJ, de Ruiter AP, Bittermann AJN, Koole R, Rosenberg A. Retrospective analysis of clinical outcomes in bilateral cleft lip and palate patients after secondary alveolar bone grafting and premaxilla osteotomy, using a new dento-maxillary scoring system. J Craniomaxillofac Surg 2021; 49(2):110-117.
29. Grayson BH, Cutting CB. Presurgical nasoalveolar orthopedic molding in primary correction of the nose, lip, and alveolus of infants born with unilateral and bilateral clefts. Cleft Palate Craniofac J 2001; 38(3):193-198.
30. Millard DR, Jr., Berkowitz S, Latham RA, Wolfe SA. A discussion of presurgical orthodontics in patients with clefts. Cleft Palate J 1988; 25(4):403-412.
31. Kecik D, Enacar A. Repositioning of premaxilla in bilateral cleft lip and palate using a "J-hook headgear". J Craniofac Surg 2006; 17(6):1198-1207.
32. Mahmood R, Flood T, Robinson S, Al-Gholmy M. Early Orthopedic Retraction of the Premaxilla in Bilateral Complete Cleft Lip and Palate: An Innovative Approach to a Difficult Problem. Cleft Palate Craniofac J 2016; 53(3):363-367.
33. Garib DG, Yatabe MS, Ozawa TO, Filho OG. Alveolar bone morphology in patients with bilateral complete cleft lip and palate in the mixed dentition: cone beam computed tomography evaluation. Cleft Palate Craniofac J 2012; 49(2):208-214.