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Current Approaches in Orthodontics

Edited by Belma Işık Aslan and Fatma Deniz Uzuner

CURRENT APPROACHES IN ORTHODONTICS

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and **Fatma Deniz Uzuner**

Current Approaches in Orthodontics
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IntechOpen Book Series

Dentistry

Volume 2

Belma Işık Aslan was born in 1976 in Ankara, Turkey. She graduated from Gazi University Faculty of Dentistry, Turkey in 1999. She received her PhD degree from the Orthodontics Department of the same university in 2005. She became an Associate Professor in 2014 and is currently working as a full-time lecturer and an academic researcher. She has published a large amount of research in various international and national journals, written book chapters, and she serves as an editorial board member and reviewer of several scientific journals. Her expertise areas are cleft lip and palate, mini-implants, fixed-functional appliances, surgery first, and Class II and Class III treatment. She is married and has two children.

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Scope of the Series

The major pathologies which dentists encounter in clinical practice include dental caries and periodontal diseases. Diagnosis and treatment of these pathologies is essential because when untreated, abscess could occur and it can even lead to the extraction of the tooth. Extracted teeth can be replaced with implants. Dentists and patients are nowadays more familiar with dental implant treatments. As a result, advanced diagnostic tools which aid in pre-operative treatment planning (cone-beam computed tomography, computer aided implant planning etc..), new implant designs improving the success of osteointegration, new materials, and techniques are introduced in the dental market.

Conditions which dentists frequently encounter in their clinical practice are temporomandibular joint (TMJ) disorders. These disorders include degenerative musculoskeletal conditions associated with morphological and functional deformities. Accurate diagnosis is important for proper management of TMJ pathologies. With the advance in technology, new materials, techniques and equipment are introduced in the dental practice. New diagnostic aids in dental caries detection, cone-beam computed tomographic imaging, soft and hard tissue lasers, advances in oral and maxillofacial surgery procedures, uses of ultrasound, CAD/CAM, nanotechnology, plasma rich protein (PRP) and dental implantology are some of them. There will be even more new applications in dentistry in the future.

This book series includes topics related to dental caries, dentomaxillofacial imaging, new trends in oral implantology, new approaches in oral and maxillofacial surgery, temporomandibular joint disorders in dentistry etc.

Contents

Preface

In the early years of orthodontics, the main goal was to properly align the dentition to create functional harmony in occlusion. Present-day developments in orthodontics focus on the aesthetic expectations of the patients, which do not only involve the dental and facial aesthetics, but also the aesthetic appearance of the appliance designs.

Current orthodontic treatment also focuses on shortening the treatment time besides the aesthetic demands. We live in a fast-paced world and the patients are impatient, not showing the same level of tolerance as before. The treatment duration needs to be shortened. Therefore, alternative methods to accelerate orthodontic tooth movement so as to shorten orthodontic treatment time, minimizing the side effects (tooth resorption etc.), and achieving stable results have gained importance.

Today, orthodontics improve dental and skeletal aesthetics and also provide more global benefits in terms of health and well-being such as in the treatment of obstructive sleep apnea.

This book documents the current technological developments and new concepts in orthodontic materials and treatment procedures. The book has been contributed to by authors and researchers from all over the world reflecting the progress in orthodontics. The main concepts of the book will scope innovations in accelerated tooth movement, present current developments in corticotomy, microperforations (MOP), piezosicion, photobiostimulation, laser in orthodontics, chemical agents, as well as complications and risks. It contains interdisciplinary managements involving surgery first, cleft lip and palate therapy, orthognathic surgery, and obstructive sleep apnea. This internationally-recognized specialty is continuously evolving advancements in technology, instrumentation, and treatment methods.

This book may be of great value to not only orthodontic practitioners but also to others who will find learning resources in connection with their fields of study. This will help them acquire valid knowledge and excellent clinical skills given that the clinicians could use all the knowledge obtained for deciding the best treatment option to meet the healthcare needs of the patient and achieving optimum treatment outcome.

Overview of the chapters of this book

First chapter: Introductory chapter - Innovations in Orthodontics written by Belma IŞIK ASLAN & Fatma Deniz UZUNER

Second chapter: 'Ceramic Brackets Revisited' written by Elekdag-Türk S. and Abulkbash H. This chapter provides information about aesthetic brackets. This comprehensive review covers the physical properties as well as rebonding and debonding of polycrystalline and monocrystalline ceramic brackets. Furthermore, this review aims to present the advantages

and disadvantages as well as the refinements of these brackets since their introduction in the late 1980s.

Third chapter: 'Accelerated Orthodontics' written by Patil A.K. and Maan A.S. In this chapter, the authors summarize the methods of accelerated tooth movements by means of pharmacological, surgical, and physical methods. The pharmacological agents (prostaglandin E_2 and E_1, 1,25-dihydroxycholecalciferol, parathyroid hormones etc.) that act as biomodulators for increased orthodontic tooth movement are discussed. Surgical methods are categorized as periodontally accelerated osteogenic orthodontics, piezocision, and micro-osteoperforations. Physical methods such as the vibratory stimulus, low level laser therapy, and low-intensity pulsed ultrasound are defined. A novel device, AcceleDent, is described as an example for vibratory stimulus.

Fourth chapter: 'The Role of Cytokines in Orthodontic Tooth Movement' written by Vujacic A. This chapter focuses on existing knowledge about the roles and dynamics of the change in three cytokines (*IL -1β, IL-6,* and *TNF*) simultaneously during the early stage of orthodontic tooth movement. Detailed information about orthodontic tooth movement and biological mechanisms of reshaping the mechanic-sensitive dental tissue is provided. The chapter discusses the benefit of pharmacological modulation of the tooth movement, especially the aspect that leads the acceleration of the process of tooth movement in the connection with the local application of cytokines.

Fifth chapter: 'Advances in Orthodontic Tooth Movement: Gene Therapy and Molecular Biology Aspect' written by Atsawasuwan P., Shirazi S. This chapter starts with general information about the biology of tooth movement. Detailed information about the molecular mechanism of orthodontic tooth movement is presented. The authors discuss how advances in gene therapy and molecular biology technology will shape the future of orthodontic treatment. The authors also emphasize the future genetic manipulation of tooth movement.

Sixth chapter: 'Micro-osteoperforations' written by Bolat E. This chapter discusses micro-osteoperforations (MOPs), which is a recent, reliable, repeatable, and minimally invasive method to stimulate alveolar bone remodeling without the disadvantages of surgery; such as the requirement of corticotomies, cuts in cortical bone, raising split-thickness flap, and decorticating the bone. Informative details about the biological basis of orthodontic tooth movement, accelerated tooth movement techniques, and application methods are mentioned. The author describes the advantages, disadvantages, effects, and possible side effects of micro-osteoperforations based on recently published literature.

Seventh chapter: '**Stability of Diastema Closure after Orthodontic Treatment' written by Carruitero M.J., Janson G.** This chapter seeks to evaluate the recurrence of upper interincisal diastemas, the factors associated with the relapse and stability, some treatment proposals, and considerations for retention. The chapter starts with the definition and epidemiology of diastemas and carries on with the development of diastemas related to the development of occlusions. The readers can benefit from practical strategies that can be applied in clinical practice to provide stability.

Eight chapter: 'Current Approaches in Orthognathic Surgery' written by ATAÇ S. M. This chapter provides information about commonly used orthognathic surgery techniques to solve deformity problems of the facial skeleton. In this comprehensive chapter, the author shared his personal experience and some technical details concerning Le fort I osteotomy

and Sagittal Split Ramus osteotomies. He mainly emphasizes the use of piezoelectric surgery rather than rotary instruments, and how surgical saws supply bloodless and neuro sensorial deficits free operations. The chapter ends with the future of orthognathic surgery and related technologic developments.

Ninth chapter: 'Ortho-surgical Management of Severe Vertical Maxillary Excess (Gummy Smile)' written by Saleh F. and Al Hamadi W. In this chapter, the authors review the treatment alternatives for excessive gingival display. They present clinical cases. The authors emphasize the orthosurgical treatment of severe vertical maxillary excess as the optimal solution to restore facial balance and harmony, an attractive smile, and patient satisfaction.

Tenth chapter: 'Surgery First Approach' written by Gülşen A. This chapter focuses on the advantages and treatment plan of SFA. The author provides information about the time for orthodontic bonding and force application in detail. The reasons of relapse in SFA are emphasized at the end of the chapter.

Eleventh chapter: 'Orthodontics in Relation to Alveolar Bone Grafting in Patients with CLP' written by Uzel A. In this chapter, the current orthodontic approaches in relation to alveolar bone grafting (ABG) in cleft patients are discussed. The factors for success of the graft are defined. The periodontal health of the surrounding graft tissues, the experience and ability of the surgeon, the timing of surgery, and orthodontic management of the cleft area before and after grafting are underlined. The chapter carries on with the treatment sequencing for pre-graft orthodontics, post-graft stabilization, and post-graft orthodontics. The chapter ends with the explanation of unfavorable conditions including wide alveolar cleft, late one grafting/prolonged orthodontic treatment, and unstable premaxilla.

Twelfth chapter: 'Contemporary Treatment Approaches to Obstructive Sleep Apnea Syndrome' written by Oğuz H.T. This chapter presents the current information about etiology, diagnostic tools, treatment alternatives of obstructive sleep apnea, and introduces dental sleep medicine to orthodontists. This chapter starts with the diagnosis and classification of Obstructive Sleep Apnea Syndrome and continues with epidemiology and etiology of this syndrome according to contemporary literature. Detailed information about the treatment options is given. The role of orthodontist is stressed in the chapter.

We would like to thank all the authors and Ms. Dolores Kuzelj and all the staff who took a role in the production of this project.

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Chapter 1

Introductory Chapter: Innovations in Orthodontics

Belma Işık Aslan and Fatma Deniz Uzuner

Additional information is available at the end of the chapter

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1. Innovations in orthodontics

In the present day, orthodontic treatment not only meets the demand for functional harmony in occlusion and the improvement of the esthetic appearance but also should be completed in the most efficient time accepted by the patient and the orthodontist. Living in a fast-paced world leads to a great interest in the techniques decreasing the orthodontic treatment duration.

Various clinical techniques were introduced for accelerated orthodontic tooth movement, including local injection of cellular mediators [1], physical or mechanical stimuli [2], and surgically assisted orthodontics [3, 4]. The rate of orthodontic tooth movement is under the control of molecular mechanisms regulating cellular behavior in the alveolar bone and in the periodontal ligament [5–7]. Therefore, it is essential to identify and control cellular regulators to safely shorten the duration of orthodontic treatment. In addition to the local injection of cellular mediators, with the rise of advanced technology in biomedical engineering and medicine, gene therapy will be a promising method in the future for accelerated tooth movement.

The common physical methods used in the present day are vibratory stimulus, low-level laser therapy, and low-intensity pulsed ultrasound.

For surgically assisted orthodontic techniques, the current surgical methods being practiced are periodontally accelerated osteogenic orthodontics, piezocision, and micro-osteoperforations. Micro-osteoperforations have emerged as a new, minimally invasive, easy-to-use, reproducible, and effective method that eliminates some of the disadvantages of surgery such as the requirement of corticotomies, cuts in cortical bone, raising split-thickness flap, and decorticating the bone [7].

Today, multidisciplinary treatment procedures are taking more place in orthodontics. The treatment of skeletal discrepancies requires orthognathic surgery in combination with orthodontic treatment to improve malocclusion, function, facial, and smile esthetics. The improvement in

orthognathic surgical techniques provides easier, comfortable confident treatments in patients with facial deformations. Nowadays, the latest trends in orthognathic surgery besides the basic techniques are tomographic assessments that provide 3D evaluations of facial proportions rather than 2D cephalometric radiographs. Additionally, 3D scanning of the teeth integrated with tomography provides to perform operation in virtual environment and also to print out 3D splints as well as the individual titanium plate screw fixation systems which eliminate surgical splints. Other advancements in orthognathic surgery are the use of piezoelectric surgery, which supplies bloodless and neurosensorial deficit-free operations rather than rotary instruments and surgical saws. It is expected that in the future a robotic surgery may take place [8].

In the past 10 years, surgery-first approach (SFA) created a broader interest [9–11]. The surgery-first approach is an alternative method that may be more satisfying for orthodontist and the patients by shortening the duration of the treatment. In the beginning of the treatment, surgery is performed without orthodontic preparation, and the orthodontic treatment is applied after the surgery. With this approach, patients benefit with the early correction of facial esthetics and psychosocial outcomes of improved body image at the beginning of the treatment [11–13] instead of worsening the facial appearance because of the presurgical decompensation of incisors as it is done in contemporary orthognathic surgery [11, 14].

Another field of orthodontics is the treatment of cleft lip and/or palate (CLP), which requires interdisciplinary care by centralization of treatment. Orthodontics is the core element of the overall treatment process in cleft patients. Alveolar bony defect is the main limiting factor for orthodontic treatment. Alveolar bone grafting is the essential implementation that necessitates a combined orthodontic and surgical involvement [15, 16].

The other interdisciplinary management in orthodontics consists of obstructive sleep apnea (OSA) syndrome patients. Sleep-related breathing disorders decrease sleep time and/or quality, which leads to excessive daytime sleepiness, fatigue, and lack of concentration, and finally decrease the life quality. In severe cases, morbidity and mortality may be observed. Orthodontists, who specialized in dental sleep medicine, can see the early symptoms of these diseases and may be effective in the treatment of snoring and mild-to-moderate obstructive sleep apnea by applying oral appliance [17, 18].

The chapters in this book provide rich information to the readers starting with the ceramic brackets. It continues with the methods in accelerated orthodontics; the role of cytokines, gene therapy, and molecular biology aspect and surgical methods such as corticotomy and micro-osteoperforations are addressed. Stability of diastema closure after orthodontic treatment is presented according to updated research. The book goes on with the current approaches in multidisciplinary treatments involving orthognathic surgery, alveolar bone grafting in patients with cleft lip and palate, and obstructive sleep apnea syndrome.

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Chapter 2

Ceramic Brackets Revisited

Selma Elekdag-Türk and
Hüdanur Yilmaz (née Huda Ebulkbash)

Additional information is available at the end of the chapter

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Abstract

Increasing demand for esthetics during fixed appliance therapy has led to a significant increase in the demand for and use of ceramic brackets. This comprehensive review covers the physical properties as well as rebonding and debonding of polycrystalline and monocrystalline ceramic brackets. Furthermore, this review aims to present the advantages and disadvantages as well as the refinements these brackets underwent since their introduction in the late 1980s. Interestingly, the introduction and development of these brackets were a part of a quickly expanding ceramic technology in many industries. The authors strongly believe that the novice will particularly benefit from this review presenting up-to-date knowledge.

Keywords: review, ceramic brackets, monocrystalline alumina brackets, polycrystalline alumina brackets, rebonding, debonding

1. Introduction

It is interesting to note that ceramic braces entered orthodontics via an indirect route. Translucent polycrystalline alumina (TPA) was developed by NASA (National Aeronautics and Space Administration) and Ceradyne, a leader in advanced ceramics for aerospace, defense, electronics, and industrial use. In 1986, a dental equipment and supply company contacted Ceradyne for an esthetic material to be used in orthodontics. Ceradyne recommended TPA. Shortly, after this contact, namely in 1987, ceramic brackets were introduced. In the same year, i.e., 1987, the production of ceramic brackets reached 300,000 pieces a month [1]. To clarify, 300,000 pieces would translate into 15,000 nonextraction patients per month! Ceramic brackets have progressed substantially since their first introduction over 30 years ago. This article aims to present an up-to-date review of the physical and clinical characteristics, i.e., properties, of ceramic brackets.

2. Ceramic bracket production: a short overview

Most ceramic brackets are produced from aluminum oxide (alumina) particles, and these brackets are available in polycrystalline and monocrystalline forms [2].

Nowadays, the majority of polycrystalline (multiple crystals) brackets are produced by ceramic injection molding (CIM). An outline of CIM is as follows: the aluminum oxide (Al_2O_3) particles are mixed with a binder. This mixture is rendered flowable through heat and pressure application and injected into a bracket mold. The binder is removed, i.e., burned out. Subsequently, sintering—the production of a coherent mass by heating without melting—is carried out. The advantage of CIM is that this technology can manufacture complex and precise items with smooth surfaces in large quantities at fast rates [3].

The production process for monocrystalline (single crystal) ceramic brackets, also referred to as sapphire brackets, is completely different. Here, the Al_2O_3 particles are melted. The resultant mass is slowly cooled to permit a controlled crystallization, leading to the production of a large, single crystal. This large, single crystal in rod or bar form is then milled into brackets with ultrasonic cutting techniques and/or diamond cutting tools. After milling, the monocrystalline brackets are heat-treated to eliminate surface imperfections and to relieve the stress caused by the milling procedure. The production of these brackets is more expensive when compared to the production of polycrystalline brackets. This increased expense is mainly due to the difficulty of milling, i.e., the cutting process [2].

3. Properties of ceramic brackets

3.1. Hardness

Ceramic brackets are known for their hardness. They are notably harder than enamel [4–7]. Thus, contact between enamel and ceramic brackets has to be avoided by all means. This type of contact can lead to severe enamel damage [8]. Particular care has to be exercised with deep bite and/or class II canine relationship patients. If required, bite opening applications must be performed to prevent enamel damage.

3.2. Tensile strength

The ultimate tensile strength, often shortened as tensile strength, is defined as the maximum stress that a material can withstand while being stretched or pulled before failing or breaking [9]. When stress is placed on a ceramic material, its unyielding atomic structure makes the redistribution and the relief of stress close to impossible [2]. Ductile materials, such as metals and polymers, experience plastic deformation before failure [10]. In other words, the elongation of ceramics at failure (brittle fracture) is less than 1%, yet the elongation of stainless steel at failure (ductile fracture) is approximately 20% [11]. Hence, ceramic brackets do not flex. This implies that ceramic brackets are much more likely to fracture than metal brackets under identical conditions [11].

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3.3. Fracture toughness

Fracture toughness is a property which describes the ability of a material containing a crack to resist fracture [6, 12]. This is an important material property since the presence of imperfections, such as microscopic scratches, cracks, voids, and pores are not completely avoidable during the fabrication of materials. These microscopic imperfections may or may not be harmful to the material, depending on a number of factors such as the fracture toughness of the material examined, the stress on the material, length of the crack, and resistance of the material to crack propagation as well as the environment of the material [6].

The higher the fracture toughness, the more difficult it is to propagate a crack in that material [12]. The fracture toughness of polycrystalline alumina brackets is higher than the fracture toughness of monocrystalline alumina brackets. This implies that crack propagation is relatively easier in single-crystal alumina brackets when compared with polycrystalline alumina brackets [12]. Polycrystalline brackets have a higher resistance to crack propagation due to crack interaction with grain boundaries (GBs). A GB is the interface between two "grains" (crystals) in a polycrystalline (multiple crystals) material (**Figure 1**). Cracks are impeded at these GBs [10]. Clinical applications that may scratch the surfaces of ceramic brackets may greatly reduce the fracture toughness, thereby predisposing ceramic brackets to eventual fracture [12]. Thus, utmost care has to be taken not to scratch ceramic bracket surfaces with instruments and stainless steel ligature wires during treatment. Also, the clinician should not overstress when ligating with steel ligature wires. This might initiate crack growth and propagation, leading to the eventual fracture of the bracket. Careful ligation is mandatory, and elastomeric modules (ligatures) or coated ligatures are advised to prevent ceramic bracket fractures, particularly tie-wing fractures [6, 13, 14]. Arch wire sequencing also has to be performed carefully. The use of resilient full-size arch wires before the placement of full-size stainless steel arch wires is recommended [7]. Furthermore, the patient has to be advised to restrain from chewing and/or biting on any hard substances [6] as well as from intraoral/lip piercings. A prudent choice is to avoid ceramic brackets with orthognathic surgery patients as well as with patients involved in contact sports.

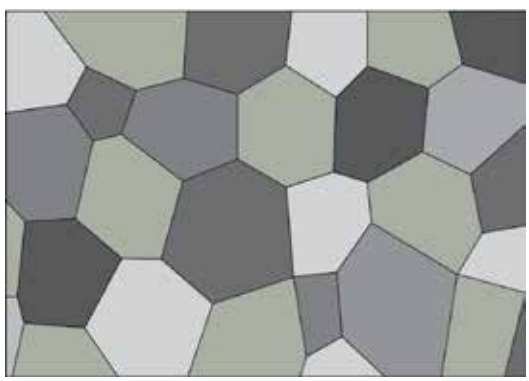

Figure 1. Schematic presentation of "grains" and GBs.

Finally, it should be noted that the exposure of alumina to water or saliva decreases fracture toughness [10]. This characteristic is important to remember when the clinician attempts to extrapolate in vitro results to the clinical setting, i.e., the oral environment.

3.3.1. Tie-wing fracture

Figure 2 pictures a tie-wing fracture of the lower second premolar bracket. Most likely this tie-wing was damaged with pliers during arch wire insertion into the molar tube.

Complete fragmentation of a damaged bracket might occur during arch wire ligation or during the course of treatment. Thus, the removal of an impaired bracket and its replacement with a new bracket is a prudent risk management strategy. The risk of ceramic fragment penetration into the patient's oral soft tissues, inhalation or swallowing by the patient does exist. Ceramics are radiolucent, i.e., ceramic bracket fragments are not visible on radiographs [15].

An interesting in vitro study [16] tested tie-wing fracture strength of polycrystalline and monocrystalline brackets after being exposed to fluoride prophylactic agents (Prevident 5000 and Phos-flur gel; Colgate Pharmaceuticals, Canton, Mass, USA). The researchers stated that the fluoride-alumina surface interaction most likely caused strain in the surface bonds of both types of brackets. Yet, this presumed bond strain only affected the fracture strength of the monocrystalline alumina brackets. The results of this study imply that the use of topical fluoride agents may increase the susceptibility of tie-wing fractures of monocrystalline brackets under clinical conditions and that polycrystalline brackets might be the appropriate choice for poor oral hygiene patients that require fluoride prophylactic agents. The authors [16] pointed out that this outcome was most likely related to the inhibition of cracks at the GBs of the polycrystalline microstructure.

The tie-wing complex of polycrystalline ceramic brackets can be manufactured as either semitwin or true twin. Semitwin differs from true twin by having an isthmus of ceramic joining the mesial and distal tie-wings, i.e., the mesial and distal tie-wings are not four independent projections from the bracket base as with the true twin configuration (**Figure 3**). This semitwin configuration has been stated to possess a better tie-wing fracture strength. It has been proposed that such a ceramic connector produces a cross-stabilizing effect [13, 17].

Figure 2. Distogingival tie-wing fracture (the red elastic ligature was used to accentuate this fracture).

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Figure 3. The semitwin tie-wing complex.

3.4. Friction

When polycrystalline ceramics were compared with monocrystalline ceramics, it was concluded that polycrystalline ceramics have a higher coefficient of friction. In fact, more than a decade ago, it was pointed out that monocrystalline brackets have frictional characteristics close to metal brackets [4].

To overcome the problem of frictional resistance of polycrystalline brackets, manufacturers carried out numerous modifications. Polycrystalline ceramic brackets with metal inserts in the arch wire slot (metal slots) were developed [18]. Nevertheless, it was reported that the sharp edges of the metal insert may "dig into" the softer arch wire material, thus increasing resistance to sliding and thereby reducing the efficiency of tooth movement [7, 19]. Another modification was the addition of bumps along the floor of the polycrystalline ceramic bracket slot. Nevertheless, these bumps were not effective in reducing frictional resistance [20].

A recent study, including ceramic and metal brackets that were manufactured by different production methods, including CIM and metal injection molding (MIM), concluded that the manufacturing technologies do not present a critical difference regarding friction [3]. It was reiterated that the complex phenomenon of friction depends on a multitude of factors, such as the bracket/ligature/arch wire combinations, the surface quality of the arch wire/bracket slot, the bracket design, and the force exerted by the ligature on the arch wire [3].

3.5. Optics

The optical properties of ceramic brackets provide an attractive option for a great number of patients. As previously mentioned, polycrystalline ceramic brackets possess a microstructure of crystal GBs. This microstructure reflects light, resulting in some degree of opacity. In contrast, single-crystal brackets lack GBs, thus permitting the passage of light, making these brackets basically clear [2, 10, 21].

As mentioned above, monocrystalline brackets have more optical clarity than polycrystalline brackets (**Figure 4**). Whether this difference is of essential importance from an esthetic point of view is a decision to be made by the orthodontist as well as the patient [2].

Apart from esthetics, the optical properties of ceramic brackets have been shown to affect the amount of light transmitted through these brackets during photocuring. The amount of light transmitted through ceramic brackets affects the curing efficiency of the light-cured adhesive. Polycrystalline brackets and polycrystalline brackets with a polymer mesh base were found to block direct light transmittance to a greater extent than monocrystalline brackets. It was pointed out that the color-coded holders designed for identification and handling of ceramic brackets also hinder light transmittance. The use of clear holders with colored edges has been suggested [22].

3.6. Color stability

The color stability of ceramic brackets throughout orthodontic treatment is an important characteristic. It has been stated that ceramic brackets, both monocrystalline and polycrystalline, undergo a color change when subjected to coffee, black tea, coke, and red wine [21, 23, 24]. It has to be pointed out that these are in vitro findings. In vivo studies concerning the color stability of ceramic brackets are lacking.

3.7. Plaque accumulation

Limited information is available about which bracket material (ceramic versus metal brackets) is less prone to the adhesion of bacteria and plaque accumulation. A clinical study performed by Lindel et al. [25] concluded that ceramic brackets exhibit less long-term biofilm accumulation than metal brackets. It was emphasized that future research should aim to determine whether the difference in biofilm accumulation between ceramic and metal brackets has a clinically significant effect on the development of decalcifications. Lindel et al. [25] pointed out that the results obtained from this type of future research might have a strong effect when choosing bracket material in patients with insufficient oral hygiene habits.

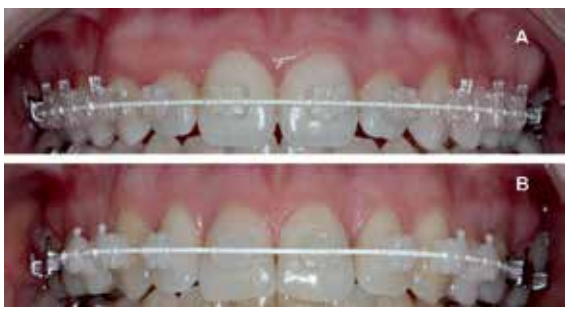

Figure 4. Intraoral image of monocrystalline (A) and polycrystalline (B) ceramic brackets.

3.8. Biocompatibility

Biocompatibility is the ability of a material to provide successful service in a host while causing minimal response [9]. It has been stated that conventional ceramic brackets are chemically stable (inert) in the oral environment and that they exhibit excellent biocompatibility with oral tissues [5].

In 2012, Retamoso et al. [26] carried out an in vitro cytotoxicity study evaluating various orthodontic brackets. These researchers reported that monocrystalline ceramic brackets had good biocompatibility. On the other hand, polycrystalline ceramic brackets with metal slots demonstrated some toxic effects. It was pointed out that the metallic slot was the essential factor responsible for a decrease of cell viability due to nickel ion release. They [26] concluded that it is essential to continue with studies evaluating cytotoxicity. If toxicity of any material is proven, alternative materials have to be used.

3.9. Magnetic resonance imaging (MRI) compatibility

Orthodontists are often asked to remove fixed orthodontic appliances prior to an MRI scan—a diagnostic tool that does not expose the patient to radiation—particularly when looking for pathology in the head and neck region or when information regarding the articular disc is required [10, 27].

Beau et al. [27] provided a detailed flowchart concerning the indications for the removal of fixed orthodontic appliances prior to MRI scans of the head and neck region. According to this flowchart, ceramic brackets do not have to be removed prior to an MRI scan. They are MRI-safe. However, ceramic brackets with any metal components, such as stainless steel slots, have to be removed if the region under examination is adjacent to these brackets. Stainless steel causes extensive artifacts, which may degrade image quality beyond clinical acceptability. The authors [27] pointed out that they did not include arch wires or removable appliances in their research, since these can be easily removed prior to an MRI scan.

4. Characteristics of ceramic bracket bases

Several retention mechanisms were developed for the attachment of ceramic bracket bases to the adhesive. These are chemical retention, mechanical retention, and a combination of both methods [21, 28]. The first developed method was the chemical retention method. This method, now obsolete, used a coating of glass on the flat ceramic bracket base and then a silane coupler to achieve a chemical bond between the glass-coated bracket base and the adhesive. The silane molecule is a bifunctional molecule; that is, one end reacted with the glass coating on the bracket base, while the other end reacted with the adhesive [11, 29]. It was pointed out that the chemical retention mechanism produced very strong bonds that harmed the tooth surface in the form of cracks and enamel tear-outs during debracketing [4, 7, 11, 29–32].

Almost three decades ago, Ghafari and Chen [33] compared the performance of chemical retention ceramic brackets to silane-treated grooved base ceramic brackets (a combination of chemical and mechanical retention). They [33] concluded that mechanical retention might reduce the negative side effects of debracketing by favoring failure within the adhesive, thus protecting the integrity of the enamel surface, i.e., the health of the tooth, as well as the integrity of the ceramic bracket.

The reports about iatrogenic tooth damage during debracketing impelled manufacturers to make changes in the base designs of ceramic brackets, relying more on mechanical retention for bond strength. In fact, the majority of ceramic brackets available today are purely mechanically retained brackets [4, 30, 34]. Mechanical retention is achieved by creating undercuts or grooves in the base of the bracket. These undercuts make a mechanical interlock with the adhesive bonding agent possible [28].

Currently, many different mechanical base designs are available, such as microcrystalline base design with a stress concentrator, button-structured base design, ball-base design with gingival ball reduction, dovetail base design, laser-structured base design, and "portal" bonding base design [4, 15, 35, 36].

An additional, interesting base design is the application of a thin layer of polymer onto the ceramic bracket base [19]. Thus, bonding takes place between the enamel and the flexible polymer mesh base. Encouraging in vitro results concerning the enamel surface after debracketing were obtained [30, 37, 38].

At this point, only two published clinical studies [14, 39] with a purely mechanical retention mechanism were encountered. There is a need for clinical studies, particularly randomized clinical studies, i.e., the gold standard for evaluating clinical procedures.

5. Rebonding of ceramic brackets

Although some major orthodontic supply companies explicitly state in their instruction sheets that their ceramic brackets are for single use only, several laboratory studies suggested various techniques for the reuse, i.e., the rebonding, of ceramic brackets [40–44]. For reuse, the bracket has to be intact in the first place.

It has been pointed out that the appropriate term is "reuse" or "recondition" instead of "recycle," since the term "recycled" implies the manufacturing of new brackets from the raw material of the original, failed brackets [12]. Nevertheless, the literature usually refers to these brackets as "recycled."

An in vitro study [43], carried out in 2016, investigated the "recycling" of polycrystalline ceramic brackets with a microcrystalline base via the following three methods: first is the erbium-doped yttrium aluminum garnet (Er:YAG) laser, and the other two are traditional methods, i.e., flaming and sandblasting. Sandblasting (50 μm Al_2O_3 particles) damaged the delicate bracket base structure and demonstrated significantly less bond strength than new

brackets. The flaming procedure yielded a bond strength that was similar to that of new ceramic brackets. However, flaming affected the esthetics of these brackets, i.e., these brackets ended up faded and dark. Er:YAG lasers completely removed the adhesive remnants from the ceramic bracket bases without damaging the base structure. Furthermore, the shear bond strength of Er:YAG laser "recycled" brackets was similar to that of new brackets. It was pointed out that the laser method may be preferred over other "recycling" methods.

Yassaei et al. [44] also concluded that the Er:YAG laser presents an efficient way for "recycling" ceramic brackets. These researchers used polycrystalline ceramic brackets with a dovetailed base in their in vitro study.

6. Debonding of ceramic brackets

Debonding usually refers to the removal of orthodontic brackets and the residual adhesives from the tooth enamel at the end of fixed appliance treatment [45].

Ceramic brackets lack flexibility. In other words, the rigid ceramic and the rigid enamel have little ability to dissipate stress when exposed to debracketing forces at the end of treatment. Thus, bracket fracture and/or enamel damage may occur during debracketing [2, 11].

Several approaches aiming to minimize the side effects associated with the debracketing of ceramic brackets exist. These are the conventional (mechanical), ultrasonic, electrothermal, and laser techniques [11, 21].

6.1. Mechanical debracketing

Three mechanical debracketing techniques have been described. These are lift-off, wrenching, and delamination [46]. The first technique uses a lift-off debracketing instrument (LODI). This pistol-grip plier is placed over the bracket, and a debracketing force is applied to the tie-wings of the bracket. It has been pointed out that the LODI cannot be used with ceramic brackets due to their brittleness [39]. The wrenching technique uses a special tool that produces a wrenching or torsional force at the base of the bracket [46]. This approach, providing a rotational shear force, can be likened to the turning of a door knob.

The delamination technique was the first technique introduced and is still reported to be the most widely accepted ceramic bracket removal technique [11, 15]. This technique involves the application of a slow squeezing force with the sharp blades of the debracketing pliers placed on the enamel surface and within the adhesive, thereby producing a wedging effect (**Figure 5**).

It has been stated that such a force—produced by a slow, gradual compression—would seem to offer the best chance for inducing crack propagation within the adhesive, leading to a cohesive failure, thus minimizing the risk of enamel damage as well as the risk of bracket fracture. A cohesive bond failure is a failure through a single material, where cohesive forces between the same atomic species are present [2, 11, 21, 46].

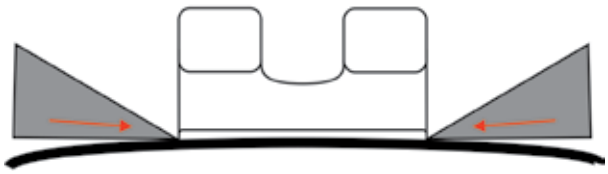

Figure 5. Schematic presentation of the delamination technique.

In 1993, Bishara and Fehr [47] evaluated the force levels produced with wide and narrow blades during ceramic bracket removal from human maxillary molar teeth. The wide blades and narrow blades were 3.2 and 2 mm, respectively. Polycrystalline ceramic brackets relying on a combination of mechanical and chemical retention were used. The findings of this in vitro study indicated that the narrow blades produced a significantly lower mean debracketing force, namely 120 kg/cm^2 than the wider blades (150 kg/cm^2). Bishara and Fehr [47] concluded that such a significant reduction (20%) in the debracketing force places less stress on the enamel surface, thereby reducing the risk of enamel damage. It has been reported and reiterated that debracketing forces larger than 138 kg/cm^2, i.e., 13.53 MPa should be avoided [11, 48, 49].

In 2000, Arici and Minors [50] carried out an in vitro study with four different methods of debracketing. They pointed out that reducing the contact area, i.e., contact area between the plier blades and the adhesive, to a minimum reduces the force necessary to initiate ceramic bracket removal. Macroscopically, no enamel damage and no bracket fractures were reported. Arici and Minors [50] used primary bovine mandibular incisor teeth and polycrystalline ceramic brackets with chemical retention. They concluded that their findings basically corroborate the findings of Bishara and Fehr [47].

Following the introduction of ceramic brackets, more than 30 years ago, serious complications during debracketing were encountered [4, 7, 11, 13, 51, 52]. Many in vitro studies, to assist in the development of more reliable and clinically safe ceramic brackets, followed. The reduction of ceramic debracketing forces, thereby protecting enamel integrity as well as bracket integrity, was the aim of these studies [11, 47, 48, 50].

Nowadays, the majority of ceramic bracket manufacturers present detailed debracketing instructions. In fact, many manufacturers have introduced debracketing instruments specifically designed for their bracket brand. These manufacturers claim that their brackets can be removed as easily and as safely as metal brackets as long as the orthodontist meticulously follows these instructions [4, 11, 13, 15]. As a risk management strategy, ceramic brackets that are not accompanied by detailed instructions for bonding and debracketing should definitely not be used.

Any shortcomings related to ceramic brackets should be reported immediately to the manufacturer [7]. "First, do no harm" should serve as the fundamental guiding principle for anyone engaged in health care [53].

6.2. Precautions for mechanical debracketing

Bonding a ceramic bracket to a compromised tooth, such as a brittle, nonvital tooth (endodontically treated tooth), a tooth with developmental defects, a tooth with demineralized enamel,

a tooth with enamel cracks and/or a large restoration, should be avoided as much as possible. This type of tooth is under greater risk for enamel damage when compared with a healthy tooth during mechanical debracketing [21, 39]. Care also has to be exercised with porcelain restorations, such as crowns and veneers [51].

During the bonding procedure, excess adhesive flash (EAF) must be removed with an explorer before the adhesive has set or with burs after setting. Only meticulous EAF removal allows the sharp-edged plier blades of the debracketing instrument to be fully seated on the enamel during ceramic bracket removal. This produces a safe force transmission and crack propagation through the adhesive, resulting in a cohesive failure within the adhesive, thereby protecting enamel as well as bracket integrity [2, 11, 21]. Furthermore, EAF removal improves esthetics by providing a clean and neat appearance.

It should be noted that a flash-free adhesive-coated appliance system was introduced. This innovative technology does not necessitate flash removal [54].

Unfortunately, debracketing may lead to bracket fracture. Bracket fragments may detach (loose fragments) or remain attached on the enamel surface. Low-speed or high-speed grinding of the bracket fragments with no water coolant may bring forth permanent damage and necrosis of the dental pulps. Therefore, water cooling is absolutely necessary during the grinding/removal of ceramic bracket fragments [10, 55]. Furthermore, high-volume suction next to the area of grinding has been emphasized in order to minimize the spreading of ceramic particles [10, 55]. These particles have been reported to cause irritation of the eyes as well as itching of the hands [55]. After the removal of these fragments, the clinician can proceed with adhesive remnant removal.

Loose, fractured ceramic bracket fragments may create serious problems, such as aspiration or ingestion of these radiolucent fragments. Furthermore, during debracketing, the "popping off" of fragments may occur. This may subject the patient as well as the orthodontist to eye injury. The use of protective eyewear and a mask is indispensable for the orthodontist. The patient should be given protective eyewear as well [21, 34, 56].

The force applied during debracketing may cause discomfort. Therefore, the orthodontist should always support the teeth with his or her fingers or make the patient bite firmly into a cotton roll during debracketing. Biting firmly into a cotton roll and/or gauze not only minimizes discomfort but also minimizes the risk of brackets and/or fragments from getting displaced into the oral cavity [21, 34]. Colored cotton rolls may facilitate the detection of fractured ceramic bracket fragments.

If pliers are used for debracketing, Bishara and Fehr [21] advised the renewal of the plier blades after the removal of 50 brackets. Pliers with nonexchangeable blades should be sharpened on a regular basis [21]. Sharp-edged plier blades are required for safe debracketing, i.e., for the induction of crack propagation within the bonding adhesive rather than the enamel or the ceramic bracket. The orthodontist should never delegate ceramic bracket debracketing to auxiliaries [21, 56].

It has been emphasized that every informed consent form signed by the patient/parents (and the orthodontist) should specifically outline the potential risks of ceramic brackets,

particularly in an ever-increasing litigious society [56]. Also, the brand of the ceramic bracket should always be recorded on the patient's file. This is of particular importance in the case of transfer to another orthodontist.

6.3. Adjunctive methods proposed for mechanical debracketing

Larmour and Chadwick [57] evaluated the ability of a commercial debonding agent, post-debonding agent (P-de-A) (Oradent Ltd., Eton, Berks, UK). This green gel, containing a derivative of peppermint oil, was claimed to facilitate ceramic bracket debracketing and adhesive residue removal. The manufacturer of P-de-A advised an application time of 1–2 min to soften the resin. Nevertheless, the P-de-A research results did not support these claims [57, 58].

In 1997, Arici et al. [59, 60] proposed the use of a crushable porous ceramic lamella as a means of facilitating debracketing. These porous lamellae were attached to the bracket base with adhesive resin. Subsequently, these bracket/lamella assemblies were bonded to the enamel of the experimental teeth (bovine incisor teeth). The authors [60] of this in vitro study reported the safe removal of these ceramic bracket/lamella assemblies, i.e., no fractures of the ceramic bracket or any evidence of enamel damage was observed. Commercial production of this type of ceramic bracket/lamella assembly was not undertaken.

In 1998, Larmour et al. [61] evaluated the possibility of reducing the complications of ceramic bracket debracketing by introducing a notch in the composite bond layer. A section of Mylar® matrix strip (0.01 mm thick and 0.75 mm wide) was placed within the bonding agent in this ex vivo investigation. After the bonding agent had set, the matrix strip was removed creating a "notched" bond layer. Larmour et al. [61] concluded that notching the bonding agent does facilitate ceramic bracket removal. Nevertheless, they emphasized that this modification is not feasible in a clinical setting due to the time needed and the technical difficulty of creating a "notched" bond layer. Furthermore, they cautioned that such a "notched" adhesive layer may lead to plaque accumulation.

In 2003, Carter [62] suggested that a hot-water bath might facilitate ceramic bracket debracketing. Patients were given a cup of hot water, supplied from a coffeemaker, and were asked to hold this water in their mouths for 1 min without swallowing. Subsequently, debracketing with suitable pliers was performed. Carter [62] emphasized that since 1986 no enamel fracture or any other iatrogenic damage occurred with this application in his clinic. Unfortunately, the exact temperature of this "hot-water bath" was not stated.

6.4. Ultrasonic debracketing

It was reported that the ultrasonic debracketing technique presents a decreased probability of enamel damage as well as a decreased probability of bracket fracture. Also, the residual adhesive remaining after debracketing can be removed with the same ultrasonic tip. Nevertheless, the debracketing time is the longest when compared with the mechanical or electrothermal debracketing techniques. It was reported that the debracketing time of the ultrasonic debonding technique is 38–50 s per bracket, when compared with 1 s per bracket with the mechanical debracketing technique. Furthermore, the contact between the "hard" ceramic bracket and the ultrasonic tip has been reported to cause wear of this expensive tip. During the ultrasonic

debracketing procedure, water spray is mandatory to prevent pulp damage. This method requires further testing and is not yet recommended for clinical use [11, 21]. No clinical studies were encountered upon a literature search.

6.5. Electrothermal debracketing

In 1986, Sheridan et al. [63] were the first who described electrothermal debracketing (ETD) for the removal of metallic brackets.

With ceramic brackets, ETD has been reported to cause a reduced incidence of bracket fracture. The reduced incidence of bracket fracture is ascribed to the small amount of force needed to break the bond after the heat-induced tip has promoted bond failure by softening/weakening the adhesive material. A relatively short debracketing time per bracket (2 ± 1 s) was reported. The possibility of pulp damage has been mentioned. Fortunately, no signs of irreversible pulp damage with ETD were described [11, 64–66]. Patient acceptance was generally positive [64].

6.6. Laser debracketing

Different types of lasers have been used for the debracketing of ceramic brackets [67]. The application of laser irradiation causes the softening of the adhesive material. This seems to be quite similar to ETD; however, with laser-assisted debracketing, the amount of thermal energy delivered to the ceramic bracket can be carefully controlled, thereby preventing the possibility of overheating [21, 68]. The time spent for ceramic bracket removal with the laser-based technique is 1–5 s. Debracketing forces are significantly reduced with lasers. As a result, enamel damage and bracket fracture risks are significantly reduced [67, 69]. The high cost of this device may be a disadvantage for the orthodontist [11, 21].

7. Last but not least

The physical properties as well as rebonding and debonding of monocrystalline and polycrystalline alumina ceramic brackets were reviewed. Ceramic brackets fabricated from polycrystalline zirconium oxide (zirconia) were not mentioned. Thus, we would like to add a short paragraph about these brackets for the interested reader.

Zirconia ceramic brackets, manufactured in Japan and Australia, have attracted interest in the past [12]. Nevertheless, problems concerning color (yellowish tint) and opacity (nontranslucency) were reported approximately three decades ago [12, 70]. Furthermore, no significant advantage of zirconia brackets over alumina brackets with regard to their frictional characteristics were reported [71]. Limited published research on zirconia (zirconium oxide) brackets exists [72].

8. Conclusion

In an increasingly demanding and litigious society, it is mandatory for the orthodontist to use carefully designed ceramic brackets. As a simple risk management strategy, ceramic brackets

that are not accompanied by detailed instructions for bonding and bracket removal should definitely not be used. These products might not have been exposed to appropriate, detailed testing procedures prior to their sale. Thus, be alert and keep updated!

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† I, Selma, dedicate this chapter to the memory of my grandmother, Anna Kirschner (1915–2004). My heart will always miss you....

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Chapter 3

Accelerated Orthodontics

Anand K. Patil and Amrit Singh Maan

Additional information is available at the end of the chapter

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Abstract

Clinical orthodontics is ever dynamic branch of Dentistry. Traditionally orthodontics was always considered as aesthetic treatment of face & also needed for proper oral oral function. This treatment may take up 2–3 years of total duration. The chapter describes changing trends in this aspect wherein we speed up the treatment by various methods thus reducing the overall time duration. These modalities include alteration in bio mechanics, pharmacological, chemical & by biological means. It is also cautioned here that the clinician has to take up these changing trends based on sound clinical knowledge & evidence based applicability.

Keywords: biology of tooth movement, acceleration of tooth movement, prostaglandins, regional acceleration phenomena, corticotomy

1. Accelerated orthodontics

Orthodontic treatment in the present day does not just require to meet the demands of creating the functional harmony in occlusion and improving the aesthetic outlook of but is should also be completed in the most efficient duration that is accepted by the patient and the orthodontist. We live in a fast-paced world where the treatment duration has clearly made the field of orthodontic treatment to revolve around it. Accelerated orthodontic tooth movement is not something that has recently emerged; it has been studied and tried out for many years. In an attempt of producing faster tooth movement during orthodontic treatment, there are numerous methods of accelerating tooth movements that have been introduced over the years which range from surgical means to the use of laser therapy. Now let us look at each method explained in this chapter.

2. Methods of accelerated tooth movements

We can categorise the methods of accelerated tooth movement into the following categories:

A. Pharmacological methods

B. Surgical methods

C. Physical methods

2.1. Pharmacological methods

Orthodontic forces cause a fluid movement in the periodontal ligament space and distortion of the matrix and cells. There is release of molecules which initiate bone remodelling for tooth movement [1]. There are a number of researches on pharmacological agents that act as biomodulators for increased orthodontic tooth movement. These are examples of such biomodulators:

- Prostaglandin E_2 and Prostaglandin E_1
- Misoprostol
- 1,25-Dihydroxycholecalciferol
- Parathyroid hormones
- Intravenous immunoglobulins

Prostaglandin E_2 (PGE_2) is an arachidonic acid metabolite is an often-tested substance to increase orthodontic tooth movement [2]. Animal studies have shown PGE_2 to increase tooth movement and facilitate bone resorption [3–6]. Camacho and Velásquez Cujar conducted a study that showed that it required repeated injections due to its short half-life [7]. Particular synthases that are required for the synthesis of PGE_2 could be targeted to control the production of the prostaglandins [8].

Another prostaglandin that has been reported to speed up orthodontic tooth movement is Prostaglandin E_1 (PGE_1). Prostaglandin E_1 (PGE_1) has also been seen to be induced by mechanical stress and cause bone remodelling, Patil and his co-workers had shown that even minimal amounts of PGE_1 injection had significant increase in tooth movement [9]. Due to the hyperalgesia that accompanies with the local injection of PGE_1, an analogue of it which is misoprostol was tried out. It was seen that it was effective in increasing orthodontic tooth movement (**Figure 1**) [10].

The parathyroid hormone (PTH) acts directly on osteoblasts and on osteoclasts indirectly by binding to the PTH type 1 receptor on osteoblasts. This causes the expression of insulin like growth factor 1. There is promotion of osteoblast survival, osteoblastogenesis and receptor activator for nuclear factor κ B ligand (RANKL) which induces osteoclast activation [2]. PTH facilitates bone remodelling in intermittent treatment by enhancing activities of osteoblasts and osteoclasts [11].

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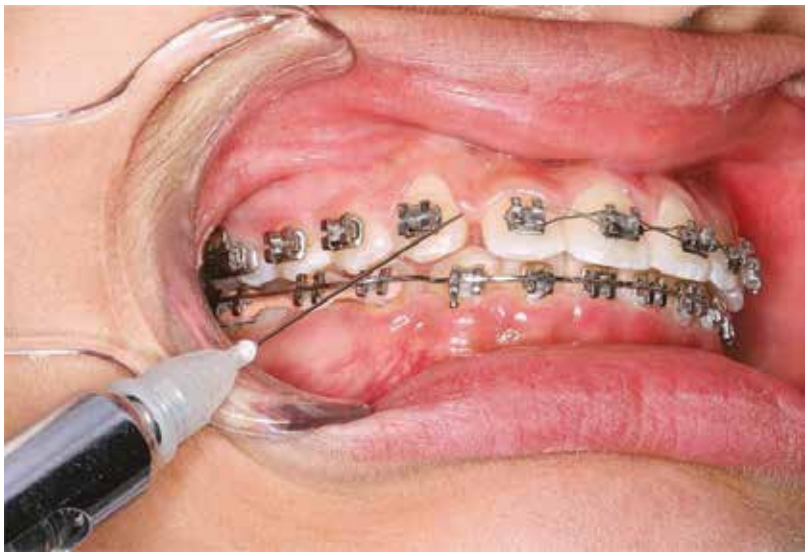

Figure 1. Injection of a biomodulator in the periodontium.

Calcitriol or 1,25-dihydroxycholecalciferol which is the most active metabolite of vitamin D acts in a similar fashion to PTH by facilitating osteoblastic proliferation and function [12]. Calcitriol facilitates alveolar bone remodelling which leads to increase in tooth movement while force application [6, 13].

Recently intravenous immunoglobulin (IVIg) preparations which are used in immunodeficient patients as replacement therapy. These preparations have been shown to induce COX-2 mediated PGE_2 and cytokine production [14, 15]. Future potential of these preparations could be used to modulate orthodontic movement via PGE_2 synthesis.

2.2. Surgical methods

Bichmalyr in 1931, put forward a surgical technique with orthodontic appliances for rapid correction of severe maxillary protrusion. First, wedges of bone were removed to reduce the volume for which the roots of the maxillary anterior teeth would require for retraction. Köle further looked into this technique in 1959 by including special movements like crossbite correction and space closure. He believed that he was able to move bony blocks using the crowns of teeth as handles as the blocks were connected by only less-dense medullary bone [16]. Currently there are few surgical methods being practiced, they are:

- Periodontally accelerated osteogenic orthodontics
- Piezocision
- Micro-osteoperforations

In 2001, Wilcko et al. had introduced a method which combines corticotomy surgery and alveolar bone grafting which is referred to as accelerated osteogenic orthodontics or recently

termed as periodontally accelerated osteogenic orthodontics (PAOO) [16]. This procedure which enables rapid tooth movement is due to a healing event that was described by Frost [17] and termed as regional acceleratory phenomenon (RAP).

RAP is the acceleration of the normal regional healing process from the original injury. It usually occurs after osteotomy, bone-grafting procedure, arthrodesis and fractures and there might be involvement and activation of precursor cells required for healing at the injury site. RAP can increase both soft and hard tissue healing processes by two- to tenfold [17]. It usually starts in the first few days of injury, peaks at the first or second month and may last for 3–4 months [16].

Orthodontic treatment can be started 1 week before or within 2 weeks after the surgery. Surgery begins with flap reflection and decortication with low-speed round burs. Bone graft is then laid over these areas of corticotomies. The flaps are then closed and sutured [18]. Several studies have been done related to corticotomies, an example is one by Uzuner and her co-workers where they showed that canine retraction assisted by corticotomy had reduced duration of retraction by 20% ratio [19]. PAOO has shown to have reduced treatment time, produce lower cortical bone resistance leading to reduced root resorption, enhancement of post-orthodontic stability, increased bone support since there is supplementation of the bone graft. However, PAOO still has risks since it is an invasive procedure and is expensive [20–24].

Since the corticotomy procedure is still invasive, Dibart et al. introduced a new minimally invasive method called piezocision. Piezocision involves microincisions which are confined to the buccal side that allows the use of piezoelectric knife and selective tunnelling which enables hard and soft tissue grafting [25]. Piezocision is usually done a week after orthodontic appliance placement. The procedure involves vertical incisions made buccally and interproximally. The mid portion of the incision between the roots enables the piezoelectric knife to be inserted. A piezotome is then inserted in the gingival openings that were made and piezoelectrical corticotomy of 3 mm is made. Hard or soft tissue grafts can then be added via a tunnelling procedure (**Figure 2**) [26].

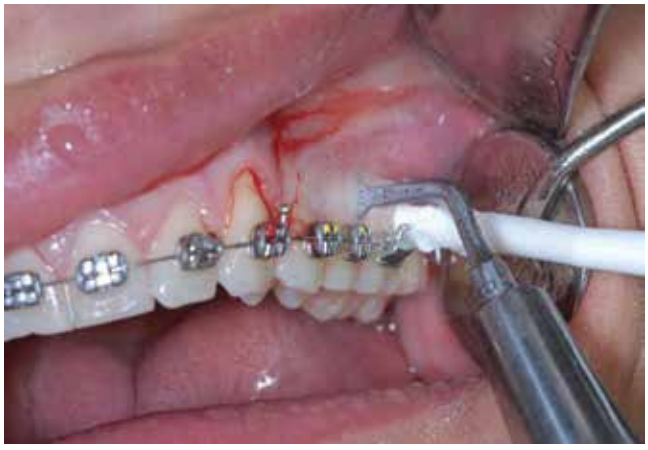

Figure 2. Piezocision.

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Piezocision can be used as an adjunct to treat a number of malocclusions and aid in rapid orthodontic treatment in adults. Since it is much more minimally invasive than corticotomy, it is having high degree of patient acceptance, short surgical time and has less postoperative discomfort [25, 26]. Dibart and coworkers in 2013 showed that there was an increase in the rate of tooth movement in their animal study and preliminary human studies are being conducted to correlate with the animal studies [26, 27].

To further reduce the amount of invasive nature of surgical intervention, a method called micro-osteoperforation (MOP). It is procedure in which small pinhole-sized perforations are created within the alveolar bone surrounding the dentition. This initiates cytokine release to call in osteoclasts to increase bone resorption. Thus, acceleration of tooth movement occurs during orthodontic treatment. The site of perforation is within the attached gingiva and close to the target teeth on the mesial and distal aspect of the roots of the teeth which will be moved. The most favourable place for placement of the perforation is the buccal cortical plate but lingual plate can also be approached with a contra-angled appliance. Two to four perforations are ideal amounts with depths of 3–7 mm into the bone [28].

In 2013, Alikhani et al. showed that MOP increased expression of cytokines for osteoclast differentiation, increased canine retraction, reduced orthodontic treatment by 62% with mild discomfort in patients [29]. In an animal study, Alikhani and co-workers found that the expression of inflammatory markers and bone resorption was significant. Their human clinical trial found distalisation was twice as much with MOP than the forces alone [30].

2.3. Physical methods

Despite all the attempts in making surgical methods being minimally invasive, they still remain as an invasive procedure. This had led to discoveries in other tools that can accelerate tooth movement during orthodontic treatment. The two most common physical methods used in the present day are:

- Vibratory stimulus
- Low level laser therapy
- Low-intensity pulsed ultrasound

Bone has the ability to respond to the mechanical stimuli that is applied to it as a mechanism to withstand functional activity. Rubin et al. showed the rate of remodelling in mechanically loaded long bones have been increased following vibrations or low level mechanical oscillatory signals [31]. In 2008, Nishimura et al. did an animal study which gave an insight on how resonance vibration could be able to accelerate tooth movement through the expression of RANKL in the periodontal ligament [32].

A novel device that was introduced by OrthoAccel Technologies is the AcceleDent device. The device has an activator and a mouthpiece. The patient bites on the mouthpiece component when in use. The activator which is extraorally positioned generates and transmits vibrations

to the teeth. It can provide 0.2 N of vibration at 30 Hz for 20 minutes. It was fabricated to work in tandem with existing bracket systems and not replace them. The device produces cyclic forces to move teeth within the alveolus via accelerated bone remodelling [33]. Pavlin and co-workers in 2015 showed low-level cyclic loading with AcceleDent increased the rate of orthodontic movement (**Figure 3**) [34].

Another treatment modality to speed up orthodontic tooth movement is by the use of low-level laser therapy (LLLT). Laser irradiation on tissues has a biostimulating effect with not more than 1°C rise in local temperature. Biostimulation potency of laser irradiation utilised by treatment are called low-level laser therapy [35]. Other than accelerating tooth movement, LLLT can enhance stability of orthodontic mini-implants [36], reduce post-adjustment pain [37], and induce bone growth in midpalatal suture area following rapid maxillary expansion [38].

Studies done by Fujita et al. and Yamaguchi et al. showed that LLLT enhances osteoclastogenesis on the compressed side of teeth being moved. There was stimulation of RANKL and macrophage colony-stimulating factor [39, 40]. Coordination of bone remodelling had been facilitated by RANKL and osteoprotegerin following orthodontic force with LLLT. LLLT stimulates bone formation on the tension side [41]. Kim et al. observed osteopontin localisation in the periodontal tissue in their study subjects, indicating LLLT may stimulate osteogenesis as well in orthodontic treatment [42]. Although much findings show LLLT stimulates osteoblast and osteoclast function, further studies are still required to optimise the effect of LLLT on tooth movement (**Figure 4**) [43].

Figure 3. AcceleDent device.

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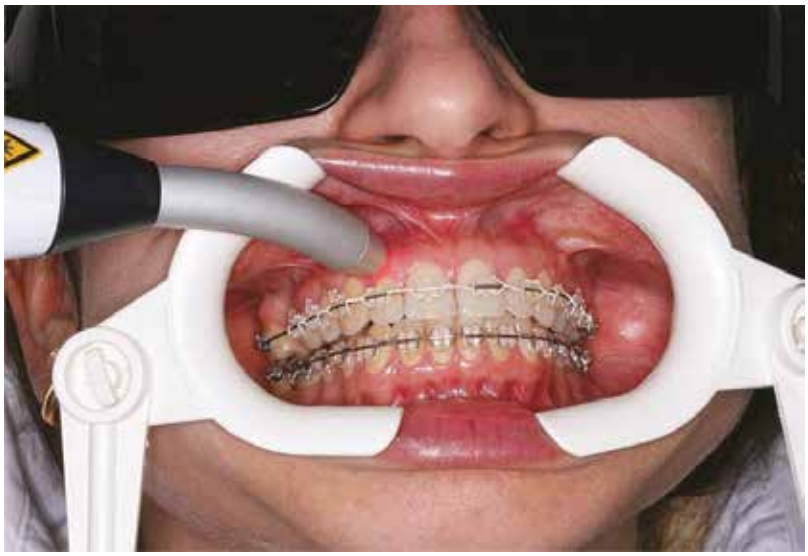

Figure 4. Low-level laser therapy.

Apart from physical agents, low-intensity pulsed ultrasound (LIPUS) has also been suggested. It uses mechanical energy which passes through the tissues as acoustic pressure waves [44]. This leads to biochemical changes at molecular and cellular levels. It can increase the healing of both soft tissue and hard tissue [45]. LIPUS is usually used at frequency pulses of 1.5 MHz with 200 μs pulse width, which is repeated at 1KHz a for 20 minutes a day with an intensity of 30 mW/cm^2 [46].

Recent studies on LIPUS using animal models by Xue et al. showed that there is induction of alveolar bone remodelling. The remodelling occurred due to an increase in the gene expression of HGF/Runx2/BMP-2 signalling pathway with LIPUS. This led to an increase in the velocity of tooth movement during orthodontic treatment [47]. El-Bialy et al. observed that LIPUS may reduce the root resorption that was orthodontically-induced by deposition of dentin and cementum to create a preventive layer from root resorption [48].

3. Conclusion

Over the years, the methods of reducing treatment time has risen along with its' demand. The options that are available on the orthodontist's plate are numerous ranging from surgical means to photostimulation. Much studies will still need to be done for newer methods to emerge and obtaining a clearer understanding on the methods that already exist. At present, the clinician should use all the knowledge obtained for deciding which treatment option is best for the patient to meet the healthcare needs of the patient and achieving an optimum treatment outcome.

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Chapter 4

The Role of Cytokines in Orthodontic Tooth Movement

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Additional information is available at the end of the chapter

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Abstract

One of the most important breakthroughs in the understanding of biological features of bones is the explanation of the role of cytokine in reshaping of the bone (remodeling) including the alveolar tooth bone exposed to the effect of the mechanical force during the orthodontic treatment. Since remodeling of the bone initiated by orthodontic forces is connected during its early stage with the inflammation of the surrounding tissue, the assumption was presented about the role of the pro-inflammation cytokine in the process of remodeling of the bone, primarily *IL-1β, IL-6,* and *TNF*. These cytokines are mediators in the reactions of the acute stage of inflammation, as well as in the processes of metabolism, stimulation of resorption, and inhibition of bone creation. In this chapter, we aimed to review the existing knowledge about the roles and dynamics of the change in these three cytokines simultaneously during the early stage of the orthodontic tooth movement.

Keywords: orthodontic tooth movement, reshaping of bones, cytokines

1. Introduction

When more than 100 years ago the world knew about the theory about the regulation of the tooth movement, cytokines were unknown to science [1]. The first experimental evidence that supported the assumption about cytokines being the potential regulators of the reshaping process (remodeling) of bones during the orthodontic treatment was obtained approximately 20 years ago [2]. Since then, until today, the efforts of researchers last in order to clarify molecular events with cytokines as mediators, which follow the orthodontic tooth movement.

The role of cytokines in the orthodontic tooth movement is considered in the context of inflammation, which occurs at the very beginning of this process as a reaction to the mechanical pressure and represents necessary precondition for the realization of all its subsequent levels. In the conjunction of mechanical and biological mechanisms, which move the teeth during the orthodontic treatment, cytokines are given great importance for their feature of transmission of biochemical signals among numerous cells of various kinds reacting to orthodontic forces. Binding themselves to specific receptors at membranes of these cells, cytokines cause in them the biochemical changes responsible for the signal transmission to corresponding genes in these cells and, consequently, to the change of gene expression in them. This orthodontic tooth movement causes the features of unusually complex processes, whose different degrees—each individually and all together—are regulated by the network of positive and negative feedbacks, in which cytokine molecules act as mutual activators or inhibitors [3].

2. Orthodontic tooth movement and force effect

Orthodontic tooth movement is a biomechanical process initiated by the effect of mechanical forces, which overpower the bio-elasticity of the support tissue [4].

The process of orthodontic movement of teeth is based on the transformation (remodeling) of periodontal tissues and is initiated by external forces and differs from the processes that occur during normal jaw function (dentition, chewing) [5]. On the basis of remodeling of periodontium, there are mechanisms, which transform the physical effort into various cell responses within the periodontal system, which primarily leads to the disturbance and then to the establishment of the periodontal homeostasis on a different basis [6]. These mechanisms provide the adaptation of the biological system of periodontium to the changed conditions emerged as a result of the effect of orthodontic forces.

Biomechanical mechanisms of the orthodontic tooth movement, because of their complexity, have been explained by various, but not mutually exclusive theories. Orthodontic dogma is considered to be the one according to which the movement of the tooth in the periodontal space occurs by the effect of two dominant forces: pressure force (compression) and tensile strength (tension) [7]. As a result of the pressure, there is resorption (suction), whereas as a result of tension to apposition (addition) of alveolar bone, the movement of the tooth occurs as a direct outcome of the reshaping of the tissue around the tooth root caused by forces. On basis of this, processes are vascular, and consequently, cellular changes of the dental tissue are caused by chemical mediators, which are created and released under the influence of orthodontic forces. Even though, in the context of this, we must not neglect the theory, which emphasizes bending of the bones as the basis of the orthodontic tooth movement [8], as well as the theory of bioelectrical signals, which emphasizes the importance of electric potentials, which are created in the tissue as a response to the application of the mechanical force [9].

Orthodontic forces lead to the change of the structural features of dental tissues at the level of cells, molecules, and genes. Mutual activities of tooth cells, periodontal ligament, bone, and bioactive substances (cytokine, chemokine, hormone, growth factor, enzymes, neuropeptides, and ligands) are necessary because they provide that during these changes, the tooth and

periodontal ligament remain clinically intact and surrounding bone is reorganized. The final outcome of these activities (the speed of orthodontic movement of teeth) may be defined as a phenotypic expression of numerous gene-controlled mechanisms, which connects the orthodontic tooth movement with hereditary basis, i.e., hereditary variations of factors which participate in this process [10].

3. Orthodontic tooth movement and biological mechanisms of reshaping of the mechanic-sensitive dental tissue

The cells of alveolar bones and periodontal ligament, gingiva, and tooth pulp react to the effect of orthodontic forces after the remodeling of extracellular matrix (ECM), which surrounds them [11]. Dental and periodontal cell responses to the applied mechanical force comprise interactions of intracellular and extracellular structural elements and mutual influences of the effects of various biochemical structures. The nature of changes in the process of reshaping is determined by the combinatory of interactions, which is different at different levels of the tooth movement [12]. The scheme no. 1 presents the main events in dental tissues, which follow orthodontic movement of the tooth.

4. Orthodontic tooth movement and the change in the structure of cytoskeleton

The function of all cells in mechanic-sensitive dental tissues is closely related to the ECM, which surrounds them and makes the corresponding microenvironment for cell activities, which emerge after the application of orthodontic force. The orthodontic treatment leads primarily to ECM periodontium deflection, which results in the changes of cytoskeleton structure of cells anchored in ECM. ECM is multicomponent tissue, which enables the transmission of mechanical signals to the corresponding cells and thus the occurrence of changes in the structure and function of a certain tissue [11]. The structural components of ECM (collagen, fibronectin, laminin, elastin, proteoglycans, hyaluronic acid, etc.) bind with the adhesive receptors at cells called integrins, via which the mechanical stimuli are transmitted into the cell causing the changes of cytoskeleton structures. The application of mechanical force outside disturbs the integrin receptors at fiber areas of periodontal ligament and gingiva and bone cells (osteoblast, osteoclast, osteocytes), and their adaptive response may increase or decrease the creation of integral elements of ECM in them and thus influence the change of the mass and morphological appearance of the bone [13].

5. Orthodontic tooth movement and reorganization of blood vessels

Blood vessels in periodontal ligament actively participate in the remodeling of dental tissues, which is related to the orthodontic tooth movement. Under the influence of mechanical forces,

the reshaping of existing and creation of new blood vessels at periodontal ligament occur. These processes occur via numerous signal paths, which are activated after the deflection of ECM, which surrounds the cells of endothelia of blood vessels. They are mostly established via integrin of endothelial cells and ECM structures, which surround the blood vessels [14] and lead to the organization of endothelial cells unto multicellular pre-capillary network [11]. The response of blood vessels of periodontal ligament to the effect of mechanical forces is expressed by increased permeability, which, on its side, increases the fluid outpouring from capillary into the interstitial space [15]. These blood vessels play an essentially important role in aseptic inflammatory reaction caused by mechanical forces, acting as a source of inflammation mediators (cytokine and neurotransmitters), which mutually react with endothelial cells of periodontal capillary network encouraging them to bind circulating leukocytes and influence their relocation into periodontal ECM.

6. Orthodontic movement of teeth and inflammation

The mechanical stimulus stemming from the orthodontic forces causes aseptic inflammatory reaction within periodontal tissues, which initiates biological processes, which are connected to the reshaping of the bone [16]. Even though in normal conditions the movement of teeth is a sterile process, the early stage of orthodontic tooth movement is observed as a type of tissue injury and it is accompanied by the acute inflammatory response.

Generally speaking, the acute inflammation is an initial stage of defense reaction of the mechanism to the tissue injury (mechanical, physical, chemical, nutritive, biological). It occurs fast and does not last long and it emerges as the result of numerous, complex, and mutually related processes via which certain proteins and cells are transmitted from blood to the damaged tissue and whose final result is the recovery of the tissue. The acute phase of the inflammation is characterized by vascular changes (vasodilatation and increased permeability of blood vessels) and consequently, plasma leakage (exudation) and relocation of leukocytes (extravasation) from blood into the injured tissue.

Immediately upon the application of orthodontic force, the disturbance of the microcirculation of periodontal ligament occurs, which results in the ischemia of local tissue, periodontal vasodilatation, and migration of leukocytes via capillaries of periodontal ligament. The changes are temporary and, by the rule, do not have pathological effects.

Even though inflammatory changes occurred during the orthodontic tooth movement are mostly the consequence of reactive processes in the support tissue, mechanical stimuli may be transmitted also to the tooth pulp and may initiate the inflammatory response within this dental tissue [17].

6.1. Orthodontic movement of teeth and inflammation mediators

Inflammatory response in orthodontic tooth movement is followed by the increased creation of inflammatory mediators (cytokines, prostaglandins, leukotrienes), enzymes (matrix metalloproteinase, lactate-dehydrogenase, alkaline phosphatase, aspartate-aminotransferase),

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growth factor (epidermal growth factor—EGF), and neuropeptides (P-SP substance, calcitonin gene-related peptide—CGRP), which indicates the participation and mutual communication of cells of immune, endocrine, and nervous system in the regulation of the bone remodeling [16, 18–22].

The primary role in the initiation of a series of biochemical processes that stimulate or inhibit cellular activities during the inflammatory changes, initiated by the effect of orthodontic

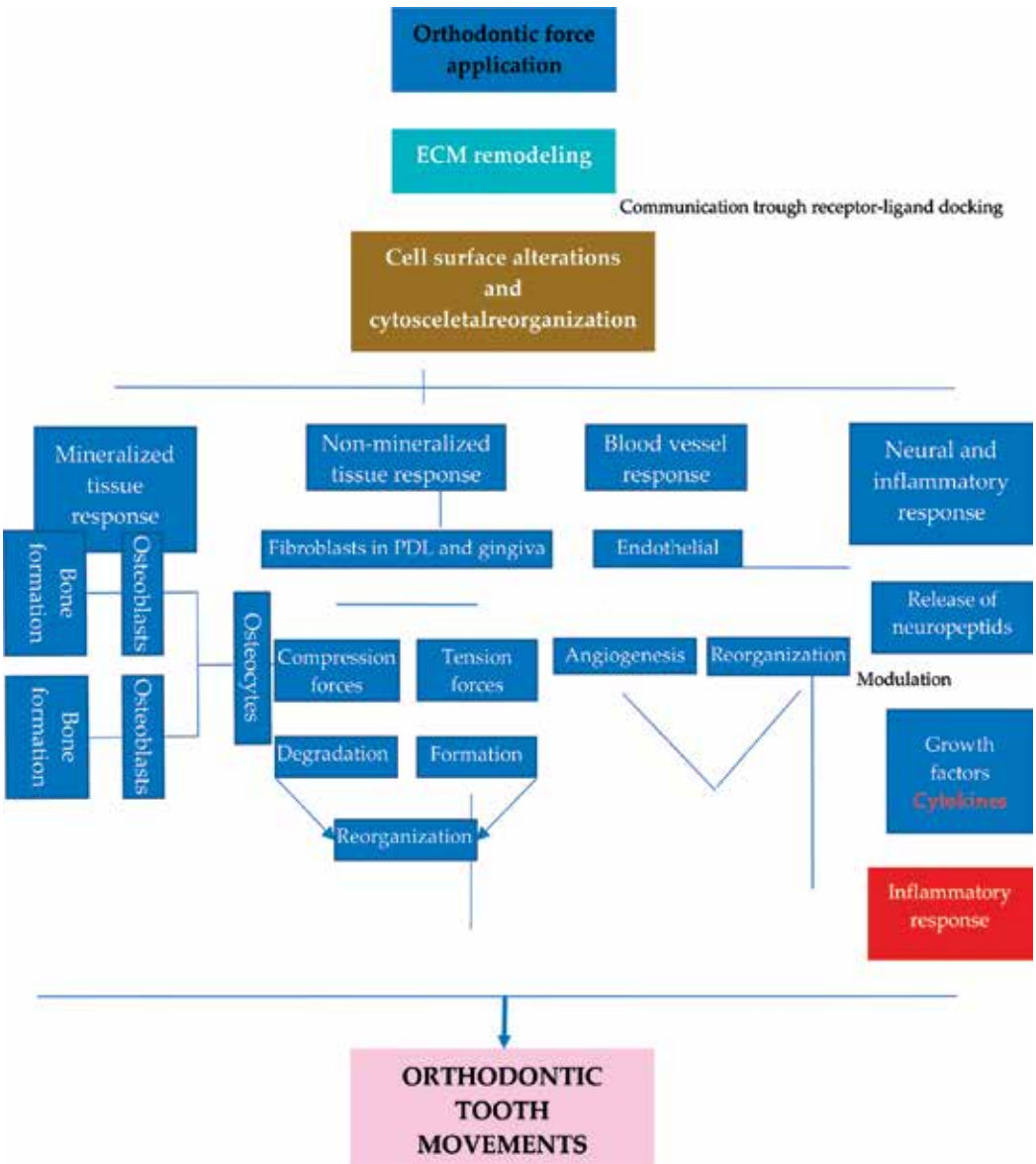

Figure 1. The sequence of events in dental tissues during orthodontic tooth movement. ECM—extracellular matrix; PDL—periodontal ligament.

forces, is attributed to cytokines [6]. Cytokines are small protein molecules, which transmit signals among cells. They are excreted by various cells as a response to external stimuli, and most frequently, they have a local effect. The effect of cytokines may be autocrine (to the cell which excretes it), paracrine (to other, adjacent cells), and endocrine (to distant cells). Cytokines express their effects by binding themselves to specific receptors at the cell membrane, which are affected by cytokines causing the biochemical changes responsible for the transmission of the signal to the corresponding genes in these cells and, consequently, to the change of the gene expression in them.

During the orthodontic tooth movement, cytokines are created by the inflammatory cells, which after the mechanical stimulus came outside widened capillaries of periodontal ligament [6, 18]. As the main regulators of the bone remodeling process during the orthodontic treatment, three cytokines are mentioned: interleukin 1 beta (IL-1β), interleukin 6 (IL-6), and tumor necrosis factor alpha (TNF-α) [6, 18, 23]. All three cytokines cause many local and systemic changes, which are the features of the acute stage of inflammation (**Figure 1**).

7. Orthodontic tooth movement and *IL-1β*, *IL–6*, and *TNF-α* effects

7.1. *IL-1β*, *IL–6*, and *TNF-α* and remodeling (resorption and apposition) of bones

The effects of *IL-1β*, *IL–6,* and *TNF-α* during the orthodontic tooth movement are going in a few connected directions and occur within the physiological process of remodeling of periodontal tissue after the application of mechanical forces. Judging by their concentration in gingival fluid, the creation of all three interleukins is already increased at the beginning stage of this process (12th and 24th hour) when leukocytes and fibroblasts of gingiva, periodontal ligament and alveolar bone make them as mediators of inflammation due to forces [6, 18, 23]. The maximum level is reached on the third day after the application of these forces [23].

IL-1β, *IL–6*, and *TNF-α* are osteotropic cytokines. They are released at the inflammatory spot and directly or indirectly (via substances whose synthesis and excretion they influenced) react with bone cells initiating the process of bone resorption [16]. Generally, it is believed that the bone resorption caused by inflammation is caused by local stimulation of osteoclast initiated by the effects of cytokines released from the infiltrated inflammatory cells [24, 25]. The process occurs in a complex signal manner via receptors of *TNF–α* (*TNF–R1*) at osteoblasts [24]. This receptor is activated by nuclear transcription factor *NF-κB*, so it is therefore called receptor activator of *NF-κB* ligand (*RANKL*) [26]. *RANKL* binds itself for the receptor at mature osteoclasts called *RANK*. Binding itself for *RANK*, *RANKL* may activate mature osteoclast and their precursors in the direction of oclastogenesis. It is prevented from doing so by natural antagonist osteoprotegerin (OPG), soluble receptor bait for RANKL, which prevents its binding for *RANK*, acting as a natural inhibitor of maturation and activation of osteoclast [27, 28]. For *TNF–α* and IL-*1β*, it is shown that together or independently from one another, *IL-1β* via *IL-1RI* receptors at osteoblasts may regulate the balance between *RANKL* and *OPG* in microenvironment of bones and mesenchymal tissue along the bone [29], not only

intensifying the expression of *RANKL* and contributing to the resorption of bones, but also that they may activate osteoclasts at RANKL independently [30]. There is evidence that *IL-1β* and *IL-6* released by osteoclasts themselves may cooperate with pro-inflammatory *IL-1β* and IL-6 in osteoclastogenesis [31].

The termination of the resorption of bones and initiation of its reformation comprises inhibition of the osteoclast function and stimulation of the activity of osteoblasts. The termination of resorption cycle includes the inhibition factors, which are created not only by surrounding cells but also by the osteoclasts themselves. They regulate negatively the activity of these cells causing their apoptosis and preventing their creation and simultaneously increasing the function of osteoblasts. This stage of normal bone remodeling is followed by lowering of the level of pro-inflammatory cytokines. The number of cells of inflammation, which are created by *IL-1β*, *IL–6*, and *TNF–α*, as well as the level of these cytokines in gingival fluid is decreased after 7–10 days since the beginning of the effect of mechanical forces [18, 23], which overlaps with the initial phase of reparation of periodontal tissue, which lasts for approximately 9 days [31]. During this stage, blood vessels are no longer excessively permeable [32]. In this stage the creation of transforming growth factor beta is intensified (TFG-β), insulin-like growth factor (IFG I and II), fibroblast growth factor (FGF), IL-10, etc. [33], which modulate the reactivity of osteoblasts and prevent the bone resorption [31]. Complicated interactions among these factors, with many of them still not being explained entirely, create the basis of the coordinated formation of a new bone at the resorption location.

7.2. *IL-1β*, *IL-6*, and *TNF-α* and orthodontic forces

Although the causal relationship of cytokine expression and orthodontic force is not entirely explained, it is believed that the direction and the nature of these forces affect the level of changes in the blood flow and thus the relationship of inflammatory mediators, which are expressed in periodontal tissues and gingival sulcus. The blood flow is decreased at the spot of compression (ligament compression) and increased at the spot of tension (ligament stretching); therefore, the response of the tissue at this location is greatly determined by the opposing forces that affect them. The inflammatory reaction occurs in both spots and the content of *IL-1β*, *IL–6*, and *TNF-α* is increased both in the zone of compression and in the zone of tension (in comparison with the control teeth), but the level of some of them in either of these zones is different [34, 35]. It is believed that these differences are the reflection of specificity of the process, which occurs at the location of effect of certain force during the orthodontic treatment [34]. Even though they are not completely harmonized, the data so far show that the level of pro-inflammatory cytokines in comparison with control teeth is generally higher in the compression zone than in the tension zone, which is connected to the role of these cytokines in the regulation of osteoclastogenesis mediated by RANKL (vide supra) and the process of bone resorption at the compression spot [34, 35]. Simultaneously, it is shown that the expression of anti-inflammatory cytokine *TGF-β* is greater at the tension spot than at the compression spot, which is attributed to its role in the process of inhibition of osteoclastogenesis and bone formation at the tension spot [35]. However, the balance between pro-inflammatory and anti-inflammatory mediators at the spots of compression and tension is still not studied enough.

The effects of cytokine in the response of the tissue to orthodontic forces are connected to the creation of nitrogen oxide (NO), which is known to be one of the important regulators of bone remodeling. For the creation of NO, the activity of two enzymes is necessary: inducible nitrogen-oxide synthesis (iNOS) and endothelial nitrogen-oxide synthesis (eNOS). The gene expression of these two enzymes is activated by pro-inflammatory (*IL-1β*, *TNF-α*) and anti-inflammatory (*IL-4*, *IL-10*, *TGF-β*) cytokines, which are created during the resorption and reparation of bones [36]. At the experimental model (rat), it was noticed that after the application of the orthodontic force iONS is the mediator in the bone resorption caused by the inflammation at the compression zone and eNOS in the bone creation at the tension zone [37].

Orthodontic forces express their effect to the dental pulp initiating the responses of fibroblast in it. Even though it is considered that the reactions of the pulp to the orthodontic treatment are very small, they still bring about changes in the blood flow and releasing of *IL-1β*, *IL-6*, and *TNF-α* from the pulp fibroblast, which results in its inflammation [17]. The process is specifically related to the pulp innervations and neurogenic mechanisms [38], and in the case of more expressed effect of mechanical forces may lead to the resorption of the tooth root [39].

7.3. *IL-1β*, *IL-6*, and *TNF-α* and "neurogenic inflammation"

The orthodontic tooth movement is accompanied by releasing neuropeptides from peripheral endings of sensor nerves, which permeate the dental pulp and periodontium, as well as from the inflammatory cells localized in the periodontal tissue. Released neuropeptides regulate the microcirculation of the pulp and mediate in inflammatory processes during remodeling of bones, characteristic for orthodontic tooth movement [19]. Such neural effect, which is generally called "neurogenic inflammation," is connected to the pain, which partially occurs during stretching and pressing of the tissue under the influence of mechanical forces and partially because of the interaction of numerous inflammatory mediators with local pain receptors [38, 40].

The main mediators of neutrogenic inflammation are neuropeptides, SP and CGRP, which are proven to have vasodilatation effect, increase vascular permeability and participate in the inflammatory processes related to the damage, and recovery of the tissue [41]. The increase of the level of these neuropeptides is recorded in gingival fluid immediately after the effect of orthodontic forces, which occurs simultaneously with the increase of the level of pro-inflammatory cytokines *IL-1β*, *IL-6*, *TNF-α* in this liquid [42]. Although the ratio of neuropeptides and cytokines included in the process of inflammation, which occurs during the orthodontic tooth movement, is still not entirely clear, the data show that SP and CGRP stimulate the excretion of *IL-1β*, *IL-6*, and *TNF-α* from fibroblast of human dental pulp, but they do not work synergistically [16, 42]. The neuropeptide SP is included in the resorption phase of the reshaping of the bone during the orthodontic tooth movement by stimulating the creation of RANKL in the cells of human dental pulp similar to fibroblasts [43]. The SP expression may be prevented by *TGF-β* [44], whose excretion overlaps with the initial stage of reparation of periodontal tissue.

The effects of neuropeptides to cytokines are not unidirectional [19, 38]. *IL-1β* and *TNF-α* secreted from inflammatory cells after the stimulation with SP lead to the creation of nerve

growth factor (NFG), which then leads to the increased production of SP and CGRP, which establishes the mechanisms of positive feedback during the inflammatory response [41].

7.4. *IL-1β, IL-6,* and *TNF-α* and other inflammatory mediators in periodontium

Apart from mutual interactive effects in the processes of inflammatory responses and bone remodeling during the orthodontic tooth movement, *IL-1β, IL-6*, and *TNF-α* frequently have effects in the combination with various other bioactive structures included in these processes.

After the primary inflow to the inflammation location and the initiation of an early stage of periodontal remodeling, these cytokines start the second tide of cytokine regulation of this process by "introducing" other relevant cytokines. It is shown that an early but not initial phase of the orthodontic tooth movement is followed by the increase of the level of *IL-8* in gingival fluid [6, 45], which is known to regulate inflammatory responses in periodontium in combination with other cytokines [46]. *IL-1β, IL-6,* and *TNF-α* stimulate the creation of *IL-8* in monocytes, macrophages, epithelial cells, and fibroblasts of periodontium, so that the IL-8 mechanism of feedback could initiate the creation of *IL-1β, IL-6*, and *TNF-α* [6], when periodontal system moves from resorptive to formative stage of bone remodeling. With IL-8 during the orthodontic treatment, the level of IL-2 also increases and it is considered to be the indicator of inflammatory activities in periodontium [47].

The increased expression of pro-inflammatory cytokines in human periodontium, due to orthodontic forces, is followed by prominent increase in the level of prostaglandin E_2 (PGE_2). This prostaglandin, which is created in various cells of mammals as one of the intermediary products of metabolism and arachidonic acid, is the mediator in the sustaining of local homeostasis, modulating numerous physiological processes including the inflammation. During the resorptive phase of bone remodeling caused by mechanical stress and initiated by acute inflammatory response, PGE_2 is created in cells of periodontal ligament (mechanically deformed osteoblasts and gingival fibroblasts), stimulating the creation of osteoclasts, which intensifies the bone resorption [16]. In this process, *IL-1β* and *TNF-α* express synergistic effects to the creation of PGE_2 stimulating the fibroblasts to the synthesis of this prostaglandin. The increased level of PGE_2 in the reaction results in the decrease of the expression of pro-inflammatory cytokines [48] and, consequently, the inhibition of the inflammatory response and stimulation of the bone formation. This dual role of PGE_2 (resorption on the one hand and bone formation on the other) is interpreted by the possibility of prostaglandin directing in different manners the bone cells: for resorption those in bone marrow and for the bone formation those at their surface.

The inflammatory response, which occurs during the orthodontic tooth movement, is followed by the increase of β_2 expression, microglobulin (β_2-*MG*), glycoproteins, which together with the pro-inflammatory cytokines initiate the process of bone remodeling [18]. β_2-*MG* occurs in soluble form in different bodily fluids of organisms including the gingival fluid and also is in the composition of the main histocompatible locus of I class (MHC class I), which is expressed at the surface of various cells, mostly lymphocytes and monocytes. In the process

of bone remodeling, after a mechanical stress, β_2-*MG* is included as a regulatory factor of the bone metabolism, with the function of a stimulator of the osteoclast activity in the resorption stage and the function of the increase of the binding for bone cells *IGF-I* in the stage of bone formation [49].

Apart from pro-inflammatory cytokines and other pro-inflammatory substances, during the orthodontic tooth movement to gingival fluid, various metabolites are released, too. For lactate-dehydrogenase (*LDH*) [21] and metalloproteinase 8 [20], it is shown that they increase their level or the activity simultaneously with pro-inflammatory cytokines, in approximately the same time, so it is considered that their existence reflects the increased periodontal remodeling caused by orthodontic forces primarily at early stages of this process. *LDH* is intracellular, cytoplasmic enzyme, which is released outside the cell under conditions of cellular necrosis or tissue degradation. It is believed that the increase of the *LDH* level at gingival fluid during the orthodontic tooth movement follows the process of bone resorption [21]; metalloproteinase 8 is the isoform of the enzyme of collagenase, which is released from periodontal fibroblasts due to the effects of mechanical forces. In the increased level in gingival fluid, it emerges during the initial stage of the orthodontic tooth movement, expressing the increased periodontal remodeling caused by these forces [20].

The damage of dental tissues caused by inflammation and its reparation are based on many elements and their coordination inside and outside cells. Even though there are differences between pathological inflammatory changes and those which accompany mechanically caused reshaping of tissues, the basic cellular responses to stimuli, regardless of their nature, express essentially the same properties [50]. Our researches of changes of individual integral parts of the immune system and ECM in normal and inflammatory gingiva [51–53] were the basis for the examination of the expression of IL-*1β*, *IL-6*, and *TNF-α* and their mutual relation in gingival fluid and tissue of gingiva of children and adults who underwent the orthodontic treatment. Analyzing the causes of gingival fluid and tissue of free gingiva of orthodontically treated teeth (experimental teeth) and their nontreated antagonists (control teeth) in four different moments in time ("zero" hour, 24th hour, 72nd hour, and 168th hour after the application of the separator), we have reached the knowledge about the dynamics of the change in the local cytokine network during the initial stage of orthodontic tooth movement and differences existing in the amplitude of these changes between children and adult examinees [54]. The results led us to assume that in the first moments of orthodontic treatment, the constitutive creation of pro-inflammatory cytokines is created and then it is overcome in the following time intervals by more expressive reaction of cells to the effects of mechanical force. The time coincidence of quantitative changes of *IL-1β*, *IL-6*, and *TNF-α* in gingival fluid and tissue expression of orthodontically treated teeth indicate that these three cytokines could be in specific interdependence during the early stage of the orthodontic tooth movement. Put into the context of the concept about the pharmacological modulation of the tooth movement, especially the aspect which leads the acceleration of the process of tooth movement in the connection with the local application of cytokine, the results could be very useful.

8. Conclusion

The early stage of the tooth movement is followed by the inflammatory response of the tissue to the effects of the mechanical force, which are conducted and regulated by pro-inflammatory cytokines *IL-1β, IL-6*, and *TNF-α*, acting as mutual activators and inhibitors.

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Chapter 5

Advances in Orthodontic Tooth Movement: Gene Therapy and Molecular Biology Aspect

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Additional information is available at the end of the chapter

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Abstract

Accelerated orthodontic tooth movement has been recently the topic of interest for orthodontic practitioners. Increased numbers of both clinical and research articles associated with the accelerated orthodontic treatment have been published in peer-reviewed journals in the last couple of years. Biochemical approaches such as administration of drugs, vitamins, and proteins and/or physical approaches such as surgery, vibration, and photobiomodulation have been widely reported and demonstrated the predicted outcome; however, the results are controversial. Very few reports addressed on genetic background of patients or utilization of molecular biological approach on the accelerated orthodontic treatment. In this chapter, we will discuss about biology of tooth movement and how the advances in gene therapy and molecular biology technology would shape the future of orthodontic treatment.

Keywords: gene therapy, molecular biology, orthodontic, accelerated tooth movement

1. Introduction

Orthodontic tooth movement is a biological process that requires the relay of mechanical loading to biological signals by periodontal ligament (PDL) and alveolar bone (AB) cells such as osteoblasts, osteocytes and osteoclasts. The mechanotransduction of signals involves dynamic cellular communication which allows for coordinated cellular response of alveolar bone remodeling and periodontal tissue homeostasis that occurs in response to orthodontic force. This complex process depends on adaptive tissue remodeling of periodontium for both anabolic and catabolic events. Compression and tension forces from orthodontic treatment create stress and strain to the PDL and AB cells and their surrounding extracellular matrices (ECM), which respond to the stress and strain from orthodontic forces by expressing and secreting

biologic mediators and inflammatory cytokines, osteoclast differentiation factors and ECM proteins such as collagen I, III, V and their modifying enzymes and proteases. These biomolecules, in turn, initiate the activation of fibroblasts, osteoblasts, osteocytes and recruitment and differentiation of osteoclasts leading to anabolic activities on the tension side and increased osteoclastic activity and low bone density on the compression side of tooth movement. These cellular and molecular events are strictly controlled at transcriptional, posttranscriptional and translational levels and the interference of these events affects the rate of tooth movement. Therefore, understanding the mechanism of cellular and molecular events of tooth movement will allow us to apply the cutting edge knowledge to improve clinical orthodontic practice using gene therapy or molecular biology approaches.

2. Orthodontic tooth movement models

Several models have been proposed for mechanism of initiation of orthodontic tooth movement as below.

1. Pressure-tension model: it was derived from the observation of experiments from animal models, in which a force of a given direction was applied to a tooth to create the tension and compression areas in periodontal tissues [1–4]. The histological studies demonstrated that bone was deposited on the alveolar wall on the tension side of the tooth in the presence of both heavy and light forces, with newly formed bone spicules followed the orientation of the periodontal fiber bundles. On the compression side, with the light forces, alveolar bone was resorbed directly by numerous multinucleated osteoclasts in Howship's lacunae (frontal resorption). In contrast, the periodontal tissues were compressed with heavy forces, leading to capillary thrombosis, cell death and the production of localized cell-free areas (hyalinization). Hyalinization phenomenon was later supported by several investigators [5–7]. At the hyalinization sites, osteoclastic resorption of the adjacent alveolar wall did not take place directly, but was initiated from the neighboring marrow spaces referred as 'undermining resorption' [8].

2. Bone bending/piezoelectric current model: it was observed that the deformation that occurred when an external load was applied to a long bone produced electrical current in the surface curvature of the bone. Increased bone concavity was shown to be associated with electronegativity and bone formation; while increased bone convexity was associated with electropositivity and bone resorption [9]. This model has major flaws given the fact that piezoelectricity does not require the presence of living cells. Dead bone produces the same effects, which appear to be generated by shearing forces acting on the collagen fibers of the bone matrix. Therefore, the stress-generated electrical potentials could be a by-product of deformation. In addition, the magnitude of the current is small and may not be sufficient to induce cellular changes [8, 10].

3. Neurogenic inflammation model: it was based on the assumption that orthodontic tooth movement was the result of inflammatory processes triggered by peripheral nerve fibers referred as neurogenic inflammation. This inflammation is characterized by the release of neuropeptides such as calcitonin gene-related peptide (CGRP) and substance P upon the stimulation of

afferent nerve endings [11]. A report showed that the nerve ending released the neuropeptides after periodontal ligament had been strained by the force applied to the tooth [12].

4. Fluid flow shear stress model: it was based on the concept that osteocytes respond to mechanical forces. Locally strain derived from the displacement of fluid in bony canaliculi of osteocytes is very important [13]. When loading occurs, interstitial fluid squeezes through the thin layer of the non-mineralized matrix surrounding the cell bodies and cell processes, resulting in local strain at the cell membrane and activation of the affected osteocytes [14]. With regard to orthodontic force, the force on the side of the tooth receiving orthodontic pressure creates shear stress and activates responses on osteocytes [15]. The shear stress on the osteocytes induces increased secretion of biological mediators from the osteocytes leading to activation of osteoclasts [16, 17]. At the same time, on the tension side, the increased pulling force on the periodontal ligament is transferred to the bone. The resulting deformation drives the fluid flow shear stress on the network of osteocytes. This shear stress induces osteocyte activation, and osteocytes respond by secreting signaling molecules that contribute to osteoclast recruitment and differentiation.

In addition, it has been shown that compressive force induces bone matrix deformation and microcracks; and the accumulation of microscopic cracks in the bone matrix may induce additional damage to osteocytes in the microcrack region [18]. Microcracks are more prevalent on the pressure-side than on the tension-side of the tooth, and it has been hypothesized that microcracks were the first damage induced by the orthodontic force to induce osteocyte apoptosis and bone remodeling. Osteocyte apoptosis has been observed at the pressure side in an experimental tooth movement model in animal models, which may be associated with the subsequent bone resorption [19, 20]. Therefore, the microcracks may play a role in the initiation of bone resorption on the pressure side of the tooth under the compressive force of orthodontic loading [21].

3. Molecular mechanism of orthodontic tooth movement

Although there are several models proposed to explain the events of orthodontic tooth movement, no single model could directly and comprehensively explain this process. The evidence from histological and animal studies has shown that this complex biological process is initiated from the application of mechanical forces onto the orthodontic appliances, which converts into the biological signals to stimulate mechanosensitive cells. (**Figure 1**) [22] Literatures showed that orthodontic force application induced physiologic adaptation of alveolar bone with small magnitude of reversible injury to periodontium [23]. Significant evidence suggests that when mechanical loading forces are relayed from the orthodontic appliances to the PDL and bone tissues, the mechanoreceptor cells percept the loading forces as shear stress and strain [24] as the tooth shifts its position in the PDL space resulting in compression and tension areas in PDL and bone tissues [25].

The sequence of biological events after loading of orthodontic force occurs as (1) fluid flow changes and matrix strain (**Figure 2**); (2) strain on mechanoreceptor cells (**Figure 2**); (3) cell activation (**Figure 3**); and (4) tissue remodeling leading to tooth movement (**Figure 4**) [15].

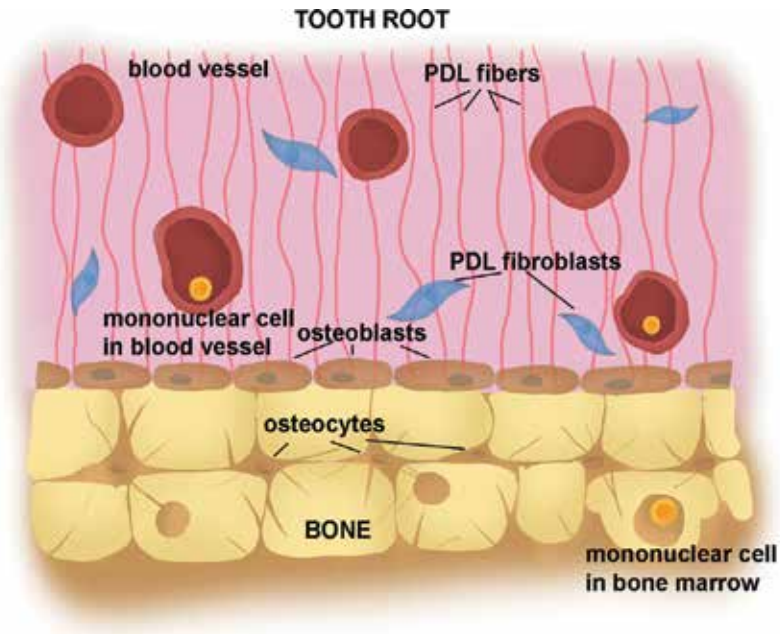

Figure 1. Illustration of cellular events of periodontal ligament and alveolar bone at non-loading state. Blood vessels and periodontal fibroblasts reside in between the periodontal ligament collagen fibers. Inactive osteoblasts line along the alveolar bone surface and quiescent osteocytes reside in their bony lacunae. Modified from Hatch [25].

The mechanoreceptor cells in periodontal tissue include osteocytes and bone lining osteoblasts in alveolar bone and fibroblasts in PDL. The final result as tissue remodeling occurs in both mineralized and non-mineralized ECM during the tooth movement [26]. Recent studies have indicated that osteocytes are capable of sensing strain in their bone lacunae following mechanical loading of the bone [21]. The mechanism of how osteocytes sense, transfer, and respond to mechanical strain remains unclear. Osteocyte processes have been shown to utilize integrins, gap junctions and ion channels to respond to mechanosensing external physical stimuli [27, 28]. Fluid flow-induced shear stress is the strain resulted from an immediate change in fluid flow in the lacunar-canalicular system leading to an increasing strain on the osteocytes. This shear stress can amplify the mechanical signals to the osteocytes [14, 29]. Several proteins such as integrins, connexin 43, osteopontin, and vitronectin, and several transcriptional factors such as c-Fos expression in the osteocytes are affected by loading forces [30–32]. In addition, the reduced number of primary cilia of osteocytes could affect their secretion of prostaglandins (PGs) and increased cyclooxygenase-2 (COX2) and RANKL/OPG ratio in osteocytes in response to fluid flow shear stress [33, 34]. Recent studies showed that osteocytes can induce both anabolic and catabolic bone signals in response to loading [35–37], yet the mechanism of how osteocytes switch from catabolic activity to anabolic activity is unclear. Under compression, osteocytes undergo apoptosis and are coupled with bone resorption [19, 38]. However, fluid flow shear stress may induce osteocytes to secrete anabolic bone proteins such as prostaglandin-E2 (PGE2) or nitric oxide (NO) [39, 40]. Several recent evidence demonstrated the significance of osteocytes during osteoclast differentiation and

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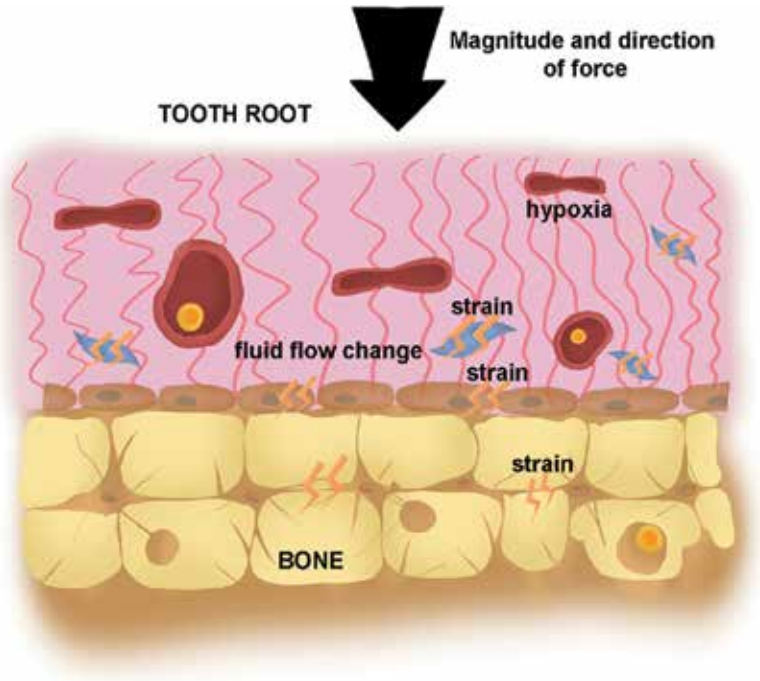

Figure 2. Initial cellular events in periodontal ligament after force loading during tooth movement. The blood vessels are squeezed then local hypoxia and fluid flow change are initiated from the loading force. The mechanical strain affects the periodontal fibroblasts and osteoblasts in the periodontal ligament space. The strain creates fluid flow shear stress and strain on the osteocytes in their bone lacunae. The mechanical strain induces secretion of inflammatory cytokines and biological signaling mediators including interleukins, prostaglandins, tumor necrosis factors, nitric oxide, growth factors, proteinases and cell differentiation factors. These mediators, in turn, activate the periodontal fibroblasts, osteoblasts and osteocytes. Modified from Hatch [25].

activation [41–43]. The osteocyte ablation *in vivo* caused a significant reduction in osteoclastogenesis and osteoclastic activity under loading forces, suggesting the important roles of osteocytes during orthodontic tooth movement [44]. Increased evidence supported the close association between osteocytes and osteoclasts during tooth movement. Experimental tooth movement in mice demonstrated increased expression of osteopontin [45], matrix extracellular phosphoglycoprotein (MEPE) [46], and receptor activator of nuclear factor-kB ligand (RANKL) [43, 47] in osteocytes. These proteins play important roles in osteoclastic activity and osteoclastogenesis because deficiency of these proteins results in significant reduction or absence of the osteoclasts and increased bone mass in the animals [43, 48]. Osteocyte apoptosis occurred abundantly on the compression side of tooth movement in 1 day after loading, and then an increased number of osteoclasts were observed until day 3, resulting increased tooth movement by day 10 [49]. It is speculated that apoptotic osteocytes may release signaling proteins such as RANKL and interleukin (IL), to osteoclast precursors, and initiate osteoclastogenesis. In contrast, when subjected to fluid flow sheer stress, osteocytes secrete NO and PGE2, which these proteins have potent, anabolic, and direct effects on osteoblasts [40, 50, 51]. PGE2 expression increased in loaded bone tissue [52]. NO secreted from osteocytes promotes osteoblast differentiation, and

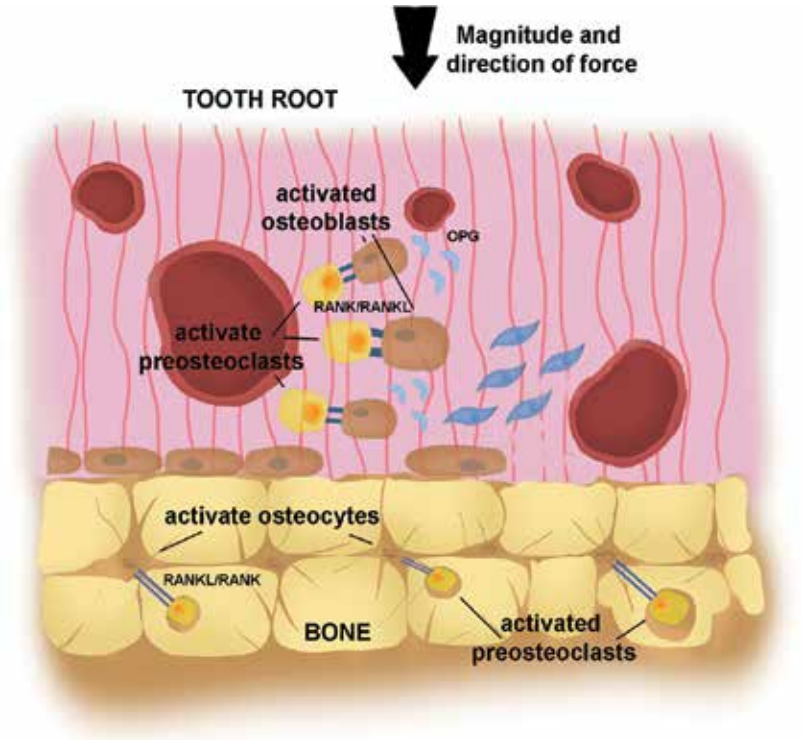

Figure 3. Intermediate cellular events in periodontal ligament during tooth movement. The blood vessels dilate due to the response to the released mediators and cytokines. The activated fibroblasts, osteoblasts and osteocytes are ready to secrete M-CSF and RANKL to activate preosteoclasts from blood and bone marrow. In addition, the activated osteoblasts release OPG to act as competitive decoys for RANKL. The PDL fibroblasts release MMPs to degrade collagen fibers in the periodontal ligaments. Modified from Hatch [25].

plays an important role in bone formation during loading [40, 53]. NO can influence bone mass and simultaneously inhibit osteoclast activity [54]. Increased NO production by osteocytes after mechanical stimulation by fluid flow modulates apoptosis-related gene expression suggesting that NO maintains osteocyte viability [55].

Beside osteocytes, preosteoblasts are also responsive to mechanical force. Mechanical force loading triggers several cell signaling pathways in osteoblasts such as calcium (Ca^{2+}), NO, IL1β and adenosine triphosphate (ATP) in a short period of time [24, 56, 57]. NO and IL are potent mediators secreted during orthodontic tooth movement [58, 59]. Preosteoblast differentiation can be induced on the tension side of tooth movement via integrin/focal adhesion kinase signaling and Ca^{2+} channels [60, 61]. Fluid shear stress can trigger Ca^{2+} signaling pathway and promotes ATP release, PGE2 secretion and proliferation of osteoblasts [24, 57]. While on the compression side, reduced blood flow in PDL and localized hypoxia occurs. The reduction in O_2 tension stabilizes hypoxia inducible factor-1 (HIF-1), a transcription factor that activates vascular endothelial growth factor (VEGF) and RANKL expression in PDL fibroblasts and osteoblasts leading to osteoclast differentiation and favoring bone resorption in areas of compression [62–64]. As mentioned above, inflammatory cascade is important for orthodontic

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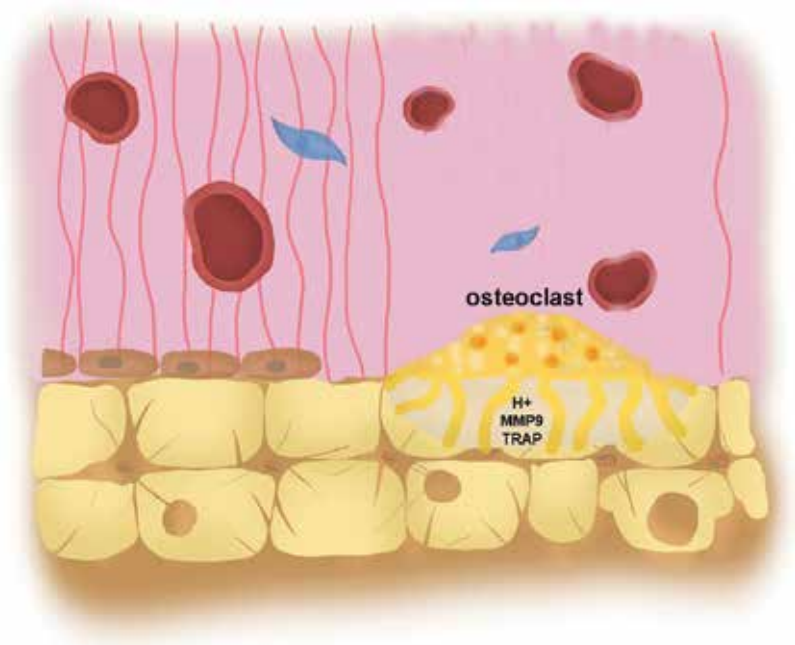

Figure 4. Late cellular events in periodontal ligament and alveolar bone front during tooth movement. The activated osteoclast is derived from the fusion of preosteoclasts, creates ruffle border to seal the bone surface area and releases MMP9, TRAP and acid to resorb bone matrix and minerals. Apoptotic osteocytes also release the biomolecules and mediators to activate osteoclast recruitment for bone resorption leading to tooth movement. Modified from Hatch [25].

tooth movement. During the process, inflammatory cytokine such as IL-1β, PGE2, tumor necrosis factor-alpha (TNF-α) and NO are secreted from preosteoblast in PDL and osteocytes in bone lacunae during the orthodontic tooth movement [59, 65, 66]. Compression is associated with elevated COX-2 which catalyzes production of PG, including PGE2, from arachidonic acid [67, 68]. Administration of PGE2 into alveolar bone of mice induces both osteoclasts and osteoblasts [26]. During orthodontic tooth movement, pain sensation occurs and, coincidentally, substance P and calcitonin gene related peptide (CGRP) are induced to be secreted during the tooth movement. These neuropeptides can enhance cellular secretion of inflammatory cytokine and in turn increase vasodilation and permeability of surrounding blood vessels [69–72]. Several evidence showed that the inhibition of inflammation hindered tooth movement [73, 74], while inflammation in the alveolar bone promoted tooth movement [75, 76].

Osteoclasts are the major key cells that play significant roles during tooth movement. Osteoclasts are multinucleated giant cells which are formed by the fusion of mononucleated osteoclast precursors derived from hematopoietic origin and function to resorb the alveolar bone during tooth movement. The osteoclast progenitor cells require macrophage colony stimulating factors (M-CSF) for their proliferation and survival. M-CSF is a secreted cytokine by osteoblasts and affects osteoclast progenitors. The RANK/RANKL/OPG system has been a crucial mechanism in osteoclastogenesis during bone resorption and tooth movement [77–79]. Receptor activator for nuclear factor κB (RANK) is a transmembrane protein and a member of tumor necrosis factor

receptor family that is expressed on osteoclastic precursors, preosteoclasts and osteoclasts. Receptor activator for nuclear factor κB ligand (RANKL) is a transmembrane protein and is a member of the tumor necrosis factor superfamily that is expressed on preosteoblasts, osteoblasts and osteocytes [80]. RANK is the receptor for RANKL and the binding between both of them stimulates the differentiation of preosteoclasts into mature osteoclasts. Osteoprotegerin (OPG) is a soluble extracellular tumor necrosis receptor protein that is secreted by preosteoblasts and osteoblasts. OPG is a decoy receptor for RANKL in regulating bone metabolism and inhibiting osteoclastogenesis and bone resorption. RANKL/OPG ratio is an important determinant of bone mass and skeletal integrity and also an indicator for the osteoclast function [78, 79]. Increased evidence demonstrated the direct association of tooth movement and activities of osteoclasts. Accelerated osteoclast resorption in alveolar bone of OPG deficient mice was observed during tooth movement [81] while inhibition of RANKL or deletion of RANKL in mice resulted in suppression of tooth movement [47]. In addition, local administration of M-CSF resulted in modulation of rate of tooth movement in animals [82].

Overall, the mechanism of tooth movement is complex and need strictly coordinated regulation of PDL, osteoclasts, osteocytes and osteoblasts. It is very challenging clinically to apply optimal force onto the tooth to avoid hyalinization. Clinically, tooth movement in patients is a result of combination of undermining and frontal resorption [83]. Compression sides involve increased expression of PGE2, TNF-α and IL-1β. PGE2 promotes osteoblast and osteoclast differentiation and activity. Activated osteoblasts secrete RANKL and OPG to trigger osteoclast differentiation and activity. TNF-α and IL-1β promote osteoclast differentiation and activity. In addition, matrix metalloproteinases (MMPs) expression is increased as well as the expression of M-CSF [84]. Loading compressive force affects osteocytes to upregulate the expression of connexin 43 [85], endothelial nitric oxide synthase (iNOS) [50], osteopontin [45], SOST [86] and RANKL [47]. These molecules recruit osteoclast precursors and activate osteoclasts to resorb the alveolar bone on the compression side. While on the tension side, increased expression of transforming growth factor-β (TGF-β), a potent ECM growth factor, was detected [87]. Several anabolic molecules such as bone sialoprotein (BSP) [88], collagen I (ColI) [89], vascular endothelial growth factor (VEGF) [84, 90], tissue inhibitors of metalloproteinases (TIMPs) [91], insulin-like growth factor (IGF) and its related receptor [92], heat shock protein 27 (HSP 27) [93] and ATP [94] were increasingly expressed on tension side during tooth movement. IL-6 around the osteocytes under loading can promote its signaling toward osteoblast pathway [53]. The presence of TIMPs around tension side is speculated to control the activity of MMP and remodeling pattern in alveolar bone. The anabolic events such as increased osteoblast activity and decreased osteoclast activity occur on the tension side of tooth movement.

4. Studies on genetic manipulation of tooth movement

Administration of proteins that affect or activate osteoclasts could be a direct approach to modulate tooth movement though the dosage and side effects such as root resorption are factors of consideration. With modern advanced technology, the manufacturer can generate a large amount of human recombinant proteins for therapeutic purposes. However, the life span

of these proteins once administered in human body is short and may not reach therapeutic level [95]. Gene therapy is a therapeutic approach that uses genes to treat or prevent diseases. Gene therapy is designed to introduce nucleotides into the cells to compensate for mutated genes or to restore the normal protein. If a mutation causes a crucial protein to be defective or missing, gene therapy may be able to introduce a normal copy of the gene to restore the function of the protein. After integration of the genes that encoded the target protein into the patient's genetic machinery, gene therapy can allow the body to produce the required protein constantly so the level of protein will be constantly high at therapeutic level [96]. The concept of gene therapy includes cloning of selected DNA/RNA fragments into a delivery system in order to administer into the host or patient. The delivery system could be viral vectors or non-viral vectors such as liposomes, peptides, polymer particles, gene gun and electric perforation [97]. The clinical application of gene therapy can be achieved with *in vivo* or *ex vivo* approaches. The *in vivo* gene therapy will include injection of vectors into the patient directly while the *ex vivo* approach includes the introduction of vector into the cells then the transfected cells are transplanted back into the patient [98–100].

Recently gene therapy has been approved to be implemented in medicine. The U.S. Food and Drug Administration (FDA) regulates all gene therapy products in the United States and oversees research in this area. In medicine, the FDA recently approved gene therapy for the treatment of some types of leukemia and inherited blindness [101]. Several experiments of gene therapy in dentistry involved orofacial pain, squamous cell carcinoma, tooth and bone regeneration, salivary gland disease and orthodontic treatment [102].

The gene therapy experiments in orthodontic treatment are still limited to cell cultures or animal experiments [103]. The purposes of previous gene therapy in orthodontic treatment were to investigate the possibility of acceleration of tooth movement or reduction of root resorption by modification of osteoclast differentiation factors such as RANKL or OPG [104–109]. The first attempt for gene therapy in orthodontic treatment aimed to transfer OPG gene into periodontal tissue to reduce osteoclast activity and inhibit tooth movement. The gene transfer approach using a hemagglutinating virus of Japan (HVJ) envelope vector carrying mouse OPG messenger RNA (mRNA) was performed in rats for 21 days of tooth movement. The vector solution was administered into rat's palatal gingiva by infiltration injection. The result showed that local OPG gene transfer reduced the number of osteoclasts and decreased tooth movement by 50% in rats in the experimental group compared to the ones in the control group. The effect of OPG gene transfer was local and did not affect bone mineral density of tibia of the animals [105]. The same group of investigators performed another experiment using the same system to transfer mouse RANKL mRNA to periodontal tissue to activate osteoclastogenesis and accelerate tooth movement in rats. The results showed that local RANKL gene transfer induced increased numbers of osteoclasts and accelerated tooth movement by approximately 150% in the rats in the experimental group compared to the control group. The effect of RANKL gene transfer was local and did not elicit any systemic effects. Interestingly, the number of osteoclasts was reduced time dependently after gene transfer [104]. Another group of investigators compared corticotomy with gene therapy using a hemagglutinating virus of Japan envelope vector containing mouse RANKL mRNA in rats for 32 days. The results showed increased level of RANKL protein 3 folds in the gene therapy group and 2 folds in the corticotomy group after 10 days; however, the level

of RANKL protein was maintained in the gene therapy group but not in the corticotomy group. The number of osteoclasts in the RANKL gene therapy group was significantly higher at day 10 with or without tooth movement compared to the tooth movement only group. The tooth movement distance was 2 times more in the RANKL gene therapy group and 1.5 times in the corticotomy group; however, the rate of tooth movement slowed down in the corticotomy and controls groups but was constant in the RANKL gene therapy group. It was concluded that gene therapy was an alternative treatment for corticotomy to accelerate tooth movement and the efficacy of treatment was higher than corticotomy to accelerate tooth movement [106]. The OPG gene transfer experiment was performed by another group of investigators using the same viral envelope packaging and delivery system to investigate the inhibition of orthodontic relapse in rats. The first molars in the rats were moved mesially for 3 weeks then the springs were removed to generate orthodontic relapse in the rats. The rats received OPG gene therapy then were observed for 2 weeks. The results showed that relapse was significantly inhibited 2 times compared to the mock and control groups. The bone mineral density and bone volume fraction of alveolar bone were significantly increased in the gene therapy group compared to the mock and control groups. No difference of bone mineral density and bone volume fraction of tibia was found among groups. The investigators stated that local OPG gene therapy to periodontal tissues could inhibit relapse after orthodontic tooth movement via osteoclastogenesis inhibition [110]. The same group of investigators further investigated the effect of local OPG gene therapy on orthodontic root resorption with the same design of experiment. They utilized a microcomputed tomogram and histological analyses. The result showed no difference between root resorption at the beginning and the end of tooth movement in the OPG gene therapy group. However, the repair of root resorption in the gene therapy group was higher than other control groups [107]. Another study investigated the effect of local OPG gene therapy using mesenchymal stem cells as carriers for plasmid containing OPG mRNA. This cell mediated OPG gene transfer was generated by insertion of plasmid containing OPG mRNA into the mesenchymal stem cells and the cells were injected into the animals. The result showed that the cells containing OPG package grew in the animals' PDL and the number of osteoclasts, level of RANKL and bone resorption were reduced significantly after single injection. The level of OPG was highest in the gene therapy group [108].

Gene therapy is a promising treatment option for a number of diseases (including inherited disorders, some types of cancer, and certain viral infections). This approach is still in the developing process as an alternative approach to treat deformity or disease that conventional method could not achieved. Although many clinical trials have shown the efficacy of the treatment, the technique remains risky and is still under processes of investigation to make sure that it will be safe and do not elicit any systemic or hereditary effects for the patients.

5. Future of genetic manipulation of tooth movement

With the rise of advanced technology in biomedical engineering and medicine, gene therapy is no longer a science fiction. Several gene therapies have been approved to treat many conditions and deformities not only in the United States but worldwide [111]. In the past decade, gene

targeting using endogenous microRNA (miRNA) has emerged as a powerful tool for targeted gene delivery. miRNAs are short, noncoding and highly conserved RNA sequences that tightly regulate the expression of genes by binding to their target sequence in the corresponding mRNAs [112, 113]. Majority of miRNA biogenesis involves transcription by RNA polymerase II to generate primary microRNA (pri-miRNA) followed by Drosha (RNase III enzyme) processing, which produces precursor miRNA (pre-miRNA). The pre-miRNA is transported to the cytoplasm via exportins/RanGTP complex. In the cytoplasm, the pre-miRNA is cleaved by another RNase III enzyme called Dicer to generate mature miRNA. The mature miRNA then forms a microRNA associated RNA-induced silencing complex (miRISC) with Argonaute proteins. The complex is steered to the target mRNA via base pairing with the target sequence of the miRNA. The degree of perfect complementarity at nucleotides 2–8 (binding sequence) in the 5′-end of the miRNA is essential for a successful action of the RISC complex. Depending on the extent of complementarity with the target sequence, gene expression is repressed either by inhibition of translation or by cleavage of the corresponding mRNA [114]. The process of gene therapy using endogenous miRNAs involves selection process of miRNA candidates, design of expression cassettes if constant expression is needed, selection of delivery carrier, and evaluation of system in cells, animal models and clinical trials [114]. Several miRNAs have been reported for their expression and roles in PDL and alveolar bones [115–118]. Under loading, several miRNAs in PDL and alveolar bone respond to the loading force and orientation of forces in different pattern of expression [119–121]. miRNA-21 has been shown to have critical roles in PDL, osteoblasts and osteoclasts [120, 122–127]. In addition, miRNA-21 deficient mouse demonstrated delayed tooth movement compared to the control mice via inhibition of osteoclastogenesis [127]. miRNA-29 was reported as a crucial miRNA for alveolar bone remodeling during tooth movement due to its expression under different orientation of loading forces and its expression profile in crevicular fluid during tooth movement in human [121, 128]. miRNA-29 expression in human PDL was up-regulated under compression but down-regulated under stretch force orientation [121] and its expression on crevicular fluid increased along the course of tooth movement [128]. Moreover, miRNA-29 sponge transgenic mice demonstrated delayed tooth movement due to the decreased numbers of osteoclasts [129]. These microRNAs could be a target candidate for gene therapy for orthodontic tooth movement. There are viral and nonviral delivery systems in clinical trials for gene therapy. Among viral vector system, lentiviral vector-based system has been developed and tested for its safety for more than 10 years. Non-integrating lentiviral vector have been investigated as a means of avoiding insertional mutagenesis. However, there is a disadvantage of this approach regarding the short-lived of the vectors in dividing cells [130]. Nonviral gene delivery systems (nVGDS) have great potential for therapeutic purposes and have several advantages over viral delivery including lower immunogenicity and toxicity, better cell specificity, better modifiability, and higher productivity. However, there is no ideal nVGDS; hence, there is widespread research to improve their properties [97]. The nVGDS system includes chemicals, peptides, liposomes, and polymers [97]. Exosomes are small (30–150 nm in diameter) extracellular vesicles that are formed in multivesicular bodies and are released from cells as the multivesicular bodies fuse with the plasma membrane. The exosomes were proposed to be used for delivery of miRNAs, protein and oligonucleotide complex [131], and were found to be cell secreted from osteoclasts [131] and in gingival crevicular fluid during the course of tooth movement [128].

Another genome editing system that has recently gained attention in research and clinical application is CRISPR/Cas9 system. The CRISPR/Cas9 system is based on CRISPR (clustered regularly interspaced short palindromic repeats) sequence and CRISPR associated (Cas) gene mechanism that are crucial for innate defense mechanism in bacteria and archaea enabling the organisms to respond to and eliminate invading genetic materials from their phages [132]. The CRISPR/Cas9 system consists of two key molecules that introduce a mutation into the DNA. First, Cas9 is an enzyme that acts as a pair of DNA scissor. It cuts the two strands of DNA at a specific location in the genome so the genome editing could be performed either addition or removal. The other molecule is guide RNA (gRNA) which consists of a small piece of predesigned RNA sequence (∼20 bases long) located within a long RNA scaffold. The long RNA scaffold binds to DNA and the gRNA sequence guides Cas9 to edit the specific part of genome. gRNA sequence is designed to be complementary to the target DNA sequence in the target gene in the genome. gRNA sequence consists of short palindromic repeats and the sequences that complement with the target genes. The target sequences should be present close to protospacer adjacent motif (PAM) sequence which increases the specificity of Cas9. After Cas9 nuclease enzyme site specifically cleaves double stranded DNA activating double-strand break repair machinery. If the DNA repair template is provided, the piece of DNA repair template will be inserted into the sequence of target genes [133, 134]. With this mechanism, the plasmid containing gRNA, Cas9 sequences, TracrRNA (transactivating CRISP RNA) and DNA repair template sequence can be introduced into cells or embryo of the animals by viral or nonviral delivery system [135]. Until now, there is no CRISPR/Cas9 experiment involving orthodontic tooth movement, however, this technology has been implemented in recent mineralized tissue research [136–139]. Future directions of gene therapy include the enhancement of the lentiviral vector-based approaches, fine tuning of the conditioning regimen, and the design of safer vectors or nonviral vector delivery system. In orthodontic field, the gene therapy approach will need several fundamental cell culture and animal experiments to demonstrate the safety and efficacy of the treatment concept. Clinical trials are required as the next step to ascertain the clinicians and patients for efficacy of the treatments.

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Conflict of interest

The authors declare no conflict of interest.

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Chapter 6

Micro-Osteoperforations

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Additional information is available at the end of the chapter

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Abstract

The researchers have tended to study on methods which will shorten orthodontic treatment periods due to the fact that orthodontic treatments are long-term treatments with increased side-effect potentials depending on the use of orthodontic force. There is a need for shortening orthodontic treatment time besides minimizing the side effects and achieving permanent results. Therefore, local or systemic application of various chemical agents, some physical-mechanic stimulus, and surgery-assisted methods are used. Surgically assisted methods have a longer clinical history with more predictable and consistent results among the acceleration methods of tooth movement. On the other hand, they include some complication risks due to the interventions as they are invasive methods, which is accepted as the disadvantage of these methods. Outstanding with its ease of implementation and repeatability, "micro-osteoperforation," which is less invasive than other surgical methods, is an up-to-date technique. Animal studies show that micro-osteoperforations increase orthodontic tooth movement rate by enhancing the cellular response. These findings are also supported by a limited number of clinical studies. Although micro-osteoperforations were reported to be included in tooth acceleration techniques as a trustable and applicable method in clinical routine, issues such as application techniques, possible side effects, and combining with different mechanics should be evaluated with further clinical studies.

Keywords: micro-osteoperforations, accelerated orthodontics, tooth movement, bone remodeling

1. Introduction

Many orthodontic patients complain about the physical and social discomfort which is associated with prolonged use of fixed appliances [1]. There are also numerous studies which report that dental and periodontal complications such as apical root resorption, subsequent

gingivitis, periodontitis, enamel demineralization, increased levels of dental caries, and open gingival embrasure spaces may occur during orthodontic treatment [2].

However, a major challenge in orthodontics is to shorten treatment time by avoiding undesirable side effects without compromising treatment outcome. The rate of orthodontic tooth movement is primarily determined by the remodeling of tissues surrounding the roots; this in return is under the control of molecular mechanisms regulating cellular behaviors in the alveolar bone and periodontal ligament [3]. Assuming that the clinician optimized mechanics and cooperation for any patient, the main factor controlling the treatment time and rate will be the patient's biological response to the orthodontic forces [4, 5]. Therefore, identifying and controlling the cellular regulators are essential to shorten orthodontic treatment time safely.

The concept of accelerating tooth movement has received increased attention recently [6] with the introduction of various clinical techniques; including local injection of cellular mediators [7], physical or mechanical stimuli [8] and surgically assisted orthodontics [9, 10]. Local or systemic pharmaceutical administration and physical or mechanical stimulation methods might not be applicable to daily clinical practice because of their possible side effects and their outcomes that await validation [11]. Over the past decade, the regional acceleratory phenomenon induced by surgical trauma has received emphasis for accelerating orthodontic tooth movement and reducing the treatment time [11]. However, many techniques which are surgically assisted involve considerably high surgical trauma limiting application of the technique currently.

A new method which uses micro-osteoperforations (MOPs) to stimulate alveolar bone remodeling without the disadvantages of surgery such as the requirement of corticotomies, cuts in cortical bone, raising split-thickness flap, and decorticating the bone has recently been identified [5, 12]. Animal studies revealed that micro-osteoperforations significantly stimulate expression of inflammatory markers and increase the number of osteoclasts and bone resorption. The increase in bone remodeling contributes to the rise in both rate and magnitude of tooth movement [4]. In addition to the requirement of additional studies, similar results are reported in clinical trials [4, 5].

In this chapter, we focused on the biologic basis of orthodontic tooth movement, accelerated tooth movement techniques and application methods, advantages, disadvantages, effects, and possible side effects of micro-osteoperforations as a new, reliable, repeatable and minimally invasive method for accelerating orthodontic tooth movement.

2. Biologic basis of orthodontic tooth movement

Application of mechanical forces to teeth causes orthodontic tooth movement as a result of the biological responses of the periodontal tissues. Alveolar bone remodeling includes selective resorption in some areas and apposition in others. The biologic response to orthodontic therapy includes not only the response of the periodontal ligament and alveolar bone but also the response of growing areas which are distant from the dentition. However, it can be stated that tooth movement is primarily a periodontal ligament phenomenon because the alveolar bone response is also mediated by the periodontal ligament [13].

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The first step of the biological response to orthodontic force is "tension and compression in the periodontal ligament" which constricts and deforms blood vessels damaging cells in the periodontal tissues. The initial aseptic acute inflammatory response is realized by releasing chemokines and cytokines from localized cells such as osteoblast, fibroblast, and endothelial cells. Most of these cytokines are pro-inflammatory providing the continuity of the inflammatory response by activating osteoclast precursors of periodontal ligament in extravascular range and inflammatory cells. Infiltration of inflammatory cells causes the increase of chemokine and cytokine levels. This enables differentiation of osteoclast precursors to multinucleated giant cells which will realize alveolar bone resorption that is required for tooth movement. The continuity of the existence of anti-inflammatory chemokines and cytokines is essential in order to suppress destructive pro-inflammatory and osteolytic processes [4]. Therefore; the pro- and anti-inflammatory responses of alveolar bone, periodontal ligament, and inflammatory cells to orthodontic force are required to be known in order to develop safe methods to shorten orthodontic treatment period.

3. Accelerated tooth movement

The methods which accelerate tooth movement can be classified in three groups:

1. Chemical applications (local or systemic applications)
2. Mechanical-physical stimulations
3. Surgical-assisted techniques [14]

3.1. Chemical applications

In this method, substances such as prostaglandins, corticosteroids, vitamin D, cytokines, neuropeptides, leukotrienes, nitric oxide, diazepam, and vasoactive medications, which are considered as physiological agents that transform mechanical forces into cellular response, are used to decrease the resistance to the strength applied during tooth movement that occurs within the cells and to change the environmental factors [15, 16].

Taking systemic effect rather than being limited to the applied area and affecting other target cells in the body are reported as a common side effect of using these hormones and medications [17]. Therefore, there is no medication treatment which safely accelerates tooth movement [18].

3.2. Mechanical-physical stimulations

It was reported that stimulating periodontal ligament mechanically or physically can increase alveolar bone remodeling and tooth movement accordingly. Physical methods frequently include equipment-assisted treatment methods [6] such as low-dose laser applications, electromagnetic field, direct electrical current, and vibrational applications. These methods take effect by inducing signal molecules such as receptor activator of nuclear factor-kappa

B (RANK), receptor activator of nuclear factor-kappa B ligand (RANKL) pathway, mitogen-activated protein kinase (MAPK), c-fos, and nitric oxide [6, 19].

Low-dosage laser applications are reported to increase osteoblastic and osteoclastic activity by stimulating cellular proliferation and differentiation and accelerate orthodontic tooth movement without creating side effects on periodontal ligament [6, 20].

It is also reported that electromagnetic field applications increased levels of a group of enzyme which is responsible from regulating intracellular metabolism and cellular proliferation accordingly by influencing sodium-calcium change speed in cell membrane [21]. Increase in osteoblastic and osteoclastic activity causes an acceleration in the tooth movement. Darendeliler et al. [22] reported that static magnetic field accelerated tooth movement by shortening unproductive period when no tooth movement is observed but the side effect can be a decrease in serum calcium and slight changes in the chemistry of blood.

Applying direct electric current is also among tooth acceleration techniques, but its clinical use is asserted as not appropriate due to some complications such as application difficulty, ionic reactions that cause damages in tissues, and replacing of bone tissue with connective tissue [23].

Resonance vibration and ultrasonic vibration are also among the methods which are used to accelerate tooth movement. Resonance vibration is reported to increase release of peptide which is called RANKL within periodontal ligament and increase tooth movement rate by providing formation, function, and continuity of osteoclasts. Tooth movement can also be accelerated via ultrasonic vibration with the same mechanism but there is a risk that the heat can cause damage in the dental pulp [19].

3.3. Surgical-assisted techniques

The most clinically used and predictable option with stable results among the techniques which accelerate tooth movement is the surgical-assisted technique; it is evaluated as a costly invasive method [6]. It is based on the idea that rapid tooth movement can be achieved by increasing the biological response which is formed in periodontal ligament and alveolar bone. Creating an injury in the bone is a potential physical mechanism which provides rapid tissue recovery. Higher regional tissue response during this tissue remodeling/regeneration period is called as regional acceleratory phenomenon (RAP) [24]. Regional acceleratory phenomenon is a complex physiological case in which initially osteoclastic activity and secondly bone density is decreased followed by a rapid osteoblastic activity and a remodeling process. Orthodontic tooth movement acceleration is provided via the increase in cytokine activity around the teeth and decrease in cortical bone resistance and formation of hyalinization tissue by activation of this recovery mechanisms [25]. The advantages of the dentoalveolar surgical intervention in orthodontic treatment of adult patients are reported as: (a) less treatment time, (b) avoidance of potential periodontal complications, (c) favorable direction of growth, and (d) extensive envelope of the tooth movement [26].

3.3.1. Corticotomy and osteotomy combinations

They were first used by Heinrich Köle who stated that the basic resistance toward tooth movement was in cortical layer so by applying corticotomies and osteotomies might accelerate

tooth movement by breaking the resistance [27]. Köle et al. applied vertical corticotomies between the roots of two adjacent teeth buccally and lingually and horizontal osteotomies subapically as to combine corticotomies buccal-lingually. They theorized that the segments which became "blocks of bone" move as a whole to achieve rapid tooth movement without devitalization in long-term monitoring. Düker et al. also reported that pulpal and periodontal tissues of the teeth which were applied orthodontic force similarly were not damaged in their study on dogs with reference to Köle's corticotomies [28].

Although Köle and Düker proposed the opposite, periodontal and pulpal damages can be formed following invasive horizontal subapical osteotomies [29]. Additionally, osteotomy surgery is an invasive intervention with possible complications such as vascular and nerve damages so it is not a widely accepted method [30]. Nonetheless, Köle's studies pioneered surgery-assisted methods which accelerate tooth movement to be involved in the literature of modern orthodontics.

3.3.2. Corticotomy-assisted treatments

Köle's horizontal subapical osteotomies were changed with horizontal subapical corticotomies by Gantes et al. [31] and Suya [32] and it was stated that treatment of the cases was completed in almost 50% less period when compared to conventional methods. Gantes et al. [31] reported that overall vitality of the tooth was preserved and none of the patients had a periodontal damage that can be clinically recognized despite minimal root resorptions and gingival recession. Suya [32] asserted that corticotomy-assisted orthodontics had less root resorption and relapse risk and pain when compared to traditional treatments. In a recent study, Uzuner et al. [33] reported that corticotomy-assisted orthodontics increased the rate of tooth movement during canine retraction in 20% ratio and also relative bodily movement was achieved with the corticotomy-combined treatment.

3.3.3. Periodontal ligament distraction

The method was introduced to orthodontic literature by Liou and Huang [34]. Initially, a distractor device which consists of canine and molar bands was applied to the patients by the researchers and following extraction of upper first premolar teeth, surgical preparation phase which consists of two vertical channels formed buccally and lingually toward canine teeth through the extraction socket and a horizontal incision on the base of the extraction socket that connects the channels was completed at the same session. Interseptal bone was not cut mesiodistally during the surgical intervention. Activation was carried out without a need for latent period different from traditional distraction method and distalization of canine teeth was completed in a short period of 3 weeks with minimal anchorage loss. None of the patients suffered from pain and minimal resorption was observed in the roots of canine teeth in the radiographies at the end of procedure. Suggesting to be used especially in orthodontic treatments with primary premolar extraction which has anterior crowded teeth, Liou and Huang [34] associate the periodontal ligament distraction with this method to the distraction which is formed in midpalatal suture during rapid maxillary expansion.

3.3.4. Dentoalveolar distraction

The method was introduced by Kişnişci et al. [35] in 2002. The researchers applied a distraction device before teeth extraction as in periodontal ligament distraction method but a more

rigid device was used. Initially, the osteotomy curved apically at a distance of 3–5 mm from the apex of the canine at the same session with the primary premolar tooth extraction as a surgical preparation. Cortical bone which remained in the buccal of the socket after the extraction was carefully removed. Direct distraction was applied by skipping latent period similar to periodontal ligament distraction method. Canine distraction was reported to be completed within 8–12 days without anchorage loss. Root resorption or vitality loss was not observed in the posttreatment evaluations.

Although a faster canine distalization is achieved with less teeth tilting in dentoalveolar distraction method when compared to periodontal ligament distraction method, it is clear to be a more invasive method [36].

3.3.5. Periodontally accelerated osteogenic orthodontics

The method which is known as "Wilckodontics" or "periodontally accelerated osteogenic orthodontics" was introduced by Wilcko and Wilcko [9, 37–40]. According to the researchers, achieving optimal tooth movement is possible by forming a bone layer of 1.5 mm or less on the root surface in the direction of movement in corticotomy-assisted orthodontic treatment. When the force is applied, soft tissue matrix and osteoid islets remaining from the demineralized layer move with the root and remineralized when the orthodontic movement is completed. Wilcko brothers explained the acceleration of tooth movement following corticotomy with demineralization-remineralization period that is generated from rapid osteoclastic activity in alveolar bone in the beginning of "regional acceleratory phenomenon" which was introduced by Frost [24]. According to this mechanism, less resorption and rapid orthodontic tooth movement is achieved as a result of the decrease in alveolar bone density and increased metabolic activity in bone tissue. Thus, the term "movement of bone blocks" which was defined by Köle [27] was replaced with the term "bone matrix transplantation."

Wilcko and Wilcko discussed the need for applying a bone graft in order to prevent dehiscences, fenestrations, and relapses which can occur in posttreatment period due to the decrease in alveolar bone density within the cases with buccal move of the roots or thin buccal bones following corticotomy. They proposed to use resorbable bone graft by saturating clindamycin phosphate or platelet-rich plasma and creating circular perforations on the surface of the bone in order to increase blooding of graft material if the cortical bone thickness is sufficient [37]. The applied surgery apart from this procedure is almost the same with Suya procedure [32]. In this technique, bonding and applying forces to teeth is initiated 1 week earlier from the surgical intervention and biweekly orthodontic examinations are recommended.

Treatments are completed in three or four times shorter periods with "periodontally accelerated osteogenic orthodontics" method when compared to traditional orthodontic treatments besides its increased root resorption and relapse risks. Its need for additional surgical intervention with an extra cost, possibility of bone loss in alveolar crest, and gingival recession following the surgery and surgical complications such as pain and edema are among the disadvantages of the procedure.

3.3.6. Corticision

The necessity to remove flaps during corticotomy-assisted orthodontic treatment makes the method invasive, decreasing its acceptability by the patients and clinicians. Therefore, corticision was introduced by Park et al. [41] as an alternative approach for corticotomy procedures. The researchers made the patients rinse their mouth with an antiseptic mouthwash and then placed a stabilized scalpel on the attached gingiva interradicularly as to make an angle of 45–60 with the long axle of root of the teeth and applied cortical incisions via a surgical hammer without removing flaps [42]. Incisions were applied as 2/3 of the roots vertically and in 10 mm depth. Corticision area must be cleaned with physiological saline solution until hemorrhage stops. Park reported that fixed orthodontic treatment of a patient without extraction was completed in a short period of 10 months through this method. Although corticision method is minimally invasive with a short surgical intervention, it is not widely accepted as it is not appropriate to grafting and the surgical technique is disturbing for the patience.

3.3.7. Piezocision

In 2009, Dibart et al. [43] introduced a minimally invasive technique that can be applied by piezosurgical tools, the piezoincision method, which can be carried out without any flap surgery as in corticision method but it allows hard or soft tissue grafting differently. In this method, small piezoelectric incisions are placed in the area from the targeted corticotomy area to periost. Piezoincisions of 3 mm length and depth are applied with the piezosaw without removing mucoperiosteal flap. In this stage, mucoperiosteal flap can be removed as a tunnel and a bone graft in pad form can be replaced if necessary. Sutures are required if grafting is applied, otherwise there is no need for sutures. It was reported that no significant difference was found in terms of tooth movement speed and root resorption in the studies which compare corticotomy-assisted methods with piezoincision method [44].

Piezoincision is a practical, minimally invasive, and effective method. Additionally, ultrasonic vibrations which occur during the procedure in piezoincision method also contribute to accelerate tooth movement [3]. Required precautions must be taken before the procedure with patients in high-risk group due to possibility of temporary bacteremia formation risk following the procedure.

3.3.8. Piezopuncture

"Piezopuncture" method, which is applied by using an ultrasonic piezosurgical tool, "piezotome" without gingival incisions in piezoincision method was developed by Kim et al. [45]. In their study on 10 dogs, the researchers created several cortical punctures both buccally and lingually on the mesial and distal sides of the teeth which will be moved and reported that anabolic activity and tooth movement were accelerated at the end of the procedure. Omidkhoda et al. who carried out piezopuncture technique on human reported that tooth movement was accelerated but they observed distal tilt in the canine teeth crowns in their case reports which consist of two cases [46].

"Piezopuncture" method also takes part in literature as a method with promising positive results but it must be evaluated by further clinical studies.

4. Micro-osteoperforations

Orthodontic tooth movement is a biological response which is created by an external force that will prevent the dentofacial complex to be in physiological balance [13]. Orthodontic force creates an aseptic inflammatory response in periodontal tissues. An increase on vascular permeability and cellular infiltration of leucocytes was reported in the early period of orthodontic tooth movement [47]. Along with native cells such as osteoblasts and fibroblasts, migrated immune cells produce inflammatory cytokines that include chemotactic factors, growth factor, monocyte-derived factor, lymphocyte, and colony-stimulating factors [48, 49]. The gingival crevicular fluid of moving teeth includes tumor necrosis factor-α (TNFα), interleukin-1 (IL-1), IL-2, IL-3, IL-6, IL-8, osteoclast differentiation factor, and interferon-γ (IFNγ) [48, 50].

Orthodontic tooth movement rate is basically found associated with the rate of bone resorption which is controlled by osteoclast activity [51]. Therefore, any factors which effect activities of osteoclast precursor cells and their transformations into osteoclasts can be assumed to have significant effects on orthodontic tooth movement. There are a lot of researches that report the increase in the activity of inflammatory markers such as chemokines and cytokines as a response to orthodontic forces [5, 25]. It is reported that several cytokines which play role in osteoclast formation and activity such as TNFα, IL-1, and IL-6 are found in gingival crevicular fluid during orthodontic tooth movement [25]. Although the roles of chemokines and cytokines during orthodontic treatment are not clearly known, they are valued as essential mediators for orthodontic tooth movement in terms of their significant roles on differentiation and activity of osteoclast cells [52–54]. Significant decrease on orthodontic tooth movement rate within the studies in which the effects of these markers are blocked via different techniques such as anti-inflammatory medication or genetic manipulation can be accepted as a proof that these factors are extremely efficient on orthodontic tooth movement rate [55–57]. Previous studies represent that surgical interventions which cause minor bone traumas increase inflammatory cytokines, bone remodeling, and orthodontic tooth movement acceleration together with regional bone density [24, 58]. Surgery-assisted techniques which are applied in order to accelerate orthodontic tooth movement should be studied under subtitles such as corticotomy and osteotomy techniques, dental distraction technique, perisegmental corticotomy, and piezoincision. Corticotomy-assisted orthodontic treatment increases bone remodeling which accelerates recovery and repair mechanisms and tooth movement rate accordingly by creating a mechanical trauma in cortical bone [59]. Although corticotomy-assisted orthodontic treatment was reported to be an efficient method in accelerating tooth movement, the significance of removing flaps is also stated to cause important postoperative complications [60]. Piezoincision technique, which is a minimally invasive technique that includes piezoelectric incisions without removing flaps, was developed in order to overcome these disadvantages [43]. Piezoincision is known to be an effective method for acceleration of tooth movement but it was reported to have high risks of damaging tooth roots [18]. Surgery-assisted techniques are invasive with disadvantages such as bone loss, postoperative pain, edema and infection, avascular necrosis besides low acceptance rates by the patients [37, 40]. Based on this, the hypothesis that small osteoperforations on cortical bone without removing flaps will increase bone remodeling and tooth movement rate accordingly by stimulating

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release of inflammatory cytokines minimizing these disadvantages was developed (**Figure 1**). Micro-osteoperforation is an up-to-date procedure which is promoted as an auxiliary dentoalveolar procedure which can accelerate tooth movement via minimum surgical interventions.

In their animal study in 2010, Teixeira et al. [25] classified 48 rats, which were applied experimental orthodontic tooth movement, into four groups as one group with only orthodontic forces, a group via application of soft tissue flaps together with orthodontic force, a group that was applied 3 small perforations on cortical plate with soft tissue flaps together and orthodontic force and a control group. The researchers stated that they formed the microperforations in the cortical bone by using a round bur and handpiece. It was observed that at the end of experimental tooth movement period, release of 37 out of the 92 cytokines increased in all experimental groups and 21 of them were at the maximum level in the group which was applied perforation. In addition to that, light and fluorescent microscopy, microcomputed tomography, and immunohistochemistry examinations, which were carried at the end of the experiment, represented a significant increase in osteoclast number which accompanied by generalized osteoporosis and orthodontic tooth rate besides bone remodeling activity [25]. Similar to previous studies, researchers also demonstrated that the increase in bone remodeling was not limited around the loaded teeth but also involved the periodontal structures of the adjacent teeth. The researchers, who argued that the observed effects of osteoperforations on tooth movement can be related to loss in bone structure rather than release of increased inflammatory cytokine, stated that perforations were applied in the smallest amount and number possible and the remaining cortical bone was healthy in order to minimize this possibility. These findings also represent that the perforations that will be applied in order to accelerate tooth movement rate are not necessarily be adjacent to the moved teeth. Stating that inflammation can cause negative effects on periodontium and tooth structure when it is uncontrolled because it is a double-sided injury, it is also reported that applying micro-osteoperforations instead of some surgical interventions such as corticotomy, which are applied for acceleration of tooth movement, can minimize the side effects.

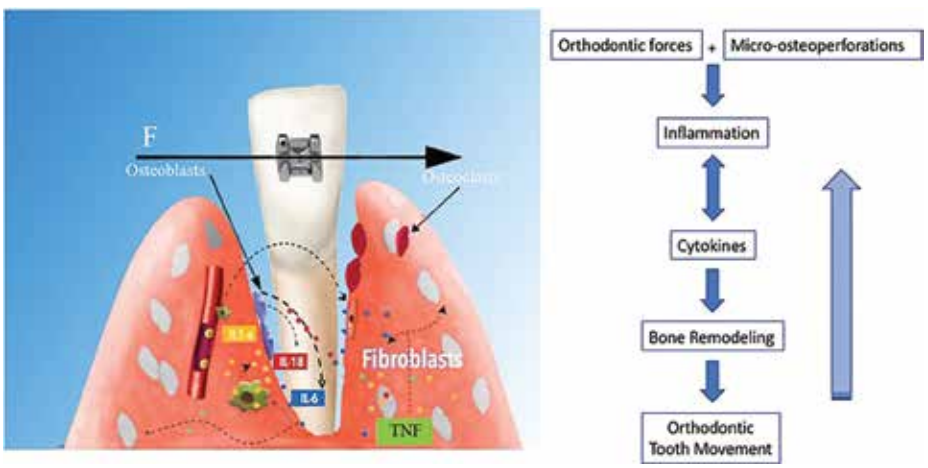

Figure 1. Micro-osteoperforations on cortical bone increase bone remodeling and tooth movement rate by stimulating release of inflammatory cytokines.

Tsai et al. [11] applied both micro-osteoperforations and corticision without removing flaps different from the previous studies and evaluated the differences between the procedures in their animal study which was carried out to evaluate the effects of micro-osteoperforations and corticisions on orthodontic tooth movement rate. It was stated that bone and bone mineral densities were significantly decreased when compared to control group and tooth movement rate was increased in both groups without any significant difference. The rise in tooth movement rate in both groups in this study is smaller than the previous studies. The researchers, who stated that there might be a direct proportion between trauma amount and remodeling rate, reported that the difference was resulted from the smaller amount of the trauma as the flap was not removed.

In 2016, Cheung et al. [12] published a study which evaluated the effectiveness of micro-osteoperforations. Mini-implants were used in order to form micro-osteoperforations different from the previous animal studies and the existence of external apical root resorption was evaluated following the procedure. Similar to previous studies, it was observed that there was a decrease on the density and volume of the bone surrounding moved molar teeth compared with the control side besides the acceleration of orthodontics tooth movement on the side where micro-osteoperforations were applied in the split mouth design study with six rats. Histological examinations also showed that new bone formation increased as well as osteoclast numbers on the side with micro-osteoperforations. This is a proof of the existence of osteoclasts-osteoblast coupling formation with decortication. Despite the fact that resorption and new bone formation were represented together, bone fraction volume and bone mineral density was decreased, which shows that resorption was more than new bone formation during tooth movement period. Contrary to conventional theories which assert osteoclast activity is limited to pressure area, the recent studies show that periodontium was remodeled as a single unit [61]. This study also shows that osteoclast rate in all alveolar bone surrounding the moving teeth is clearly on the pressure areas.

Alikhani et al. [5] designed a clinical trial to evaluate tooth movement with or without micro-osteoperforations in order to investigate whether this phenomenon occurs in human or not. They called the technique of creating micro-osteoperforations in the bone as "alveocentesis." In their study, 20 adults were divided into 2 groups as control and experimental groups. Micro-osteoperforations were applied in the experimental group on one side of the maxilla using a disposable micro-osteoperforation device designed for this purpose by Propel Orthodontics (Ossining NY) and the control group did not receive any micro-osteoperforations. Following 28 days of canine retraction period, amount of the tooth movement was measured and also the activity of inflammatory mediators was determined in gingival crevicular fluid with an antibody-based protein assay. Additionally, the presence of pain or discomfort was evaluated with a numeric rating scale. Results of this first clinical trial about micro-osteoperforations in the literature reported significant increase in the levels of inflammatory markers and also 2.3-times rise in the rate of tooth movement with micro-osteoperforations. However, significant pain or discomfort during or after the procedure was not reported by the patients; also, any other complications were not observed.

It was stated that orthognathic surgery, corticotomies (applying several incisions and perforations with removing flaps), piezoincision (creating injury on the bone with small incisions

without removing flaps via a piezoelectric device), and even tooth extractions will increase the release of inflammatory markers and bone remodeling by creating injuries similar to micro-osteoperforations on alveolar bone effecting the rate of tooth movement accordingly [5]. Unfortunately, increase of inflammatory marker release cannot continue for a long period of time and a decrease in cytokine activity is observed regardless of the severity of the trauma after 2–3 months [4] which reveals the necessity for repeating the procedure during orthodontic treatment. Therefore, the extraction is suggested to be applied at the same time with major tooth movement for the patients whose tooth is planned to be extracted. There will not be a need for micro-osteoperforation requirement to accelerate tooth movement process as remodeling will speed up. In other words, it would be appropriate to prefer micro-osteoperforations in the treatments without tooth extraction or when extraction is carried out long after the orthodontic treatment because its effects are similar to tooth extraction. It is considered that as the level of trauma increases, inflammatory response will also rise. The rise on tooth movement rate when micro-osteoperforation number is increased can be shown as a proof. It was stated that regional acceleratory phenomenon (RAP) took effect in 1 or 2 days following the surgical intervention and reached its maximum level in 1 or 2 months [9]. Aboul-Ela et al. [59], Al-Naoum et al. [62], and Leethanakul et al. [63] reported that tooth movement speed was high during 2 months following the creation of traumas in cortical layers but it gradually decreased in this period. Alikhani et al. also stated that cytokine activity decreased after 2 months following micro-osteoperforation so they proposed that the procedure shall be repeated after a month break [4]. Micro-osteoperforations are repeatable which can be considered as an advantage because application time can affect the results in all surgically assisted procedures. On the other hand, there is not a clear information on how frequently micro-osteoperforations can be applied in order to achieve an optimum acceleration in tooth movement rate.

In a recent thesis study in Akdeniz University, Faculty of Dentistry, Department of Orthodontics (Antalya, Turkey), efficiency of micro-osteoperforations on molar distalization rate with cervical headgear was evaluated on 3D digital models [64]. About 17 patients whose molar relations were bilateral class II (minimum teeth to teeth) with class I skeletal anomalies or normal or low vertical growth pattern were included in the study which is regarded as the first study to evaluate the efficiency of micro-osteoperforations on molar distalization. Propel device (Ossining, NY) was used to form three micro-osteoperforations—one mesially and two distally—of 1.5 diameter and 5 mm depth on a random molar teeth, in the session where distalization was initiated with cervical headgears in the split-mouth designed study. Micro-osteoperforations were renewed at least twice during the distalization period every 8 weeks in each sample. The procedure continued until class I relationship was achieved on the side where tooth movement was considered to be slower. Studies were carried out on 3D digital models which were taken just before and after molar distalization in order to compare molar distalization rate and tilt and rotation rate in molar teeth. While no significant difference was observed between tilting and rotation rates, it was concluded that on the micro-osteoperforation side, more tooth movement was seen.

Lee et al. [65], who stated that in the studies which evaluated the efficiency of micro-osteoperforations on orthodontic tooth movement, micro-osteoperforations were generally applied on healthy alveolar bone, planned a study considering that the effects of

bone remodeling process that was activated by applying orthodontic tooth movement and micro-osteoperforations on the atrophic ridge could be different. The researchers, who created atrophic alveolar ridge model on eight beagle dogs, evaluated tooth movement rates and atrophic alveolar ridge area on the sides with and without osteoperforations in their study which they planned via split mouth design. Micro-CT based histomorphometry analysis similar to the previous studies suggested that osteoperforations accelerated tooth movement with a decrease in bone density without any differences in atrophic ridge volume. This up-to-date finding can be evaluated as an indicator that the efficiency of micro-osteoperforations on bone remodeling is more related to resorption mechanism and osteoclast activation.

In the literature, there is a limited number of studies which evaluate the effects of micro-osteoperforations on tooth movement in human. Current studies indicate that micro-osteoperforation is a safe method that can accelerate tooth movement but it must be taken into consideration that several factors such as occlusal relations, movement type, applied mechanics, age and gender of the patient, oral hygiene, periodontal illnesses, alveolar bone loss, systemic diseases, and medication use effect tooth movement rate in human. Therefore, the efficiency of micro-osteoperforations must be evaluated with long-term studies in which study groups are standardized as much as possible considering these variable where different tooth movement types (distalization, intrusion, eruption of impacted tooth, etc.) and mechanics are applied containing more sample numbers.

4.1. Micro-osteoperforation application techniques

Micro-osteoperforation is an up-to-date method among the surgery-assisted techniques of accelerating tooth movement. Therefore, in the literature, there are studies in which different methods are used in order to create MOPs on alveolar bones.

We observe that low-speed handpiece and round bur combinations [11, 25] and mini-implants [12] are used within the animal studies which evaluate effects of MOPs on tooth movement and both methods are accepted as effective. Even if in a limited number of clinical trial; micro-osteoperforations are typically applied with a handheld disposable appliance designed by Propel Orthodontics (Ossining, NY). Propel is an appliance which is designed to apply alveocentesis procedure. The foremost part of the device which is like an orthodontic stainless steel screw is patented, allowing perforation of alveolar bone traumatically over keratinized gingiva and moving mucosa. Contrary to other rotatory devices, Propel was reported to have a slight effect on soft issue. It is a device which enables tissue remodeling and micro-osteoperforations between tooth roots over both stable and moving tissue of 1.5 mm diameter and 3, 5, and 7 mm depth without flap surgery in order to accelerate tooth movement. The appliance has an adjustable length and a light signal that turns on when the clinician achieved desired depth [5, 64]. Micro-osteoperforations, which are applied on the defined depth, reach to medullar bone from cortical bone increasing inflammatory mediators. The device was also reported not to cause soft tissue damage while enabling remodeling process [5].

While it is not always possible to create homogenous perforations of same size using micro-osteoperforation methods such as round burs, Propel device which is designed in order to

form MOPs has not included in routine clinical use yet. Thus, mini-implants are considered more advantageous than other methods as they are included in clinical routine and frequently used by orthodontists for different purposes and easily tolerated by the patients. On the other hand, in the literature, there are limited studies in which mini-implant-facilitated micro-osteoperforations are carried out with human. Aksakalli et al. [66] applied three micro-osteoperforations distal to the canine teeth with miniscrews just before canine distalization period. In their case report, they reported that MOP method with miniscrews accelerated canine distalization in their 14-year-old male patient with class II malocclusion by almost 1.5-fold and also without harmful effects on root and periodontal structures. On the contrary, Alkebsi et al. [67] could not find any differences of anchorage loss, canine rotation, and tipping between the MOP and control sides in their randomized controlled clinical trial where they investigated the effectiveness of miniscrew-facilitated MOPs on the rate of canine distalization.

All of the methods are applied without a need for additional periodontal surgeries which is considered as a significant advantage but additional clinical studies are required in order to evaluate the efficiency of each technique and their advantages and disadvantages over each other in detail.

4.2. Advantages of micro-osteoperforation over other surgical techniques

When micro-osteoperforations are compared with several surgical techniques, which are proved to accelerate tooth movement, they are considered as more advantageous because they are less invasive with no need for removing flaps eliminating possible side effects of the surgery [4, 5]. Additionally, all techniques which make use of micro-osteoperforation do not include an invasive surgical procedure represent that they are easily applicable in the clinics by the orthodontists and can be added to clinical routine. Patients did not report any pain or discomfort in the clinical studies with micro-osteoperforations which shows that it is easily accepted and tolerated by the patients who are under orthodontic treatment [5, 64]. These advantages also enable the micro-osteoperforations to be periodically repeated until the desired results are achieved [4, 64].

4.3. The relation of micro-osteoperforations with pain and root resorption

There is a limited number of studies that evaluate patients' pain and discomfort levels among the clinical studies in which micro-osteoperforations are applied. Alikhani et al. [5] asked their patients to scale their pain and discomfort levels via a numeric rating scale on the day they replaced the device, the day they began canine distalization, 24 h, 7th and 28th days after canine distalization in their study in which they evaluated the effect of micro-osteoperforations on canine distalization. In this scale, which is reported as having high credibility, "0" presents no pain, while "10" stands for the existence of the worst pain. Data analyses showed that the patients had the most pain in 24 h following canine distalization but no significant difference was observed between experimental and control groups. The patients defined a slight and resistible pain on the micro-osteoperforation side which does not require taking painkillers but no statically significant difference was found. The similar feedbacks were taken in Boz's thesis study in 2018 concluding that micro-osteoperforations did not cause a

significant pain or discomfort [64]. In line with these findings, it is possible to state that micro-osteoperforations are easily tolerated by the patients and can be applied to routine clinical use.

Orthodontically induced inflammatory root resorption (OIIRR) is included in negative side effects of orthodontic tooth movement as a frequent research subject. Although its etiology and predictors are not fully understood, it is considered to be resulted from complex interaction of individual sensitivity [68], applied mechanics [69, 70], and specific dental predisposition [71]. Orthodontically induced inflammatory root resorption was stated to be related with periodontal ligament remodeling which is a result of the pressure applied to tooth root during tooth movement and removal of hyalinized necrotic tissues after trauma. Excessive pressure that causes ischemic necrosis [72] of periodontal ligament and root resorption related to orthodontic tooth movement is reported to be frequently observed in the areas in which excessive pressure is applied to periodontal ligament [73]. The underlying biological process of both orthodontic tooth movement and root resorption covers local inflammatory response. Animal studies show that many pro-inflammatory cytokines are common in both pathways. Cytokines such as IL-1, TNFα, and chemokines as IL-8 and MCP-1 are known to have significant roles to initiate and ease root resorption process [74]. These inflammatory mediators have significant roles on activation of tooth movement and osteoclast activity [75]. Inhibition of cytokine activity decreases osteoclast and odontoclast rate as well as tooth movement and root resorption [76]. It is known that accelerated tooth movement techniques increase inflammatory cytokine activation.

In the literature, there are several studies that evaluate the effects of accelerated tooth movement and decortication on root resorption [5]. The cytokines which promote inflammation are also reported to activate cementoclasts which cause root resorption increasing root resorption risk accordingly [77]. On the contrary, there are findings in the literature which state that decortication and demineralization of alveolar bone decrease the pressure toward tooth movement enabling an ease for the movement and decreasing root resorption risk accordingly [12]. It was found out that the effects of tooth movement accelerated via corticotomy on orthodontically induced inflammatory root resorption were similar to conventional orthodontic treatment but it was also reported that periapical radiographies may not be reliable for assessing root resorption in two studies which was carried out by evaluation of periapical radiographies [78, 79]. In an animal study which evaluates the effects of corticotomies on tooth movement, it was found out that there were not any differences between control and experimental groups in terms of root resorption [80].

In the literature, there is a limited number of studies which evaluates the effects of micro-osteoperforations on root resorption. Tsai et al. reported in their study in which they compared the efficiency of micro-osteoperforations and corticisions that root resorption creation risk of minor surgical interventions is lower when compared with conventional orthodontic treatments. The hematoxylin and eosin analysis of the researchers showed that micro-osteoperforation-assisted accelerated tooth movement was resulted in decreased root resorption [11]. Similarly, in the study in which Cheung et al. evaluated effects of micro-osteoperforations on experimental tooth movement in rats, root resorption was observed on the MOP application side on the samples which were colored with hematoxylin eosin reporting that 3D volumetric analysis did not show any volumetric difference in the root

of upper molar teeth which was moved [12]. Although all findings support the idea that micro-osteoperforations do not increase root resorption risk showing that mini-implants can be safely used for MOP procedures in terms of root resorption, it is clear that there is a need for further supportive studies with increased sample number.

Cheung et al. [12] included 20 patients whose upper primary premolars are planned to be extracted for orthodontic purposes in their study which is known as the initial clinical study to evaluate the effects of MOPs on OIRR. About 150 g buccal tipping force was applied to premolar tooth which was planned to be extracted and micro-osteoperforations of 5 mm depth were applied to mesial and distal parts of the premolar teeth on the experimental side via Propel device (Propel Orthodontics, San Jose, Calif) evaluating the patient's opposite side as the control group. Following 28 days of tooth movement period, premolar tooth on both sides was extracted and examined by microcomputed tomography, volumes of root resorption craters were calculated and compared. Volumetric root loss average of the premolar tooth on the side which micro-osteoperforations are applied was found to be 42% more than the premolars on the control side. The researchers represented that the side to be applied micro-osteoperforations was not randomly selected (decision was taken in accordance with the availability of the distance between the roots) and evaluation was carried out following a short-term tooth movement as the limitations of the study and suggested further studies with treatments of longer terms and larger sample numbers. Nonetheless, when the findings of the study are considered, it can be stated that special attention shall be paid on planning micro-osteoperforations with patients who are individually prone to root resorption.

As a result, there are studies which report that micro-osteoperforations can cause positive and negative effects on root resorption. Their relations were not clearly explained so there is a need for further studies.

5. Conclusions

- Among several techniques which are defined to accelerate orthodontic tooth movement and treatment periods accordingly, due to numerous studies, surgical techniques that have the most predictable results have been an essential part of modern orthodontics.
- Micro-osteoperforations are outstanding as a minimal invasive, easy-to-use, repeatable, and efficient new method that can eliminate some disadvantages of surgery among the defined invasive techniques.
- Experimental studies have proved that micro-osteoperforations accelerated bone remodeling process and orthodontic tooth movement accordingly together with an increase in osteoclast number and new bone formation and a decrease on bone volume and density. A limited number of clinical studies also support the findings.
- Different techniques are defined to apply micro-osteoperforation in the literature but there is a need for studies that evaluate differences between techniques in order to determine the ideal method.

- Further studies are required for the ideal timing and frequency of the application in order to achieve optimum tooth movement acceleration.
- Although it is reported that side effects such as pain or root resorption are not observed due to micro-osteoperforations, long-term studies with more samples are required.

Conflict of interest

I declare that I have no conflict of interest regarding the publication of this chapter.

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Chapter 7

Stability of Diastemas Closure after Orthodontic Treatment

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Additional information is available at the end of the chapter

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Abstract

The prevalence of diastemas varies greatly according to age and ethnic group. In permanent dentition, it varies from 1.7 to 38%. Its etiology is multifactorial. In the deciduous and mixed dentition phases, interincisal diastemas are considered normal. There are several approaches used in the treatment of anterosuperior diastemas, which vary according to the present etiologic factor. Orthodontic treatment also has the function of treating any other associated occlusal problem and helping in the elimination of parafunctional habits. Some authors agree that orthodontic closure of diastemas without subsequent surgery for removing the abnormal labial brake greatly increases the frequency of relapse in the postretention period, while others concluded that the fibrotomy of periodontal fibers together with the retainer had a positive effect on the stability of space closure. Buccal and lingual teeth inclinations have greater tendency to relapse, while mesial and distal movements, with a period of containment, are stable movements. Removable retainers are not considered a good choice. Depending on the type of initial malocclusion, the use of retainers throughout life is recommended. Fixed retention is often cited as the only satisfactory method to promote stability at the closure of previous diastemas.

Keywords: diastemas closure, stability, diastema, relapse, corrective orthodontics

1. Introduction

The presence of diastemas in the anterosuperior region used to be considered unpleasant, being one of the main reasons why patients seek orthodontic treatment. However, some studies report that the stability of the orthodontic closure of the diastemas, particularly of the median diastema in the permanent denture phase, is low. Long-term stability is one of the main objectives in orthodontic treatment; however, it is one of the most difficult to reach.

In the anterosuperior region, this stability is especially desired and verified by patients. Most of the publications on anterosuperior diastemas consist of clinical cases and reviews of the literature on the etiology of the problem and its possible treatments. But, there is a shortage in the literature of studies on the stability of the treatment of the upper interincisal subjects. The present chapter seeks to evaluate the recurrence of the upper interincisal diastemas, the factors associated to the relapse and stability, some treatment proposals, and considerations for retention.

2. Definition and epidemiology of diastemas

At the beginning of the last century, Angle [1] described median diastema as a common form of malocclusion where there is a space between the upper central incisors and, very rarely, between the lower central incisors. According to the author, the width of this space usually varies from 1 to 4 mm and always presents an unpleasant appearance, and may interfere with speech, depending on the width. Later, according to Bishara [2], diastemas were described as just clinically visible spaces between two or more adjacent teeth.

In publishing the Six Keys for normal occlusion, Andrews [3] stated in Key V that there must be fair points of contact throughout the dental arch, in the absence of dental size discrepancy. According to this information, diastemas are also considered a malocclusion by Andrews.

2.1. General prevalence

The prevalence of diastemas varies greatly according to age and ethnic group [4, 5]. In permanent dentures, the prevalence of diastemas varies from 1.7 to 38% [4].

2.2. Prevalence according to age

The literature demonstrates that the prevalence of diastemas decreases with age, mainly due to the development of occlusion. Richardson et al. [6] observed 5307 children between 6 and 14 years of age and, as a result, obtained a prevalence of 38% of children with median diastemas above 6 years of age, 56% at 8 years, and 18% at 14 years.

In a longitudinal study, Bergström et al. [7] aimed to observe the behavior of median diastemas in relation to the presence of the labial frenulum, in children of age 9 years on average, followed up after 2, 5, and 10 years. In the first two follow-ups, the group submitted to the removal of the labial frenulum presented a larger number of cases with closed diastemas than the control group. However, 10 years after the first consultation, there was no difference between groups. The authors found that the number of children with diastema declined with age and that there were a considerable number of individuals where diastemas closed from 14 to 19 years.

Steigman and Weissberg [8] found a prevalence of 50% in 1279 adolescents with spaced permanent dentures, ranging in age from 12 to 18 years. On the other hand, Steigman et al. [9]

found that the number of diastemas per individual decreased with age, but 79% of the pre-existing spaces remained, resulting in a percentage of 38% of young adults with spacing between the teeth.

2.3. Prevalence according to gender

Richardson et al. [6] observed a higher percentage of 6-year-old girls with median diastemas; however, at age 14, the opposite occurred. The authors believe that this is due to an earlier maturation of the girls in relation to the boys.

The findings of Steigman and Weissberg [8] corroborate those of the other authors, since they found dimorphism only in the subgroup between 14 and 16 years. There was no difference between genders in the 12- to 14-year-old and 16- to 18-year-old groups. Another study published by Steigman et al. [9] supported this finding, demonstrating that there was no difference between genders regarding the number of spaces in the arches in a sample of patients aged 16–22 years. It was also found that the number of diastemas decreased with age in both genders, but this occurred earlier in females than in males.

2.4. Prevalence according to race

In an epidemiological study on the prevalence of diastemas in white and black ethnic groups, Richardson et al. [6] observed, in a sample of children between 6 and 14 years of age, a higher frequency of diastemas in blacks at almost all ages. A prevalence of 23% of blacks at 14 years of age with diastema was found, differing from 14% of whites of this age with central interincisive spaces.

Similarly, Lavelle [4] found a higher incidence of central interincisive diastemas in melanodermas (5.5%) than in leucodermas (3.4%) and a lower incidence in xanthomas (1.7%). Likewise, McVay and Latta [5] found a very significant difference between the prevalence of blacks (29%) compared to the percentage of 20% of adults presenting this space in both the white and yellow race.

3. Diastemas and occlusion development

It is a consensus in the literature that, during the development of occlusion, in the deciduous and mixed dentition stages, the presence of diastemas is a characteristic of normality [2, 10–14]. Nevertheless, Richardson et al. [6] suggested that median diastemas are more than a phase of development eliminated over time, due to the high prevalence found in 18% of adolescents with medium diastemas. Edwards [15] stated that if the median space is greater than 2 mm, it is unlikely to close spontaneously. It was observed in a longitudinal study in adolescents that there was stability in most of the existing diastema, although some were eliminated even after the eruption of permanent canines [9].

3.1. Primary dentition

Authors have suggested that these spaces in the primary dentition are normal and would have the function of assisting in the eruption of permanent teeth [10, 16]. In describing the

biogenetic course of the deciduous dentition, Baume [11] classified the dental arches according to the spaces designated by him as type I, with anterior spaces, and type II, without such spaces. In this work, the author cites that one type cannot progress to the other.

Moyers [14] reported that there is a generalized spacing in the anterior region of the upper and lower arches in the deciduous dentition, which increases significantly after this dentition has been completed.

3.2. Mixed dentition

In the mixed dentition phase, the diastemas continue to appear as a physiological characteristic, mainly the diastema between the permanent maxillary incisors. The germs of these teeth remain separated within the maxilla, respecting the intermaxillary suture interposed between them [16].

Broadbent [12] published a study of 5000 individuals observed over 12 years. The author called the "ugly duckling stage" the period that ranges from the eruption of the upper incisors, around the age of 7 years, until approximately 10 years of age, when the upper canines erupt. At this stage, the lateral incisors remain with the converging roots until the maxilla size is sufficient to assume a more vertical position. With sufficient increase in the size of the subnasal area, and in the presence of normal growth, the canines move down, forward, and laterally to the lateral incisor roots. Correction of the ugly duckling stage will occur in the period between 8 and 12 years of age.

Burstone [13], observing the normal changes during development, identified that during the "ugly duckling phase" of Broadbent [12], protrusion of the crowns, overjet, and anterosuperior spaces occurs. He also reported that the upper central interincisive diastema would be closed with the eruption of the other permanent teeth, especially the canines, and that the overjet of the incisors would be corrected by the pressure of the labial musculature and the irruption of the other permanent teeth.

3.3. Permanent dentition

In the permanent dentition, the etiology of the diastemas is multifactorial [2, 16–19] and can be associated to the following factors: abnormal labial frenulum [1, 2, 16, 17, 20–22], microdontia, agenesis of maxillary incisors [2, 10, 16, 17, 20], dental discrepancy [2, 17, 23], shape of the anterior teeth (barrel) [10], brachyfacial pattern [17], a positive tooth-bone discrepancy [2, 16, 17], overbite, congenital anomalies such as soft tissue fissure, hybrid brake, or supernumerary teeth [2, 10, 16, 24], cysts and tumors [2, 24], periodontal disease [2, 25], macroglossia or neuromuscular imbalance of the tongue [10], acromegaly [26, 27], and orthodontic treatment [2, 20, 24].

The etiology of diastemas is also very well explored in a review article published by Bishara [2]. In this study, the authors divided diastemas into two categories, according to the etiology: (1) those not caused by orthodontic treatment, present before its accomplishment; and (2) those that appeared during or after orthodontic treatment. In the first category, the etiological factors mentioned are: physiological spaces in the deciduous dentition; developmental spaces in the mixed dentition phase, which are closed, according to the authors, after the canine

eruption; genetic factors such as large jaws and small teeth; tooth size, interarch discrepancy, agenesis, and micro-diseases; characteristics relating to ethnic groups; low insertion of the upper labial frenulum, preventing the mesial migration of the maxillary central incisors during canine eruption; dental rotations; supernumerary teeth, among which a classic example is the mesiodens; pathological conditions, such as proximal caries, periodontitis, cysts, and tumors; and, finally, the deleterious habits, exemplified by the lingual interposition and sucking lip. In the second category, the author refers to dental extractions and occasional dental size discrepancies, caused by extractions as diastematic agents during orthodontic treatment.

Steigman et al. [9] evaluated the stability of permanent denture spaces during adolescence in untreated patients, and also investigated the association of tooth spacing with tooth size and dental arch dimensions. They observed that women with spaced dentition had smaller dental widths and similar arch dimensions than those without dental spacing. In them, the spaces were equally distributed in both arches. On the other hand, in men, there was an equivalence of the dental dimensions, but the intercanine distances and superior interpremolars were greater in those with a spaced dentition. This is the reason why, in males, a greater number of spaces were found in the maxilla than in the mandible.

Oesterle and Shellhart [27] mentioned that the presence of generalized spaces in the dental arches may be the result of discrepancies between the size of the teeth and their respective apical bases, muscular imbalances, deleterious habits, and loss or absence of teeth. The large jaw and/or jaw combination with normal or slightly reduced teeth size is related to inherited characteristics, but may also be a sign of endocrine imbalances, which result in excess growth hormone, such as acromegaly.

Gass et al. [18], when evaluating the correlation between the heredity and the presence of the medium diastemas in leucodermas and melanodermas, found that the genetic expression was more significant for the whites than for the black ones, where the existence of an interincisive diastema is more related to environmental factors, such as excessive protrusion of the incisors, a predisposing periodontal tissue, habits, and absence of teeth. However, in relation to the stability of the treatment, an association between heredity and median diastema recurrence has been reported [28].

In the same year, Mondelli et al. [24], in a comprehensive review work describing the etiology and the various diastema treatments, add that there are diastemas of iatrogenic etiology. As examples, the authors cited the rapid expansion of the maxilla and a type of mechanics inadequate for diastema closure, where an elastic band is positioned on the central incisors, but slides in the cervical direction, causing periodontal damage and root approximation, with consequent divergence of the crowns, making the diastema even wider.

4. Treatment of anterosuperior diastemas

In the deciduous and mixed dentition phases, interincisive diastemas are considered normal [11–13, 16]. However, in the permanent dentures, diastema is frequently associated with several occlusal problems, which include missing teeth, dental anomalies, abnormal bone

structures, and excessive horizontal and/or vertical trespass [24]. It should be remembered that the diastema of racial and genetic etiologies are considered normal and the treatment will be dispensable, unless the patient considers the diastema an esthetic problem [24].

Patients' perception of the need for treatment for anterosuperior diastemas is influenced by the epoch and culture in which they live [29]. There is also a great contribution of the media to the opinion of people. In the 1960s, Gardiner [20] referred to a famous movie actor who presented a medium diastema and suggested, between the lines, that this feature, when smooth, may be well accepted for those individuals with a rather pleasant facial appearance. However, this cannot be considered for the majority of the population. The author's opinion was retracted through the following statement:

It was demonstrated [30] that when patients self-evaluate, they perceive a greater need for orthodontic treatment, when the problem is located in the anterior region, compromising aesthetics, as is the case of the anterosuperior diastema and anteroinferior crowding.

4.1. Treatment of diastemas in the mixed dentition

In the majority of cases, the central interincisive diastemas in the mixed dentition period are a temporary physiological feature of the "ugly duckling phase" [12, 13], which will be closed gradually with the eruption of the permanent lateral incisors and then with the eruption of the permanent canines. However, orthodontic interception is indicated in exceptional cases, where the diastema is preventing normal eruption of permanent teeth, stimulating the appearance or maintenance of deleterious habits, or compromising the child's self-esteem [16].

4.2. Treatment of diastemas in permanent dentition

There are several approaches used in the treatment of anterosuperior diastema, which vary according to the present etiologic factor [2, 16, 17]. The success of such treatment will depend on the elimination of these factors [17].

4.2.1. Orthodontic treatment

The orthodontic approach can be performed with the following objective: close the diastema or redistribute the spaces for a posterior reanatomization of the anterior teeth. Orthodontic treatment also has the function of treating, if present, any other associated occlusal problem and helping in the elimination of parafunctional habits. In cases where there is discrepancy of dental size, orthodontic treatment alone is not able to offer the best results. Therefore, these diastemas must be closed by means of composites, facets, or prosthetic crowns. However, for a better esthetic result of these restorations, orthodontic movement is indicated to redistribute the spaces before the cosmetic procedure [31].

Proffit, in the new volume of his book [32], divides the protocol of treatment of anteroposterior diastema according to two basic groups: (1) incisors with diastema and vestibular inclination and (2) diastema in the upper midline. In the first group, the diastema is usually caused by deleterious habit, which must be removed before the space closes, which can only

be performed with a removable device, retracting the incisors. In the second group, the author recommends the closure of the space, followed by frenectomy, in case there is excess tissue pressed in the midline.

In cases where there is a deep overbite, it is interesting that it is corrected previously. This usually increases the horizontal overpass, making it possible to close spaces together with an anterior retraction [24].

4.2.2. Frenectomies

Labial frenulum is considered abnormal when it is enlarged and/or inserted near the gingival margin [15]. Some authors [16, 33] recommend frenectomy to be performed after orthodontic closure of space, since diastema closure and interdental papilla compression may act as a stimulus to promote atrophy of the fibrous tissue interposed between the incisors.

4.2.3. Restorative treatment

Peck and Peck [34] stated that teeth are, by nature, perfect structures. However, a tooth with altered anatomy can often form a malocclusion. For the authors, the orthodontist should increase their understanding of the limitations of orthodontic therapy, and know the value of procedures to change dental forms. Only in this way can treatment success be achieved.

Andrews [35] cited in his book "Straight Wire: Concept and Apparatus" that when there are spaces between teeth due to discrepancy of tooth size, where there are small teeth, orthodontic correction is contraindicated, and recommend the restoration of these teeth with composites or prosthetic crowns.

However, even if there is a discrepancy of dental size, orthodontic treatment may be an auxiliary tool to redistribute the spaces between the teeth before the restorative procedure. This allows the dentition in the anterior teeth to be performed according to the golden ratio, obtaining a better esthetic result [24, 31].

4.2.4. Other types of treatments

Bell [36] argued for the immediate closure of diastema by subapical and interdental osteotomy, justifying that there is a great unpredictability of diastole orthodontic closure stability and that this approach is difficult and takes a long time. In addition, the author believes that the alveolar bone is the major factor responsible for the difficulty in the orthodontic movement of the teeth and for the final stability, as opposed to the majority of authors who consider the labial frenulum and adjacent soft tissues as the main factor for relapse.

5. Stability of diastema treatment

Concern over the stability of results obtained with treatment has existed for more than a century [37].

Riedel [38], in reviewing the problem of containment in the literature, proposed nine theorems that should be considered for greater stability of treatment results. In the first, the author mentioned that orthodontically moved teeth tend to return to their original positions. In Theorem 3, it is reported that the etiological factors of malocclusion should be eliminated for greater stability. Theorem 6 states that the bone and adjacent tissues should have a time for reorganization around the new tooth position, so some type of holding device should be used. And finally, in Theorem 9, it is ensured that the higher the tooth movement, the less recurrence.

The work of Ormiston et al. [39] observed that the greater the severity of malocclusion, the greater the recurrence. This means that the greater the severity of malocclusion, the greater the movement required and the greater the relapse. In this way, they oppose Riedel's Theorem 9 [38].

There are few scientific studies that evaluate the diastema recurrence after orthodontic closure [15, 28, 40, 41].

Orthodontic corrections in the growth and eruption phases of the teeth are considered more stable by some authors. According to Reitan [37], there will be little or no recurrence after orthodontic movement of an erupting tooth due to the fact that the supporting tissues are in a proliferation stage as a result of the eruption process. New fibers will be formed as the root develops, and these new fibers will help maintain the new tooth position.

Almeida et al. [16] have speculated that the closure of diastema in the mixed denture phase is more stable than if treated in the permanent denture. Therefore, it does not require definitive containment, as long as the habit is removed and, in case of interincisive gingival hypertrophy, surgical correction is performed before removal of the device.

Edwards [15] cited several factors responsible for the reopening of the diastema: incorrect axial inclination of the central incisor roots, tooth size discrepancies, deleterious habits, deleterious occlusal patterns, such as displacement from centric relation to maximal habitual intercuspation or other mandible position, which generate lateral forces on the central incisors, anatomy of the teeth (wider cervical region than the incisal region), and possibly some muscular imbalance in the oral cavity. But in addition to these, the labial frenulum, along with associated tissues, is cited as the most frequent etiological factor in relapsing interincisal diastemas.

The influence of muscular imbalances on the maintenance of bad dental positions varies according to three factors: duration, frequency, and intensity. Studies [10, 22] emphasized that when an etiological factor, such as atypical lingual pressure, cannot be eliminated, it is very difficult to keep the space closed, without the use of permanent retention, especially in adult patients. Attia [10] mentioned that in these patients, the chances of success of improving the lingual posture with only exercises are minimal and indicates the glossectomy.

Shashua and Ärtun [28] evaluated the proportion of diastema recurrence and the possible variables that may have contributed to this recurrence in a sample of 96 patients 4–9 years after the end of treatment. Diastemas varied from 0.5 to 5.6 mm in the pre-treatment stage. The following variables were analyzed: initial diastema size, tooth size discrepancy, overbite, upper incisor inclination, maxillary incisor mobility, labial frenulum, intermaxillary

septal cleft, root parallelism, anterior maxillary spacing, periodontal bone loss and heredity. Pre- and posttreatment data were obtained from the available documentation (models, radiographs, photographs, and clinical records). Post-retention data were collected from a follow-up visit of 37 patients (group A) and a telephone interview of 59 patients (group B). The incidence was 49% in the total, and 46% in group A, considering the patients with the diastema observed, or already portrayed by restoration or orthodontically, and still those who make continuous use of the retention by perceiving a tendency to reopen of space. However, in group A, the space in patients with recurrence ranged from 0.3 to 0.6 mm, but the mean was 0.1 mm. Logistic regression analysis revealed that the initial width of the diastema and the family tendency were risk factors for relapse. Although patients with abnormal braking had earlier initial diameters than those with a normal brake, no association was found between recurrence and the presence of an abnormal or crevice brake in the intermaxillary bone crest. The mobility of the upper incisors was the only parameter found in the postretention period that could be associated with the reopening of the space.

Surbeck et al. [41] evaluated the influence of occlusal changes on the stability in the anterior region of the maxilla. The sample was selected from study models for the posttreatment stage and was divided into three groups: (1) with anterosuperior spaces in this phase; (2) with crowding in the anterosuperior region; and (3) with perfect alignment. In group 1, it was found that the presence of spaces before treatment, as well as at the end of the treatment, increases the risk of increased spaces after treatment is finished. The authors suggest that the contention strategy should be altered according to the presence of spaces before treatment and their severity. Also, an association was observed between the increase of the spaces in the post-containment period and the reduction of the intercanine distance during the treatment. It was also found that reopening of spaces was associated with increased arch length and intercanine and intermolar distances in the posttreatment period.

5.1. The upper lip frenulum and its relation to the recurrence of diastema

Some authors [15, 21] agree that orthodontic closure of diastemas caused by an abnormal labial frenulum, without subsequent brake removal surgery, greatly increases the frequency of relapse in the post-retention period.

Edwards [15] evaluated the relation of the abnormal labial frenulum with both the etiology of diastema and the stability of its treatment. In this study, the author concluded that frenectomy had a great contribution to increase the stability of the orthodontic treatment of the median diastema and commented, citing that the upper labial frenulum is one of the most relevant factors for the reopening of orthodontically closed diastema.

On the contrary, Shashua and Ärtun [28] did not consider the central labial cleft lip and fissure in the central interincisive bone crest as risk factors for diastema recurrence, after evaluation of diastema relapse 4–9 years after the end of treatment. The authors [28] observed that the presence of the abnormal brake at the beginning of the treatment influenced the initial width of diastema. However, they emphasized that this type of brake can remodel spontaneously with the closing of the space.

5.2. Relation between periodontium and recurrence of diastema

It is believed that alveolar bone played the most important role in reopening orthodontically closed spaces [36]. However, several authors [15, 42] consider supra-alveolar gingival tissues as a primary factor in the relapse of dental positions.

Transseptal fibers are part of the supra-alveolar fibers group of the periodontal ligament and are composed mainly of collagen fibers. They are firmly inserted into the cementum, close to the cementum-enamel junction, and are responsible for holding together the adjacent teeth. They do not have elastic fibers, but their elastic characteristic is due to their structural form of tiny springs, which form as the fibers mature [25]. It is believed that their role in medial diastema recurrence is due to this elastic property, pressing the incisors mesially and pulling them incisively distally to the initial position [25, 42].

When they examined microscopically the interdental papilla of patients with median diastema, Campbell, Moore, and Matthews [42] observed that the insertion fibers of the brake had continuity with the gingival fibers of that area. The authors also verified an excess of accumulated and compressed gingival tissue in this area after the orthodontic closure of the space, which would act to reopen the spaces. In conclusion, it was suggested that the fibrous network of interincisive supra-alveolar tissues may be one of the main factors responsible for median diastema recurrence.

Similar situation, where a compression of gingival tissues was observed in orthodontically closed dental extraction sites, had already been observed by Edwards [15] and Parker [43]. In the aforementioned study, Edwards [15] noticed that the transseptal fibers, especially those near the alveolar ridge, reorganized and appeared normal after 10–14 days with retention of space closure. The author suggested that this was caused by the heavy forces applied during the final closure of space and by the approximation of the roots (parallelism), causing ischemia at the site and subsequent destruction of the compressed transseptal fibers. These, in turn, would be completely replaced by new fibers. Ten Cate et al. [44] have mentioned that it would be possible to remodel the transseptal fibers by a process called fibroblastic activity, where the fibroblasts are able to synthesize and degrade the collagen simultaneously, controlling its remodeling in the periodontal ligament and in the deeper transseptal areas.

In addition, Parker's findings [43] demonstrated that transseptal fibers within 60 days did not readapt to the new tooth position and were responsible for the reopening of the space. In this study, the effect of transseptal fibers on the stability of space closure was histologically evaluated, where the first permanent molar was extracted and the second premolar was retracted. In addition, the authors observed that the parallelism of the roots at the end of the closure increased stability and concluded that the fibrotomy of these fibers together with the containment had a positive effect on the stability of the retraction.

Bell [36] believed that the interdental septum was responsible for the instability of diastema closure and advocated subapical osteotomy in the space to be closed to facilitate movement and increase the stability of space closure.

5.3. Mesiodistal angulation of maxillary incisors

It has been mentioned in the literature that there is a greater stability of extraction space closure if the roots end up parallel, because when the roots also follow, the roots are also accompanied, a compression is generated in the place, causing ischemia and consequent destruction of the fibers transseptals. Subsequently, new fibers are formed and configured according to the new tooth position [33, 43].

Mulligan [45] stated that divergent roots are more conducive to keeping the space closed. He believes that when the roots are parallel, the vector of functional forces goes through the long axis of the tooth. However, when the roots are divergent, there is a space between the force vector and the tooth resistance center. This creates a moment that favors the approach of the crowns. The author also added that each patient has a moment of stability. And if within a period of up to 6 weeks without orthodontic treatment, the space remains closed, the ideal moment has been reached. But if there is a recurrence of the diastema, it would be necessary to further diverge the roots.

According to Reitan [37], the stability of the tooth position is greatly influenced by the direction in which the tooth was moved. The vestibular and lingual inclination of teeth has great chances of relapse. However, the mesial and distal movements, with a period of containment, are stable movements, except in cases of extrabuccal force for Class II correction.

Morais et al. reported no association between relapse of interincisor diastema and root parallelism. While midline diastema relapse occurred in 60% of the sample, lateral diastemas closure remained stable after treatment. Only initial diastema width and overjet relapse showed association with relapse of midline diastema [46].

5.4. Retention

In a study by Edwards [15], a recurrence rate of 88% was found in patients with abnormal labial frenulums who used retention for 16–22 months. In another similar group, but with a retention time of 8–10 months, this proportion was similar, 91% of cases with reopening of diastema. This may suggest that, in patients with abnormal labial frenulum, stability is not influenced by retention time, but rather by labial frenulum surgery.

Depending on the type of initial malocclusion, the use of retention throughout life is recommended [38].

Fixed retention is often cited as the only satisfactory method to promote stability at the closure of the previous diastema [32, 47]. However, the commonly available fixed retention present undesirable characteristics for long-term use, since they restrict access to gingival tissues, making their hygiene difficult. Currently, there is a type of fixed retention through small magnets attached to the lingual surface of the incisors, in previously diastematic areas [48].

Removable retention is not considered a good choice in cases of interincisive diastema because, soon after removal, the teeth already begin to move away [32, 45]. In addition, prolonged use of this retainer category would generate back-and-forth movements, which are potentially damaging.

6. Conclusions

The origin of the anterosuperior diastemas is multifactorial. In this sense, the clinician has several approaches for its treatment, which must be selected according to the etiological factor.

The etiology of the diastemas is associated to: abnormal labial frenulum microdontia, agenesis of maxillary incisors, dental discrepancy, shape of the anterior teeth, brachyfacial pattern, a positive tooth-bone discrepancy, supernumerary teeth, cysts, tumors, macroglossia, neuromuscular imbalance of the tongue, and orthodontic treatment.

Orthodontic treatment is a good alternative for diastemas closure, because it can also treat any other associated occlusal problem and help to eliminate parafunctional habits, if any.

When the width of the initial diastemas is very pronounced, the use of retainers can be considered throughout life. Fixed retention is often cited as the only satisfactory method to promote stability in the closure of previous anterosuperior diastemas, while removable retention is not considered a good option.

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Conflict of interest

The authors declare to have no conflicts of interest.

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Chapter 8

Current Approaches in Orthognathic Surgery

Mustafa Sancar Atac

Additional information is available at the end of the chapter

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Abstract

The orthognathic surgical procedures are performed for the correction of abnormalities of the facial skeleton that are present from the birth or arise during growth or acquired secondarily during lifetime. Due to the cover of this book as orthodontics, I would prefer to summarize some commonly used techniques to correct the dentofacial deformities. Even we have published all these techniques at their popular time with our orthodontist colleagues; skeletal anchor systems, some basic interdental osteotomies, or complex mechanics that are applying orthopedic corrective forces are currently being used by the orthodontists rather than surgeons. Le Fort I osteotomy in maxilla and sagittal split ramus osteotomies (SSRO) in mandible are commonly used techniques to solve the deformity problems of the facial skeleton; therefore, the scope of this chapter is going to be including my personal experience and some technical details with Le Fort I and SSRO.

Keywords: orthognathic, surgery, dentofacial deformity, Le Fort I osteotomy, sagittal split ramus osteotomy, maxillary osteotomy, mandibular osteotomy, airway, facial esthetics, genioplasty, facial harmony, piezoelectric surgery, piezosurgery

1. Introduction

The orthognathic surgical procedures are performed for the correction of abnormalities of the facial skeleton that are present from the birth or arise during growth or acquired secondarily during lifetime. The variety of the underlying reasons of the facial deformities would require different types of surgical strategies, but mainly orthognathic surgical techniques are preferred for the rehabilitation of the deformities. Due to the cover of this book as orthodontics, I would prefer to summarize some commonly used techniques to correct the dentofacial deformities.

Skeletal anchor systems, some basic interdental osteotomies, or complex mechanics that are applying orthopedic corrective forces are currently being used by the orthodontics rather than surgeons. Even we have published all these techniques at their popular time period with our orthodontist colleagues. Therefore in this chapter, I will focus on the most commonly used surgical applications to solve the skeletal discrepancies mainly called as Le Fort I osteotomy and BSSRO. These techniques can be applied as a single-jaw surgery or double-jaw surgery depending on the magnitude of discrepancy of the jaws to each other. Besides these basic techniques can also be preferred in association with distraction osteogenesis or orthodontic elastic traction forces for gradual and slow motions of the segments as well, but I think it is better to discuss the indication not in this chapter. Therefore the scope of this chapter is going to be including my personal experience and some technical details with Le Fort I and SSRO.

2. Bilateral sagittal split ramus osteotomy (BSSRO)

The history of orthognathic surgical techniques goes back to the mid-1800s. Both in Europe and the USA, the corrective jaw surgeries at their first steps begun to be heard by the population. Schuchardt was the author who first described the sagittal split in 1942 in German literature [1]. In English literature, Trauner and Obwegeser were the pioneers in 1957 who described and discussed the technique in detail [2]. Dal Pont [3], Hunsuck [4], and Epker [5] all added modifications to the technique such as to maintain more surface contact between bony segments at outer cortex, obtaining a minimal horizontal cut in the medial surface of ramus just beneath the lingula and minimal muscle detachment to avoid postoperative blood loss and hematoma.

2.1. Indications

BSSRO is a perfect option for surgical treatment of dentofacial deformities including the mandibular deformities. The mandible can be repositioned in three dimensions of the space such sagittal direction (as advancement, setback), transversal direction (asymmetries and shifting from one side to the other or transversal rotation), and axial plane (clockwise or counterclockwise rotation). For each movement a patient-specific osteotomy and muscle dissection should be kept in mind to avoid postoperative relapse and skeletal disfigurement. On the other hand, while planning such osteotomies, the facial harmony, speech, tongue size, esthetics, and most importantly the airway should be precisely evaluated. As an example huge magnitude of setback of the mandible would result as decreased airway volume and in advance as OSAS. The preoperative evaluation should include a 3D airway evaluation. If such compromise occurs, other osteotomy techniques should be analyzed such as mandibular ostectomy, IVRO. Also in macroglossia patients, tongue reduction surgery should be kept in mind before or during operation in setback surgeries to avoid dentoskeletal relapse. The incision would be placed on the ascending ramus in proximal segment and be carried inferiorly on the distal segment over the external oblique linea, while a mouth gag or a position stabilizator is placed on the opposite side with maximum mouth opening. Care must be taken to avoid dislocation

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of any condyle during this point (**Figure 1**). An electrocautery knife or a 15 blade would be preferable depending on the surgeon. My choice is the Colorado Needle which may help us for delicate incision without necrosis of the soft tissue with a good bloodless vision. The trick with the electrocautery is to perform repetitive coagulations on the same soft tissue plane to avoid tissue necrosis. A layer-by-layer dissection of mucosa muscles and periosteum is necessary. Adequate amount of soft tissue should be remained on the attached gingival side for wound closure. The mucoperiosteal full thickness flap is raised on the external oblique linea. If the temporal muscle tendons are identified, they can also be reflected. An Obwegeser ramus retractor or a curved hemostat is positioned on the tip of coronoid process. The anterior inferior border of the mandible which is close to the external oblique linea can also be reflected, and during bone osteotomies an Obwegeser channel retractor should be placed on the inferior mandibular border. The lingula and the mandibular foramina should be identified on the medial surface of the mandibular ramus and a Williger or Obwegeser raspatorium is used to protect the neurovascular bundle above its insertion to foramen mandibularis. This maneuver will guide to horizontal medial bone osteotomy just above the foramina. There is no need to perform posterior dissection to the posterior border of the mandible. It is better to create a soft tissue tunnel subperiosteally and keep it just to the posterior of the foramina. This will also reduce the possible intraoperative excessive bleeding arising posteriorly and medially from the adjacent muscle and vessel structures (**Figure 2**). Once all the bony aspects of the desired osteotomy lines have been approached, depending on the surgeon's preference, a cutting instrument like saws, rotary handpiece, or piezoelectric surgery handpiece can be used. If the patient has no cardiac disease like arrhythmias or is not a pacemaker user, my first choice is piezoelectric ultrasonic surgery. The piezoelectric ultrasonic surgery is a well-defined system, the first steps of which were discovered by Pierre and Jacques Curie Brothers in 1880. Piezoelectricity is the electric charge that accumulates in certain solid materials (such as crystals certain ceramics, and biological matter such as bone, DNA and various proteins) in response to applied mechanical stress. The first ultrasonic alveolar bone cut was performed by Horton on the dog alveolus in 1975 [6]. In piezoelectric surgery, the cavitation phenomenon

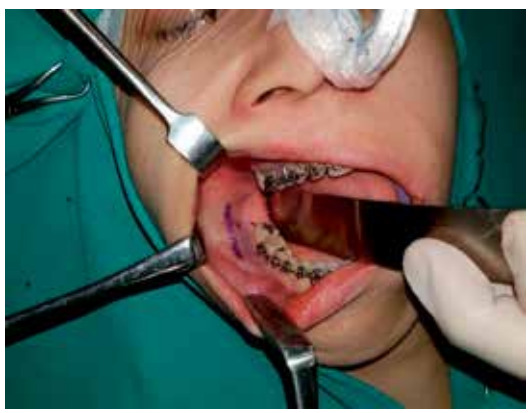

Figure 1. The incision line placed over the external oblique line and ascending ramus. Carrying it to high and medially will cause herniation of temporoparietal fat pad (Bichat's fat pad).

Figure 2. Ramus single white arrow: alveolaris inferior neurovascular bundle; double white arrow: Obwegeser ramus retractor; and three white arrows: Obwegeser ramus channel retractor. Please note that the Obwegeser periosteal raspatorium is placed above the foramina and protecting the neurovascular bundle. Also a sub periosteal dissection has been performed creating a tunnel to avoid haemorrhagia. Also pay attention to pencil drawing along the ascending ramus declining on the external oblique linea finalizing at the anterior most inferior edge of the linea. This cut will create a separation between distal and proximal segments.

describes the process of vaporization, bubble generation, and subsequent implosion (growth and collapse of bubbles) into many minute fractions of its original size (microscopic gas bubbles) that will occur in a flowing liquid because of the decrease and increase in pressure that is caused by the ultrasonic vibrations. In ultrasonic osteotomy, the cavitation phenomenon helps to maintain good visibility in the operative field by dispersing a coolant fluid as an aerosol that causes the blood to essentially be washed away. Furthermore, the cavitation effect will bring about hemostasis, which results in a bloodless surgery. Walmsley et al. have suggested that the cavitation effect fragments the cell walls of bacteria and therefore has an antibacterial efficiency [7]. Also, piezoelectric device has a selective cutting effect and has a sound alert warning system when the tip has reached to a nonresisting part such as neurovascular bundle or sinus membrane that avoids trauma to soft tissues. Therefore, hemorrhagic and neurosensory deficits are rare in piezoelectric surgery. Afterward Vercellotti published his experience with piezoelectric surgery [8]. In orthognathic surgery Landes et al. shared their experience with piezo-osteotomy feasibility as a substitute for the conventional saw in orthognathic surgery that was evaluated regarding operative technique, blood loss, time requirement, and nerve and vessel integrity. Fifty patients who had orthognathic surgery procedures using piezo surgical osteotomy showed that piezoelectric osteotomy reduced blood loss and inferior alveolar nerve injury at no extra time investment, compared with 86 patients who received conventional saw and chisel osteotomies [9]. We have also evaluated piezosurgery in PhD thesis and compared the conventional rotary instruments with piezosurgery. The aim of this study is to comparatively evaluate primary postoperative patient complaints such as

edema, paresthesia, and pain and patient satisfaction and operation times of orthognathic surgical operations performed with conventional drills and piezoelectric surgery. A total of 200 patients with completed skeletal growth and malocclusion due to mastication dysfunctions were retrospectively evaluated. These patients were divided into two main groups named control group and piezo group. Each group was divided into three subgroups about the type of surgical procedure performed, which are, Le Fort 1 subgroup, bilateral sagittal split ramus osteotomy (BSSRO) subgroup, and bimaxillary subgroup. The evaluation between the groups is made at postoperative 1 day, 1 week, and 1, 3, and 6 months. To evaluate each subgroup, the data acquired from a specific subgroup was compared to the subgroup with the same name in the other main group. After piezoelectric surgery edema, neurosensory dysfunction and pain levels were found to be lesser than conventional techniques. Patient satisfaction was found to be higher in piezoelectric surgery patients. When operation times were compared, piezoelectric surgery was discovered to take longer to finish the osteotomy because of its lower cutting efficiency. This study shows that the selective cutting ability of the piezoelectric surgery device provides an extremely safe osteotomy for patients by performing a selective osteotomy, thus preserving critical adjacent soft tissues [10]. The piezosurgery begins with the medial aspect of the ramus just over the mandibular foramina, and the cutting tip of the handpiece will be directed 45° angle at posterior start point with around a depth of 2 mm and comes anteriorly. On the ascending ramus, the tip is applied without angulation as deep as possible through the cortex to reach the medullary bone and declines inferiorly on the external oblique linea. If the procedure is a mandibular setback surgery, it is better to extend the osteotomy till the anterior border of the external oblique linea which would help the removal of bony segment for desired positioning of the distal segment similar to Hunsuck-Epker modification. In my experience if a rotational or laterognatic corrective sagittal split osteotomy is going to be performed, the anterior vertical osteotomy on the lateral cortex would be like Obwegeser technique that goes posteriorly to the angulus of the mandible. Besides if the rotation amount is excessive, care must be taken not to dislocate the proximal segment laterally. If such situation occurs, a greenstick fracture must be performed on the posterior part of the distal segment distal to the last molar. In thin and small volumetric mandibles generally, piezoelectric ultrasound surgery is enough to complete the whole osteotomy running from the superior cortex border to the inferior bony cortex border through a fashion without chisel and hammer or separators. Also, this would avoid unfavorable fracture of the segments during rotation split maneuver of the osteotomes that are placed between proximal and distal segments. To avoid undesired inferior border, splitting piezoelectric surgical instruments tips can be directed to the inferior border to maintain a bone cut at the inferior border initiating from the end of the anterior vertical osteotomy back to the posterior end just inferior to orthogonal projection of the lingula. The osteotomy and separation of the proximal and distal segments will simultaneously be completed via this technique without chiseling and hammering. I personally call this technique as Piezotomy Technique®. If the osteotomes are needed to complete the osteotomy, thin osteotomes would be helpful to complete cortex osteotomies keeping them away from the neurovascular bundles. While osteotomizing the inferior border of the mandible, the Obwegeser channel retractor is essential to guard the underlying soft tissues and vital structures. It is better to use a thin osteotome to start separation of the segments seen in **Figure 3**. Completing the osteotomies bilaterally and

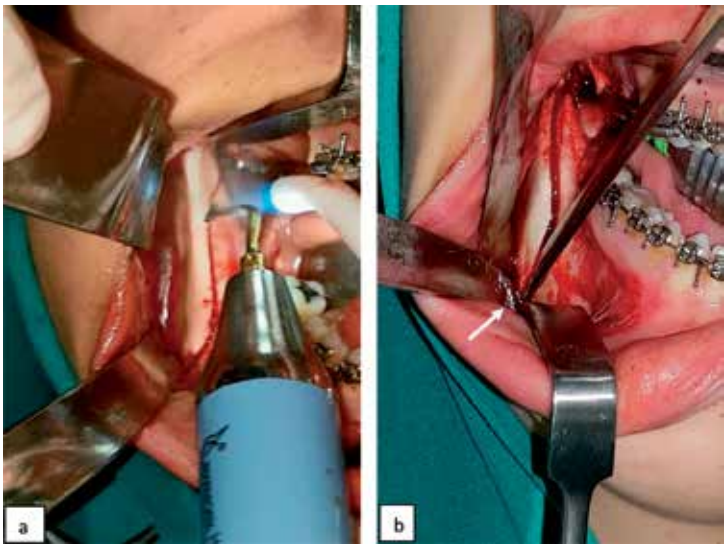

Figure 3. (a) Osteotomy line starting from medial surface of ramus down to ascending ramus to he external oblique linea inferiorly with ultrasonic surgery; (b) thin osteotome is placed for separation of outer cortex of the inferior border.

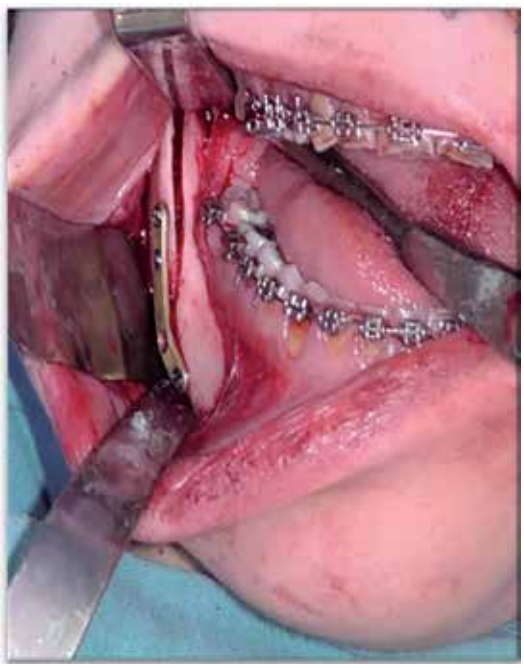

Figure 4. The sagittal mini plate has been bended and passively adopted to proximal and distal segments with monocortical mini self-drive screws and let mandible for early functioning.

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detaching the muscle attachments that are resisting for mobilization of distal segment, the surgical splint is placed, and an exact intermaxillary fixation is secured. In this period if it is a setback surgery, the excess bone is removed. At this point, for rigid fixation, the surgeon can prefer lag screws or miniplates with screws to stabilize the segments. My personal choice is to use sagittal plates with self-drive mono cortical screws for rigid fixation. This is essential to avoid condylar sag and distortion of proximal segments as well, especially in the laterognatic and asymmetric cases that require shifting or rotation of the mandible. Also bending the plates geometrically to create a step would stabilize the segments in passive but stable position avoiding the relapse phenomenon. The proximal and distal segments are rigidly fixated (**Figure 4**). After bleeding control the wound edges will be sutured with 3.0 resorbable sutures. Suctioning drains are placed if required.

3. Le Fort I osteotomy

The maxillary discrepancies and the midface abnormalities can easily be corrected by Le Fort I level osteotomy with variable surgical modifications. In 1927, Wassmund performed the first Le Fort I osteotomy for an open-bite deformity [11]. The advancement of maxilla was performed by Axhausen [12], and Bell was the first to show the vascular supply and safety of the procedure at this osteotomy level [13]. The quadrangular Le Fort I was first described by Obwegeser in 1969 [14].

3.1. Technique

The patient is intubated via nasotracheal preformed intubation tube (RAE tube) to avoid nasal deformation during the surgery. Prior the incision a circular infiltrative local anesthesia is administrated with vasoconstrictor agent. A mucosal incision at the vestibular fornix region from one premolar to another is initiated with Colorado Needle electrocautery (**Figure 5a**). To avoid repetitive coagulation, layer-by-layer single application is essential for deeper soft tissue structures down to the periosteum. A mucoperiosteal flap is raised both on sagittal and vertical plans. Laceration of the periosteum would result in discomfortable bleeding and herniation of Bichat fad pad. Depending of the level of the osteotomy, the reflection of the flap may extend up to the infraorbital region. A curved periosteal elevator would be essential for the elevation of the nasal mucosa on the medial sinus wall and midpalatal suture for each side (**Figure 5b**). If a collective fashion of haemorrhagia occurs, a fine rat tail tamponade with tranexamic acid solution will control it. Using piezoelectric ultrasonic surgery device, osteotomy will be initiated at the apertura priformis back to the crista zygomaticus and through the tuberosity of the posterior maxilla. Also via the piezosurgery tip, the medial antral wall osteotomy may be completed. Thanks to its selective cutting effect with cavitation phenomenon, also the palatine bone can be cut via piezosurgery. Even in some cases, from anterior nasal spine to posterior nasal spine, septal separation from midpalatal suture can be performed via piezoelectric surgery (**Figure 5c**). For each maneuver special piezo cutting tips can be preferred even for the pterygomaxillary junction region (**Figure 5d**). When all the

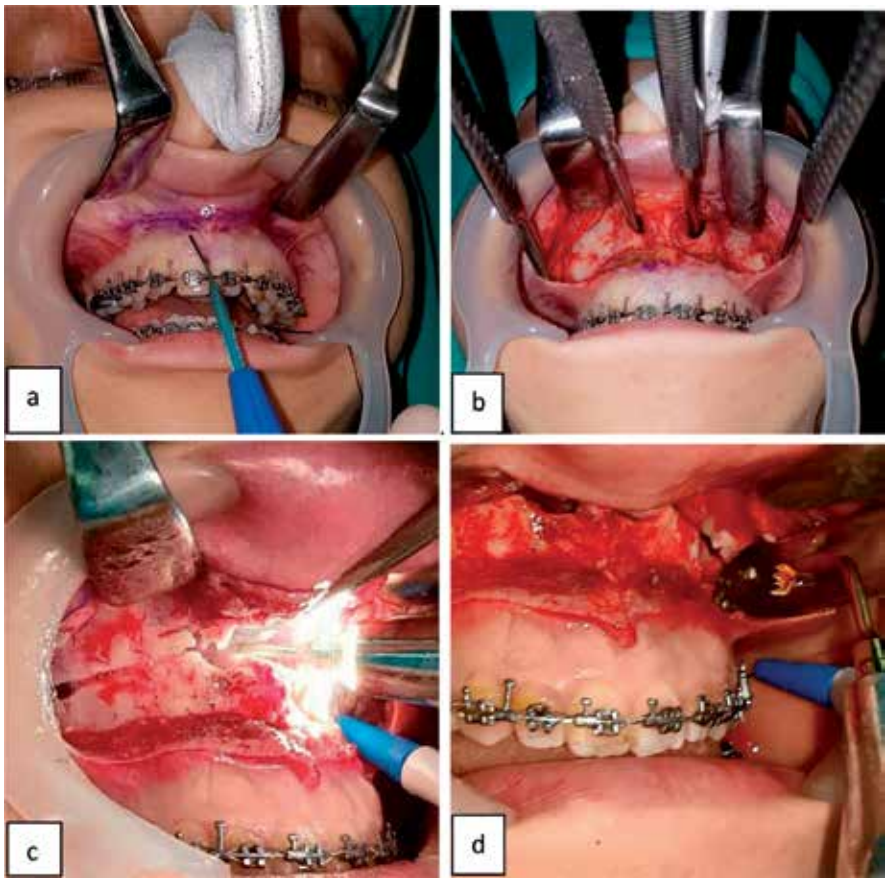

Figure 5. (a) Nasotracheal intubation via RAE tube, Colorado Needle for soft tissue incision, and the at very deepest sulcus, the incision is initiated between premolar regions; (b) soft tissue flaps created by subperiosteal tunneling; (c) anterior nasal spine to posterior nasal spine and also septal separation from mid palatal suture can be performed via piezo electric surgery; and (d) piezo cutting tips can be preferred even for the pterygomaxillary junction region.

osteotomies are performed via piezoelectric surgery, a bi-digital mild pressure on the anterior incisors is applied by the surgeon in vertical and sagittal directions for down fracture of the maxilla. In cases that osteotomies are performed by rotary handpieces or saws, the osteotomy lines are quite similar to piezosurgery, but care must be taken while malleating curved pterygoid osteotome to separate the pterygomaxillary junction. The surgeons' eye should follow the upper occlusion, and the osteotome should be parallel to this line to avoid directing it cranially. One should place the point finger of the assisting hand to the pterygoid hamulus intraorally to feel the tip of the pterygoid osteotome (**Figure 6**). If such malpositioning occurs, there is the risk of laceration of maxillary artery close to the posterior region. Also during medial antral wall osteotomy, care must be taken not to go so far posteriorly due to laceration risk for descending palatinal artery. Because around 3.5 cm posteriorly, the sound will change and a resistance will occur during chiseling. This means that one has reached to the perpendicular lamina of the palatine bone that descending palatine artery is

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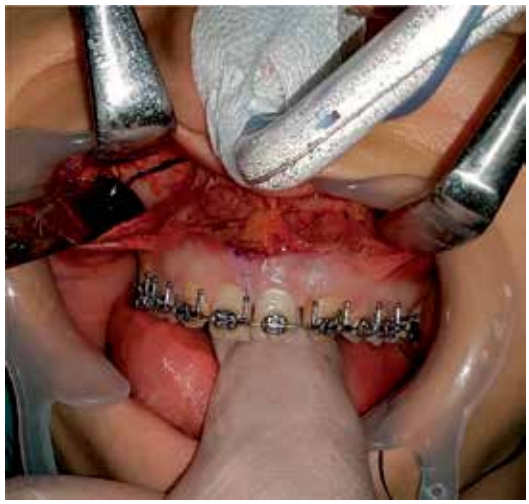

Figure 6. Pterygoid osteotome placed at the pterygomaxillary junction, and the assisting point finger is positioned on the hamulus pterygoideus to feel the disjunction and the tip.

running inside of it. If the surgeon is inexperienced, it is so easy to face an aggressive hemorrhage from the palatine artery. It can be controlled easily by direct packing with a immediate down fracture procedure to isolate the bleeding source. The septal cartilage and midpalatal bone would be separated using nasal septal osteotome. The assisting hand's point finger should be placed posterior-to-posterior nasal spine to feel the tip of the septal osteotome at the end (**Figure 7**). After the down fracture, Tessier mobilizers or Rowe forceps are used to mobilize the maxilla freely. At this point the lacerations on the nasal mucosa is sutured, and posterior reflection of the soft tissues was completed (**Figure 8a**, **b**). If the maxilla is going to be impacted, the septal cartilage would be trimmed as required to avoid deviation of the septum. If the anterior nasal spine is problematic for columella or nasal shape, it can also be removed till the desired level. If the magnitude of the advancement, impaction, or rotation is quite much, surgeon will face resistance; then decompression of the palatal artery and removal of the palatal bone around are necessary. These risky maneuvers can easily be done via piezoelectric surgery without bleeding. As an alternative, a round diamond rotatary burr can be preferred. Surgical saws may lead to hemorrhage. The maxilla will be repositioned with a surgical prefabricated splint followed by intermaxillary fixation. The bone edges are controlled, and interference of excessive bones is removed. When maxilla and mandible act as one unit due to intermaxillary fixation, rotation is completed by gentle force application on both sides of the mandibular premasseteric notches, and rigid fixation is performed (**Figure 9**). If regular micro- or miniplates are going to be used for rigid fixation; I suggest to use two plates on each side in inferior repositioning and advancement surgeries. If the maxilla is impacted, one four-hole L-shaped plate is adequate on each side. For the first time in the literature in 2008, I have published stress distribution both on plates (single- or double-plate fixation on each side) and facial skeleton after rigid fixation in Le Fort I osteotomies for all scenarios, and since 10 years, I clinically follow the guidelines of my publications to choose the number of the plates for each case [15–17].Also as an alternative, I

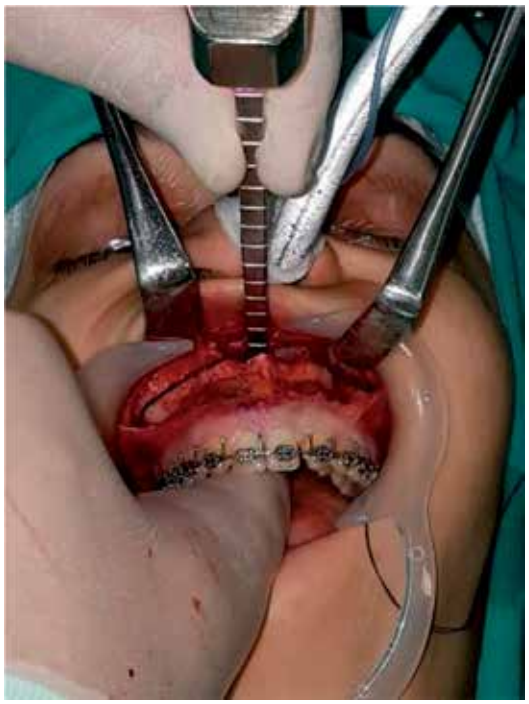

Figure 7. Nasal osteotome is placed on the midline just below the septum to separate the septal cartilage and the mid-palatal bone junction. Assisting point finger placed on to the posterior nasal spine.

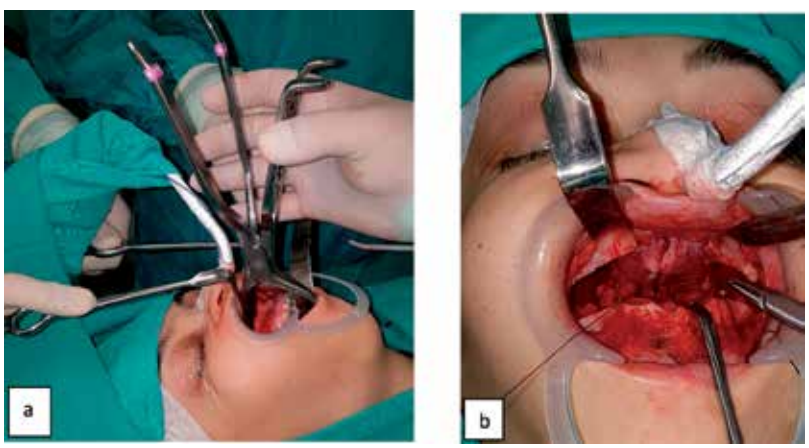

Figure 8. (a) Rowe forceps placed bilaterally on the down fractured maxilla to complete the disjunction, (b) suturing the nasal mucosa ruptures.

suggest to use preformed Le Fort microplates that are currently available on the market with different shoulder sizes (**Figure 10**). Another current advancement in rigid fixation materials is the patient-specific titanium miniplates that are manufactured via 3D printers. The use

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Figure 9. Intermaxillary fixation with a surgical splint, and superior repositioning of the complex with gentle pressure that has been applied bilaterally on both premasseteric notches.

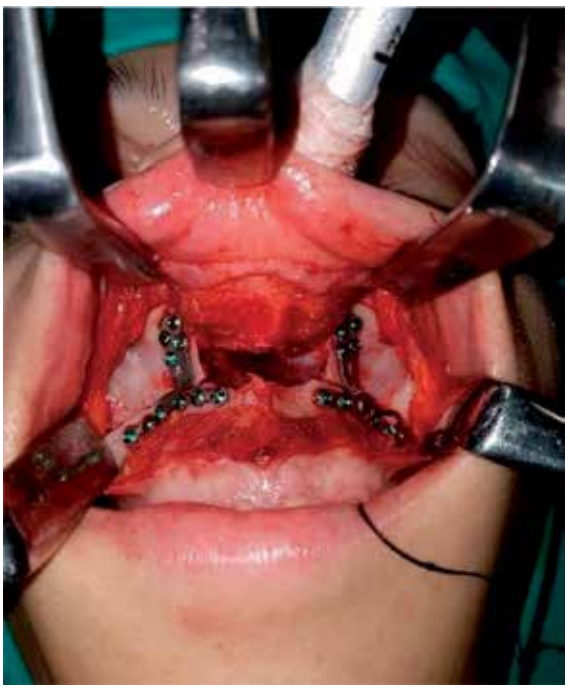

Figure 10. Preformed Le Fort miniplates and self-drive screws used for rigid [18] fixation. This special form supplies enough rigidity with just one plate and there is no need to place posterior plates around zygomatic buttress.

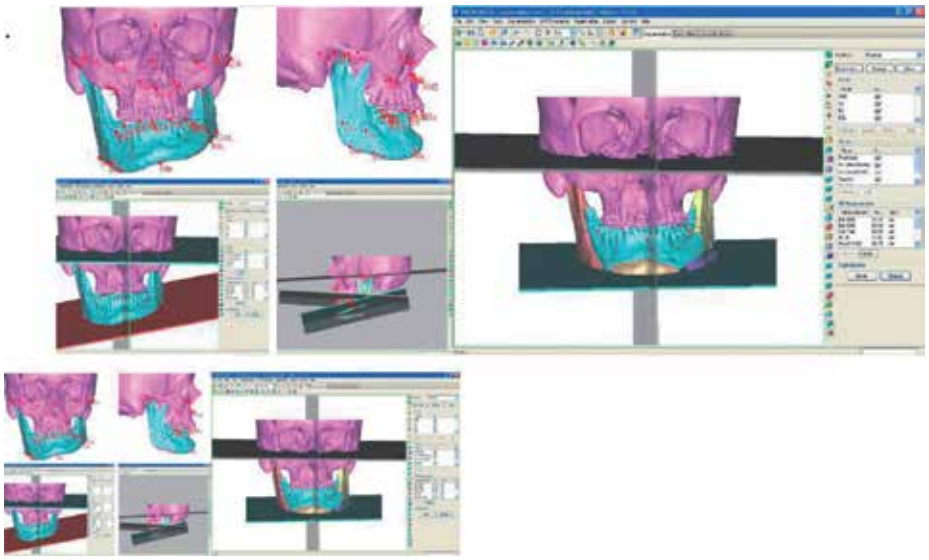

Figure 11. 3D evaluation and planning of a hemimandibular hyperplasia case using DICOM data with a surgical software, published in Journal of Cranio-Maxillofacial Surgery in 2009.

of 3D surgical planning in orthognathic surgery cases is on the market more than 10 years. Meanwhile we also published a paper with 3D tomographic DICOM data transferred to software and completed the operation virtually in 2009 (**Figure 11**) [18]. Depending on surgeons and orthodontist's choice, currently there are many alternatives to each other starting from conventional cast model articulating and model set up on casts, to 3D analyze and 3D printing of surgical splints and titanium fixation plates. On the other hand, higher technology for planning and manufacturing with 3D printers increases the financial expenses. After rigid fixation of the maxilla, the alar wings, nasal tip, and nostril symmetry and projection of the upper lips and the gums should all be evaluated. If the maxilla has been impacted, the nostrils and the nasal soft base will be wider, and alar chinch suture is essential to control the width. Also, a V–Y closure will help to improve the philtrum projection.

4. Bimaxillary surgery

The sequence of these surgeries may vary from surgeon to surgeon. My traditional way to do double jaw is to start with the maxilla first then complete the mandible. But in one indication, one should always start with mandible first which is the double-jaw counterclockwise surgery. Especially Class II with maxillary excess with gummy smile cases associated with OSAS or airway limitations, there is indication for counterclockwise double surgery. The surgical strategy should be mandibular advancement surgery with posterior inferior repositioning and anterior superior rotation. The space created at posterior occlusion is then filled with inferior rotation of the posterior maxilla with anterior maxillary impaction with Le Fort I osteotomy. The pivot point to perform this maneuver is premolar and zygomatic buttress region.

5. Conclusion and the future

As I have briefly explained the basic techniques that are commonly used in orthognathic surgery, for 5 years we are already beyond the future. The current advancements are tomographic evaluation including 3D reformatted frames integrated to advanced software programs with facial scanning to analyze the facial proportions rather than 2D cephalometric radiographs and 3D scanning of the teeth integrated with tomography that let to us perform operation in virtual environment and print out 3D splints. Besides other advancements are; 3D printing of the individual titanium plate screw fixation systems will avoid producing surgical splints, as well as using piezoelectric surgery rather than burs and saws supplies for bloodless and neuro-sensorial deficit-free operations, finally last but not the least, a totally robotic guided surgery will take place within a couple of years.

Conflict of interest

I declare that there is no conflict of interest.

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Chapter 9

Orthosurgical Correction of Severe Vertical Maxillary Excess: Gummy Smile

Fayez Saleh and Wisam Al Hamadi

Additional information is available at the end of the chapter

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Abstract

Gummy smile which is commonly described as excessive gingival display in full smile has become a major esthetic concern for patients and healthcare providers in recent years. Because of the complex etiologic factors and severity of this orofacial deformity including skeletal, dentoalveolar, muscular, and gingival, it is essential that a differential diagnosis be established so that a relevant therapy can be selected from a wide range of treatment options. The complex etiologic factors of this dentofacial deformity necessitates coordination between a team of health care providers, especially between orthodontist and maxillofacial surgeon, to improve diagnosis and treatment planning, accurately predict the outcome, and minimize orthosurgical complications. In this chapter, the author will present several clinical cases and briefly review available treatment modalities for excessive gingival display in full smile. Emphasis will be put on orthosurgical treatment of severe vertical maxillary excess as the optimal solution to restore facial balance and harmony, attractive smile, and patients' satisfaction.

Keywords: gummy smile, vertical maxillary excess, dentofacial deformity, orthosurgery, interdisciplinary approach

1. Introduction

Orthosurgical correction of dentofacial deformities aims to obtain more harmonious and esthetically pleasing facial proportions in addition to normal and stable functional occlusion. Facial esthetics cannot be considered apart from an attractive smile and vice versa.

Despite the variety of ethnic, racial, and individual perception of facial attractiveness and a pleasant smile, there are general consensus among health care professionals that harmony and

balance of the facial features are equally important as intrinsic dentogingival, lips, and perioral soft tissue components to be considered before designing a beautiful smile [1–3].

Gummy smile caused by vertical maxillary excess and dentofacial disharmony cannot be treated satisfactorily with adjunctive nonsurgical approaches such as botulinum toxin injection or a crown lengthening procedures or temporary anchorage devices (TDAs). An ideal treatment option for vertical maxillary excess (VME) is the reduction of the maxillary vertical excess by LeFort I osteotomy with superior maxillary repositioning [4, 5]. However, when gummy smile is not associated with severe skeletal discrepancy in three dimensions (3Ds), intrusion of anterior teeth using TDAs may be indicated provided that the anterior esthetic occlusal plane and smile arc are well preserved [6, 7].

Many nonsurgical orthodontic treatment modalities proposed to treat gummy smile in adult patients with severe skeletal facial problems were of limited use with no benefit on the skeletal vertical discrepancy [8]. Depending on the magnitude and severity of the gummy smile, combined orthosurgical techniques offer better esthetics and long-term stability [9].

Increased vertical dimension in class III cases is the most important and deciding factor to be considered when formulating a treatment planning and deciding the technique of orthognathic surgery [10].

Computer simulation of orthosurgical outcome which was once of great concern that showing predictions to patients might lead to unrealistic expectations and disappointments. Nowadays, image prediction is becoming more likely satisfying and acceptable as it allows patients to visualize possible soft tissue profile changes and help them to decide whether surgery would be worth in terms of the additional risk and cost [11, 12].

2. Orthosurgical treatment modalities of vertical maxillary excess: gummy smile

Because not all dentofacial deformities are alike, different treatment modalities were developed and advances in surgical orthodontics added a new dimension to the health profession and assured patients' satisfaction. The following case series intended to share experience with colleagues and students.

2.1. Case #1

A healthy patient (39 year 9 month old) presented with a chief complaint of "I am not happy with my smile, arrangement of my teeth, and prominent chin." Cursory examination revealed that the patient has skeletal and dental class III malocclusion with reverse overjet, narrow maxilla, long face, concave profile, and gummy smile. The case was complicated by old extraction of lower left first molar. Thorough clinical examination and diagnostic data collected assured limitation of orthodontic treatment alone to achieve treatment objectives and therefore,

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combined orthosurgical correction was suggested and the patient agreed after detailed discussion and joint meeting with the clinicians.

2.1.1. Diagnosis, treatment objectives, and treatment planning

Figures 1 and **2** explain the extra-oral, intra-oral clinical examination and radiographic assessment of the dentofacial components in 3Ds.

2.1.2. Treatment objectives

The objectives were directed to the listed problems to include: achieving esthetic facial features and pleasant smile; in addition to restoring normal occlusion in three dimensions (3Ds) as a prerequisite to ideal lip-teeth relationship. Retaining the treatment outcome is equally important, and all measures were taken to establish effective retention means before appliance removal.

Figure 1. Initial clinical (extra-oral and intra-oral) and radiographic examination that allow to formulate an appropriate treatment planning and setting the treatment objectives.

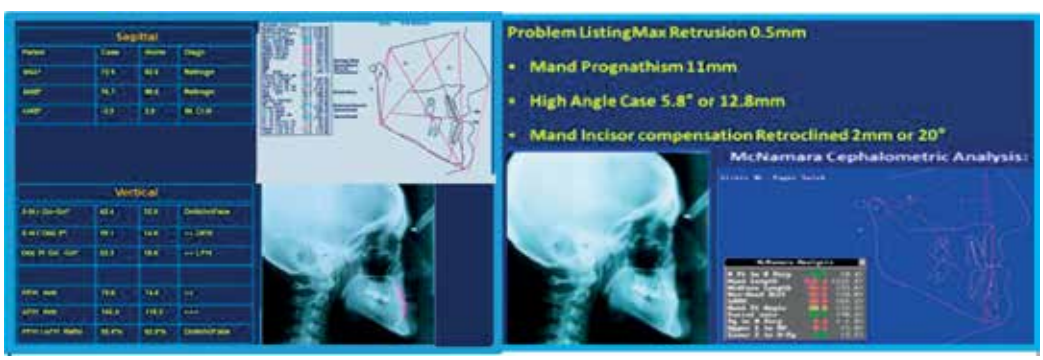

Figure 2. Jarabak and McNamara lateral cephalometric analysis, as adjunctive to clinical examination, to assess objectively the sagittal and vertical dentofacial deformity.

2.1.3. Treatment progress

2.1.3.1. Presurgical phase

Preparing the dentoalveolar arches for orthognathic surgery necessitated leveling and alignment of teeth, decompensating the retroclination of lower incisors, and achieving ideal arch form that allows surgical expansion through median maxillary split. **Figures 3** and **4** depict the sequence of orthodontic work and the simulation of surgical movements in 3Ds.

2.1.3.2. Postsurgical phase

Maintaining normal anterior overjet and overbite, plus ideal incisors, canines, and molar relationship during this phase is crucially important. The healing of bony parts during detailing of occlusion and any minor corrections if they ever exist is the key for success at this stage. Full-time wear of heavy elastics for 2 weeks after surgery to assure full dental interdigitation, arch symmetry, and stable treatment outcome. 4 month later, the appliance was removed, and the necessary retainers were constructed, the prosthodontist took good care of the four maxillary incisors upon the patient's request. Follow up for almost 5 years was planned, and the interdisciplinary approach yielded realistic adequate treatment outcome (**Figures 5**–**7**).

Figure 3. Treatment progress: decompensation of the incisor inclination and uprighting buccal teeth, leveling and alignment of teeth, flattening curve of Spee, restoring normal arch form, preparing model surgery, and constructing surgical hooks prior to surgery.

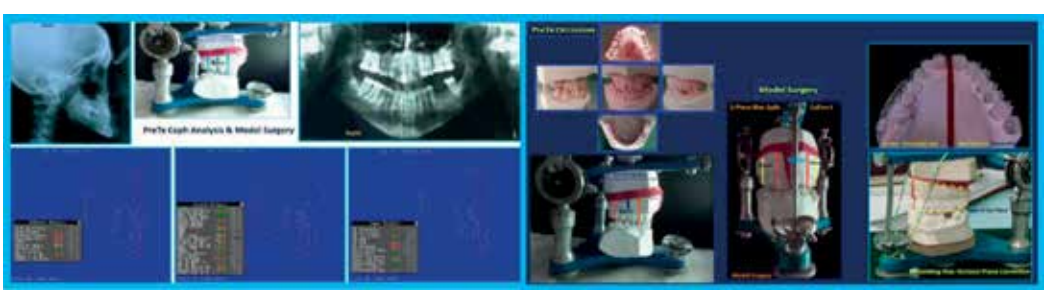

Figure 4. Models of the presurgical arches were constructed and mounted on semi-adjustable articulator, simulation of the surgical act included: superior maxillary repositioning and advancement, splitting the maxilla into two pieces for expansion, displaying final movements of the jaws and cant of the occlusal plane, and monitoring any possible occlusal interference to be resolved.

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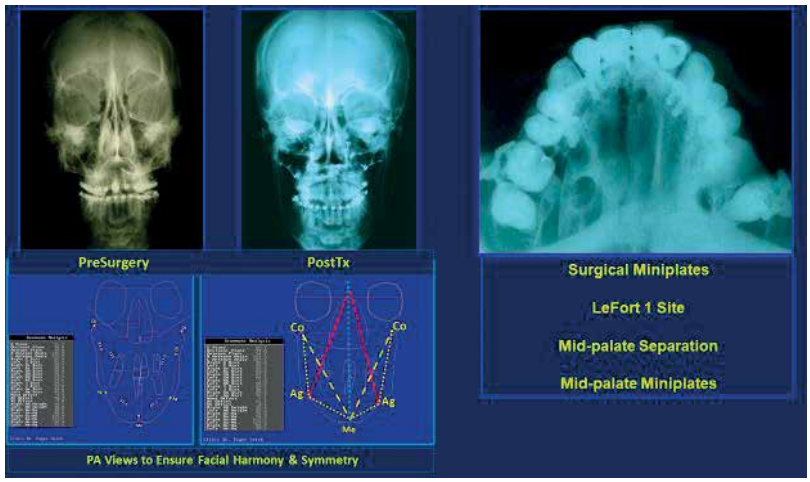

Figure 5. Pre- and postfrontal (PA) cephalometric analysis and occlusal view depicting the transverse surgical expansion of the maxilla with relevant min-plates in situ.

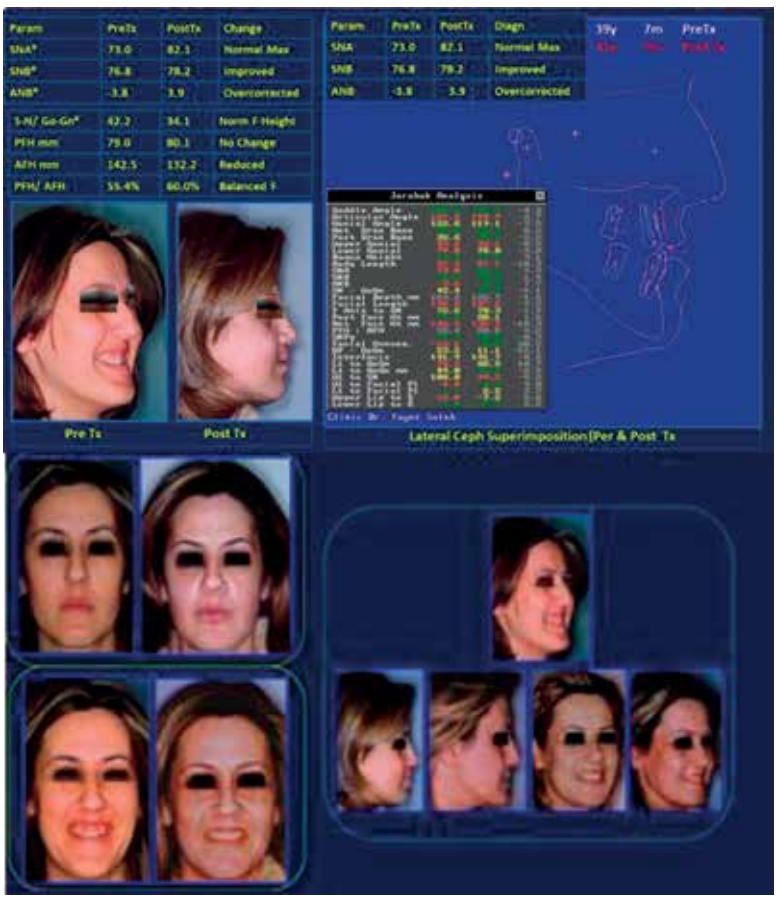

Figure 6. Pre and post photos and Lat Ceph analysis of Jarabak depicting the changes in profile.

Figure 7. Pretreatment occlusion, presurgical orthodontic preparation, postsurgical occlusion, and final prosthetic reconstruction of occlusion.

2.1.4. Treatment results

The following photos and radiographs reveal the impeccable improvement in the harmony and balance of the facial form in 3Ds, successful surgical expansion, and superior repositioning of the maxilla resulted in ideal functional and static occlusion. Postoperative radiographs have shown a well seated condyle in the glenoid fossa in an appropriate physiologic rest position. Patient's satisfaction was rewarding (**Figures 8–10**).

Figure 8. Pretreatment, presurgical preparation phase, postsurgical phase, and postsurgical radiographs.

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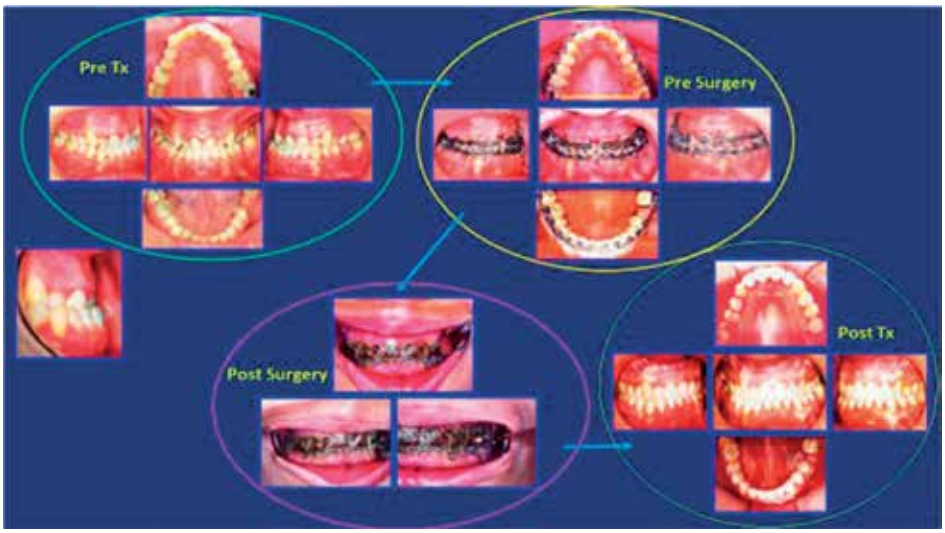

Figure 9. Pretreatment occlusion, presurgical preparation phase, surgical procedures, postsurgical phase to detail the occlusion, and posttreatment occlusion.

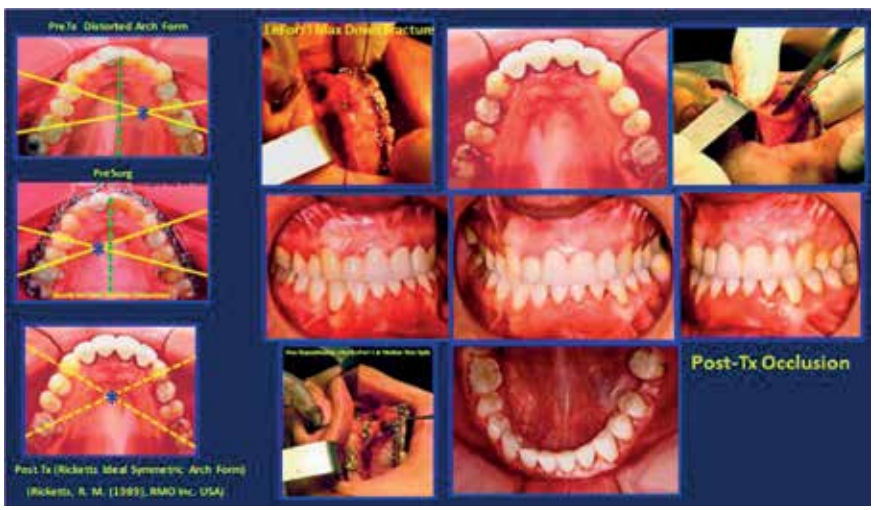

Figure 10. The final occlusion 5 year postretention focusing on the median maxillary surgical split to restore ideal arch form stable occlusion.

2.1.5. Conclusions

Attempt to treat dentofacial deformities without comprehensive data analysis and collaboration with other health care providers will not yield acceptable results and may need retreatment. Medical ethics obligate us to consider the patients' interest and welfare. This case, from the very beginning, was diagnosed as severe skeletal deformity in 3Ds and disproportionate dentoalveolar arches. The dentofacial imbalance and disharmony imposed the

orthosurgical treatment approach which was agreed upon by orthodontist and maxillofacial surgeon and readily accepted by the patient.

2.2. Case #2

A healthy female (24 year 5 month old) presented with apparent long face syndrome, convex profile, vertical maxillary excess causing gummy smile, incompetent lips, and deficient chin. Intra-oral examination revealed a class II division 1 malocclusion associated with narrow maxilla, deep palatal vault, micrognathia, moderate crowding mild anterior open bite, and distorted occlusal planes.

2.2.1. Diagnosis

Figures 11–14 exhibit the clinical and diagnostic aids that facilitate the formulation of appropriate treatment planning to achieve optimum treatment outcome.

2.2.2. Treatment planning and treatment objectives

Because of the diagnosed severe skeletal dentofacial deformity, joint consultation and thorough patient's data analysis took place by the orthodontist, and the maxillofacial surgeon, the treatment modality offered to the patient, after detailed discussion and displaying of the possible outcome, was a combined orthosurgical care which was willingly accepted.

Figure 11. Pretreatment facial, dentoalveolar, and radiographic photos.

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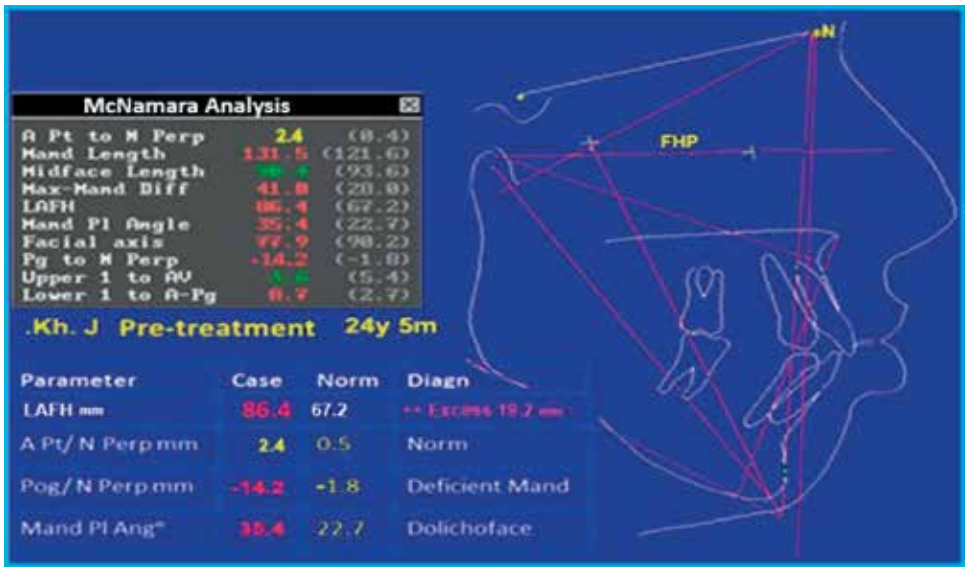

Figure 12. Pretreatment lateral cephalometric analysis of McNamara confirming the long face deformity and retrognathic mandible. Tipped down occlusal plane buccally caused backward rotation of the mandible.

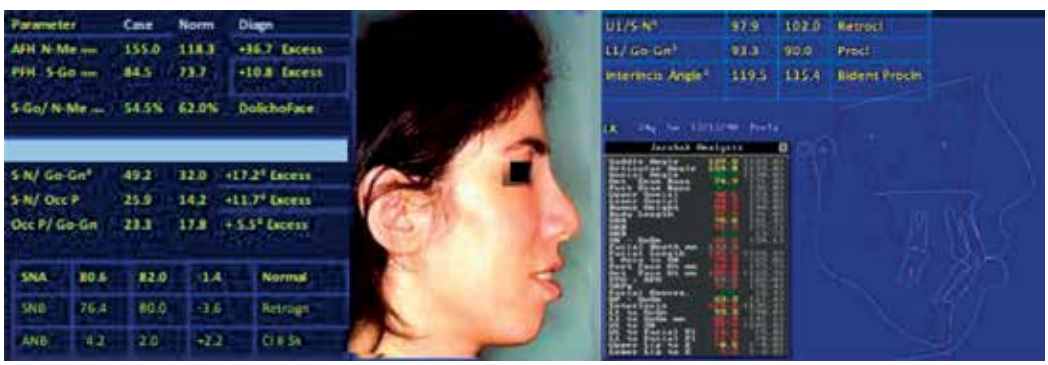

Figure 13. Pretreatment cephalometric analysis of Jarabak (sagittal, vertical, and dentoalveolar findings).

2.2.3. Treatment progress and results

Presurgical orthodontic phase intended to restore ideal arch form and dentoalveolar alignment (teeth decrowding, root parallelism, and occlusal plane leveling). This will facilitate the surgical mobilization of the arches in 3Ds; so that the appropriate canting of the occlusal plane and proper lip-teeth relationship secures satisfying treatment outcome (**Figures 15** and **16**).

2.2.4. Conclusions

When malocclusion is a consequence of facial musculoskeletal imbalance, any attempt to restore facial esthetics and beautiful smile by orthodontic dentofacial orthopedics alone will

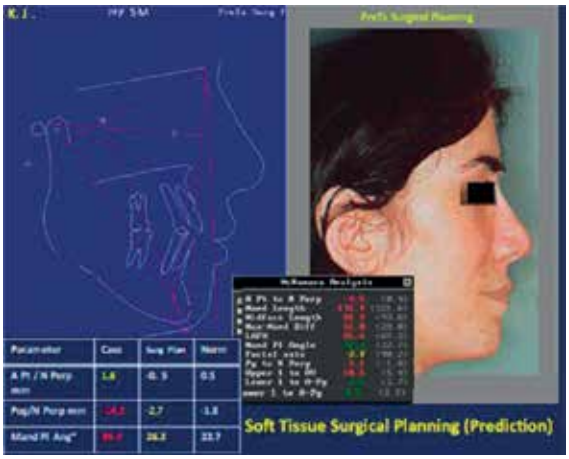

Figure 14. Soft tissue and dentoalveolar surgical planning of the case, surgical mobilization of the maxilla, and chin to restore harmony and balance of the facial profile, in addition to dentoalveolar sagittal and vertical orthodontic movements.

Figure 15. Phases of treatment (facial esthetics, pleasant smile, and ideal occlusion).

relationship in 3Ds. Surgical hooks were then constructed to start up the surgical phase surgical mobilization of the arches and bring occlusion into normal treatment outcome, and **Figure 5** shows the end up with straightening of teeth within orofacial disharmony and unaesthetic smile. Formulating treatment planning in such cases needs mutual collaboration between different health care providers and in particular between orthodontist and maxillofacial surgeon from the very beginning if the treatment objectives and patient's satisfaction are to be realized as in the case presented.

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Figure 16. Radiographic representation of LeFort I osteotomy with maxillary superior repositioning and genioplasty for chin augmentation achieved treatment objectives and patient's satisfaction. Phases of treatment (facial esthetics, pleasant smile, and ideal occlusion).

2.3. Case #3

A healthy female patient (23 year old) presented with a chief complaint "I am frustrated with my elongated face, unaesthetic smile, and jam-packed teeth that prevent me from biting objects or chew food efficiently." She was reluctant to wear braces before, but is ready to go through orthodontic treatment to re-establish a pleasant dentofacial appearance.

Diagnosis clinical examination revealed typical long face features with increased lower facial height and almost straight profile. Vertical maxillary excess broke the balance and harmony of the facial parts that caused a clear gummy smile and tipped occlusal plane (**Figure 17**).

Intraorally, the sagittal relationship showed a class III malocclusion with negative overjet and a complex anterior open bite vertically, where second molars are in occlusion (pivoting) and crowded maxillary arch; the transverse relationship exhibited bilateral buccal crossbite, high palatal vault, and narrow maxilla contained in the normal mandibular arch form. There were extraction spaces in the lower arch as appears in the panoramic radiograph **Figure 1**.

2.3.1. Cephalometric assessment of vertical and sagittal features

Jarabak and McNamara lateral cephalometric analyses demonstrated the severe skeletal origin of the dentofacial deformity (**Figure 2**) that cannot be treated by orthodontic approach alone. The vertical maxillary excess caused severe gingival display during full smile and backward rotation of the mandible that surpassed any physiologic eruptive compensation of anterior teeth to develop anterior open bite (**Figure 18**).

Figure 17. Extra-oral and intra-oral photos showing the dentofacial deformity to be objectively assessed by other diagnostic aids (radiographs).

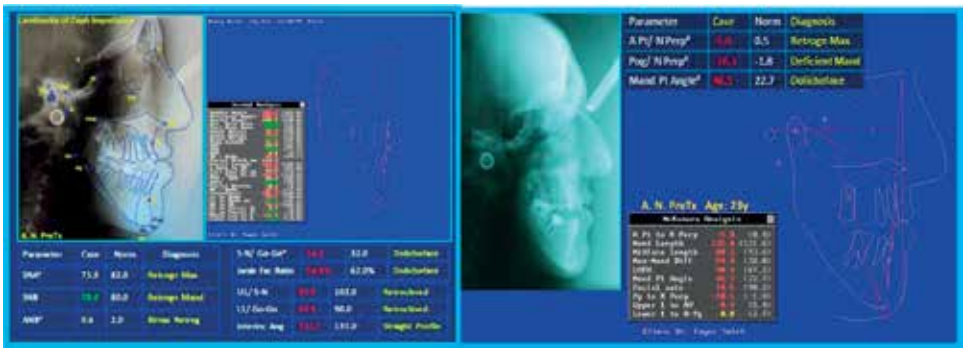

Figure 18. Lateral cephalometric analysis of Jarabak revealing vertical facial height excess, canted occ. plane, straight profile, deficient chin, and high gonial angle.

2.3.2. Problem listing

Data collection and analyses (**Figure 3**) endorsed the orthosurgical approach of treatment after joint patient's consultation with the health care team and her enthusiastic approval (**Figure 19**).

2.3.3. Treatment objectives

Realistic treatment objectives (**Figure 20**) were strictly reviewed to formulate the appropriate treatment planning, appliance design, and different presurgical orthodontics, surgical, and postsurgical phases of treatment (**Figure 21**).

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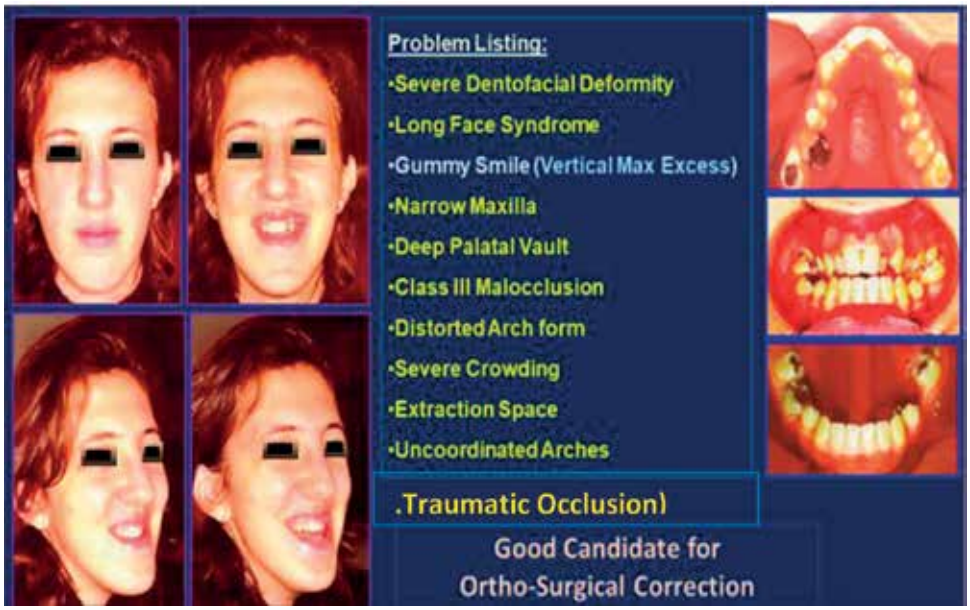

Figure 19. Listing the complex discrepancies of skeletal, dentoalveolar, and soft tissue origin is to justify the orthosurgical approach of interdisciplinary treatment for the benefit of the patient.

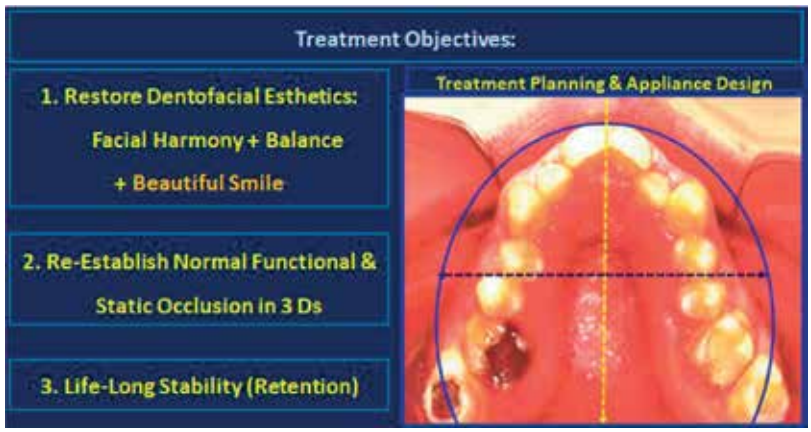

Figure 20. Treatment objectives allowed clinicians to formulate proper treatment planning.

2.3.4. Treatment progress

Maxillary arch expansion with quad helix appliance initiated simultaneously with leveling and alignment of teeth. Almost ideal arch form was attained, and the occlusal planes were flattened. When the presurgical phase was accomplished, surgical hooks were constructed as shown in **Figure 22**. LeFort I maxillary osteotomy with superior maxillary repositioning then took place to be followed by postsurgical detailing of occlusion and prosthetic replacement of the missing teeth.

Figure 21. Model surgery and diagnostic set up possible for prosthetic construction.

2.3.5. Results and conclusions

Despite controversy, combined orthosurgical treatment of severe maxillary excess concomitant with facial skeletal disharmony remains the only treatment option. The treatment results as seen in **Figures 22–24** were remarkably promising and satisfactory.

2.4. Case #4

A healthy patient (24 year and 10 month old) presented with a chief complaint "I am unhappy with my unaesthetic smile, lack of biting on my teeth, and prominent chin." The necessary data were collected and analyzed, and the patient was informed that orthodontic treatment

Figure 22. Pre and post treatment cephalometric analysis and panoramic views.

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Figure 23. Phases of treatment, pretreatment, presurgical, postsurgical, and post prosthetic replacement of extraction spaces.

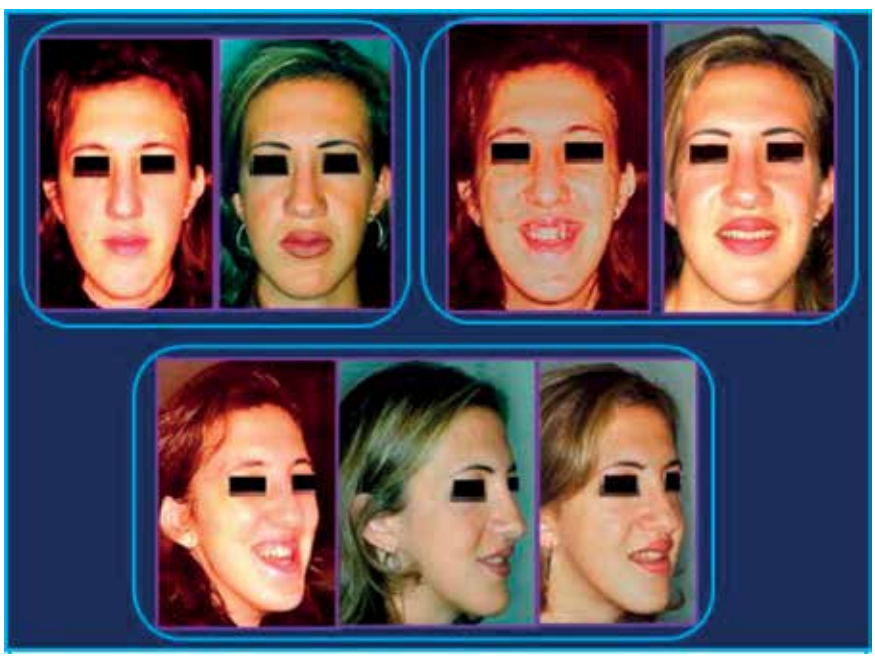

Figure 24. Frontal and lateral photos of the face showing significant esthetic improvement and pleasing smile shaped by combined orthosurgical treatment.

alone cannot be of benefit to you. A combined orthosurgical treatment modality was offered, and a joint meeting with the maxillofacial surgeon was arranged; details of the treatment phases, surgical risk, and cost effectiveness were explained to the patient who showed great interest in undergoing the treatment in a positive manner. The following figures reveal the steps and efforts exerted by the healthcare team and the cooperation of the patient that made the achievement possible.

2.4.1. Diagnosis

The diagnostic significance of clinically examining the dentofacial deformities lies in observing the soft and hard tissues functioning, so any abnormality is detected, and further investigations are assigned. Esthetic smile is a function of oral and perioral muscles and their relationship to the teeth and gingiva. Clinical examination of the patient revealed the severity of the skeletal, dentoalveolar, and soft tissues components as listed in **Figure 25**. However, objective judgment of the deformity necessitated other diagnostic aids including radiographs and study models to measure the discrepancy and formulate appropriate.

2.4.2. Treatment objectives

To restore normal facial harmony and balance, correct the severe malocclusion, design a pleasant smile, and insure life-long postoperative stability.

2.4.3. Treatment planning

Because of the complexity and severity of dentofacial deformity, the healthcare team decided to follow the logic sequence of orthosurgical treatment phases, presurgical orthodontics to be followed by bimaxillary surgical phase. LeFort I osteotomy with superior repositioning of the maxilla intended to reduce the increased facial height and level the tipped back occlusal

Figure 25. Pretreatment extra and intraoral photos describing the severity of the dentofacial deformity and the problems to be solved.

plane; bilateral sagittal split osteotomy of the mandibular ramus to setback the prognathic mandible bringing narrower mandibular arch in favor of the normal transverse occlusion, anticlockwise rotation aided in correcting the open bite. Retentive means were designed to guarantee stable outcome (**Figures 26–32**).

2.4.4. Conclusions

Mutual collaboration between healthcare providers allows for the proper selection of treatment modality relevant to the dentofacial deformity presented. Bimaxillary surgery helped in

Figure 26. Pretreatment lateral cephalometric and panoramic radiographs (essential diagnostic aids).

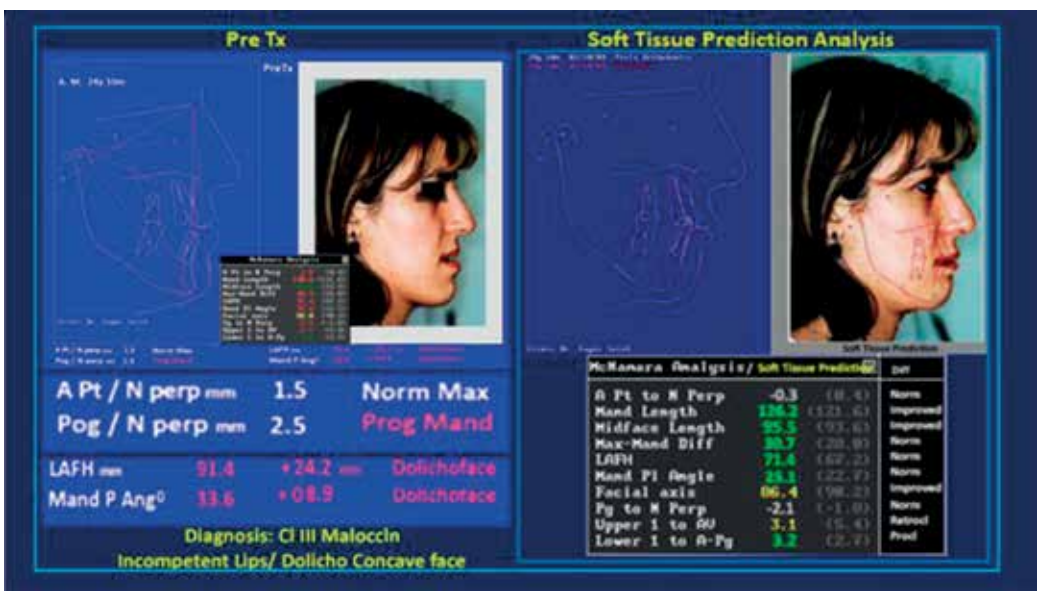

Figure 27. Pretreatment lateral ceph (McNamara) analysis and soft tissue surgical planning (prediction).

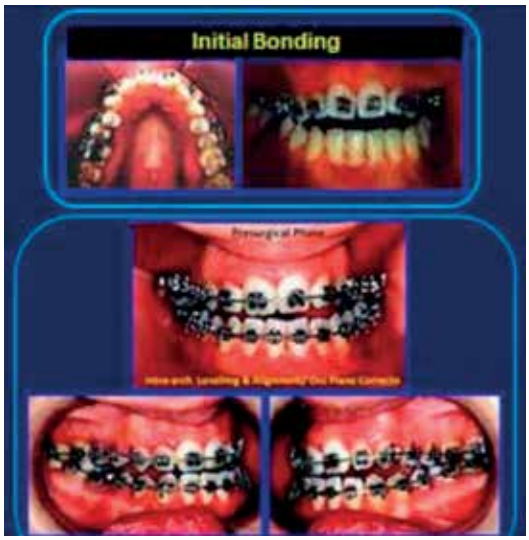

Figure 28. Initial bonding followed by inserting leveling arch wires to prepare the case for surgery.

Figure 29. Postsurgical occlusion and the role of inter-maxillary elastics in stabilizing the occlusion and then setup retentive means.

Figure 30. Pre and post cephalometric and panoramic radiographs.

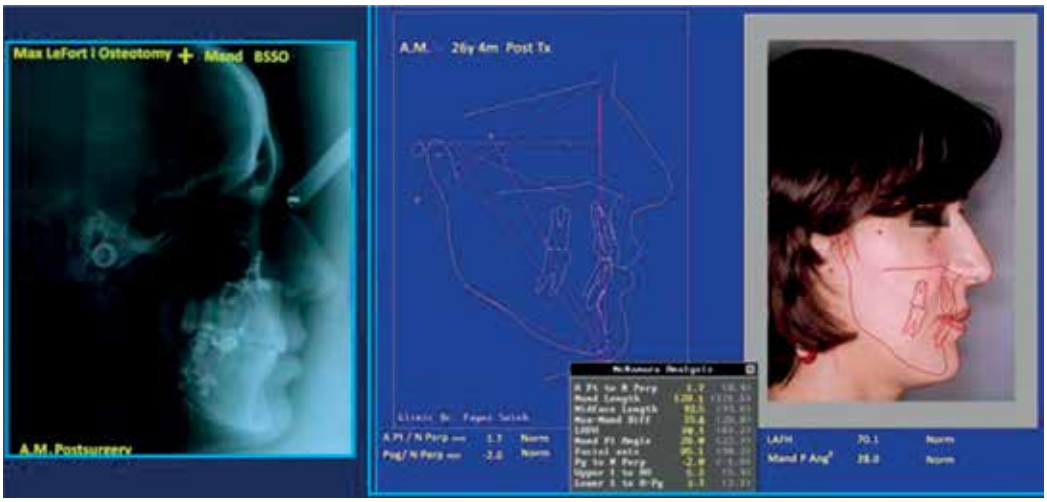

Figure 31. Post treatment cephalometric analysis reflecting the restructural improvement of the profile.

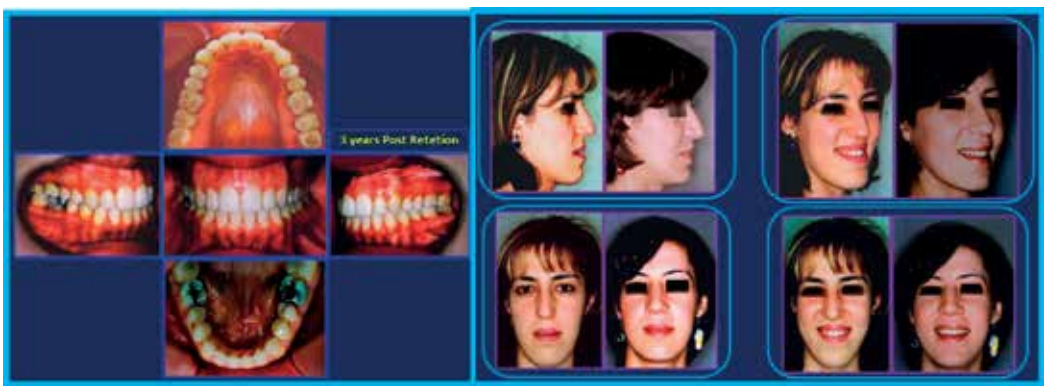

Figure 32. Occlusion 3-year post retention. Pre and post treatment facial features.

restoring harmony and balance of the face and establishing an ideal occlusal relationship. Patient cooperation and her family support were a key factor in achieving the satisfactory treatment outcome. Follow up for 5 year post treatment confirmed the successful procedures performed.

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this chapter.

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Chapter 10

Surgery First Approach

Ayşe Gülşen

Additional information is available at the end of the chapter

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Abstract

The surgery first approach (SFA) was presented by some researchers in earlier years, but SFA in a combined treatment, with the surgery first and the orthodontic treatment second, as introduced by Brachvogel et al. and by Nagasaka et al., has gained attention in the past 10 years. The advantages of SFA were reported in the literature, and the research into this method continues. One of the advantages of the SFA is the shorter total treatment time, and another is that patients begin treatment with a much improved face esthetically. The protocol of presurgical orthodontics is well known in dentofacial anomalies, but in SFA, especially in complex cases, the meticulous treatment is very important. In this chapter, SFA will be discussed.

Keywords: surgery first, orthognathic surgery, orthodontics

1. Introduction

The treatment of skeletal discrepancies requires orthognathic surgery in combination with orthodontic treatment to improve malocclusion, function, facial, and smile esthetics.

In the 1960s, the surgeons performed orthognathic surgery without orthodontic treatment [1–3]. But it was clearly understood that mandibular or maxillary movement was limited without tooth movement. For example, amount of mandibular setback was limited by the overjet in Class III cases. To achieve a proper setback and to have a good the occlusal and facial esthetics results, orthodontic alignment of malaligned teeth and solving the compensation of teeth to the malposed jaws are required before surgery [4–6]. After the 1970s, orthognathic surgery in combination with orthodontic treatment began to have good standards and showed popularity [7–11].

In conventional orthognathic surgery approach, the surgery follows the orthodontic treatment (orthodontic-first approach). Teeth are tended to compensate for skeletal discrepancies to have functional occlusion. The presurgical orthodontic treatment is needed to solve the dental decompensation that reveals the true extent of the skeletal deformity to align the teeth and to fit the maxilla and mandible into a good occlusion after surgery [11, 12]. Following the orthodontic treatment, orthognathic surgery corrects the skeletal discrepancy to obtain a good jaw alignment with good facial proportions. As the direction of presurgical orthodontic treatment is opposite to that of natural dental compensation forces, the orthodontic treatment time is said to require time to overcome the natural compensation forces [13]. The presurgical orthodontic treatment period which includes aligning dental occlusion, reversing incisor decompensation, correcting tooth rotation, and arch coordination lasts for 12–36 months depending on the complexity of case and also for a period after the surgery [13].

In last 10 years, surgery first approach (SFA) has begun to be implemented in some centers [14] and created broader interest [15–20].

The surgery first approach (SFA) is the orthognathic surgery approach that the orthognathic surgery precedes the orthodontic treatment. In the beginning of the treatment, surgery is performed without orthodontic preparation, and the orthodontic treatment is done after the surgery.

Historically, the SFA was presented by some researchers in earlier years [2, 21–24], but SFA in a combined treatment, which was introduced officially by Brachvogel et al. [25] and by Nagasaka et al., has gained attention in the past 10 years [26].

Among the published studies about SFA regarding the type of malocclusion, Class III is the most prevalent. Class III with openbite and asymmetry cases with SFA are the other published studies. SFA in Class II cases and in some deformities like TMJ disorders or condylar hyperplasia is rare [27–29].

2. The advantages of SFA

The advantages of SFA reported in literature continue. One of the advantages of the SFA is the shorter total treatment time [13, 15, 30]. Other advantages are that patients begin treatment with a much improved face esthetically in the beginning of the treatment and that the patient's chief complaint, dental function, and facial esthetics are achieved and improved in the beginning of the treatment [31, 32] and a psychosocial benefit of improved body image in the beginning of the treatment instead of worsening the facial appearance because of the presurgical decompensation of incisors [31, 34]. Improved corporation of the patient during the treatment may be the other advantage of SFA due to rapid profile improvement [33, 35]. SFA is also preferred in early correction of obstructive sleep apnea patients. On the other side, due to the early correction of skeletal and soft tissue problems, orthodontic treatment may be easier due to normalized surrounding soft tissue [23]. It was reported that the patients with preexisting TMJ dysfunction might experience a significant improvement of TMD signs and symptoms after SFA [29].

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One of the reasons for the shorter duration of treatment in SFA is the regional accelerated phenomenon (RAP) which is the increase of the osteoclastic and metabolic activities due to the surgery. Selective bone injury activates stimulus for anabolic and catabolic responses in the periodontium adjacent to the osteotomies performed during orthognathic surgery and increases bone reorganization [32, 36–45]. It was reported that RAP in humans began in a few days after surgery and peaked at 1–2 months and took 6 months to more than 24 months to subside [39]. Liou et al. also studied the causes of rapid postoperative orthodontic treatment time in SFA cases, and they found that the levels of serum alkaline phosphatase and C-terminal telopeptide of type I collagen (ICTP) increased, which supported the postoperative accelerated orthodontic tooth movement caused the orthognathic surgery [15]. Zingler et al. found that crevicular fluids in SFA cases were higher levels of bone remodeling factors for fracture healing [32].

The other reason for the shorter duration of treatment in SFA than in the conventional approach may be improvement of function. Choi and Bradley reported that teeth tended

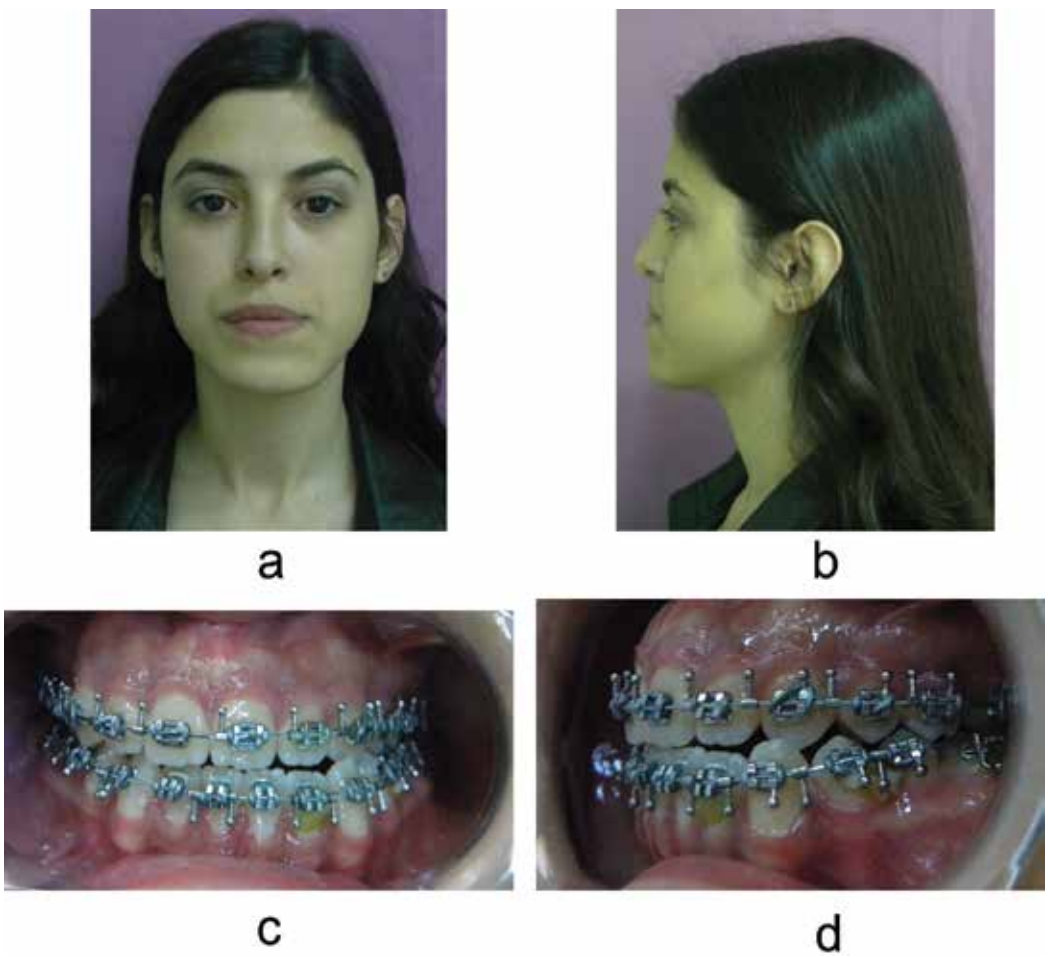

Figure 1. a-d: Facial asymmetry case. Passive arch wires were inserted the day before surgery.

to move in the direction of decompensation to perform the function following the surgery [46]. Postoperative orthodontic direction and function improve the efficiency of decompensation. Additionally, orthodontic movement via a more rapid natural dental adaptation by facilitating natural compensation may be performed easier with less occluded occlusion.

Orthodontic treatment time depends on the complexity of case. The shortest reported treatment time is 4 months (**Figure 1a-d**, **2a-d**) but generally it takes 6–12 months [17, 26, 28, 33, 35, 47–50]. Tooth extraction is the factor that influences the total treatment time [13], and in some cases, the time range was reported between 10 and 19 months [51–53].

Figure 2. a-d: Three months later after surgery. Total treatment time 3 and half months.

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3. Treatment plan in SFA

SFA is indicated more common in some cases like well-aligned to mildly crowded anterior teeth, flat to mildly curve of Spee, and normal to mildly proclined/retroclined incisor inclination. The protocol of presurgical orthodontics is well known in conventional approach [4, 8]; however, treatment plan including orthodontic treatment is questioned in SFA especially in complex cases. The orthodontic management and treatment plan are different in SFA compared with the conventional approaches.

In treatment plan, accurate and detailed prediction of the postoperative orthodontic treatment is required at the beginning of all treatment [50]. Following the analysis of occlusion with model mounting, of detailed clinical and cephalometrics, presurgical orthodontic setup that is useful for accurate prediction and simulation of postsurgical orthodontics and cephalometric setup may be required before the surgery [13, 51].

The model surgery is a setup according to the cephalometric and molar relationship. Three stable occlusion points between the upper and lower dentitions are required [38]. Liou et al. reported that the molar relationship could be set up in Class I in cases of nonextraction or bimaxillary first premolar extraction, Class III in cases of lower first premolar extraction, and Class II in cases of maxillary first premolar extraction [16].

Following cephalometric, model, and clinical diagnosis, the aim is to optimize the position of facial components to attain the most desirable results in esthetics, function, and stability. The skeletal movements in all anteroposterior, vertical, and transverse directions are determined to obtain good facial proportions, smile esthetics, and occlusion.

Liou et al. have made some suggestions in treatment plan of SFA [16]. In Class III cases, to correct the decompensation of maxillary incisor, first premolar extraction and retraction of anterior teeth can be done by orthodontics or by anterior segmental osteotomy. If the case has moderately retroclined and crowded lower incisors, the molars in a Class I relationship with an excessive incisor overjet can be planned. In cases with severe crowding and retroclination in mandible, first premolar extraction and lower anterior setback osteotomy can be planned. In Class III cases with deep curve of Spee, leveling of Spee can be corrected before the surgery or can be corrected with lower anterior segmental osteotomy surgically to avoid upward-forward rotation of mandible postoperatively, which is not preferred in Class III cases. The chin cap therapy may be used to prevent the skeletal postsurgical relapse after surgery for 3 months [16]. In Class II cases, in mandibular retrognathia with deep curve of Spee, mandibular advancement with surgical intrusion of anterior segment to advance mandible properly or mandibular advancement followed by orthodontically intrusion of lower incisor postsurgically is proposed to obtain a better chin profile. Otherwise, the mandible cannot be advanced properly and lower face can be longer with correction of posterior openbite after surgery, and this cannot be preferred in some long face case. But in some cases where advancements are not required much, correction of posterior openbite only with posterior extrusion can be preferred [54].

4. Time for orthodontic bonding and force application

On the basis of simulated model surgery setup, surgical guidance splint is prepared. Before the surgery, orthodontic bracket bonding/banding is placed but no arch wire is used. Bonding orthodontic bracket was reported as immediately before surgery [26, 47, 48], 1 week before surgery [16, 26, 38], and 1–2 months before surgery [50]. Some studies reported the usage of passive archwire before the surgery [49, 50, 52, 53, 55]. Passive arch can be used 1–3 days before the surgery [17, 35]. In some cases, the orthodontist can prefer minimal orthodontic preparation during 6 months [49] before the surgery, and then, they are continuing the orthodontic treatment after the surgery. Intermaxillary fixation of jaws during the surgery can be done by bony screws following the surgical guidance splints placements in cases without arch wires [47, 50, 51]. Kim et al. maintained intermaxillary fixation without surgical splint for 2 weeks but used intermaxillary elastic [50]. The osteotomized bones are fixed by rigid fixation.

Postoperatively, surgical splint is left for 2–4 weeks [34, 50, 53], and intermaxillary elastics usage may begin after orthodontic wire was placed.

There is no definitive consensus about postsurgical orthodontic force application time. But generally, the orthodontic treatment in SFA begins in 1 or 2 weeks after surgery. The surgical splint and inter-maxillary fixation were removed for the tooth movement. Liao et al. reported that postsurgical orthodontics begun immediately after surgery [17]. This is beneficial to shorten the orthodontic treatment time due to the regional accelerated phenomena. The studies showed that the orthognathic surgery triggers a 3- to 4-month period of higher osteoclastic activity, serum findings, and metabolic changes and that in the dentoalveolar bone postoperatively [15, 56]. Archwire changes took place every 2–3 weeks. Arch coordination may be managed with transpalatal elastics or active transpalatal arch. In segmental surgery patients, passive continuous arches which were placed before surgery are changed with sectional arches at first orthodontic appointment after surgery.

5. Relapse in SFA

The short- and long-term relapse rates in SFA have been investigated, and the results are good by comparison with the conventional surgical approach with a maximum follow-up of 3 years [19, 57–59]. Without presurgical orthodontics, the patients may have likely to develop unstable occlusion after surgery leading to relapse. However, some of the comparative studies between conventional and surgery first approach showed no statistical differences in relapse and almost equal for those achieved using the more traditional orthodontics-first approach [17, 19, 51, 53, 59–63]. Advancement of fixation system enabled more stabilized results due to more stable fixation of bony segments. On the other side, based on one research and on the meta-analysis, SFA showed more relapses than in the conventional approach [57, 64]. Larger overbite, a deeper curve of Spee, a greater negative overjet, and a greater mandibular setback were reported to affect stability in SFA cases [59].

Although there are benefits of the SFA, there are some difficulties like the prediction of final occlusion, instability of postsurgical transient occlusion [10, 65], the requirement of presurgical orthodontic setup before surgery in some complex cases, and requirement of frequent orthodontic appointment due to RAP. The treatment plan requires detailed and meticulous planning.

6. Conclusion

The surgery first approach is an alternative method that may be more satisfying for orthodontists and patients by minimizing the treatment time required for orthodontic treatment compared to conventional approach.

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Chapter 11

Orthodontics in Relation with Alveolar Bone Grafting in CLP Patients

Aslıhan Uzel

Additional information is available at the end of the chapter

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Abstract

Alveolar bone grafting is an essential step in the overall management of patients with cleft lip and palate (CLP). The numerous advantages of this procedure have been reported in the literature. Failure to rehabilitate the alveolar cleft may give rise to a variety of problems. Lack of investing alveolar bone often precludes the correction of anterior tooth irregularities and limits orthodontic treatment and/or prosthodontic rehabilitation. The success of the graft is multifactorial. The periodontal health of the surrounding graft tissues, the experience and ability of the surgeon, the timing of surgery, and orthodontic management of the cleft area before and after grafting are shown to be the most important factors in this issue. In this chapter, current orthodontic approaches in relation with alveolar bone grafting (ABG) in cleft patients will be discussed.

Keywords: cleft palate, alveolar bone grafting, orthodontics

1. Introduction

Cleft lip and palate (CLP) are among the most common of all congenital facial deformities which affect approximately 1 in every 600 newborn babies worldwide [1]. Congenital CL/P can arise in isolation or together with other syndromes. Alveolar cleft (osseous defect in the alveolus of upper jaw) affects approximately 75% of cleft lip and palate patients [2, 3].

The rehabilitation of individuals with CL/P requires interdisciplinary care by centralization of treatment [1, 4].

As the facial cleft affects the whole stomatognathic system, orthodontics is a core element of the overall treatment process. The orthodontist should aim to provide a dentition that

functions well and is capable of lifetime maintenance by routine oral hygiene and dental care. However, the underlying skeletal deformity that reflects intrinsic variation and the consequences of surgery severely restricts occlusal change [5].

There are two major factors which effect orthodontic treatment in patients with CLP:

1. Facial growth disruption

It is well known that facial growth in patient with CP is disturbed. Besides the intrinsic defect, surgery itself contributes to further disruption [3, 6]. A significant feature of facial growth in repaired CP patients is that the maxilla fails to grow at the same rate as the mandible during the adolescent growth spurt. Progressive midfacial retrusion is usually seen by the mid- to late teens. The results of the facial growth studies revealed the general characteristics of the individuals with UCLP: a short retrusive maxilla and vertical elongation of the anterior face, a retrusive mandible, and a reduction in posterior face height [6–9].

2. Alveolar bone deficiency

Lack of the alveolar bone may give rise to a variety of problems, including oronasal fistula, fluid reflux, speech pathology, impaired tooth eruption, lack of bone support for the anterior teeth, dental crowding, periodontal recession and eventual loss of teeth, and maxillary and facial asymmetry. Alveolar defect also limits orthodontic treatment and/or prosthodontic rehabilitation [10–12].

Thus, orthodontic treatment for children with cleft should aim to achieve an optimal occlusion and dentofacial esthetics within the constraints imposed by the underlying skeletal pattern [5].

The integration of orthodontics into the overall treatment of CLP starts any point between birth and end of the teens and highly related to surgical procedures, including lip repair, alveolar bone grafting, distraction, and orthognathic surgery [5].

This chapter reviews current orthodontic approaches in relation with alveolar bone grafting (ABG) in nonsyndromic cleft patients.

2. Alveolar bone grafting

Alveolar bony defect is the main limiting factor for orthodontic treatment. Elimination of the bony defect is provided by alveolar bone grafting (ABG). Since the introduction of secondary alveolar bone grafting (SABG) in 1972, this technique has become an essential step in the overall management of patients with cleft lip and palate (CLP). Providing bone tissue for cleft has following benefits: [10, 12–21]

1. To permit eruption of the permanent canine in the cleft site into sound bone.

2. To provide bony support for teeth on either side of the cleft site.

3. To permit orthodontic tooth movement.

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4. To obviate or minimize the need for prosthetic replacement of teeth in the cleft site.
5. To permit placement of osseointegrated implants into the cleft area when indicated.
6. To stabilize maxillary dental arch.
7. To facilitate fistula closure.
8. To improve the contour of the alar base.

The long-term success of the alveolar graft is crucial for providing and lifetime maintaining optimal occlusion and dentofacial esthetics in patients with CLP.

Postgraft stimulation of maturation through remodeling of the graft is extremely important and is provided primarily by natural tooth eruption. Thus, it is generally agreed that the optimum timing for ABG is in the mixed dentition stage (8–11 years), just before the eruption of the permanent canine in line with the cleft side [10, 13, 16, 19, 22–29]. There is no precise recommended chronological age but, when one-half to two-thirds of the canine root is formed [10]. Canines are mostly the reference teeth because lateral incisors in patient with CLP are frequently absent. However, if lateral incisor is present, earlier bone grafting can be indicated at an age around 7–8 years. It has been found that the success rate is significantly reduced when ABG is performed after eruption of the canine. Resorption of the bone graft is a common situation, and the success of ABG depends on several factors. The periodontal health of the surrounding graft tissues, the experience and ability of the surgeon, and graft material are also shown to be the general factors determining success [13, 19, 20, 23, 30, 31].

The importance of the orthodontist in planning, preparations, and follow-up around ABG procedure is also widely recognized. Successful alveolar bone grafting necessitates a joint orthodontic and surgical involvement pre-, peri-, and postoperatively [10, 32, 33].

Before treatment, orthodontists should be able to explain the predicted outcome of bone grafting to patients and their parents [34].

At that point, diagnostic information is very important for planning pre- and post-orthodontic management.

3. Advantages of CBCT in diagnosing and treatment planning of patients with CLP

A bony bridge with sufficient height and width is important for successful bone grafting in the alveolar cleft and to guide eruption and movement of permanent teeth [15, 35–37]. The outcome of the procedure is considered satisfactory when a sufficient volume of remodeled bone tissue is obtained; otherwise, the surgery has to be repeated. Thus, volumetric measurements of CBCT images have been using to evaluate the success of alveolar graft outcomes in the current literature [38]. CBCT, an alternative approach to conventional CT that provides similar diagnostic information with much less radiation exposure, avoids the problems associated with traditional 2D imaging such as image enlargement and distortion, structure

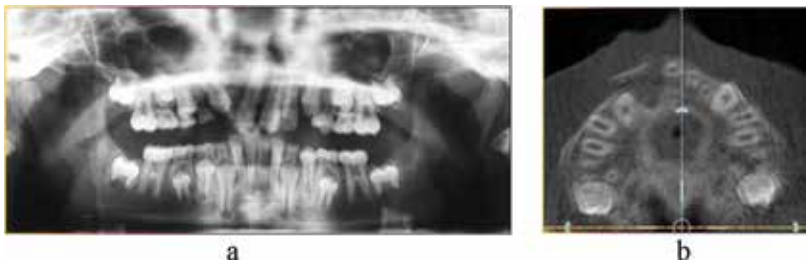

Figure 1. Superimpositions on the 2D imaging might led to misdiagnosis. (a) Grafted area seems to be filled successfully on the pantomograph; (b) axial view of the CBCT image shows that graft is not successful as seen on the panoramic radiograph.

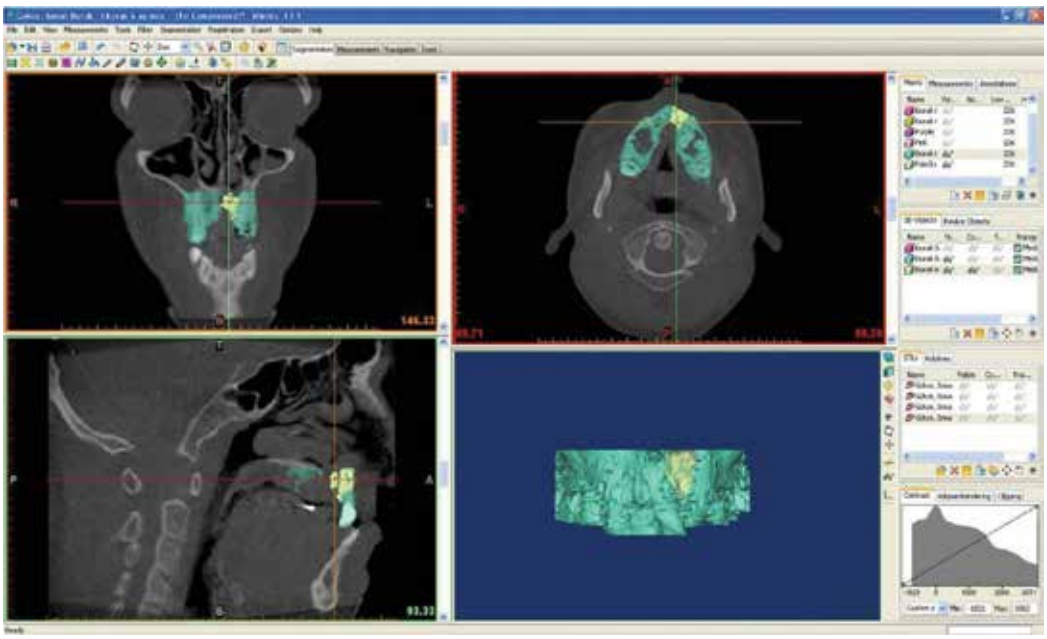

Figure 2. The precise volume and density can be measured by using various software.

overlap, positional problems, and limited number of identifiable landmarks (**Figure 1**). It has been used to quantify the average volume of the graft, location of the bone loss, and periodontal bone support of the cleft-adjacent teeth. CBCT-derived volumetric assessment of alveolar grafts has been reported as a reliable method [24, 29, 37, 39, 40] (**Figure 2**). The success rate of an alveolar graft has been found to be significantly lower with volumetric evaluation than that with conventional radiographic imaging [39, 41, 42].

4. Mixed dentition stage (phase I orthodontic treatment)

Orthodontic treatment of patients with CLP takes much more time (is more extensive) than routine treatment because of the underlying skeletal pattern. There is no need to attempt

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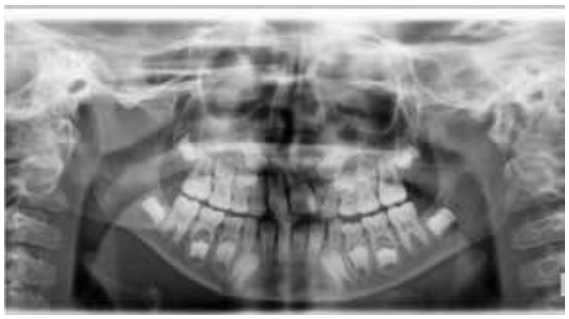

Figure 3. Malpositioned lateral incisor in the cleft area should be extracted 3–4 weeks before ABG.

orthodontic treatment in deciduous dentition stage. This will need much more retention procedures and will impose unnecessary burden of care [5].

Monitoring eruption of the teeth is important by the age 6–7.Radiographic evaluation is needed at the age of 8–9 (after eruption of upper incisors) to detect any possible teeth positioned in the cleft area and to see if lateral incisor is missing or not. If lateral incisor is present, earlier bone grafting can be planned. It is often advisable that any supplemental, deciduous teeth and also malformed and/or malpositioned lateral incisors in the cleft area should be extracted 3–4 weeks before surgery that permits healing of the mucosa (**Figure 3**). Thus, CBCT 6–8 months before ABG is recommended for detailed evaluation of bony support and position of the cleft-related teeth. This time frame will provide enough time to accomplish all necessary pregraft preparations such as tooth extraction and/or orthodontic tooth movement on time and not to delay bone grafting. Sometimes an additional CBCT just before the grafting might be needed to assess the root position of the cleft-related teeth after orthodonticmovement.

4.1. Pre-graft orthodontics

Presurgical orthodontics plays an important role in correcting misaligned incisors or repositioning displaced maxillary alveolar segments. Severe central or canine inclination toward the cleft defect can also interfere with cleft mucoperiosteal dissection. Presurgical orthodontics allows the surgeon better access for placement of the graft and closure of the soft tissue (**Figure 4**). Furthermore, correction of central incisor rotation and inclination prior to SABG enables patients to achieve better oral hygiene and prevents plaque formation. This can therefore prevent chronic, low-grade inflammation activating proteases that degrade grafted bone [5, 32, 33, 38].

One of the presenting problems which occurs early in both unilateral and bilateral clefts is the anteroposterior malpositioning of the incisors. If the anteroposterior malpositioning of the incisors is not corrected, lingual lock of the anterior teeth will further inhibit the development of the maxilla. The proper overjet relationship will allow appropriate growth of the maxilla [19]. By age 7–8, incisor alignment and correction of anterior crossbite can be provided to maximize the forward development of the maxillary dentoalveolar process. However, orthodontic movement of maxillary anterior teeth must be done with great caution because of the closeness of the roots to the bony defect. A very thin bony covering of the central incisor next to the cleft site is a common feature. Often there is just a lamina dura with no cancellous bone.

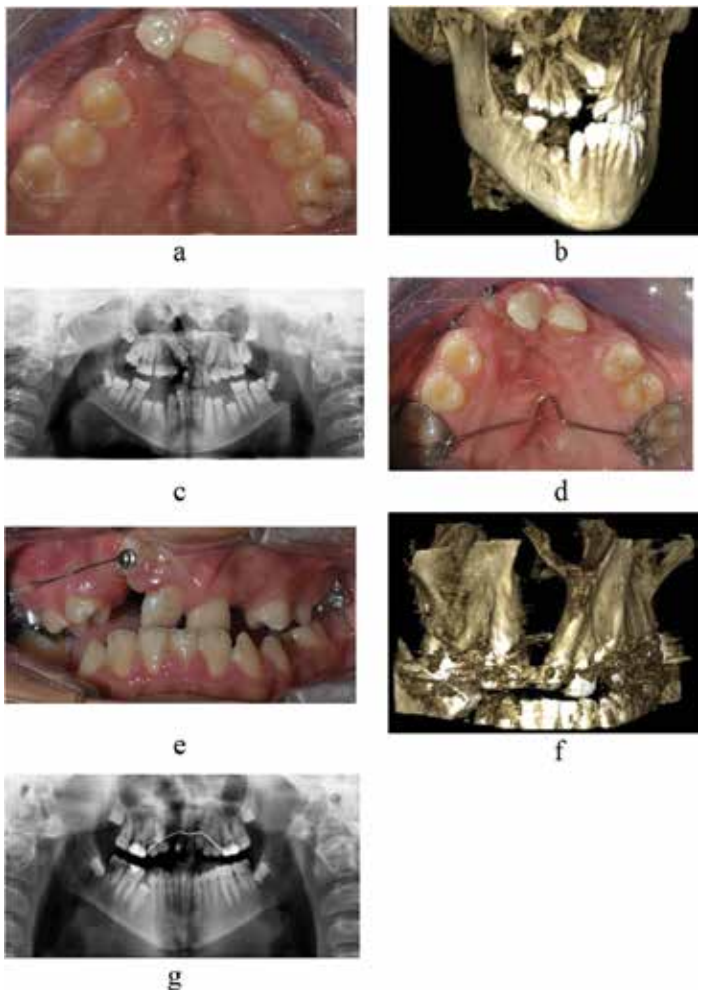

Figure 4. Severe canine inclination toward the cleft defect should be corrected before ABG. (a,b,c) initial views, a. intraoral, b. 3D image c. panoramik radiograph of the patient. (d,e) orthodontic traction of the cleft related canine. (f,g) pregraft 3D image and panoramic radiograph.

The incisor should not be bodily uprighted before successful ABG because of the possibility of bone loss and fenestration of the thin cortical lamina [10, 43].

4.2. Transverse expansion

Constriction of maxillary segments is a very common situation in patients with cleft palate. As the individuals with complete cleft lip and palate do not have midpalatal suture, constriction occurs mostly by the rotation of the lateral segment(s) inward, toward bony defect. Both the absence of the midpalatal bone and the soft tissue traction produced by lip and palate repair promote arch constriction [5, 44, 45].

Significant segmental displacement requires pre-bone graft expansion to rotate the lateral segment(s) outward, facilitate placement of the graft, and provide the surgeon working

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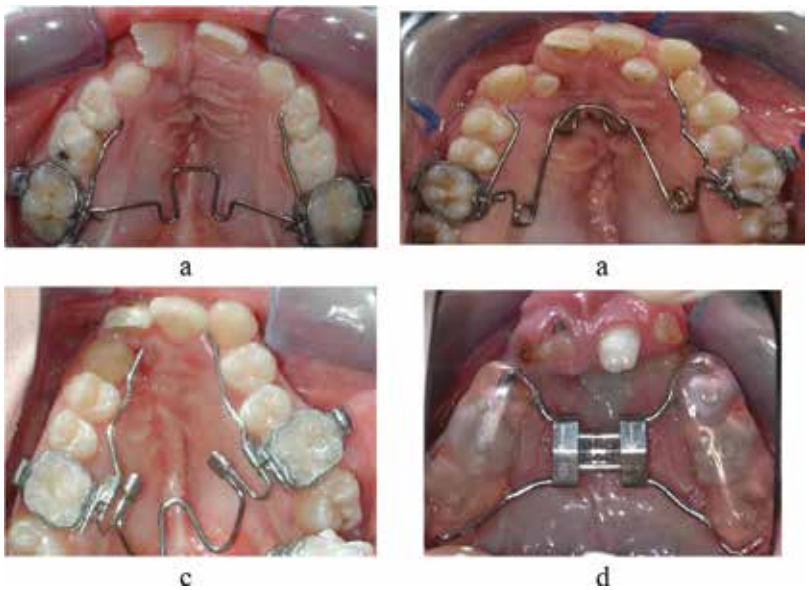

Figure 5. Various types of expanders for maxillary expansion. (a) TPA with lateral expansions, (b) quad helix, (c) NiTi expander, and (d) hyrax.

facility during surgery. If possible, transverse expansion can be combined with the correction of incisor irregularities. In the mixed dentition stage, arch expansion is very important because this process also normalizes the morphology and induces eruption of the canine into the symmetrical maxillary arch [10, 13, 19, 46].

Several types of expanders have been used, and there is no universal protocol for maxillary expansion prior to secondary alveolar bone grafting (**Figure 5**). Both slow maxillary expansion (SME) and rapid maxillary expansion (RME) have been advocated [5, 47, 48].

SME using the quad helix or its variations is frequently used for segmental repositioning as selective expansion anteriorly is required [5]. It has been shown that slow expansion forces are apparently already sufficient to allow a skeletal expansion of the maxilla in complete cleft palate patients [45, 47, 48]. There were *no differences* found between the dentoalveolar effects of SME and RME in patients with BCLP [45, 47].

RME with Haas type or hyrax expanders is also widely used for correcting the maxillary constriction. Asymmetric expansions were found by several authors [46, 48–53]. Isaacson and Murphy reported no correlation between the cleft location and the relative amount of lateral movement of each maxillary segment, emphasizing that RME laterally repositioned the maxillary segments in an unpredictable manner [49]. For greater amount of anterior displacement of the maxilla, fantype or double-hinged RPE expanders have been advocated [54].

According to our clinical experience, there is usually no need for RPE in patients with UCLP. Quad helix or TPA with lateral expansions can solve the problem. However, in some patients with BCLP, significant constriction of the segments necessitates RPE. Thus, patient selection is important in this issue.

5. Postgraft stabilization

The quad helix and/or stabilizing archwire used in BCLP may be removed during the bone grafting procedure for improved surgical access, but these appliances should be replaced before the patient leaves the operating room and left in place for 3 months.

As the bone grafting alone cannot be relied upon to maintain the expansion, a simple palatal arch would be advisable until the permanent dentition has erupted.

Stabilizing a mobile premaxilla with orthodontic arch wire is needed in patients with complete BCLP. Typically the arch wires will be removed during surgery and replaced at the end of the operation to provide retention [5, 10, 19].

6. Postgraft orthodontics

If the graft is done at proper time, before eruption of the cleft-related permanent canine, observation of the permanent dentition is generally all that is necessary. The status of cleft side unerupted teeth does need careful monitoring [5]. Physiologic eruption of the adjacent canine will provide enough stimulation for the alveolar graft. Sometimes orthodontic traction might be needed if the position of the canine is not appropriate for spontaneous eruption. High degrees of canine inclination indicate risk for altered eruption and impaction [5, 32].

If graft is done at age 7–8, correction of incisor irregularities provides also favorable stimulation to the graft.

Orthodontic movement of the cleft-adjacent teeth in the direction of the grafted bone can be instituted at an average of 3 months after the bone grafting, if needed. Combined interceptive bone grafting and orthodontic treatment at an early age avoid more extensive prolonged treatment later in the patient's life [5, 13, 19, 34].

It has been recommended not to delay orthodontic treatment more than 6 months after grafting, in cases in which an a-p crossbite or a residual transverse posterior crossbite exists. One- to two year delay in stimulation of the ABG of the premaxilla (by orthodontic treatment) can lead to serious postoperative problems. Where there is no stimulation of the graft, there tends to be "locking" or lingual collapse of the maxillary central incisors and collapse of the premaxillary arch [19].

6.1. Maxillary space management choice

In patients with CLP, the lateral incisor is missing in about 50% of cases in the permanent dentition. There are two options when maxillary lateral incisors are absent: space closure or space preservation [5].

The success of the bone graft is the determinant factor for this choice. When bone graft is properly done before the eruption of the permanent canine, canine can simultaneously migrate

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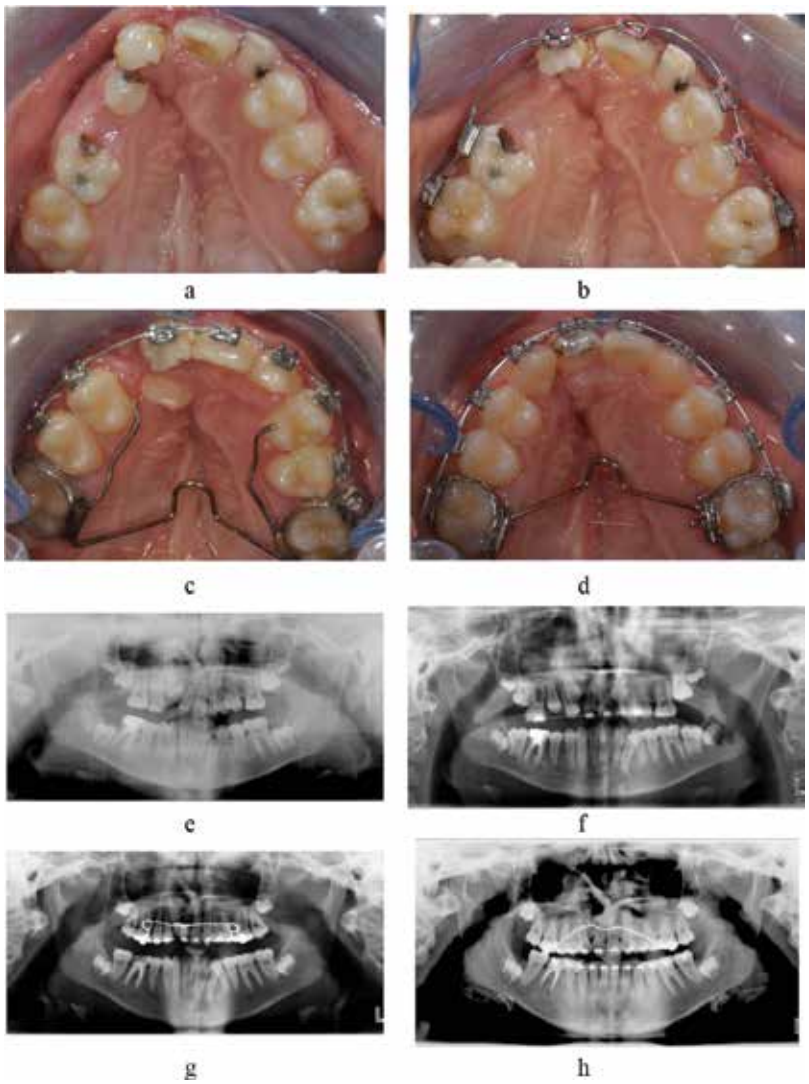

Figure 6. Space closure with canine substitution in patient with successful ABG. (a-d) intraoral occlusal views of the case. a. pre-graft, b. post-graft, c. after eruption of the canine, d. levelling of the upper dentition. (e-h) panoramic radiographs of the case. a. pre-graft, b. post-graft, c. eruption of the canine, h. levelling of the upper arch.

into the newly formed bone and increases its vertical height. Maintaining the alveolar bone height in the cleft area is important to prevent long-term complications, such as gingival retractions and periodontitis [32, 33].

Moreover, the natural dentition has the best prognosis for long-term health of the dentition. Thus, space closure with the canine substitution should be the first treatment choice for patients with CLP [5, 32, 33] (**Figure 6**). The functional stress imposed by orthodontic treatment influences the volume and prevents resorption of the grafted bone. Higher grafting success was found in the case of space closure than in the case of space openings [5, 33].

However, in patients with severely impaired maxillary growth, multiple missing teeth, and/or failure of bone graft, orthodontic space closure may not be feasible, and some form of prosthesis might be needed.

6.2. Extraction choice

Extraction of maxillary teeth may be required in UCLP in non-cleft quadrant, either because of crowding or to allow correction of the dental midline. As the second premolar is frequently malformed, it is the most commonly removed tooth. In some patients removal of the non-cleft lateral incisor allows the rapid restoration of the symmetry. However, this should be considered when compliance with space closure is assured [5].

In the lower arch, the absence of the second premolars is frequent and should be assessed carefully where extractions are necessary to relieve lower incisor crowding.

Extraction of the lower teeth to compensate class III skeletal pattern should be avoided in the early teens.

7. Management of maxillary deficiency in growing cleft patients

Hypoplastic maxilla and progressive midface retrusion are typical characteristics of patients with CLP.

Therefore, maxillary protraction (MP) has been frequently applied in the orthodontic treatment of growing patients with cleft lip and palate to improve the maxillomandibular relationship, occlusion, and facial esthetics. Optimal timing for initiating maxillary protraction for non-cleft children is shown as in the early mixed dentition before age 10. Early mixed dentition is favored over late, because of the closure of the sutures of the nasomaxillary complex [55].

However, SABG is optimally carried out between 9 and 11 years, and there is no consensus on the treatment sequencing of maxillary protraction and SABG in patients with CLP.

Two studies of three-dimensional finite element analysis suggested the advantage of SABG before maxillary protraction [55–57].

In a recent clinical study, short-term results showed that facemask therapy after alveolar bone grafting led to enhance maxillary skeletal advancement and minimize mandibular clockwise rotation more than those in the ungrafted group.

Maeda-lino found that the root lengths of U1 were comparatively short on the cleft side in patients with UCLP treated with MPA before SABG. Thus, they concluded that orthodontic force exerted by the MPA before SABG might result in short dental roots [58].

Moreover, it has been advocated that protraction of severely retruded cleft maxilla, even at an early stage, does not provide lasting skeletal benefit. Its effect in individual cases with CLP is difficult to predict, and many patients require orthognathic surgery after MP treatment [5, 59, 60] (**Figures 7** and **8**). Thus, explanation of the expected effects and associated problems should be given to the patients and parents before MP treatment.

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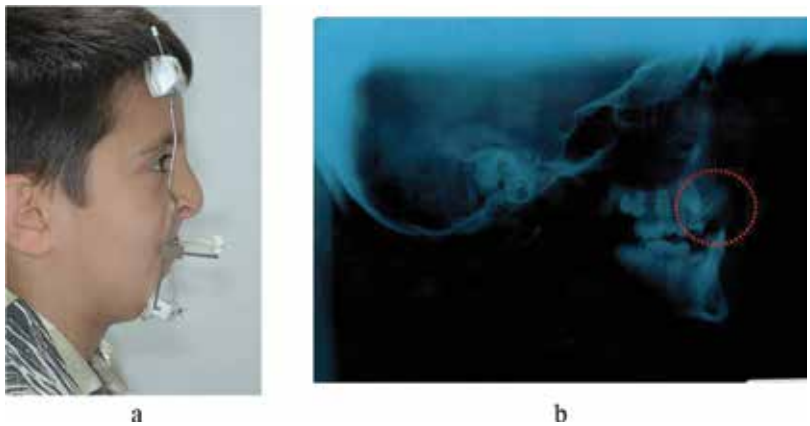

Figure 7. Face mask application using bonded hyrax in patient with BCLP. (a) extraoral view of the patient. (b) initial lateral cephalogram.

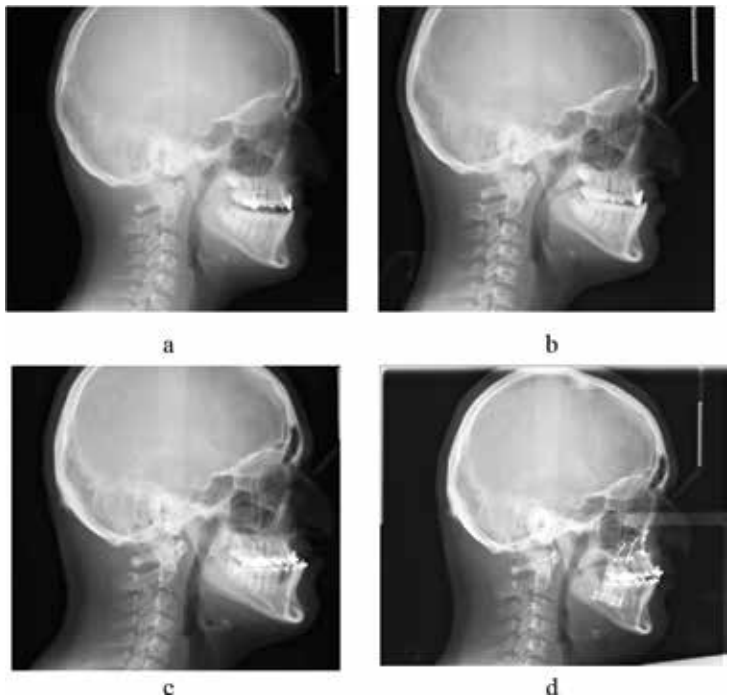

Figure 8. Cephalometric radiographs of the patient after facemask therapy between 10 and 19 years of age and after orthognathic surgery. (a) age 10, after face mask therapy, (b) age 14, follow-up, (c) age 19, ğre-orthogntic surgery, (d) after orthognatic surgery.

7.1. Patients with no to mild skeletal discrepancy

Providing proper overjet relationship at the early mixed dentition by correcting lingual lock of the anterior teeth using either removable or fixed appliances will be sufficient to maximize the forward development of the maxillary dentoalveolar process. Facemask can be a valuable source of anchorage for advancing posterior teeth during space closure after SABG [5, 19].

7.2. Patients with moderate to severe skeletal discrepancy

Early determination of the eventual need for maxillary osteotomy is a very important decision for the orthodontist.

Extracting lower premolar to correct anterior crossbite and trying to camouflage skeletal discrepancy are not appropriate in growing children. In that case, leveling only the upper arch, finishing with crossbite, and monitoring the growth are the best options. Early surgical options can be considered if needed [5].

7.3. Summary of treatment sequencing for mixed dentition stage

Evaluation CBCT–maxillary expansion and/or ortho-tooth movement–fistula closure and alveolar graft–maxillary protraction if needed.

Maxillary protraction protocol: 350–450 gram per side protraction force is adequate 14–15 hours a day for 6–12 months. MP can be started 4–6 weeks after SABG.

Part of this force should be transmitted as intermittent force to the maxillary anterior teeth through oral appliances such as an arch wire and/or lingual arch.

7.4. Unfavorable conditions

If maxillary deficiency accompanies with a wide alveolar cleft and/or fistula, it will be more challenging for both orthodontist and surgeon to treat growing patients [54] (**Figure 9**).

Late bone grafting or prolonged orthodontic treatment prior to bone grafting leads to loss of orthodontic control, marked instability of the premaxilla, and difficulty in maintaining

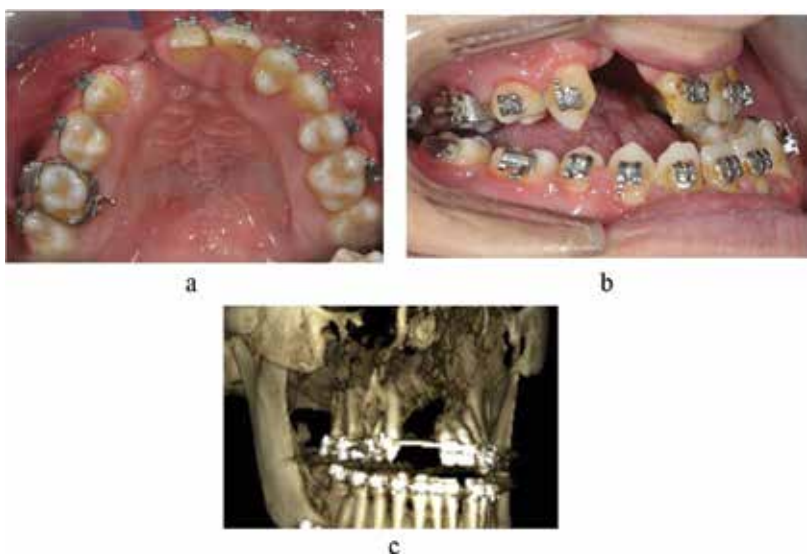

Figure 9. Wide alveolar cleft limits both orthodontic treatment and maxillary osteotomy. (a, b) intraoral views of a wide cleft, (c) 3D image of the case.

anteroposterior growth. In patients with BCLP, the unstable premaxilla with the small amount of maxillary bone attached to the vomer is usually incapable of being maintained with good stability without grafting.

When the premaxilla has been effectively grafted, any need to forward the maxilla at a later date can be accomplished by Le Fort osteotomy with a much diminished possibility of relapse [19].

8. Summary

The success of the orthodontic treatment and SABG is strongly interrelated. Carefully coordinated orthodontic and surgical involvement is critical for the well-being of the patients with CLP.

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Chapter 12

Contemporary Treatment Approaches to Obstructive Sleep Apnea Syndrome

Handan Tuğçe Oğuz

Additional information is available at the end of the chapter

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Abstract

Upper airway diseases decrease sleep time and/or quality, which leads to excessive day time sleepiness, fatigue, and lack of concentration. Upper airway diseases can be classified into two groups: upper airway resistance syndrome and sleep apnea syndrome. Sleep apnea syndrome is a disease, which occurs during sleeping, characterized by apneic and hypopneic events, low oxygen levels in lungs, blood oxygen desaturation, sleep arousal, and/or awakening. It increases morbidity and mortality of the patients. Sleep apnea syndrome has three types: central, mixed, and obstructive. Central and mixed apnea can be treated only by medical practitioners. But in the treatment of obstructive sleep apnea, orthodontists and maxillofacial surgeons have an important role. The aim of this chapter is to present current information about etiology, diagnostic tools, and treatment alternatives of obstructive sleep apnea and to introduce dental sleep medicine to orthodontists.

Keywords: dental sleep medicine, obstructive sleep apnea, dental treatment approaches for obstructive sleep apnea, pediatric obstructive sleep apnea

1. Introduction

Sleep-related breathing disorders are pathological changes in respiratory pattern during sleep. They increase morbidity and mortality, decreasing the life quality of patients. The patients complaining about waking up restless and headache, daytime sleepiness, lack of concentration, emotional changes, and unbalanced blood pressure may probably have a secret sleep-related breathing disorder.

Dental sleep medicine is an area of dental practice that focuses on dental therapy to treat sleep-disordered breathing, including snoring and obstructive sleep apnea. Dentists who are

specialized in dental sleep medicine can diagnose the early signs and symptoms of potential obstructive sleep apnea.

2. Upper airway diseases

Upper airway diseases decrease sleep time and/or quality, which leads to excessive daytime sleepiness, fatigue, and lack of concentration. Upper airway diseases can be classified into two groups: upper airway resistance syndrome and sleep apnea syndrome.

Snoring is the most evident symptom of these syndromes, but it is not a disease. Snoring is just a sound resulting from soft tissues in the upper airway vibrating during inspiration, due to the increased velocity of air, caused by the decrease in size of airway space. Almost all patients with upper airway resistance syndrome and sleep apnea syndrome snore. However, snoring patients may or may not also have sleep apnea or upper airway resistance syndromes. Prevalence of snoring is estimated in 35–40% of the population. Snoring is a sign that some type of resistance is occurring in the upper respiratory system.

Upper airway resistance syndrome (UARS) occurs when resistance of upper respiratory system increases and breathing effort crosses over from harmless snoring. The transformation of snoring to UARS can be caused by aging (muscle tone in the throat decreases in time) and weight gain. In upper airway resistance syndrome, breathing needs much effort due to the narrow upper airway, and this extra effort leads to sleep arousal, frequent nocturnal awakenings, chronic insomnia, and excessive daytime sleepiness. Apnea and hypopnea are not symptoms of this disease.

UARS and obstructive sleep apnea syndrome (OSAS) have similar etiologies and symptoms, so differential diagnosis is very important. The key differences between UARS and OSAS consist of apnea-hypopnea existence, gender, and weight differences. In UARS apnea and hypopnea are either absent or very low. UARS patients are often in average weight, but OSAS patients are generally overweight or obese. UARS affects two genders equally, but OSAS affects men more than women.

UARS treatment consists of behavior and lifestyle changes, oral-dental appliances, and continuous positive airway pressure (CPAP) therapy. When UARS is untreated or treatment is unsuccessful, it can end up developing sleep apnea syndrome.

Sleep apnea syndrome is a chronic, progressive, and life-threatening disorder which occurs during sleeping and characterized by apneic and hypopneic events, hypoxia, blood oxygen desaturation, sleep arousal, and/or awakening. Sleep apnea has three types: central, obstructive, and mixed. In central apnea respiratory muscles need extra effort because breathing is limited or completely blocked due to the obstruction in the upper airway. Mixed apnea is the combination of central and obstructive apnea. Central and mixed apnea can be treated only by medical practitioners. But in the treatment of obstructive sleep apnea, dentists have an important role [1–4].

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3. Obstructive Sleep Apnea Syndrome (OSAS)

Obstructive sleep apnea syndrome is a sleep-related breathing disorder that increases morbidity and mortality in patients, which is characterized by recurrent episodes of partial or complete upper airway obstructions and blood oxygen desaturation during sleep. This syndrome leads to severe long-term health problems as a result of the decrease in blood pressure during apnea and hypopnea episodes. Increased risk of high blood pressure, heart disease, heart arrhythmias, heart failure, and stroke can be seen in OSA patients.

OSAS prevalence is estimated at 3–7% in men and 2–5% in women. People from all ages, even pediatric and adolescent ages, can be affected from OSAS, but middle ages have the highest prevalence.

There are some definitions and indexes in order to define and classify the syndrome:

Apnea is the arousal of breathing for 10 seconds or more. It is a full obstruction of the airway when a person is asleep.

Hypopnea is a reduction in ventilation of at least 50% that results in a decrease in arterial saturation of 4% or more. It occurs due to partial airway obstruction.

Apnea index (*AI*) shows the average number of apneas and hypopneas per hour of sleep. The term respiratory disturbance index (RDI) can be used instead of AHI.

Arousal is a sudden change from a deeper sleep stage to a superficial sleep stage. It ends up in apnea and hypopnea.

Arousal index shows the average number of arousals per hour of sleep.

Minimum oxygen saturation is the minimum oxygen saturation level recorded in the whole sleep time [5–9].

4. Pathogenesis of obstructive sleep apnea

Upper airway has a great tendency to obstruction. Any pathological change that narrows the airway leads to OSAS. The pharynx is the site of upper airway obstruction during sleep. The size of the pharyngeal lumen depends on the balance between the forces that narrows and dilates the airway. The force that narrows the upper airway is the suction type, negative air pressure occurring during inspiration, and personal anatomical factors. The force that dilates the upper airway is the tension of the genioglossus and tensor veli palatini muscles surrounding the airway and tension of other small muscles attached to the pharynx. The difference between the narrowing and dilating forces are called transmural pressure. During sleep the tension of the muscles surrounding the airway decreases. The tongue and soft palate displace to the posterior wall of the oropharynx and narrows the upper airway. The airflow rate increases due to the narrowing airway dimension. The increased airflow applies more negative, suction-type

pressure to the airway lumen. The increased negative airway pressure, decreased muscle activity, and airway lumen diameter lead to upper airway obstruction and apnea. During apnea the blood oxygen desaturation decreases, carbon dioxide saturation increases, and pH decreases. This change leads to the stimulation of central nervous system chemoreceptors, and awakening occurs to end up in apneic process. When the patient awakens, the tension of the muscles increases, which ends up in the obstruction of the upper airway. Blood oxygen saturation increases, carbon dioxide saturation decreases, pH increases, and the patient falls asleep again. This sleeping, apnea, awakening cycle continues during total sleep time [3, 5, 6, 8].

5. Etiology of obstructive sleep apnea

The etiology of OSAS is multifaceted. The factors that lead to OSA can change in adults and pediatric and adolescent patients.

5.1. Adult OSAS etiology

General factors: Age, gender, obesity, alcohol consumption, sedative medicine consumption.

Anatomical factors: Lesions and anatomical variations, nasal obstruction, neck circumference, head and neck position, airway shape, and dimension.

Mechanical factors: Supine position, upper airway resistance, upper airway compliance, intraluminal pressure, extraluminal pressure, thoracic caudal traction, mucosal adhesive affects, vascular factors.

Neuromuscular factors: Upper airway dilator muscles, upper airway reflexes, dilator muscle, and diaphragm relations.

Central factors: Hypocapnic apneic threshold, periodical breathing, arousal, and cytokines.

5.2. General factors

1. Age: OSA can be seen in all ages but mostly seen in middle ages. The tissue elasticity, ventilation on central, and pulmonary and cardiovascular functions decrease with age, and those factors lead to OSAS.
2. Gender: Men are affected more than women because of testosterone hormone inhibits respiration and men have more pharyngeal and supraglottic airway resistance than women.
3. Obesity: There is a close correlation between obesity and apnea. Obese patients have more fat deposits around soft palate. The fat infiltration from lateral to upper airway decreases the airway area and leads to apnea.
4. Alcohol and sedative medicine consumption: Investigations indicate that the activity of genioglossus and tensor veli palatini muscles decreases when the patient is asleep in a supine position. Alcohol and sedative medicine consumption can exaggerate this condition; the airway area decreases leading to apnea.

5.3. Anatomical factors

1. Lesions and anatomical variations: Changes in craniofacial form, inadequate muscle activity, soft tissue anomalies, micrognathia, retrognathia, macroglossia, enlarged soft palate, steep mandibular plane angle, nasal obstruction, adenotonsillar hypertrophy, reduced airway lumen, vocal cord dysfunction, edema of the epiglottis, polyps, tumors, acromegaly, nasal obstruction, juvenile temporomandibular ankylosis, and syndromes (Franceschetti-Treacher Collins, Apert, Crouzon, Pierre Robin, and Down syndromes) may cause OSA.
2. Neck circumference: Neck circumference is a significant predictor of OSA. Men and women have a greater risk for OSA if they have neck circumferences of 17 and 16 inch or greater, respectively.
3. Head and neck position: When a patient changes from upright to supine position, the thickness of the soft palate increases, and the anteroposterior oropharyngeal cross-sectional area decreases. The decrease in cross-sectional area is most evident in posterior of the soft palate.
4. Airway lumen diameter and shape: The investigations have shown that the OSAS patients have narrower airway area even they are awake and upright position. The airway has an anteroposterior configuration in normal people, but OSAS patients' airway has horizontal configuration [1–12].

6. Symptoms of obstructive sleep apnea

Major symptoms: Snoring, apnea, excessive daytime sleepiness.

Cardiopulmonary symptoms: Systemic hypertension, pulmonary hypertension, nocturnal arrhythmia, atypical breath ache, acute pulmonary edema, reversible proteinuria.

Neurobehavioral and social: Restlessness and headache in the morning, insomnia, depression, anxiety, mood disturbances, tendency to accidents, nervousness, forgetfulness.

Other symptoms: Dry mouth, night transpiration, nocturnal cough, hearing loss, gastroesophageal reflux [1–21].

7. Classification of OSAS

The severity of OSAS can be classified into three groups according to the apnea-hypopnea index (AHI). An AHI score smaller than 5 is considered normal. An AHI score between 10 and 20 represents mild obstructive sleep apnea, AHI score of 21–40 represents moderate obstructive sleep apnea, and AHI score greater than 40 represents severe obstructive sleep apnea [10–12, 22].

8. Diagnostic methods of OSAS

OSAS is a life-threatening syndrome which affects cardiovascular, pulmonary, psychological, and neurological systems. Because of this reason, the diagnosis and treatment plan of this syndrome should be done by a multidisciplinary team. The team should consist of medical practitioners, sleep specialists, psychiatrists, and dentists. The diagnosis and treatment plan should be done after a comprehensive medical and dental history and examination [10, 12, 13, 22].

The diagnostic methods of OSAS can be done in steps:

1. Clinical examination
2. Endoscopical examination
3. Polysomnography (PSG)
4. Radiological examination

8.1. Clinical examination

a. Nose-ear-throat and head-neck inspection: Inspection of the tonsil, soft palate, and tongue; inspection of maxillofacial characteristics; inspection of craniofacial characteristics, neck circumference, and natural head position.

b. Dental inspection: Malocclusion, maxillar and mandibular deficiency, narrow maxilla and maxillar segment, and dry mouth.

c. Systemic examination: Weight and inspection of pulmonary, cardiac, and neurobehavioral functions.

8.2. Endoscopic examination

Endoscopic examination is frequently used for the inspection of upper airway from the nose to the glottis. The dynamic upper airway changes and the level of collapse in upper airway can be seen in this method. Especially before surgical treatments, the level and severity of collapse can be seen, and the operation can be planned according to these data.

8.3. Polysomnography (PSG)

Nocturnal, laboratory-based polysomnography is the gold standard diagnostic tool for obstructive sleep apnea syndrome. It is used for diagnosing, determining the severity of disease, and evaluating various other sleep disorders that can exist with or without OSAS. It is also used after OSAS treatment for the evaluation of the treatment effectiveness. A classical PSG consists of electroencephalogram (EEG), electromyogram (EMG), and electrooculogram (EOG) records. PSG measures sleep cycles and stages by recording airflow in and out of lungs during breathing, the level of oxygen in blood, breathing effort and rate, brain waves, electrical activity of muscles, eye movements, and heart rate. The most important parameters measured by PSG are apnea index (AI), hypopnea index (HI), apnea-hypopnea index (AHI), and minimum

oxygen desaturation. PSG can be done either at a sleep center or in the patients at home. But home-based PSG records do not have the same reliability as sleep center-based PSG records.

8.4. Radiological inspection

Upper airway imaging should be performed in OSAS patients in order to examine the anatomy of the pharynx, surrounding the craniofacial and soft tissue structures. Lateral cephalometry, computerized tomography (CT), and MR imaging (MRI) are the most commonly used radiological inspection methods of OSAS.

8.4.1. Lateral cephalometry

It is the most commonly used radiological inspection method of OSAS. It allows to investigate the sagittal and vertical positions of the maxilla and mandible according to the cranial base, maxillomandibular relationships, the sagittal and vertical position of hyoid bone, soft palate anatomy, and head posture. It is cheap and noninvasive but documentation can be done in two dimensions.

Cephalometric analysis alone is insufficient to diagnose OSAS. But some cephalometric parameters can accept a signal for OSAS risk. Increased mandibular plane angle, steep occlusal plane, over-erupted posterior dentition, large gonial angle, anterior open bite, adipose tissue placed in the submental and parapharyngeal region, larger and wider soft palate, and increased linear distance from the mandibular plane to the hyoid bone (a distance bigger than 15.4 mm) create OSA risk [10, 13].

8.4.2. Computed tomography (CT)

The high-resolution and three-dimensional evaluation of the skeletal system and soft tissues in maxillofacial complex can be performed by computed tomography. CT imaging is done in supine position and can give us the opportunity of recording the changes in upper airway cross-sectional area in different phases of respiration and predicting the collapse area in OSAS patients. The disadvantages of this technique are as follows: being expensive, high radiation level, and the low-resolution images of fat tissues around upper airway according to MR imaging [14].

8.4.3. Magnetic resonance imaging (MRI)

Magnetic resonance imaging allows 3D imaging and accurate measurement of the upper airway, soft tissues, and skeletal structures. MRI is performed in supine position, it allows high-resolution visualization of soft tissues and fat deposits around pharyngeal airway, and the volume and area of the airway can be measured accurately. With ultrafast mode dynamic observation can be done. But it is expensive and cannot be used in patients who have claustrophobia, ferromagnetic clips, or pacemaker [15].

9. Treatment of adult obstructive sleep apnea syndrome

OSAS is a life-threatening disease. Because of this reason, early, accurate, and individualized treatment should be performed by a multidisciplinary team. The aim of OSAS treatment is

to decrease the number of obstructive episodes and severity and the collapse tendency and increase the airway area, blood oxygen saturation, and life quality.

Treatment options in OSAS are:

1. Behavioral modification
2. Surgical treatments
3. Use of nasal continuous positive airway pressure device (nCPAP)
4. Use of oral appliances

9.1. Behavioral modification

Weight loss, quitting smoking and alcohol, changing sleep positions and head posture, and avoidance of central nervous system depressors may be beneficial for some patients. Weight loss may decrease OSA symptoms by reducing the size of the tongue and soft palate. Quitting alcohol and depressor drugs prevents the relaxation of upper airway muscles. Alteration of sleeping position can prevent the tongue and mandible moving backward and narrowing the airway. The patients are instructed to sleep on their sides rather than their backs.

9.2. Surgical treatment

OSA resulted from anatomic obstruction of the upper airway, unsuccessful behavioral therapies, and inability to tolerate CPAP or oral appliances, which are inclusion criteria for surgical treatment. Tracheostomy, tonsillectomy and adenoidectomy, genial advancement with or without hyoid myotomy, uvulopalatopharyngoplasty, laser glossectomy and lingualplasty, maxillomandibular advancement, and epiglottoplasty are the surgical techniques used for the treatment of OSAS.

9.3. Nasal continuous positive airway pressure (nCPAP) device treatment

It is a device which has a small air pump connected to either a sealed face or nose mask. The device opens the pharyngeal airway and prevents the soft tissues from collapsing and blocking the airway. It is the gold standard treatment option for moderate to severe OSAS cases, and the success rate is about 75%. However, patient compliance is poor because of the pump noise, the irritation of nasal mucosa because of the airflow to the nose, xerostomia, and poor retention. The patient non-compliance ratio was reported to range from 46 to 83%. The ordinary usage of nCPAP device for 4–6 weeks decreases the volume of the tongue and increases the pharyngeal volume. Inclusion criteria for nCPAP therapy are moderate and severe OSAS patients with AHI score greater than 20, mild OSAS patients with AHI scores between 10 and 20 but has excessive daytime sleepiness and cardiopulmonary or cerebrovascular risks, anatomical-based OSAS patients whose medical health condition is inappropriate to surgical treatment, and mild-to-moderate OSA patients who have failed behavioral modification therapy and unable to tolerate oral appliances [1–10, 16, 17].

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10. Oral appliance therapy

Oral appliance treatment was first introduced in the 1980s and is a very effective treatment option for mild-to-moderate OSAS and seven patients with severe OSA who cannot tolerate CPAP or refuse surgical therapy. The American Sleep Disorders Association reported that oral appliance therapy is the primary treatment for patients with mild OSA and a secondary treatment option for moderate to severe OSA. For severe OSA patients, reduction in AHI score occurs, but it cannot turn into the normal range. If the AHI score cannot be decreased to 20, long-term health risks will continue. Oral appliances can be successful if only they are used after the etiological factors are eliminated. Only obstructive sleep apnea can be treated with oral devices; they are not indicated for central and mixed apneas [10–12, 16–21, 23–32].

10.1. Oral appliance therapy indications

1. Snoring patients
2. Upper airway resistance syndrome
3. Mild OSAS patients who have failed behavioral modification therapy
4. Moderate to severe OSAS patients who refused or failed CPAP therapy or surgery

10.2. Oral appliance therapy contraindications

1. Central or mixed obstructive sleep apnea
2. TMJ diseases
3. Periodontal diseases
4. Insufficient oral hygiene
5. Anatomical based OSAS

11. Types of oral appliances

Oral appliances can be classified into four groups according to their affect mechanisms:

1. Mandibular advancement devices (MADs)
2. Tongue retaining devices (TRDs)
3. Palate lifting appliances (PLA)
4. OPAP appliances (oral appliances + CPAP device)

12. Mandibular advancement devices (MADs)

MADs were first described by Pierre Robin in 1934 in the treatment of a patient with micrognathia as a modified monobloc in order to reposition the mandible in a more forwarded position and open the airway. This advancement makes the attached soft tissues and tongue stretch and stabilize; by this way, oro- and hypopharyngeal airways enlarge.

MAD appliances can be divided into several groups: monobloc-style one-piece or twin block-style two-piece, available to activation or unavailable to activation, teeth-supported or teeth- and tissue-supported, and soft- or hard-materialed. All of these appliances increase the distance between soft palate and posterior wall of the pharynx and enlarge the space between tongue root and posterior region of the oropharynx.

Tooth- and tissue-supported, soft-materialed, activation-optioned MAD appliances are reported to be more successful in OSAS treatment. One-pieced or two-pieced appliance design does not affect the treatment success.

MAD-type oral appliances are found the most effective type of oral appliances in OSAS treatment [10–12, 17, 19, 23, 25–30].

12.1. MAD indications

Normal or reduced facial height, patients have at least eight teeth per arch, patients who have normal soft palate thickness and normal positioned soft palate, and obese patients.

12.2. MAD contraindications

The patients with thick and enlarged soft palate, periodontal disease, and TMJ problems.

MAD appliance construction:

a. Upper and lower dental impressions are taken.

b. Maximum opening, left and right lateral excursion, and maximum protrusion are measured.

c. The appliance is constructed using a position 75% of the patients' maximum protrusion. Vertical opening amount changes individually. But it is recommended to open the bite at least 5 or 6 millimeters.

d. Bite registrations in centric occlusion and advanced position are obtained. A George gauge and light-body impression material can be helpful in stabilizing the construction bite position.

e. The MAD appliance fabricated from soft acrylic, hard acrylic, silicone-based, or Essix-based materials according to the type of the chosen appliance.

12.3. MAD treatment mechanism

MADs displace the mandible, suprahyoid, and genioglossus muscles anteriorly. This anterior movement prevents oropharyngeal airway obstruction. The forward and downward

displacement of the mandible also decreases the gravitational effect of the tongue in supine position and enlarges the velopharynx by stretching the palatoglossal and palatopharyngeal arches (**Figures 1** and **2**).

Figure 1. One-piece, non-activation MAD appliance.

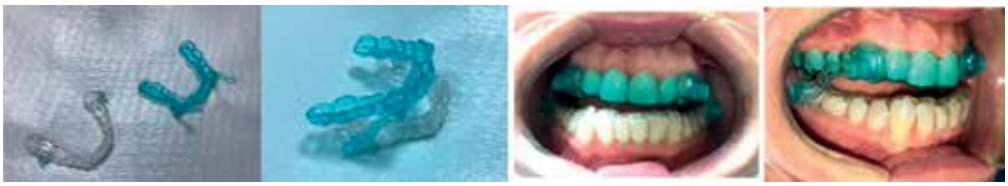

Figure 2. Two-piece, activation-optioned MAD appliance.

13. Tongue-retaining devices (TRDs)

Tongue-retaining devices were first described by Cartwright and Samelson in 1982 [28]. During sleeping and in supine position, the tongue and all gravity-dependent tissues tend to fall posteriorly. With TRDs, the tongue is prevented from dropping posteriorly by suction created when the patient forces the tongue into a hollow bulb built into the device. The forward position of the tongue increases the volume and decreases the resistance of upper airway. The superiority of TRDs over MADs is that they can be used for edentulous patients [10–12, 16, 17, 19, 20, 23, 25, 31].

13.1. TRD indications

Edentulous OSA patients, OSA patients who have TMJ disorders, OSA patients with big tonsils and large tongues, OSA patients who have less than six teeth per arch, hypothyroidism, and sleep position–related apnea-hypopnea occurrence.

13.2. TRD contraindications

Severe periodontal diseases, bruxism, chronic nasal obstruction, patients who are unable to move their tongue anteriorly, and obese OSA patients.

13.3. TRD appliance construction

The TRD appliance can be fabricated from soft copolymer materials by the technician to the OSA patient individually, or standard fabricated appliances can be used to the patient.

13.3.1. Individualized TRD appliance construction

a. Upper and lower dental impressions are taken.

b. Maximum opening, left and right lateral excursion, and maximum protrusion measured.

c. The appliance is constructed using a position 75% of the patients' maximum protrusion. Vertical opening amount differs patient to patient, but it is more than MAD vertical opening amount, in order to place the hollow bulb part.

d. The appliance is fabricated from soft copolymer material like a monobloc with a hollow bulb part to place the tongue.

13.3.2. Fabricated TRD appliance construction

a. The tongue is measured by wrapping a piece of dental floss around the tongue. Then, the floss is removed and measured.

b. The fabrication appliances have two types, edentulous and dentulous, and three sizes, small, medium, and large.

c. The correct type and size are chosen for the patient.

d. The bulb appliance is moistened and compressed and the tongue is inserted.

TRD-type appliances are found to be effective in mild to moderate OSA treatment. They decrease AI, HI, AHI, oxygen desaturation, and excessive daytime sleepiness and increase minimum oxygen desaturation and genioglossus muscle activity. But the patient compliance is low with TRDs because of the irritation in the tongue, hypersalivation, uncomfortable feeling, and non-esthetic appearance.

14. Palate lifting appliances (PLA)

They are used in patients who have a thick and enlarged soft palate. The appliance supports and stabilizes the soft palate, prevents soft palate blocking the airway, and reduces the vibration of soft palate leading to snoring. But they are uncomfortable appliances and the patient compliance rates are very low. Because of this reason, they are not frequently used in OSA treatment [10, 11].

15. OPAP appliances (oral appliance + CPAP device)

nCPAP device applies very high pressure and high airflow rate which leads to irritation of nasal, oral, and throat mucosa and decreases patient compliance. When MAD-type oral appliances are combined with CPAP device, the pressure given by nCPAP diminishes; irritation

of nasal, oral, and throat mucosa decreases; and patient compliance and treatment success increase. The adaptation of CPAP and MAD appliance should be done by a dental sleep specialist [10, 18].

16. Side effects of oral appliance therapy

There are very few side effects of oral appliances when compared to other treatment alternatives. Researches have reported that patients prefer treatment with an OA over surgery or CPAP appliance, and compliance with OA has been reported to be 40–80%.

The side effects of OA therapy can be divided into two groups:

1. Short-term side effects of OA therapy: Excessive salivation or dry mouth, TMJ sounds and/or TMJ pain, tooth pain, odd bite feeling in the morning, gum irritation, tenderness in masticatory muscles, or myofascial pain. These complications are generally mild and transient [10, 11, 19, 20].
2. Long-term side effects of OA therapy: OA therapy for OSAS patients is a lifelong treatment. Skeletal, dental, and occlusal changes can occur from OA therapy in 2 or more years' time. Craniofacial changes related to long-term oral appliance use can be determined with cephalometric investigations. Skeletal changes generally occur after average treatment duration of 5 years. The changes can be summarized as more downward and forward position of the mandible, increased lower facial height, decreased overbite and overjet, retroclination of the maxillary incisors, proclination of the mandibular incisors, changes in molar relationship, and curve of Spee flattening. Long-term use of OA also makes differences in upper airway configuration. Palatal length decreases and pharyngeal area increases in time. These changes can be due to the loss of edema caused by snoring and repetitive apneas [10, 32–38].

17. Efficacy and success of oral appliance therapy

OA therapy is an effective and safe long-term therapy for patients with snoring, mild to moderate OSAS. However, the efficiency varies on many factors including the type of OA, materials used for fabrication, piece number of appliance (monobloc or bibloc), titration ability (titrable or untitrable), and degree of sagittal and vertical mandibular displacement.

The efficacy criteria of oral appliance therapy in OSA are:

a. Positive changes in PSG test: Decrease in AHI, AI, HI, arousal score, increase in blood oxygen saturation, sleep time, and efficiency

b. Positive subjective feedback of patients: Decrease or quitting snoring and decrease in daytime sleepiness

c. Enlargement of pharyngeal airway area [10–12, 25, 28, 31, 32]

17.1. To summarize

1. MADs are reported more effective than other types of OAs.
2. Monobloc-type one-piece and soft-materialed OAs are found to be more efficient in reducing AHI, AI, and snoring.
3. Forward and downward displacement of the mandible is recommended for enlargement in pharyngeal airway area. But this displacement amount should be assessed individually.
4. No definite conclusions can be drawn regarding which type or design of OA has a beneficial influence on subjective treatment efficacy. Individually determination of appliance is very important in OA treatment success [10–12, 16, 17, 19, 20, 23, 25–28, 30–32].

18. Intraoral appliance treatment guideline for OSAS patients

The American Sleep Disorders Association (ASDA) has prepared a protocol for OA treatment in OSAS patients. A dental sleep specialist should follow up the stages below in the treatment of an OSAS patient [21, 24]:

1. Detailed medical and dental history.
2. PSG and medical specialist's report investigation.
3. Dental investigation: Soft tissue and intraoral inspection, periodontal-occlusal-TMJ inspection, intraoral habits and parafunction inspection, teeth and restoration inspection, and radiological inspection.
4. Upper-lower dental impressions and bite registration are obtained.
5. Selection of OA type for the patient individually. (The success and compliance rate varies case to case, and choosing the right type of OA is the most important stage of OA treatment protocol.)
6. OA fabrication.
7. Adjustment of the OA. Appliance fit and comfort are controlled, and usage instructions are given to patient.
8. Follow-up PSG and lateral cephalogram are taken after appropriate appliance adjustment/titration and patient adaptation period. Objective determination of the treatment efficiency can only be observed by PSG test with the appliance in place. Nearly all patients report positive change subjectively; however, they should be proven with PSG reports.
9. If the treatment is found to be effective in the first year of the therapy, four follow-up appointments are recommended. After 1 year, two follow-up appointments per year are enough. At the follow-up appointments, appliance fit, comfort, effectiveness, and patient

compliance are investigated. If the OA is found to be less effective, the titration of OA or fabrication of a new OA should be done.

19. Pediatric and adolescent obstructive sleep apnea

Obstructive sleep apnea in pediatric and adolescent patients is characterized by episodic partial or complete upper airway obstruction during sleep. All children with OSAS snore. It has been estimated to occur in 5–6% of children. It is most seen in preschool children; the peak age is 3–6 years, which coincides with the growth of adenoids and tonsils. Pediatric OSAS is similar to adult OSAS, but there are differences. Sleep disruption occurred by respiratory pauses less than ten seconds. Hypopneic episodes can be seen, usually more than five to ten episodes per night with oxygen saturations less than 85%. Pediatric OSAS effects both gender at the same ratios, different from adult OSA. Seven percent to nine percent of children snore every night; 18% of them snore in nasal, ear, or throat infection periods. The prevalence of pediatric OSAS is estimated about 0.5–3%. AHI scores of pediatric sleep apnea is controversial. Some protocols of AHI score greater than 1 should be accepted as a pediatric OSA predictor; some protocols accepting AHI score greater than 5 is pathognomonic.

19.1. Pediatric and adolescent OSA etiology

Hypertrophic adenoids, hypertrophic tonsils, maxillary transverse deficiency, class 3 maxillary skeletal deficiency, class 2 mandibular skeletal deficiency, overweight and obesity, and craniofacial anomalies. (Pierre Robin Sequence, Goldenhar syndrome, Crouzon syndrome, Apert syndrome, cleft lip and/or palate, vertical face anomalies, Marfan syndrome, and associated 22q deletion syndromes).

19.2. OSA symptoms in children

Abnormal breathing during sleeping, frequent awakenings or restlessness, frequent nightmares, enuresis, difficult awakening, excessive daytime sleepiness, hyperactivity-behavior problems, daytime mouth breathing, poor or irregular sleep patterns, early recognition of mouth breathing and airway obstruction; symptoms of recurrent blocked nose; recurrent nasal, ear, and throat infections; parents concerned about snoring should alert the dental professional for definitive diagnosis for pediatric OSA.

19.3. Differences between pediatric and adult OSA

a. Pediatric OSAS effects both genders equally.

b. Snoring is irregular and interrupted with apneic-hypopneic events in adult OSAS. In pediatric OSAS snoring can be continuous.

c. Daytime sleepiness is mostly seen in adult OSA. In pediatric OSA behavioral changes and growth retardation are seen more than daytime sleepiness.

d. Mouth breathing is mostly seen in pediatric OSA [39–42].

e. Obesity is mostly seen in adult OSA; in pediatric OSA, the patient can be of normal weight or thin.

f. The etiology of pediatric OSA is mostly hypertrophic adenoids and tonsils. In adult OSA retropharyngeal and retrolingual pathologies are more effective.

g. The gold standard treatment protocol for adult OSA is CPAP appliance. In pediatric OSA adenotonsillectomy is advised rather than CPAP therapy.

19.4. Clinical inspection in pediatric sleep apnea syndrome

a. Nose-ear-throat and head-neck inspection: Craniofacial, orofacial, and maxillofacial characteristics are investigated. In craniofacial inspection symptoms of various syndromes are investigated. In orofacial inspection features of the tongue, soft palate, and tonsils are investigated. In maxillofacial inspection maxillofacial characteristics like facies adenoidalis, rhinolalia clausa, long-thin face, narrow maxilla and maxillary segment, overlenghtened teeth, hypoplastic mandible, and septum deviation are investigated.

b. Systemic inspection: Includes inspection of pulmonary, cardiac, physical, and mental condition functions and weight.

20. Pediatric OSA treatment protocol

1. Surgical approaches: In pediatric patients dentists should examine oropharynx carefully. Both the lingual and pharyngeal tonsils can be visible intraorally, but the adenoids will not. When hypertrophic tonsils are observed clinically or radiographically, referral for endoscopic evaluation and possible surgical removal by a pediatric otolaryngologist should be made. Early removal of these tissues can prevent the long-face growth pattern with narrow upper and lower dental arches and anterior open bite.

2. Weight loss and behavioral changes

3. Rapid maxillary expansion (RME): Cephalometric and acoustic rhinometry studies report that with RME maxilla, the palate and floor of nasal cavity expand, which leads to increase the volume and decrease the airflow resistance in nasal cavity. It also makes statistically significant changes in tongue size position and hyoid position. The expansion in maxillary dentition gives the tongue a greater space and more forward positioning to the tongue. The widened maxillary basal bone on the velum, the superior pharyngeal constrictor muscles, and the surrounding orofacial musculature can increase the muscle tonus.

4. Class 2 growth modification therapy: When the pediatric and adolescent patients have both mandibular deficiency and obstructive sleep apnea, mandibular advancement devices like Herbst, twin block, monobloc, bionator, Frankel 2, etc. can be used for the treatment.

5. Class 3 growth modification therapy: When the pediatric or adolescent patients have both class 3 maxillary deficiency and OSA, class 3 growth modification therapy is recommended. In this treatment maxilla is widened first with RPE, followed by orthopedic traction with protraction face mask (reverse pull headgear).
6. CPAP therapy: This therapy can be advised to severely affected adolescent OSAS patients. In pediatric and adolescent population, the long-term use of CPAP can cause craniofacial side effects. The force applied from the elastic strap of CPAP mask applies a restraining force on the maxilla. So, CPAP therapy can lead a class 3 skeletal deficiency in pediatric and adolescent OSA patients. But it is unclear how much of the malocclusion results from CPAP and how much results from an underlying adverse growth pattern.

21. Conclusion

Sleep-related breathing disorders are complicated problems, which decrease life quality and increase morbidity and mortality in patients.

Dentists, who are specialized in dental sleep medicine, can see the early symptoms of these diseases and can be frontline screeners for potential OSAS diagnosis. In the treatment of snoring and mild-to-moderate obstructive sleep apnea, the oral appliance therapy was found to be a very effective treatment option.

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This book provides information on the current technological developments and new concepts in orthodontic treatment procedures. The main concepts of the book are scope innovations in accelerated tooth movement, new developments such as corticotomy, microperforations (MOP), piezosicion, photobiostimulation, laser in orthodontics, chemical agents, as well as complications and risks. The book contains interdisciplinary managements involving surgery first, cleft lip and palate therapy, orthognathic surgery, and obstructive sleep apnea. This internationally-recognized specialty is continuing to experience advancements in technology, instrumentation, and treatment methods.

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