CLINICAL CROWN LENGTH, GINGIVAL RECESSION DEVELOPMENT AND SAGITTAL INCLINATION CHANGES IN INCISORS ASSOCIATED WITH ORTHODONTIC TREATMENT: A RETROSPECTIVE STUDY

By

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ABSTRACT

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AIM: To retrospectively investigate the clinical crown length changes and the development of gingival recession in the labial aspect of the maxillary and mandibular incisors associated with orthodontic treatment and relate them to the observed changes in their sagittal inclination.

MATERIALS AND METHODS: Eighty-two consecutive subjects, treated by means of fixed orthodontic appliances in both dental arches, with good quality pre- and post-treatment dental casts and lateral cephalometric radiographs, were selected from the archives of a private orthodontic clinic. Incisor clinical crown length before and after orthodontic treatment, as well as the presence or absence of recession, were measured on digitized study models. Sagittal inclination changes were assessed on lateral cephalometric radiographs and categorized as proclination, retroclination or no change (±1°). Spearman's correlation coefficient, one-way analysis of variance and chi-square tests were used for analysis.

RESULTS: The mean change of clinical crown lengths for the maxillary incisors was from -0.24 to 0.01 mm, and for the mandibular, from 0.06 to 0.10 mm. The inclination changes were -1.78° and 1.03°, respectively, but no correlations were observed with changes in clinical crown length. Overall, no statistically significant differences were observed regarding clinical crown length changes and the presence of gingival recession between proclination, retroclination and no change groups.

CONCLUSIONS: The change of incisor inclination during treatment did not seem to affect labial clinical crown length increases and gingival recession development in this specific sample.

DEDICATION

With great gratitude, I would like to dedicate this thesis to my beloved parents, who have never stopped giving of themselves in countless ways, who have been very patient and supportive throughout this journey. Thank you both for giving me strength to chase my dreams.

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DECLARATION

I declare that all the content of the thesis is my own work. There is no conflict of interest with any other entity or organization.

Name: Shatha R. Al-Khalidy

Signature:

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1. INTRODUCTION

The harmony of a smile is particularly determined by the shape, position and color of the teeth (Townsend, 1993). In addition, an adequate mucogingival complex, in which the mucogingival tissues can sustain their biomorphologic integrity and maintain an enduring attachment to the teeth, as well as the underlying tissue, is essential (Dodwad, 2001).

Gingival recession has been defined as the denudation of the tooth root surface due to the displacement of the gingival margin apically to the cementoenamel junction (Gorman, 1967). It can be localized or generalized and associated with at least one tooth surface. Recessions are more frequently localized on the buccal surfaces (Kassab and Cohen, 2003); their development is multifactorial and their severity is correlated with age as recessions are more prevalent in older than younger individuals (Al-Wahadni, 2002). Furthermore, they are more frequently observed in males than females (Susin et al., 2004; Slutzkey and Levin, 2008). The mandibular incisor teeth have been shown to be the most prone to such adverse gingival changes (Khocht et al., 1993). Excessive gingival recessions can negatively affect the appearance of the dentition by causing esthetic impairment, and also increase the susceptibility for dentine hypersensitivity, root caries, cervical wear and the accumulation of dental plaque (Smith, 1997; Lawrence et al.,1995; Al-Wahadni, 2002). The periodontal tissues usually adapt when teeth are moved orthodontically, but tissue adaptability can be reduced when teeth acquire extreme positions (Artun and Krogstad, 1987). Current information suggests that adverse changes to the mucogingival complex can be encountered during orthodontic treatment (Wennström, 1996). However, the level of available evidence is low; the

amount of recession found in studies showing statistically significant differences between proclined and non proclined teeth is of questionable clinical consequence and should be treated with caution until additional research becomes available (Joss-Vassalli et al., 2010).

2. REVIEW OF THE LITERATURE

A beautiful smile is the best ornament a face could have and is the most primitive form of human communication. The harmony of the smile is especially determined by the shape, position and color of the teeth. People of all ages are becoming increasingly concerned about their smiles and overall appearance (Townsend, 1993). Thus, correction of malocclusion and the achievement of attractive smile esthetics may lead to significant improvements for individual patients in terms of dental health, psychological and social well-being, as well as quality of life (Proffit and Fields, 2012). Orthodontic treatment of malocclusions and dentofacial deformities, by ensuring proper alignment of teeth accompanied by harmonious occlusal and jaw relationship, may improve mastication, phonation and facial esthetics. These beneficial effects on the general and oral health play a positive role in improving individual's comfort and self-esteem and their overall quality of life (Graber et al., 2004). Therefore, the treatment objectives are consistent with the aims of medical interventions; namely ensuring health, the "state of complete physical, mental and social well-being", as perceived by the World Health Organization (World Health Organization, 1946). However, during orthodontic treatment patients may experience undesirable side-effects, such as discoloration, demineralization and cavity formation, enamel cracks and tooth surface loss, root resorption and adverse changes to the periodontal tissues (Graber et al., 2004). Therefore, it is very important to assess the risks of treatment as well as the potential gains and beneficial aspects of orthodontic therapy before deciding to treat a malocclusion. The psychological trauma of having orthodontic treatment, or indeed not having treatment, should not be overlooked and is an important consideration in the treatment planning decision making process (Justus, 2015).

2.1. Risks of orthodontic treatment

Orthodontic treatment involves the application of various materials and procedures that can have local effects on the teeth and surrounding tissues as well as systematic complications. Despite a lack of any incontrovertible evidence of a direct connection between treatment and specific pathological sequelae, studies have attempted to link orthodontically induced variables, such as treatment longevity, force magnitude, temporomandibular joint (TMJ) complaints, periodontal problems, allergic responses and even post-treatment changes, with the occurrence of various deleterious effects. In general, orthodontic treatment has the potential to cause significant damage to hard and soft tissues, but the risk of harm is considerably limited compared to surgery or other invasive medical procedures. Therefore, if orthodontic treatment is to be of benefit to a patient, the advantages it offers should outweigh any possible damage it may cause (Shaw, 1991).

An increased risk of complications might contraindicate the orthodontic therapy or influence its objectives, phases and conduct, aspects directly linked with the quality of the final outcome and prognosis. Generally speaking, the consequential benefits of the medical intervention must be greater than any potential damage. Legal regulations on medical conduct put emphasis on the patient's rights in treatment decision making and the need to obtain informed consent on the benefits and possible risks that may occur due to treatment after receiving a rigorous risk profile analysis. In cases where side effects appear, the avoidance of informing patients about possible complications associated with the medical intervention may lead to malpractice complaints, or even lawsuits (Graber et al., 2004).

Krishnan, (2009) emphasized the importance of establishing proper communication between the patient and the orthodontist to promote trust and improved oral hygiene habits. Whenever iatrogenic damage is observed, the patient should be immediately

referred to the appropriate specialist in order to avoid exacerbating problems. Patient selection plays a vital role in minimizing the risks of treatment, and the clinician should be vigilant in assessing every aspect of patients' situations and their malocclusion.

Nevertheless, abnormal force application and poor oral hygiene can lead to damage to teeth and paradental tissues. All components of the craniofacial complex may be affected, including the dental enamel, pulp, cementum, gingival, periodontal ligament (PDL), osseous structures, cartilages and muscles. Inflammation is the most significant process in force-induced tissue remodelling and tooth movement. The magnitude, direction and duration of force will determine the nature of the inflammatory changes that might occur during the orthodontic treatment (Giannopoulou and Catherine, 2009).

2.2. Classification of risks and complications of orthodontic treatment

A classification of the risks and complications associated with orthodontic treatment has been proposed by Graber et al. (2004) and includes the following:

a. Based on the localization of the condition:

- local effects, with manifestations on dento-maxillary apparatus structures (teeth demineralizations and discolorations, root resorption, periodontal disease),
- systemic effects (allergic reactions to nickel or latex and infective endocarditis).

b. According to the severity of the condition:

- mild, reversible (gingivitis),
- moderate, reversible (fracture of a ceramic crown),
- moderate, irreversible (enamel fracture during debonding),
- severe, irreversible (multiples caries and decalcifications, severe root resorption).

c. Based on orthodontist's role in the occurrence of side effects:

- standard inherent complications: these include side effects where the orthodontist's role is irrelevant (enamel changes due to acid etching when resins are used as bonding material),
- complications related to the patient's individual susceptibility or disease status not disclosed during evaluation, possibly unknown even to the patient (allergic reaction for which the history data were inconclusive; risk of severe root resorption and demineralisations present in association with a metabolic disease unidentified at the initial diagnostic assessment),
- conditions arising as a result of a passive operator intervention, associated with a lack of proper monitoring (lack of monitoring and proper prevention methods in cases with severe root resorption or decalcifications),
- medical errors induced by inappropriate medical objectives and deficient treatment conduct (enamel damage due to improper debonding technique; tooth movement into an area with alveolar bone defect causing severe loss of periodontal attachment).

2.2.1 Localized side effects

There are numerous localised side effects linked to orthodontic interventions. Fixed orthodontic appliances may induce both quantitative (enamel loss during bonding and debonding procedures) and qualitative enamel changes (discolorations). On a root level, the most undesirable side-effects of orthodontic treatment is the induction of severe root resorption. This process is associated with root shortening that may lead to insufficient ability on the part of the tooth to withstand the forces of normal oral function, which, in extreme cases, may result in early tooth loss. Regarding the pulpal reactions, orthodontic force application may cause a decreased oxygenation of pulpal tissue and this may vary

according to the force magnitude and duration of its action. Usually, the inflammatory reactions are transient and reversible, but severe irreversible changes, such as necrosis may occur. Greater risk of pulpal reactions is present in teeth with a history of severe periodontal injury during certain orthodontic procedures, e.g., during intrusion and extrusion (Bauss et al., 2008).

A) Enamel damage during bonding and debonding of the orthodontic devices

Enamel is characterized by a high mineral content (96 wt%) and low content of organic matter (0.4-0.8 wt%) and water (3.2-3.6 wt%). The mineral phase is generally described as calcium hydroxyapatite. The tightly packed, hexagonal, needle shaped crystallites of hydroxyapatite are the real unit of enamel, and these are arranged in prisms (Øgaard et al., 2004).

Enamel damage that occurs as a side effect to orthodontic therapy is largely related to the bonding and debonding techniques. According to current knowledge, bonding of orthodontic appliances may induce irreversible changes in tooth surfaces. After bracket bonding, demineralised enamel remains uncovered by resin. However, usually remineralisation occurs and reduces the risk of caries development. Bonding techniques have evolved, and self-etching adhesive resins have been introduced onto the market. Despite these adhesives producing less enamel damage, they have the disadvantage of a lower bond strength (Fjeld and Øgaard, 2006). During debonding and removal of the residual material, there is a risk of tooth damage such as enamel loss and cracks. These complications are irreversible and sometimes difficult to avoid. The frequency and severity of enamel loss are usually reduced when metallic braces and bonding materials based on glass ionomer cements are used. More severe effects are seen when ceramic brackets and adhesive resins are used as the materials of choice. The orthodontist has a crucial role in preventing this irreversible enamel damage by using the most appropriate

debonding techniques. A safe debonding technique aims to break the bracket and adhesive interface, and great caution is advised; especially in cases where there is softened, demineralised enamel adjacent to the bracket base. The residual bonding material is best removed with tungsten carbide burs at low speed, followed by surface polishing, in order to decrease the roughness and prevent subsequent plaque accumulation (Graber et al., 2004). Horizontal enamel cracks present after debonding bear a direct relationship to the orthodontic technique used, while vertical ones are often present in individuals who had never received previous orthodontic treatment (Øgaard and Fjeld, 2010).

B) Carious complications associated with the orthodontic intervention

As the orthodontic techniques have developed, concerns regarding tooth damage by carious lesions during treatment have increased. Caries development and progression is considered as one of the most frequent unwanted side effect associated with orthodontics. Decay damage associated with orthodontic technique presents some specific characteristics. It appears with increased frequency on the tooth surfaces where the bracket has been bonded, adjacent to its base. It is usually of low severity, being most frequently encountered in the form of white spot lesions, usually localised to the gingival and distal to the base of the bracket than mesially or occlusally. Evidence shows that the prevalence of this unwanted side effect is nearly 70% for white spot lesions and less than 5% for cavities (Al Maaitah et al., 2011). According to Chapman et al. (2010), more than 30% of maxillary incisors present decalcification after orthodontic intervention, with consequent negative effects on the patients' esthetics (Chapman et al., 2010). Demineralisation around brackets mainly occurs due to insufficient and/or improper oral hygiene maintenance, while the presence of orthodontic appliances increases the number of plaque retentive areas and decreases the self-cleaning capacity of the saliva. In orthodontic patients, plaque coverage is 2 to 3 times higher than the levels present in high plaque forming adults not

receiving this type of treatment (Klukowska et al., 2011). Orthodontic appliances also facilitate a decrease in salivary pH and an increase in the level of Streptococcus Mutans and Lactobacillus, all of which are elements favouring carioactivity (Vizitiu and Ionescu, 2010). Thus, maintaining good oral hygiene is mandatory during orthodontic treatment so as to reduce the risk of the aforementioned side-effects.

In addition, learning new skills on how to perform oral hygiene, and the use of additional auxiliaries, such as interdental brushes, is also required. Consequently, there are higher financial (the tooth brush wears faster, investments in auxiliary devices like interdental brush or oral shower) and time-related expenses (more time spent on ensuring a good oral hygiene) that are associated with the maintenance of adequate oral hygiene during the whole course of orthodontic treatment (Preoteasa et al., 2012).

In decay prevention, even if the patient plays the leading role by maintaining a good oral hygiene, the orthodontist's role is not negligible., It is recommended that the orthodontist evaluate carioactivity and oral hygiene habits before starting orthodontic therapy, while sometimes orthodontic treatment with fixed appliances may be postponed until adequate oral hygiene is evident. Primary prevention methods may be used; these include instructions of how to maintain good oral hygiene, dietary advice and the usage of fluoride releasing materials for bracket bonding and band cementation. When necessary, secondary prevention methods must be promptly applied, especially when white spot lesions are observed, and thus the patient's compliance can be reinforced through the operator's active intervention) (Benson et al., 2004).

C) Color alterations linked to the orthodontic treatment

Color alterations after orthodontic treatment are of multifactorial origin and some variables are directly linked to the technique itself. The frequency of these alterations is considerably higher, and increased in severity, when fixed appliances are used in comparison to removable ones. When resins are used for bracket bonding, enamel changes are unavoidable. The resin tags cannot be removed by cleaning procedures without significantly altering the enamel surface. Irreversible changes regarding enamel surface morphology include increased roughness and texture changes that negatively influence its reflective properties, luminosity and optical perception. Evidence shows that adhesive resins used for bracket bonding do not present good color stability over time. Food dyes, ultraviolet light and corrosion products from the orthodontic appliances induce color alterations, with a tendency to modify colors towards the yellow tones (Faltermeier et al., 2008).

In the presence of orthodontic forces that induce variation in pulp vascularization, it is also possible that endogenous discoloration appears, with a premature aging of the tooth. Additionally, if white spots lesions are present, even if remineralisation occurs, most probably the final outcome will be somehow different from the initial enamel structure. This is due to the fact that the new mineral is not identically composed as that of the unaffected enamel, with a possible influence on color properties. White spot lesions rarely progress to caries and generally are not registered as carious lesions requiring restorative treatment according to the DMFT/S indices (Gorelick et al., 1982).

After bracket removal, patients frequently wish to enhance their appearance by means of tooth whitening. This procedure presents particular problems, especially when resins were used as the bonding material, due to the remaining resins tags. The residual adhesive behaves differently to the adjacent enamel during whitening, which makes it important to

accurately evaluate the situation before whitening in order to avoid undesirable outcomes (Preoteasa et al., 2012).

D) Dental wear associated to the use of orthodontic appliances

Another potential dental alteration present in the orthodontic patient is tooth wear secondary to the contact between brackets or tubes to teeth of the opposing arch. An increased severity of this process has been noticed when ceramic brackets were used, with a severity from 9 to 38 times higher compared to metallic brackets (Lau et al., 2006; Viazis et al., 1990).

It is recommended, especially during certain phases of orthodontic treatment, to avoid the usage of ceramic brackets in order to minimize the irreversible treatment complication of dental wear. For example, when deep bite is present, ceramic brackets on the lower anterior teeth should not be used until sufficient overjet and overbite have been created in order not to induce wear of the maxillary incisors, a side effect with an increased negative impact on the aesthetics of the final result. Precautions must be taken when using ceramic attachments on the mandibular canines that are in a Class II relationship, and also during maxillary incisor retraction (Graber et al., 2004).

E) External apical root resorption in orthodontic therapy

The earliest reports of root resorption in permanent teeth, as cited by Ketcham (1927) were made by Bates in 1856, Chase in 1875, and Harding in 1878. The first study of apical root resorption associated with orthodontic procedures was reported by Ottolengui in 1914 (Papaconstantinou, 2004).

Root resorption due to orthodontic treatment can be categorized into three major groups according to Proffit (2000).

Severe localized root resorption is observed in 2-3% of orthodontic patients. Usually,

maxillary incisors are the affected teeth with loss of more than 1/4 of the root length and this can be related to orthodontic treatment. Another type is moderate generalized root resorption of 1-2 mm during treatment, something which is probably an inevitable consequence of orthodontic therapy affecting almost all teeth, and which is not accompanied by severe clinical consequences. The last category is severe generalized root resorption, which can happen in the absence of orthodontic treatment as often as in treated patients, and its exact etiology remains unknown. It may be related to some type of autoimmune response; it is not a subject of the orthodontists' responsibility and, as yet, there is no reliable method to predict or diagnose susceptible patients.

Apical root resorption is an unavoidable complication of orthodontic treatment and microscopic studies have shown a prevalence of almost 100% when assessed at the end of treatment. A systematic review and meta-analysis found a mean value of root shortening after orthodontic treatment of 1.421 +/- 0.448 mm (Segal et al., 2004).

The signs and symptoms of root resorption are usually absent and tooth mobility rarely reaches higher than 1st degree on the Miller scale. If the root resorption severity is mild or moderate at the end of the treatment, the tooth prognosis is not greatly compromised. It has been demonstrated that 4 mm of root shortening due to this pathological aspect is equivalent to 20%loss of the periodontal attachment, and 3 mm of loss is equivalent to 1 mm loss of the periodontal attachment (Kalkwarf et al., 1986).

In order to minimize the severity of root resorption, a good knowledge of its etiopathogenic mechanism is mandatory. Two main categories of factors have been incriminated for root resorption appearance, namely: related to patient characteristics, and to the orthodontic technique. Indicators of high risk patients may be the signs of root resorption prior to orthodontic therapy, and the presence of root resorption in first-degree relatives. Genetic factors play an important part in root resorption presence with the polymorphism of the IL-1beta gene being associated with severe root resorption (Lages et

al., 2009). Some studies suggest that this unwanted side effect varies between ethnic groups. There is a decreased frequency of root resorption in Asians compared to Caucasians or Hispanics (Lopatiene and Dumbravaite, 2008). Modified general health status has been linked to more severe root resorption process, and the associated diseases include allergies, asthma, diabetes, arthritis and endocrine disorders (Graber et al., 2004). A greater risk of root resorption is present in teeth with long and narrow roots, abnormal root shape in the apical part of the root, especially eroded, pointed, deviated or bottle shaped (Artun et al., 2009).

The most important risk factors of root resorption are related directly to orthodontic technique, treatment time, the amount of root apex displacement, the type and amount of orthodontic force, and also the type of orthodontic appliances used (Segal et al., 2004). Once orthodontic treatment has begun, an initial follow-up is recommended at 6 months which should include periapical radiographs of all maxillary and mandibular incisors, since these are the teeth most susceptible to root resorption. It is important that the root apices can be viewed on at least two radiographs taken from different directions. In order to standardize the assessment of root resorption, a four-point index can be applied. If there are no radiographic signs of root resorption at this stage of treatment, the risk of severe resorption at the end of therapy is minimal. Resorption at 6 months of treatment indicates a risk of progressive resorption as treatment proceeds. This risk may be reduced by a temporary suspension of active treatment for 2 to 3 months. In teeth showing progressive resorption, further radiographic follow-up every 3 months is recommended (Malmgren and Lavender, 2004).

A study by Cheng el at (2009) by using microcomputed tomography (micro-CT) to evaluate cementum repair at 4 and 8 weeks of retention after 4 weeks of continuous light and heavy orthodontic forces. The effects of age and tooth movement were investigated. They concluded that root resorption continued for 4 weeks after orthodontic force stopped.

Resorptive activity was more pronounced after the heavy forces. the reparative processes were different between the light and heavy forces, with marked individual variations. Repair seemed to become steady after 4 weeks of passive retention following 4 weeks of light force application, whereas most repair occurred after 4 weeks of passive retention following 4 weeks of heavy force application. Root resorption crater volume positively depended on tooth movement and negatively correlated with chronologic age.

Another histological study performed by the same author Cheng et al (2010), And they concluded that Root resorption cavities have the potential to repair regardless of the orthodontic force magnitude. less of the root resorption cavity seemed to be repaired by new cementum after heavy orthodontic force application and short retention time when compared with light orthodontic force with the same retention time; the reparative processes seem to depend on time, with a longer retention time yielding the most amount of repair.

A good knowledge of the variables associated with severe root resorption is essential for the identification of high-risk patients, as well as for the selection of the best suited treatment alternative in terms of minimizing the probability of root resorption occurrence.

F) Side effects related to tooth supporting tissues

One of the stated objectives of orthodontic treatment is to promote better oral health, so periodontal evaluation is considered an important requisite for the patient prior to orthodontic therapy. Periodontal problems can often be prevented by careful case planning and oral hygiene control (Preoteasa et al., 2012).

The complications related to the tooth supporting hard and soft tissues are the most actual side effects linked to orthodontics, and, often, constitute the basis of malpractice complaints (Preoteasa et al., 2012). The gingiva is the only dental

tissue visually accessible for direct clinical evaluation for the classic signs of inflammation. During fixed appliance orthodontic therapy, redness and swelling are commonly observed. Orthodontic appliances may act as a retention area for plaque. Plaque retention causes bacterial invasion and consequently, inflammation (Atack et al., 1996). In patients without orthodontic appliances, plaque levels range from 10.3% to 13.3%, depending on the population (White et al., 2006). In a study by Klukowska et al. (2011), plaque levels ranged from 5.1% of tooth surfaces to as high as 85.3% in patients undergoing treatment with fixed appliances. The mean plaque coverage in their study was 41.9 ±18.8%. If this plaque is not adequately removed, the ensuing gingival inflammation may lead to periodontal breakdown and, therefore, recession (Kassab and Cohen, 2008). A longitudinal clinical and microbiological study by Kipioti el al (1987) concluded that the use of bonded tubes rather than bands on molars and premolars may be more appropriate in eliminating unwanted stagnation areas. Plaque retention is increased with fixed appliances and plaque composition may also be altered. There is an increase in periopathogenic anaerobic organisms and a reduction in facultative anaerobes around bands. Thus, bands may cause more gingival inflammation than bonded tubes, which is not surprising since the margins of bands are often seated subgingivally.

Orthodontic side effects related to tooth supporting tissues can be manifested in various forms, from gingivitis to periodontitis, dehiscences, fenestrations, interdental fold, gingival recession or overgrowth, and black triangles. Severe damage can significantly interfere with teeth prognosis. Etiopathogenesis is complex and involves factors related to the individual patient (e.g., previous conditions present, increased susceptibility, poor oral hygiene) and the orthodontic technique. Gingivitis usually occurs due to the incorrect

maintenance of oral hygiene and the presence of the orthodontic appliances favours plaque accumulation. Research has shown that during orthodontic therapy, gingival enlargement occurs, but approximately 3 months after the removal of the appliance, in most cases, the gingiva presents a similar aspect as before treatment (Kouraki et al., 2005).

During initial assessment, patients with factors predisposing to a worsening of the periodontal condition (e.g., presence of diabetes or epilepsy treated with drugs that induce gingival enlargement) need to be identified. During orthodontic therapy, it is recommended to insist on the importance of maintaining good oral hygiene, to monitor the periodontal status (at least every three months to perform an examination and dental cleaning) and to take the necessary measures in order to control the risk factors (Preoteasa et al., 2012).

The literature suggests that orthodontic treatment does not affect the periodontal status of patients in the long term. However, Zachrisson and Zachrisson (1972) reported that even after maintaining excellent oral hygiene, patients usually experienced mild to moderate gingivitis within the 1-2 months after appliance placement, but that the gingival changes were transient and no permanent damage to the periodontal tissues could be demonstrated. A retrospective study by Boke el al. (2014) confirmed that visible plaque and gingival inflammation values increased between the start and the end of treatment in all patients in their study sample, and their results showed that, regardless of the quality of plaque control, most subjects undergoing fixed orthodontic treatment developed generalized gingivitis within a short time. These increases were statistically significant in patients treated with fixed orthodontic appliances.

Kloehn and Pfeifer (1974) studied the effect of orthodontic treatment on the periodontium, concluding that inflammatory and hyperplastic changes in the

gingiva, occurring during the treatment, were reversible upon appliance removal and that the periodontium was in better health following treatment. Overall, orthodontic treatment did not cause any irreversible periodontal destruction and a direct relation between oral hygiene and periodontal disease was noted. Moreover, it has been suggested that orthodontic treatment does not aggravate the status of periodontally compromised dentitions (Zasciurinskiene, 2016).

In order to test the effects of orthodontic tooth movement on reduced periodontium, several experimental animal studies have been conducted. Ericsson et al (1977) studied orthodontic tooth movement in dogs and concluded that healthy and inflamed periodontal tissues react differently. The movement of teeth in reduced but healthy periodontium did not cause additional attachment loss (Ericsson et al., 1978). In addition, mesial movement into infrabony defects in rats (Vardimon et al., 2001; Nemcovsky et al.,2007) and intrusion movement in monkeys (Melsen, 2001), and extrusion movement in dogs (van Venrooy and Yukna, 1985), were all performed without additional loss of periodontal support, provided oral hygiene was maintained. In contrast, orthodontic movement of teeth with inflamed infrabony pockets was found to increase loss of connective tissue attachment (Ericsson et al., 1977; Wennstrom et al., 1987; Melsen, 2001). Findings in animal studies with experimentally induced periodontal disease cannot be easily extrapolated to human conditions because natural periodontal destruction is unknown in monkeys and it only occurs in much older dogs than those used in the studies. Attachment loss in humans occurs relatively slowly over a much longer time (Harrel et al., 2006), and underlying modifying host responses possibly influence it. Finally, orthodontic tooth movement has been implicated as a possible etiological factor

for apical soft tissue relocation and gingival recession (Joss-Vassalli et al., 2010). As teeth are moved to new positions, the spatial relationships of the soft and underlying osseous tissues are altered to accommodate changes in aesthetics and function. Thus, it seems likely that, under certain circumstances, teeth acquire unfavourable positions relative to their supporting structures, which could possibly lead to apical soft tissue relocation, an increase in clinical crown length and eventually recession formation (Wennstrom et al., 1987).

G) Soft tissue alterations

During orthodontic treatment, intra- and extraoral (face and neck) soft tissue alterations may appear. For the oral lesions, the etiological mechanism involves the direct contact of gingiva and mucosa with brackets, bands, tubes and arches, and is also related to the incorrect handling of orthodontic instruments. The outcome usually involves erosions and ulcerations on the buccal, labial, lingual or gingival mucosa. Pain and discomfort are associated, but by using orthodontic wax it may be possible to alleviate, to some extent, the symptoms. Improper hygiene in the removable orthodontic appliances is sometimes associated with stomatitis due to Candida Albicans (Shah and Sandler, 2006). Headgear appliance was linked to facial and intraoral trauma as an effect of an accident during playing, sleeping or due to incorrect handling. The consequences were dramatic and ranged from impairment of vision to hand movement perception or less severe issues (Hareuveni et al., 2004). After several cases of trauma associated with headgear devices were reported, modifications of its design were made by the industry in order to prevent severe complications (Hareuveni et al., 2006).

H) Temporomandibular disorders

It has been claimed that the appearance of signs and symptoms of temporomandibular disorders involving TMJ modifications, as well as muscle and dental impairments, may be associated with orthodontic therapy. Current scientific evidence does not clearly elucidate any relation between temporomandibular alterations and orthodontic intervention, with studies reporting contradictory and varying results. Some studies claim that the state of morphofunctional equilibrium present after orthodontic intervention provides the optimal conditions for preventing TMDs. Others believe that the premature occlusal contacts present during therapy are a significant risk for the development of TMDs (Bourzgui et al., 2010).

Before starting orthodontic treatment, every patient must be examined in order to detect previous temporomandibular disorders and identify the high risk individuals. Aspects like inflammatory bone and muscular disorders (rheumatoid arthritis), head and neck trauma, chronic head pain or high stress level must be taken into account. If signs and symptoms of temporomandibular disorders are present, reaching a diagnosis and establishing their degree of severity is mandatory. It is not recommended to start orthodontic therapy if the patient presents acute or severe signs of pain due to temporomandibular dysfunction. If severe symptoms are observed during treatment, depending on case particularities, it might be decided to refer the patient for the management of the symptoms or, in extreme cases, to discontinue orthodontic therapy (Athanasiou and Graber, 2004).

2.2.2. Systemic side effects

A) Allergic reactions

Orthodontic treatment has been linked to allergic reactions. Hypersensitivity reactions can occur due to well-known allergens such as nickel, cobalt, chromium, latex and polymers. The most frequent form is contact dermatitis of the face and neck, but lesions can also appear on the oral mucosa and gingiva, and even, although rarely, systemic reactions may occur. Nickel allergies are the most frequent ones in the industrialized countries, usually manifesting as a type IV hypersensitivity reaction. Orthodontists are often required to treat patients with super-elastic nickel-titanium (Ni-Ti) archwires during the initial phase of orthodontic treatment. These archwires contain about 47-50% nickel (Eliades and Athanasiou, 2002). Stainless steel archwires also contain about 8% nickel, which is also present in bands and metallic brackets. The allergic signs may vary from limited appearance of a rash on skin or mucosa to generalized dermatitis. In high severity cases, the manifestations may necessitate the discontinuation of orthodontic treatment. Another allergen to be taken into consideration in the process of orthodontic treatment is the latex contained in medical gloves, elastomeric ligatures, elastic chain, rubber dams and other materials or devices. The prevalence of latex related allergies is reported at percentages lower than 1% in the general population but greater than 5% among dental professionals (Leite, 2004). Furthermore, Kolokitha et al. (2008) conducted a meta-analysis regarding the prevalence of nickel hypersensitivity and found that orthodontic treatment is not associated with an increase in the prevalence of nickel hypersensitivity unless subjects have a history of cutaneous piercing associated.

Types I and IV hypersensitivity reactions may appear, and the most severe type I can be life threatening. In order to ensure safe medical treatment, it is important to identify allergic patients before starting any intervention. Patients with higher risk have a previous history of complex or repeated surgical interventions (prolonged contact with rubber

drains and tubes), those with spina bifida, and those who report itching and redness from contact with rubber objects and allergies or contact dermatitis. A definitive diagnosis is established by combining the anamnestic data with the clinical data and hypersensitivity tests. When allergic reaction to latex is identified, alternative latex-free devices should be used, and it is also recommended avoiding nickel-based components (Kolokitha et al., 2008).

B) Infective endocarditis

Infective endocarditis is rarely associated with orthodontic interventions, but if it does, it can present severe complications that are potentially life threatening. The American Heart Association recommends prophylactic methods in order to prevent infectious endocarditis in patients with a prosthetic cardiac valve, previous infective endocarditis, congenital heart disease and cardiac transplantation with cardiac valvulopathy. The prophylaxis is mainly indicated in dental procedures concerning the oral and maxillofacial surgery, endodontics and periodontics spectrum, while in routine orthodontic procedures it is not mandatory. Prophylactic therapy may be indicated in some particular orthodontic phases where bleeding is expected during interventions such as tooth extraction, mini-implant placement used for anchorage control, interventions of orthognathic surgery and sometimes during placement and removal of orthodontic bands (Wilson et al.,2007).

In conclusion, the risks associated with orthodontic treatment are a reality. The complications are a result of various etiologic factors, including aspects related to the individual patient, the orthodontist and the technical features of orthodontic appliances and procedures. These can be prevented or limited through identification and implementation of best treatment alternative for each individual case. Patient compliance is an important factor that can contribute to a high standard of outcome, with minimal side effects (Preoteasa et al., 2012).

2.3. Gingival recession

Gingival recession is defined by the American Academy of Periodontology 2017 as "the migration of the marginal soft tissue to a point apical to the cemento-enamel junction of a tooth or the platform of a dental implant".

In order to better understand recession, a review of the soft tissue attachment apparatus is necessary. In a buccal-lingual cross section, the gingival epithelium forms a crevice around the tooth. On the tooth side, the gingival epithelium is termed the sulcular epithelium, which, along with the tooth, forms the boundaries of the gingival sulcus. The area apical to the unattached sulcular epithelium, termed the junctional epithelium, forms an epithelial attachment to the tooth surface itself. This epithelial attachment is the most coronal portion of the periodontal attachment apparatus, and provides apical resistance when a periodontal probe is inserted into the sulcus. When healthy, the level of the epithelial attachment to the tooth is usually at, or slightly coronal, to the level of the cementoenamel junction (CEJ). Immediately apical to the epithelial attachment, densely packed collagen bundles are anchored into the cementum, forming the connective tissue attachment. Apical to the connective tissue attachment is the periodontal ligament (PDL). Therefore, the element of space that healthy gingival tissues occupy between the sulcular base and the underlying alveolar bone is comprised of the junctional epithelial attachment and the connective tissue attachment. The combined attachment width is identified as the biologic width. Garguilo et al. (1961), found that, in the average human, the connective tissue attachment measures 1.07 mm, and the junctional epithelial attachment measures 0.97 mm.

In clinically healthy gingiva, the free gingiva is in close contact with the enamel surface, and its margins located 0.5 to 2 mm coronal to the CEJ after the completion of tooth

eruption. The attached gingiva is firmly attached to the underlying alveolar bone and root cement by connective tissue fibres and is, therefore, comparatively immobile in relation to the underlying tissue (Thilander, 2004).

The muco-gingival complex consists of the free and attached gingiva, the muco-gingival junction and the alveolar mucosa. An adequate mucogingival complex, in which the mucogingival tissues can sustain their biomorphologic integrity and maintain an enduring attachment to the teeth together with the underlying soft tissue, is always essential for the harmony of the smile. When a mucogingival problem occurs, there are basically two ways in which it presents itself: either as a close disruption of the mucogingival complex resulting in pocket formation, or as an open disruption resulting in gingival clefts and recession (Dodwad, 2001). In addition, gingival recession may negatively affect the appearance of the dentition, often causing esthetic impairment and can increase the susceptibility to dentine hypersensitivity, root caries, cervical wear, and erosion because of exposure of the root surface to the oral environment and an increase in the accumulation of dental plaque (Susin et al., 2004). Although many dental conditions go unnoticed by patients, gingival recession may often be detected by patients, leading them to seek dental advice (Mathur et al., 2009). When such a problem affects the anterior teeth, the esthetic problem is amplified, and anxiety about tooth loss due to progression of the destruction may appear (Löe et al.,1992).

2.3.1. Epidemiology of gingival recession

Initial observations suggested that recession might be a physiological process related to aging (Woofter, 1969). Indeed, population-based studies have shown that the development of gingival recessions is correlated with age. The prevalence is lower at younger ages and increases over time (Ainamo et al., 1986; Susin et al., 2004). As there is some doubt regarding diagnosis of gingival recession before 12 years of age with certainty (Volchansky and Cleaton, 1976), it is believed that apparent recession in younger children is due to a delay in the maturation of the gingivae of the adjacent paired tooth, rather than to true recession of the gingivae of the apparently affected tooth. However, convincing evidence for a physiological shift of the gingival attachment has never been presented (Leo, 1967).

In a sample of 299 children and teenagers, Ainamo et al. (1986) showed that at least one gingival recession of 0.5 mm or more, measured from the CEJ to the gingival margin was present in 5% of 7-year-olds, 39% of 12-year-olds, and 74% of 17-year-olds. Susin et al. (2004) examined 1,586 individuals aged 14 years and older. Measuring recession clinically from the CEJ to the gingival margin, they demonstrated that recessions of more than 3 mm were present in 6%, 24%, and 54% of patients aged 14 to 19, 20 to 29, and 30 to 39 years, respectively. Albander and Kingman (1999) used a sample of 9,689 persons from the data collected in the third National Health and Nutrition Examination Survey (NHANES III) and demonstrated that the prevalence, extent and severity of gingival recession increased in individuals aged 30 and above. In the same study, 56% of individuals aged 40-49, 71% of individuals 50-59, 80% of individuals aged 60-69, 87% of individuals 70-79, and 90% of individuals aged 80-90 showed 1 mm or more of gingival recession on at least 1 tooth.

Some teeth exhibit a significantly higher prevalence of gingival recession than others. From the NHANES III data (Albander and Kingman, 1999) it was noted that in the maxillary arch, the two teeth that presented with gingival recession most frequently were the first premolars and first molars. In patients aged 30-55 years, 20.3% of first maxillary premolars showed recession on the labial surface, compared to 21.1% of maxillary first molars. The same pattern was noted in the maxillary arch for patients aged 56-90 years. For this group, 39.5% of first premolars exhibited recession, and 48.4% of first molars showed recession. In the mandibular arch, recessions were most prevalent in the central incisors and first premolars. Approximately 19.7% of central incisors and 19.5% of first premolars showed recession in the 30-55 year old group. The lateral incisor and second premolar were the next highest, exhibiting 13.2% and 14.5%, respectively. For the 56-90-year-old group, 49.7% of central incisors and 43.5% of first premolars were affected. The lateral incisor was the next highest in this group, with 42% of teeth showing recession.

Recession also appears to be much more prevalent on the facial surface when compared to the lingual surface of teeth. In the study by Ainamo et al. (1986), gingival recession was measured on the facial and lingual surfaces of 299 Finish school children aged 7-17 years. Of the 5,895 teeth examined, recession was found on the facial aspect of 512 teeth (8.7%) and on the lingual aspect of only 16 teeth (0.3%). Data from a long-term epidemiological study by Loe et al. (1992) showed increased prevalence of recession on the facial surface as opposed to the lingual surface. It was also demonstrated that lingual recession seems to appear later in life. In the same study, the prevalence of gingival recession in two cohorts of individuals who were receiving regular dental care with a reasonable level of oral hygiene was investigated. The two cohorts were

from Norway (1969-1988) and Sri Lanka (1970-1990), covering the age range from 15 to 50 years. In the Norwegian cohort, gingival recession began early in life. It occurred in greater than 60% of the 20-year-olds and was primarily found on the facial surfaces. At 30 years of age, 70% had recession, mainly on the facial surfaces. As the group approached 50 years of age, more than 90% of individuals had gingival recession. Twenty-five percent of the facial surfaces, 15% of the lingual surfaces, and 3% of the interproximal surfaces were involved. In the Sri Lankan cohort, 30% showed recession before 20 years of age. By 30 years, 90% had recession on facial, lingual, and interproximal surfaces. At 40 years, 100% of the Sri Lankans cohort exhibited gingival recession. As they approached 50 years, recession occurred on 70% of the facial, 50% of the lingual, and 40% of the interproximal surfaces. However, the occurrence of gingival recessions was significantly higher in a population without any dental care. Additional demographic features that appear to be related to gingival recession include sex, ethnicity, and socio-economic status. Using the NHANES III data, Albander and Kingman (1999) demonstrated that males aged 30 or more had significantly more recession than females of the same age. The results of Susin et al. (2004) corroborate these findings. In their study, males consistently exhibited a higher prevalence and extent of gingival recession than females. However, in subjects younger than 30, their sample showed no significant gender differences. Ainamo et al. (1986) reported similar results. In their sample, no significant gender differences were noted among 17-year olds. Therefore, it appears that there are no gender differences at the younger ages, but, with aging, males exhibit more recession than females. The NHANES III data also indicated that of the three racial/ethnic groups studied, non-Hispanic blacks had the highest prevalence and extent of gingival recession

(Albandar and Kingman, 1999). Another investigation showed that the percentage of teeth with recession was significantly higher in the lower socioeconomic groups, irrespective of age (Susin et al., 2004).

Mandibular incisors have been shown to be the teeth most prone to adverse gingival changes (Khocht el at., 1993). Recession localized in these teeth might be related to their path of eruption and the buccolingual thickness of the alveolar process. Specifically, a mandibular anterior tooth that erupts in labioversion, possessing a thin and high symphysis, is most likely to develop gingival recession (Artun and Krogstad, 1987). Ruf et al. (1998) observed that 13% of their sample of 98 patients with Class II malocclusion had recession of the mandibular incisors prior to Herbst treatment.

2.3.2. Etiology of gingival recession

The etiology of gingival recession is considered to be multifactorial. Several factors may play roles in the development of gingival recession, but not necessarily simultaneously or equally (Kassab and Cohen, 2003), including gingival inflammation and periodontal disease, faulty tooth-brushing technique (Kassab and Cohen, 2003; Loe et al., 1992) and muscle fibre pull (Sognnaes, 1977). Iatrogenic damage to the delicate periodontal tissues caused by applying force vectors in the extreme vestibular direction, tongue thrusting, trauma from occlusion and fronto-lateral bruxism have been associated with the initiation and ⁄ or progression of gingival recessions (Abboud et al., 2002). Trauma from occlusion has been suggested in the past, but its mechanism of action has never been demonstrated (Newman, 2012). A relationship may also exist between smoking and gingival recession (Gunsolley et al., 1998), due to the reduction in gingival blood flow (Preber and Bergstrom, 1985). The multifactorial

mechanism may also include changes in the immune response, such as decreases in the phagocytic function of polymorphonuclear leukocytes and a reduction in the production of immunoglobulins (Johnson et al., 1990). Gingival trauma and gingival abrasion from tooth brushing are thought to play an integral part in the etiology of recession. Toothbrush abrasion also may cause wear at the CEJ resulting in the destruction of the supporting periodontium (Litonjua et al., 2003). The relationship of tooth brushing to recession also depends on the population under study. In populations with little access to care, recession is associated with poor oral hygiene and calculus deposits (van Palenstein Helderman et al., 1998), whereas in populations with a high use of dental services, recession is primarily associated with excessive or forceful tooth brushing (Loe et al.,1992).

Specific anatomical attributes may also place an individual at an increased risk of recession. Susceptibility to recession may be influenced by the position of the teeth in the arch, the root-bone angle, and the mesiodistal curvature of the tooth surface (Trott and Love, 1966). There is also evidence suggesting that the quantity of the hard and soft tissues adjacent to the teeth may play an important role in the development of recession, with the thicker gingival tissue biotype imparting some resistance to recession, while the thin gingival tissue biotype is more prone to recession (Melsen and Allais, 2005)

Many authors have hypothesized that on rotated, tilted, or facially displaced teeth, the bony plate is thinned or reduced in height. Subsequent pressure from mastication or aggressive tooth brushing damages the unsupported gingiva and may produce recession (Gorman,1967). This hypothesis seems to be supported by the literature. To determine the prevalence of gingival recession, as well as the etiologic factors associated to it, Parfitt and Mjor (1964) examined 668

school children aged 9 to 12 years and found that 8% of this group had 2-5mm of facial gingival recession associated with the mandibular incisors. A tooth-size arch-length discrepancy was found to be the most commonly associated factor with gingival recession. Actually, the authors stated that 80% of the affected teeth exhibited such a discrepancy. Trott and Love (1966) investigated a group of 766 high school students aged 14-19 in a similar study. The facial surfaces of the mandibular incisors were examined for recession. Factors most associated with the recession were also studied. Of the teeth examined, 1.8% were reported to have recession greater than 3 mm. Tooth malposition was the factor most commonly associated with recession. Gorman (1967) examined 164 subjects aged 16-86 years for recession. In teeth in pronounced labioversion, 61% were found to have some degree of gingival recession, as well as 15% of teeth in pronounced linguoversion. Again, malposition of the teeth was the variable most frequently associated with recession. The above studies have shown that gingival recessions are often found at tooth surfaces where alveolar bone dehiscences are also present. In other words, a root dehiscence may provide an environment which may lead to the loss of gingival tissue. If this assumption is correct, it would imply that as long as orthodontic tooth movement is performed entirely within the alveolar bone, no apical shift of the gingival margin is likely to take place. However, if teeth are moved out of the alveolar envelope and a dehiscence is formed, there is a risk that gingival recession may occur (Wennstrom et al., 1987). Orthodontic treatment may promote development of recession (Bollen et al., 2008; Dorfman, 1978), but the mechanism by which orthodontic treatment influences the occurrence of recessions remains unclear. In summary, treatment duration, treatment type, the skeletal or dental relationship, age, gender or race did not have an influence on

the development of recessions during treatment. The presence of gingival inflammation and baseline recession, a thin gingival biotype, a narrow width of keratinized gingiva or a thin symphysis were all found to correlate significantly with the development, or increase in, gingival recession (Joss-Vassalli et al., 2010).

2.3.3. Consequences of recession

Gingival recession can negatively affect the appearance of the dentition, often causing esthetic impairment, and may increase the susceptibility for dentine hypersensitivity, root caries, cervical wear, and erosion because of exposure of the root surface to the oral environment and an increase in the accumulation of dental plaque (Susin et al., 2004). The results of the long-term follow up studies indicate that time may be a critical factor in the development of gingival recessions following orthodontic treatment. Animal studies, through histologic analysis, have demonstrated a conservation of the soft tissue attachment up to 5 months after tooth movement was ended despite the formation of a bone dehiscence. The result of this process was the development a long epithelial attachment (Karring et al., 1982; Wennestrom et al., 1987).

There is a need in the orthodontic literature to further evaluate the long-term prevalence of gingival recession following orthodontic tooth movement. There are only three studies (Yared et al., 2006, Artun and Grobety, 2001, Artun and Krogstad, 1987) that focus on this subject, and they offer conflicting results. Although it is reasonable to believe that a similar situation occurs in humans, the actual process that occurs is unknown, because findings from animal studies cannot be easily extrapolated to humans. Moreover, the relationship of alveolar dehiscences and recession in the long term is also unknown, since the histologic analysis in the animal studies were completed only 3 weeks to 5 months after treatment.

2.3.4. Orthodontic treatment and gingival recession

The periodontal tissues adapt to teeth that are moved orthodontically along the dental arch. However, tissue adaptability may be reduced when teeth are moved into extreme positions. Thus, orthodontic treatment may contribute to improving the periodontal health status but is sometimes associated with periodontal complications**.**

Current information suggests that there is a potential loss of tooth supporting structures during orthodontic treatment, expressed as crestal bone loss, gingival recession and sometimes root resorption. On the other hand, orthodontic therapy is occasionally used to treat pre-existing infrabony defects, and suggestions have been made that orthodontic therapy may be applicable in the treatment of mucogingival problems (Coatoam et al., 1981). In cases where minimal attached gingiva is present, even the slightest loss of this tissue might result in a mucogingival problem, Furthermore, mucogingival problems noted after orthodontic therapy are often the result of a pre-existing condition. There are conflicting opinions as to whether the problem is better managed before or after orthodontic therapy. This uncertainty is due, in part, to the lack of controlled longitudinal investigations evaluating the effect of orthodontic therapy on the width of keratinized gingiva (Coatoam et al., 1981). Alterations in the mucogingival complex will occur during orthodontic tooth movement, but these are independent of the apico-coronal width (height) of the gingiva (Wennström, 1996). In the same study, it was also found that the integrity of the periodontium can also be maintained during orthodontic therapy in areas that have only a minimal zone of gingival; but the important factors to consider are the direction of tooth movement and the bucco-lingual thickness of the gingiva**.** In an animal experiment carried out on dogs, it was shown that dehiscences or fenestrations can be produced in the buccal alveolar plate by moving teeth in a facial direction and that the bone will reform when the teeth are moved back to their original positions. It was also demonstrated that such tooth movements are not necessarily accompanied by loss of

connective tissue attachment (Thilander et al., 1983). However, results of animal experimental studies cannot be applied directly to humans. The concept of 'envelope of discrepancy' presents zones in three planes of space beyond which the above-mentioned morphological side-effects are likely to occur (Proffit and White, 1991; Sarver and Proffit, 2005). It has been suggested that transverse expansion and/or labial proclination of the teeth are valid alternatives to extraction in cases of crowding. However, the labial development of bone dehiscences has been demonstrated as a side effect subsequent to excessive displacement of dental units (Allais and Melsen, 2003).

The influence of orthodontic therapy on periodontal tissue has been studied extensively but with very debatable outcomes (Dannan, 2010). Some articles showed an association between incisor labial proclination and the development of recessions (Slutzkey and Levin, 2008; Dorfman, 1978; Boke et al.,2017)**.** In a cross-sectional study design, Slutzkey and Levin (2008) found that the prevalence and severity of recession was worse in orthodontically treated patients when compared to patients who had not received orthodontic treatment, and that tooth movement outside the labial or lingual alveolar plate can lead to dehiscence and gingival recession. Similarly, in another study, it became apparent that in a small percentage of cases, visible mucogingival changes occurred and could be statistically correlated with the magnitude and direction of tooth movement (Dorfman, 1978). It was concluded that more proclined teeth had a higher occurrence, or severity, of gingival recession compared with less proclined or untreated teeth. However, the differences were small and the clinical consequences questionable. In addition, a retrospective study by Boke et al. (2017) found a positive correlation between lower incisor retraction and gingival recession which showed the cuspids were the teeth with the highest prevalence of gingival recession. Within the limits of research design, orthodontic treatment and/or the retention phase may be risk factors for the development of labial gingival recessions. In orthodontically treated subjects, mandibular incisors seem to be the

most vulnerable teeth for the development of gingival recessions (Renkema et al., 2013a). On the other hand, some authors have claimed no association between orthodontic tooth movement and clinical crown length, gingival recession, or attachment loss. Several reports have downgraded the influence of orthodontic treatment to the periodontal health of incisors (Kloehn and Pfeifer,1974; Vasconcelos et al., 2012; Ruf et al., 1998; Artun and Grobety, 2001; Renkema et al., 2013b; Allais and Melsen, 2003; Melsen and Alais, 2005; Morris et al., 2017; Kamak et al.,2015).

Allais and Melsen (2003) showed that controlled proclination under maintenance of good oral hygiene can be carried out in most patients without risk to the periodontium. A recent study by Morris et al. (2017) also concluded that there was no relationship between the amount of mandibular incisor proclination during treatment and the amount of gingival recession, either during,or after treatment. Similarly, Kamak et al. (2015) studied retrospectively the effect of changes in lower incisor inclination on gingival recession and also concluded that the change of inclination of lower incisors during orthodontic treatment did not lead to development of labial recessions. In addition, Vasconcelos et al., (2012) noted that vestibular gingival recession of mandibular incisors after orthodontic treatment is of minor prevalence and severity. Ruf et al., (1998) concluded that no interrelation was found between the development of gingival recession and the orthodontic proclination of lower incisors in children and adolescents when treated with the Herbst appliance. Kloehn and Pfeifer (1974) claimed that orthodontic treatment did not cause any irreversible periodontal destruction and changes in the lengths of clinical crowns during treatment was of no statistical significance. Renkema et al., (2013b) agreed with all the previous studies and concluded that the change of inclination of lower incisors during orthodontic treatment did not produce labial recession. Another study conducted by the same authors included re-assessment of the same sample 5 years after post-orthodontic treatment and concluded that the proclination of mandibular incisors did not increase the

risk of development of gingival recession in comparison to non-proclined teeth (Renkema et al., 2015). In addition, Artun and Grobety (2001) highlighted that pronounced advancement of the mandibular incisors may be performed in adolescent patients with dentoalveolar retrusion without increasing the risk of recession. It has also been suggested in a study conducted by Yared et al. (2006) that more proclination during treatment may be accepted for a low initial inclination than for a high initial inclination. Indeed, they showed that a final lower incisor inclination of more than 95° in relation to the mandibular plane was directly related to more frequent and more severe recession in the mandibular central incisors; the amount of proclination was not important but the final inclination was. Future studies should consider both the amount of proclination during treatment and the final inclination. Nevertheless, when comparing the thickness to the final inclination, the periodontal characteristics had greater relevance to recession.

Recent systematic reviews (Joss-Vassalli et al., 2010; Aziz and Flores-Mir, 2011) did not show significant relation between pronounced labial movement of incisors during orthodontic treatment and gingival recession. Factors that may lead to gingival recession after orthodontic tipping and/or translation movement were identified and included reduced thickness of the free gingival margin, narrow mandibular symphysis, inadequate plaque control and aggressive tooth brushing. However, the precise mechanism by which orthodontic treatment influences the occurrence of recessions remains unclear. Other factors might play roles in the development of gingival recession, but not necessarily simultaneously or equally (Slutzkey and Levin, 2008).

A comprehensive review of the evidence-based literature in the fields of periodontics and orthodontics noted that proper orthodontic treatment in patients with excellent oral hygiene in the absence of significant periodontal disease should not pose any significant periodontal risk (Sandler, 1999). Furthermore, the above-mentioned contrasting findings could be the result of methodological issues in study design, such as the moment of

assessment (immediately after treatment versus long-term) or sample composition (subjects with a given type of malocclusion versus subjects with various types of malocllusion) (Pandis et al.,2007).

Further clinical studies are needed to clarify the effect of orthodontic changes in incisor inclination and the occurrence of gingival recession in order to prevent harmful side effects.

3. AIM

3.1. Aim of the thesis

To retrospectively investigate the clinical crown length changes and the development of gingival recession in the labial aspect of the maxillary and mandibular incisors associated with orthodontic treatment, and relate them to the observed changes in their sagittal inclination.

3.2. Objectives of the thesis

a. To quantify the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment and relate them to the observed changes in their sagittal inclination.

b. To measure of gingival recession in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment and relate them to the observed changes in their sagittal inclination.

3.3. Null hypotheses

a. There is no association between the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment and the observed changes in their sagittal inclination.

b. There is no difference in the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment between the groups of patients whose incisors underwent proclination, retroclination or remained stable during treatment.

c. There is no difference in the presence of gingival recession in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment.

d. There is no difference in the presence of gingival recession in the labial aspect of the maxillary and mandibular incisors after orthodontic treatment between the groups of patients whose incisors underwent proclination, retroclination or remained stable during treatment.

4.MATERIALS AND METHODS

The present retrospective investigation constituted an observational before and after study, where a sample of orthodontically treated subjects was assessed before the initiation, and after the completion, of orthodontic treatment.

Ethics approvals were obtained from the Medical Faculty Ethics Committee of the University of Mohammed Bin Rashid University of Medical and Health Sciences, Ethical reference number: EC1016-004.

4.1. Subjects

The archive of patients who had received comprehensive orthodontic treatment by means of fixed appliances in both dental arches by two orthodontic specialists in a private orthodontic clinic in Dubai, United Arab Emirates, was searched. The files on all subjects contained a complete medical and dental history, intra- and extra-oral photographs, panoramic X-rays, lateral cephalometric radiographs, and study models, which were taken as part of routine diagnostic record procedures. The study models and lateral

cephalometric radiographs, before orthodontic treatment (T1) and after orthodontic treatment (T2), were used for this investigation. Sample size analysis was not performed before the beginning of the study, but all eligible subjects were included in the investigation.

The subjects included in the current study satisfied the following criteria:

Inclusion criteria

1. Medically fit.
2. Adolescent and adult patients.
3. Comprehensive orthodontic treatment with fixed appliances in both dental arches.
4. All mandibular incisors fully erupted before treatment.
5. No incisors extracted during treatment.
6. No generalized visible wear of incisal edges occurring during the course of treatment.
7. No previous orthodontic treatment.
8. Study models with an excellent quality of incisor morphology and gingival configuration available before treatment (T1) and after treatment (T2).
9. Lateral cephalometric radiographs of excellent quality available before treatment (T1) and after treatment (T2).
10. Information about treatment duration.

Exclusion criteria

1. Combined orthodontic/surgical treatment.
2. Change in incisor crown morphology.
3. Study models of poor quality.

Finally, eighty-two consecutive subjects, treated by means of fixed appliances in both arches and with good quality pre- and post-treatment dental casts and lateral cephalograms satisfied the inclusion criteria.

4.2. Methods

4.2.1. Measurements on digitized dental casts

The dental casts were scanned and digitized blindly by means of the Ortho Insight 3D® Scanner (Motion View Software LLC, Chattanooga, Tennessee, USA).

The clinical crown length of all maxillary and mandibular incisors was measured blindly by one investigator (BS) on the digitized pre- and post-treatment study models from the incisal edges of the incisors to the deepest point in the curvature of the gingival margin (Renkema et al., 2013). The measurements were made with the software installed in the Ortho Insight 3D® Scanner.

The same investigator assessed blindly the presence or absence of gingival recession in all maxillary and mandibular incisors on the digitized pre- and post-treatment study models. Labial gingival recession was scored as "present" when the labial CEJ was exposed (Renkema et al., 2013).

Individual teeth with visible wear of the incisal edges which occurred during the course of treatment, or with poor gingival margin anatomy were excluded from further consideration and analysis. From the cases where all maxillary or mandibular incisors had been assessed, the prevalence rate of gingival recession in at least one maxillary or mandibular incisor was calculated.

4.2.2. Validity of digital measurements

The validity of measuring clinical crown lengths and identifying the presence of recession on digital dental casts was investigated in a pilot study.

Thirty casts were selected at random. The investigator responsible for the cast measurements measured blindly the original plaster casts with an electronic caliper [Accurate to 0.0005inch (0.01mm)] the clinical crown length of all maxillary and mandibular incisors from the incisal edges of the incisors to the deepest point in the curvature of the gingival margin and identified the presence of gingival recession when the labial CEJ was exposed (Renkema et al., 2013). The same measurements were repeated after scanning and digitizing of dental casts by means of the Ortho Insight 3D® Scanner and the installed software. After one month, the same procedure was repeated.

4.2.3. Measurements on lateral cephalometric radiographs

Pre- and post-treatment lateral cephalometric radiographs were digitized blindly by one investigator (SA) and used to measure maxillary and mandibular incisor inclination by means of the Viewbox software (dHal, Kifissia, Greece).

In order to assess the maxillary incisor inclination, the following variable was measured:

Maxillary incisor inclination (U1/NL): the angle between the line passing through the long axis of the maxillary incisor (incisal edge-incisor apex) and the line at the connecting the ANS and PNS points (Riolo, 1974).

According to the changes observed in maxillary incisor inclination before (T1) and after orthodontic treatment (T2), subjects were categorized into three groups; a. Retroclined group (ΔU1/NL<1^o); b. Stable group (ΔU1/NL$\pm$1^o), and c. Proclined group (ΔU1/NL>1^o).

In order to assess the mandibular incisor inclination, the following variable was measured:

Mandibular incisor inclination (L1/MP): the angle between the line passing through the long axis of the mandibular incisor (incisal edge-incisor apex) and the line at the lower border of the mandible tangent to the gonion angle and profile image of the symphysis (Downs, 1948).

According to the changes observed in mandibular incisor inclination before (T1) and after orthodontic treatment (T2) subjects were categorized into three groups; a. Retroclined group (ΔL1/MP<1^o); b. Stable group (ΔL1/MP±1^o), and c. Proclined group (ΔL1/MP>1^o).

4.3. Method error

To determine the intraobserver reliability of the measurements, 30 pre-treatment and post-treatment casts and lateral cephalometric radiographs were randomly selected and blindly measured by the respective investigators and re-evaluated after one month.

To assess possible systematic errors in the measurements of clinical crown lengths and inclination of the incisors, intraclass correlation coefficients and Wilcoxon tests were applied between both series of measurements. Furthermore, random errors were calculated by the Dahlberg formula (Dahlberg, 1940).

$$s(i) = \sqrt{\frac{\Sigma(X_a - X_b)^2}{2N}}$$

To assess possible errors in the strength of agreement for scoring the presence of recessions between both series of measurements the kappa statistics were used.

4.4. Statistical analysis

Following collection, all data were entered into Microsoft® Excel® sheets (Microsoft® Corporation, USA) and analyzed using the SPSS® version 24 software (SPSS® Inc., USA). The assumption of normality was investigated using the Shapiro–Wilk test.

In the study investigating the validity of the digitized measurements, the Spearman's correlation coefficient and the Wilcoxon test were used to analyze the difference between the plaster and digitized cast measurements of incisor clinical crown lengths. The kappa statistic was used to quantify the agreement between the plaster and digitized cast assessments for the presence of recessions.

The association between the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment with the changes in their sagittal inclination was assessed using Spearman's rho correlation coefficient. The differences in the changes in clinical crown length between the retroclined, stable and proclined groups was assessed with the Kruskal-Wallis and Mann-Whitney tests. The association of the changes with age and treatment duration, as well as the difference between genders and Angle Classes was assessed with Spearman's rho correlation

coefficient, the Mann-Whitney test and the Kruskal-Wallis test, respectively.

The differences in the prevalence of gingival recession in the labial aspect of the maxillary and mandibular incisors, before and after orthodontic treatment, between the retroclined, stable and proclined groups were assessed with the Fisher's exact test. Statistical significance was set at $p<0.05$.

5. RESULTS

5.1. Validity of digital measurements

The Spearman's correlation coefficient for the measurements of incisor clinical crown lengths on the plaster and digitized casts was 0.924. No statistically significant differences were found with the Wilcoxon test. The level of agreement between the plaster and digitized cast assessments for the presence of recessions was very good (kappa scores >0.800).

5.2. Method error

Intra-observer repeatability was excellent for the linear and angular measurements. The intraclass correlation coefficients for the measurements of incisor clinical crown lengths and incisor inclination are presented in Table 1.

The application of Wilcoxon tests between both series of measurements did not reveal any statistically significant differences. Random errors for the measurements of incisor clinical crown lengths ranged from 0.10 to 0.18 mm and for incisor inclination ranged from 1.1 to 1.3^{o}. The level of agreement between the two series of measurements for the presence of recessions indicated good agreement (kappa scores >0.800).

Table 1. Intraclass correlation coefficients [ICC] and 95% Confidence Intervals [95% CI].

Maxillary teeth	ICC [95% CI]	Mandibular teeth	ICC [95% CI]
12 clinical crown length	0.978 [0.955-0.990]	**32 clinical crown length**	0.950 [0.896-0.976]
11 clinical crown length	0.972 [0.941-0.987]	**31 clinical crown length**	0.954 [0.901-0.979]
21 clinical crown length	0.983 [0.965-0.992]	**41 clinical crown length**	0.974 [0.946-0.988]
22 clinical crown length	0.976 [0.950-0.989]	**42 clinical crown length**	0.980 [0.958-0.991]
U1/NL angle	0,982 [0,963-0,991]	**L1/MP angle**	0,964 [0,926- 0,983]

5.3. Sample characteristics

Twelve male and 70 female patients were included in the final sample. Most of them received treatment on a non-extraction basis. The basic characteristics of the subjects are presented in Table 2. Overall, the maxillary incisors retroclined, whereas the mandibular incisors proclined during treatment.

Table 2. Age characteristics of the sample (years), treatment duration (years), and inclination changes (°).

	Median	Minimum	Maximum	Interquartile range
Age before treatment	16.12	10.50	40.66	12.50
Age at the end of Treatment	17.70	10.66	42.33	11.38
Treatment duration	1.71	0.08	5.25	1.35
Change in U1/NL	-1.2631	-30.64	18.56	9.66
Change in L1/MP	0.9698	-20.13	23.31	7.89

5.4. Clinical crown length

The changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment are presented in Table 3. Overall, clinical crown length decreased in the maxillary and increased in the mandibular teeth.

Table 3. Changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment (mm).

Tooth number	Median	Minimum	Maximum	Interquartile range
12	-0.0350	-1.28	1.84	0.74
11	-0.2400	-1.63	0.82	0.72
21	-0.2550	-1.77	1.52	0.80
22	-0.1100	-1.76	2.15	0.64
32	0.0700	-1.57	2.00	0.79
31	0.1300	-1.62	1.77	0.98
41	0.0000	-1.20	2.41	0.96
42	-0.0850	-0.86	2.35	0.89

The Spearman's rho correlation coefficients for the association between the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment, and the observed changes in their sagittal inclination, respectively, are shown in Table 4. No association was noted for any of the investigated variables.

Table 4. Correlation coefficients between the changes in clinical crown length and the sagittal inclination of maxillary and mandibular incisors.

Tooth number	**Changes in U1/NL**	
	Correlation Coefficient	**p value**
12	0.144	0.215
11	0.169	O.136
21	0.064	0.574
22	0.118	0.307
	Changes in L1/MP	
	Correlation Coefficient	**p value**
32	0.060	0.602
31	-0.057	0.624
41	-0.001	0.994
42	0.061	0.593

The Spearman's rho correlation coefficients for the association between the changes in clinical crown length in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment, and treatment duration are shown in Table 5. Overall, no association was noted apart from the changes in clinical crown height in tooth number 12.

Table 5. Correlation coefficients between the changes in clinical crown length and treatment duration.

Tooth number	Correlation Coefficient	p value
12	0.228	0.047
11	0.41	0.723
21	-0.081	0.474
22	0.111	0.335
32	0.151	0.184
31	-0.003	0.980
41	0.096	0.399
42	0.180	0.115

No statistically significant differences in the clinical crown length changes were noted between males and females (Table 6).

Table 6. Differences in clinical crown length changes between males and females (mm). [Med: median; min: minimum; max: maximum; IR: interquartile range].

Tooth number	Males				Females				
	Med	**Min**	**Max**	**IR**	**Med**	**Min**	**Max**	**IR**	**p-value**
12	0.3950	-0.63	1.84	0.70	-0.0750	-1.28	1.20	0.69	0.053
11	-0.3100	-1.03	0.61	1.06	-0.1400	-1.63	0.82	0.64	0.552
21	-0.1300	-0.82	0.95	0.68	-0.2550	-1.77	1.52	0.75	0.647
22	-0.0600	-0.68	2.15	0.58	-0.0450	-1.76	1.60	0.68	0.890
42	-0.1800	-0.68	1.07	0.59	-0.0150	-0.86	2.35	1.06	0.221
41	0.3100	-0.72	0.95	0.49	-0.1200	-1.20	2.41	0.98	0.171
31	0.2150	-0.54	1.69	0.68	0.0700	-1.62	1.77	1.07	0.115
32	0.2650	-0.79	1.32	1.24	0.0650	-1.57	2.00	0.92	0.897

No statistically significant differences in the clinical crown length changes were noted between Angle Classes (Table 7).

Table 7. Differences in clinical crown length changes between Angle Classes (mm). [Med: median; min: minimum; max: maximum; IR: interquartile range]

Tooth number	Class I				Class II				Class III				
	Med	**Min**	**Max**	**IR**	**Med**	**Min**	**Max**	**IR**	**Med**	**Min**	**Max**	**IR**	**p-value**
12	0.800	-0.79	1.20	0.82	-0.06	-1.28	1.84	0.72	0.09	-0.59	0.57	0.88	0.743
11	-0.14	-1.63	0.82	0.51	-0.22	-1.56	0.61	0.84	-0.01	-1.00	0.62	1.06	0.905
21	-0.13	-0.79	1.52	0.62	-0.29	-1.77	0.64	0.50	-0.23	-0.82	0.76	1.22	0.044
22	0.0250	-1.76	1.60	0.63	-0.17	-1.59	2.15	0.76	-0.17	-0.54	1.02	0.95	0.355
42	0.0750	-0.81	2.35	1.29	-0.04	-0.68	1.24	0.52	0.0150	-0.48	0.83	0.70	0.575
41	0.0950	-0.96	2.41	1.07	0.2600	-1.16	1.25	1.11	-0.180	-0.76	0.52	0.64	0.459
31	0.2200	-0.79	1.77	1.05	0.2300	-1.62	1.69	1.06	-0.235	-1.37	0.77	1.50	0.664
32	0.0950	-1.57	2.00	0.90	0.2300	-1.27	1.32	1.29	-0.025	-0.91	0.79	1.03	0.555

No statistically significant differences in the clinical crown length changes were noted between the retroclined, stable and proclined groups, except for tooth number 12 (Table 8).

Table 8. Differences in clinical crown length changes between retroclined, stable and proclined groups. [Med: median; min: minimum; max: maximum; IR: interquartile range]

Tooth number	Retroclined				Stable				Proclined				
	Med	Min	Max	IR	Med	Min	Max	IR	Med	Min	Max	IR	p-value
12	-0.06	-1.28	1.20	0.99	-0.53	-0.68	0.11	0.60	0.0550	-0.79	1.84	0.68	0.042
11	-0.13	-1.56	056	0.70	-0.52	-0.67	0.03	0.59	-0.255	-1.63	0.82	0.71	0.298
21	-0.25	-1.77	0.79	0.87	-0.28	-0.83	0.31	0.64	-0.260	-0.82	1.52	0.65	0.532
22	-0.07	-1.59	1.03	0.60	-0.50	-0.61	0.32	0.57	-0.045	-1.76	2.15	0.78	0.170
42	-0.13	-0.74	2.35	0.74	0.03	-0.81	0.83	1.18	-0.08	-0.86	1.28	1.06	0.855
41	0.13	-1.19	1.64	1.07	-0.18	-1.20	1.08	1.29	0.055	-1.16	2.41	0.91	0.760
31	0.1850	-1.62	1.69	0.90	0.385	-1.35	0.77	1.13	-0.01	-1.49	1.77	1.09	0.973
32	0.0850	-1.27	1.97	0.89	-0.02	-0.82	1.54	1.63	0.09	-1.57	2.00	0.94	0.451

The application of paired Mann-Whitney tests for the clinical crown length changes of tooth 12 between the retroclined, stable and proclined groups, revealed a statistically significant difference between retroclined/stable and proclined/stable but not between retroclined/proclined (Table 8).

Table 9. Paired differences in clinical crown length changes of tooth number 12 between the retroclined, stable and proclined groups.

	Retroclined/Stable	Proclined/Stable	Retroclined/Proclined
p-value	0.073	0.013	0.217

5.5. Gingival recession

Very few gingival recession sites were found, either before or after treatment (Table 9). The differences in recession presence between the various teeth, as well as, before and after treatment, were not statistically significant ($p>0.05$).

Table 9. Changes in gingival recession presence in the labial aspect of the maxillary and mandibular incisors before and after orthodontic treatment.

Teeth	Gingival recession |Before treatment		Gingival recession |After treatment	
	Absence	**Presence**	**Absence**	**Presence**
12	74	2	75	1
11	77	2	77	2
21	77	3	75	5
22	75	2	75	2
42	77	1	77	1
41	77	2	77	2
31	74	2	74	2
32	78	1	77	2

Similarly, no statistically significant differences in recession presence were noted between the retroclined, stable and proclined groups.

The prevalence of gingival recession in at least one maxillary or mandibular incisor before treatment was 3.7% (3 out of 72) and 1.2% (1 out 72) respectively. The prevalence of gingival recession in at least one maxillary or mandibular incisor after treatment was 7.3% (6 out of 72) and 2.4% (2 out 72) respectively. The difference was not statistically significant ($p>0.05$). Similarly, no statistically significant differences were noted between the retroclined, stable and proclined groups ($p>0.05$).

6.DISCUSSION

6.1. Summary of evidence

Gingival recessions may compromise the orthodontic therapeutic outcome. As teeth are moved orthodontically to new positions, it is possible that they acquire unfavourable positions which could lead to apical soft tissue relocation, an increase in clinical crown length and, eventually, recession formation (Wennstrom et al., 1987). Overall, the findings of the present study suggest that change in incisor inclination during treatment does not affect labial clinical crown length or gingival recession development.

Despite the differences in the extent and direction of lower incisor inclination during treatment, the increase in clinical crown heights was similar across the various groups, which is in agreement with other investigations (Ruf et al., 1998; Artun and Grobéty, 2001; Djeu et al., 2002; Melsen and Allais, 2005; Djeu et al., 2002; Renkema et al., 2013b; Kamak et al., 2015; Renkema et al., 2015; Konikoff et al., 2007). Teeth exhibiting gingival recession generally show an increase in clinical crown length because of the accompanying decrease in the vertical height of the attached gingiva. However, clinical crown length is not necessarily an accurate indicator of gingival recession because teeth can be extruded without any damage to the gingival, while at the same time these teeth exhibit an increase in crown height that is not considered pathological/detrimental/deleterious. Nonetheless, clinical crown length was included in this study to provide a secondary measure of gingival recession. A recent study by (Kamak et al., 2015) showed that neither changing the inclination of mandibular incisors nor maintaining them in the original positions during orthodontic treatment has any influence on the development of

gingival recessions in the mandibular incisor region. Although they found an increase in the clinical crown height in tooth number 32, the difference was limited to a single tooth and the change in clinical crown heights of the remaining incisors was comparable. A retrospective study by Coatoam et al. (1981) evaluated the effect of orthodontic therapy on the width of the zone of the keratinized gingival and showed statistically significant increases in the clinical crown during orthodontic therapy are not reflected in statistically significant decreases in the width of the keratinized gingiva. Minimal widths of keratinized gingiva (less than 2 mm) are capable of withstanding the stresses of orthodontic mechanics. This change is partly related to the position of the tooth in the dental arch and to the pre-existing condition of the keratinized gingiva. The greatest loss of width of keratinized gingiva occurred in relation to the lateral incisors. Both maxillary and mandibular lateral incisors in this investigation were often in a lingual position. In addition, Bowers (1963) found that these lingually displaced teeth often had the greatest width of keratinized gingiva. It appears that when these teeth are brought into proper alignment through orthodontic therapy, the result is a decrease in this width. Similarly, Dorfman (1978) concluded that lingual tooth movement will result in an increased bucco-lingual thickness of the tissue at the facial aspect of the tooth, which results in coronal migration of the soft tissue margin (decreased clinical crown height). On the other hand, facial tooth movement will result in a reduced bucco-lingual tissue thickness and thereby a reduced height of the free gingival portion and an increased clinical crown height.

Renkema et al. (2013a) noted that only 6.6% of patients exhibited gingival recessions. In the present study, recession was assessed on digital casts, whereas Renkema et al. (2013a) evaluated recession on plaster casts. The higher prevalence of patients with gingival

recessions following orthodontic treatment noted by Slutzkey and Levin (22.9%) could be explained by the fact that they evaluated recession at 18-22 years of age, meaning that their sample was older than the average age of our sample. In the present study, the prevalence of recession after orthodontic treatment was similar to, or slightly less than, that reported for untreated patients. Ainamo et al. (1986) reported that 8.7% of teeth among untreated 17-year olds showed recessions, whereas Susin et al., (2004) noted recessions on only 2.9% of teeth among untreated 14 to 19-year olds.

Based on the present findings, change of incisor inclination during treatment did not affect gingival recession development. Allais and Melsen (2003) showed that controlled proclination under the maintenance of good oral hygiene can be carried out in most patients without risk to the periodontium. It should, however, be underlined that new recessions developed in 10% of the investigated teeth, but also improved in 5%. In 85% of their study sample there was no change. Other studies, however, found association between a change of inclination of lower incisors and increased risk of gingival recessions (Sperry et al., 1977; Artun and Krogstad, 1987; Renkema et al., 2013a; Yared et al., 2006). Artun and Krogstad (1987) studied 40 adult individuals who underwent surgery for mandibular prognathism. Their patients were older at the start of treatment (18-33 years), which is important because the ability of the periodontium to withstand orthodontic treatment appears to decrease with age (Ruf and Pancherz, 1998). They reported that excessively proclined incisors (more than 10^{o} to the mandibular plane) exhibited a significant increase in clinical crown height causing gingival recession, both during appliance therapy and the 3 years of the retention period compared with patients with minimal change of incisor inclination. However, the long-term prognosis for such teeth with extensive gingival recessions may not be critical. The noted discrepancies

may also be due to the fact that IMPA was the only variable measured in lateral cephalograms without noting whether or not the incisors were protracted or retracted. In addition, it might be due to the more complex etiology of gingival recession, in which orthodontic treatment is only one factor among others in its development (Bollen et al., 2008). Overall, the level of available evidence is low, the degree of recession found in studies with statistically significant differences between proclined and non proclined teeth is of questionable clinical consequence and should be treated with caution until additional research becomes available (Joss-Vassalli et al., 2010).

6.2. Limitations

Although we followed a similar methodology to other investigators (Artun and Grobety, 2001; Djeu et al., 2002; Allais and Melsen, 2005), the fact that assessments were carried out immediately post-treatment, may somewhat circumscribe the applicability and generalisability of the observed results. Artun and Krogstad (1987) have suggested an observation time of 3 years; as up to 3 years the clinical crown height increased significantly more in the patients with excessive proclination than in the patients with minimal change in incisors inclination, but thereafter the differences between the groups were not significant. Renkema et al. (2015) also observed no difference in recession 5 years after treatment between individuals with an average final IMPA of 90.80 and another group who finished at 105.2°. Immediately after treatment, it is possible that gingival inflammation and enlargement can mask recession. However, in the present study high quality clinical pictures of thin gingival margins and pointed papilla were present.

In the present study, the possible influence of the width of the attached gingiva on the observed results could not be assessed. However, this is a clinical parameter that is impossible to measure from study casts (Djeu et al., 2002). Furthermore, measurement of

gingival recession and the inclination of lower incisors were measured on pre-treatment and end-of-treatment records, but not during treatment. It is possible that in some subjects, the lower incisors were moved labially to correct a discrepancy, which may have caused a recession, and then were moved lingually (retroclined) before the final records were taken. In such cases, the transient recession would not be detected by an observer relying on the pre- and post-treatment records. Finally, information on smoking and oral hygiene were not reported in the retrieved records, so the influence of these variables could not be assessed and incorporated into the correlation analysis. Another significant limitation concerns the use of lateral cephalograms. This methodological issue is something that can be found in the majority of the existing literature. One of the main problems in relating incisor proclination to gingival recession and identifying patients susceptible to this kind of side effect, is possibly correlated with the amount of bone supporting the tooth roots in the buccolingual dimension. The problem with 2D representation is that it does not provide any information regarding the hard tissue biotype. It is well documented that bone thickness in anterior maxillary and mandibular areas can range from really thin to absent; meaning that dehiscences and fenestrations are maybe present before the initiation of orthodontic treatment. In addition, cortical bone thickness can vary according to the skeletal pattern of the patients. A study using CBCTs as an assessment method to correlate bone thickness, incisor inclination and the development gingival recessions could be of benefit for the diagnosis and possibly create some cut-off values for both parameters and aid the prediction of susceptibility and risk of future gingival recession development following the completion of treatment.

Although some differences have been detected among biotypes, the data indicate that biotype does not play a fundamental role in influencing alveolar BT, whereas other variables (i.e., TT, sex, age, and smoking habit) do influence alveolar BT. Further studies are needed to better understand the extent of influence arising from each clinical variable

(El Nahass and Naiem, 2014).

Kook et al., 2012 carried out a study comparing alveolar bone loss around incisors in normal occlusion samples and surgical skeletal class III patients and concluded that special care should be taken to avoid aggravating preexisting alveolar bone loss in the anterior teeth, especially in the mandible, in skeletal Class III patients, who may be more vulnerable to alveolar bone loss during orthodontic treatment.

6.3. Recommendations for future research

Additional prospective, long-term, clinical studies, including clinical assessment of oral hygiene and gingival condition before, during and after treatment are needed to clarify the effect of orthodontic changes in incisor inclination and the occurrence of gingival recession.

7. CONCLUSIONS

Overall, orthodontic treatment, as well as, the change in incisor inclination during treatment did not seem to affect increases in labial clinical crown length and gingival recession development in the maxillary and mandibular incisors of this specific sample. Further long-term studies are needed in order to fully evaluate the effect of orthodontically induced changes on soft periodontal tissue support and clinical crown length.

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