

**LONG-TERM PREVALENCE OF GINGIVAL RECESSION FOLLOWING
LABIAL ORTHODONTIC TOOTH MOVEMENT**

A Thesis

by

JASON W MORRIS

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Chair of Committee,	Peter Buschang
Committee Members,	Phillip Campbell
	Larry Tadlock
	Jimmy Boley
Head of Department,	Phillip Campbell

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ABSTRACT

The effects of labial incisor movements are controversial, and the effects of expansion of maxillary posterior teeth remain unknown. Therefore, this study was designed to evaluate the long-term prevalence of gingival recession following orthodontic tooth movements.

Records of 205 patients (162 female, 43 male) were obtained from two private practice orthodontists who were careful to avoid excessive labial movement of mandibular incisors and maxillary posterior teeth. Using pre-treatment (14.0 ± 5.9 years) and post-treatment (16.5 ± 6.0 years) lateral cephalograms and dental models, changes in mandibular incisor inclination and maxillary arch widths were determined. Gingival recession was measured based on post-treatment and post-retention (32.3 ± 8.5 years) intraoral slides. Associations between tooth movements and gingival recession were evaluated statistically.

There was only limited recession at the end of orthodontic treatment (5.8% of teeth exhibited recession; 0.6% exhibited recession greater than 1 mm). Recession increased long-term (41.7% of teeth), however the severity was limited (7.0% over 1 mm). Both incisor proclination and retroclination occurred, resulting in no statistically significant treatment change ($-0.05 \pm 6.4^\circ$). Proclination was greatest in individuals with the most upright incisors at the beginning of treatment ($R = -0.575$; $p < 0.001$). There was no

relationship between the treatment changes in mandibular incisor proclination and the post-treatment changes in gingival recession. There was also no difference in recession between incisors that finished treatment at an incisor to mandibular plane angle (IMPA) of less than 95° versus those that finished at an IMPA greater than or equal to 95°. Expansion of the maxillary posterior teeth was limited (1.1 ± 2.0 mm, 0.2 ± 2.6 mm, and -0.2 ± 2.2 mm for the first premolars, second premolars, and first molars, respectively). There were weak positive correlations (0.173 to 0.407) between increases in maxillary arch widths and recession long-term.

Orthodontic treatment is not a risk factor for the development of gingival recession.

Proclination of the mandibular incisors does not increase the risk for recession long-term if care is taken to avoid excessive labial movements. Expansion of the maxillary premolars and first molars does increase the risk of long-term recession, but the increase is limited.

DEDICATION

I would like to dedicate this thesis to my incredible wife, Jennifer, for her unfailing love and support through my long years of continuing education. I would also like to dedicate this to my beautiful daughter, Katelyn, who was born shortly before this project was completed.

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Significance of gingival recession

By clinical definition, gingival recession refers to the exposure of the root surface by an apical shift in the position of the gingiva.[1] It can be localized or generalized, and associated with one or more tooth surfaces.[2] Several aspects of gingival recession make it clinically significant. First, recession signifies a loss of attachment. In areas of recession, the epithelial attachment has migrated apically from the CEJ. Second, root surfaces exposed as a result of gingival recession are more susceptible to caries. In a sample of 452 adults aged 65 or older, Lawrence et al [3] showed that indicators of poor periodontal status, including gingival recessions, were significantly correlated with an increased risk of root caries development. Additionally, studies that have examined the prevalence of root caries have shown higher levels of lesions in patients with periodontal disease and recession compared to patients without recession.[4-6] Third, abrasion or erosion of the cementum exposed by recession leaves an underlying dentinal surface that can be sensitive. Al-Wahadni and Linden [7] demonstrated that gingival recession of 3 mm or more was the best independent predictor of dentin hypersensitivity. In another study, Costa et al [8] showed that gingival recession was associated with increased dentin hypersensitivity in 1,023 adults aged 35 or older in Brazil. In addition to these complications, authors have proposed other clinical problems associated with recession,

including difficult maintenance of oral hygiene [1, 9] and compromised esthetics.[9-11] For instance, Rocha et al [11] showed that when the esthetic perception of smiles was evaluated by 160 dental students, there was a statistically significant difference between smiles with gingival recession and smiles without. This presents a problem because esthetics is a major motivational factor for patients to seek orthodontic treatment.[12, 13] For example, Reidman et al [12] they showed that for 75% of patients seeking orthodontic treatment, dental esthetics was their primary motive. Therefore, if orthodontic treatment contributes to the formation of gingival recession, there are a number of significant clinical and psychological problems that may result.

Anatomy of the attachment apparatus

In order to better understand recession, a review of the soft tissue attachment apparatus is necessary. In 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 cemento-enamel junction (CEJ). Just 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 [14] found that, in the average human, the connective tissue attachment measures 1.07 mm, and the junctional epithelial attachment measures 0.97 mm.

Measurement of gingival recession

Gingival recession is typically measured during a clinical examination with a periodontal probe. It is recorded as the distance in millimeters from the CEJ to the gingival crest.[1] To better understand recession, however, it helps to distinguish between the actual and the apparent positions of the gingiva. The *actual position* is the level of the epithelial attachment on the tooth, whereas the *apparent position* is the level of the crest of the gingival margin. The difference between the actual and apparent position of the attachment is the sulcus depth. As mentioned previously, in a completely erupted tooth with healthy periodontal tissues, the coronal portion of the epithelial attachment is located near the CEJ.[1] In the strictest sense, the severity of recession is determined by the actual position of the gingiva, not its apparent position, and is measured from the CEJ to the actual position.[1] However, measuring the *actual position* of the gingiva is

inherently difficult, and therefore the most widely accepted measurement of gingival recession is from the CEJ to the gingival crest.[1]

Methods of measuring recession without having to perform a clinical examination have been developed. The two most widely utilized substitutes are dental casts and intraoral photographs. Dental casts have been successfully used to measure recession as a dichotomous level variable. Renkema et al [15] assessed the validity of using dental casts for scoring recessions on 30 adults. The clinical exam and dental cast comparison produced a mean kappa score greater than 0.80, suggesting good agreement. Intraoral photographs have also been used to measure recession as a dichotomous level variable. To assess the method error in evaluating gingival recession from intraoral photographs, Ruf et al [16] conducted replicate analyses of photographs of 10 randomly selected subjects and found a concordance in 80% of the subjects and 92.5% of the teeth. In both of the studies mentioned above, recession was considered present if the CEJ was exposed. Allais and Melsen [17] measured the reliability of both of these methods and found that the number of unreadable teeth was larger when performed on casts than when assessed on intraoral photographs, and the variation in the error of the method was smaller for the photographs than for the cast analysis. The authors speculated that the reliability was better for the intraoral photographs compared to the dental casts because the color contrast between the enamel and cementum helped to distinguish the CEJ in the photographs. Also, dental casts may have artifacts around the gingival margin.

A method for measuring recession as an interval level variable using a combination of dental casts and intraoral photographs was established by Djeu et al [18], based on a method originally applied by Coatoam et al. In the study by Coatoam et al [19], the width of keratinized gingiva on the facial surface of the mandibular incisors was measured from slides that were calibrated for size using the corresponding dental casts. They reported an error of the method of 0.1 mm. Trentini et al [20] compared this method of using dental casts and intraoral photographs with the corresponding measurements made during a clinical examination. The average difference between the clinical measurements and those calculated using the orthodontic records was small and not statistically significant. The method error was determined to be 0.43 mm for the clinical measures and 0.32 mm for the measures calculated from orthodontic records. The reliability of the orthodontic records measurement was slightly greater than the direct clinical measurement, with intra-class correlations of 0.93 and 0.90, respectively. They concluded that carefully taken photographs and study models provide accurate measures of keratinized tissue width. Furthermore, the method errors suggest that an examiner may actually be more reliable measuring from dental casts and intraoral photographs compared to measuring directly in the mouth. Djeu et al [18] utilized this method to measure gingival recession as opposed to the width of keratinized gingiva. Using dental casts and intraoral slides, examiner reliability was evaluated on 10 randomly selected patients from their study. Paired t-tests indicated that there was no significant difference between the original and repeated values.

Prevalence and demographics of gingival recession

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.[21-24] For example, in a sample of 299 children and teenagers, Ainamo et al [21] showed that at least one gingival recession of 0.5 mm or more, measured from the CEJ to the gingival margin, was present on 5% of 7 year olds, 39% of 12 year olds, and 74% of 17 year olds. Susin et al [24] 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. Using a sample of 9,689 persons from data collected in the third National Health and Nutrition Examination Survey (NHANES III), Albander and Kingman [22] demonstrated that the prevalence, extent and severity of gingival recession increased in individuals aged 30 and over. In this 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 have a significantly higher prevalence of gingival recession than others. For example, from the NHANES III data, Albander and Kingman [22] noted that in the maxillary arch, the two teeth that presented with gingival recession most often 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 had recession. In the mandibular arch, the central incisors and the first premolars showed the most recession. Approximately 19.7% of central incisors showed labial gingival recession, 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 were affected, and 43.5% of first premolars. The lateral incisor was the next highest in this group, with 42% of teeth showing recession. This pattern of recession has been demonstrated in other non-orthodontic samples.[21, 24-27]

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 [21], gingival recession was measured on the facial and lingual surfaces of 299 Finish school children aged 7 to 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%). The increased prevalence of recession on the facial surface as opposed to the lingual surface is supported by a study by Loe et al.[28] They also demonstrated that lingual recession seems to appear later in life. In the study by Loe et al, the prevalence of gingival recession in two cohorts of individuals 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, primarily 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. Unfortunately, recession was measured only on the facial surface in the NHANES III data.

Additional demographic features that appear to be related to gingival recession include sex, ethnicity, and socioeconomic class. Using the NHANES III data, Albander and Kingman [22] demonstrated that males aged 30 or more had significantly more recession than females of the same age. The results of Susin et al [24] 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 sex differences. Ainamo et al [21] reported similar results. In their sample, no significant sex differences were noted among 17 year olds. Therefore, it appears that there are no sex differences at the younger ages, but as individuals age, males exhibit more recession than females. The NHANES III data also indicated that of the three

race/ethnic groups studied, non-Hispanic blacks had the highest prevalence and extent of gingival recession.[22] Mexican Americans had similar prevalence and extent of gingival recession compared with non-Hispanic whites.[22] Finally, in the sample investigated by Susin et al [24], the percentage of teeth with recession was significantly higher in the lower socioeconomic groups, irrespective of age.

Attrition and eruption effects

The correlation between age and recession has led some investigators to assume that recession may be an age related physiologic process, and that maturation brings about greater exposure of the tooth outside its investing soft tissues. When the teeth reach their functional antagonists, the gingival attachment is still well above the CEJ, and the clinical crown is approximately two-thirds of the anatomic crown. The relative apical movement of the attachment after this results more from vertical growth of the jaws and the accompanying eruption of the teeth than from apical migration of the gingival attachment. By the time vertical growth of the jaws has slowed to the adult rate, which is typically in the late teens, the gingival attachment is usually near the CEJ. At one time, it was thought that *passive eruption*, which is the exposure of the teeth by apical migration of the gingiva, played a large role in this process. However, it now appears that as long as the gingival tissues are entirely healthy, this sort of downward migration of the soft tissues does not occur.[1] What was once thought to be passive eruption

during the teens is really *active eruption*, compensating for the vertical jaw growth occurring at that time.[29]

The effect of aging on the location of the junctional epithelium has been the subject of much speculation. Some reports show migration of the junctional epithelium from its position in healthy individuals (i.e., near the CEJ) to a more apical position on the root surface, with accompanying gingival recession.[30] In other animal studies, however, no apical migration has been noted.[31] With continuing gingival recession, the width of the attached gingiva would be expected to decrease with age, but the opposite appears to be true.[32, 33] Alternatively, the migration of the junctional epithelium to the root surface could be caused by the tooth erupting through the gingiva in an attempt to maintain occlusal contact with its opposing tooth as a result of tooth surface loss from attrition. This has been termed *physiologic recession*.

There is some evidence that supports physiologic recession. Human skull studies have demonstrated the continuous eruption of the mandibular molars throughout life, without a compensatory migration of the surrounding hard tissue.[34, 35] In a study by Whittaker et al [34], the eruption and alveolar bone levels of the mandibular first molars was evaluated on the jaws of an eighteenth-century population whose tooth wear had been minimal. The measurements of eruption and bone levels were made on 122 human skulls using a stable reference point, the inferior alveolar canal. Over a 40 year period, they showed that the mandibular first molar erupted 2.8 mm on average, while the

alveolar crest remained relatively stable. The alveolar crest increased by only 0.7 mm over the 40 year period. In a very similar study, Varrela et al [35] showed similar results in a population that had much more attrition. Their sample consisted of 244 individuals from the medieval period. Again, the inferior alveolar canal was used as a stable reference point. Their results showed that the first molar experienced attrition of 2.99 mm over a 30 year period, and erupted 2.25 mm over that same time period. Like the study by Whittaker et al, the alveolar crest remained stable, indicating that despite continuous eruption of the teeth, there was no concomitant growth of the alveolar crest. In this study, only a weak negative correlation was found between eruption and attrition (Spearman's rho = -0.15). However, in other similar studies on human skulls, stronger correlations have been found, such as in the studies by Newman and Levers [36], Levers and Darling [37], and Whittaker et al [38]. Therefore, it is possible that as we age, if there is significant attrition and compensatory eruption, it may be a contributing factor in recession.

Etiology

Numerous factors have been implicated in the etiology of gingival recession, including faulty tooth-brushing technique (gingival abrasion) [2, 39], gingival inflammation and periodontal disease [2, 28, 40], friction from soft tissues (gingival ablation) [41], and iatrogenic dentistry.[1] Trauma from occlusion has been suggested in the past, but its mechanism of action has never been demonstrated.[1] A relationship may also exist

between smoking and gingival recession. [42, 43] The multifactorial mechanism may include alterations in the immune response, such as decreases in the phagocytic function of polymorphonuclear leukocytes [44, 45] and reduction in the production of immunoglobulins.[46] Reduction in gingival blood flow as a result of smoking is also a possible contributing factor.[47, 48] Finally, orthodontic treatment has been implicated in the development of gingival recessions.[9, 49-54]

Orthodontic treatment may cause recession because fixed appliances act as a retention area for plaque. In patients without orthodontic appliances, plaque levels range from 10.3% to 13.3%, depending on the population.[55, 56] In a recent study by Klukowska et al [57], 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 $41.9 \pm 18.8\%$. If this plaque is not adequately removed, the ensuing gingival inflammation may lead to periodontal breakdown, and therefore recession.[2, 49, 50]

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.[58-60] Moreover, there is also evidence suggesting that thin gingival tissue (thin biotype/phenotype) is more prone to recession.[61, 62] 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.

This hypothesis seems to be supported by the literature. To determine the prevalence of gingival recession, as well as the etiologic factors associated it, Parfitt and Mjor [60] examined 668 school children aged 9 to 12 years. Eight percent of this group was found to have between 2 and 5 mm 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 had a discrepancy. Trott and Love [58] 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. Finally, Gorman [59] 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 studies mentioned above have prompted the postulation that gingival recessions are often found at tooth surfaces where alveolar bone dehiscence 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 result.

Effects of incisor proclination on surrounding hard and soft tissues

In 1981, Steiner et al [49] utilized monkeys to evaluate dehiscence formation during labial tooth movement. They moved the central incisors of five *Macaca nemitrina* monkeys a mean distance of 3.05 mm over 13 weeks. The movement was followed by a stabilization period of 3 weeks. Afterwards, the teeth and surrounding tissues were evaluated with periodontal flap surgery. Using an amalgam marker on the facial surfaces of the teeth as a stable reference point, they were able to measure the changes in the distance to the marginal bone, the connective tissue attachment, and the gingival margin. Their results showed that all three measurements were significantly different compared to controls. For the marginal bone height, the experimental group showed an average of 3.96 mm of labial bone loss compared to controls. The connective tissue attachment and gingival margin migrated apically, 1.04 mm and 0.81 mm, respectively, compared to controls. In this case, the marginal bone loss was much greater than the amount of soft tissue loss. Eight months later, Engelking and Zachrisson [63] took the same animals and moved the same teeth back lingually into position with fixed appliances. The incisors were retracted a mean distance of 1.8 mm and then retained for

5 months. The animals were sacrificed and clinical and histological examinations were performed. The marginal bone levels recovered, relative to their original levels, an average of 2.5 mm and 3.1 mm for the maxillary and mandibular incisors, respectively. Bone histomorphometric analysis with tetracycline labels demonstrated significant osteogenesis in the periodontium of the retracted teeth.

In 1982, Karring et al [64] performed a similar study using beagle dogs. They evaluated the effect of facial tipping on the maxillary second and third incisors. Orthodontic appliances were used to tip the incisors on the left side of the maxilla in a facial direction through the alveolar bone plate. After five months of movement, the appliance was reversed so that the teeth were brought back to their original position over a subsequent five month time period. At the same time, the incisors on the right side of the mouth were tipped out to a position corresponding to that attained on the left side, and held in this position while the left side was moved back. Both sides were retained in their final positions for 5 months, and then the animals were sacrificed. The teeth of three untreated dogs were used as controls. Meticulous care was taken so that all teeth during the study were free of plaque and gingival inflammation. The average distance between the CEJ and alveolar bone crest in control teeth was 2.2 mm (± 0.5 mm). The average distance between the CEJ and the bone crest in the test group, in which the incisors were retained in a tipped position, was 4.1 mm (± 2.1 mm). Lastly, for the incisors that were moved back after tipping, the measurement was 1.8 mm (± 0.4 mm). In all dogs, including the controls, the apical termination of the junctional epithelium corresponded

to the cement-enamel junction (CEJ), indicating that soft tissue migration was not evident. This study supports the findings of Steiner et al [49], that labial tooth movement can create a dehiscence, but that the defect can be at least partially repaired if the tooth is moved back into position.

In 1987, Wennestrom et al [50] again showed similar results. In their study, the maxillary central incisors of 5 *Macaca Cynomolgus* monkeys were protracted an average of 3.3 mm over a period of 3-4 months. The teeth were then stabilized for a period of 1 month. All ten of the experimental teeth experienced facial bone loss. On average, the facial bone migrated apically 2.08 mm compared to control teeth. In contrast, only five of the ten test teeth exhibited apical migration of the gingiva. The mean apical movement of the gingival margin of the five teeth that did experience recession was 0.4 mm. In eight of the ten test teeth, the apical termination of the junctional epithelium was positioned at the CEJ. Two test teeth, both in the same animal, showed a small loss of connective tissue attachment (0.3 mm and 1.4 mm). The authors noted that on the two teeth that did experience a loss of connective tissue attachment, the gingival tissues showed clear signs of gingival inflammation.

The three studies highlighted above demonstrate three important issues. First, a dehiscence can be produced in the alveolar bone by tipping the teeth in a facial direction. Second, bone has the capacity to regenerate in such defects when the teeth are moved back to their original position. Finally, the soft tissue attachment does not migrate

apically nearly as much as does the marginal bone when teeth are tipped facially. In fact, in the studies reviewed above, more times than not it stayed in the original position.

Effects of expansion on surrounding hard and soft tissues

One of the negative aspects of orthodontic expansion is uncontrolled tipping of the clinical crowns. In 1996, Lundgren et al [65] utilized light forces (50cN) in a human model to expand the maxillary premolars. They noted movement in all three planes of space. In fact, the apices of the premolars moved palatally in 49 of 56 cases, and they tipped between 0.2 to 22.9 degrees. In 2009, Paventy et al [66] evaluated the effects of comprehensive treatment with the Damon System. Nineteen patients with moderate to severe crowding (5 mm or more) were treated following the published Damon System protocol. Treatment in the transverse dimension was limited to expansion with the normal archwire sequence used in the Damon System. The study showed effective expansion of the dental arches with increased arch perimeter. However, the increases in arch width was in part due to tipping of the crowns. On average, the first and second maxillary premolars expanded more at their cusp tips than at their lingual gingival margins. For the maxillary first premolar, the difference was 1.7 mm, and for the maxillary second premolar the difference was 1.6 mm. In 2011, Cattaneo et al [67] evaluated transverse movements and buccal bone modeling in humans after orthodontic archwire expansion. Sixty-four patients were randomly assigned to treatment with either active (In-Ovation R) or passive (Damon 3MX) brackets. Outcomes were evaluated

with digital models and pre and post-treatment cone beam CT radiographs. They found that in all but one patient, transverse expansion was achieved through buccal tipping. Specifically, the Damon group showed 11.7 degrees of tipping at the first premolars and 13.5 degrees of tipping at the second premolars. The In-Ovation group had 11.8 degrees and 13.0 degrees of tipping of the same teeth. Kraus et al [68] used foxhound dogs to evaluate archwire expansion using mechanics similar to those used with the Damon system. Over eight weeks he saw an average of 3.5 mm of tooth movement. The buccal movement was accompanied by a significant amount (15.8 degrees) of tipping.

Poor tooth position and excessive tipping are not the only undesirable effects of orthodontic expansion. Buccal or labial crown movement may be producing deleterious amounts of stress on the surrounding hard tissue. The experiment by Kraus et al, mentioned above, showed similar results to those in which the incisors were proclined in monkeys. Namely, a dehiscence can be formed. After expansion of the premolars, he found an average of 2.9 mm and 1.2 mm of marginal bone loss at the mesial and distal roots, respectively.[68]

In Paventy's thesis, the human clinical trial described previously, a loss of buccal bone height after expansion with the Damon system was also noted.[66] Statistically significant buccal bone height loss occurred at the maxillary first premolars, mandibular first and second premolars, and mandibular first molars. Also, statistically significant facial bone width loss was evident 3 mm apical to the bony crest of the maxillary first

and second premolars and first molars, as well as the mandibular right first premolar, second premolars, and first molars. In 2010, Paventy re-evaluated some of the same subjects 6-12 months post-treatment (5 of the 19 subjects could not be contacted).[66] He noted that all teeth except one showed a small amount of recovery of facial bone height and width, but none of the improvements were statistically significant. In the randomized clinical trial described earlier by Cattaneo et al, loss of buccal bone was also seen.[67] They found that the buccal bone area lateral to the second premolar decreased 23% and 18% (right and left sides) with Damon and 17% and 12% (right and left sides) with In-Ovation. Cattaneo and coworkers also found that the bone loss that occurred with the inter-premolar expansion was positively associated with buccal tipping.

In 1985, Quinn and Yoshikawa published a review of the literature on force magnitude in orthodontics.[69] When evaluating different tooth movements, they noted that with tipping, forces were concentrated on the crestal bone, and when these forces surpass physiological levels, they can become deleterious.

Recession and orthodontic treatment

Without evaluating tooth movements, several studies have shown inconsistencies in the prevalence of recession following orthodontic treatment in humans. In a cross-sectional design, Slutzkey et al [53] found that the prevalence and severity of recession was worse in orthodontically treated patients when compared to patients who had not received

orthodontic treatment. They measured recession clinically as the distance from the CEJ to the free gingival margin on 303 consecutive military cadets, aged 18-22 years. Twenty three percent of patients who had received orthodontic treatment exhibited gingival recession, whereas only 11.4% of patients who had not received orthodontic treatment had recession. Also, 8.4% of patients who had orthodontic treatment had at least one tooth with 3 mm or more of recession, whereas only 0.9% of the patients who did not have orthodontic treatment experienced a recession of 3 mm or more. In contrast, Alstad and Zachrisson [70], as well as Polson et al [71], found no significant difference in gingival recession when comparing groups of orthodontically treated patients with matched control groups. In the study by Alstad and Zachrisson, the periodontal status of the maxillary teeth of teenagers (mean age 11.7 ± 1.4 years), excluding the second molars, was evaluated by measuring from the base of the pocket to the CEJ on the facial surface with a periodontal probe. Five months after appliance removal, they reported no significant difference between the 38 individuals in the experimental group and the 39 matched controls. Polson et al [71] evaluated 112 subjects at least 10 years after orthodontic treatment was completed (mean age 29.3 ± 4.2 years). Recession was recorded clinically as the distance from the CEJ to the free gingival margin on each tooth of both the experimental group and a control group, which consisted of 111 adults with untreated malocclusions. They found no significant difference in gingival recession between the treated and untreated samples.

Renkema et al [15] investigated both the prevalence and pattern of labial recession during and following orthodontic treatment. They measured recession on the facial surface of all teeth on the dental casts of 302 orthodontically treated patients. Recession was considered present if the CEJ was exposed. Measurements were taken at 4 time points: the beginning of treatment (mean age 13.6 ± 3.6 years), the end of treatment (mean age 16.2 ± 3.5 years), 2 years after treatment (mean age 18.6 ± 3.6 years), and 5 years after treatment (mean age 21.6 ± 3.5 years). The authors pointed out that the frequency of recession in their cohort was overall somewhat lower than in non-orthodontic samples.[21, 25, 26] They mentioned that this could indicate that orthodontic treatment does not pose a risk for the development of gingival recessions. However, in their conclusion they note that methodological differences, a wide range of ages at the assessments, and other confounders preclude the conclusion that orthodontic treatment does not pose a risk for the development of gingival recessions. Another important aspect of their results was the pattern of labial gingival recessions. At 5 years post-treatment, the two most commonly affected teeth in the maxillary arch were the first premolar and first molar. Approximately 14% of the maxillary first premolars and 6% of first molars exhibited labial gingival recessions. In the mandibular arch, the central incisors (10%) and the first premolars (8%) showed the highest prevalence of recession. The authors stated that the results from their sample were similar to that observed in epidemiologic studies for both orthodontic [51, 53, 72] and non-orthodontic samples.[21, 22, 24]

Recession and the labial movement of incisors

Several studies have sought to assess the relationship of gingival recession following labial movement of mandibular incisors in humans. A few have demonstrated the development of recession after this type of movement.[51, 52] Artun et al [51] evaluated patients with surgically treated mandibular prognathism. In their study, 29 individuals with more than 10 degrees of proclination of the mandibular incisors (as measured by the incisor to mandibular plane angle [IMPA]) and 33 individuals with minimal change in incisor proclination (less than 2 degrees) during the presurgical orthodontic phase were evaluated. Records were taken before treatment, after removal of appliances, and at 3-year follow up. A total of 21 patients in the proclination group and 19 in the minimal proclination group were available for a long term follow-up. The mean postoperative times at this examination were 7.8 years and 8.1 years, respectively. The number of teeth with recession was determined from the intraoral photographs. Recession was recorded if the facial CEJ was exposed. Among the patients with excessive proclination, significantly more recession occurred on the mandibular incisors (0.79 ± 0.98 mm) compared to the group with minimal change in IMPA (0.09 ± 0.29 mm) during active treatment ($p < 0.001$). This was also true from the end of treatment to the 3-year post-treatment follow up. Mandibular incisors in the excessive proclination group (0.52 ± 0.87 mm) had significantly more recession than the minimal change group (0.06 ± 0.02 mm) from the end of treatment to the 3-year follow up ($p = 0.01$). The increases from the 3-year post-treatment follow-up to the longer term follow-up showed

no statistically significant difference. The authors hypothesized that bone dehiscences occasionally occurred due to the labial movement of the incisors. These areas then underwent a relatively rapid gingival recession during the first couple of years and then stabilized after that.

Yared et al [52] evaluated the mandibular central incisors of 34 individuals (aged 18 to 33) during a follow-up examination ranging from 7 months to 3 years 11 months after orthodontic treatment. They measured recession during a clinical examination using a digital caliper on the buccal surface of the central incisors. Proclination of the lower incisors was evaluated using cephalometric superimposition measurements (IMPA). Statistical analyses showed no significant correlation between recession and the total quantity of proclination. However, they noted that in patients who developed gingival recession, 93% showed final incisor inclinations equal to or greater than 96 degrees. Their results showed that for the mandibular right central incisors, there was significantly more recession when the final inclination was greater 95 degrees ($p = 0.022$). The association was not statistically significant for the mandibular left central incisor, however, the same trend was observed. The authors concluded that the final inclination of the mandibular central incisors is much more important than the total amount of proclination of these teeth in the development of recession.

Other investigations have failed to support a relationship between lower incisor proclination and gingival recession.[16-18, 72, 73] For example, Ruf et al [16]

evaluated the facial gingiva of 98 patients treated with the Herbst appliance. Mandibular incisor movement was measured using pre- and post-treatment cephalograms. Recession was measured prior to treatment and 6 months after completion of treatment using intraoral photographs. Recession was recorded positive if the CEJ was apparent. Their results showed that overall recession was not significantly different before and after treatment. No correlation was detected between the amount of proclination of the lower incisors (measured using IMPA) and the incidence of recession. Djeu et al [18] had a sample of 67 individuals treated with fixed appliance therapy. Pre- and post-treatment records were available. Lower incisor movement was measured using the cephalographic measurements of IMPA and lower 1 to A point/Pogonion (L1-Apo). In their sample, the average IMPA at the beginning of treatment was 96.05 ± 7.1 degrees, and the average IMPA at the end of treatment was 97.09 ± 7.5 degrees. The average change in IMPA during treatment was 1.04 ± 6.62 degrees. Recession was measured from the CEJ to the gingival margin using intraoral slides, and corrected for magnification using the corresponding dental casts. Their results showed no significant correlation between labial incisor movement and recession. Furthermore, they separated their sample into two groups based on the amount of incisor movement. One group experienced proclination during the treatment, the other did not. A two-sample t-test was performed for IMPA to determine if there was a statistically significant difference between the proclined group and the group without proclination in regards to an increase in gingival recession. The t-test showed no difference between the two groups. As mentioned earlier, several other studies have come to similar conclusions.[17, 72-75]

Recession and expansion of maxillary teeth

Recession in the maxillary arch following expansion during orthodontic treatment has not been studied nearly as much as recession following proclination of the mandibular incisors. The few studies that have been completed have primarily been conducted following rapid maxillary expansion. Similar to the mandibular incisors, the results are inconsistent. In a study by Vanarsdall and Herberger [76], 55 patients (ages 8 to 13 at the time of treatment with a Haas expander) were recalled 8 to 10 years after rapid palatal expansion (10 to 10.5 mm over 3 weeks) and compared to 30 control patients (matched for age) who underwent edgewise non-expansion treatment. Recession was evaluated by comparing clinical crown heights. The study indicated that of the expanded cases, 20% of patients showed labial recession on one or more teeth, compared with only 6% in the non-expanded control patients.

Handelman et al [77] evaluated 47 adults (mean age 29.9 ± 8.0 years) who underwent rapid maxillary expansion as part of their treatment plan, and compared it to a control group of 52 adults (mean age 32.7 ± 7.4 years) who had orthodontic treatment without rapid maxillary expansion. All patients who received rapid maxillary expansion therapy had a Haas type expander and were expanded 0.25 mm per day until the maxillary palatal cusps were contacting the lingual inclines of the mandibular buccal cusps and stabilized for 12 weeks. Crown height was measured on the buccal surface of the maxillary premolars and first molars on the dental casts and used as a proxy for

attachment loss. From the beginning of treatment to the end of orthodontic treatment, the average changes in crown height for the premolars and first molars were between 0.3 to 0.6 mm. When compared to the control group, there were no significant differences for males. For females, the first premolars and first molars were significantly different. On average, the adult females who underwent rapid maxillary expansion experienced 0.5 mm more increase in crown height compared to the control group for both the first premolar and first premolar. The authors had access to long-term records for the Haas expander group for 21 out of the original 47 patients, but not for the control group. To evaluate recession at the long-term follow up, the clinical crown heights were measured again at least 5 years later. The average changes from the end of treatment to the long-term follow up were 0.5 ± 0.8 mm for the first premolar, 0.6 ± 0.7 mm for the second premolar, and 0.6 ± 0.8 mm for the first molar. The authors concluded that the amount of clinical crown increase was not clinically significant.

Time considerations following orthodontic treatment

Most of the studies that have not established a relationship between recession and orthodontic tooth movement in the labial direction have evaluated recession shortly after orthodontic treatment. Many neglect to evaluate the possible association of mandibular incisor movement and the development of recession in the long term. Only three studies have evaluated recession at least six months after appliances were removed, two of which were mentioned above. Artun et al [51], which was reviewed earlier, did show a

correlation. They demonstrated an increased incidence of gingival recession in the group that was proclined more than 10 degrees at a 3-year follow up appointment. The same primary investigator conducted another study.[72] Sixty-seven treated Class II patients were studied. Utilizing mandibular superimpositions, 45 patients treated with reverse-pull head gear to the mandibular dentition with a minimum of 1 mm of advancement at the CEJ (mean 2.18 ± 0.87) and a minimum of a 2 mm of advancement at the incisal edge (mean 3.87 ± 1.34) were identified. Additionally, 30 patients treated without headgear and who finished treatment at a similar time and age without any advancement of the CEJ (mean -0.43 ± 0.53) and a maximum of 1 mm advancement at the incisal edge (mean -0.26 ± 1.15) were also identified. A total of 30 patients from the pronounced advancement group and 21 patients from the no advancement group could meet for a follow-up examination a mean period of 7.83 years (± 4.44) and 9.38 years (± 4.39) after treatment, respectively. Prior to treatment, the mandibular incisors were more upright relative to the mandibular plane angle (IMPA), as well as relative to the line from A-point to pogonion, in the patients with pronounced advancement than in those with no advancement of the mandibular incisors. No differences in the final position of the incisors were found in the final position of the lower incisors at the time of appliance removal. Using intraoral photographs to assess recession, no differences in the number of mandibular incisors that developed recession from before treatment to after treatment, and from after treatment to follow-up, was detected. It was concluded that pronounced advancement of the mandibular incisors may be performed in adolescent patients with dentoalveolar retrusion without increasing the risk of

recession.[72] Yared et al [52], which was previously discussed in detail above, also showed no correlation in the amount of incisor proclination and recession at a follow up ranging from 7 months to 4 years. However, they noted that in patients who developed gingival recession, 92.86% showed final incisor inclinations of equal to or greater than 96 degrees. In this manner, the results showed that, for the mandibular right central incisor, there was statistical significance in relation to greater recession when the final inclination was above 95 degrees. Although the coefficient did not reach significance for the left central incisor, the same trend was noted. The authors concluded that the final inclination of the mandibular incisors, much more than the amount of proclination of these teeth, is an important factor.

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. The animal studies discussed earlier demonstrated a conservation of the soft tissue attachment up to 5 months after tooth movement was ended despite the formation of a bone dehiscence.[49, 50, 64] The result of this process was the development a long epithelial attachment. This was definitively shown in the histologic analyses completed by Karring et al [64] and Wennestrom et al [50]. Although it is reasonable to believe that a similar situation occurs in humans, the actual process that occurs is unknown. Moreover, the relationship of alveolar dehiscences and recession in the long term is also unknown, since in the animal studies mentioned above, the histologic analyses were completed only 3 weeks to 5 months after treatment. Again, a conservation of soft tissue

attachment was observed during that period. However, some of the authors speculated that given more time, recession may have ensued. Steiner et al [49] noted that the facial gingiva appeared stretched and thinned by the tension created by the facially directed orthodontic force. They also noted a persistent inflammation on the marginal gingiva. In this study, unlike the studies by Karring et al and Wennestrom et al, there was some loss of soft tissue attachment and recession, although it was not as great as the marginal bone loss. Wennestrom et al took more care to remove plaque from the area under study. However, in the one animal that did experience a loss of soft tissue attachment, inflammation was noted. Both authors speculated that the stretched and thinned tissue may have favored the destructive effect of the plaque associated inflammation. Hence, the thin inflamed facial tissue, which resulted following labial tooth movement, may have rendered the site more vulnerable to a process of recession and loss of attachment. This assumption is validated by the observation that in the presence of plaque-induced gingivitis, a thin gingival unit is more susceptible to complete breakdown than a thick one.[78-80]

The thin, unsupported soft tissue attachment may not only be more vulnerable to plaque induced inflammation, but to the other causes of recession as well, such as improper tooth brushing technique. Such speculations are reinforced by the high frequency of gingival recessions observed on the labial aspect of prominent teeth in persons who have not received orthodontic treatment.[58-60, 81] It has also been established that the distance between the apical end of the junctional epithelium and the crest of the alveolus

remains constant (average 1.07 mm). This distance, in addition to the normal length of the junctional epithelium (average 0.97 mm), constitute the biologic width.[14] It is possible that the failure of many of the studies that have evaluated the relationship between labial movement and gingival recession is due to the fact that the gingiva has not had enough time to be exposed to the trauma of tooth-brushing, mastication, and plaque-induced inflammation, as well as the re-establishment of the biologic width. Put another way, teeth with a thin unsupported soft tissue attachment may be more prone to the detrimental effects of inflammation and trauma over time.

In summary, 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 a few studies that focus on this subject, and they offer conflicting results. The literature on the maxillary posterior teeth is particularly scarce. The present study will further evaluate the long term prevalence of gingival recession following orthodontic movement of the mandibular incisors and maxillary premolars and first molars. The clinical orthodontist is regularly faced with crowded arches and must determine whether or not to expand the arch in order to make room for teeth. Limits must be established, and this study aims to help the clinical orthodontist in recognizing those limits.

CHAPTER II

LONG-TERM PREVALENCE OF GINGIVAL RESSION FOLLOWING LABIAL ORTHODONTIC TOOTH MOVEMENT

Introduction

By clinical definition, gingival recession refers to the exposure of the root surface by an apical shift in the position of the gingiva.[82] Recession is important because it can lead to poor esthetics [10, 11], tooth hypersensitivity [7], loss of periodontal support [83], difficulties in maintenance of oral hygiene [1, 9], and increased susceptibility to caries.[4-6] Although its etiology is not fully understood, periodontal disease [2, 28, 40] and mechanical trauma [2, 39, 41] are considered the primary factors in the pathogenesis of gingival recession.

Orthodontic treatment might also promote the development of gingival recessions.[54] It has been well established that orthodontic forces can move roots close to or through the alveolar cortical plates, leading to bone dehiscences.[49, 50, 64] In such instances, the marginal gingiva, without proper alveolar bone support, might be expected to migrate apically and lead to root exposure. This assumption is based on the fact that in areas of recession, a subjacent alveolar bone dehiscence is always present.[84] However, animal experiments have demonstrated little or no recession over the short-term associated with excessively proclined teeth, despite the development of bony

dehiscences.[49, 50, 64] This suggests that either more time may be necessary for recession to develop, or recessions do not necessarily occur when a dehiscence is created.

Clinically, the association between mandibular incisor proclination and recession remains unclear. Most studies evaluating recession shortly after treatment show no relationship.[16-18, 72, 74] However, the few studies that have investigated the long-term relationship between mandibular incisor proclination and recession are controversial.[51, 52, 72] Two of the studies showed no relationship [52, 72], while one study did.[51] However, in the one study that did indicate a relationship, the sample was relatively small (N = 40) and composed of adult individuals who underwent surgery for mandibular prognathism.[51] Only one long-term study has been completed on adolescents, which represent typical orthodontic patients.[72]

The purpose of the present study is to evaluate the long-term prevalence of gingival recession following orthodontic movement of the mandibular incisors and maxillary premolars and first molars. It will be the first study to evaluate the long-term effects of maxillary expansion on the buccal soft tissues. The specific aims are to:

1. Evaluate the prevalence and extent of recession immediately after orthodontic treatment and after a long-term follow up period.

2. Evaluate the relationship between mandibular incisor proclination during treatment and recession long-term.
3. Evaluate the relationship between maxillary expansion and recession long-term.

Materials and methods

Sample

A retrospective sample of 327 patients from two private orthodontic practices was evaluated. The selection criteria included records at the beginning of treatment (T1), the end of treatment (T2), and at long-term follow up (T3). Long-term follow up was defined as at least two years after appliances had been removed. A total of 205 patients met the inclusion criteria. Partially missing records or records taken too close to the appliance removal date were the primary reasons patients were omitted from the study. Approximately half of the sample was treated with premolar extractions. Most of the patients were in retention for three years; some had their retainer removed by their general dentist prior to three years. To eliminate the possibility of inflamed gingiva obscuring gingival recession, patients were excluded if the final treatment models and intraoral photographs were taken less than 2 weeks following debonding of the appliance.

Records

All necessary records were digitized and saved electronically. For each subject, the frontal and buccal intraoral photographs were scanned at the time orthodontic treatment was completed (T2), and at the long-term follow up (T3). In addition, the cephalometric radiographs were scanned at the beginning of treatment (T1), and at the end of orthodontic treatment (T2). Five standardized photographs of the dental models were taken at all three time points (T1, T2, and T3), including maxillary occlusal, maxillary frontal, maxillary right buccal, maxillary left buccal, and mandibular frontal. The following information was obtained from the patients' charts: ethnicity, Angle classification, expansion type (RPE or arch wire), extractions, retention type, retention duration (when available), and finally, the dates at each of the three time points.

Scanning and photograph specifications

Intraoral photographs were scanned on a HP Scanjet G4050 Photo Scanner at a resolution of 300 pixels per inch and saved as jpeg files. A limited number of previously digitized intraoral photographs were saved as jpeg files. The cephalometric radiographs were scanned using an Epson Perfection 4990 Photo Scanner at a resolution of 300 pixels per inch and saved as jpeg files.

The models were photographed using a Canon EOS Rebel T2i digital SLR camera with an EF-S 60mm f/2.8 Macro USM lens and Metz 15 MS-1 ring flash. A millimeter ruler was placed at the base of the model in order to calibrate the image in the photograph. The camera was mounted on a tripod and the distance between the camera lens and the model was fixed at 20 inches. Photographs of the models were taken with a black felt background using the auto-focus setting.

Measurements of gingival recession

Recession was measured on the lower incisors and the maxillary premolars and first molars on each side of the arch at the end of orthodontic treatment (T2) and at the long-term follow-up (T3). Recession on the mandibular incisors was defined as the distance from the gingival margin to the cementoenamel junction (CEJ) on the mid-facial surface.[15-17, 52, 53] Since the location of recession is more variable on the facial aspect of maxillary first molars, recession was defined as the maximum distance from the gingival margin to the CEJ anywhere on the facial aspect of the maxillary first molars. Whenever possible, intraoral photographs were used to measure recession because they have been shown to be more reliable than dental models.[17]

All of the images were imported into Viewbox 4 Cephalometric Software™. The dental models were calibrated using the millimeter ruler. The intraoral photographs were calibrated based on the ratio of the mesial-distal width of the maxillary central incisor at

its broadest point, as measured on the dental model, to the same width measured on the intraoral photographs:

Mandibular Incisor Recession = Photographic measured recession \times (mesial-distal width of maxillary central incisor measured on the model \div mesial-distal width of maxillary central incisor measured on photograph)

To measure recession at the maxillary premolars and first molars, a ratio was established based on the distance from the gingival margin to the cusp tip of the maxillary posterior teeth on the models, and the same measurement taken on the intraoral photographs:

Maxillary Posterior Recession = Photographic measured recession \times (distance from gingival margin to cusp tip of premolar or molar measured on the model \div distance from gingival margin to cusp tip of premolar or molar measured on the photograph)

When intraoral photographs were not available or the quality was poor (approximately 20% of the time), recession was measured on the dental models using Viewbox 4 Cephalometric Software™. Using models to measure recession has been shown to be both valid [15] and reliable [17].

Technical errors were based on randomly selected sets of replicates: 20 replicate intraoral photographs and 20 casts and associated intraoral photographs. The systematic

error for intra-oral photographs was not statistically significant, and the intra-class correlations ranged from 0.962 to 0.981. Systematic difference showed slightly (0.026 mm) larger cast than intra-oral photograph measurements for the maxillary first molars. Intra-class correlations between cast and intra-oral photograph measurements ranged from 0.931 to 0.959.

Measurement of changes in incisor inclination

Mandibular incisor inclination was measured using the pre- and post-treatment cephalometric radiographs. The radiographs were traced using Dolphin Imaging Software™. Nine cephalometric landmarks were recorded at T1 and T2, including Sella, Nasion, A point, B point, Pogonion, Menton, Gonion, Lower incisor tip, and Lower incisor root apex (Figure 1). The landmarks were used to calculate three angular measurements: IMPA ($L1_{tip}L1_{apex}/Go-Me$), Lower 1 to NB ($L1_{tip}$ to Nasion-B point in degrees), and finally the mandibular plane angle ($S-N/Go-Me$).

Based on replicate measures of 20 randomly selected cephalometric radiographs, there were no statistically significant systematic errors. The intra-class correlations were 0.997 for IMPA and 0.991 for L1 to NB.

Measurement of changes in arch width

The maxillary molar and premolar movements were measured using occlusal photographs of the pre- and post-treatment models (T1 and T2). The models were imported into Viewbox 4 Cephalometric Software™ and calibrated using the millimeter ruler at the base of the model. The inter-molar and inter-premolar distances were measured at the most lingual point at the lingual gingival margin to the same position on the corresponding contralateral tooth (Figure 2).

Based on replicate measures of 20 randomly selected casts, there were no statistically significant systematic errors. The intra-class correlations were 0.999 for all arch width measurements.

Statistical procedures

The data were analyzed statistically using SPSS software (SPSS Inc., Chicago, IL). Recession was not normally distributed, therefore it is reported in three ordinal groups: no recession, 0.1 to 1 millimeter (mm) recession, and greater than 1.0 mm recession.

Results

The overall age at the beginning of orthodontic treatment was 14.0 (± 5.9) years (Table 1). The overall age at the end of treatment was 16.5 (± 6.0) years, and the mean treatment time was 2.5 (± 1.1) years. The overall mean age at the long-term follow up appointment was 32.3 (± 8.5) years, and the overall time between the end of treatment and the long-term follow up was 15.8 (± 6.3) years.

Gingival recession

There was very little recession at the end of orthodontic treatment (Table 2). The vast majority of teeth (86.5-97.8%) displayed no recession. The mandibular central incisors showed the most recession, with 12.8% exhibiting 0.1 to 1.0 mm of recession and 0.7% with greater than 1.0 mm. The maxillary first premolars showed the second highest prevalence, with 7.9% exhibiting 0.1 to 1.0 mm of recession, and 1.2% exhibiting more than 1.0 mm of recession. The mandibular lateral incisors, as well as the maxillary second premolars and first molars all showed more limited amounts of recession, with less than 4% of the teeth showing recession at the end of orthodontic treatment. Overall, 18.3% of patients demonstrated gingival recession on at least one tooth after treatment, and only 5.8% of the teeth examined exhibited recession.

Each tooth showed significant ($p < 0.001$) increases in recession between the end of orthodontic treatment and the long-term follow up. The maxillary first premolars displayed the highest prevalence of recession at T3. Recession was evident for almost 60% of maxillary first premolars, but only 7.7% showed greater than 1.0 mm of recession. The mandibular central incisors showed the second highest prevalence of recession at T3, with almost 53% exhibiting recession, and 10.3% showing greater than 1 mm of recession. The mandibular lateral incisors, the maxillary second premolars, and the maxillary first molars all showed similar amounts of recession at the long-term follow up, with 32-37% of the teeth exhibiting recession. Overall, 55.7% of patients demonstrated gingival recession on at least one tooth at the long-term follow up, and 41.7% of the teeth examined exhibited recession.

From the end of treatment to the long-term follow up, the incidence of recession was highest for the maxillary first premolars and the mandibular central incisors (Figure 1). Recession occurred on the maxillary first premolars 58.1% of the time; 6.7% of the time it was greater than 1 mm. For the mandibular central incisors, recession occurred 51.2% of the time; 12.1% of the time it was more than 1 mm. The incidences of recession for the mandibular central incisors and maxillary first premolars were not significantly different ($p = 0.070$). The incidence of recession was less than 36% for the mandibular lateral incisors, maxillary second premolars, and maxillary first molars. The incidence of recession was significantly less ($p < 0.05$) for these three teeth than for the mandibular central incisors and maxillary first premolars. Recessions for the mandibular lateral

incisors, maxillary second premolars, and maxillary first molars were not significantly different from one another.

A significant ($R = 0.154$; $p = 0.033$) positive correlation was found between recession of the mandibular right central incisor and age. However, the left central incisor and both lateral incisors showed no statistically significant correlations. All of the maxillary teeth showed significant correlations with age ($p < 0.050$).

There was no statistically significant difference in post-treatment recession between Class I and Class II patients. The only significant sex differences pertained to the right ($p = 0.003$) and left ($p < 0.001$) first premolars, with females exhibiting more recession than males. There also was no consistent difference between non-extraction and premolar extraction patients. Only the right ($p = 0.050$) and left ($p = 0.012$) mandibular central incisors showed significant differences, with extraction patients exhibiting more recession.

Changes in lower incisor inclination

The pre-treatment incisor inclination, as measured by the incisor to mandibular plane angle (IMPA), was 93.5 ± 7.1 degrees. The IMPA did not change significantly ($p = 0.901$) during treatment. At the end of orthodontic treatment, the average IMPA was 93.3 ± 6.1 degrees. The post-treatment lower 1 to NB (L1-NB) was also not significantly

different than the pre-treatment measurement ($p = 0.629$). The maximum increase in angulation during treatment was 16.3 degrees, and the maximum decrease was -18.9 degrees. There were statistically significant differences in IMPA and L1-NB changes between extraction and non-extraction patients. In non-extraction patients, the IMPA increased an average of 1.8 ± 6.0 degrees, and in extraction patients it decreased 1.5 ± 6.6 degrees (Table 3). L1-NB showed similar differences.

There were no significant differences in incisor inclination as measured by IMPA or L1-NB between Class I and Class II patients ($p = 0.067$), or between male and female patients ($p = 0.210$).

The more retroclined the mandibular incisors were at the beginning of treatment, the greater they were proclined during treatment. This relationship was evident for both IMPA ($R = -0.575$; $p < 0.001$) and L1-NB ($R = -0.673$; $p < 0.001$). There was no statistically significant difference in mandibular incisor recession between patients whose final IMPA was $>95^\circ$ (69 patients) and patients whose final IMPA was $\leq 95^\circ$ (136 patients).

Changes in maxillary arch width

Pre-treatment inter-first premolar, inter-second premolar, and inter-first molar arch widths were 24.9 ± 2.6 mm, 29.5 ± 2.6 mm, and 31.2 ± 2.6 mm, respectively. Arch widths increased significantly ($p < 0.001$) at the first premolars (1.1 ± 2.0 mm), increased slightly at the second premolars (0.2 ± 2.6 mm), and decreased slightly at the first molars (-0.2 ± 2.2 mm). Arch width changes at the second premolars and first molars were not statistically significant. There was significantly ($p = 0.048$) more expansion of the first premolars in extraction (2.6 ± 1.7 mm) than in non-extraction (1.0 ± 1.9 mm) patients (Table 3). In contrast, there was significantly more expansion of the second premolars and first molars in non-extraction patients. Second premolar widths increased 1.7 ± 2.5 mm in non-extraction patients, and decreased -1.1 ± 1.7 mm in extraction patients ($p < 0.001$). The first molar widths increased 1.1 ± 2.0 mm in non-extraction patients, and decreased -0.9 ± 1.8 mm in extraction patients ($p < 0.001$).

No statistically significant differences in arch width changes were found between Class I and Class II patients. The only statistically significant ($p = 0.043$) difference between males and females occurred at the second premolar, which was constricted -0.6 ± 2.2 mm in males and expanded $0.4 (\pm 2.7)$ mm in females.

Relationship between tooth movements and recession

Of the eight possible associations, only the recession of the mandibular left lateral incisor and L1-NB were significantly related ($R = 0.279$; $p = 0.011$). The relation between recession of the mandibular left lateral incisor and IMPA changes approached significant levels ($R = 0.211$; $p = 0.057$).

There were several significant positive correlations between expansion and recession of the maxillary premolars and first molars (Table 4). Eight of the sixteen possible correlations between arch width changes and post-treatment changes in recession were statistically significant. However, there was no observable pattern.

Discussion

There were only limited amounts of recession at the end of orthodontic treatment. Approximately 18.3% of the patients in the present study demonstrated gingival recession on at least one tooth after treatment, but only 5.8% of the teeth exhibited recessions. Slutzkey and Levin [53] found that 22.9% of patients had gingival recessions following orthodontic treatment, while Renkema et al [15] noted that only 6.6% of patients had gingival recessions. In the present study, recession was assessed on color slides, whereas Renkema et al evaluated recession on plaster casts. Assessment of recession on intraoral slides has previously been shown to be the preferred method due

to the high number of unreadable teeth on plaster casts.[17] The higher prevalence noted by Slutzkey and Levin could be explained by the fact that they evaluated recession at 18-22 years of age, which is older than the average age at the end of treatment in the present study's sample (16.5 years).

In fact, the prevalence found after orthodontic treatment in the present study was similar to or slightly less than prevalences reported for untreated samples. Ainamo et al [21] reported that 8.7% of teeth among untreated 17 year olds had recessions, compared to 5.8% in the present study. Susin et al [24] noted recessions on only 2.9% of teeth among untreated 14 to 19 year olds, but they only recorded recession if it was 1 mm or greater. In the present study, only 0.6% of teeth had recessions 1 mm or greater after orthodontic treatment. This suggests that the recession after treatment in the present sample was typical for individuals of that age, and not caused by orthodontic treatment.

Substantial amounts of recession occurred during the 15.8 years that the patients were followed after orthodontic treatment. At approximately 32.3 years of age, 55.7% of the patients in the present study demonstrated gingival recessions on at least one tooth, and 41.7% of all teeth exhibited recessions. However, the severity of recession was limited, with only 7% of teeth exhibiting more than 1 mm of recession. Renkema et al [15] did not record the severity of recession in their study, but noted that 37.7% of their sample had recessions on at least one tooth five years after orthodontic treatment. Their lower prevalence may be explained by the different methodology used for recording

recessions, as well as their shorter follow-up duration (5 years vs. 16.5 years). Focusing only on the mandibular incisors of treated individuals, Allais and Melsen [17] noted recessions of 0.5 mm or more on 26.1% of teeth (average age 33.7 years), which was similar to the value of 25.4% observed in the present study. Considering the extent of recession, Thompson et al [85] showed that in treated individuals, 6.9% of teeth exhibited recessions of 1 mm or more at age 26, which compared closely to the 7% of the teeth identified in the present study.

The increases in recession observed after orthodontic treatment appear to be largely age related. Using data representative of the United States population (NHANES III), Albander and Kingman [22] showed that the prevalence and extent of recession in untreated individuals increases steadily with age, regardless of the threshold (i.e., 0 mm, 1.0 mm, etc.) used in defining recession. Approximately 38% of the individuals in their youngest age cohort (30 to 39 years) had gingival recessions on at least one tooth, and 8.6% percent of teeth exhibited at least 1 mm of recession. Their prevalence was slightly less than that in the present study, but the extent of recession was slightly greater. Methodology differences could again explain the discrepancy, because they measured recession during a clinical exam, which may be less sensitive than measuring recession on color slides that can be enlarged and manipulated. Susin et al [24], who evaluated a representative untreated sample from Brazil, also demonstrated significant increases in the prevalence and extent of recession with age. Approximately 96% of individuals in their 30 to 39 year old cohort demonstrated gingival recessions on at least

one tooth, and 44.3% of teeth exhibited recessions of 1 mm or more, both of which are much higher than the present study. Untreated reference data clearly show that significant increases in recession occur as individuals age, and that most of the increases in recession observed in the present sample were most likely normal aging effects.

The lack of association between mandibular incisor proclination and recession also demonstrates that the changes which occurred on the incisors were largely age related. The NHANES III data [22] demonstrated that 19.7% of mandibular central incisors and 13.2% of lateral incisors had recessions of 1 mm or more in individuals 30 to 55 years old. This compares very favorably with the results from the present study, where the central incisors showed recessions equal to or greater than 1 mm 10.3% of the time, and the lateral incisors 3.9% of the time. The lower prevalence in the present study can be explained by the younger age (32.3 years on average) of the sample.

Post-treatment gingival recession is greater for the mandibular central than lateral incisors. Compared to the laterals, the prevalence of recession on the mandibular central incisors was 15.8% more, and the extent of recession (≥ 1 mm) was 6.4% more. Ruf et al [16] showed that the prevalence of recession on the central incisors of treated individuals was 11.2% more than the laterals, while Renkema et al [15] noted that it was approximately 6% more. The central incisors have been shown to have a higher prevalence of recession in untreated samples as well. Among 30 to 55 year olds, the prevalence of recession greater than 1 mm was 6.5% more for the central than lateral

incisors.[22] Susin et al [24] reported an 8% difference in recession of 1 mm or more among untreated 14 to 30 year olds. It has been hypothesized that the difference is due to the more limited thickness of facial bone adjacent to the roots of the central than lateral incisors.[81, 86] The increased prevalence of recession on the central incisors does not appear to be related to differences in the thickness of the gingiva, because gingival thickness just apical to the base of the pocket on the central and lateral incisors is not significantly different in young adults.[87]

Importantly, the amount of mandibular incisor proclination that occurs during treatment does not appear to be related to the development of gingival recession. While some authors have postulated an association, the results of the present study, as well as numerous others [16-18, 72, 73], show no association. Moreover, animal experiments have demonstrated little or no recession in the short term on excessively proclined teeth, despite the development of bony dehiscences.[49, 50, 64] This suggests that more time may be necessary for recessions to develop, or recessions do not necessarily occur when a dehiscence is created. Of the few clinical studies that have investigated the long-term relationship between proclination and recession, only one has found a relationship.[51] However, their sample was relatively small (N = 40) and composed of adult individuals who underwent surgery for mandibular prognathism. In contrast, the present study, as well as two other long-term studies [52, 72], show no relationship. In the present study, as well as another study showing no relationship [72], the incisors that were proclined

the most during treatment were more upright prior to treatment, which may obscure a relationship.

This has led some authors to postulate that the final position of the mandibular incisors may be more closely associated with long-term recession than the amount of proclination that occurs during treatment.[52] However, the present study showed no difference in recession between individuals whose final IMPA was greater than 95° and those whose incisors finished at 95° or less. Renkema et al [88] also found no difference in recession 5 years after treatment between individuals with an average final IMPA of 90.8° and another group who finished at 105.2°. In contrast, Yared et al [52] reported more recession among patients whose final IMPA was greater than 95° compared to those who finished treatment at 95° or less, but their results were statistically significant for only the right central incisor. Moreover, their patients were older at the start of treatment (18-33), which is important because the ability of the periodontium to withstand orthodontic treatment appears to decrease as individuals age.[15] The discrepancies among studies may also be due to the fact that IMPA only measures changes in inclination; it does not measure whether or not the incisors were protracted or retracted.

While incisor proclination was unrelated, expansion was related to recession of the maxillary posterior teeth. Animal studies have previously demonstrated the development of buccal bone dehiscences after posterior expansion.[64, 68] In

orthodontic patients, buccal dehiscences in the maxillary arch have been demonstrated following arch wire expansion [67], as well as rapid and slow maxillary expansion.[89] Within a year after treatment, small but significant losses of attachment on the maxillary posterior teeth have been reported for patients who underwent rapid palatal expansion compared to those who did not.[77, 90] Longer term studies also indicate a relationship between expansion and recession.[76] The quantity of the hard and soft tissues adjacent to the maxillary posterior teeth may play an important role in the development of recession. Thicker gingival tissue has been reported facial to the maxillary second premolars than the first premolars and molars [87], which may impart some resistance to recession on the second premolars. This is important because thin gingival tissue (thin gingival biotype) is more prone to recession.[52, 73] Additionally, the amount of buccal bone adjacent to first premolars has been shown to be thinner than the bone adjacent to the other maxillary posterior teeth.[91-93] For instance, Horner et al [92], who evaluated the buccal cortical and medullary bone thickness from the maxillary canine to the first molar, showed that the cortical bone thickness was similar, but the medullary bone gets noticeably thinner from posterior to anterior. In the present study, the maxillary first premolars were expanded the most, which is typical in patients who have been expanded.[67, 94] The first premolars also exhibited the highest prevalence and extent of recession, which is in agreement with other treated samples [15], but different from untreated samples [22, 24] Therefore, a treatment effect seems likely, considering the different patterns of recession seen in the maxillary arch between treated and non-treated samples.

Treated and untreated samples exhibit different patterns of maxillary posterior recession. In the present study, as well as in other treated samples [15, 53], the maxillary first premolars exhibited the most posterior recession. While they showed almost twice as much recession as the second premolars and first molars, most of the recession was minor. Recession of 1 mm or more was present only 7.7% of the time. Renkema et al [15] reported that the maxillary first premolars exhibited recession approximately 15% of the time 5 years after treatment, compared to only 2% for the second premolars and 5% for the first molars. Again, their lower prevalences can be explained by the use of plaster casts, and to their shorter follow-up period. In contrast, most untreated samples have shown that the maxillary first molars exhibit more recession than the premolars.[22, 24] For example, Albander and Kingman [22] demonstrated that the first molars of individuals 30 to 55 years old exhibited slightly more recession (0.8%) than the first premolars, and substantially more (9%) than the second premolars. Susin et al [24] showed maxillary first molar recession approximately 26% of the time in individuals 30 to 49 years old, whereas the first premolars showed recession only 15% of the time.

Clinical implications

Orthodontic treatment does not appear to be a risk factor for the development of gingival recessions. Proclination of the mandibular incisors during orthodontic treatment is not a risk factor for recession if the incisors were upright to begin with. Expansion of the maxillary premolars and first molars does appear to be a risk factor for recession. However, the recession is very limited.

CHAPTER III
CONCLUSIONS

1. Gingival recession was limited after orthodontic treatment was completed.
2. Gingival recession increased long-term, however the severity of recession was limited.
3. There was no relationship between the amount of mandibular incisor proclination and gingival recession long-term.
4. There was a small but definite link between the amount of maxillary expansion and the amount of gingival recession.

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APPENDIX A

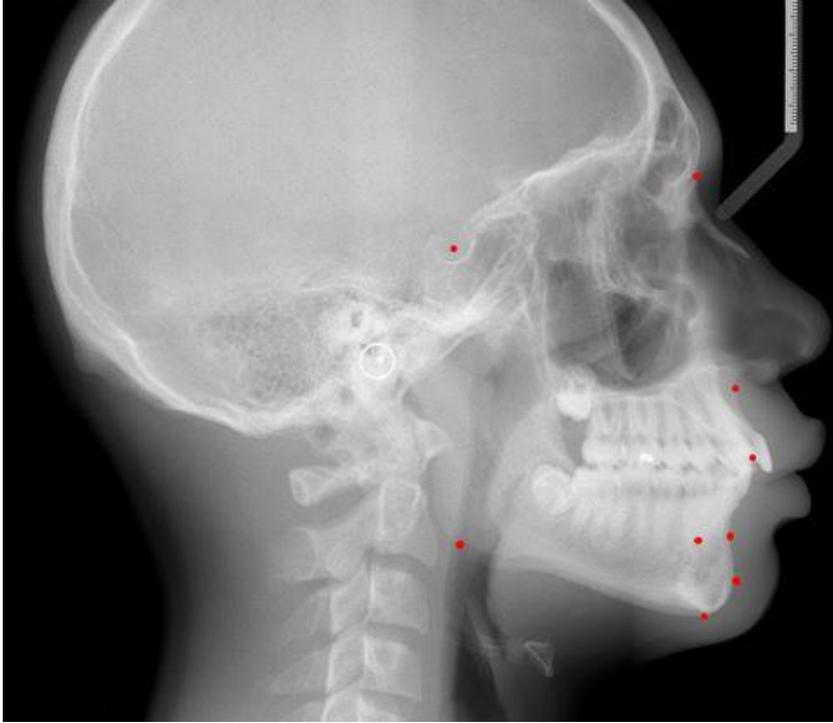


Figure 1. Cephalometric landmarks.

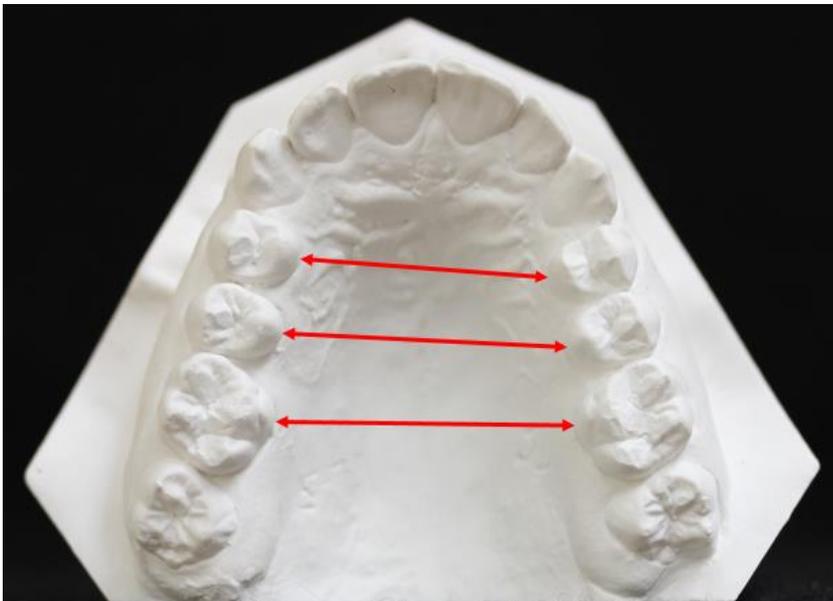


Figure 2. Inter-premolar and inter-molar width measurements.

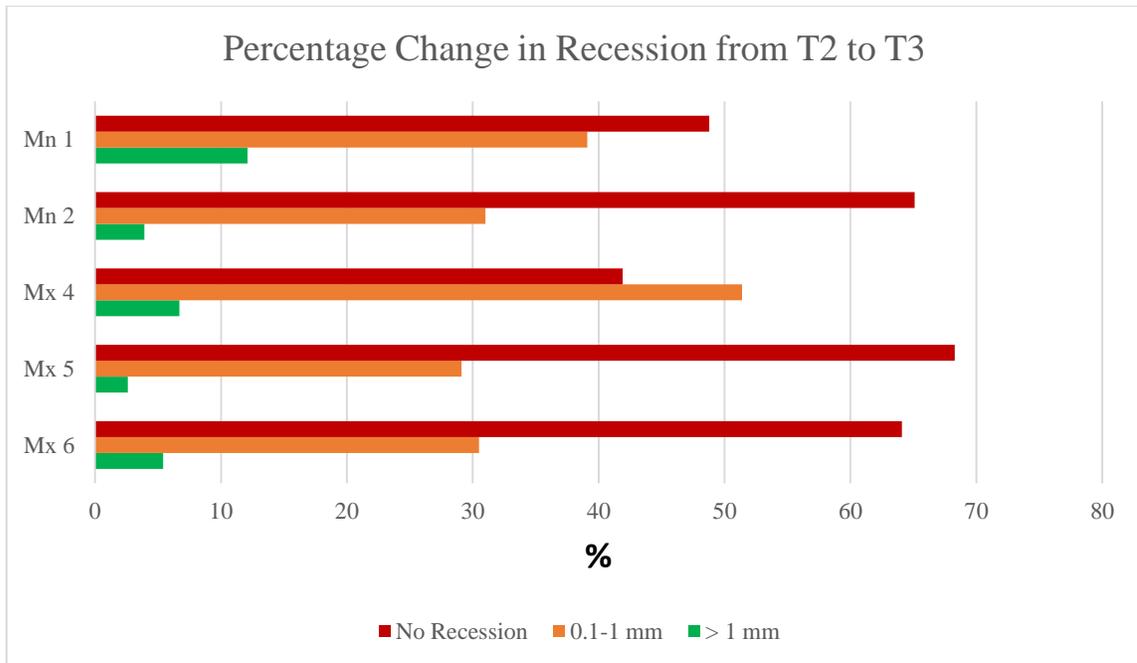


Figure 3. Percentage change in recession from the end of orthodontic treatment (T2) to the long-term follow up (T3).

APPENDIX B

Table 1. Sample sizes and ages (years) at pre-treatment (T1), post-treatment (T2), and long-term follow up (T3) for the overall sample and subgroups.

Groups	Subgroups	N	T1		T2		T3	
			Mean	SD	Mean	SD	Mean	SD
Overall	--	205	14.0	5.9	16.5	6.0	32.3	8.5
Sex	Male	43	13.2	2.4	15.6	2.3	30.5	7.3
	Female	162	14.2	6.5	16.8	6.6	32.7	8.7
Treatment¹	Non-Ext	84	13.7	5.7	15.8	5.9	29.6	8.2
	Ext	109	13.8	5.5	16.7	5.5	34.1	7.7
Angle Class²	I	90	14.6	5.9	17.1	6.1	33.3	8.7
	II	108	13.0	5.3	15.6	5.3	30.9	8.1

¹ Premolar extraction patients only

² Class III patients omitted

Table 2. Technical errors [intra-class correlations (ICC) and systematic differences (SD) with probabilities] of measuring recession. Comparisons include replicate intraoral (IO) photograph measurements and intraoral photograph versus cast measurements.

Tooth	IO Photographs			IO Photographs vs. Cast		
	ICC	SD	Prob. Diff.	ICC	SD	Prob. Diff.
Lower 1s	0.981	-0.015	0.393	0.959	< 0.001	1.000
Lower 2s	0.974	-0.013	0.405	0.943	0.003	0.906
Upper 4s	0.973	< 0.001	1.000	0.951	-0.033	0.224
Upper 5s	0.979	0.003	0.767	0.931	0.013	0.303
Upper 6s	0.962	< 0.001	1.000	0.947	-0.026	0.040

Table 3. Percentage of teeth with gingival recession at the end of orthodontic treatment (T2) and the long-term follow up (T3).

Time point	Recession (mm)	Teeth				
		Mn 1s (%)	Mn 2s (%)	Mx 4s (%)	Mx 5s (%)	Mx 6s (%)
T2	0	86.5	96.3	90.9	97.8	97.2
	0.1 – 1.0	12.8	3.4	7.9	1.2	2.5
	> 1.0	0.7	0.3	1.2	1.0	0.3
T3	0	47.3	63.1	40.4	67.8	64.2
	0.1 – 1.0	42.4	33.0	51.9	29.1	30.2
	> 1.0	10.3	3.9	7.7	3.1	5.6

Mn: Mandibular; Mx: Maxillary

Table 4. Treatment changes in IMPA and arch width (AW) for non-extraction and premolar extraction patients, with probability differences.

Variable	Non-Extraction		Premolar Extraction		Prob. Diff.
	Mean	SD	Mean	SD	
IMPA (°)	1.8	6.0	-1.5	6.6	p < 0.001
L1-NB (°)	1.8	5.9	-1.7	6.6	p < 0.001
AW Mx 4s (mm)	1.0	1.9	2.6 ¹	1.7	p = 0.048
AW Mx 5s (mm)	1.7	2.5	-1.1	1.7	p < 0.001
AW Mx 6s (mm)	1.1	2.0	-0.9	1.8	p < 0.001

¹ Patients in which second premolars were extracted instead of first premolars

Table 5. Correlations between recession and maxillary arch width changes during treatment.

Recession	Changes in Arch Width					
	4s		5s		6s	
	R	Prob	R	Prob	R	Prob
Mx Right 6	0.202	0.035	0.173	0.026	0.182	0.008
Mx Right 5	0.233	0.015	0.141	0.052	0.086	0.121
Mx Right 4	0.306	0.003	0.354	0.002	0.135	0.109
Mx Left 4	-0.093	0.203	0.407	<0.001	0.090	0.204
Mx Left 5	-0.063	0.283	-0.081	0.179	-0.076	0.153
Mx Left 6	0.212	0.029	0.063	0.240	0.094	0.110