Gingiva and Orthodontic Treatment

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Orthodontic appliances, as well as mechanical procedures, are prone to evoke local soft-tissue responses in the gingiva. These effects can either be of positive nature, (physiologic recontouring), helping tooth movement, or negative ones, which should be avoided. The main source of negative outcomes involves orthodontic attachments, which inhibit efficient removal of bacterial biofilms (dental plaque). Undesirable complications are often due to an understandable lack of awareness while the orthodontist focuses on biomechanical matters. While conscientious attention to biomechanical progress justifies this focus, close attention should be paid to infection control and the possibility of iatrogenic side effects. This article considers the issues of ideal orthodontic clinical management as well as those of inadequate patient compliance and infection management. Exactly how therapeutic, prophylactic, and anti-infective issues are assumed or delegated by the orthodontist, patient, or the referring dentist is a matter of individual practice style and an integral part of the doctor-patient covenant. This article attempts to provide current information regarding clinical, microscopic, and molecular level effects of orthodontic tooth movement on gingival tissues during fixed appliance therapy, or remedial methods once orthodontic appliances are removed. (Semin Orthod 2007;13: 257-271.)

The periodontium can be divided anatomically between the gingival unit (the soft tissue coronal to the bony crest of the alveolus in health), and the periodontal attachment apparatus, defined by the cementum, the periodontal ligament (membrane), and the cribriform plate of the alveolus. While gingival disease must precede periodontal infection, not all gingival diseases progress to periodontitis. Because of the unpredictable nature of the disease progression, all orthodontic patients with inflamed gingiva must be considered to be at risk for periodontal damage. For the purposes of syntactical clarity the words "periodontium" and "periodontal diseases" will refer to both anatomical elements, unless otherwise specified.

Gingival and periodontal diseases are influenced by a wide variety of factors, such as host resistance, social and behavioral characteristics, which affect belief values and compliance, respectively, compromised systemic resistance (eg, human immunodeficiency virus status), genetic predispositions, tooth level, and finally both quantitative and qualitative compositions of the bacterial biofilm (dental plaque) at the gingival margin. As new discoveries in molecular genetics and the science of virology and bacteriology progress, refinements in concepts of disease risk factors emerge almost annually.[1] Thus, it behooves the orthodontists to understand both the physiology and the pathophysiology of the foundational tissues, as well as the coronal elements

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that have traditionally defined the specialty. Within these anatomical and disease entities, tooth anatomy, appliance design, and composition of dental plaque are considered to be paramount local factors, which influence periodontal health.[2] Among tooth level risk factors contributing to the etiology of periodontal diseases (gingivitis and periodontitis), arch length deficiency (crowding) and direct soft-tissue impingement are most salient.[1-3] The exact mechanism contributing to disease in any individual patient is still not clearly defined or foreseeable. However, the malalignment of teeth can adversely affect gingival health, since the amount of plaque at the gingival margin around teeth correlates rather strongly with gingival inflammation and bleeding. It was reported that at extremes of oral hygiene, the pathological contribution of malocclusion may be eclipsed by more profound etiological agents, causing gingival or periodontal disease.[1,2,4] However, this correlation does not necessarily deny the role of arch length deficiency (ALD), or direct gingival traumatic impingement, as risk factors in nonextreme cases. The good news for the practicing orthodontist is that given adequate instruction, gingival infection can be brought under reasonable control. For example, Addy and coworkers reported that all children who were included in their study sample and in need of orthodontic treatment were plaque free or free from gingival bleeding on probing.[4] Yet, in keeping with the concept that ALD may be a risk factor in non extreme cases, Ashley and coworkers reported that overlapping incisors had a direct effect on gingivitis.[5] Furthermore, Ainamo stated that the degree of oral cleanliness and extent of periodontal disease were worse around malaligned teeth than around properly aligned teeth.[6] Thus, it may be concluded that a crowded dentition can complicate oral hygiene procedures, leading to increased plaque retention and subsequent gingival inflammation.

This article attempts to provide current information regarding clinical, microscopic, and molecular level effects of orthodontic tooth movement on gingival tissues during fixed appliance therapy, or remedial methods once orthodontic appliances are removed.

Clinical Changes

The introduction of fixed orthodontic appliances into the oral cavity in the form of orthodontic bands and resin-bonded attachments often evokes a local soft-tissue response inconsistent with health or esthetic treatment goals. The proximity of these attachments to the gingival sulcus, plaque accumulation, and the impediments they pose to oral hygiene habits further complicate the process of efficient salutary orthodontic care.[7-10] The effects seen clinically following the insertion of orthodontic appliances into the oral cavity can contribute to chronic infection, inflammatory hyperplasia, gingival recession, irreversible loss of attachment (permanent bone loss), and excessive accumulation of tissue, inhibiting complete extraction space closure. The following discussion addresses each of these pathological issues in detail.

Inflammatory Changes

Orthodontic mechanotherapy is capable of producing local changes in the oral microbial ecosystem and altering the composition of the bacterial plaque qualitatively and quantitatively. Generally, as plaque accumulates, especially subgingivally, relatively benign Gram-positive cocci (commensal organisms) forms relent to the development of more pathogenic Gram-negative rods, spirochetes, and motile forms that define the pantheon of putative pathogens (periodontopathic bacteria), many of which are uncharacterized and not culturable for in vitro analysis. The development of a stable pathogenic milieu tips the host-parasite homeostasis in favor of the pathogen and manifests as clinical inflammation. This trend is evident by the increased severity of gingival inflammation observed immediately after fixed appliance placement. Fixed appliances frequently encroach on the gingival sulcus, inhibiting effective oral hygiene maintenance.[11] Zachrisson and Zachrisson reported that even after maintaining seemingly excellent oral hygiene, patients usually experience mild to moderate gingivitis within 1 to 2 months of appliance placement. These infective changes are generally quiescent, with no permanent damage introduced to tissues, except for 10% of adolescents, who might show considerable irreversible periodontal attachment apparatus destruction.[7,8] This finding is similar to that

of Kloehn and Pfeifer, who evaluated pretreatment gingivitis in prospective orthodontic patients with the help of Russell's periodontal index, and reported a gingivitis prevalence of approximately 8%. When an orthodontic appliance was placed, there was a sudden drop in the number of patients who could maintain an excellent oral hygiene from 20% to 6.5%. However, a dramatic improvement in the gingival condition was observed 48 hours after appliance removal, as indicated by very low Russell index scores.[9]

Clinical studies used various indices for evaluating gingival inflammation after orthodontic appliance placement. The plaque index, gingival index, bleeding on probing, pocket probing depth, Quigley-Hein index (for bonded maxillary and mandibular molars), bonded bracket index (for bracketed teeth), and a modified gingival index, all were used for assessment of pre- and post-treatment gingival conditions.[12-17] Virtually all studies have reported that orthodontic appliances act as protective havens for bacterial plaque accumulation, providing an encumbrance to oral hygiene procedures.

Mucogingival Problems

The zone of attached gingiva in health is defined as the amount of keratinized tissue from the gingival margin apical to the mucogingival junction, minus the depth of the gingival sulcus. Assessment of the mucogingival status is considered to be a very important part of the intraoral examination, if orthodontic treatment is to be planned and rendered. It has been surmised by anecdotal evidence and case studies that extreme labial or lingual positioning of teeth may be associated with gingival recession and an inadequate zone or thickness of attached gingiva.[18,19] However, a strong predictive coefficient of correlation has not yet been unequivocally demonstrated. Thus, while some recession (gingival dehiscence) may be predicted in orthodontic cases epidemiologically, the lack of strong correlation coefficients makes individual patient proclivity so problematic and the emergent pathosis so unforeseeable by the practicing orthodontist. This unpredictability is why a policy of prophylactic soft-tissue grafting and preorthodontic periodontal consultation may be recommended as prudent for all orthodontic patients wherever the attached gingiva is minimal or thin.[20-23]

Direct Gingival Traumatic Impingement

In patients with a Class II, Division 2 malocclusion, functional trauma from incisor impingement on the mandibular soft tissue can result in marginal recession of facial gingiva of mandibular incisors.[24] Similarly, extreme cases of deep bite (complete deep bite), direct trauma to the gingiva from the incisal edges of mandibular incisors can contribute to gingival recession palatal to maxillary incisors.[18] Such traumatic damage to gingival tissues might result in the complete ablation of the gingival unit providing a portal of entry through which infection could spread to the subjacent periodontal attachment apparatus.[25] When such a process is allowed to occur, the periodontal status of the patient is compromised, because the depth of the pocket beyond 3 mm inhibits complete bacterial biofilm removal subgingivally by common home care techniques, and even professional scaling and root planning. This of course complicates the application of orthodontic mechanics, or renders it absolutely contraindicated because, as Wennstrom and Pini Prato[26] have speculated, tooth movement can convert supragingival plaque subgingivally. However, a prudent orthodontic treatment plan and a few simple precautions can ameliorate or entirely preclude such unfortunate complications as gingival recession or periodontal attachment loss[11]:

1. A low profile appliance, which facilitates access for effective oral hygiene management.
2. A complete and explicit informed consent of complications and sequelae before treatment commencement, which sets a goal of gingival health to improve the esthetic relationship of the gingival margin.
3. Maintenance of interproximal health, to preclude development of papilla loss (black triangle phenomenon) when incisor ALD is corrected.

After full discussion of risks and benefits, the orthodontist should allow the well-informed patient to determine whether the benefits of orthodontic treatment outweigh the side effects on gingival health. There is no doubt that the insertion of an orthodontic appliance makes

maintenance of oral hygiene difficult. Most patients undergoing fixed appliance treatment experience an increased incidence of gingivitis throughout the duration of therapy, but if the qualitative nature of the gingival infection is noncariogenic, and nonprogressive into the periodontal attachment apparatus, the permanent destructive effects may be minimal, remediable, or precluded entirely.

Gingival Enlargement

One of the most common problems with gingivitis associated with orthodontic treatment is gingival overgrowth (Figs 1, 2, and 3). The affected tissue is generally edematous, and may bleed when gently probed.[14] The first review regarding gingival overgrowth appeared in 1933, in volume 3 of *The Angle Orthodontist*.[27] Kloehn and Pfeifer evaluated in detail the nature and degree of gingival enlargement after orthodontic appliance placement. They reported that the average incidence of gingival enlargement was 4 times greater around posterior teeth compared with incisors and canines.[9] They listed the following causes:

1. Mechanical irritation by bands, more on posterior than on anterior teeth,
2. Chemical irritation produced by cements used for banding,
3. Food impaction, because of the proximity of the arch wires to the soft tissues, and
4. Less efficient oral hygiene maintenance.

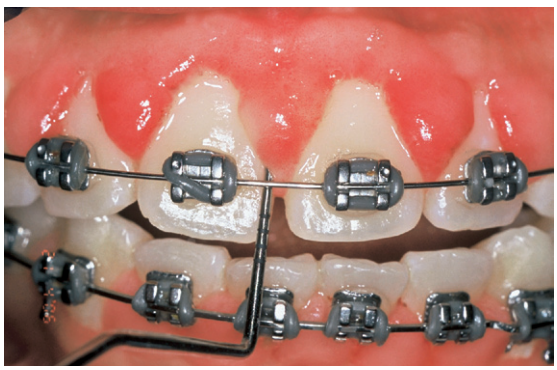

Figure 1. This kind of papillary hypertrophy is caused by bacterial biofilm accumulation below the interproximal gingival margin. Prolonged presence converts hypertrophic tissue to fibrous hyperplasia that must be removed either during fixed appliance therapy to facilitate adjustments or around the debonding appointment. Caution must be exercised to distinguish between hypertrophy that regresses and permanent hyperplasia that may look normal but represents redundant tissue growth covering a portion of the anatomic crown. (Color version of figure is available online.)

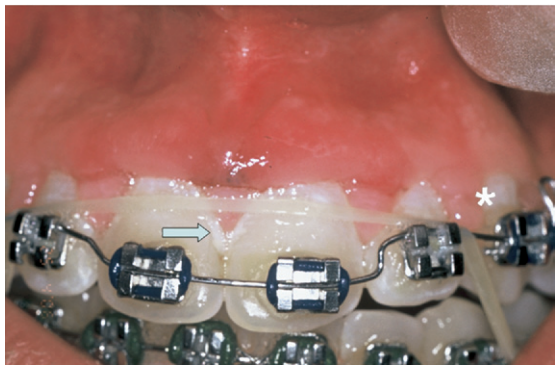

Figure 2. Inaccurate placement of elastics can inhibit oral hygiene efforts accumulating plaque (arrow) and adding mechanical irritation to the hypertrophied tissue. Black asterisk marks line of erroneous elastic placement on gingiva and maxillary canines. (Color version of figure is available online.)

Those investigators also reported greater incidence of gingival enlargement at the interdental region compared with the facial aspect of the gingiva margin. They concluded that as long as a band is in place, it is prone to produce gingival irritation, leading to enlargement. This situation can be prevented only by properly fitting each band and making it self-cleansing.[9] Those findings were in contrast to those of Zachrisson, who found that the mandibular incisor region harbors the highest risk for the development of

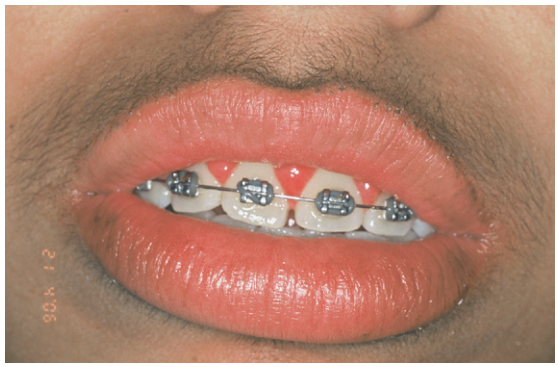

Figure 3. Lip incompetence can contribute to gingival enlargement by dehydrating the gingival during mouth breathing. (Color version of figure is available online.)

gingival hyperplasia.[7,8] Zentner and coworkers evaluated the proliferative response of cells of dentogingival junction to mechanical stimulation in male rats, and reported that junctional epithelium rapidly adapts to mechanical stimulation by cell proliferation. Their findings were contradictory to the existing dogma, and they concluded that orthodontic tooth movement need not necessarily produce any detrimental effect on the stability of the dentogingival junction.[28]

Published reports indicate that there are definite changes in gingival characteristics once orthodontic appliances are in place. Zachrisson reported consistently higher gingival index values and deeper periodontal pockets at interproximal surfaces, once orthodontic banding is completed.[7,8] Recent studies have also demonstrated an increase in probing depth after placement of orthodontic appliances.[13,15,16] Existing evidence supports the hypothesis that these defects may be only pseudo-pockets, which may or may not return to a normal topography once the appliances are removed. Kloehn and Pfeifer could not find any radiographic evidence of pathology in patients with changes in clinical crown height. They could also demonstrate a dramatic reduction in gingival hyperplasia within 48 hours of appliance removal,[9] but there is no definitive proof that "normal" gingival margins will necessarily return to a physiologically optimal position at the cemento enamel junction. Normal is a mathematical term meaning "most, median, or arithmetic mean" on a Gaussian distribution. This definition, however, is not synonymous with optimal health.

Gingival Recession and Loss of Attachment

Gingival recession is defined as "the exposure of the root surface by an apical shift in the position of gingiva."[29] It depends on the existence of a subjacent alveolar bone dehiscence and is always the result of a loss of attachment.[26] To date, no single causative factor has been identified as a singular etiologic agent, but many predisposing and precipitating factors have been anecdotally implicated in its development. The predisposing factors are anatomical, whereas the precipitating factors consist of trauma or exacerbation of acceleration of gingival inflammation, and alveolar bone dehiscences.[30]

An association between orthodontic tooth movement and gingival recession has been mentioned in both the orthodontic and the periodontal literature, with some reports arguing on behalf of a causal connection and others arguing against it.[31-33] Geiger reported that the incidence of gingival recession with fixed orthodontic appliances ranges from 1.3% to 10%. It is accepted that a 2-mm-wide attached gingiva is adequate to withstand orthodontic forces and prevent gingival recession. Moreover, it is argued that preexisting mucogingival problems can be exacerbated with orthodontic force application.[34] Therefore, it seems to be prudent and useful to identify and localize gingival areas at risk, where recession occurs, and advise patients of the anecdotal association, accordingly.

Dorfman suggested that mandibular incisors may be more prone to recession than any other teeth.[35] He attributed this recession to a thin or nonexistent labial plate of bone, inadequate or absent keratinized gingiva, and labial prominence of teeth. When excessive forces are applied, which do not permit repair or remodeling of bone during tooth movement, teeth with inadequate attached gingiva might show localized recession. It should be noted, however, that Dorfman explicitly noted that this association was unpredictable; thus inference of causal connection may be intemperate. An additional predisposing factor that may be more relevant is chronic marginal gingivitis, or chronic necrotizing ulcerative gingivitis, which may rapidly destroy the marginal alveolar bone and gingival attachment, even during application of modest orthodontic forces.[34] While this hypothesis is disputed by some,[36] research by Aleo and coworkers[37] and others[38,39] suggests that bacterial biofilm factors may inhibit cell proliferation necessary to adapt to a repositioning of the dental root. Further research demonstrating a lack of tissue proliferation in the presence of marginal dental plaque would support such an interpretation by compelling in vitro data.

Other biomechanical therapeutic modalities associated with anterior gingival recession logically include transverse expansion and intermaxillary (interarch) springs and elastics. In such cases, a breakdown of fragile gingival attachment may occur.[40,41] However, despite these reports, the movement of teeth within their alveolar bone envelope was suggested as safe by

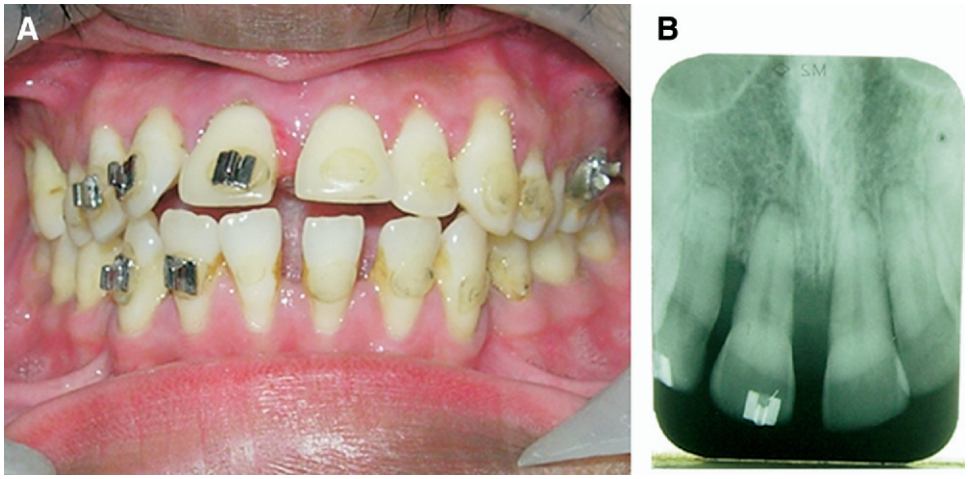

Figure 4. Image (A) shows the consequence of faulty mechanics by a general practitioner. Note improper bracket placement, improper removal of adhesive flash resulting in plaque harboring areas, and gingival recession in the mandibular anterior region. Note also the position of molar band, which is prone to induce attachment loss, gingival recession, and infrabony pocket formation. Image (B) is the radiograph of the maxillary anterior region from the same patient. Note the amount of bone loss and root shortening. (Picture courtesy of Arun Sadasivan, MDS, periodontist, Trivandrum, Kerala, India.) (Color version of figure is available online.)

Wennstrom and coworkers.[42,43] Those investigators reported that as long as a moving tooth remains within the envelope of alveolar bone (and presumably the plastic limits of epigenetically determined phenotype), the risk of harmful side effects in the gingival tissue is minimal. Wennstrom and coworkers concluded that the careful examination of the quality of the tissue, in addition to merely its linear dimensions, is important before applying orthodontic mechanics.[42,43]

Whatever the predisposing or precipitating agents, a combination of causative factors are paramount; gingival recession can lead to poor esthetics, root sensitivity, loss of periodontal support, difficulties in maintenance of oral hygiene, and achieving successful periodontal repair, as well as promoting increased susceptibility to caries[32] (Figs 4 and 5). However, a number of recent studies seem to support the hypothesis that orthodontic mechanics per se rarely produces gingival recession, and that a poor correlation exists between the degree of mandibular incisor proclination and gingival recession. Melsen and Allais reported that only 15% of teeth experience either the development or aggravation of gingival recession with orthodontic mechanics.[30] They listed local anatomical factors and the periodontal health status of these teeth as predisposing factors to this process. Besides gingival recession, the essential changes during periodontal destruction include loss of attachment and proliferation of pocket epithelium beyond the cemento-enamel junction.[8] Zachrisson and Alnaes demonstrated loss of attachment in orthodontic patients, and attributed it to variations in the gingival condition, the traumatic effect of increased thoroughness of tooth brushing, and

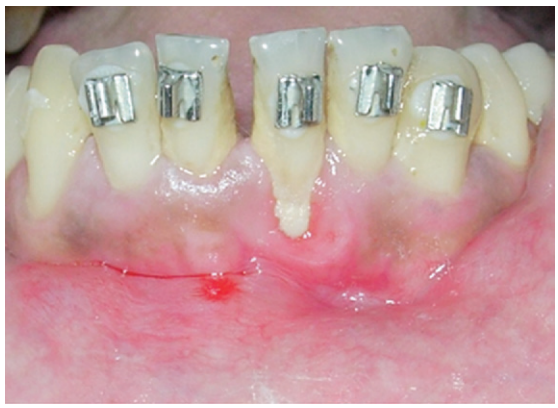

Figure 5. Another case of poorly executed orthodontic treatment. Note the improper bracket placement, improper removal of adhesive flash resulting in plaque harboring areas, and gingival recession in relation to mandibular anterior. (Picture courtesy of Arun Sadasivan, MDS, periodontist, Trivandrum, Kerala, India.) (Color version of figure is available online.)

placement of orthodontic bands. They found a mean attachment loss of 0.41 mm in patients wearing fixed appliances, compared with 0.11 mm in the control group, and concluded that this difference was not significant clinically.[8] However, it should be kept in mind that individual orthodontists treat individual patients and arithmetic means may have value as general guidelines to treat and inform patients but have little if any predictive (statistical forecasting) robustness for the next patient requiring treatment.

In other words, although the literature reports conflicting findings on possible associations between gingival recession and orthodontic mechanics, it seems prudent to emphasize the importance of a careful clinical examination, application of optimal forces, and control over tooth movement as a means to avoid or prevent this problem.

The Gingiva and Closure of Extraction Spaces

The application of retraction forces, as well as compressive forces in extraction sites for tooth approximation, often result in accumulation of gingival tissue and enlargement of interdental papillae. This type of gingival cleft formation is also observed when a couple is applied with the help of elastic chains for rotation corrections (Fig 6). Adjacent to this accumulated tissue, vertical invaginations or clefts, of both epithelium and connective tissue, are formed on both buccal and lingual sides.[44] It is suggested that transseptal fibers may be compressed or displaced, rather than remodeled during tooth movement, and that the invagination is the result of passive folding of gingival tissues.[45,46] Histological and histochemical studies have shown that hyperplasia of epithelium and connective tissues is associated with a loss of collagen and an increase in glycosaminoglycans.[44,47] The soft-tissue invaginations formed may vary from a shallow groove to a definite cleft extending to the alveolar bone surface, exceeding 1 mm in depth. Its presence may even be augmented with a large osseous defect in the pressure side of the moving tooth.[48] It was suggested that the anatomical configuration of the accumulated tissue causes difficulty in plaque control, and might also result in extraction space reopening, as well as relapse.[44]

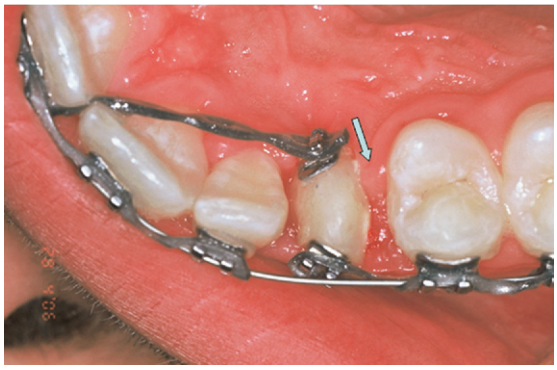

Figure 6. Note the accumulation of gingival tissue between the cuspid and bicuspid (arrow)—gingival cleft formation. This may inhibit tooth movement and surgical excision may be needed after debonding to establish physiologic soft-tissue contour. (Color version of figure is available online.)

Gingiva in Systemic Diseases and Drug Intake

Gingival enlargement is a common finding associated with some pathologic conditions. Erythematous gingival enlargement is often associated with uncontrolled diabetes. Inadequate nutrition and systemic hormonal stimulation often leads to puberty- and pregnancy-associated gingival enlargement, respectively.[49] Enlarged, edematous gingiva, soft and tender to touch, which bleeds easily on mild trauma, is a characteristic feature of acute monocytic, lymphocytic, and myelocytic leukemia. In addition, thrombocytopenia and thrombocytopathy can cause gingival enlargement and bleeding. All these conditions may get worse, if oral hygiene maintenance is poor and the rate of plaque accumulation is high.[50]

The literature contains many references suggesting that some drugs consumed for systemic diseases may contribute to gingival enlargements. Phenytoin sodium, nifedipine, and cyclosporine are the most cited drugs contributing to this type of reaction, that can include both interdental papilla and marginal tissue.[51,52] Some generalized syndromes are also known to exhibit characteristic gingival enlargement. These include Rutherford syndrome, Cross syndrome, Ramon syndrome, and Laband syndrome.[53,54] In all of these conditions, orthodontic attachments might act as plaque-harboring areas exacerbating the predispositions of the general syndrome itself. Clinicians treating such individuals should always be aware of the consequences they might face, try

to minimize appliances that would contribute to plaque retention, and ensure that aggressive infection control measures are taken by appropriate personnel.

Microbiological Changes

Placement of an orthodontic appliance in a patient's mouth is often associated with alterations in the oral hygiene habits and periodontal health, as a local change in the oral ecosystem alters the qualitative nature of the local bacterial biofilm. Literature regarding this effect of orthodontic treatment has outlined the changes in microbial environment associated with the appliance placement, along with the increase in the amount of supra- and subgingival plaque.[55-57] Specifically, orthodontic appliances seem to offer an opportunity to shift plaque composition from a predominance of aerobic Gram-positive cocci to more destructive putative pathogens, comprised mainly of facultative and strictly anaerobic Gram-negative species.[57,58] This shift results in populating the area with potential periodontopathogens such as fusiform bacteria spirochetes, prevotella, and bacteroids. These bacteria have the potential to produce cytotoxic products, which include an array of enzymes capable of hydrolyzing gingival tissues.[59]

The gingival/microbiologic changes associated with fixed orthodontic appliances can be attributed to the presence of rough-surfaced banding material acting as a "plaque trap" and irritant to gingival tissues. The plaque-retaining areas created by the orthodontic appliances increase the possibility of transforming reversible gingivitis to irreversible and self-perpetuating periodontitis.

This exact biochemical process is due to the production of endotoxins and lipopolysaccharides (LPS) from the cell wall of Gram-negative bacteria on cell death. LPS is demonstrated in subgingival plaque as well as in gingival crevicular fluid of orthodontic patients. LPS is capable of producing inflammatory reactions, which appears to be the predominant mechanism of tissue destruction, leading to periodontal attachment loss, alveolar bone loss, and gingival recession. Aside from actively producing destructive processes, the pathologic process can also induce several biologic pathways at the same time that it inhibits the healing capacity of gingival tissues. LPS is capable of activating the complement system and inducing inflammation through macrophage release of inflammatory mediators, such as interleukin (IL)-1, tumor necrosis-alpha (TNF-α), IL-6, and IL-8. This activity can, in turn, stimulate bone resorption and inhibit osteogenesis. Knoernschild and coworkers demonstrated that orthodontic brackets retain an affinity to LPS, which is dependent on bracket material composition, surface energy, and surface porosity.[60]

Sinclair and coworkers demonstrated an increase in the percentage of streptococci and a decrease in percentage of actinomyces in subgingival plaque from orthodontic patients. These findings, which are concordant with other authors, suggest that the increase in streptococcal flora can also lead to a higher incidence of caries. However, their study is encouraging to clinicians, because it failed to demonstrate either an increase in the plaque level around the appliances or in the percentage of potentially pathogenic Gram-negative organisms. This observation is consistent with anecdotal evidence that a high level of oral hygiene maintenance adopted by the study subjects can reduce plaque accumulation to reasonable and less pathogenic levels.[14] Davies and coworkers reinforce this perception in a report that suggests behavioral factors rather than orthodontic appliances, treatment plans, and force modules per se were responsible for the degree of oral hygiene and gingival health in patients wearing fixed orthodontic appliances.[61]

Hagg and coworkers recently evaluated quantitative and qualitative alterations in the carrier rate of candida species, enterobacteria, and associated changes in the plaque index during orthodontic treatment with fixed appliances. They could isolate eight coliform species (*Klebsiella pneumoniae*, *Enterobacter sakazakii*, *Enterobacter cloacae*, *Enterobacter gergoviae*, *Pseudomonas aeruginosa*, *Enterobacter agglomerans*, *Acinetobacter*, and *Yersinia* species) from clinical study patients. They could also observe a change of *Candida albicans* to a carrier state from a noncarrier state, once fixed appliances were placed.[62]

It is clear from the ongoing discussion that fixed appliances retain a direct effect on plaque index and microbiological quantity. The appliance might interfere with oral hygiene practices

and an astute clinician should always place an emphasis on strict instructions regarding oral hygiene, as well as appliance hygiene, in orthodontic patients.

Histological Changes

Due to inherent problems in planning and conducting clinical studies concerning gingival conditions in humans, a number of researchers have used animal models to perform histological studies that may define actual tissue-level pathogenic mechanisms. Histological sections of orthodontically treated tissues characteristically reveal increased numbers of mononuclear infiltrates, along with hyperplasia and proliferation of pocket epithelium. Throughout the duration of these studies a dense accumulation of chronic inflammatory cells occupying large areas of connective tissue was observed.[44,63]

Redlich and coworkers outlined the histological changes at sites of extraction space closure, in the form of papillary epithelial hyperplasia. The newly formed collagen in these regions was coiled and compressed, in the shape of a "football."[44] There are, however, other reports that state that the space closure mechanics can lead to loss of collagen in the hyperplastic gingiva.[64] After tensile force application in rabbit incisors, Van de Velde and coworkers demonstrated trauma, characterized by tears, ulcerations, and ruptures in the gingival epithelium, which can provide facile access of invasive bacteria and cytotoxic products to subjacent bone. Leukocytes were present in the histological sections, attributed to the production of chemoattraction factors following local destruction of tissues. Longer periods of tensile force application resulted in deeper penetration of leukocytes, with greater degrees of ulcerations and tears. It was concluded that these higher levels of damage to the gingiva occurred as soon as 24 hours after the initiation of tooth movement. It is clinically important to note, however, that the gingiva seems to recover by 72 hours after appliance removal.[65]

Therefore when gingival inflammation, hypertrophy, and incipient periodontitis is imminent, temporary removal of an arch wire, to facilitate flossing and brushing efficacy, may be prudent and well within the spectrum of reasonable orthodontic treatment planning. In the event of untoward tooth relapse during the course of treatment, at worst the clinician has observed potential relapse. This is a physiologic phenomenon, better identified while brackets are in place even without arch wires. Alternatively, relapse after all brackets are removed is often perceived by patients as the end of treatment, and the relapse as a sign of treatment failure.

Biochemical and Vascular Changes

The gingival tissue tolerance to orthodontic banding was evaluated by Cheraskin and Ringsdorf, with the help of biochemical tests evaluating fasting blood glucose levels in humans. The authors correlated tissue tolerance to fasting blood glucose levels and stated that subjects who showed no worsening in blood glucose values (grouped around mean) had good tissue tolerance. In contrast, subjects with poor tissue tolerance showed blood glucose values widely dispersed around the mean values. They suggested using fasting blood glucose variability or homeostasis as a predicting criterion to assess tissue tolerance of an orthodontic patient.[66]

Using laser Doppler flowmetry in human gingiva, Yamaguchi and Nanda measured changes in blood flow after orthodontic force application. They measured blood flow through the infraorbital artery, the branch of the maxillary artery, and demonstrated an initial reduction in this parameter within 2 to 3 seconds of force application. There was a gradual recovery in blood flow to the resting level in the attached gingiva when force application was continued, which was assumed to be the result of indirect blood flow from adjacent capillary loops and network of vessels. This flow was possible because of the existence of an open microcirculatory system in gingival tissues. When the force was removed, a reactive hyperemia was observed, the magnitude of which was correlated to the decreased blood flow. The duration of the reactive hyperemia was positively correlated to the decreased blood flow observed earlier. That investigation illuminates various changes in blood flow associated with orthodontic mechanics, implying that force modulation would provide optimum force for optimal tooth movement.[67]

Molecular Level Changes

Collagen fibers, the main structural component of the extracellular matrix (ECM) of the gingiva, retain a higher turnover rate in the periodontal ligament than in the gingival unit, coronal to the alveolar osseous crest. Whenever a force is applied, these fibers are compressed, retracted, or even become hypertrophic.[44] Ultrastructural analysis with transmission electron microscopy (TEM) revealed an increase in the diameter of collagen fibers in both tension and compression areas. Degradation of fibers inside the compressed papilla can be seen, with longitudinal splits, but without the typical bending pattern.[44,68]

Redlich and coworkers examined the effect of mechanical force on gene expression of collagen type 1 (COL-1) and matrix metalloproteinase-1 (MMP-1) in cultured gingival fibroblasts. They found that the cells under pressure expressed higher levels of COL-1 and lower levels of MMP-1 mRNA, compared with the control cells. Assuming that gene expression at gingival ECM components is also affected in vivo, they performed another study in dogs. There they observed no change in mRNA levels of COL-1, tissue inhibitor of metalloproteinase (TIMP)-1 and -2, at both pressure and tension regions of the gingiva. An interesting finding was the time-dependent regulation of gene expression of MMP-1, and increased activity of this enzyme following application of force. In the pressure side, a rise in MMP-1 level was observed at day 7, followed by a decrease at days 14 and 28, whereas in the tension side, a rise in mRNA level of MMP-1 was noted at day 3, with a further increase in day 7, followed by a decrease in day 14. This pattern was followed by a 10-fold increase at day 28. It was concluded that the responsiveness of MMP-1 to force is not only the result of tissue injury and inflammatory reactions, but also of the mechanical stresses themselves.[69]

Bolcato-Bellemin and coworkers, through cell cultures following application of mechanical stretch, demonstrated an increase in mRNA gene transcription of integrin subunit $\beta 1$ in gingival fibroblasts. Integrin subunit $\beta 1$ is present in basal and parabasal cells of the gingival epithelium, and is involved in the formation of focal contacts, where integrin cytoplasmic parts are linked to cytoskeleton components via a bridging molecule, focal adhesion kinase (FAK). It was demonstrated that $p125^{FAK}$, located primarily at the cell periphery, is activated by tyrosine phosphorylation on binding of $\beta 1$-integrins to an ECM ligand, triggering signal mechanotransduction. They observed a rise of $p125^{FAK}$ in fibroblastic cultures subjected to mechanical strain, providing a preliminary report on a potential future research on the role of gingival fibroblasts in tooth movement.[70]

Long-Term Gingival Effects of Force Application

It is clear from the ongoing discussion that orthodontic treatment has a direct influence on gingival health. Glans and coworkers reported on a marked and statistically significant improvement in gingival health of patients with initially crowded dentitions, from 12 weeks after bonding until debonding. They attributed this finding to leveling of the dentition performed within these 12 weeks, making oral hygiene measures effective, while at the same time evoking patient motivation by creating better esthetics.[71] A similar result was reported by Davies and coworkers, but they interpreted it as a behavioral change, rather than as the outcome of orthodontic treatment per se. They concluded that regular visits to the orthodontist are the most likely reason for the improvement in oral hygiene and gingival health.[61] It may be prudent to secure the supportive comanagement of a periodontist or referring dentist where indicated.

Management or Reduction of the Side Effects of Orthodontic Treatment on the Gingiva

Although various methods have been used to improve oral hygiene, optimum mechanical removal of plaque by brushing and by professional scaling is considered to be the most important function.[72-74] End-tufted brushes saturated with bactericidal disinfectants such as chlorhexidine gluconate, supplemented with floss threaders or stiff plastic floss that can be threaded beneath the arch wires, are particularly useful. However, required daily time commitment for effective plaque abatement is often in the range of 15 to 30 minutes. Thus, family, professional, and staff

encouragement for the patient is more helpful than repetitious admonitions.

During orthodontic treatment, the importance of a regular brushing routine, as a measure of preventing or reducing gingival disease, should be emphasized to all patients as an integral and ongoing part of therapy. For this reason, a specially designed manual, as well as electric toothbrushes, for use by orthodontic patients may be effective for some but are a disservice when used as a substitute for diligent individual care. An electric toothbrush used as a "crutch" can be harmful to oral health by giving false confidence of gingival health. Trimpeneers and coworkers compared electric and manual toothbrushes for their efficacy in plaque removal, and concluded that manual toothbrushes are most effective in orthodontic patients.[75] A study by Kilicoglu and coworkers even found that specially designed orthodontic toothbrushes were not superior to classic toothbrushes in terms of plaque-removing efficacy.[15] These results, however, have not remained unchallenged. In a series of studies that followed, the efficacy of electric toothbrushes, when compared with their manual counterparts, were constantly superior.[15,76-78] Hickmann and coworkers conducted a detailed evaluation regarding this controversy in 63 patients, with the help of a plaque index, gingival index, mouth rinse with water, interdental bleeding index, and assessment of tissue trauma. The results they obtained were in favor of powered toothbrushes with dedicated orthodontic heads.[76] All these studies point to the importance of oral hygiene measures rather than just evaluation of different types of tooth brushes—classic, manual orthodontic, or powered. The factor that is most important clinically is the motivation of the patient to accomplish daily efficient and effective removal of dental plaque, a process for which a team effort is often necessary and in which all patients should be carefully instructed.

Pharmaceutical Aids

Preventive clinical plaque control methods with chlorhexidine mouthwash have been used in orthodontic patients. In the literature, conflicting results on the efficacy have been reported, with studies reporting favorable results while others report unfavorable results.[79-81] Anderson and coworkers evaluated this issue recently and stated that chlorhexidine in addition to regular oral hygiene habits was effective in the reduction of plaque and gingivitis in orthodontic patients. They also assessed the discoloration or staining in the tooth surface with long-term use of chlorhexidine and stated that it was neither clinically nor statistically significant.[82]

Removal of dental plaque in orthodontic patients with various other measures is also reported. Isotupa and coworkers tried polyol gums in orthodontic patients for plaque control and observed a reduction in plaque and in *Streptococcus mutans* numbers, showing its efficacy.[83] In another study, Othman and coworkers combined orthodontic composite resins with benzalkonium chloride, an antimicrobial agent, for bonding orthodontic brackets. The results demonstrated effective antimicrobial action by this compound, without altering the mechanical properties of the composite resin.[84] The effect of combined application of antimicrobial and fluoride varnish to orthodontic patients for the purpose of reducing plaque and gingivitis was studied by Ogaard and coworkers. They reported a significant reduction in *Streptococcus mutans* count in plaque during first 48 hours of treatment with fixed appliances. They also observed a significant reduction in the amount of plaque and gingivitis in this study sample.[85]

The Timing of Soft-Tissue Augmentation: Prophylactic Versus Therapeutic?

Even though prophylactic management of gingival recession in at-risk orthodontic patients remains controversial, there were numerous reports that suggest that universal prophylaxis in cases of doubt enhances ultimate treatment efficiency.[20-23,33] Preorthodontic gingival augmentation procedures are indicated in patients with thin gingival tissue and in areas of possible arch expansion, but not if tooth movement is constrained to the envelope of the alveolar process. The primary therapeutic goal is to increase the buccolingual thickness of the marginal tissues over teeth that might develop alveolar bone dehiscence during tooth movement. The rationale behind this procedure is that increasing the gingival thickness creates more robust marginal tissues, which are less susceptible to trauma or plaque related inflammation and subsequent re-

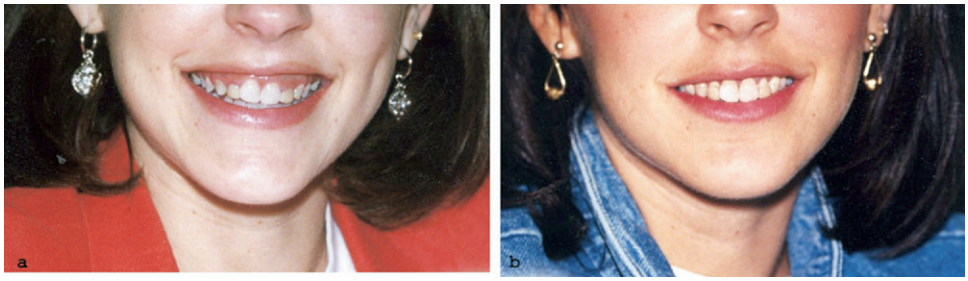

Figure 7. These two portraits of the same patient demonstrate that attention to and treatment of gingival enlargement can add significantly to the esthetic orthodontic outcome in total facial esthetics. (Color version of figure is available online.)

cession. The subepithelial free connective tissue grafting for increasing the apicocoronal width of keratinized gingiva and establishing root coverage in areas of marginal tissue recession is the most preferred method. The efficacy of this procedure was evaluated by Holmes and coworkers in dogs, and they reported favorable results, which were stable throughout the orthodontic treatment period.[86]

Interdental clefts at the site of extraction space closure contributing to poor periodontal health and orthodontic relapse are often treated by either electrosurgery or conventional surgical gingivectomy.[87] Malkoc and coworkers compared the efficacy of both techniques and found no statistically significantly difference between the results. However, they cautioned against the use of electrosurgery in patients bearing cardiac pacemakers. They stated that, with proper attention to safeguards, both techniques can be used effectively to remove gingival invaginations and overgrowth—hyperplasia or hypertrophy[48] (Fig 7).

The Gingiva After Removal of Orthodontic Appliances

The favorable as well as the harmful effects that can occur during orthodontic treatment are well understood. The fate of these effects, once the appliance is removed, was addressed by Sallum and coworkers. They reported a significant reduction in plaque index, bleeding on probing, and probing depth, the three most important parameters indicating clinical gingival health, once orthodontic appliances are removed. The removal of orthodontic appliances, along with professional scaling and proper instruction on oral hygiene, leads to significant reduction in the periodontopathogens harbored in the oral cavity, such as *Bacteroides forsythus* and *Actinobacillus actinomycetemcomitans*, from both supra- and subgingival plaque samples.[88]

At the molecular level, Redlich and coworkers observed a gradual increase in both COL-1 and TIMP gene expressions concomitant with a decrease in MMP-1 after removal of orthodontic appliances. These findings indicate progressive renormalization of collagen metabolism in the gingiva, once orthodontic appliances are out of the mouth.[69]

Conclusions

This review has attempted to outline all the effects that fixed orthodontic appliances are prone to produce on gingival tissues. It is evident that the mere placement of orthodontic appliances can contribute to undesirable changes, such as the formation of plaque-harboring areas, a change in oral ecosystem, a shift from normal flora to microbes marked as periodontopathogens, gingival inflammation, irreversible gingival hyperplasia, permanent loss of periodontal attachment (bone loss), and bony or gingival dehiscence (recession). While these effects can be controlled by proper oral hygiene measures, failing to adhere to such a regimen might result in initiation of destructive periodontal disease through the breach in natural protective barriers. These findings point to the importance of stressing oral hygiene and effective infection control as an integral part of every visit to the orthodontist. Repetitive and the "recovery always accompanies . . ." which is "obvious" to the poorly informed, is clearly not supported by periodontal literature. A thorough, evidenced-

based, candid informed consent recruits the patient and parent as collaborative informal "cotherapists." As important and legitimate stakeholders in optimal outcome, they must share the responsibility for any untoward events, side effects, or complications that may be permanently damaging to the underlying soft tissue and bone. When a damaging side effect is found, the orthodontist should consider obtaining a consultation with a periodontist, and remove irritating forces, as well as attachments and appliances, which may be construed as contributing factors, so that further destruction is prevented.

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