Chapter

Orthodontics and the Periodontium: A Symbiotic Relationship

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Abstract

The force applied by the orthodontist to facilitate the orderly movement of teeth to their new position may have deleterious effects on the most important structure involved in the procedure—the periodontium. This chapter endeavors to provide an overview of the biological processes that play a role in achieving the patient's as well as the orthodontist's objective.

Keywords: accelerated tooth movement, inflammation, periodontal ligament, alveolar bone, cytokines

1. Introduction

The art and science of orthodontics have certainly come a long way from the era of treatment using removable appliances, which mainly produced tipping movements of the teeth, to the fixed appliances that bring about bodily movements of the teeth to their desired destination. But this has not been a journey without hiccups. The quest for the perfect smile or the ideal occlusion has been marred by quite a few stories of botched-up cases, which, sometimes, could be the result of unscientific methods being utilized to achieve the promised results. But it also seems to be a story of due respect not being accorded to the most important structure in the treatment process—the periodontium. This chapter discusses the relationship that the periodontium (*and the periodontist*) shares with the specialty of orthodontics, one which, at times, tends to be taken for granted.

2. Theories of tooth movement

The force applied to the teeth during orthodontic treatment results in remodeling of the alveolar bone. The exact mechanism of how this force is converted to biological activity has not been elicited till now. Various theories try to explain this phenomenon:

1. Bone-bending/biological electrical

2. Pressure-tension

3. Neurogenic inflammation

4. Fluid-flow sheer stress

Whatever be the mechanism, it seems obvious that it is the periodontium that plays a big role in achieving the treatment goals.

3. The periodontium

The periodontium has two main functions: protection and attachment. The former function is carried out by the gingiva and the latter by its three remaining parts, namely the cementum, periodontal ligaments, and the alveolar bone. It is only the gingiva that is visible in the oral cavity, the rest being covered and protected by it.

3.1 Gingiva

The primary function of the gingiva is protection, as stated earlier. Orthodontic treatment tends to be associated with gingivitis in many patients. As the presence of plaque is one of the main factors in the development of gingivitis, it could be the interference of the orthodontic brackets and elastics with effective removal of dental plaque, which might be resulting in gingivitis. However, it has been shown that because of orthodontic treatment a shift in the composition and type of bacteria can occur [1]. According to J. van Gastel, et al. [2], fixed orthodontic treatment may result in localized gingivitis, which rarely progresses to periodontitis. E. Bimstein and A. Becker [3] state that, following placement of a fixed appliance, a small amount of gingival inflammation will be visible, which could be transient in nature and does not lead to attachment loss, in the majority of the patients.

Gingival recession is considered to be one of the more common complications of orthodontic tooth movement (OTM). But in a study conducted among 205 orthodontic patients, Morris JW, et al. [4] found that "orthodontic treatment is not a major risk factor for the development of gingival recession." They further state that "although greater amounts of maxillary expansion during treatment increase the risks of post-treatment recession, the effects are minimal."

3.2 Cementum

Any treatment procedure that a human being undergoes, whether surgical or medical, can have side effects or risks involved, and orthodontic treatment is no exception. Similar to other medical procedures, the aim in orthodontics is also to minimize the risks involved in the maximum number of patients possible.

Microscopic resorption of the cementum of erupted as well as unerupted teeth is a common phenomenon. This occurs without involving the underlying dentin in the majority of cases. The resorption may be caused by local or systemic factors or can be idiopathic. Trauma from occlusion, malaligned erupting teeth, periapical as well as periodontal infections, replanted or transplanted teeth, orthodontic movement,

etc. are the local factors that may result in cementum resorption. According to some authors [5–7], root resorption is the most common side effect of orthodontic treatment and occurs within 6 months of commencement of the treatment. Anja Pejicic, et al. [8] have described three degrees of severity of orthodontically induced root resorption (OIRR): the first degree wherein there is only surface resorption of the cementum which will regenerate or remodel fully. The second degree shows deep resorption with cementum and outer dentin layers involved and this is usually repaired with cementum. In this, the original shape of the root may or may not be achieved following the repair. In their third degree, there is full resorption of the apical hard tissues evidenced by shortening of the root.

Biologic as well as mechanical factors have been highlighted as probable causes of OIRR, with tooth root morphology, abnormal root shape, previous history of trauma or root resorption, genetic and systemic factors, endodontic treatment, habits and oral health, etc. likely to be the biologic factors. Mechanical factors could be the amount of apical displacement, the magnitude of force applied, duration of treatment, whether it is continuous or intermittent force, type, extent and direction of tooth movement, etc., according to Anja Pejicic, et al. There are studies [9–12] that suggest that it is the trauma due to over-compression of the periodontal ligament that causes OIRR. It seems to be an accepted fact that orthodontic movements are not entirely translatory due to mechanical laws. This results in the concentration of orthodontic forces at the apical region. During OTM, hyalinization does occur because it is virtually impossible to prevent the occlusion of blood vessels totally. Because of this, root resorption might begin at the hyalinized region of the necrotic periodontal ligament. As hyalinized necrotic tissue develops in almost 100% of patients during orthodontic treatment, some authors believe that root resorption occurs in almost every orthodontic patient [13, 14]. It is believed that the aggressiveness of the resorbing cells, the vulnerability and sensitivity of the tissues involved and individual variation and susceptibility will decide the extent of resorption [8].

The use of light orthodontic force has been shown to minimize the extent of root resorption, especially with horizontal and vertical displacement. This could be because the apical third of the root is covered with cellular cementum, whereas the coronal third is covered with acellular cementum. Because of the increased proliferation activity, cells of the cellular cementum might be easily damaged, whereas the slowly dividing cells of the acellular cementum might be more resistant to those forces [15–17].

Nitric oxide (NO), the intra- and the intercellular signal molecule, is synthesized by the activity of neuronal, endothelial, and inducible isoforms of NO synthases (NOSs). The primary sensor of NO is soluble guanylate cyclase (sGC). It plays a very important part in many physiological as well as pathological processes and conditions and also in NO signaling. The enzymatic activity of sGC is boosted when NO binds to it. In inflammation, sGC is oxidized and becomes insensitive to NO. Inflammation of the periodontium induces the resorption of cementum by cementoclasts and the resorption of the alveolar bone by osteoclasts. Korkmaz Y, et al. think that if medication can be used to activate sGC in periodontal tissues of patients suffering from periodontitis in nitric oxide and heme-independent manner, it could result in a novel treatment to stop cementum resorption for such patients. They reached this conclusion after studying the α1- and β1-subunits of sGC in cementoclasts of healthy and inflamed human periodontium using double immunostaining for CD68 and cathepsin K. They compared this with those of osteoclasts from the same sections and noticed that under inflammatory conditions, cementoclasts showed a decreased staining intensity for both the subunits [18].

Yufei Xie, Ning Zhao, and Gang Shen [19] investigated the anti-resorptive mechanisms of cementocytes during orthodontic tooth movement. They concluded that under fluid-flow sheer stress, cementocytes stimulate the differentiation of osteoblasts and inhibit the activation of osteoclasts, showing greater potential for bone protection than alveolar bone osteocytes. And according to them, "cementocytes might play an important role in preventing one of the most common complications of orthodontic treatment – root resorption."

According to Alberto Consolaro [20], teeth with OIRR do not need:

1. restraints, as they do not become mobile or painful. If either of these symptoms is present, he suggests that other etiological factors like recently removed orthodontic braces, trauma from occlusion, bruxism, cervical bone loss, bone loss due to periodontitis, etc. should be looked for.

2. endodontic treatment as the dental pulp does not undergo ischemia, infarction, or necrosis during the orthodontic movements.

3. replacement with dental implants, as the cervical third of the root is responsible for 60% of periodontal support.

3.3 The alveolar bone

The alveolar bone is a mineralized connective tissue and is made up of around 67% inorganic material by weight. The inorganic content is primarily calcium and phosphate, with the mineral content being typically in the form of hydroxyapatite crystals. Around 20% of the alveolar bone consists of organic material, containing both collagen and non-collagenous materials. Water constitutes the rest of the weight of the alveolar bone- ~ 15% [21]. The inner wall of the tooth socket, known as the alveolar bone proper, contains many openings through which the periodontal ligament connects with the neurovascular bundles of the cancellous bone. The interdental bone or septum is made up of cancellous supporting bone within cortical walls.

Adjacent to the PDL space is a plate of compact bone called the lamina dura, whereas the majority of alveolar bone is trabecular in nature. The alveolar bone contains many different types of cells such as adipocytes, endothelial cells, macrophages, osteoclasts, osteoblasts and osteocytes. But the crucial detail of maintaining the function as well as homeostasis of the alveolar bone is carried out by the last three types of cells. There are some differences between the osteoblasts which form bone, and the osteoclasts, which resorb bone. The former (and the osteocytes) descend from mesenchymal cells, whereas the latter originates from the monocyte or hematopoietic cells. At the same time, the osteoclasts are formed by the fusion of multiple monocytes and thus are multinucleated while the osteoblasts are mononucleated. Type I collagen, which is the most abundant protein in vertebrates, can be made by both fibroblasts and osteoblasts and it is this collagen that forms the structural and mechanical matrix of the alveolar bone. The osteoblasts contain the master switch Runx2, which helps in the differentiation of osteoblasts from the progenitor mesenchymal cells [21]. As age advances, there is a disproportion between bone deposition and resorption and this is because the number of osteoblasts decreases as we age [22]. While apposition of bone is taking place, osteoblasts get enclosed in the mineralized bone and these cells are known as the osteocytes. A lacuna can form around such an osteocyte by deposition of minerals such as calcium carbonate, hydroxyapatite and

calcium phosphate, during bone formation. The lacunae connect with each other through canaliculi, which are narrow channels through which the dendrites of osteocytes correspond using gap junctions. The bone-resorbing osteoclasts express various substances such as osteoprotegerin (OPG), chloride channel 7 (ClCN7), cathepsin K, and tartrate-resistant acid phosphatase (TRAP). Bone matrix proteins such as elastin, collagen, and gelatin are catabolized by the protease cathepsin K, whereas ClCN7 maintains osteoclast neutrality by shuffling chloride ions through the cell membrane. OPG, though a member of the TNF receptor family, is secreted and acts as a cytokine.

Among all the periodontal tissues, alveolar bone is the least stable because it is in a constant state of flux. Local factors that cause internal remodeling include age-related changes as well as functional requirements on the tooth. Mechanical strains caused by orthodontic movements are thought to be resulting in physiologic bone adaptation together with minor injuries to the periodontium, which are reversible [23]. The pressure-tension theory of tooth movement proposes that a tooth moves in the periodontal space by creating a pressure and tension side. According to this theory, the tooth shifts its position within the periodontal ligament (PDL) space, resulting in PDL compression in some areas and PDL tension in others within a few seconds of force loading and this is brought about by chemical, rather than electric, signals as the stimulus for cellular differentiation and ultimately tooth movement. Bone resorption occurs at the compression side and bone formation at the tension side, with blood flow being decreased on the compression side and is maintained or increased on the tension side. Within minutes of force being applied, the alteration in blood flow changes the oxygen tension and the chemical environment by releasing biologically active agents such as prostaglandins and cytokines [24]. This happens especially if there is sustained force. This alteration results in less oxygen levels on the pressure side due to compression of the periodontal ligament and vice versa. It has been observed that low oxygen tension causes decreased adenosine triphosphate (ATP) activity [25]. These changes act on cellular differentiation and activity, bringing about bone resorption at the compression side and bone formation at the tension side. Schwarz (1932) correlated the tissue response to the magnitude of force, with capillary blood pressure. If the force exceeds the pressure of ~20–25 g/cm^2 of the root surface, tissue necrosis can occur due to the strangulated periodontium [26]. It has been shown that with the application of heavy force, blood flow tends to be cut off resulting in cell death under compression. According to Al Ansari et al., these cell deaths also include some osteocytes and osteoblasts in the adjacent alveolar bone. This causes acute inflammatory response with the release of chemokines that could attract other inflammatory and precursor cells into the extravascular space from the blood vessels. According to Taddei et al., during orthodontic movement, the chemokines known as monocyte chemo-attractant protein-1 (MCP-1) is released attracting the monocytes. These monocytes become either macrophages or osteoclasts once they exit the bloodstream and enter into the tissue. The release of other inflammatory mediators is also seen within the first few hours of tooth movement.

If the cessation of blood flow occurs because of heavy orthodontic force being applied, a delayed differentiation or recruitment of osteoclasts from adjacent bone marrow space also may occur resulting in "undermining resorption" that removes the lamina dura next to the compressed PDL. This is because no osteoclast differentiation occurs within the compressed PDL space. Under such a condition, tooth movement will take place only after this "undermining resorption" is completed, meaning only after a week or two. This also explains why tooth movement occurs within 2 to 3 days when light force is applied, because the light force will only reduce the blood flow

permitting the quick recruitment of osteoclasts either from within the periodontal ligament space or from blood. This will result in the removal of the lamina dura by the process of "frontal resorption." At the same time, it is a fact that tooth movement is a result of a combination of "undermining" as well as "frontal" resorption. This is because some degree of hyalinization almost always occurs as it is virtually impossible to clinically prevent the occlusion of blood vessels completely [24].

3.4 The periodontal ligament

Yes, the force applied by the orthodontic appliance provides the impetus for the tooth to move. But without the PDL it would be impossible for the teeth to move through the bone and reach their intended destination, in an orderly manner. The PDL, like all ligaments in the body, connects the hard tissue structures, either the cementum of adjacent teeth to each other or the cementum of the tooth to the alveolar bone.

The PDL also connects with the neurovascular bundles of the cancellous bone through the alveolar bone proper *via* the numerous openings. The PDL is a dense fibrous connective tissue structure that consists of collagenous fiber bundles, cells, vascular and neural elements, and interstitial fluid. Its primary function is to support the teeth in their sockets and at the same time allow them to withstand considerable masticatory forces. The average width of the PDL space is around 0.2 mm, with the space decreasing as age advances. The space is occupied mostly by Type 1 collagen bundles and is the thinnest near the middle third of the root. These collagen fibers are mainly divided into principal, accessory, and elastic fibers. Sharpey's fibers are the term used for the terminal portion of these fibers that insert in the alveolar bone and cementum. The principal fibers can be subdivided into the transseptal fiber (or interdental ligament) and alveolodental ligament. Some authors consider the transeptal fibers as gingival fibers because they do not have an osseous attachment. These fibers connect the cementum of adjacent teeth, with their duty being maintaining the alignment of teeth, and the alveolodental ligament group of fibers helping teeth withstand compression forces during mastication. The accessory fibers prevent rotation of the tooth and run from the alveolar bone to cementum in different planes, in a tangential manner. Many cells occupy the PDL space, namely 1) synthetic cells like fibroblasts that make up to around 60% of the total PDL cell population, osteoblasts, and cementoblasts; 2) resorptive cells such as osteoclasts, cementoclasts; 3) progenitor cells including undifferentiated mesenchymal cells; 4) defense cells such as macrophages, mast cells, and lymphocytes; and 5) remnants of the epithelial root sheath of Hertwig, which are epithelial cells. The PDL space also contains interstitial fluid, which is contributed by the circulatory system [24]. This helps the PDL space to transmit the masticatory forces (which can range from 70 to 150 newtons) etc., onto teeth, thus acting as a shock absorber.

Some of the more frequent complications of orthodontic treatment are dehiscence or fenestration of the alveolar bone. These can result in root exposure, gingival recession, and relapse of the condition.

4. Inflammation and orthodontic tooth movement

As discussed earlier, tooth loading, physiologic or otherwise, causes areas of compression and tension on the soft tissues surrounding the teeth also—the PDL,

nerves, blood vessels, etc. In the PDL, there is an intimate relationship of the nerve endings with the blood vessels. Neurotransmitters such as calcitonin gene-related peptide (CGRP) and substance P are released when the nerve endings get distorted and these cause vasodilation and increased permeability of the blood vessels resulting in plasma leakage [23, 26]. OTM is achieved by the remodeling of the PDL and alveolar bone. These remodeling activities and the movement of the teeth result in an aseptic inflammatory process with the consequent increase in mediators such as prostaglandins (PGs), interleukins (ILs. IL-6, IL-7 & IL-17), the tumor necrosis factor (TNF)-α superfamily, and the receptor activator of nuclear factor (RANK)/RANK ligand (RANKL)/osteoprotegerin (OPG).

Current scientific literature suggests that arachidonic acid (AA) pathway plays a very important role in many human diseases such as cardiovascular problems, carcinogenesis as well as inflammatory conditions such as asthma, arthritis. Periodontists have been exploring the role of AA in periodontitis for some time, it being an inflammatory condition. AA can be metabolized by three specific enzyme systems, that is, cyclooxygenases, lipoxygenases, and cytochrome P450 (CYP) enzymes. One of the derivatives of the AA cascade—the prostaglandins (PGs) are produced within seconds of cell injury. PGE2 is the most abundantly seen PG in various tissues and is known for its all-around physiologic and pathological actions. It increases vascular permeability and chemotactic actions by acting as a vasodilator and at the same time, it increases bone resorption and osteoclast formation. An increase in PGs levels in the PDL and alveolar bone has been reported by Ngan, et al. [27], during orthodontic treatment. PGE2 levels in the gingival crevicular fluid (GCF) increased during OTM, according to Shetty et al. [28]. Leiker et al. [29] demonstrated that exogenous prostaglandins enhanced the rate of OTM in rats. The administration of PGE or prostaglandin receptor EP4 also enhanced the rate of tooth movement [30, 31]. It has also been demonstrated that indomethacin, a specific inhibitor of prostaglandin synthesis, reduces the rate of OTM in rats [31, 32]. As mentioned earlier, the cytokines also increase during OTM. IL-β in particular is involved with inflammation and stimulates bone resorption. It has been reported that IL-1β is produced by both macrophages and neutrophils, and is increased in inflamed gingival tissues. IL-6 is a multifunctional cytokine produced by immune cells and induces osteoclastic bone resorption. IL-17 is an inflammatory cytokine that is produced by activated T cells and it has been reported that IL-17 induces osteoclastogenesis from monocytes.

RANK ligand (RANKL) and its receptor RANK are present on osteoblasts and precursor osteoclasts, respectively. They are considered to be the key factors that stimulate osteoclast formation and osteoclastogenesis. RANKL is required for osteoclast formation with macrophage-colony-stimulating factor (MCSF) from precursor monocyte/macrophages. Osteoprotegerin (OPG) inhibits RANK–RANKL interactions [24]. It binds to RANKL and prevents RANK–RANKL ligation. Therefore, OPG prevents osteoclast differentiation and activation. Kanzaki et al. demonstrated that compression forces upregulate RANKL expression through induction of COX-2 in human PDL cells *in vitro*. They also [33] demonstrated that the amount of rat experimental tooth movement is accelerated by the transfer of the RANKL gene to the periodontal tissue, while it is inhibited by OPG gene transfer. Additionally, compression force increases RANKL and decreases OPG secretion in human PDL cells *in vitro*. The GCF levels of RANKL are increased, and the levels of OPG are decreased in experimental canine movement. Therefore, it is suggested that the RANK–RANKL system is directly involved in the regulation of orthodontic tooth movement.

All these studies suggest that these and other inflammatory cytokines may be intricately entwined with one another during OTM, and may play important roles in bone remodeling. But studies on OIRR seem to suggest that these mediators might also be the cause for the most common complication in orthodontics-root resorption.

5. Accelerated orthodontic tooth movement

A systematic review of prospective studies on the duration of orthodontic treatment suggests that the duration of orthodontic treatment varies widely but takes less than 2 years to complete, on average [34]. Most patients would like their treatment to be done in a much shorter period and in addition, longer treatment periods might increase the chances of root resorption, decalcification, etc. This has resulted in orthodontists as well as manufacturers trying to shorten the duration by using various methods to accelerate OTM (AOTM). Some of the techniques advocated in this quest to accelerate OTM are as follows:

A. Those aimed at altering orthodontic mechanics

1. Limited orthodontic treatment.
2. Self-ligating and varying bracket designs
3. Customized appliances

B. Altering biological response to force

1. Medications such as corticosteroids, vitamin D3, parathyroid hormone, thyroxin, prostaglandins, platelet-rich plasma.
2. Micro-vibration
3. Low-intensity Laser
4. Photobiomodulation aka low-level light therapy
5. Electromagnetic fields
6. Direct electrical current

C. Surgical methods

1. Micro-osteoperforations
2. Piezocision
3. Corticotomies
4. Osteotomies/PDL distraction
5. Surgery first

Miles P [35] reviewed the studies involving the aforesaid techniques and was of the opinion that the technique of photobiomodulation may be of benefit but suggested that since there is limited evidence to support it, more studies will be needed before it can be applied routinely. With regard to the use of corticotomy, also he says that only low-level evidence is available and he concludes his review by suggesting that rigorous, well-designed randomized controlled trials with longer follow-up periods are necessary for all the techniques before they can be recommended.

The effects of most of the above AOTM procedures on the periodontium do not seem to have been studied in detail. The more commonly performed and studied one seems to be the corticotomies, *viz.*, the periodontally accelerated osteogenic orthodontics (PAOO).

5.1 Periodontally accelerated osteogenic orthodontics

In 2001, Wilcko, et al. [36] introduced the "periodontally accelerated osteogenic orthodontics" (PAOO) technique that they claimed shortened the duration of orthodontic treatment. This involved flap design, selective decortication, alveolar augmentation, membrane coverage, and closure using sutures. They radiographically assessed the presence of transient demineralization followed by remineralization at the corticotomy level. According to them, reversible osteopenia occurs both within the alveolar bone proper and on the surface and with this, the collagenous bony matrix also moves with the root in the same direction as the OTM. Once the OTM is completed and the teeth are retained in their predetermined position, remineralization of the matrix takes place. They claim that this demineralization-remineralization is complete in adolescents but not so much in adults and termed it the "regional acceleratory phenomenon" (RAP) of bone remodeling. They thought that this "bone matrix transportation" had made it possible to design a surgical approach, which permits extraction space closure in 3 to 4 weeks. The duration of RAP is claimed to last for 3–4 months by these authors and the amount of tooth movement during this period was double around 1 mm/month, in animal studies conducted by Iino S, et al. [37].

5.2 History of osteotomies/corticotomies to speed up tooth movement

The use of techniques to speed up orthodontic tooth movement by utilizing alveolar surgery has a history dating back to more than a century. But it is Heinrich Kole [38] who has been credited with refining the process. He proposed the idea of accelerating orthodontic movements by displacing bone blocks, more than 6 decades ago. He hypothesized that it was the cortical bone that slowed the orthodontic movement of teeth and so why not weaken it by osteotomizing it? He advocated buccal and lingual interdental corticotomies together with supra-apical horizontal osteotomies connecting the two vertical cuts. Though accelerated orthodontic movements were achieved, he encountered quite a few complications like the non-vitality of teeth. It should be noted that Kole achieved the tooth movements using removable appliances fitted with adjustable screws. Others tried to build on this technique with Duker in 1975 [39] sparing the crestal bone in his corticotomies and Suya [40] replacing the supra-apical osteotomy with a corticotomy in 1991.

According to T. Gellee, et al. [41], PAOO also allows larger tooth displacements, a reduction in the risk of root resorption, and a gain in stability after the removal of orthodontic devices.

6. Periodontal therapy and orthodontics

According to the American Association of Orthodontists, one in four orthodontic patients is an adult. Some studies suggest that almost 40% of the patients are adults. As more and more adult patients seek orthodontic treatment for various reasons, it can be challenging for the orthodontist to tailor his/her techniques to the specific patient. Many of these patients might have underlying periodontal problems that can affect the treatment process as well as its outcome. The periodontist can play an active role in ensuring the success of the orthodontic treatment—in adult patients or adolescents. This role can be before, during, or after the orthodontic treatment.

6.1 Pretreatment

A thorough periodontal examination/charting is of utmost importance for every orthodontic patient, especially if skeletal growth has been completed. This is to identify and manage active conditions such as gingivitis and periodontitis as well as conditions that result in deficiency of soft or hard tissues or both. In ideal conditions, and with good oral hygiene, gingival health can be maintained with as little as 1–2 mm of keratinized gingiva. But soft tissue grafting might be indicated under the following circumstances:

1. when the buccal displacement of the roots is planned during the treatment or when treatment might result in thinning of the gingiva.

2. chronically inflamed areas of keratinized gingiva or no area of keratinized gingiva where the alveolar mucosa prevents optimal plaque control.

3. minimal areas of attached gingiva compromised by a shallow vestibule or frenum pull.

4. advancing gingival recession.

The techniques that are available to correct these conditions include the following:

1. Laterally or coronally advanced pedicle grafts.

2. Coronally advanced flaps alone or in conjunction with barrier membranes or enamel matrix proteins.

3. Free gingival grafts.

4. Subepithelial connective tissue autografts.

5. Allografts.

Every patient should undergo professional plaque removal and root debridement before the start of the treatment. Oral hygiene instructions should be reinforced because it has been shown that orthodontic bands, elastics, etc., tend to retain plaque, resulting in gingivitis, which may then proceed to periodontitis. Orthodontically

induced remodeling process may have a positive effect on bone, so extensive osseous surgery is usually not indicated at this time. But sometimes osseous surgery might be indicated in the following conditions:

1. **osseous craters**—these two-wall defects are mainly on the mesial and distal surfaces of the roots. If they are shallow craters (4–5 mm pocket depth), they might be left alone but more extensive ones might have to be treated.

2. **3-wall intra-bony defects**—these may be treated using bone grafts with or without membranes. Even though autografts are considered the gold standard for bone grafts, allografts also have been proven to give good results.

3. **Furcation defects**—this might be treated by open flap debridement followed by placement of the graft, etc., depending on the severity of the defect.

4. **Crown lengthening**—when the clinical crown is small, there might be a need to lengthen the crown utilizing gingivectomy procedures alone or in combination with osseous resection and apical repositioning of the gingiva.

The American Academy of Periodontology's systematic review on whether periodontal phenotype modification therapy (PhMT) involving hard tissue augmentation (PhMT-b) or soft tissue augmentation (PhMT-s) has clinical benefits for patients undergoing orthodontic treatment concluded that PhMT *via* corticotomy with particulate bone grafting (PhMT-b along with CAOT) may provide clinical benefits of augmenting periodontal phenotype, accelerating tooth movement, expanding the scope of incisor movement, and enhancing post-orthodontic stability of the mandibular anterior teeth. This study also says that the benefits of PhMT-s alone during orthodontic treatment remain undetermined because of the limited studies available [42].

6.2 During orthodontic treatment

The maintenance of oral hygiene during treatment is of paramount importance. During each visit reinforcement of oral hygiene instructions have to be carried out along with motivating the patient to do so. Periodontal evaluation every 6 months and radiographic examination once in a year would be ideal. Procedures like frenectomy might have to be carried out during the treatment period if the orthodontist feels that diastema closure, etc. are being hampered by an aberrant frenum.

6.3 Post-orthodontic phase

Regular periodontal charting should be carried out in patients who have completed their treatment. Depending on the case, circumferential supracrestal fiberotomy (CSF) may have to be carried out during the end part of the treatment or after the treatment is over. This is expected to release the tension on the supra-alveolar fibers following tooth de-rotation, thereby reducing the relapse risk. Reham Al-Jasser, et al. [43] in their study found that "post-treatment rotational relapse of anterior teeth subjected to CSF was minimal and statistically insignificant after 1 year of follow-up."

7. The periodontally compromised patient and orthodontics

Most orthodontists may be worried about carrying out orthodontic treatment in periodontally compromised patients and with good reason. At the same time, studies show that a large percentage (~65%) of patients with moderate to severe periodontitis are interested in such treatment for esthetic and functional changes caused by pathologic tooth migration [44].

Many questions need to be answered in these periodontally compromised patients who opt for orthodontic treatment. Some of the findings of a comprehensive search on PubMed focusing on "ortho-perio treatments" are as follows [45]:

1. Best time to start orthodontic treatment following periodontal therapy.

   According to the authors, in periodontally compromised cases that have undergone periodontal therapy, it is better to start orthodontic treatment as follows:

   1. 3 to 6 months after non-surgical/surgical periodontal treatment and

   2. 9 to 12 months after regenerative surgical procedures.

2. Acceptable periodontal status for orthodontic treatment

   It is important to achieve low rates of full-mouth plaque and bleeding on probing after active periodontal treatment with scores <25% of previous ones. They recommend that these low scores (i.e., optimal plaque control without clinical gingival inflammation) be reached and maintained during the entire phase of orthodontic therapy and they think that without these conditions, orthodontic tooth movement should be discontinued.

3. Biologic efficacy of orthodontic treatment

   A combined periodontist-orthodontist diagnostic and treatment endeavor in periodontally compromised patients can result in improved masticatory efficiency by a more balanced occlusion brought about by a realignment of the migrated teeth. The realignment may also result in the periodontal structures being better able to carry out their assigned functions.

According to Lindhe J and Ericsson I [46], a healthy periodontium with reduced height has a capacity similar to that of a normal periodontium to adapt to traumatizing occlusal forces. Wennström JL, et al. [47] state that sites with the horizontal bone loss after periodontal therapy will not be negatively influenced by the type of tooth movement once the individualized orthodontic mechanics are established (i.e., an appropriate ratio force/% remaining periodontal support). According to Polson A, et al., if teeth are moved through or into vertical bone defects, it can increase the rate of destruction of these periodontal structures. At the same time, if the OTM into infrabony pockets is done after successful elimination of subgingival infection, it will not result in adverse effects. They concluded that this movement/treatment will not bring about changes in the periodontal ligament attachment level; instead, the formation of a long junctional epithelium is what will be achieved [48].

According to Melsen B, et al. [49], "orthodontic intrusion at healthy sites can lead to new cementum formation and new collagen attachment, whereas for sites lacking proper oral hygiene, results vary from a moderate new attachment development to a worsening of the alveolar bone loss." And in a subsequent study, Melsen B [50] recommends that "the intrusion movement should be carefully planned as it can increase the risk of other adverse effects not desired in patients with a reduced periodontium, such as alveolar process reduction and root resorption."

Cassio Volponi Carvalho, et al. [51] studied the effects of orthodontic movement in the periodontal tissues of 10 adult patients with aggressive periodontitis and compared them with 10 patients with healthy periodontium. They evaluated the probing pocket depth, clinical attachment level, bleeding on probing, and dental plaque index before, during and 4 months after orthodontic treatment. They found improvement in all the above parameters, 4 months after orthodontic treatment.

Despite advances in therapeutics as well as our increased knowledge of the biological effects of orthodontic treatment, it might be better to avoid OTM in conditions such as uncontrolled infection/inflammation, inadequate anchorage, conditions where periodontal health might not improve despite periodontal therapy.

8. Conclusion

This chapter has attempted to portray the roles the periodontium, inflammation, and periodontal therapy play during the planning and execution of orthodontic treatment as well as once it is completed. It also discusses orthodontics in the periodontally compromised patient. There is a huge void in our knowledge about various aspects of the orthodontic movement of teeth and their effects on the periodontium. It is also evident that for the long-term success of orthodontic treatment, especially in the periodontally compromised patient, joining forces of the orthodontist and the periodontist would benefit patients as well as both the specialties.

Conflict of interest

The authors declare no conflict of interest.

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