Salient Periodontal Issues for the Modern Biologic Orthodontist

Leena Palomo, Juan Martin Palomo, and Nabil F. Bissada

The term synergy refers to the interaction between many agents acting in concert to create an effect greater than the sum of the separate parts; in other words the whole is greater than the sum of the parts. This definition helps one understand the biologic basis of periodontal orthodontic relationships in either therapy or disease. Synergistic collaborative treatment between the specialists also helps develop the harmony that results in less damage to both components of the periodontium, the soft tissue “gingival unit” around the crown, and the “attachment apparatus” that connects the root to bone. Orthodontists should recognize possible periodontal risks to patients undergoing orthodontic treatment and any “damage” to the periodontium or enlargement of the gingiva is often an inevitable and reversible side effect. The goal is to minimize any harmful effects as any good surgeon seeks to minimize an inevitable scar. This is best done by interdisciplinary collaboration in controlling infection with chemicals, erupting teeth through keratinized gingiva, empowering patients with modern regenerative surgery, and using orthodontic therapy to minimize the amount of bone that is removed during periodontal osseous surgery. Thus, with synergistic collaboration, orthodontic therapy can enhance periodontal health and periodontal therapy can enhance the orthodontic outcome. (Semin Orthod 2008;14: 229-245.) © 2008 Published by Elsevier Inc.

The Question as to Whether Orthodontic Therapy Contributes to Periodontal Disease

The periodontium consists of two parts: the “gingival unit,” the gingival margin to the most coronal connective tissue fiber attached to the osseous crest and the remainder of the periodontium, the periodontal ligament (PDL), alveolus to
which it is attached and root elements. This second major anatomical unit is referred to as the “attachment apparatus” on which is based the term “attachment loss.” The latter term is more appropriate than the ambiguous “bone loss” when referring to periodontitis.

Maintaining a healthy periodontal status in orthodontic patients is a perennial and ubiquitous challenge to most orthodontists, and the entrance of an increasing number of adults into the orthodontic treatment population increases the risk of iatrogenic periodontal damage. Many researchers worldwide have investigated the effects of fixed orthodontic appliances on patients in relation to periodontal attachment loss and gingivitis.1-6 Most periodontal challenges are gingival since fixed appliances can indirectly contribute to an exacerbation of any inflammation. Chronic inflammation is generally reversible on appliance removal or following patient compliance with standard periodontal recall appointments and personal oral hygiene regimens. However, chronic inflammation can cause fibroplasia in most areas of the human body and the oral cavity is no exception. Orally that is manifest as permanent gingival hyperplasia known as postorthodontic gingival enlargement. Gingival hyperplasia is difficult to differentiate because superficial fibrosis mimics gingival health so the orthodontists should actively investigate the possibility of pernicious damage. Alexander, and Atack and others7-9 suggested that orthodontic appliance removal leads to similar periodontal conditions as those present before treatment, but their conclusions beg a larger question of hyperplasia and gingival pocket management.

Although most problems are gingival, it is difficult to ignore the findings of Hamp and coworkers10 who found that “in conclusion, during a long-term period of active orthodontic treatment with fixed appliances a slight but significant loss of periodontal support was observed despite the establishment and maintenance of proper oral hygiene standards.” The word “long-term” is emphasized. An excellent review of periodontal bacteriology has been authored by Loesche and Grossman,11 which suggests that the orthodontist should always be prepared for the individual patient who does not conform to professional instructions that should ensure periodontal health. Contemporary commercial emphasis on esthetics, “invisible” appliances, and practice management, however, have somewhat eclipsed the biological imperatives that form the basis of orthodontic care and protect the clinician from untoward destructive side effects. In one very real sense the sciences of osteology and orthopedics, as integral parts of all orthodontic tooth movement (OTM), can never be seriously considered irrelevant to any orthodontic treatment. They can only be ignored. This article aims to make that science understandable and easily incorporated into daily practice.

**Mechanisms of Tissue Damage**

Unfestooned orthodontic bands are particularly suspect as possibly complicating factors jeopardizing interproximal periodontal support, and at the present time “special periodontally friendly bands” are being designed in research and design laboratories. These challenging effects of band impingement may directly compromise local resistance related to subgingival pathogens in susceptible patients and result in damage to both interproximal gingival tissues and alveolar crestal bone in a manner similar to that produced by faulty crown margins. Diedrich and coworkers12 observed apical migration of the junctional epithelium and defects in orthodontic cemented bands in 85% of the subjects studied.

Årtun and Urbye (1998)5 suggest that the periodontal damage witnessed in their study of orthodontic patients is corroborated by others13 and, beyond the well known mechanisms of traumatic occlusion, that periodontal support might also be damaged during tooth intrusion where patients have active periodontitis or gingival infection significant enough to convert to periodontal disease. In these kinds of susceptible patients a screening examination for the interleukin (IL) family of inflammatory mediators may be wise. The details of genetic screening, discussed elsewhere in this issue, study the genetic potential of exaggerated immunologic reactions of host response to bacterial challenge such as those that recruit IL-1β.14,15 Although such tests may not be specific for periodontitis it has been associated with a number of serious health problems.16

Thus, where available, an assessment of elevated inflammatory mediator may be prudent, as it represents some of the latest scientific at-
tempts that eclipse gross clinical observation or radiographs in its ability to clarify systemic etiologic elements of periodontal health and disease. This enhances the value of care to orthodontic patients or their parents who are paying for the “best” modern orthodontics with a strong biologic rationale.

C-reactive protein (CRP) has also been associated with both cardiovascular disease and oral infection. Noack and coworkers have noted an increase in CRP levels in periodontitis patients with severity of the disease even where age, smoking, body mass index, triglycerides, and cholesterol have been statistically eliminated as confounding variables. Also, there are elevated levels of CRP associated with infection with subgingival organisms often associated with periodontal disease, including a number of notorious periodontal pathogens.

Therefore, for orthodontists who are interested in distinguishing themselves as an integral part of the health care profession, any evaluation of genetic susceptibility and systemic health threats associated with the infection can meaningfully enhance their orthodontic treatment as more than merely minor cosmetic adornment. While the details and validity are still being worked out at major research centers, the modern orthodontist may wish to remain educated about emerging oral tests of gingival crevicular fluid (GCF) to assess systemic health and diseases that relate to easily diagnosed genetic polymorphisms.

The etiology of periodontal problems may not simply rely on exaggerated host immunologic reactions. Mattingly and coworkers and others reflect the view that long-term fixed appliances can contribute to unfortunate but predictable qualitative alterations in the subgingival bacterial biofilms that become progressively periodontopathic with time.

One cannot make the airtight claim that there is absolutely unequivocal proof of causal connection among all orthodontic patients and inevitable contributions to periodontitis. However, the same can be said of orthodontic benefits. We cannot claim that orthodontic treatment will universally guarantee a more productive life and self-esteem. So to dismiss periodontal damage by reasoning that it is a minor event in most patients is fallacious. Extrapolations and common sense must prevail at this point in the absence of absolute proof that microbiological changes of crevicular fluid components prove that orthodontic fixed appliances singularly “cause” periodontitis to worsen. At this point, as in many multifactorial causal systems, it remains at most a “risk factor” and quality-of-life issue just as much as physiologic dental alignment. The degree to which any orthodontist seeks to personally protect his or her patient from all risk factors is a matter of individual preference and style of practice. Sufficient circumstantial data exist, however, to convince the modern biologically oriented orthodontist to at least inform the patient of the possibility of risk in a formal informed consent document and ensure that regular prophylaxis is maintained. Once that is done, the contributions to periodontal health by conscientious orthodontic therapy are virtually undeniable.

On a practical level it seems that an absence of bleeding on probing is a better forecasting parameter of health than bleeding on probing is a predictor of progressive disease. In other words, an absence of bleeding on probing, despite the pocket depth can justifiably be used as a test of “healthy gums.” On the other hand, while bleeding on probing is certainly an indication of infection of the gingivae, it is one of many risk factors associated with progressive bone loss due to periodontitis. However, the test is not spontaneous bleeding or even bleeding on brushing and flossing. That elicits only superficial disease, one that contributes significantly to caries and marginal decalcification. The best test is “bleeding on probing” elicited by stroking the sulci with a flexible plastic periodontal probe at a comfortable range of force between 10 to 20 g. Those orthodontic patients who present with persistent bleeding on such probing should be notified that they are “at risk” and that prudence dictates a more intensive regimen of periodontal therapy than those who present with little or no bleeding on probing.

Since bleeding swollen gingiva is ubiquitous in the orthodontic population universal caution should be employed and supportive periodontal care recommended routinely as an integral part of orthodontic therapy. Boyd and Baumrind and others have pointed out the importance of a full-mouth examination, six sites per tooth, for a comprehensive description of periodontal status in orthodontic patients. Thus, it is
still not clear if these circumstantial data of pernicious effects of orthodontic therapy might permanently influence the susceptibility to periodontal attachment loss in healthy patients undergoing orthodontic treatment, but it is certainly nothing that can be ethically or scientifically ignored. The responsibility, however, should be shared, and delegation to trained, competent ancillary personnel with educational talents is very effective under general supervision by the orthodontist.

The words of Sanders give us an excellent summation of the pertinent literature:

Orthodontic bodily tooth movement into plaque-induced infrabony defects can be successfully performed, provided that the periodontal lesion is eliminated before tooth movement is begun and that excellent oral hygiene is maintained. Although there is no unfavorable effect on the level of connective tissue attachment under these optimal conditions, neither does there appear to be a gain in soft-tissue attachment. On the other hand, if subgingival plaque and periodontal disease are not controlled before and during orthodontic treatment, an accelerated loss of attachment may result.28

This is particularly sage advice when recent evidence suggests that popular methods of non-extraction therapy, rapid palatal expansion, per se may cause a reduction in the amount of buccal periodontal bone support.29 The conclusion that seems most logical is that some periodontal damage may occur, particularly in those patients who exhibit poor oral hygiene during fixed appliance therapy, but the contribution of orthodontic care is generally minor, occasionally severe enough to justify periodontal therapy and prevalent enough to indicate concomitant supportive periodontal therapy as a routine preventive tactic during fixed appliance therapy. It is advisable that professional scaling and root planning, where indicated, be performed by a periodontist and alternated with restorative dentists who can also provide prophylactic measures for caries control, for example, sealants, fluoride varnish, and with alternating recalls every 3 months with the periodontist.

With the trend toward the use of bonded bracket by orthodontists, removal of arch wires and brackets for restorative care is understandable, but routine removal of arch wires for subgingival scaling and root planing does not appear to be necessary. Fixed appliance removal can also risk orthodontic relapse and interfere with expedient tooth movement. Therefore, routine appliance removal every 3 months is not necessary and should be requested only under specifically difficult circumstances.

**Treating the Infected Patient**

Since the human mouth is impossible to sterilize, the orthodontist should consider all patients more or less “infected.” This begs many questions of management given that one cannot easily distinguish between benign commensal residents, those that limit their damage to the gingival unit, and more virulent pathogenic forms. As universal precautions are employed for OSHA requirements, “universal caution” should be employed with all orthodontic patients and every patient should be (1) informed for the risks of periodontal damage and (2) explicitly encouraged to participate in “formal initial therapy” before and during all orthodontic treatment with fixed appliances.

The classic relationship between periodontitis and orthodontics lies in managing plaque-induced infection with different degrees of virulence. The key in delivery of appropriate treatment is distinguishing reversible gingivitis from self-perpetuating and progressive periodontitis, an infection that requires the diagnostic and therapeutic abilities and interventions of a clinician. Therapy for both is aimed at the reduction of etiologic factors that damage tissue directly from bacterial toxins and indirectly due to the host’s destructively excessive inflammatory response.

Bone loss is the hallmark of periodontitis. It involves loss of the “attachment apparatus” of which bone is a part and may or may not be amenable to regeneration depending on the pattern of destruction and an individual patient’s biologic capacity for regeneration. With notable exceptions, children and adolescents tend to develop gingivitis (hypertrophic or hyperplastic) without massive loss of attachment during orthodontic therapy when compliance is poor. Zachrisson and Alnaes30,31 noted decades ago that although periodontal attachment (bone) loss is not a common threat to the orthodontic patient on average, some individual patients may show some pernicious damage to the periodontal attachment.

In fact, the patient with root resorption of 3 mm is less compromised than the patient with
marginal bone loss due to the conic nature of the root form.

Interestingly individual patients in the 13- to 18-year-old age group may harbor infection and frank periodontal attachment loss patterns that do not conform to the norm. Significantly, Capelli and coworkers noted that 25.7% of the students in a study of Hispanic patients exhibited early-onset periodontitis (EOP) with 1.7% diagnosed as localized juvenile periodontitis (LJP). This fact alone should alert orthodontists that the teenage populations may not be able to be treated with impunity.

Encouraging is the report of Johal and Lee that simple modifications in orthodontic protocols can ameliorate any iatrogenic damage that threatens the clinical outcome. Simple supportive care, if not ensuring against periodontal side effects, at least can allow patients to modulate any pernicious effects with supportive care. Diedrich and coworkers noted that the connective tissue attachment damage at the cemento-enamel junction of banded teeth was so severely damaged on the mesial surface and the pocket epithelium proliferated toward the apex, meaning progression from established gingivitis to an initial periodontal lesion. It is these kinds of individual problems that most experienced practitioners guard against, because statistical data and general trends do not necessarily capture the entire domain of private practice cohorts especially when data are pooled or experimental selection bias (eg, dental students as subjects) occurs in the research.

Moreover, although it may be comforting to consider that orthodontic patients are, “on average,” less prone to problems, averages may not be of significance to the individual patient. Informed patient consent and awareness of risks versus rewards are strongly advised. Attachment loss in an adolescent with bleeding on probing and the presence of bacteria that can invade tissue may have serious systemic implications, and treating without informing both parents and patient is untenable in light of modern research data on oral bacteriology and disease research in cardiovascular disease. Ironically, treating adult patients may be less risky because disease is more prevalent and less obscure and both clinicians and patients are likely aware of the patient’s medical condition. This is especially true of patients who smoke tobacco or are compromised with systemic conditions such as diabetes, connective tissue disorders, endocrine disorders, neutrophil abnormalities, HIV, and those with a family history of dentures or periodontitis. Therefore, a comprehensive orthodontic diagnosis that includes assessment of the periodontal foundation is the critical step in determining an appropriate treatment plan for individual cases. Attachment loss cannot be diagnosed simply by even the best radiographs (eg, a full series of films using a parallel technique with a superimposed 1-mm grid on bitewings and periapical exposures), which essentially measure trabecular patterns of the endostome, not the medullary bone where periodontitis begins. This is because radiographs underestimate the degree of attachment (bone) loss unless it is correlated with periodontal probing below the contact point. Likewise line angle probing misses incipient lesions that initiate the destructive process apical to the col.

Attachment loss may actually begin during eruption of permanent teeth into an arch length deficiency and may produce permanent attachment loss even before full eruption according to Waerhaug. Lest the orthodontist becomes too preoccupied with periodontal problems, we must remember that orthodontic therapy generally helps more than it hurts. Apparently, according to Waerhaug, arch length deficiencies and distortion of contact point/embrasure form nearly always ensure at least some gingival pocketing where crowding (arch length deficiencies) drive plaques subgingivally. Moreover trauma associated with faulty bracket position, partial bonding, and untoward trauma during individual tooth movement can drive supragingival plaque subgingivally to initiate pathological changes in the subjacent attachment apparatus. Some evidence suggests that trauma per se may create qualitative deterioration of subgingival biofilm to more virulent bacterial profiles.

The mechanism of accelerated periodontal destruction around teeth with occlusal trauma and increased mobility remains unclear. One possible mechanism is that tooth mobility creates a subgingival environment conducive to overgrowth by periodontal pathogens. One authoritative study compared the subgingival microflora in mobile and nonmobile teeth of 35 supportive therapy adult patients and 15 untreated adults with periodontitis. Grant and
coworkers found that pockets around mobile teeth harbored significantly higher proportions of known periodontal pathogens than pockets with nonmobile teeth. These researchers suggested that tooth mobility may pose a risk for periodontal breakdown due to an increased subgingival occurrence of specific periodontal pathogens.

This potential brings up a practical issue for most practicing orthodontists. Since a great degree of task delegation keeps orthodontic treatment affordable, task delegation may be inadequate supervision of disease activity. Thus delegation to a general dental supervision or periodontal specialist may be the most prudent course. A simultaneous “tandem treatment” with conventional fixed appliance therapy and standard nonsurgical “initial therapy” emerges as the optimal protocol for most orthodontic patient groups.

The monitoring should involve recording probing pocket depth in even seemingly mild cases because a mere 5-mm interproximal pocket on the mesial of a maxillary first molar threatens to produce horizontal progression into the trifurcation rendering treatment more problematic with a relatively poorer prognosis. A Michigan O probe with Williams markings (Hu-Friedy Manufacturing, Chicago IL) is recommended because its precise scoring helps ensure accuracy. The actual probing force rarely needs to be more than 20 to 25 g of force, a force generated by little more than the weight of the probe itself (~17 g) (Fig 1). It is critical to realize that gingival recession, zone of attached gingiva, attachment level of some infrabony defects entirely within the medullary bone may escape diagnosis in periapical or bitewing radiographs. However, if the diagnosis is made by a collaborating professional, the orthodontist need only provide a screening examination, calibrate probing accuracy with the comanaging professional, and inform the patient of potential risks and then concentrate on the biomechani-

Figure 1. (A) Probing at static points such as line angles may miss defects that are not located in those areas. (B) A common error is not advancing the probe far enough interproximally and under the contact point. To more accurately detect defects, the periodontal probe is “walked” along the junctional epithelium. This allows the clinician to follow the attachment around the tooth and reach the depth of the pocket. A slight angulation may be necessary to “walk” the probe underneath the contact point. (Color version of figure is available online.)
cal protocol. This kind of shared responsibility ensures that periodontal pockets (pathologically deepened sulci) will be managed as well as possible. Ultimately it is the patient’s responsibility to assume treatment risks on well-informed written consent.

It is important to understand some of the histology of the pocket to understand health and disease (Fig 2). The area of the pocket, the so-called forefront of the “periodontal lesion,” is most vulnerable because it is bounded by hyperplastic epithelium, which is not keratinized. This lack of stratum corneum has given rise to the theory that the gingival sulcus is a susceptible point of entry to subjacent connective tissue and bone and even influences the spread of microorganisms’ toxins into the systemic environment. This is why periodontitis has not escaped the attention of the medical community, which is taking a renewed interest in the systemic effects of chronic untreated oral infection.

The junctional epithelium, apical to the base of the clinical pocket, forms the attachment between tooth and gingiva. The apical extent of this epithelium is thinner, just a few cell layers thick, has two basal laminas, one to attach to connective tissue and the other to attach to tooth surface. These “functional” epithelial cells turn over every 4 to 6 days and play a significant role in breakdown of the periodontal attachment. Healing of these cells is known as “repair by long functional (or junctional) epithelium.” In contrast, “new attachment” procedures or “regeneration” of periodontal attachment refers to the restoration of an entirely new tissue complex of cementum, ligament fibers, and bone previously destroyed by disease. The nuance is important because a long junctional epithelial (not connective tissue) attachment to the root can mimic regeneration of a new periodontal attachment when viewed clinically by an inexperienced clinician. Disease can look like health to the untrained eye.

**Treatment Options and Rationale**

Once periodontitis is diagnosed two major modalities of care have proven effective. The first is anti-infective treatment. The goal is to stop the progression of disease by minimizing the amount and influence of a primary etiologic element, that is, bacterial plaque biofilms. This is often elusive because the natural communal aggregations of microbes that form on all wet surfaces appear to the naked eye identical to virulent bacteria-laden fluids of the mouth and the surface of the tooth.

The second major objective is regenerative therapy to restore structures that have been destroyed by disease with new attachment: bone, cementum, and periodontal ligament. Initial therapy is directed at the reduction of oral bacteria and associated calcified deposits and non-calcified biofilm. Good home care is stressed to improve oral hygiene and plaque scores are used to quantify the extent of the improvement. Local irritating factors like calculus and defective restorations are removed. Orthodontic arch wires and brackets usually need not be removed, depending on extent and severity of disease. Removal of calculus and biofilms is achieved by hand, sonic, ultrasonic, or piezo instruments. Topical antibacterial agents, with active ingredients such as chlorhexidine diglucone or triclosan (5-chloro-2-(2,4-dichlorophenoxy)phenol), help to reduce supragingival bacterial plaque.

However, the problem is that topical liquids may not reach the subgingival environment effectively, even when delivered under pressure, because they do not remain in close contact with the root surface for long enough periods of time. There is protection by the architecture of dental plaque, a dynamic bacterial biofilm “community” that has formidable microbiologic defense mechanisms. Treating the surface of the gingiva only “masks” deeper disease. Professionally controlled delivery of solids or gels, however, is effective because these materials remain active for a long period of time. The latter chemical agents, deep inside periodontal pockets, are very effective, according to Drisko, and although the process is more time consuming than superficial brushing, the outcomes confirm the hypotheses of other experts in the field that they can be an effective means of maintaining periodontal health.

**Gingival Hyperplasia**

Overgrowth of tissue during orthodontic therapy is also a threat to periodontal health and is often more pernicious because its fibrotic superficial appearance can mimic health (Fig 3). Gingival enlargement (see Waldrop elsewhere in
Figure 2. Illustration showing what is measured using a periodontal probe. (A) Healthy environment showing a sulcus, also known as periodontal pocket if infected and/or pathologically deepened; (B) due to gingivitis, the value measured may indicate a “deep” pocket, but there is not bone loss present. This is also known as pseudopocket. (C) Illustration showing both attachment and bone loss, and what could be measured as a “deep” pocket. Even though the linear depth may be lower in this situation than in the previous, the bone loss present categorically indicates a worse prognosis. (Color version of figure is available online.)
Managing Patients at Risk

In patients with active periodontal disease, progressively resorbing bone, the threat of complications is more imminent but does not categorically contraindicate OTM. Initial therapy may be followed by surgical treatment to improve tissue topography by surgical osteoplasty/ostectomy or guided tissue regeneration (GTR). The choice of treatment plan depends on factors such as plaque score, width of the zone of keratinized tissue, and osseous architecture. The extent of the osseous surgery and predictability of regeneration depend on the extent and morphology of the osseous defect (Fig 4). Therefore, a well-orchestrated leveling and aligning of teeth can actually minimize the need for surgical bone removal during periodontal surgery and enhance bone development through forced eruption.

Some two-walled defects with shallow craters up to 4 mm may be corrected with minor bone removal (ostectomy) and an apically positioned flap. For deeper bone defects, however, new advances in tissue engineering can be used to manage three-walled defects that are deeper than 4 mm, some hemiseptal defects, and some molar furcation defects with limited tooth mobility. Currently, bone grafts using autogenuously harvested bone, allografts, such as mineralized or demineralized freeze-dried bone, with or without a resorbable or nonresorbable membrane, are commonly used with a high degree of predictability in the treatment of periodontitis. The bone graft generally serves as a scaffold for native bone growth and the introduction of growth factors can generate new bone, even ectopically in animals, by a process of osteoinduction (creating bone de novo). How, specifically, surgical mechanisms of osteoinduction relate synergistically with bone created by OTM has not yet been clearly defined. Nonetheless discoveries in tissue and genetic engineering are most promising and can add an exciting new dimension to the daily practice of dentofacial orthopedics. Clinicians who incorporate periodontal surgery with orthodontic therapy might treat new patients who would otherwise avoid orthodontic care.

The purpose of the resorbable and nonresorbable tissue membranes is to exclude epithelial ingrowth into the bony defect and provide space between the mucoperiosteal flap and periodontal bony lesion to protect osteogenesis and new attachment to the root. If gingival epithelium and connective tissue can be delayed from migrating into the defect during early wound healing, reconstruction of the periodontal tissues can progress from pluripotent cells of the periodontal ligament and alveolar bone. Membranes and bone grafting materials are often used together to reduce micromovement of the graft and are well-accepted modalities to maintain the alveolar crestal bone height and thickness.

In the past, prudence dictated that periodontists delay orthodontic tooth movement until the regenerated tissues were mature. However, recent clinical research by Wilcko and Ferguson and coworkers (see Wilcko and coworkers in this issue) have shown that tooth movement through a healing graft is not only safe but may actually
Figure 4. Bone loss analysis based on morphology. (A) One-wall defect, (B) two-wall defect, and (C) three-wall defect. More walls contribute to an improved prognosis for regeneration. (Color version of figure is available online.)
enhance the orthodontic clinical outcome and obviate any need for tooth extraction. The “periodontally accelerated osteogenic orthodontics” protocol (PAOO) presents a most promising frontier for future interdisciplinary synergy between the specialties of orthodontics and periodontists. (PAOO is a trademark of Wilcko-odontics, Inc., Erie, PA.)

**Gingival Reconstructive Procedures**

Reconstructive surgery is performed on abnormal structures that may be congenital, developmental, results of trauma, infection, or disease. Mucogingival surgery (often referred to as “periodontal plastic surgery”) is an example of reconstructive surgery available to orthodontic patients. The goal of mucogingival procedures is to restore periodontal health through the reconstruction of lost hard and soft tissue. Improvement of esthetics is another goal of the mucogingival procedures.

Whether the nature of periodontal attachment (regenerated attachment apparatus versus a long junctional epithelium or both) has a significant impact on the clinical result is not yet clearly defined in the literature and cannot be confirmed in each patient because of ethical strictures; that is, the nature of the attachment can only be confirmed by experimenting on live humans.

Tooth malposition, alveolar bone dehiscence, impinging restorative or orthodontic appliances, and chronic infection are all risk factors that contribute to mucogingival lesions known as “gingival stripping” or “runners” in the clinical orthodontic terminology. While the lesion may appear during orthodontic tooth movement no clear correlation has been demonstrated between tooth position and gingival dehiscence and may represent one of the most unfortunate misconceptions in the field of orthodontics. It is important to realize that in adolescents or transitional dentition patients, a “precocious” passive eruption, compared with adjacent retarded passive eruption, may mimic true gingival dehiscence. This is easily diagnosed by palpating the cemento-enamel junction (CEJ). If the gingival margin is at the CEJ and palpable with a probe, the lesion is a kind of “pseudo recession,” and if sufficient connective tissue thickness lies at the cervix of the tooth, no treatment is needed.

True gingival recession and bony dehiscences, however, are a constant threat that seem to be minimized by a thick buccolingual dimension of alveolar bone. Where this is absent and extraction therapy is contraindicated due to facial profile esthetics, the PAOO procedure introduced by Wilcko and coworkers serves as a well-founded alternative to orthognathic surgery in some cases. What is particularly encouraging about PAOO is that it lends greater stability than conventional orthodontic biomechanics while it accelerates the tooth movement rates by at least 200% with both outcome quality and periodontal impunity.

However, a simple prophylactic soft tissue gingival augmentation may be reasonable especially in patients with thin periodontal biotype. Treatment of gingival recession by soft tissue grafting for complete root coverage requires adequate vascular supply. This may be obtained from the subjacent underlying bone and periodontal ligament and from the gingival flap overlying the graft. The early surgeries for root coverage employed laterally positioned flap (LPF), rotational flaps, and repositioned flaps. An average of 67% root coverage can be attained by LPF. Free gingival grafts evolved from a desire to expand the zone of keratinized tissue to thicker ones with greater predictability for root coverage. Root coverage expected from free gingival grafts was estimated to be 69% of the root. Free gingival grafts have a single source of blood supply from the underlying bone, periosteum, and periodontal ligament. Coronally positioned flap procedure advances tissue already present by a distance equal to the extent of recession. Existing papillae are deepithelialized and overlapped by the newly created gingival tissue. An average of 61% root coverage can be achieved with the main limitation being the zone of keratinized tissue that is present. If the existing keratinized tissue is limited, the potential to advance a flap is also limited.

Recently, with the development of tissue engineering, enamel matrix derivative applied to coronally positioned flap has been reported to enhance root coverage to a mean coverage of 86% of the root. Enamel matrix derivative (EMD) is a resorbable, implantable material consisting of hydrophobic enamel matrix pro-
teins extracted from a developing porcine embryo. When applied in a gel form to the exposed root, the protein assembles into an insoluble three-dimensional matrix. New bone and periodontal ligament formation are reported using EMD. An average of 98% defect coverage is obtained because of the dual blood supply from the underlying periosteum and overlying flap. Since reflecting a flap interferes to some extent with the vascular beds, a newer, no-flap, tunnel preparation has been developed to slide connective tissue graft underneath the overlying flap.

Over the last few decades, connective tissue grafting has expanded the possibilities for correcting mucogingival defects. With the advent of tissue engineering, guided tissue regeneration (GTR), to a mean coverage of 86% of the root, has also been employed for root coverage using a bioabsorbable or nonresorbable membrane. The membrane is sutured in place to prevent epithelial ingrowth. The membrane is then covered completely with a coronally positioned flap because an exposed membrane is often subject to infection and may compromise the results. One benefit of this technique is that the GTR has the potential to regenerate new bone. An advantage of GTR is that there is no need for a second surgical site to obtain donor tissue. More recently, the use of acellular dermal matrix (ADM) was introduced to substitute connective tissue grafting. ADM is harvested from cadavers and treated to remove all cells but preserve the extracellular matrix including vascular structures.

A short clinical crown is another esthetic problem in orthodontic patients. Altered passive eruption is when a tooth has reached the occlusal plane and the gingival margin, is at the junction between the cervical and middle thirds of the clinical crown or in the coronal third of the clinical crown in the absence of inflammation, hypertrophy, or hyperplasia of the gingiva. This may give the appearance of excessive gingival display. Gingival overgrowth, associated with orthodontic therapy and poor plaque control, also results in the appearance of short clinical crowns (Fig 5). These conditions are treated with a flap procedure instead of a gingivectomy. Flap surgery is actually less morbid and allows complete visualization of any need for osseous resection. Sometimes altered passive eruption of soft tissue is accompanied by arrested active eruption and the crest of the alveolus is coronal to the CEJ. In such situations simple gingivectomies will fail because they do not attend to an immutable biological width, a fundamental mechanism of spatial periodontal homeostasis. Several parameters such as zone of keratinized tissue, location of alveolar crest relative to cemento-enamel junction (CEJ), and biologic width of attachment apparatus are used to determine surgical treatment modality. Lasers are also reported to have similar effects as traditional scalpel gingivectomy, without hemostasis complications, but are not shown superior to the traditional method.

**Visualizing and Exposing Impacted Teeth**

When a permanent tooth fails to erupt, a reconstructive procedure may serve to surgically expose the anatomical crown of the tooth. This should be done without compromising periodontal support. Often damage can occur when canines are erupted through infected mucosa or stainless steel ligatures are used to “lasso” the impacted tooth at its cemento-enamel junction. The primary goal of modern periodontal procedures is to expose the crown to place an orthodontic bracket, while minimizing periodontal defect on adjacent teeth. The best way to protect the erupting tooth is to erupt it within a wide
zone of keratinized tissue. The best way to keep
the tooth in keratinized tissue is to use an api-
cally positioned flap or to use a gingival graft.
After the surgery the rate of eruption may be
accelerated and orthodontic adjustments can be
made biweekly.

Determining the most suitable flap design
before surgery is very subjective due to the diffi-
culty in determining the exact tooth location in
the alveolus. A conventional two-dimensional ra-
diograph with inherent distortion makes locating
the unerupted tooth inaccurate. An innova-
tion rapidly gaining popularity in the 21st
century is cone beam CT (CBCT). The three-
dimensional CBCT scan provides the exact loca-
tion of the unerupted tooth (Fig 6). It provides a
three-dimensional image without the distortion
that is present in a panoramic radiograph. This
modern imaging simplifies presurgical flap design
planning and allows for smaller incisions and flaps,
less anesthetic, a more rapid surgical procedure,
and less postoperative discomfort. Using CBCT for
surgical exposure of impacted teeth is particularly
cost effective when pretreatment records have also
employed CBCT imaging.

Orthodontic Solutions to Periodontal
Problems
Orthodontic treatment to correctly position
teeth before placing dental implants or before
periodontal reconstruction must be offered, as

Figure 6. Sagittal views using CBCT technology of a patient presenting with supernumerary teeth in the upper central area. The image clearly shows lack of contact and damage from part of the supernumerary teeth. The image also shows that access through the palate would probably be an efficient approach without the need for removal of the deciduous teeth.
indicated, to each patient seeking treatment (Fig 7). Orthodontic treatment can reduce the likelihood of unnecessary or excessive removal of healthy bone by leveling and aligning teeth to restore alveolar crest topology. Appropriate treatment planning is essential. Orthodontic treatment is needed to achieve and maintain periodontal health. An example is where a deep dental bite that impinges on periodontal tissues may result in trauma to the periodontium. Histologically the latter will show a chronic inflammatory infiltrate, ulceration, and incomplete keratinization. Prevention of this type of situation is indicated. In addition, by correcting severe malocclusions, local factors contributing to periodontitis may be eliminated (Figs 8, 9). For example, plaque retentive contact relationships that may lead to plaque induced inflammation, and trauma from interfering occlusal contact, which in the presence of inflammation can lead to attachment loss, can be corrected. Orthodontics can also utilize equilibration (occlusal adjustment) techniques to reshape form where positional changes cannot be corrected or where anterior esthetics are a function of incisal edge shape, not only malalignment. The functional alignment of teeth beyond their static esthetic form is underestimated as a method of directing occlusal forces along the long axis of the tooth to improve periodontal architecture and to improve tooth position relative to the alveolar housing. Changing the location of malposed teeth within the alveolus can increase the width and thickness of keratinized tissue and axial loading strengthens the cribriform plate when primary occlusal trauma reduces mobility.

Figure 7. Male patient (17-year-old), with congenitally missing permanent upper lateral incisors, and three lower incisors. This is an example of treatment that requires multidisciplinary coordination.

Figure 8. Use of a temporary anchorage device (TAD) is a more modern alternative to orthognathic surgery that could aid in the intrusion of the posterior segment of the maxilla. (Color version of figure is available online.)
In certain clinical situations, vertical bony defects may be corrected by orthodontically repositioning teeth. Similarly, in restorative cases where, due to the extent of decay, a crown impinges on the needed biologic width of gingival attachment to tooth, and surgical crown lengthening is not practical, orthodontic extrusion of the tooth can be used to recreate biologic width. Caution in the restoration of the tooth is needed in these situations, as crown-to-root ratio ideally should be less than 1:1 to try to ensure a good long-term prognosis, even though this is a relative objective. Many teeth with severely shortened roots function well when both functional and para-functional forces are equilibrated to a mutual protected schema. Similarly, in cases where implant therapy is planned, and there is not adequate vertical height of bone, forced orthodontic extrusion can be a means of attaining adequate alveolar ridge height. However, without an appreciation of microtrauma the longevity of the implant is jeopardized.

**Conclusions**

Attaining optimal outcomes in daily clinical practice may involve bringing the expertise of more than one dental specialty. This requires coordination, communication, and sometimes compromise. Modern technology allows easier specialty interaction. Biology provides the fundamental basis, while technology provides the seamless vehicle needed in everyday clinical synergy. The new orthodontist who wishes to build a career that is uniquely compatible with emerging sciences of the new century would be wise to consult the Web for a plethora of valuable insights, policies and, even in the absence of hard unequivocal data, review reports that may assist in providing information into periodontally sound dentofacial and dentoalveolar orthopedics. The best local source of data is an experienced, responsible and enlightened periodontist. In this regard, collegial humility is no vice and orthodontic overconfidence is no virtue in interdisciplinary en-
References


