1

Orthodontic Tissue Engineering: A 20-Year Retrospective and Philosophical Polemic©

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Dedication

This chapter is dedicated to Professor Spiro Chaconas, Founder and Chairman Emeritus, Section of Orthodontics, UCLA School of Dentistry. Professor Chaconas, presently enjoying a well-deserved retirement in Sothern California, was an exceptional leader, friend, and mentor for over three generations of orthodontists at UCLA. He taught his protégés to enter private practice with confidence and engage the inevitable vicissitudes of our careers – many rather brutal – with professional élan, stoic indifference, transcendent vision, and personal humility. We did.

Thank you, Spiro.

Introductory Rationale

Since 2001, this author – dual-certified in both orthodontic and periodontic – has collaborated with a number of orthodontists and periodontists in an effort to engineer a novel alveolus bone that could accommodate the full complement of human dentition. This was attempted to liberate a naturally "full" smile from the strictures of skeletal malalignment and so-called arch length deficiencies. By the year 2023, we were able to develop protocols that achieved that goal and accelerate the rate of tooth movement three to fourfold. In addition, instances of pernicious side effects like apical root resorption and periodontal attachment loss were predictably minimized or nonexistent compared to traditional edgewise therapies. In that regard, our protocols, both surgical and nonsurgical in a phrase, proved to be "faster, safer, and better." These revelations were brought into high relief by a 20-year retrospect as attested to by studies cited herein.

Orthodontists' attempts to enhance the esthetic value of the patients' lower face are indeed laudable goals. However, the widespread popularity of extraction therapy presents a sobering challenge because it notoriously has been haunted by the unfortunate and unpredictable side effects of premature lower face aging and unsightly flattened (so-called "dished-in") profiles in maturity. Moreover, since these unsightly facial profiles often become most apparent years after active therapy has ended, they are subtle assaults on facial beauty. While evident to the general population, the iatrogenic deformity presents a pattern that is vaguely unsightly but

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nondescript to laymen. This chapter explains that cell- and tissue-level biology is often ignored in orthodontic curricula and sacrificed by inordinate preoccupations with gross anatomy. But periodontology revels in cell-level dynamics and affords us, as specialty science integrators, to reveal a universe of new orthodontic science, we call "orthodontic tissue engineering (OTE)."

If dentists claim a desire for "best care," we must ask how one defines that superlative term. By definition, a superlative is an absolute, and whatever treatment most closely approximates that ideal is reasonably argued as "best." Is "best care" which is predictable, fastest, with fewest pernicious side effects, least painful, most stable, and most compatible with contemporary cultural values?

This chapter is an attempt to organize a compelling rationale for this new protocol in terms of the underlying cellular dynamics that allow it to achieve case outcome stability superior to the very unstable outcomes that are predictably disappointing in traditional care. Ironically, the new concept of "accelerated orthodontic therapy" was met with political opposition and excessively cynical skepticism by established practitioners. Yet truth prevails and the luddites and clinical nay-sayers of the 20th century were proven wrong by 21st-century science. This chapter boldly addressees this controversy as a tribute to intrepid clinicians who preceded us and as a scientific reminder that hard data and scientific epistemological inquiry, however, disruptive to prevailing thought and wishes, will out.

This definition seems sensible to us. Therefore, the aim of this chapter is to describe the ramifications of an emerging perspective and clinical protocol in those terms. Although unheard of by some, the subject is neither new nor novel because it has been evolving over time within the ebb and flow of scientific evolution. It nonetheless brings disruptive issues and protocols that are certainly more predictable, faster, with fewest pernicious side effects, less painful in adjustments, more stable, and more compatible with contemporary cultural values than traditional extraction alternatives. The problem with science is that it has no master. So, it flies in the face of convention and traditional bias.

Late 20th-century science has delivered a collection of empirical observations and corticotomy protocols that are embodied under the collective rubric, surgically facilitated orthodontic therapy (SFOT) which in this book will be called orthodontically driven osteogenesis (ODO). This term referring to a particular histological reaction, subsumes a number of protocols that are mere variants of the same basic biological phenomena. These terms include but are not limited to "selective alveolar (-us) decortication" (SAD), decortication without a bone graft, "stem cell orthodontic therapy" (SCOT), "stem cell alveolar therapy" (SCAT), "corticision" when a scalpel is used, "accelerated osteogenic orthodontics" (AOO) where a bone graft is combined with SAD, "periodontal(ly) accelerated osteogenic orthodontics" (PAOO) synonymous with AOO, and here, "orthodontic tissue engineering" (OTE) referring to a 21st century protocol focusing on permanent alveolus bone phenotype alteration. We posit that emerging periodontal sciences, the biology of healing bone, and cell-level biology, which underlie ODO, are as integral to orthodontics, as civil engineering is to good architectural design. ODO is an example of the clinical science of engineered morphogenetic bone modeling – pioneered by the Russian orthopedist Professor Gavriil Ilizarov – synthesized with traditional orthopedic biomechanics (Figure 1.1). This Russian orthopedic surgeon proved beyond doubt and under great oppression that bone is malleable and can be reshaped to a more physiological form at will. That principle applies to the alveolus bone as well (Figure 1.1).

We pose a challenge to traditional biomechanics to enhance clinical efficacy, ameliorate pernicious side effects, and advance the orthodontic specialty beyond the strictures of simple mechanical art. The issues discussed in this

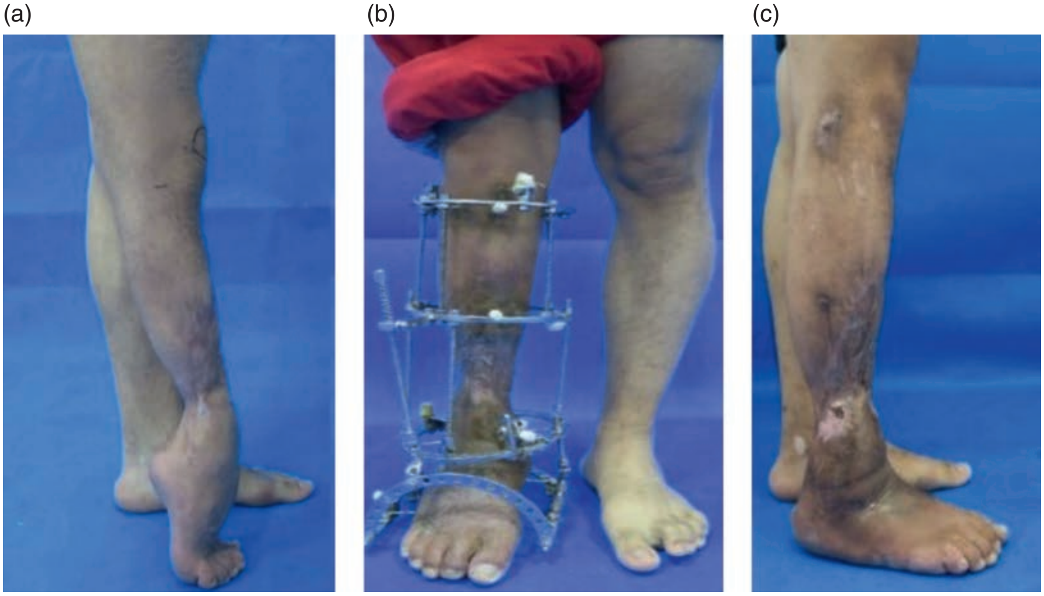

Figure 1.1 This treatment demonstrates what can be done to correct a deformed long bone. The same principle used by Gavriil Ilizarov can be applied to the alveolus bone in correcting dentoalveolar deformities. The leg deformity in (a) represents a deformed bone. (b) The Ilizarov orthopedic device with his surgical protocol can lengthen long bones at the rate of 1 mm/day. (c) Demonstrates an improved esthetic appearance and function. This is what OTE attempts to do with the dental alveolus bone. *Source:* Dong *et al.* (2021)/Reproduced with permission from Tsinghua University Press Ltd.

polemic are based on the dual-certified author's 50 years of integrating periodontics and orthodontic in an urban private, but academic, practice and 20 years of understanding ODO. It also reflects the combined work of a growing global community of biologists and dentists, formally trained, or passionately interested in reengineering the mass and shape of the foundation of the human dentition. Hence, we write in the first-person plural, not to imply lock-step concordance, but rather a general agreement that is compatible with a wide variety of readers. Some repetition will be noticed in this discourse, but that serves as an intentional pedagogical device. The subject matter is quite novel to some readers because we employ a technical prose of molecular biologists, tissue engineers, periodontists, and orthodontists. Moreover, repetition of a new concept within several different contexts can only enrich the conceptualization. Hopefully, the literary device will edify and not distract.

Our objective here is not to proselytize but rather to serve examples of what *can* be achieved by others who wish to minimize extraction therapy side effects. But we hope this is received in a provocative manner to stimulate a meaningful dialectical exchange rather than contentious debate, misinformation, distortions, and misrepresentations that have marred the development of this topic. What is practiced by others is beyond our scope of control. So, we do not call for the immediate condemnation of those who are uncomfortable with protocols presented herein. We focus on scientific advances in ancillary biological fields too fascinating to ignore. And, these innovations can serve as both a beacon and safe harbor for those who are dissatisfied with the limits of the *status quo*. We are here to show a better path but only for those who wish to embark upon it.

We do not seek to condemn legitimate extraction therapy categorically, but we are intentionally provocative about the perfunctory use of healthy tooth extraction merely for the sake of mindless expediency. We also object philosophically to the excuse of "art" as a refuge from the inarguable demands of science. The fact is that ODO is here to stay, and its use will most likely continue to proliferate worldwide, as any scientific innovation always does. A recent review of the subject published by Hoogeveen *et al.* (2014) concludes that SFOT "… might effectively shorten the duration of treatment, but *careful treatment planning*, early activation of appliances, and short intervals between check-ups are recommended. SFOT … is not associated with complications such as loss of tooth vitality, periodontal problems, or severe root resorption … prospective research is still needed … " (Emphasis added).

The lack of prospective, multi-replicated, double-blind, placebo-controlled, multisite, and human clinical studies to which they allude, does not invalidate the ODO innovation. Rather, the lack of such a prospective gold-standard analysis speaks to the impracticality of such studies, the lack of funding, and the inchoate nature that SFOT shares with *many effective clinical disciplines*. In this chapter, we elaborate on the necessary items of interest that accord with "careful treatment planning."

Topical Issues

Since the dawn of the orthodontic specialty a philosophical debate has reigned over the intellectual life of most orthodontists: Are we artists or scientists? One reconciliation of this ostensible dichotomy is that art is a goal, and science is the method. The corollary presumption is that an artful outcome *via* science also represents an optimal physiologic state, e.g., mutually protected occlusion.

Many orthodontists believe that only the most efficient methods of biomechanical loading should define our specialty. To them, periodontal biology is considered an ancillary and often irritating companion. We disagree. We propose that the periodontium is a useful but ignored asset in the creation of optimal function and esthetically harmonious outcomes. These are the axioms upon which we base our posits: Tissue is a collection of *cells* serving a common function. An organ is a collection of *tissues* serving a common function. Engineering is the physical manipulation of a natural phenomenon toward a predesigned schema. *OTE* in the title refers to the burgeoning science of manipulating the dentoalveolar complex and rerouting its architectural development to a predetermined and improved pattern. A plethora of protocols have blossomed over the last two decades and presently constitute a complex collage of techniques. In this proliferation, critical problems of clinical management and intellectual development have arisen that we have solved. These solutions we share.

As a civil engineer can manipulate the course of a river to convert fluid dynamics to electromagnetic potentials, we proffer nothing less *vis a vis* alveolar bone physiology. We propose that the orthodontist can manipulate the alveolus bone to facilitate *orthodontic tooth movement* (OTM) and in the process *make better bone*. The materials of OTE are a collection of surgical and nonsurgical periodontal protocols that are applied to areas of bone beneath a dentition needing orthodontic treatment. The principal method is a collection of *selective alveolar decortication* (SAD) protocols referred to in the aggregate as "Surgically Facilitated Orthodontic Therapy" (SFOT). For the sake of convenience, in this review, we shall use the acronyms SFOT, OTE, and ODO roughly synonymously, while the latter encompasses more nonsurgical modalities.

Since 1981, this author has attempted to integrate tissue dynamics into the biomechanical procedures of clinical orthodontics. At that time, tissue engineering was just beginning to appear in the biological literature. In retrospect, we see that the manipulative techniques of OTE paralleled similar 21st-century bioengineering protocols in other fields of the human body from dermatology to consciousness (Nilforoushzadeh *et al.*, 2022).

The popular emergence *of* SFOT in the early 1990s in the United States brought a promise of sustaining real-world benefits to patients. Faster care, less infection, and significant reduction of that embarrassing ghost of orthodontics, relapse, are OTE's most salient merits. In 2006, we were able to organize these new clinical observations with new science in a seminal book chapter published by the Harvard Society for the Advancement of Orthodontics. The title, *Tissue Engineering for Orthodontists – a modest first step* (Murphy, 2006) foretold this chapter, a modest *second step*. We look back over 20 years and address the growing pains of this new approach to alveolus bone development and liberation from the strictures of working within a deformed bony base. Further steps in this advancing science we relinquish to readers and their place in a new century. In this regard, the past is prologue indeed, but only for those who wish to travel its rocky albeit compelling path. The intellectual foundation for SFOT has enjoyed a long history in the scientific literature reviewed a decade ago (Murphy *et al.,* 2012). The first significant attempt was Cunningham in 1894. So, the title of this chapter should more accurately read " … a 130-year retrospective." But 20 years is more manageable and experiential for retrospective musings.

The early procedures, mainly luxated osteotomies, remained in the German literature for decades until published in English by Kole in 1959. These rudimentary surgeries of the early 20th century were materially refined by Suya (1991) to make them more predictable. After Suya, SFOT was carried into the 21st century by a global consortium of creative clinicians. With subsequent analyses and decades of clinical success, SFOT now enjoys increasing popularity. At this point, OTE, both surgical and nonsurgical, has certainly earned a secure albeit inchoate place in the pantheon of legitimate contemporary orthodontic protocols.

Early attempts to combine minor surgery with traditional OTM were inspired by the universal concern that slow treatment is not necessarily the best treatment. Indeed, the pernicious side effects of OTM, all correlate with treatment duration (Artun and Brobakken, 1986; Kurol *et al.*, 1996).

So, it appears that *good treatment is fast treatment* when biological imperatives are respected, and manipulation of the alveolus bone is understood. The fact is that SFOT-induced stability exceeds the common standards reported by Professor Little's revelations (Little *et al.*, 1988) of the University of Washington database (Ferguson *et al.*, 2014, 2016) (Wilcko, 2023).[1] OTE was also sanctioned *de facto* by the American Association of Orthodontists (AAO) Council on Scientific Affairs. It selected Dr. Susan Baloul (2016) as the winner of the 2010 Milo Hellman Research Award for research excellence. Dr. Baloul's experiment demonstrated that osteoblast/osteoclast "coupling," a natural event, defined the mechanism by which *loaded* selective alveolus decortication (SAD) elicits fundamental changes in alveolus morphology. This was achieved by analyzing animal RNA markers with micro-CT measurement and clarifies the cellular mechanisms of accelerated OTM. Despite the professional impediments of healthy skepticism and unintentional misrepresentation, the Baloul confirmation of natural biology plants the flag of legitimacy for SFOT beyond doubt.

Twenty-first century orthodontists are already incorporating SFOT into their therapeutic repertoire and the American standard of care demands that it be proffered as a *valid alternative* when patients ask that inevitable question, "How long will treatment last?" Now, the orthodontist can justifiably say, "You have two choices of safe and effective treatment duration, *fast or slow*." The disadvantage for some patients will always be the minor (2–8 mm deep) surgery. But we ask our audience, "… compared with the traditional extraction of healthy teeth and dentoalveolar units is

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[1] Personal communication, 2023.

not our non-extraction proposal actually *less morbid*?" The obvious advantage of SFOT is speed, but the biological effect goes *beyond speed*. Pretty clinical outcomes and the high relapse rates (Little *et al.*, 1988; Bernabe *et al.*, 2017) have been, respectively, the boon and the bane of the orthodontic specialty for over a century. Alas, Little reports, "post-retention anterior crowding is both unpredictable and variable and no pretreatment variables … seem to be useful predictors." (Little, 1990) SFOT changes that. It is predictable and consistent.

Many doctors in many countries have documented that SFOT works well for most patients. But several skeptical authors and a few iconoclastic cynics are those who have either failed to achieve clinically significant results or explicitly turned down offers to attend our free lectures. This opens the possibility that the reader might universalize the experimental failures of a few to the experience of *all* SFOT. Such a universal misinterpretation is a big mistake. We have found that the limitations of the skeptical group are usually due to inadequate intervention, inadequate training, or a lack of long-term adjunctive therapy. Our rejoinder to insinuations that OTE does not work is a "black swan argument," i.e., "It only takes one black swan to disprove the notion that all swans are white." We have seen too many "black swans" to deny OTE efficacy. So, we interpret failed experiments as proof that critics simply *do not know* how to do it. Other critics of OTE are too inexperienced in the SFOT to manage subtle healing variance. They acknowledge it works for a short period of time. But they criticize the half-way treatment because it does not last long and, therefore, lacks practical clinical relevance (Buschang *et al.*, 2012). This conclusion is an error.

A negative result is caused by misunderstanding the whole protocol. After the first intervention, the tissue engineer must sustain the consequent osteopenic state with an orchestrated combination of osteopenia management and bone-stimulating OTM. And, that is the second half of the protocol which many critics miss. It appears critical research groups may have been entrapped by a kind of "Newtonian bias," i.e., a preoccupation with mechanical engineering, not the fascinating biomathematical concepts like fractal geometry and the emergent states of nonlinear complexity that manifest in well-executed OTE (see: Mandelbrot; Kaplan, Recommended Readings.) Such is the novitiate status of many OTE practitioners.

When applied force bends (strains) bone in an osteopenic state normal recalcification is delayed. In orthopedic medicine, a persistent osteopenic state in healing fractures is termed *a hypertrophic nonunion*. But in clinical orthodontics, engineering transient osteopenia facilitates excellent outcomes in a little as 3–12 months. Some critics claim that when interruptions occur a second surgery to reinstate the osteopenia would be needed. But a second SFOT surgery is *not necessary* when appropriate biomechanical forces are levied and maintained. Wire-stressing teeth strain bone, and this is sufficient to sustain the osteopenic state indefinitely. The so-called latent period in OTM is bone decalcifying sufficiently to cause tooth mobility. That is basic orthodontic biology. And when the bone is more osteopenic, though latent periods may still prevail, the teeth move faster. It is that simple. The key to success is to sustain osseous strain at intervals no longer than 1–2 weeks.

Occasionally, misplaced loading in OTM causes strain maldistribution or the treatment is interrupted for longer than 4 weeks. When this occurs the osteopenic state will wither as the bone becomes recalcified. The osteopenic state, i.e., the regional acceleratory phenomenon (RAP) can also be resumed by sequential perturbation of the alveolus bones every 1–4 weeks *via* small bur perforations discussed below. But when SFOT creates a functional osteopenic state it is *sustained indefinitely* if teeth are constantly in motion. This accelerated bony state allows semiweekly adjustments and on occasion daily adjustments. Thus, constant surveillance and constant bone straining by OTM should obviate any fear of closure of the so-called "limited windows of opportunity." There are only incomplete treatments; *there are no windows.*

A major milestone arrived when Wilcko *et al.* published the results of adding a bone graft to SAD. This is referred to as *PAOO*,[2] and it stimulated an international flurry of creative progress, with even a comparison of transmucosal bur perforations. Furthermore, when combined with injections of platelet-rich plasma (Gulec *et al.*, 2017), orthodontics has achieved an entirely new professional identity. Two decades of universal success, international endorsements, and a plethora of scientific articles have proven that SFOT is *faster, safer, and better* than the traditional art of wire bending. For this reason, it may be unwise in the future to treat orthodontic patients without offering this biology-based protocol in documents of informed consent. The contents of this second edition book lend credence to a new identity and the authors personify the spirit of free inquiry, insatiable curiosity, and intrepid perseverance necessary to sustain it. Science, once liberated from the bottle of obscurity, proceeds on its own momentum. We posit most humbly that we are merely the messengers of this new clinical science.

In reviewing the rocky sojourn of SFOT through history, some major themes emerge. The first is whether the essence of orthodontic practice is art or science. The resolution of this dichotomy is that orthodontics is neither and both. Art and science are merely two different but complementary epistemological perspectives of the world; they are the Platonic and the Aristotelian. Synthesized appropriately, the ontological insights of both, coordinated well, are critical to the successful practice of orthodontics.

A second and increasingly less contentious but nonetheless salient perspective facing orthodontics is the perennial conflict between extraction and non-extraction protocols. This is germane to our discussion of SFOT because traditionally the decision to extract or not extract was a matter of style or a biased choice from a myriad of cephalometric norms. But SFOT introduces new objective data that fortifies the non-extraction option to enhance a *new standard of esthetics*. No longer constrained by anterior limits of arch form, expansion to protrusive smiles (in contrast to Angle's ideal profile of Apollo Belvedere) appears to be emerging as the new standard for social esthetics. And yet, a kind of cultural inertia and drag on professional progress delays a definitive reconciliation of extraction and non-extraction philosophies. Our new approach can obviate any need for the former and facilitate the latter.

The Alveolus Bone Is Not a Process

Recent revelations in the literature about OTE suggest that knowledge of the alveolus bone has suffered greatly from two major misconceptions. First, it is not a *process*. A bony process, like the zygomatic or mastoid, is a projection of a larger body for muscle attachment or mechanical advantage. So, physiology of an anatomical "process" should be identical to that of its basal bone. But the alveolus bone is somewhat independent of the maxillary or mandibular corpus in origin, function, and fate. In congenital anodontia, it is absent, and its behavior reflects that of the tooth roots, not the subjacent corpi.

In dentistry, since the term "process" has been used for centuries, it is naturally assumed to be a structural and behavioral extension of the basal bone, i.e., maxillary or mandibular body. But observing the absence of alveolus in congenital anodontia one must logically question the legitimacy of that term. Thus, we propose it be referred to as simply "the alveolus." The alveolus bone is

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[2] Periodontally accelerated osteogenic orthodontics.

developmentally, structurally, and functionally unique, similar to and *contiguous with* the underlying body of bone. But it is *neither anatomically continuous nor physiologically identical*.

The alveolus bone lives, thrives, and dies by virtue of root positions.

If we posit that arch length deficiency (crowding) reflects ectopic eruption and the alveolus bone emerges only upon tooth eruption, then logically the teeth are not erupting ectopically because of a "small bone." The opposite is true: *the bone is small because the teeth are crowded*. Since the bone development will "follow" the root to some degree, we posit that one should evaluate potential space (available space) by measuring the circumference of the labial alveolus rather than relying on mere visual inspection or adding the sums of mesiodistal widths of teeth.

The second major misconception about the alveolus is that its form, its phenotype, is immutable and thus, risks dehiscence when arches are expanded. Our experience with the alveolus bone suggests that it its *not immutable*; it is in fact, under the right conditions, very malleable and invites expansion to accommodate an entire natural dentition.

Epigenetics and the Waddington Landscape

In 1957, Conrad Waddington, a biologist and polymath, described mammalian development as unidirectional,[3] which means that embryonic stem cells develop into a more mature differentiated state. Explaining interruptions in the developmental expression of the genotype, he drew a "developmental landscape" (Figure 1.2).

Waddington's Epigenetic Landscape (Figure 1.2) is a visual metaphor showing how stem cell differentiation is analogous to a ball rolling down an incline. Here, we use it to demonstrate epigenetic *alteration* of a developmental trajectory with OTE. At the top of the incline, the ball symbolizes tissue in development during healing, where it mimics its original ontogeny. The pull of gravity down the incline symbolizes the force of nature in natural development or, in our case, the force of therapeutic manipulation.

During the process, the cells become specialized by deleting or inactivating unnecessary genetic information. Since normal cells do not lose differentiation potential, i.e., transformative information during their differentiation, they can differentiate into virtually any tissue element. And tissue mimics that cellular differentiation. In cell development genes are just silenced but can be reactivated by exposure to defining stimuli called "epigenetic perturbation." We submit that SAD, PAOO, and other OTE protocols constitute the same. This is the key to alveolus bone malleability and the orthodontic stability it renders. Loading a healing bone overcomes any determinative barrier, termed "canalization" (ridges in the landscape).

The term "epigenetics" was coined by Waddington to introduce the idea that some threshold environmental phenomena can modify the expression of chromosomes. He contended that a focus on natural selection as a determinate of structural phenotype must also consider the nonheritable role of epigenetic dynamics. The limit is dictated by a physiologic negotiation between the genomic options and the environmental perturbation which the genome "recognizes" and uses to select from a variety of expressions depending on the developmental environment. That is, DNA is not destiny nor a blueprint for a fixed and immutable phenotype. The DNA is rather like a survival manual, telling the tissue how to react depending on whatever environmental challenge presents. This "challenge" is termed an "epigenetic perturbation." This recognition and allowance for alveolar phenotypic expansion in turn depends on the *threshold* of epigenetic perturbation, (infection, surgery,

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[3] Dedifferentiation and trans-differentiation had not yet been introduced in the vocabulary of developmental biology.

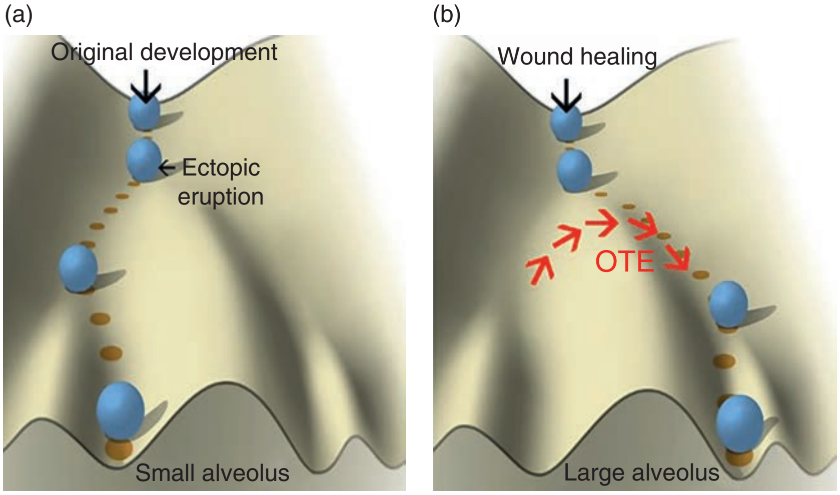

Figure 1.2 Phenotype development and recapitulating regional ontogeny in the alveolus bone is demonstrated, yet modified from Waddington's epigenetic landscape. The original landscape on the left is changed to demonstrate how SFOT and TMP can cause such an epigenetic perturbation that sufficient drive over the canalization ridges will redirect local healing toward a new form of bony phenotype. This stabilizes the orthodontic outcome better than traditional methods of retention which have an 80% to 90% failure rate over 10–20 years. The tissue engineering methods explained in this chapter not only provide a new theory of oral tissue dynamics but also comport exactly with independent demonstrations of stability in medical long bone surgery. This happens because the cells in a loaded healing wound can recapitulate regional ontogeny. In both schematics the rolling ball of this visual metaphor represents tissue development in ontogeny and local wound healing; the end of its trajectory is full development and complete healing. The landscape's valleys (canals) represent optional and alternative developmental pathways and the ridges represent phenotype stability which will manifest depending on different local developmental environments. OTE techniques are "epigenetic perturbations" that overcome the symbolic ridges' impediments to stable phenotypes. *Source:* Mitchell (2015) / PLOS / CC BY 4.0.

metabolic disorders, methylation, etc.). These possible perturbing factors include the influence of orthodontic forces loading a *healing* bone. Siegal and Bergman (2002) argued that phenotype is "robust to changes," and we interpret that as a reference to the potential stability of orthodontic clinical outcomes. The robustness they refer to in epigenetic terminology is visualized as "canalization," analogous to "energy wells," which is the result of "long-term natural selection for optimal phenotypes." We contend that in wound healing, which is a regional redux of embryological development, this process is mimicked and, thus, can be applied to the wound healing of decorticated bone and stem cells, endogenous or grafted.

Without an influential perturbation, a healing bone normally reverts to its original phenotype. In the case of infection, fibroplasia replaces parenchymal regeneration thus forming scar tissue, itself a qualitative change in phenotype. If a broken bone is immobilized and heals well, it will endorse normal environmental stresses, e.g., running, walking, load bearing, and return to its original form. However, if fixation is inadequate and movement occurs under a cast, the movement is an exogenous influence sufficient to overcome normal robustness (canalization). Then a hypertrophic nonunion may occur. If there is an inadequate blood supply to the fracture, an atrophic nonunion will develop. Each of these forms of clinical outcome represents an altered trajectory down a developmental canal. Sufficient to maintain an osteopenic state, the orthodontic load will also prevent recalcification, providing less resistance to OTM. The fundamental biological principle at work in this phenomenon is the fact that a healing wound mimics the original development of phenotype. That is, *a healing wound recapitulates regional ontogeny*.

In Figure 1.2, Waddington's epigenetic landscape model, (a) represents malocclusion development as an unperturbed genetic expression hits an ectopic eruption problematic enough to qualify as an "epigenetic perturbation." (b) Illustrates a perturbation during healing and reprogramming of development to an alternative morphotype along a novel developmental trajectory. Ridges represent barriers to differential development and valleys (canals) represent stable morphotypes. Overcoming the ridges requires a kind of "energy of activation" and a threshold of "epigenetic perturbation." This may take the form of infection as in scar formation, surgery skill in the hands of a plastic surgeon, or PAOO in the hands of a skilled orthodontist–periodontist team. The therapeutic intervention is designated by the red arrows, changing trajectory "a" to trajectory "b." The result is a reengineered alveolus, morphotype (a) to morphotype (b) a larger alveolus bone, which is secure in orthodontic stability by deep canalization.

Thus, rather than the DNA acting as exact "blueprint" for a fixed and immutable phenotypic form, the type of genetic expression and the final configuration of the alveolus bone depends upon (i) the genomic options available, (ii) the physical resources available, e.g., grafted scaffolding, (iii) the limits to which soft tissue periosteum can be extended (stretched or "relieved of tension") during surgery, and (iv) general regenerative capacity of the individual set by age, physical health, metabolic robustness of local tissue, atrophic, and degree of vascularity or fibrosis, i.e., the cell/fiber element ratio.

One of the great advantages of SFOT is that alveolar bone can be enlarged sufficiently to accommodate an idealized dental arch rather than modifying a normal *healthy dentition*, with odontoplasty or healthy teeth extractions, *to match inferior bone*. In a way, the ability to "build a better bone" renders, the extraction–expansion debate somewhat moot as a simplistic and false dichotomy, just as epigenetics has rendered the nature–nurture debate into an anachronistic dichotomy.

From Osteotomy to Corticotomy to Tissue Engineering

When reviewing the history of the corticotomy, one discovers that it originated in attempts to minimize the harsh side effects of major segmental osteotomy. The history is complicated by the fact that early writers used the terms osteotomy and corticotomy synonymously. So, much of the early literature is vague and prone to misinterpretation. An osteotomy starts with a linear decortication of bone and ends with a physical "movement" or "mobilization" (read: fracture) of a section of bone the way one might break a twig from the branch of a tree. Thus, "mobilization" is a euphemism for a kind of purposeful fracturing of bone sometimes literally done with a mallet and chisel. Whereas a corticotomy is limited to gentle incisions *without any luxation* or fracture. When studying OTE one must keep in mind the fundamental effects and esoteric mechanisms which facilitate the phenomena.

These effects, "observed" in the mind of the doctor during OTE occur sub-clinically at the tissue and cell level. They are less clearly defined than clinic-level gross anatomy, a level to which most orthodontists are accustomed. Therefore, new thinking must occur which could not have been appreciated by the specialty's earlier advocates. Ironically, the mechanism that made OTE successful may have been singularly intuited as early as the 19th century by John Nutting Farrar (1839–1913) around 1888. He was referring to orthodontic effects from a "whole bone" perspective when he wrote,

> ... The softening of the socket breaks the fixedness or rigidity of the tooth leaving it comparatively easy to move, either by resorption of the tissues or by *bending of the alveolar process or both* (Emphasis added)

Farrar Revisited: Bending Bone and CPO

Farrar's writing invites natural queries about the optimal threshold for bone fractures but is more germane to OTE, the thresholds for therapeutic bone modeling. The answers lie just beyond the scope of classical orthodontic literature residing instead within the fields of recent orthopedic and osteology (Mavcic and Antolic, 2012).

This bending of the *whole alveolus bone* stimulates regenerative osseous metabolism, *compensatory bone resorption, and deposition,* which occurs in areas of *shear loading*. The net result is a reconfiguration of cortical and trabecular architecture consistent with Wolff's Law (Wolff, 1892). For example, in Figure 1.3 if a premolar is loaded with a buccal vector, the alveolus is bent buccally causing it to assume a relatively convex surface on the palatal aspect and a concavity on the buccal aspect. This buccal bending produces *shear tension* on the palatal convex surface signified by (−) and *shear compression* signified by (+) on the labial surface (Figure 1.3).

Close analysis of a loaded alveolus depicted in Figures 1.3a, b, an exaggerated diagram of OTM, shows a much more complicated vector system. One key to reconciliation of the medical–dental paradox is to imagine the cribriform plate and periodontal ligament as analogous to endosteal elements. Figure 1.3b is a closer look at the complicated vector system in Figure 1.3a.

In Figure 1.3, orthodontic force (F) is applied to the palatal side of a bicuspid. The alveolus palatal cortical plate and the buccal cortical plate are distorted, ("bent") buccally in the

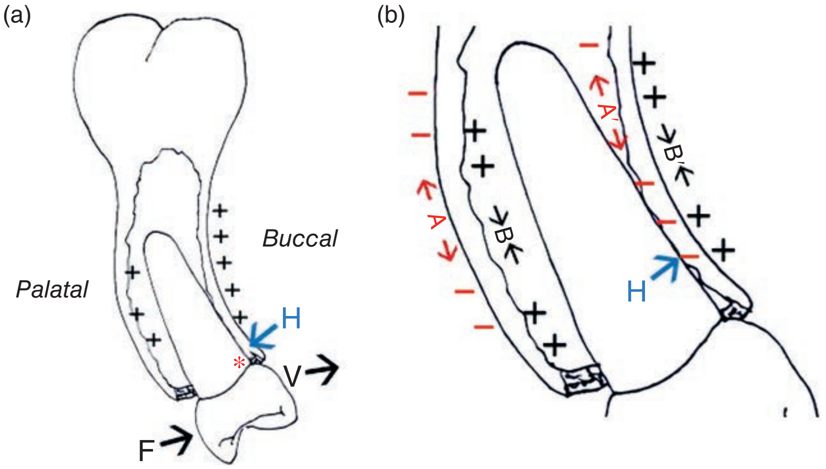

Figure 1.3 This figure is intended to demonstrate the entire load dissipation when a force is applied to a tooth. The traditional "pressure-tension" concept is so elementary that it is misleading and archaic. The figure demonstrates shear compression and shear tensions on the alveolus bone and explains why the cell-level responses to the orthodontic load "bend" the cribriform plate in such a way that the dynamics go beyond simple crestal pressure and tension. It is the *bending* of the alveolus bone and the shear compression and shear stretching of the cribriform plate and bony cortex periosteal tissue that results in phenotypic change. A plus sign (+) represents osteogenesis at areas of shear compression. A minus sign (−) represents osteoclastic resorption at areas of shear tension.

direction of the sum vector (V). Note the asterisk representing the area of hyalinization, an ischemic necrosis (infarct) of the crestal periodontal ligament.

Observe in Figure 1.3b, how force (F) induces *shear tension* on the palatal subperiosteal cortical surface and shear *compression* on the palatal cribriform plate. On the buccal alveolus cortex, the palatal surface of the buccal alveolus cortex also "senses" *shear* tension. As the same force bends the alveolus in the direction of vector (V), it produces *shear tension* on the palatal aspect of the buccal cortex and *shear compression* on the buccal aspect of the buccal cortex. In periodontal terminology, this shear loading-osteogenesis creates "buttressing bone" and is often attributed to trauma from occlusion.

These buttressing bone phenomena are limited by genotypic codes, but the expression of those codes depends upon the environmental perturbations they encounter. In summary, we propose that the bone apposition and resorption patterns seen in the periodontal ligament during OTM are due to differential shear forces during alveolus "bone bending," *simultaneous with* periodontal ligament infarction, i.e., (hyalinization "H" in Figure 1.3). We contend that the surrounding osteopenia caused by the bone bending is amplified by the OTE-induced Regional Acceleratory Phenomenon (RAP). Further the mobility phenomenon is referred to, in periodontal terms, as primary mobility. When mobility is caused by progressive loss of the attachment apparatus the appropriate term is secondary mobility. The concern about the exaggerated OTE and ODO-induced mobility is not to be confused with secondary mobility caused by attachment loss. Patients should be explicitly reassured that OTE/ODO mobility is a therapeutic asset and is completely reversible in the retention period as the bone quickly recalcifies to its original density.

The two seemingly contradictory clichés of orthopedics and orthodontics about how load affects bone physiology, (load causes osteoclasia in dentistry; load causes osteogenesis in medical orthopedics) are thus reconciled if one considers the cribriform plate and periodontal ligament as a kind of "modified endosteum," and a distinction is made between the orthodontic model of *compression loading* and the medical orthopedic model of *shear loading*. As force (F) is applied to the tooth, in Figure 1.3, it does indeed move in the socket, but pressures and tensions are applied with a myriad of component vectors. Therefore, we propose that the "pressure–tension" metaphor is so simplistic that it acts as a kind of facile intellectual red herring." This intellectual "detour" inhibits a full conceptualization of bone dynamics during PAOO and alveolus enlargement. This leads the reader away from a more sophisticated and accurate bone physiology that allows alveolus phenotype change and, therefore, increased orthodontic stability.

In other words, the reaction of orthodontic load we posit is better explained by the *shear* tension or *shear* compression analysis of the bone cortices and periosteum. Hyalinization of the periodontal ligament provides little knowledge about "whole bone" periosteal strain. This mechanism to explain OTM within the alveolus bone and cribriform plate is superior to the pressure–tension hypothesis because it is consistent with basic science and general orthopedic principles. Looking beyond the ligament with a "whole bone" perspective gives a more realistic and comprehensive assessment of bone-under-load. It also explains the universal clinical successes of both SFOT in general and ODO (or PAOO) in particular.

This principle of compensatory periosteal osteogenesis (CPO) can be seen when a *palatal alveolus bone – not* dental – expander (Figure 1.4a) is activated. The purpose, to expand the *palatal* alveolus bone with acrylic panels, and stimulate bone deposition on the concave *buccal* surface of bone. This appositional behavior of woven bone on the buccal alveolar aspect can only be explained by what we call *CPO*. Figure 1.4 demonstrates a real-life application of the schema in Figure 1.3. The behavior of the convex buccal

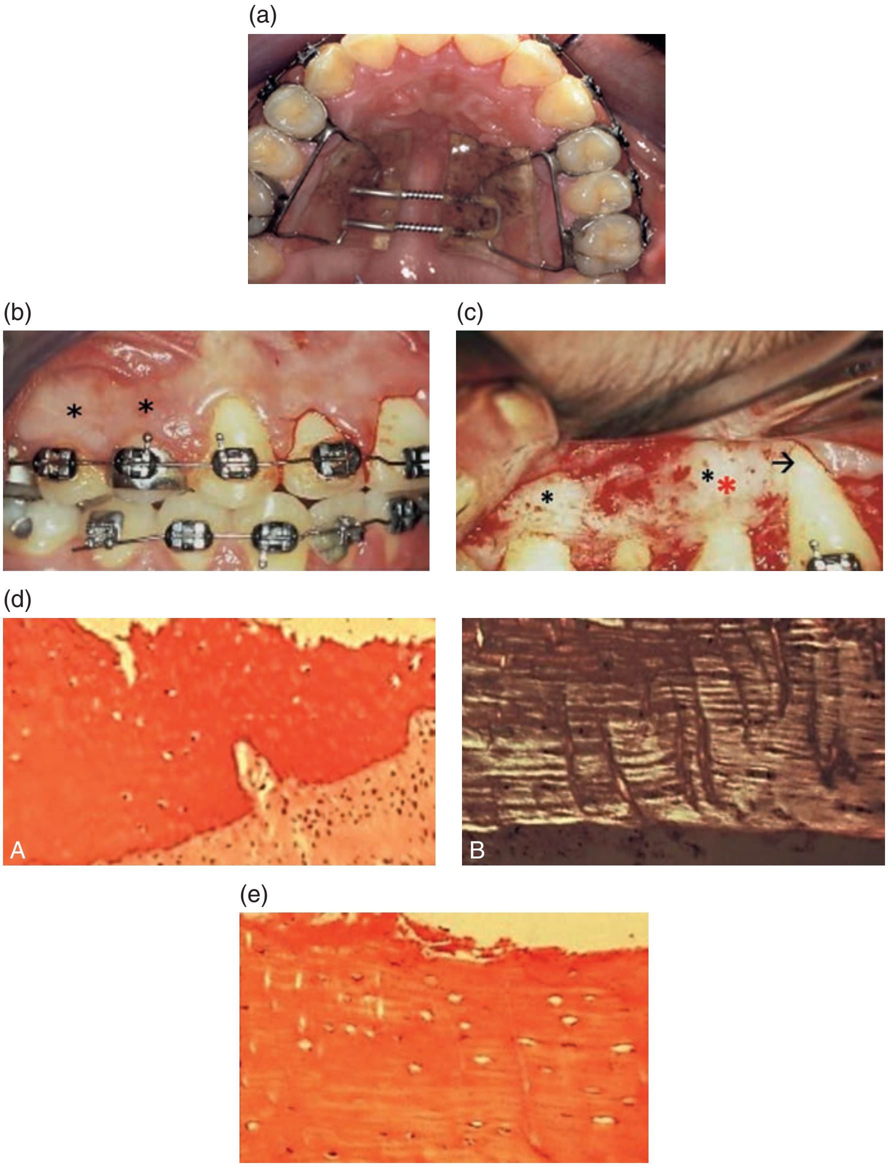

Figure 1.4 (a) The Max-2000® appliance demonstrated here uses two acrylic panels for *nonsurgical alveolus development*. The alveolus expander seeks to enlarge the alveolus bone by placing 150 g of continuous force on the palatal shelves of bone between the molars and first bicuspids. This appliance disregards any expansion of the subjacent maxillary corpus and induces slow dental arch development which is more stable than rapid palatal expansion (RPE) or a more morbid surgically assisted rapid palatal expansion (SARPE). (b) Demonstrates the buccal aspect of the dentition after the alveolus expansion device has placed the maxillary buccal segment congruent to the mandibular dentition. Note the thick "buttressing bone" (at the black asterisks) created by slow alveolus expansion. (c) Demonstrates the reflection of a mucoperiosteal surgical flap after alveolus expansion by the Series 2000®, Max 2000 appliance. Note buttressing bone (at the black asterisks) labial to the expansion region of the palatal acrylic panels. The red asterisk at tooth #5 represents the site of a biopsy sample. Note the buttressing bone (black asterisks) that formed on the molar and bicuspid areas. This labial bone was directly in the force vector sum trajectory of the palatal acrylic panels. Also, note the bony dehiscence on the canine (arrow) which was expanded with an archwire. (d) In (A, left) one sees a routine hematoxylin and eosin-stained histological section of the buccal aspect biopsy of alveolus bone at tooth #5 (Note woven bone) directly buccal to the force trajectory from the palatal acrylic panels of the Max-2000 appliance. Note the *absence* of lamellar pattern which would normally define this biopsy specimen as homeostatic mature bone in a so-called "steady state." The polarized specimen in (B, right) highlights the steady state pattern of bone after *osteogenic modeling*. (e) The image (A) is a control specimen from the patient's nonloaded alveolus that shows no histological evidence of load application or convexity deformation of the bone. The architectural structure in the hematoxylin and eosin-stained specimens clearly shows the lamellar pattern of "steady state" bone and new bone modeling in the patient using a Max 2000 appliance.

subperiosteal bony surface is osteogenic when the alveolus expander panels are subjected to 150 g of lateral load. This nonsurgical phenotype alteration is how a larger alveolus bone is developed in a natural way that accommodates a fully aligned dentition (Figure 1.4a–d).

Histophysiology of Orthodontically Driven Osteogenesis

The "whole bone perspective," discussed above, is a new way of looking at alveolar bone reactions to orthodontic loads and vector sums. The focus on periodontal ligament change is legion in our literature. But to fully understand OTE one must go *beyond the ligament* of the periodontium. The "hyalinization phenomenon" occupies an inordinate place in orthodontic literature. In 2008, Williams and Murphy documented the effects of "bone bending" with unequivocal biopsy images after using a Series 2000® appliance (Max-2000®). It developed a low-force magnitude for slow *alveolus expansion* in a case where healthy tooth extraction would compromise facial esthetics. The effect of the appliance captures the optimal loading threshold necessary for subperiosteal, coupled osteoclasia/osteoblastic activity in the alveolar cortices, as the bone is slightly distorted ("bent") buccally. It is thus demonstrated here that the concept of "moving the root out of a (supposedly) static and immutable state" is misleading. The idea that the entire "alveolar housing" is immutable must be corrected in the orthodontic literature.

So, when patients refuse surgery to correct a posterior crossbite a safer, more stable, and periodontally healthier alternative lies with biomechanics of *alveolus* expansion. When speed is desired, PAOO can do the job. At cell level, it appears that the Max-2000® appliance and PAOO accomplish the same objective. Surgery simply elicits the phenomenon more dramatically and faster. Long-term stability also appears to be the same. This theoretical concept of osteogenesis by shear loading has been alluded to previously in medical orthopedic literature and expanded in Melsen's excellent textbook (Melsen, 2022), where she refers to a "… change in surface curvature of the alveolar walls." All contemporary orthodontists should read this most elegant summary of basic alveolar osteology to fully understand bone *shear strain* in all patients.

This "whole-bone" perspective posits the alveolus bone, *cf.* alveolar "process," as a separate operative organ independent of its contiguous corpus. As the whole bone is orthodontically bent, buccally each osteon and indeed, each cell is deformed. We propose a kind of "peri-orthodontic hypothesis",[4] which contends that this bends protein molecules and DNA, opening obscure binding sites on important developmental molecules. This, in turn, elicits an epigenetic perturbation and redesigns the morphogenesis to a novel homeostatic phenotype unique in alveolus shape, mass, and volume at the molecular level. The value of this new perspective is that it conforms well with contemporary basic biological sciences, particularly molecular biology, and epigenetics (Allis and Caparros, 2015).

In this regard, alveolar subperiosteal tissue and periodontal *ligament* act no differently than the periosteum and endosteum, respectively, in any long bone. Indeed, the father of limb-lengthening surgery, Dr. Gavriil Ilizarov, mimicked the SFOT for the lengthening of legs and restructuring limbs deformed by accident, congenital factors, or poor management of prior fractures. Therefore, a lot of recent medical and basic orthopedic science can be transferred to and from alveolus bone science. This phenomenon, facilitated by corticotomy protocols, may be employed to reduce the degree of clinical relapse that still plagues orthodontics after 100 years of clinical trial and error. Standard orthodontic protocols, merely moving teeth without surgery, cannot overcome the natural tissue phenotype "canalization"

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[4] See Recommended Reading, Allis, Lanza, Bilezikian, Alberts.

that resists change (Figure 1.2). Traditional orthodontic biomechanical schemes create internal tension which rebound like an orthodontic elastic. The rebound is manifest as relapse, necessitating life-long retention.

Cell-Level Orthodontics

Bone cells, as homologs in other tissues as well, sense changes in their mechanical environments, internally throughout the cytoskeleton and externally through focal adhesions to the extracellular matrix (Ingber, 2003, 2008). This area of cell-level biomechanics was essentially beyond the control of most orthodontists who relied instead on gross anatomical and clinical events to intuit cellular activity. With the introduction of tissue engineering concepts and a revival of corticotomy-facilitated orthodontics, a new interest in cell- and tissue-level phenomena has emerged. This is how the alveolus bone lives, thrives, and dies by virtue of its functional matrix, tooth roots (Moss, 1997a–d). This is because of the behavioral imperatives identified by Wolff's law and Frost's "mechanostat" model (Frost, 2003).

What skeletal muscle can do to bone morphogenesis at the gross anatomical level is analogous to effects of microstrain at the cell level. Engineering bone morphogenesis is a threshold phenomenon; too much or too little is dysfunctional. The influence of mechanical stimuli at the cell and tissue level, mechanobiology, is not the domain of bone alone; indeed, the pathoses of atherosclerotic cardiovascular disease are directly related to mechanobiological changes in vessel walls. With modern analytic methodologies and a body of science, too extensive for the scope of this writing,[4] responses to all tissue can now be studied and modulated, be they integumental or neuronal, mucosal, or bony. This is the essence of tissue engineering science. Thus, orthodontic scientists have a legitimate claim to equity in *mechano-biology* as well as *biomechanics*.

Timid Surgery and Its Consequences

Where adequate bone volume is present SAD is the treatment of choice. In the SAD protocol perforations and linear decortication 2–8 mm into the bone are performed with impunity. Although the depth of penetration depends upon the surgeon's discretion, this author recommends deep perforations. Many doctors are happy with a simple flap reflection and a placement of small but effete divots 2–4 mm deep (Figure 1.5). But, this will not give access to medullary stem cells and elicits only a mild osteopenia. Then the doctor erroneously concludes "SFOT does not work", (Figure 1.5).

In Figure 1.5, note the rather shallow, diffusely located, and bloodless (arrows) "divots" used as cortical decortication. This pattern achieves little more than simple flap reflection would create; it renders effete osteopenic (RAP) effects. So, in one respect, a failure to deliver definitive, deep, and hemorrhagic punctate decortications squanders the opportunity to engineer an efficient OTM course. This may be the reason why critics of SFOT encountered experimental failures. *Nothing works well when it is done incorrectly.*

Thus, complete decortication is correctly aggressive after elevation of a mucoperiosteal flap. This area has sufficient bone to decorticate without concomitant bone grafting. In most SFOT, the postoperative discomfort is not

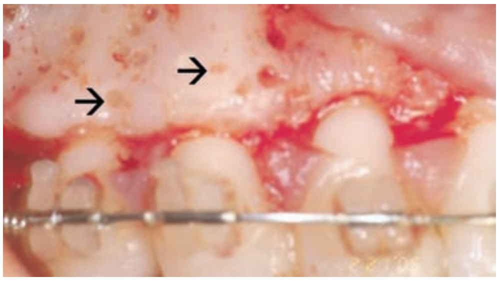

Figure 1.5 Here, shallow, diffusely located, and bloodless (arrows) "divots" achieve little more than simple flap reflection and effete osteopenic (RAP) effect. Compare this timid decortication with the deep and focused clusters of definitive decortications elsewhere in this treatise.

necessarily related to the degree of decortication. When a bone graft is added to a SAD, the appropriate term is PAOO. A previously employed term is accelerated osteogenic orthodontics (AOO[5]), but the two acronyms can be used interchangeably. The cells may be of autogenous origins (Nowzari *et al.*, 2008) or donor stem cells (viable cell allograft) (Murphy *et al.*, 2012). Experience suggests that in most cases, demineralized human bone or xenogeneic graft suffices (Brugnami *et al.*, 2018, 2021a, b). But this author prefers s*tem cell alveolus therapy* (SCAT) for regeneration because of its superior osteogenic potency.

Finally, it is important here to interject a practical guide to discomfort management. Aggressive and timid decortication being equally benign, discomfort comes mainly form protracted time spent in flap reflection, unnecessarily excessive flap reflection, and dehydration of tissue. So, in properly executed SFOT, most discomfort is handled by appropriate doses of nonsteroidal anti-inflammatory pharmaceutical agents with little need for opioid analgesics. Prescribing a single dose of nonsteroidal anti-inflammatory drugs (NSAIDs) *prior to* surgery is a helpful prophylaxis in pain amelioration. Also, remember that pain has both physical and psychological components. So, combining analgesics with a selective serotonin reuptake inhibitor (SSRI), e.g., benzodiazepines PRN is also helpful.

Conceptual Foundation Through Historical Evolution

The SFOT, we describe here, purposely executes OTM through a healing bone wound, or bone graft, eliciting *purposefully delayed wound maturation*. This occurs by perpetuating an "early" bone "callus" or regional osteopenia until all the teeth are ideally aligned, coordinated, and detailed. This kind of surgery is decidedly *not* "rearranging luxated anatomical *parts*" like so many Lego® children's toys. Parts rearrangement is the stuff of orthognathic osteotomies. In stark contrast, OTE does *not* create anatomical fragments or separate "parts." *Loaded corticotomies reengineer physiology*, SFOT, PAOO, and ODO seek to *reengineer* the basic *physiology* of healing but in doing so it seeks a change in the ultimate morphogenesis at the molecular level. This engineers DNA expression and stem cell differentiation.

German Roots

Many historians cite Cunningham's German article of 1894 as the first published article to address SFOT, after his lecture in Chicago the previous year. While having some rudimentary characteristics in common with modern corticotomies, scrutiny of Cunningham's SFOT procedure suggests it has no relation to modern SFOT beyond a crude surgical approach. Cunningham's treatment was really a luxated segmental osteotomy in today's clinical parlance.

His singular goal of making teeth move faster has since evolved to more global objectives and variations on the corticotomy theme. But his prototype has indeed spawned interesting incarnations throughout the 20th and 21st centuries in many different countries and cultures.

Ironically, despite numerous derivative articles describing surgical facilitations of orthodontic treatment in the German literature, it languished there for more than half a century. For example, in 1921 Cohn-Stock, citing "Angle's method," removed the palatal bone near the maxillary teeth to facilitate retrusion of single or multiple teeth. Five years later, Skogsborg (1926) wrote about "permanent fixation of the teeth after orthodontic treatment" by dividing the interdental bone, with a procedure he called "septotomy."

Then, just before World War II, Bichlmayr (1931) described a surgical technique to accelerate tooth movement and reduce relapse.

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[5] Accelerated Osteogenic Orthodontics (AOO) and Periodontally Accelerated OsteogenicOrthodontics (PAOO) are trademarked by Wilckodontics, Inc. Erie, Pennsylvania, USA.

This surgery was performed in the palatal aspect of the anterior sextant by seemingly ablating large areas of cortex and medullar bone (spongiosa) (Figure 1.6b). This ablative technique was apparently employed also with canine retraction after bicuspid extraction, by excising the buccal and lingual cortical plates at the extraction site. Although this procedure seemed bold to American orthodontists, it became popular in the German scientific community. Later, Ascher (1947) published a similar procedure, claiming that it reduced treatment duration by 20%–25%. Analyses of these procedures were combined with a critique of the Bichlmayr's procedure by Neuman in 1955.

The Introduction Into English

The seminal American work, published in English by Köle, built upon the German predecessors and summarized a series of surgical techniques to facilitate orthodontic therapy. One was a decortication of the dentoalveolar process to facilitate OTM. He compared his interdental decortication with the more morbid procedure of Bichlmayr claiming that only the cortex should be removed.

He specifically mentioned the advantage of singular cortex removal leaving the spongiosa intact to serve "… as a nutritive pedicle to the bone denuded of its mucoperiosteum." With some notable refinements, this is the basic technique that is used today without a subapical osteotomy. The Kole surgery (Figure 1.6a) was limited to the cortex of the dental alveolus, but subapical decortication was embellished by extending buccal and lingual cortical plate incisions until they communicated through the subapical spongiosa. Buccolingual communication is now considered unnecessarily morbid and eschewed by evidence-based SAD and PAOO protocols.

The rationale for the decortication as Kole explained was, "… the main resistance to movement is encountered in the cortical layer," thus, ignoring the change in physiology (RAP) that Frost has so vividly discussed. The literature in the early half of the 20th century seems to miss the central point, *viz*. SFOT is *not* intended to eliminate a mechanical impediment as Kole implies. It is somewhat illogical that the cortex should be the main impediment to efficient OTM because the teeth move through medullary bone, not cortical bone. SFOT is a *physiology modulation*. Figure 1.8 demonstrates this

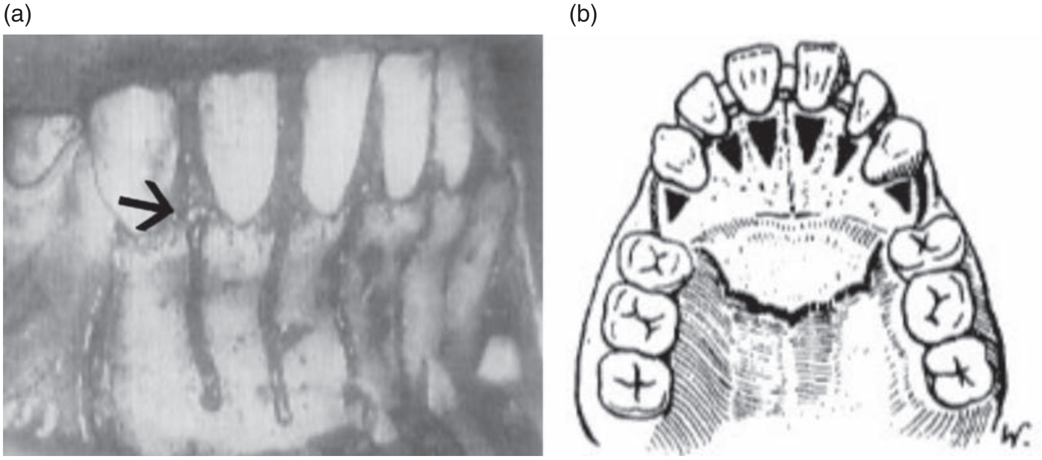

Figure 1.6 Kole contrasted his technique with more morbid prior attempts to accelerate orthodontic treatment. This publication marks a didactic milestone because Kole focuses on alveolus bone "physiology re-engineering", the conceptual basis of OTE, not mere mechanical bone ablation. *Source:* Kole (1959)/with permission from Elsevier.

point dramatizing the stark difference between normally calcified bone and osteopenic bone.

Early bias and the lack of cell-level perspective are reasons why the literature of this period is merely anecdotal, dismissive, and often incorrect. Ironically, this body of data is still used to justify specious criticisms of 21st century SFOT. In Figure 1.6, we can see how Kole contrasted his technique (Figure 1.6a), with that of Bichlmayr's technique (Figure 1.6b). The Bischlmayr technique seems to ablate an entire segment of palatal bone prior to incisor retraction. Kole's method however, is similar to the modern technique employed by contemporary periodontist–orthodontist teams. Note, however, that Kole carried the linear decortications to the crest of the alveolus bone (arrow). Most modern surgeons categorically disdain this extension.

But it should be noted that integrating SAD with OTM will not grow new bone mass. In fact, in a steady-state alveolus, it may reduce alveolar bone mass (Figure 1.23 (1b) and (1c). This is what "moving bone out of the alveolar housing" means. So, applying Cunningham's derivatives indiscriminately can indeed result in a *net loss* of supporting bone. This dilemma of risking bone loss for the sake of faster OTM was solved by the prodigious efforts of many doctors in the Wilckodontics® research group, representing the academic aegis of Case Western Reserve University. But the inspiration for all the American innovators belongs to a Japanese doctor.

Suya and the Asian Connection

Suya (1991), a Japanese clinician, stimulated significant academic interest in the United States and rejuvenated the Asian orthodontic community for nearly two decades. For example, his paper reintroduced refinements of Kole's work into the American mind with a report on "corticotomy-facilitated orthodontics." Suya's contention was well received because it demonstrated that conservative intervention could yield dramatic results. He also substantiated that opinion by reporting his experiences with over 300 patients. Importantly, Suya did not connect the buccal and labial incisions like Kole (1959) but relied purely on linear interproximal decortication. Although Suya, unfortunately, contended that the facilitated tooth movement was the result of "bony block" movement, his contribution to the world literature evoked many questions about the exact pattern of cortical cuts. We now know that the style of decortication, divots, lines, or other patterns is irrelevant. Only the sum of therapeutic perturbations is significant. For some patients, minor divots will suffice. Others might require a nearly complete cortex decortication. But biodiversity rules all. So, experience and surgeon discretion are important drivers of success. In any case, the decortication must elicit bleeding from the spongiosa.

Suya's refinement of Kole's methods has essentially set the standard for decortication procedures that followed in the postmodern era. Korean literature included a discussion by Kim (S-C), as early as 2003, and a thorough discussion of the corticotomy phenomenon in that year, which stimulated further inquiry in the Korean peninsula very soon. Practical application of the protocols was published in 2009 (Kim *et al.*, 2009a, b). The fact that Suya's method requires a surgical flap reflection was recognized as an intimidating requirement for some clinical and patient cohorts. So, knowing how fast mucosal wounds heal encouraged other investigators to develop more conservative protocols. Kim (SJ) followed with a published transmucosal procedure (TMP) called *corticision* the same year (Figure 1.7). Corticision is a so-called "flapless decortication".

1980s: Anholm and the Loma Linda Investigation

Following communications with prior visionaries of the 1980s, periodontists and

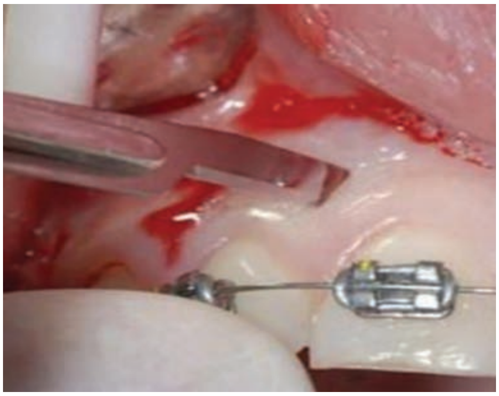

Figure 1.7 Corticision is the use of transmucosal incisions into the bony alveolus cortex. It relies upon palpation or tomography to ensure adequate facial alveolar bone is present. The salutary effects of this procedure are less morbidly effected if TMP is employed.

orthodontists collaborated in the first major university studies of the phenomenon at California's Loma Linda University (LLU). A series of excellent research papers were created at that school, but support for the SFOT was tempered. Anholm *et al.* (1986), sobered by minor attachment loss in a male patient, were cautious in their praise of SFOT. Some limited crestal attachment loss can certainly occur if the periodontal (mucoperiosteal) flap is reflected off the bone for a protracted period; it gets dehydrated, and papillae may temporarily shrink (Figure 1.20). So, effective decortication surgery should be *sterile, sure, and swift*.

Gantes (1990), also at LLU, was a little more supportive of SFOT's merits when he reported decortication on buccal and lingual surfaces on five orthodontic patients, with immediate orthodontic activation. His interproximal "vertical grooves" extended apically "beyond the apex levels of the teeth" with horizontal connections at the apical end. It is surprising that at a university follow-up was possible since patients are often handed off to new orthodontists as classes graduate. Nonetheless, it is to the school's credit that the Gantes data demonstrate a clinically significant 50% reduction in treatment duration.

After the completion of the orthodontic treatment in five patients, the outcomes according to Gantes, "... showed that the corticotomy procedure caused minimal change in the periodontal apparatus" offsetting Dr. Anholm's fears. The parameters for periodontal health assessment were standard well-accepted indices: plaque scores, probing depths, and probing attachment levels. So, it is ironic that Gantes advised that a corticotomy should be avoided on patients having "any form of periodontal pathology or deformity." Under this rubric of "untouchables," he included gingival recession, buccal or lingual bony dehiscence, and teeth with reduced periodontium. We disagree with his proscriptions. We have successfully treated every one of these periodontal problems with SAD or PAOO/SCAT. Patients who have reduced (compromised) periodontal support are no exception. As the case presented in this chapter demonstrates, reduced periodontium and active periodontal infection need not be contraindications for SFOT, as long as the proper technique is employed.

Even if no bone is used, and SAD is the only surgery employed, there is no reason to believe that dehiscence would worsen by virtue of the surgery. Indeed, recession is commonly treated *with periodontal surgery*. Regarding gingival recession, if the surgical flap is replaced coronally over receded gingival margins, and the root surface is properly prepared, a new attachment or a stable long-epithelial attachment can be achieved as reasonable clinical outcomes. And, the outcomes are stable if maintenance achieves low bleeding indices. Orthodontic non-extraction may exacerbate a dehiscence, but surgery would not necessarily affect this unfortunate phenotype. It is unclear why Gantes included this proviso because no rationale was given. The categorical and vague nature of this admonition discredits it in the context of 21st-century periodontal science.

A 20th Century Summary

From Bichlmayr in Germany to Köle in the English literature, to the turn of the new century, it becomes obvious that thinning of the alveolar volume in the direction of the tooth movement is a critically important consideration in the protocols of any orthodontic-driven corticotomy procedure. When one applies this nuanced philosophy to the surgical preparations, it becomes much easier to keep the entire OTM treatment times under 12 months except in cases of severe Class III or Class II skeletal dysplasia.

Yet, even severe skeletal dysplasia can be treated fast and better if SFOT is coordinated with orthognathic surgery. This is when SFOT accelerates the universal first step in OTM, leveling and aligning. Orthognathic surgery can achieve the second phase, arch coordination (congruency) within hours. The third phase of OTM, "detailing" is perhaps the most difficult part of OTM. But SFOT makes this phase easier also. Moreover, the immediate post-orthognathic surgical condition is notorious for displaying excellent physiologic tooth mobility. Rather than waiting to attempt detailing until the surgery "heals" (read: recalcifies), it is better to intervene early in the postoperative course to take advantage of the generalized iatrogenic mobility. This author begins OTM during the surgical procedure after the last suture is placed. This has been achieved with clinical impunity for over a decade.

Most treatment times range from 3 to 9 months with Class I and mild Class II malocclusions. Twelve months in severe Class II cases and less than 3 months in Class I moderate crowded cases are reasonable OTM goals. Independent clinicians often claim that severe skeletal posterior cross bites and anterior open bites are universally daunting challenges, but they may also be treated successfully in about 10–12 months despite the fact they represent a basal bone skeletal dysplasia. Where clinicians face maxillary transverse deficiency, it is doubtful that PAOO has any effect on the basal bone. But it can be employed in Class II cases if decortication and grafting are performed on this basal component. Wilcko has demonstrated this principle in his famous case of Patient X (Figure 1.8) whom he treated with serial PAOO at pogonion and gnathion. However, for transverse deficiencies, it should be noted that expansion of the palatal suture and maxilla *per se* is

Figure 1.8 The case of patient X demonstrates that sequential PAOO protocols can be used as an alternative to genioplasty or mandibular advancement surgery if the mandibular corpus is grafted at B-point and pogonion. Images courtesy of Dr. M. Thomas Wilcko Erie, Pennsylvania USA.

irrelevant if the alveolus bone itself is sufficiently enlarged. This is what PAOO is designed to achieve.

As the century closed, the specialty of dentoalveolar surgical orthodontics was reaching the end of clinical art and the beginning of a protracted journey through the gauntlet of scientific scrutiny. SFOT specialists remained dutifully skeptical but undaunted because, with the consistently gratifying clinical results and unimpeachable anecdotal consistency around a shrinking world, their collective confidence continued to grow.

21st Century Pioneers

The Entry of Academics

By the year 2007, intellectual collaboration among clinical researchers and other universities around the world inspired by scholastic leadership at Case Western Reserve University resulted in significant documentation of the SAD and PAOO efficacy. Clinical researchers resolved for the last time much of the contention among the earlier clinicians by subjecting SAD to detailed analysis and rigorous standards of evidence-based science. This is important because it positioned corticotomy procedures at the exalted level of university-based analysis and the kind of controlled experimentation that it demands. At the time, these early 21st-century studies of SFOT were unparalleled; in retrospect, their findings seem epochal.

“Wilckodontics,” PAOO^{TM}/AOO^{TM}, and Tissue Engineering

After studying the publication of Suya (1991), Wilcko group led us intrepidly into the 21st century. Dr. M. Thomas Wilcko, a periodontist, collaborated with his brother William, an orthodontist, in Erie, Pennsylvania, United States, to develop a more stable and benign SFOT. Concerned with the destructive periodontal effects of banded therapy and encouraged by the results of SAD, the brothers surmised that alveolus inadequacy might play a factor in relapse. So, in 1996, they conducted case studies to a very high standard of excellence. Finally, they added an allogeneic bone graft between the decorticated cortices and replaced flaps. The Wilckos also popularized the notion that a corticotomy induces a regional osteopenia (Figure 1.9), the so-called RAP.

The Wilckos’ computerized tomography of the results also documented a definitive alteration of alveolus morphology, Wilcko *et al.* published their seminal academic works within the next 7 years, referring to the grafted protocols as AOO^{TM}.[7] Later, it was renamed the PAOO^{TM} technique. The difference between the two is merely semantic, so they are essentially synonyms.

Sadly, many dental schools initially shunned these innovative efforts. Even worse, some prejudiced professional associations even expressed hostility to the Wilcko efforts. This is, however, the personal cost which innovative leaders predictably must pay for novel data which appears to be iconoclastic. New data can threaten some intransigent vested interests simply by being new. But, undaunted by institutional cynicism, they began teaching their innovation in a proprietary school called “Wilckodontics.” They trademarked the name of the school for the protection of the unique protocol lest it be adulterated to the detriment of patients’ welfare. Among cynics, the school’s name and trademark were mistakenly misinterpreted as a legal gambit to monopolize the surgical technique. But nothing could be further from the truth.

The legal trademark was intended to protect patients and the integrity of the school’s name. Ironically, the trend of “patenting science” is now becoming increasingly popular, but this is still not the case with the Wilcko philosophy. The trend toward healthcare corporatization is inevitable and was heralded by Paul Starr’s prescient work, *The Transformation of*

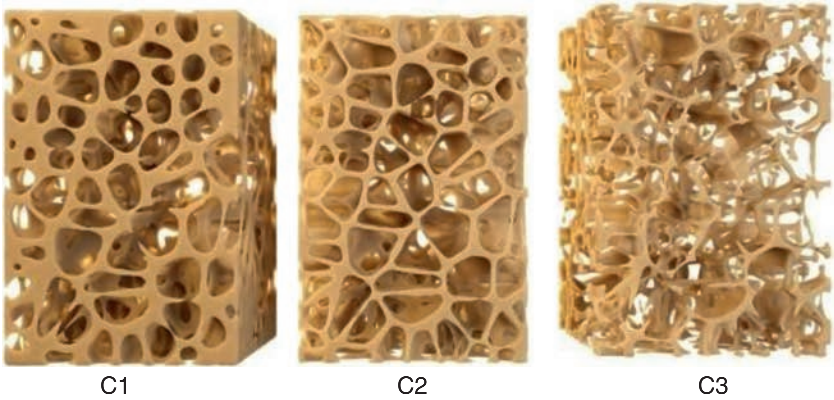

Figure 1.9 Sections of bone in various stages of porosity, C1 normal, C2 osteopenic, C3 osteoporotic. In this schematic reconstruction, the dense architectural pattern in normal bone in C1 compares to less density of simulated osteopenia bone in C2 and C3 where selective decortication, TMP, and other OTE surgical modalities capitalize on a reversible osteopenic state to accelerate tooth movement and ensure greater clinical outcome stability. This improved stability is due to the epigenetic change in phenotype due to OTE. The osteopenic state of RAP differs from the disease state of osteoporosis in that the therapeutic osteopenic state is predictably physiologic, intentional, quantizable, aseptic, therapeutic, and fully reversible. The osteopenic state is the keystone to all OTE procedures. Essentially, the osteopenia in RAP is not a pathological state because it is reversible. *Source:* https://www.drnesterenko.com/2019/04/26/osteoporosis-and-spine-health/.

American Medicine, two decades earlier. The 1982 sociological treatise is still worth reading for those interested in the future of healthcare delivery patterns. But the Wilcko mission was scholastic, not commercial. Interestingly, as with Mary Shelly's gothic novel, *Frankenstein*, the school's name was transmogrified by some readers and misappropriated for the creation itself. So, the term "Wilckodontics" in contemporary vernacular is a general reference to any and all SFOT iterations.

Since the 1990s, the Wilcko collaborators have remained true to their noble mission, relying on empirical proof of their brainchild, collegially sharing data through their school, wisely eschewing crass commercialization, and validating the worth of their protocol by independent corroboration internationally. The combination of grafting, decortication, and accelerated adjustment schedules was an ingenious addition to the orthodontic profession because it enriched the studies at the Loma Linda School of Dentistry with a rationale and depth of scholarship others seemed to misunderstand.

The Wilcko group protocols went even further by developing protocols for space closure in extraction cases, as Kole did before them. But the Wilcko wisely added more. They combined buccal and lingual grafted corticotomies with total extraction site ablation. This unnamed procedure leaves only the mesial and distal cribriform plates of adjacent teeth during extraction site closure. As the adjacent teeth move toward one another, a new alveolus naturally forms around them, aided by the graft of demineralized freeze-dried bone allograft (DFDBA).

At the dawn of the 21st century, further refinements of PAOO and other variations of SFOT were investigated under the aegis of Case Western Reserve University and the profoundly rigorous analyses of Professor DJ Ferguson. This data spread thereafter throughout the world. Ironically, some of the most disciplined studies came out of the so-called Third World.[6] The introduction of PAOO to the

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[6] Wilcko MT, personal communication, 2023.

SFOT collage marked a particular quantum leap into 21st-century tissue engineering. Remember, the word "engineering" means the art of manipulating natural forces to a predetermined design. The tantalizing dynamics and biological phenomena of this clinical approach evoke the biomathematics of nonlinear complexity and fractal geometry. These progressions and patterns expose the enigmatic patterns of mammalian morphogenesis and geometric destiny of individual cells. These topics are too profoundly esoteric for the scope of this practical discussion but warrant further study.[7]

As with many great innovations in science, the difference between profundity or meaningless novelty, innovation, or apostasy often depends on the consistency of observation, psychosocial acceptance, degree of institutional inertia, political advocacy, and even the serendipity of fickle fortune. All these determinants are presently alive and beneficent for the young visionary orthodontist.

According to the strict Wilcko protocol, 1998–2020, tooth movement was typically initiated sometime during the week preceding the surgery. So, the surgery at that time was performed in strained bone *ab initio*. Thereafter, the strain was perpetuated by activation of the orthodontic fixed appliance every 2 weeks. Since the time PAOO was introduced, timing of the adjustment periods has been shortened by this author to semiweekly or even daily adjustments. SFOT is so effective that simple space closure can be achieved with finger pressure in less than a minute.[8] The creation of bone *de novo* was a revelation to many periodontists. Prior to this observation, it was considered axiomatic that one "… cannot grow bone on a flat surface." Yet, PAOO did just that. The secret is coincident and *nonresonant wound loading*.

The first publication by Wilcko *et al.* was preceded by 8 years of research and clinical investigation that reduced morbidity and, in some cases, even obviated the need for orthognathic surgery. What began as performing corticotomy surgeries without bone grafting on both younger adolescents and adults flowered into a major subspecialty of dentoalveolar phenotype morphogenesis. The Wilcko group made it obvious to everyone that accelerated care reduces bacterial load, enhances stability, reduces the pain of mechanical adjustments, precludes damage to the attached gingiva, and can even increase the zone of attached gingiva. All these historical banes of traditional fixed appliance care are reduced with PAOO and conform to the definition of "best" discussed earlier in this chapter.

Prior to this research, corticotomy-based surgeries were only recommended for individuals 18 years of age or older following the cessation of growth. This age limit now appears to be antiquated. Indeed, due to the tremendous rate of adolescent development, leveling, and aligning can be achieved in a matter of weeks, not months in adolescents younger than 18. High-resolution surface scans of computerized tomography clearly showed a demineralization–remineralization process at work and stable, enlarged alveoli. The explanation Dr. Wilcko first suggested was "bone matrix transportation" rather than a bony block movement.

Building on that astute observation, we introduced a modified term combining specific technical terms derived from orthopedic, periodontal, and orthodontic literature. Our term is *periodontal matrix translation* (*PMT*). This term is entirely consistent with Frost's RAP and Wilckos' thesis. But PMT is more accurate. The terminology refinement proposes that only the decalcified cribriform bone, periodontal ligament, and root are the translating entities. The two terms are essentially synonymous, but the one we proffer is more traditionally consistent. Thus, in summary, the PAOO is

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[7] See Recommended Reading, Liebovitch, Kaplan.

[8] Murphy NC, YouTube: "15 Second Orthodontics": https://youtu.be/i265QQfXaKU).

purely a natural osseous reaction not an idiosyncratic phenomenon. PAOO requires no modification of fixed appliances. It is also indifferent to any style of biomechanical adjustments (Begg, Tweed, etc.), as long as the internal osseous strain is constant.

The Small Window Myth

Critics who have not taken formal instruction in SFOT or are inexperienced often claim that the SFOT procedures have a limited window of opportunity. They reason that, since an osteopenic state, (purportedly but fallaciously) lasts for such a limited time, it cannot facilitate OTM (Buschang *et al.*, 2012). Even though they acknowledge acceleration is possible, e.g., a doubling of movement rate, they fall victim to the myth of short duration. The truth is: *there is no window*.

Quoting Buschang, "the amount of time during which corticotomy have an effect on tooth movements is limited to 1–2 months." This is true *only* if the OTE protocols are executed erroneously, or therapy is interrupted for a protracted period. However, when instructed well, the orthodontists' biomechanical protocol can load the bone in a way that elicits a *constant strain* in the healing alveolus. So, there is really no such thing as a small 1–2 month "window of opportunity." The small window concept is a myth. The osteopenic state (RAP) can be maintained indefinitely allowing recalcification to occur in the retention phase of treatment. This is perfectly analogous to a fracture nonunion that recalcifies once total immobilization is secured. In case the treated alveolus recalcification occurs too soon, an osteopenic state will return within a couple of weeks if strain is reinstated and/or transmucosal perforations are employed. The clinical truth is this: *RAP (transient osteopenia) can be sustained indefinitely*.

Even when RAP is evinced, sometimes, bodily translation of a tooth through the long axis of the alveolus may be delayed to a millimeter per month when no significant entry into interproximal bone is made. This is the error of timid surgery. But sometimes even with appropriate intramedullary perforation is achieved a complete treatment failure can occur; such is the nature of biological systems. It is a wise man who acknowledges that every island of knowledge which science discovers is surrounded by a shore of ignorance. These are idiopathic occurrences. They are idiosyncratic and rare.

Despite the knowledge that sometimes things simply do not work in idiosyncratic patients, so-called "failure" must be minimized by assiduous scholarship and confident intervention. For example, a timid punctate decortication (Figure 1.5), in the opinion of this author, should be abandoned in favor of deep linear decortication 4–8 mm deep and in clusters no more than 4 mm apart. The millimeter depth is only a general guideline that must be individualized for each patient and each location within that patient. The goal is *intramedullary* penetration *through* the cortex, to elicit hemorrhage, not merely *within* a cortex. This will ensure medullary osteopenia which is much more important than its cortical counterpart.

Therefore, if a thin cortex perforation elicits bleeding with a 2 mm decortication, then it is the correct procedure for that *individual* patient. Whereas other patients may require a 4–5 mm perforation. And, if more mobility is needed, the penetrations should be doubled in each example. Decortication does not always mean *partial* decortication; it *can also refer to a complete decortication in situ*. In cases of very dense bone, a *total resection of the cortices* is necessary. PAOO and non-grafted SFOT principles, however manifest in the hands of an experienced surgeon, complete many cases as fast as 4 mm per month. This will *not* be achieved with a reticent surgeon.

The "Need" for Extraction Myth

Bicuspid extraction therapy is often presented as "needed" because the alveolus bone supposedly cannot be moved facially to accommodate arch expansion. It is also claimed that "the teeth should not be moved beyond the limits of the alveolar housing" (presuming the alveolus is immutable). Second, the extraction choice is often justified because the doctor wishes to reduce morbidity. That is, he or she claims a desire to reduce the risk of "periodontal damage, *e.g.,* gingival dehiscence." Third, the doctor decides *unilaterally* that the patient's profile would be better served if the teeth were not protrusive. This is the kind of specious reasoning that leads to perfunctory extractions which on closer inspection are neither indicated by contemporary science nor desired by hapless patients. Too often biomechanical extraction protocols are little more than short-order recipes for convenient and predictable tooth alignment without consideration of the patients' subjective opinion of their own facial appearance. Improper extraction faces get even worse through the ravages of time (Figure 1.13).

We propose that none of these excuses are justified. First, the erroneous notion of alveolus immutability of the alveolus has been disproven by years of "black swan" arguments. Professor Ferguson's intrepid work is particularly impressive in this regard because of his unabashed scholastic excellence. He has the uncanny ability to breach obscured scientific truths and present them in pedagogically facile ways. His research culminated in a new definition of the tooth movement envelope. The *Ferguson Limits of Movement* (Figure 1.14) clearly defines the new limits to which the alveolus bone can be expanded. Second, even if periodontal dehiscence occurs, (unlikely when infection is managed) a gingival graft to restore the zone of attached gingival is far less morbid than extracting two to four healthy teeth. Third, the definition of facial beauty ultimately is a *patient decision*, and with changing culture, more protrusive, so-called "full smiles" are in vogue.

Where the projection of future facial form is in doubt or where the patient is ambivalent, a resort to Pascal's Wager maximizes future options. If the non-extraction course is followed only to discover that it produces too much lower facial protrusion, patients still have the option to change their minds and retract anterior sextants. The perfunctory and injudicious extraction of bicuspids to suit the doctor's singular artistic vision is a misappropriation of patents' prerogatives. In cases where SFOT can enlarge the alveolus, ignoring patients' opinions, for the sake of expediency or a doctor's artistic sentiments is simply too facile and solipsistic to serve seriously deliberative and rational decision theories.

Some may claim that only the doctor is uniquely trained in the definition of good esthetics. Often what they believe are benignly authoritarian decisions on esthetic matters is couched in expressions like "… work well for most patients" or "… art that works well in my hands." However, when that arbitrary decision involves injudicious tooth extraction to match a deformed bone, the retreat into the orthodontist's singularly subjective sense of beauty rings hollow as a banal rationalization.

On the contrary, we claim that routine extraction therapy "recipes," with their unpredictable potential to flatten facial profiles in maturity, evoke serious questions about what constitutes universal definitions of desirable facial esthetics. The generation of PAOO-augmented profiles with serial augmentation protocols call for guidelines about how far one should go with augmentation therapy. But a fear of excessive protrusion is an idle fear. A critical discussion of traditional orthodontic standards is in order here.

On Beauty

Existing concepts of optimal esthetics are available to all orthodontists and are integral to their training. But as cultural values change, some clinicians appear to be relying on outdated concepts of facial beauty, starting with Angle's veneration of the Apollo Belvedere profile. It is noteworthy to remember that this

Victorian standard also coincided with the eugenics movement and social Darwinism where prognathic lower facial profiles were considered simian and "primitive." It is the extraction protocols that tend to produce this archetype for better or worse. Now, relative bimaxillary protrusions reflect an emerging social standard of high esthetic value. The Apollo profile is beginning to fade as a desirable standard. We propose that the Victorian profile is too flattened, *viz*. "dished-in," to meet contemporary standards of social esthetics, as judged by a naso-pogonion "E-line" (Ricketts, 1968) (Figures 1.10 and 1.11).

Marquardt, a facial reconstruction surgeon, heartily endorses the use of his ideas in orthodontic therapy. His Golden Decagon can serve as a geometric template or intellectual metaphor (Jefferson, 2004; Bashour, 2006) (Figure 1.11). Mathematical imperatives seem to appear more prominently in popular facial profiles. These have been studied for decades by Dr. Steven Marquardt with a profound justification. It seems that universal geometric pattern, expressed as a ratio called *Phi,* is a ubiquitous phenomenon in nature and even in purely inanimate physical systems. As part of what Plato called the "World of Forms" and Jungian Archetype, it is thought that *Phi* can predict the social acceptance of some faces over others.

Calling upon an eclectic literature ranging from physics to biology, to metaphysics, Marquardt has developed an esthetic matrix that can be overlaid on photographs to quantitate how much deviation a facial form differs from his geometrical archetype (www.beautyanalysis.com). These kinds of precise quantitations are superior to subjective evaluations, outdated notions, or popular esthetic nostrums. They give both plastic surgeons and orthodontists a very useful instrument to confirm professional assessment (Figure 1.12).

Figures 1.11, 1.12, and 1.13 harshly illustrate just how extraction treatments – often

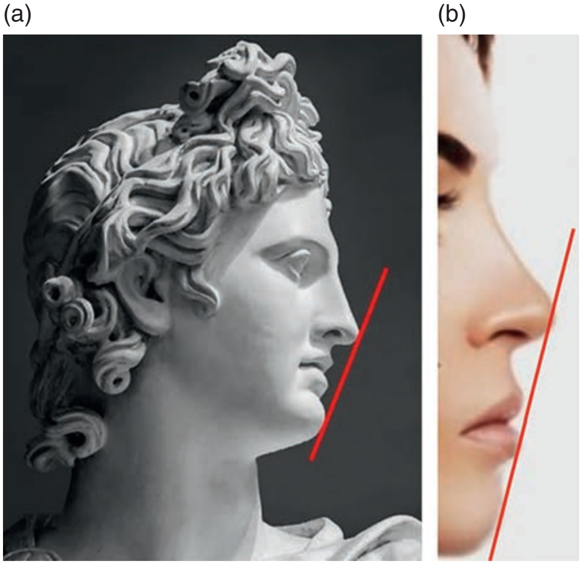

Figure 1.10 (a) The classic Victorian profile of Apollo Belvedere, unfortunately favored by many 20th-century orthodontists, seems antiquated as an esthetic reference in contemporary social standards. Many 21st-century orthodontists see healthy tooth extractions as tantamount to amputations. (b) The more modern esthetic profile that comports with contemporary standards of the facial esthetic ideal archetype. Where so-called arch length deficiencies argue for healthy tooth extractions but are contraindicated due to prominent nose and chin profiles, OTE provides a predictive alternative to healthy tooth extractions. This safe alveolus bone expansion ensures less premature ageing ("dished-in profiles") of the lower human face and a prettier clinical outcome from patients' perspective. *Source:* Used with permission from Wayfair LLC.

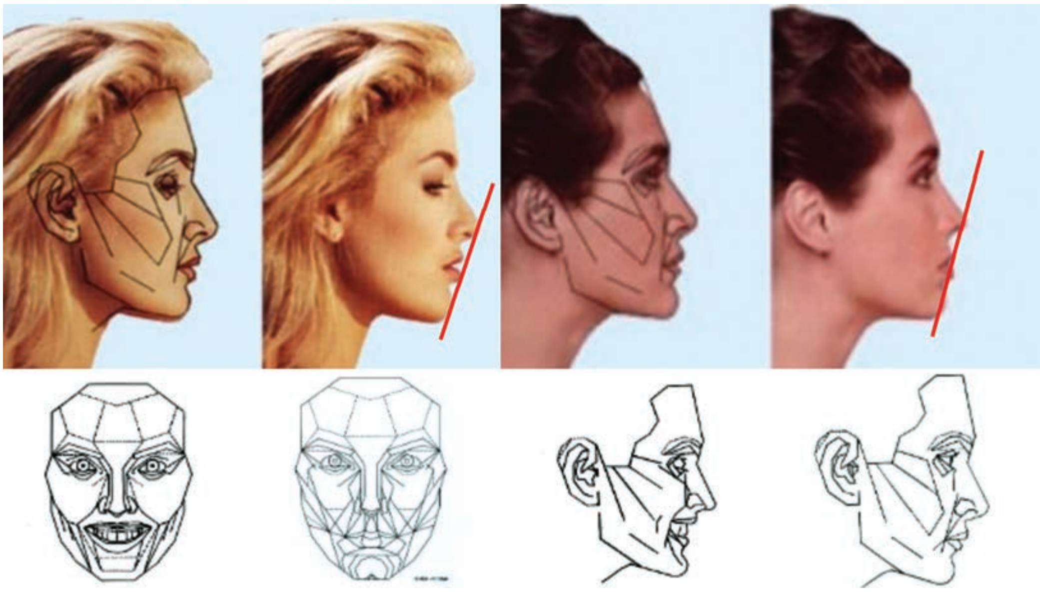

Figure 1.11 Beauty may be somewhat "in the eye of the beholder," but it also lies within predictable geometrical patterns. These patterns can be quantitated and revealed with an overlay template, the "Marquardt Mask," a two-dimensional geometric form very compatible with the E-line reference. The template consists of integrated decagons that we propose are nothing more than natural "emergent patterns" in a bio-morphogenic nonlinear complex progression. These patterns are common in natural phenomena and derive from natural morphogenetic development to create a "steady state" of tissue homeostasis. In this regard, orthodontic non-extraction therapy can serve to liberate natural forms from epigenetic developmental impediments. *Source:* Image courtesy of Dr. Stephen R. Marquardt. www.bautyanalysis.com. Used with permission.

unnecessary in the age of PAOO/SCAT – can unpredictably devolve into unattractive lower faces in adulthood. Even when a midline discrepancy is obvious, a generous display of teeth presents a more esthetic appearance than iatrogenically flattened profiles, as demonstrated in Figure 1.12 (Figure 1.13).

A prominent nose and chin combined with arch length deficiencies has always presented a conundrum for previous generations of orthodontists with no good alternatives: Not extracting teeth would allow a malocclusion to prevail. Even extracting healthy posterior teeth and allowing facial profile to remain static can be very unpredictable and biomechanically challenging. When adult teeth are extracted in an adolescent, postpubertal predictions of facial form can be very unreliable. Finally, deferring treatment to maturity is unacceptable to older adolescents and adults. So, extraction became *de rigeuer* and injudicious extraction became more common from the earliest days of orthodontics. It was thought that extractions rendered a more stable outcome. But Little *et al.* (1988) found that the 80%–90% relapse rate was equally evident regardless of extraction or non-extraction treatment. Both plans are equally unstable because most orthodontists are not yet aware of the fact that *traditional OTM does not change alveolar phenotype*; it merely strains it. And *what is strained relapses.*

However, the success of PAOO vitiates the previous imperative to extract healthy teeth simply to align the dentition or render less relapse. Now that PAOO allows expansion of the alveolus bone with impunity, alveolar bone augmentation is emerging as the professional standard of care to build a better bone.

The unesthetic outcome gets worse with age. Figure 1.13 illustrates that injudicious extraction of healthy teeth is tantamount to amputation of a facial growth site. The predominant

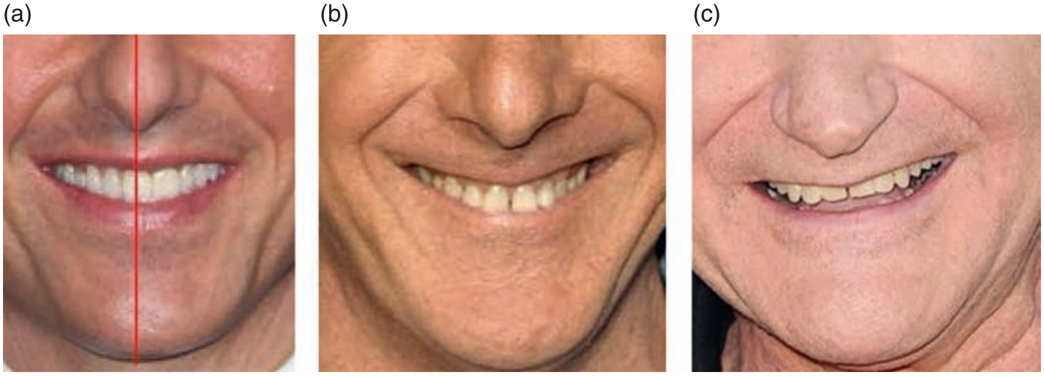

Figure 1.12 Even when a midline discrepancy (a) is obvious, a generous display of teeth presents a more esthetic appearance than the flattened profiles (b, c). In the initial diagnostic analysis, the presentation of a prominent nose and chin should alert the thoughtful orthodontist that PAOO is a preferred option. The more prominent the smile, the more esthetic is the clinical outcome according to patients' preference.

Figure 1.13 The retrusive lower face due to injudicious dental extraction becomes more pronounced with age. Hence, the nondescript esthetic deformity, mildly evident during treatment, is more starkly disfiguring after traditional orthodontic extraction therapy has ended. The regression of the low facial profile in reference to Ricketts' E-line, as a function of age is evident in this longitudinal case document. The age of the patient from left to right – (a–c) – is 12, 14, and 30, respectively. If a severe ALD is present the orthodontist may have no choice but to extract bicuspid teeth, but that is not a categorical imperative. With PAOO this dilemma is no longer a vexing problem. Simple out-patient OTE now allows treatment of a so-called "arch length deficiency" (ALD) without extraction. Image courtesy of Karin Badt, 2021/from Medium.com.

idea of years past is that a recessive low face was less primitive looking than a prominent dentition seen in primates. This patient in Figure 1.13 approximates that of Apollo Belvedere, the Victorian archetype, and eugenics ideal. As she ages her lower facial profile becomes relatively retruded. Such profiles are even today cited for their "strong chin" a reference to pseudoscience physiognomy, the philosophy that character can be assessed by the shape of bones. Such a notion finds comfort in social prejudice and misplaced art; anathemas to science.

Professor Ricketts set an enviable standard in 1968 that helped define optimal esthetics and is still widely employed standard of facial value. He stated that the lips in the Caucasian are *"... contained within a line from the nose to the chin, the lower lip is closer to the line than the upper, the lips are smooth in contour, and the mouth is closed with no strain"* (The so-called "E-line," "E" for esthetics, as displayed in Figures 1.10b and 1.11).

In retrospect, the E-line and the Marquardt mask have worked well in establishing attractive facial profiles for generations of orthodontists and have such an enduring quality that they should not be summarily dismissed. The closer we can come to predictable beauty the stronger the profession becomes. And yet, conformity does not necessarily produce the most beautiful facial esthetics. Average faces are attractive, and yet, exceptional beauty is not average (Alley and Cunningham, 1991). So individualization is still a strong clinical imperative. We must ask patients what *they* call "attractive." The correct measure of appropriate case development, the *sine qua non,* is *concordance*, a "meeting of minds," between and doctor and the patient. This can only be forged with fully informed consent, meaningful dialogue, and an adroit use of Pascal's wage.

From PAOO to SCAT; Dead Cells to Living Tissue

The original component of PAOO that made a significant difference in alveolus augmentation was the addition of Demineralized Freeze-Dried Bone Allograft (DFDBA), a common component in infrabony defect regeneration. Mixed with clindamycin, the trademarked protocol of Wilckodontics® proved to be successful in "growing bone on a flat surface" heretofore thought to be impossible. The reason the Wilcko group was so successful was that their graft was placed on decorticated bone and – most importantly – *it was loaded*. The loading stimulated endogenous mesenchymal stem cells (MSCs) (pericytes) to differentiate into osteoblasts. PAOO employs the potentials of "dead cells" to serve as an osteoinductive scaffolding for endogenous bone growth. But if the host's stem cells are minimal or effete so is the regeneration.

PAOO, despite its manifest efficacy, lacked a method of ensuring the graft was robust in a way to quantify the number of robust cells *in situ*. Fortunately, the introduction of living stem cells to the graft solved this problem. Supplemental *exogenous* multipotent stem cell allografts[9] with potent growth factors can fortify in a complementary way, the minimal numbers, and effete stem cells of the host (Murphy *et al.*, 2015). One no longer needs rely on merely *endogenous* elements liberated from the medullary bone and blood when an excellent FDA-approved stem cell product has been extensively in medical orthopedics for many years.

The exogenous stem cells, euphemistically referred to as "viable cell matrices," are attached to a DFDBA "vehicle" for easy handling. The stem cells are taken from biopsies in healthy patients under the age of thirty to ensure safety and robustness. The stem cell product boasts millions of multipotent MSCs/cc in a DFDBA vehicle that is easily handled. Originally, the population was not expanded in cell culture. But since its introduction cryogenic technology has changed to allow expansion and "banking" of robust MSCs, in either endogenous or allogeneic grafts.

DFABA is only osteoinductive stimulating the regenerative potential *in situ,* and in older patients, a robust potential is absent. Nowzari, (2008) was the first to recognize this and published a variation on the Wilcko protocol. By using an autograft harvested from the mandibular ramus. Nowzari added extra robustness to the healing wound by the autograft and presumably engineered faster regeneration of the new alveolus form. The first disadvantage to the Nowzari technique is that it requires a second surgical site for harvest. The second disadvantage is the weakness of autogenous cells in older patients. Refined protocols using a *viable* stem cell admixture to an autograft makes a productive enhancement. The stem cell approach to alveolus expansion surgery was first published by this author (Murphy *et al.*, 2012) during a prolific period of clinical innovation inspired by the Wilcko group's "revolution." And the SCAT is even better in

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[9] OsteoCel Plus®, Ace Surgical Supply, Inc. Rockton, MA USA.

time. Because the allograft contained *living cells,* the healing is more subdued and even recruit endogenous latent stem cells to differentiate as well. This is a manifest advantage in regenerative surgery. This author's protocol is demonstrated step-by-step in Figure 1.22a–h.

The development of stem cell regeneration to facilitate this standard more predictably in orthodontic therapy is discussed in length elsewhere (Murphy *et al*., 2012, 2015), but a brief reiteration is appropriate. The progression of human development, from the conceptual to the empirical, from the rudimentary to the sophisticated, and from the unpredictable to the routine is a trajectory of fits and starts. Likewise, from the perfunctory extraction of healthy teeth to the molding of beautiful faces, intellectual leaps, retreats, and rational adjustments are to be expected. Such was the quantum leap of viable MSCs into medical orthopedics, periodontal regeneration, and OTE. MSC regeneration of the dental alveolus is exceptionally compatible with the standard PAOO protocol published by Wilkco.

Because of supportive literature and our own extensive experience, we propose that the clinical standard for long bone regeneration (Chaparro and Linero, 2016) and the emerging choice for a variety of regenerative protocols (Safari *et al*., 2018) should also be considered the preferred choice for alveolus augmentation in PAOO. SCAT has been the treatment of choice in the author's office for over a decade with commendable and predictable outcomes. While not yet universally appreciated, when proffered fairly, SCAT is also the first choice of many patients. The Ferguson research group contributions, 2005–2014, have strengthened the PAOO rationale and demonstrate how far we can go with it in their "new envelope of movement."

SCAT achieves that goal more robustly and more predictably than DFDBA or autografts, and in the future, stem cell grafting will constitute a 21st-century gold standard (Figure 1.14).

The Ferguson Envelope of Movement tells us that the alternative is here.

There will probably never be a total replacement for bicuspid extraction or its justification in clinical art. For the exigencies of private practice extraction therapy,

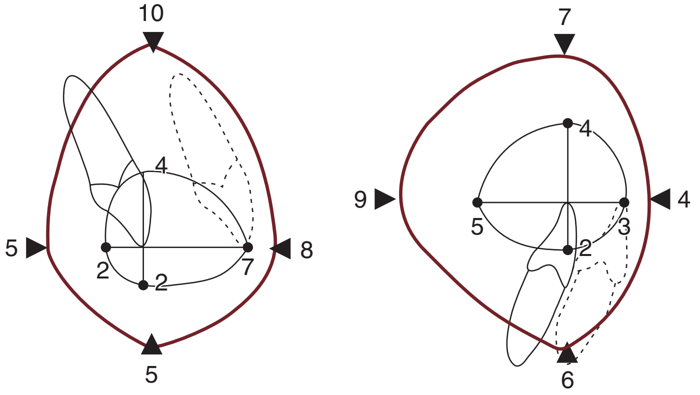

Figure 1.14 The "Ferguson New Limits of Tooth Movement" revises the Proffitt model by demonstrating the range of OTM within an alveolus to be enlarged by PAOO. Note particularly the distance that incisors can be moved anteriorly. This new science argues assiduously for non-extraction therapy, an enlarged alveolus bone via tissue engineering, and a prominent facial profile as seen in many bi-maxillary protrusion cases. The black circle represents the old limits of orthodontic tooth movement. The red circle represents the new limits of orthodontic tooth movement enhanced by OTE protocols. *Source:* Image courtesy of Dr. Donald J. Ferguson. https://www.sciencedirect.com/science/article/abs/pii/S1073874615000353.

despite its limitation, is inescapable. The need for “camouflage-biomechanics” in cases of Class II skeletal dysplasia and correction of disfiguring bimaxillary protrusion certainly define a perpetual need to occasionally sacrifice some dental units. Even SCAT and a singular PAOO protocol to reconstitute more esthetic protrusive lip posture have limits. But SCAT is an emerging first choice in many erstwhile orthopedic surgical theories, so it must be considered in orthodontists who value facial beauty over simple tooth alignment.

Stem cell engineering of bone tissue even has a place in extraction therapy. Stem cell supplements aid ridge retention and extraction site healing. Moreover, surgical manipulations of the vacant alveolus can accelerate anterior teeth retraction *en masse* safely without anchorage loss. Because of these merits, unproductive, inert synthetic material for bone tissue engineering will probably lose favor in the future among enlightened doctors. Thus, it behooves all progressive clinicians, to learn about SCAT.

Beyond the Ligament with Mechano-biology

Given a basic understanding of stem cell biology, we turn to its practical application in clinical practice. One aspect of “cell-level” orthodontics that merits greater consideration in orthodontic curricula is mechanobiology (Van der Meulen and Huiskes, 2002). This is a relatively new science that investigates the effects of exogenous force reflected on and within cells (Ingber, 2008). A good education in stereochemistry and protein chemistry is extremely helpful in understanding clinical outcomes. This is the kind of biology which essentially defines the biology of orthopedically strained stem cells and the reason predoctoral educational requirements demand significant proficiency in organic chemistry and physics. It is these disciplines which form the conceptual basis of molecular and mechano-biology.

The concept of an engineered alveolus phenotype introduced at the start of the new century, (Murphy, 2005a, b) acknowledges the traditional explanation of tissue behavior in the periodontal ligament but asks for more. This focus on the ligament alone has occupied orthodontists for too long, and using it to singularly describe the full spectrum of mechano-physiology in the subjacent bone is inadequate.

Newer concepts go *beyond the ligament* to champion the theories of subperiosteal cell dynamics as a determinate of treatment stability. The original model of hyalinization of the periodontal ligament was always at odds with the medical orthopedic model, and the two divergent views were only recently reconciled. Yet the so-called “pressure–tension model,” despite the early criticism by Baumrind half a century ago (Baumrind, 1969) persists anachronistically to dominate the clinical worldview and the curricula of many academic institutions. The pressure–tension model was published around the end of the 19th century (Sandstedt, 1904, 1905) and should not be consumed whole as a comprehensive explanation of alveolar bone physiology.

The pressure–tension hypothesis been criticized as effete and parochial as late as 2006 by Meikle who just as easily could have been explaining PAOO. He argues,

> Contrary to the impression gained from the literature, tooth movement is not confined to events within the periodontal ligament. Orthodontic tooth movement involves two interrelated processes: (1) deflection or *bending of the alveolar bone* and (2) remodeling (*sic.*) of the periodontal tissues. (Emphasis added)

The new model, “appositional osteogenesis,” a compensatory subperiosteal phenomenon, when bone is distorted, did not get much recognition until Ilizarov popularized distraction osteogenesis. Ironically, an indirect reference to the phenomenon antedates Ilizarov's data by nearly a century. A 19th-century observation by Farrer (1889) notes that orthodontic biomechanical adjustments can bend the alveolus bone *en toto*. This "whole bone" concept has been alluded to by Melsen (Burstone, 1988) and others. It appears that bending of the whole alveolus bone results in shear compression which affects the subperiosteal cells' cytoskeletons, causes canaliculi fluid perturbations, and, at the cell level, transduces strain to biochemical and behavioral changes inside the stem cell cytoplasm.

The OTE based on this "Peri-orthodontic Hypothesis" seeks to explain the effects of both PAOO and even some nonsurgical appliance therapy where the classic "pressure–tension hypothesis" falls short. The peri-orthodontic hypothesis beings with Popper's (2002) falsification principal *vis a vis* the ligament focus. We propose that it is the physiologically adaptive mechanisms in the entire periosteum, not just the PDL, which effect a reengineering of the alveolus form to a larger or more accommodating phenotype after PAOO and to a more robust form with SCAT. The healing of the bone under load amplifies the natural appositional modeling of bone that effects osteoclasia on the convex side (Figure 1.15) of a bone and osteoblastic osteogenesis on the concave surface of a bent bone (Figure 1.3). This bending, we proffer, accounts for the striking success seen in Wilckos' tomographs (Figure 1.23).

But neither surgery alone nor orthodontic pressure alone can achieve permanent change in the alveolus or ensure impressive stability; *both* must be orchestrated simultaneously. Happily, there is no need for any orthodontist to change his or her favorite biomechanical style. All biomechanical protocols will work with PAOO and SCAT if the adjustment appointments are scheduled no farther apart than 1–2 weeks to perpetuate the RAP and activate stem cells.

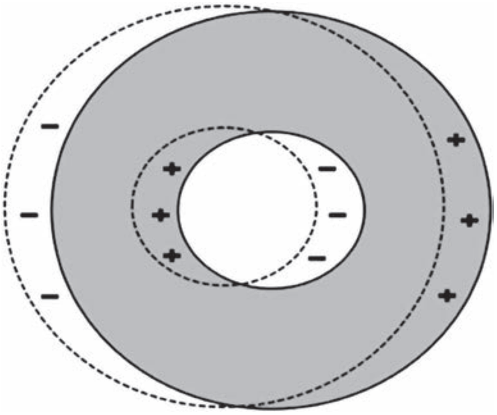

Figure 1.15 This cross section of a long bone demonstrates how bone is reshaped by osteogenesis and osteoclasia in shear load environments. The minus signs represent osteoclastic activity and the plus signs demonstrate subperiosteal compensatory osteogenesis. *Source:* Schlecht (2012)/Stephen Harold Schlecht.

This hypothesis is biologically valid, and the creation of a more accommodating alveolus phenotype is possible, because – at the cell level – *wound healing recapitulates regional ontogeny, viz*. the basic function of a stem cell *in utero* is not much different from its function in a healing SFOT "wound." In other words, the stem cell *in situ* simply responds to local epigenetic perturbations and does not "know" if it is residing in a developing fetus or the healing wound of a mature adult. Any alveolus healing wound, supplemented with MSCs and subjected to physiologic orthodontic forces, creates a phenotype reflecting epigenetic perturbations which the orthodontist (*cum* tissue engineer) decides to induce. Reiterating, at the cell level, a stem cell responds *only* to local environmental chemical or physical stimuli.

Some criticisms of this new heuristic model have been presented, but most are specious because they lack a sufficiently authoritative biological basis and rely on the intergenerational didactics among clinical artisans for validation. But a challenge to a clinic-level paradigm is not without merit; a heretical idea that works certainly has precedent in science. But if new theory is to have scientific *gravitas*, it must be appropriate for cultural needs, and contribute to better results, albeit inconvenient and disruptive. When new ideas are algorithmically sound, supported by biological science, and logically consistent, they should enjoy reasonable appeal to open-minded fellows and earn a legitimate place in the pantheon of accepted clinical therapeutics.

The Mechanosome

At cell-level biology, there is a missing link between genetic expression and clinical skeletal change. That link may be the mechanosome. In 2003, Bidwell and Pavalko proposed a theory of mechanotransduction that involved a hypothetical entity called a "mechanosome" (Figure 1.17). The mechanosome hypothesis explains that deformation of bone deforms bone cells, and that in turn ultimately deforms target genes. That is to say, *bending bone bends genes*. This DNA physical distortion ostensibly is the "instructional mechanism" which alters the gene's morphogenetic expression, i.e., overcomes Waddington's canalization. The Bidwell and Pavalko hypothesis suggests that bending bone bends DNA in a way analogous to stereochemical phenomena.

In 2010, they explained that the mechanosome is not a single object or intracellular organelle but rather a mechanosome transduction *pathway*. This pathway is a cascading series of molecular interactions. The origin of mechanosomes, they explained was

> "... the transient formation of complexes comprised of adhesion-associated molecules and nucleocytoplasmic shuttling ATFs, transferred information from the membrane to the nucleus." They cited β-*catenin/LEF-1* and *Nmp4/p130C* ... as mechanosomal archetypes.

In 2010, Bidwell noted that

> ... the load-induced nuclear translocation of β-*catenin/LEF-1* is accepted as a ... significant component of bone mechano-transduction, and *Nmp4* has been characterized as a general inhibitor of bone anabolism.

Intracellular and transmembrane cytoskeletal proteins (Figure 1.16) target specific genes

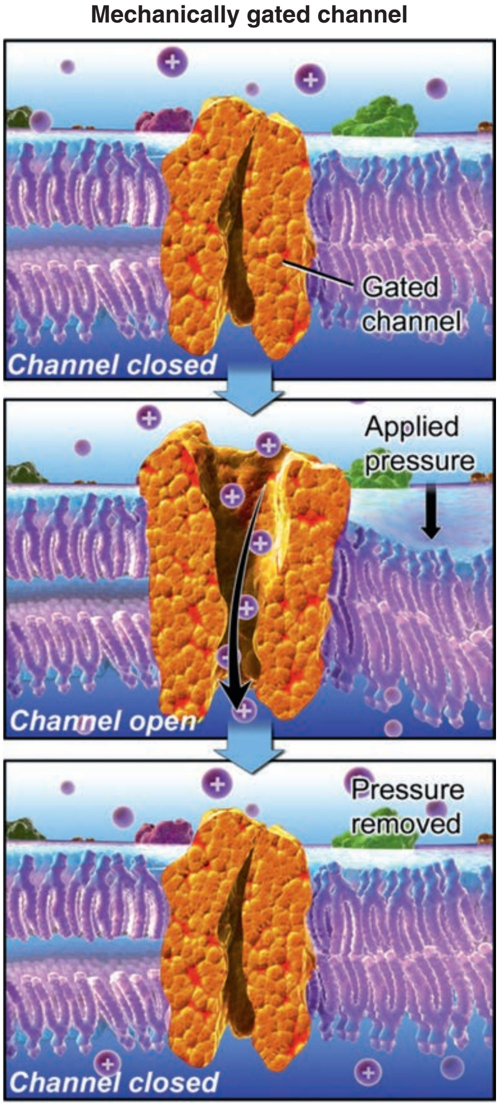

Figure 1.16 This is a theoretical representation of a cell membrane mechanical gates channel which is hypothesized to be an important part of the mechanome pathway involved in bone phenotype alteration. *Source:* BruceBlaus/ Wikimedia Commons/CC BY-SA 4.0.

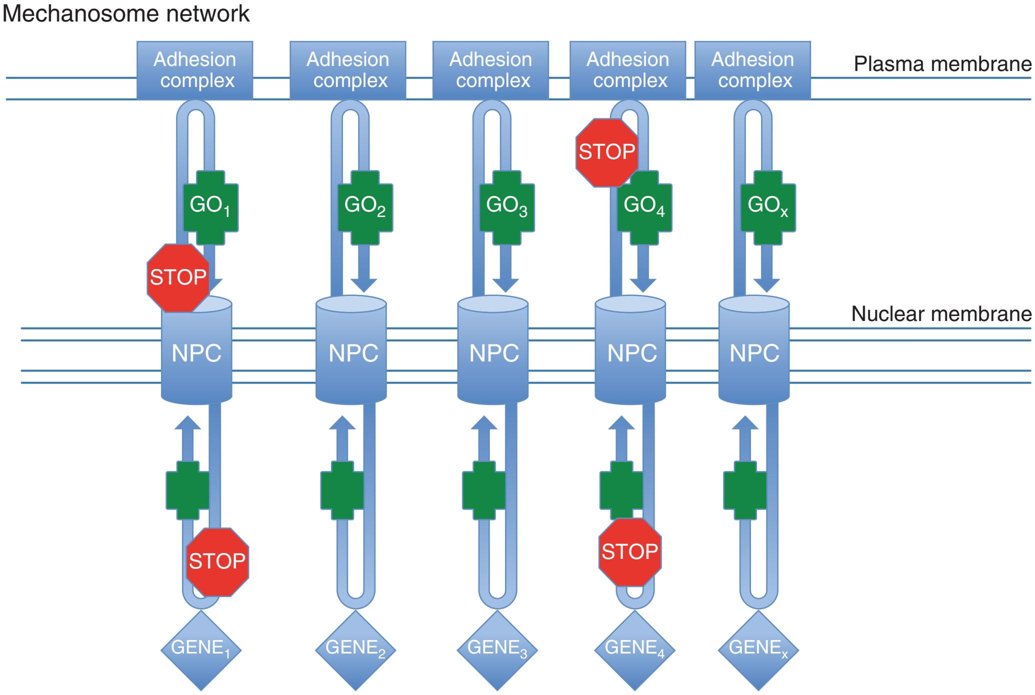

Figure 1.17 The mechanosome network is displayed here by Bidwell and Pavalko (2010) explaining mechanical transduction of exogenous loading into morphogenetic alteration. In the words of Pavalko and Bidwell, "Nucleocytoplasmic shuttling transcription factors cycle between the cytoplasm and nucleus. PTH-and/or load-induced deformation of the membrane launches adhesion-associated molecules from the adhesion complexes. Adhesion proteins with the transcription factors for 'GO' mechanosome which cause bone formation and 'STOP' mechanosomes which minimize 'GO' mechanosome activity strike a balance between morphological change and steady state homeostasis." The launching of adhesion- and cytoskeletal-associated proteins into the nucleus toward target genes appears to be a common mechanism for regulating cell response to changes in its mechanical microenvironment. *Source:* Bidwell and Pavalko (2010)/Springer Nature.

sensitive to mechanical stimulation via this transduction cascade (Figure 1.17).

This ultimately "reprograms" DNA with a new "transcription message," in a way not dissimilar to new software reprogramming a computer hard drive. The Bidwell–Pavalko hypothesis of mechanotransduction is intriguing as a working model until future explications reveal a better explanation.

In summary, orthodontic force application, immediately translated to gross anatomical deformation of alveolus bone, changes the conformation of cytoplasmic proteins, and distorts cytoskeletal tensional integrity (tensegrity) with presumptive strains. This is consistent with the findings of Ingber *et al.* (2003, 2008) and Mao (2002) widely respected clinical tissue engineers. The physical stimulation of the membrane launches mechanome activation at focal adhesins while concomitantly putting genes into position for direct chemical interaction with the mechanosome messengers. Upon arrival at the target gene, this transcription factor conceptually adds data to the physically altered DNA conformation to influence the desired genetic expression as the new DNA "message" forms a more stable architectural form.

Apparently, these phenomena are determinate engines involved in techniques to engineer novel genetic expression and morphogenesis and are predictable due to the fact that *wound healing recapitulates regional ontogeny*. It is crucial to recall that a wound or load separately is not capable of eliciting such profound change in permanent alveolus architecture. It is *synergistic interaction* that delivers the clinical outcome, an emergent event from a multifaceted chaotic, or nonlinear complex dynamical system.

Enter the Exosome

A natural progression from the Wilkos' PAOO flows to stem cell regeneration and then to MSC products, passing ultimately to exosome injections.[10] Significant exosome developments have occurred in the last decade. An exosome is a small, spherical, membrane-bound vesicle containing genomic material which is released by short-lived MSCs. They are produced and released by all stem cells regardless of embryologic characteristics. Exosomes, once released from stem cells, are termed "extracellular vesicles" (EV) (Figure 1.18a–c).

EV's liaise cell-to-cell communication by transferring cellular data, proteins, and RNA, among cells. This intercellular communication is important for regulating immune responses, cellular signaling, and waste removal. Overall, exosomes are commercially available[11] and can play a crucial role in the regenerative process. We have found that adding exosomes to bone grafts of any kind can add significant regenerative potential which may potentiate the effects of epigenetic perturbation and orthodontic loading.

TMP and MOP Nonsurgical Alternatives

Where sufficient labial bone is present, one may ask if a nonsurgical alternative can evoke an effective perturbation. Just as the surgical evolution trended toward less morbidity, the same evolution applies to nonsurgical techniques. The question begged is: Can a minimal-intervention technique be employed successfully and comfortably in an out-patient setting to achieve the same outcomes as the surgical techniques. This question was answered affirmatively in 2005 when we introduced a reasonable alternative to surgical flap reflection (Murphy, 2005a, b, 2006). A significant cohort of patients absolutely will never submit to elective surgery yet desire a well-treated smile. The objective of this alternative was to solicit RAP without the stigmata of oral surgery.

After studying PAOO and understanding the RAP phenomenon, the author designed a *non*-SFOT. Encouraged by the results of physiologic manipulation of the osteogenic/osteoclastic dynamic, a series of small alveolus bone perforations were made directly through the gingiva without reflecting a surgical flap. The procedure demonstrated excellent accelerated OTM and is called "Trans-Medullary Perturbation", (TMP), *technically a kind of "medull-otomy"*. It worked very well when sufficient bone can be identified with tomography and the arch length deficiencies were minor. It is also important to note that the perforations went 4–8 mm deep into the alveolus medulla (spongiosa). The effect appeared within a week and created a profound increase in physiologic tooth mobility. It is especially noteworthy when serial perforation is employed a fortnight apart. In such cases, movement of the teeth could be achieved with simple finger pressure within a few minutes. And, in such cases no attachment loss was evident (Murphy, 2014) if the perforations did not impinge within 3–4 mm of the alveolus osseous crest.

This nonsurgical perforation was first introduced to orthodontists in 2005 at the Eighth

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[10] ChatGPT enhanced

[11] ExoFlow®, Direct Biologics, St. Louis, Missouri USA. Sometimes experts will advise against using NSAIDs (non-steroidal anti-inflammatory drugs, e.g. ibruprofen) because they can delay OTM. But this occurs only in cases of chroic dosing. If high doses are prescribed for immediate post-operative discomfort in this protocol, it analgesic effects will occure in the normal OTM latent period, and thus irrelevant.

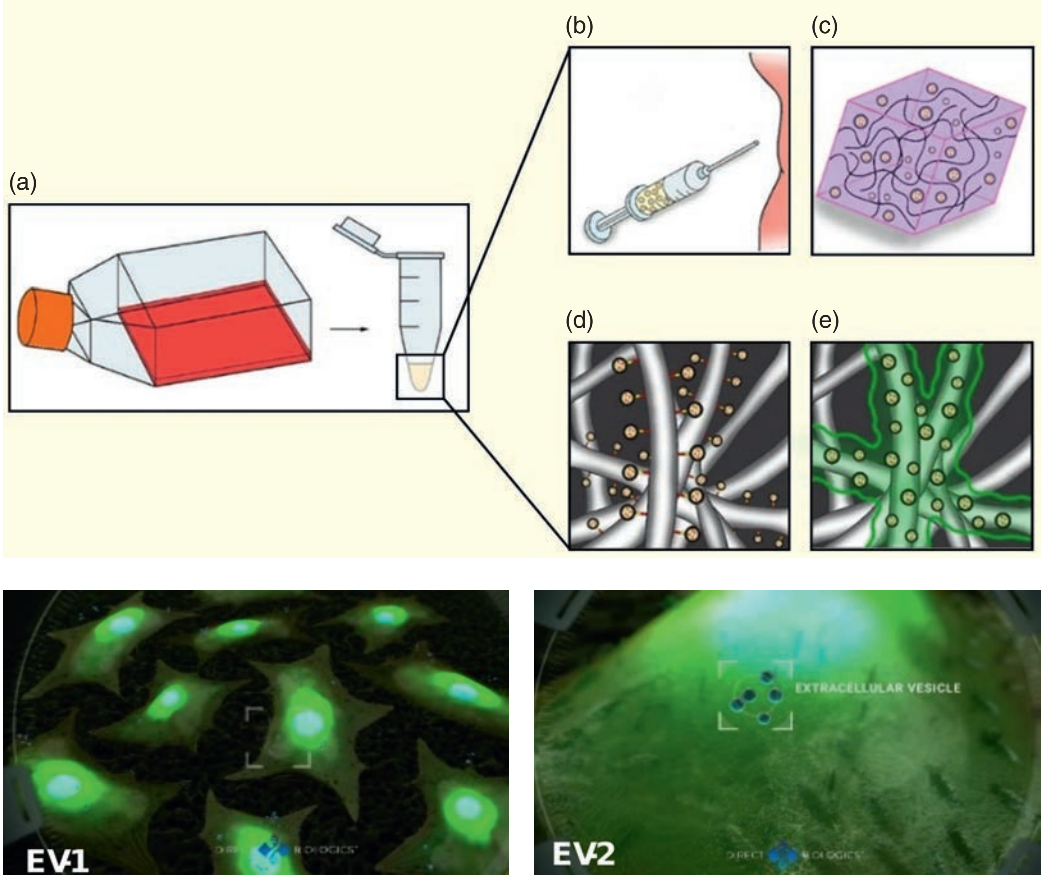

Figure 1.18 (a) Applications of EV in OTE: The isolated EV filtrate (a) is employed in regenerative medicine and regenerative periodontics, separately or in concert with other therapeutics. *Via* (b) direct injection into tissue or general circulation. (c) An EV/hydrogels complex (d) chemical linkers, antibodies, or specific tags engineered onto the EV. (e) Biodegradable gels such as fibrin, which creating a long-term pharmacodynamic entity during gradual degradation. *Source:* De Jong (2014)/Frontiers Media/CC BY 4.0. (b) Applications of EV in OTE; The image EV-1 represents MSCs with endosomes which upon release from the cell (EV-2) are referred to as exosomes, i.e., extracellular vesicles (EV). Added to a bone graft of viable cells, EVs facilitate healing by employing regulatory proteins and RNA contained in EVs to facilitate intercellular communication, aid in revascularization, and advance healing potentials and phenotype development. *Source:* Direct Biologics LLC / https://directbiologics.com/exoflo/ / last accessed November 17, 2023.

International Conference of the Harvard Society for the Advancement of Orthodontics in Phuket, Thailand and again at the annual meeting of the American Academy of Periodontology the same year. It is significant and encouraging that the procedure was well received in both venues. So as of this writing, TMP has been working well for nearly 20 years.

A subsequent replication of TMP was introduced in New York City and validated in humans 7 years later (Alikhani *et al*., 2012). The procedure, less definitive than TMP was called *micro-osteoperforation* (MOP). The MOP perforation technique differs significantly from TMP because it cuts less deeply into the cortex. It has been shown to be effective by local advocates but when combined with a flap is identical to the punctate perforations that the Wilcko brothers introduced in 2001.

Nonetheless, it has been shown by Alikhani *et al*. (2012) that TMP can double the rate of dental translation and may be an effective treatment adjunct. The scientific justification for TMP was essentially a replication of the Wilcko (2000, 2001) technique only “flapless.” It is noteworthy that superficial perforation may fail to recruit significantly more RAP just as timid surgery fails. That is why TMP was designed to penetrate 4–8 into the medullary bone for maximum effectiveness. Others have confirmed the efficacy of the TMP procedure by replicating the protocols in laboratory animals (Teixeira *et al.*, 2010) and humans (Alikhani *et al.*, 2012; Alansari *et al.*, 2017; Feizbakhsh *et al.*, 2018).

TMP is executed under local anesthesia with epinephrine-fortified lidocaine. This method helps form a blood clot to protect the perforated site; patients report little to no pain. The mild post-operative discomfort is ironic since a deep penetration elicits a transient but profoundly therapeutic osteopenia. Their discomfort is easily managed with one to two doses of NSAIDs. The images herein, (and in Figure 1.19) were first presented in 2005 as mentioned above, at that presentation it was emphasized that TMP success depends upon a release of medullary stem cells in addition to the osteopenia. It is the deep medullary stimulation that accounts for the efficacy of TMP in contrast to the effete response to MOP penetration of merely the cortical bone.

Figure 1.19 This demonstrates the original (2005) method of transmucosal alveolus perforations (TMP), a nonsurgical method of inducing regional osteopenia for accelerated and efficient orthodontic tooth movement (OTM). When TMP is executed properly a clot immediately forms to protect the perforated site and patients report little to no pain.

This author’s theory thus successfully ran the gauntlet of skepticism and is now successfully employed for several clinical challenges. The difference between TMP and its derivative, MOP is that the latter produces divots too shallow for many cases. But for minor tooth movement, MOP should suffice. TMP can be used for any case normally planned for surgery, but not in cases where bony support is questionable. Conceptually, molecular-level alterations of genetic expression with surgical perturbation and TMP are the same. So, TMP may also enhance stability if phenotype is altered significantly. But such stability studies have not yet been published.

The clinical success of the TMP innovation has been summarized well in a review by Chou and Alikhani (2017). But there is some disagreement on how well shallow perforations may work. The authors note “Three MOPs with a depth of 4 mm can be … effective method to increase the rate of tooth movement. However, … depths of 4–7 mm does not additionally enhance tooth movement.” (Ozkan and Arici, 2021) Our experience is just the opposite. We speculate that no coordinated osseous strain accompanied the authors’ perforations. This is why, we generally sink perforations at least four to eight deep and perturb the medullary bone with sweeping movements within 3–4 mm of each other. This is intended to elicit medullary osteopenia, the medium through which teeth move. Medullary RAP will not follow innocuous shallow divots in thick cortices. In our experience – given wide biological diversity – that greater depth penetration can indeed turn a failing case into success. But often the failure is declared too early in treatment. Some failure to accelerate OTM can also be attributed to the natural OTM latency period seen in traditional OTM. Thus, patience and the willingness to apply multiple doses of TMP will usually salvage a refractory response.

The failure of Chau and Alikhani to achieve RAP with deeper perforations brings out a basic problem with clinical studies; they do not always replicate protocols faithfully. A good example of inadequate penetration and inadequate clinical management was published by Swapp (2015) and Cramer (2019).

To their credit, Swapp *et al.* (2015) claimed the effect of accelerated tooth movement requires penetration *into the medullary bone* which confirms our observations. Referring to the corticision study of Kim and Tae (2003), they say:

> ... tooth movements that were 3.75 times faster than the controls.
>
> ... the blade was *driven up to 10 mm* into the cortical bone; this is significantly deeper than any reported corticotomy procedure and might have been expected to damage both the cortical and trabecular bone surrounding the teeth that were moved. These studies suggest that the awl injuries produced in this study *might not have been deep enough* to affect the bone mesial to the root being moved. (Emphasis added.)

All cases must be individualized beyond the perfunctory compliance with TMP protocols. This is why, we generally sink perforations four to eight deep *with a sweeping motion* and cluster them within 3–4 mm of each other. Swapp's research affirms that the term MOP should be relegated to shallow penetration, as the term TMP refers to more definitive spongiosa management and less on mere cortical management pe se.

It should be noted that some cases (in concordance with biological principles of wide variance) will demonstrate almost complete recalcitrance to therapy, regardless of extreme decortication efforts. From a logical point of view, definitive perforations of TMP as an SFOT intermitent supplement can release endogenous medullary stem cells which are necessary for augmentation success and morphotype modification. This produces a more profound immediate effect (Ozkan and Arici, 2021) which can lend greater stability over time.

The Problems with Orthodontics

A failure to achieve sufficient osteopenic state to escape canalization and facilitate clinical OTM will occur for specific reasons, some known and preventable, e.g., inadequate depth of perforation, poor patient compliance, and a myriad of idiopathic factors common to biological systems. The job we have at hand is to elaborate on the phenomenon for the edification of our colleagues and to protect patients from untoward events. Therefore, it is not our responsibility, in this chapter, to prove that TMP *can* work. That has been demonstrated. As the burden of proof that all swans are white is vitiated by an observation of one black swan, our cases for over a decade document many "black swans."

All that critics have achieved with their experimental failure was to demonstrate that their experimental design was inappropriate, or they have documented the obvious: biology is not always predictable. The results of individual failures do *not* demonstrate a universal truth. Logically, they may only claim that a technique is not universally possible for all doctors. True. It is not possible to replicate protocol if one performs it wrong.

Papillary Slough as a Troubling Complication

Complications to OTE generally appear as standard surgical problems, viz. bleeding, pain, and infection. The orthodontic complications are generally limited to an inability to sustain a *constant osseous internal strain*. However, slough of the interdental papilla is also a troubling complication that one must bear in mind because it is so easy to prevent. The best way to minimize this event is to be ever conscious of vascular richness of the gingiva and preserve it. Interdental papillary erythema is a sign that slough has occurred (Figure 1.20).

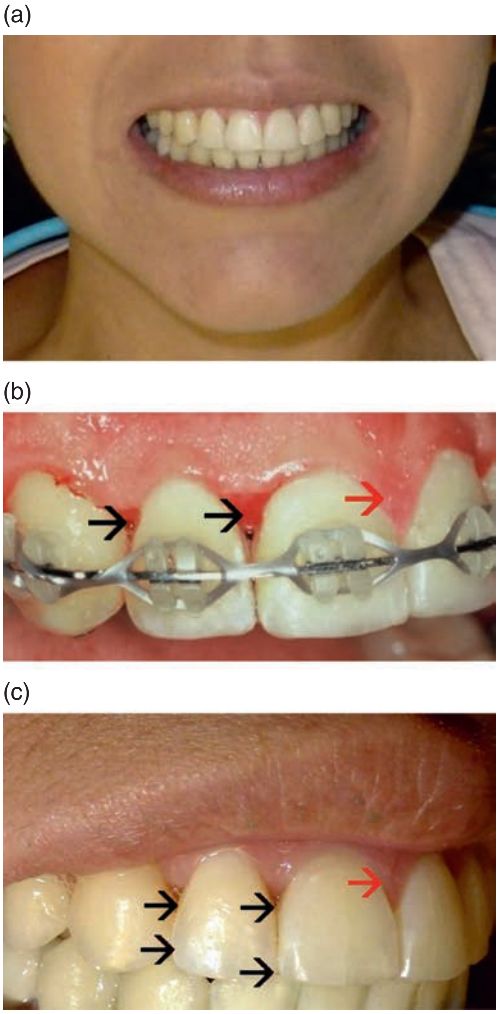

Figure 1.20 If a papillary slough opens gingival embrasures, black triangular areas (a) appear obvious to the patients (b) (black arrows). The cosmetic disappointment has been ameliorated by interproximal odontoplasty known as "re-proximation," interproximal (enamel) reduction (IPR)," or "inter-dental stripping" in orthodontic parlance. Note the large flat contact areas in (c) (apico-coronally between the black arrows) and the papillary integrity of the incisive papilla where the mucogingival flap was not reflected (red arrow).

The *black triangle* generally appears in adults when incisor crowding is eliminated during the aligning stage of OTM. So, its appearance should not be categorically attributed to poor surgical technique. However, taking SFOT surgery within 3–4 mm of the osseous crest can indeed cause loss of papillary integrity and recession. If a "black triangle" does appear, the patient should be reassured that it can be eliminated by interproximal odontoplasty. This is also called interproximal enamel reduction (IPR) or "interdental stripping." This procedure is often used to prevent the notorious rotational relapse of canines and incisors. Rotations are difficult to retain because contact *points* offer less resistance to rotation than contact *areas*. The wide flat contact area produced by IPR offers countervailing resistance to rotation, as flat walls would prevent the rotation of a cube. The cosmetic effect is to reduce the height of the gingival embrasure to the point of obscurity.

Managing Periodontal Infection

A misapprehension has haunted the development of OTE from its inception. The challenge is to manage SFOT in patients who present with periodontal inflammation. Is SFOT contraindicated in patients with periodontal inflammation? No. It is only contraindicated in patients with *untreated periodontitis*. The important question to ask is whether the surgical treatment of the infection should occur prior to SFOT or *simultaneously with* SFOT. We claim the latter. This inappropriate contraindication that SFOT and other OTE should not be delivered in patients with periodontal disease is sadly widespread. It derives from the fact that active periodontal *infection*, progressive self-sustaining subgingival disintegration of the periodontal attachment apparatus, *is conflated with inflammation*, a reversible phenomenon. Periodontitis is inflammatory but the reverse is not true. Infection is not inflammation. The former requires a unique confluence of host immunologic reactions and the pathogenic potentials of gram-negative anaerobic pathogens. The conversion factor that changes the reversible gingival inflammation (the "established lesion") toward a self-sustaining periodontitis. Progressive attachment loss, is not yet fully defined and may differ from one patient to another, indeed from site-to-site in any one patient as a function of time. (Lindhe *et al.*, 1980).

But as indicated above, we do not agree with the notion that initial therapy must categorically precede surgery. Surgery itself more efficiently eliminates active infection. Indeed, that is why, it was introduced to periodontology. After flap reflection and osseous recontouring in cases of periodontitis, additional decortication can be rendered, and bone grafts placed according to the PAOO protocol. Thus, the fallacious admonition that PAOO should not be performed in fields of infection is ill-conceived.

Subjecting the patients to an additional periodontal surgery prior to PAOO just for debridement is superfluous and only increases financial cost and morbidity. Protracted initial therapy, subgingival scaling, root planning, and subgingival curettage are likewise ill-conceived in regenerative procedures because they compromise healing potential. PAOO is akin to regenerative periodontal surgery and success requires as many helpful tissue growth factors as possible. Root planning and soft tissue debridement can commence immediately after flap reflection not *before* the surgery. Scaling and root planning allow tissue fibroses to develop rather than aiding the regenerative potential of a highly inflamed tissue, so-called "hot lesions." When scaling and root planning are finished in an open surgical field there is no more active infection, only inflamed tissue that receives stem cell grafting better than fibrotic tissue.

PAOO and SCAT Step-by-Step

PAOO/SCAT and corrective periodontal surgeries can be conducted *simultaneously according to expert consensus*. The protocol for using PAOO/SCAT with infected periodontia is illustrated in Figure 1.22a–h. The protocol follows:

1) Place all brackets on the labial surface of teeth as the manufacturer suggests.
2) Reflect a full mucoperiosteal surgical flap to a depth necessary to visualize the alveolus bone. It is not always necessary to reflect the flap completely into the vestibule because that will cause excessive postoperative edema and facial ecchymosis. But do not reflect the maxillary incisive papilla (Figure 1.21, black arrow). Flap reflection may occur on the labial surface only coupled with TMP on the palatal surface as the incisive papilla is untouched.
3) Debride all accretions, root-plane the root surface, then proceed with periodontal osseous recontouring at the surgeon's discretion.
4) Decorticate the alveolus cortex until copious medullary bleeding is observed but do not approach within 3–4 mm of the osseous crest.
5) Soak the root surfaces with ethylenediaminetetraacetic acid (EDTA).
6) Suture the surgical flap with a continuous ("sling") suture at doctor's discretion forming a pouch to accept the graft. This ensures easy graft containment.
7) Apply graft abundantly to the root surfaces and "overfill" as desired.

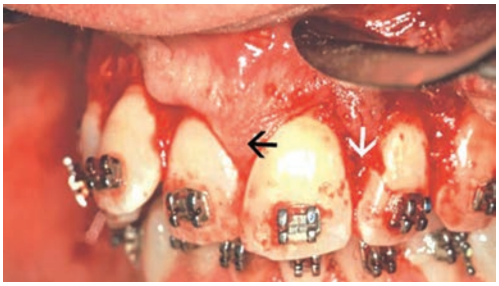

Figure 1.21 This demonstrates that papillary integrity (black Arrow) can also be preserved with a single *split-thickness* labial flap (white arrow) leaving the palatal interproximal tissue intact. These practical gambits may compromise RAP somewhat since buccal and lingual flaps are dictated in the original PAOO protocol. But we have been very pleased with the outcomes of a singular labial split-thickness flap.

8) Coronally position the surgical flap 1–2 mm coronal to cementoenamel junctions (CEJ).
9) Immediately activate the "wound" with a nickel–titanium archwire after the last suture is tied.
10) Cover the sutured wound with 2-octyl-cyanoacrylate dressing and prescribe NSAIDs[12] for discomfort. Proceed with orthodontic therapy activating the wires weekly or semiweekly, as the orthodontic success dictates.
11) After a few weeks, advise the patient of the activation objective and tell them to return when that objective, e.g., space closure, can be achieved, *in days or even hours* after activation.
12) Continue to activate as necessary depending upon the patient's unique response. Avoid any arbitrary schedule, e.g., fortnight intervals. Adjustment goals may be accomplished within days or hours when the osteopenic state is well managed.

Flap reflection in the esthetic zone risks loss of papillae in the case of a flap marginal slough, a rare but significant event. But if a labial flap is reflected off the maxillary midline the appearance of an open midline, embrasure may unfairly be attributed to the clinical style of the surgeon. Another alternative to PAOO that might ensure embrasure integrity in the esthetic zone is to operate with a split flap (Figure 1.21, white arrow) or place initial incisions solely within the attached gingiva, not the sulcus (Figure 1.21).

Figure 1.21 demonstrates papillary integrity (black Arrow) can also be preserved with a single *split* labial flap (white arrow) leaving the palatal interproximal tissue intact. These practical gambits may compromise RAP somewhat since buccal and lingual flaps are reflected as dictated in the original PAOO protocol. But we have been very pleased with the outcomes of a singular labial flap combined with lingual TMP.

Figure 1.22a–g demonstrates the management of these so-called "hot lesion" (severely inflamed periodontal infection). Initial therapy was excluded from the treatment plan to take advantage of the rich supply of growth factors and endogenous undifferentiated MSCs in such lesions. A protracted initial therapy of scaling and subgingival root planning eliminates these robust healing agents. It is well known that there is no difference in clinical outcome quality if scaling and root planning are performed during surgery or before. The advantage of performing initial therapy is to become familiar with the psychological state of the patient. The other advantage of initial therapy is that it reduces surgical time. However, the loss of healing potential in regenerative surgery is too great a price to pay for excessive initial therapy. Note, in Figure 1.22c, d, the *copious bleeding* that was created by the deep and numerous decortication pattern. This bleeding is a blessing to any regenerative procedure. So are "hot lesions."

After the periodontal lesions were debrided and the teeth were root-planed, physiologic topography was created with standard periodontal osseous recontouring. But where deep infrabony pockets precluded aggressive osseous recontouring, e.g., the circumferential defect around tooth #25 regenerative treatment with viable stem cell allograft was selected by the patient. Immediately after the periodontal lesion was debrided, a generalized corticotomy ensued. The sequence of osseous recontouring *vis a vis* decortication lies within the surgeon's discretion. The stem cell allograft was placed according to the PAOO protocol. The mucoperiosteal flap was sutured to place and followed by a cyanoacrylate dressing; "wound activation" here was achieved with a 0.018" nickel–titanium arch wire in full bracket engagement.

A subsequent biopsy and microscopic inspection (Figure 1.22h) of the grafted site demonstrated abundant osseous regeneration (white arrow) adjacent to a DFDBA matrix/carrier (green arrow). OTM did not interfere with osteogenesis, as clinical tooth alignment

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[12] Syndrome (def.) – a group of signs and symptoms which consistently occur together.

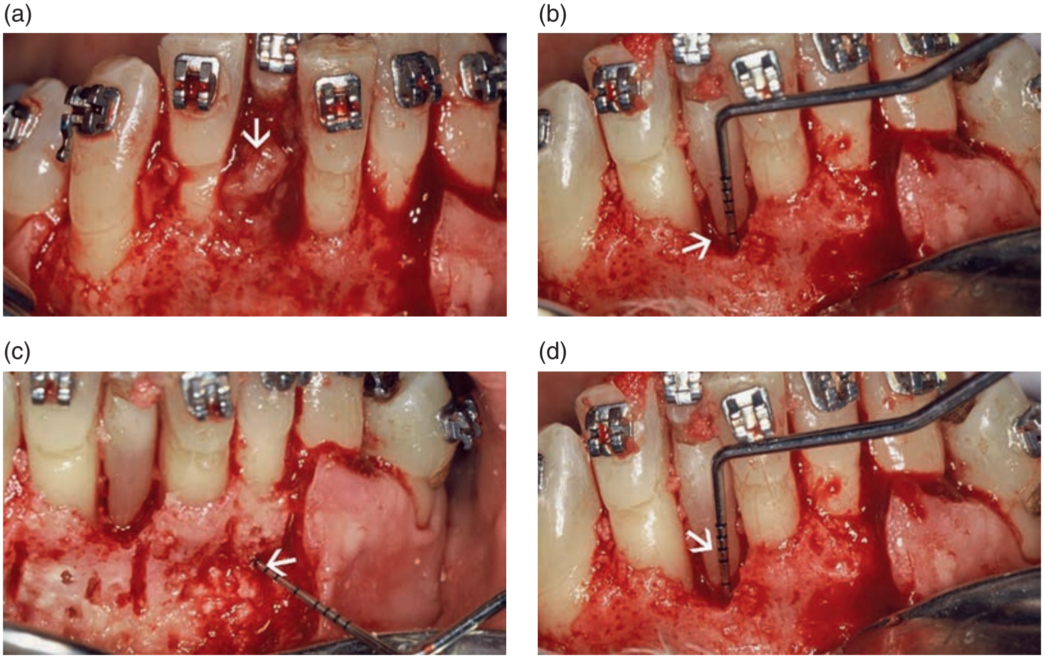

Figure 1.22 (a) Flap reflection reveals a massive infrabony defect at tooth #25 and an infected granuloma (white arrow). This case demonstrates severe periodontal disease with a deep infrabony pocket. The patient elected simultaneous periodontal regenerative-PAOO surgery with OTM to avoid excessive cost and maximize the regenerative potential of OTE. The fixed appliances have been bonded but not loaded. An 0.018″ round nickel–titanium archwire was placed into the bracket 0.022″ slots after the last suture was placed. (b) All granulation tissue has been removed and the roots have been debrided of infected accretions. The most severe infrabony defect is defined (white arrow) and decorticated at its base. The infrabony defect is totally decorticated to release endogenous pluripotential stem cells and growth factors. (c) The labial alveolus bone is decorticated with caution over root surfaces. Where a 1–2 mm cortex covers the root, decortication may be precluded at the doctor's discretion. (d) (Optional) The total denudation of root surface is measured to quantitate success and the sharp rim of the circumferential infrabony defect is flattened. (e) The graft is a viable multipotent stem cell bone matrix, combined with a cancellous freeze-dried demineralized bone allograft (DFDBA) from the same donor. The stem cells are naturally adherent to the surface of the cancellous bone and consist of mesenchymal stem cells (MSCs) and osteoprogenitor cells. After thawing, pouring off the supernatant ensured facile handling characteristics of the "living graft". (f) The graft is placed inside the sutured "pouch," packed copiously into the decorticated infrabony defect floor, and "plastered" generously over the decorticated buccal surface. Here, a continuous sling (circumferential) suture loosely enables the graft to rest secure in a surgical flap "pouch." The suture is tightened like a purse string after the grafting is complete. In this image, a 2-0 resorbable suture is employed. If too much graft is used and the flap cannot be tightened to its original location, the flap should be released from tension at its apical base and more coronally re-positioned. (g) Some bone regeneration is appearing (arrow) under the replaced flap by six to eight weeks. After grafting with archwire activation, a new attachment apparatus is developing, and the robust alveolus is prominently displayed. (h) A biopsy and microscopic inspection of the bone regeneration demonstrates healthy osteogenesis (white arrow) and a sheet of long-lived "bone-lining cells" (red arrow) begins to grow on the demineralized bone matrix (DBM) carrier (green arrow) which may remain up to 15–20 years. *Source:* NuVasive, Inc., La Jolla, California USA.

was accelerated. The asterisk in Figure 1.22f indicates the location from which the biopsy was taken after 3 months. It is true *as a generality* that tooth movement should not be done in areas of *active but untreated* periodontal infection. But occasionally periodontal infection treatment can indeed be done *simultaneously* with orthodontic treatment in this coordinated manner. The bone is just as stable because an improved phenotype was *created by load* placed on the graft differentiation, as the roots were moved into the desire location

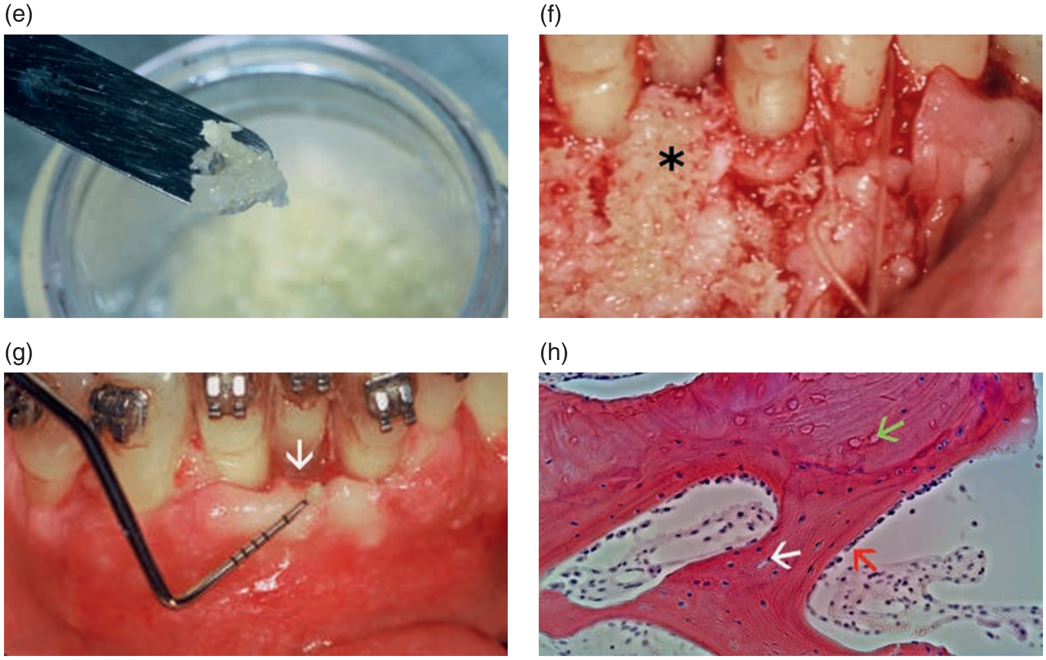

Figure 1.22 (Continued)

during bone healing. This phenomenon is entirely consistent with Moss's functional matrix theory: *the roots of the teeth constitute the functional matrix of a new alveolus phenotype* (Figures 1.23 and 1.24).

Figure 1.23, a Wilcko document, illustrates how copious bone can be created *de novo* with the PAOO protocol. The original surgery in this case was performed on January 7, 1997. Surgical reentry to inspect the bone stability was performed 8 years later January 6, 2005. In Figure 1.24a, note the bony dehiscence on teeth #27 and #29. These are common sites of bony dehiscence, gingival recession, and minimal zones of keratinized attached gingiva. In Figure 1.24b, the outlined dehiscence and fenestrations are more discernable. Often these cases present with the clinical topography of an old-fashioned washboard. In such cases, this "washboard effect" has often been proposed as an indication for bicuspid extraction regardless of how it might affect facial appearance. This extraction indication is no longer valid.

In cases of retrusive and thin lips, an injudicious bicuspid extraction would seriously compromise facial appearance. When we have the fate of a child's face for a lifetime, prudence and serious consideration of PAOO as an explicit element of informed consent is, in our modest view, a professional imperative. Figures 1.23 (2b) and (2c) demonstrate how much stable bone can be created with PAOO. Since this is a new phenotype both the dental pattern and the integrity of the alveolus bone are extremely stable. An eight-year stability has been clinically demonstrated in this case. Basic logic tells us that stability of bone at eight years argues for stability indefinitely if the roots are maintained as the functional matrix of the new phenotype.

In summary, stability of the bony phenotype is caused by *loading a healing bone graft*. A labial loading stimulates endogenous and grafted stem cells to differentiate into a new bony form. That is, the key to success lies in the *simultaneous interaction of bone graft differentiation and load*. Neither the singular effect of incisor advancement nor grafts alone will suffice. Unloaded grafts on the labial alveolus cortex will normally resorb over time when the

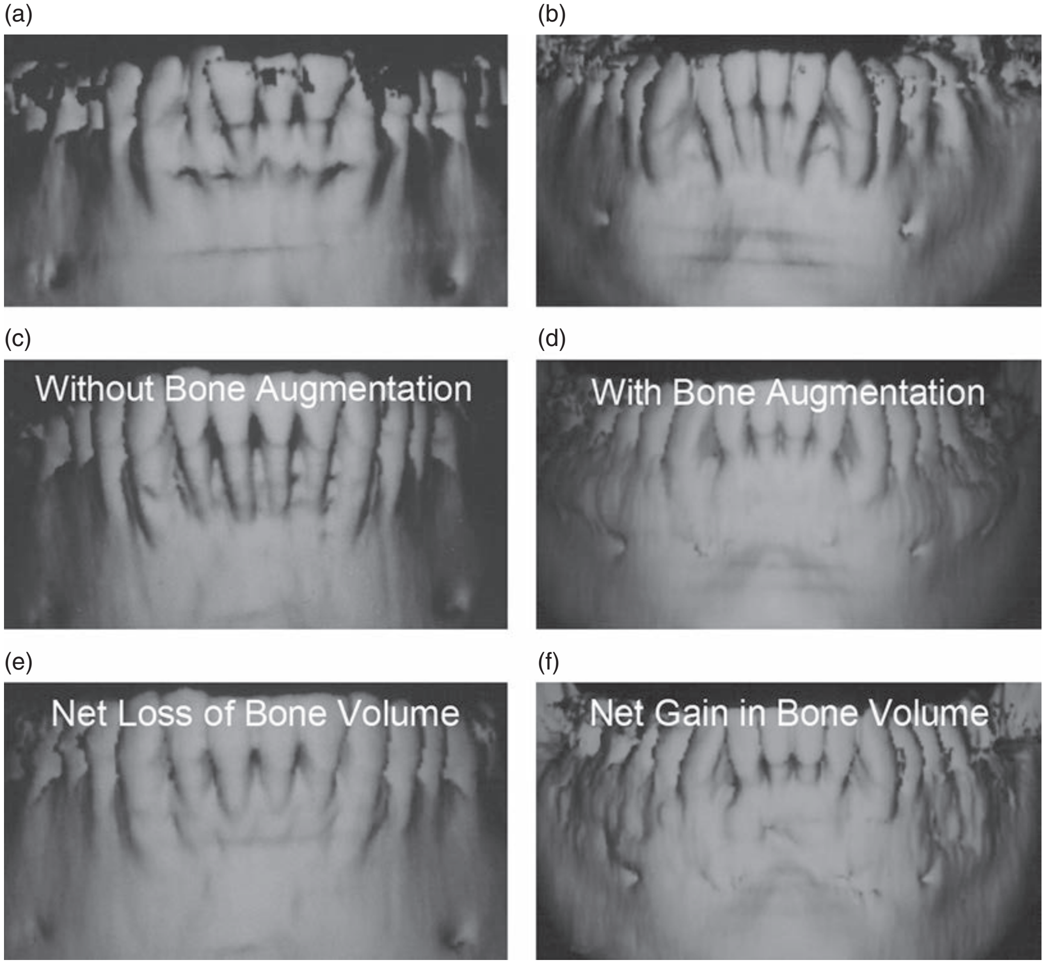

Figure 1.23 This is the predictable appearance of the fully developed alveolus bone when PAOO is employed to augment the bony base. The stable dentition is due to the thickness of the new alveolar morphotype. Usually, 1–2 years are needed to manifest fully calcified regeneration on a tomograph. Thus, early tomography will demonstrate less calcified bone appearing to be a graft "failure" or osteogenesis less copious than expected. *Source:* Courtesy of Dr. M. Thomas Wilcko, Erie, Pensylvania, USA.

graft is not loaded during healing and graft differentiation, and incisor advancement will only bring minimal bone with it. This is not the case with PAOO/SCAT; it is manifestly a *bone-generating machine*.

Ortho-Infection

A discussion of OTM is painfully incomplete without addressing the potential for its predictable and irreversible tissue damage by other oral infections, e.g., hyperplastic gingivitis and focal decalcification. The syndrome[12] is so closely related to traditional fixed appliance therapy that we refer to as, *Ortho-Infection*.

Hyperplastic gingivitis, periodontitis, and focal decalcification (so-called "white spots") are the most common side effects of fixed-appliance OTM. Some speculate that SFOT may amplify the damage of the side effects. This is *not true*; indeed, the opposite is true. Accelerated orthodontic care *reduces* the chance of side effects most notably, apical root resorption, adjustment pain, caries, and periodontal infection. All these complications are time-sensitive. Periodontal infections are

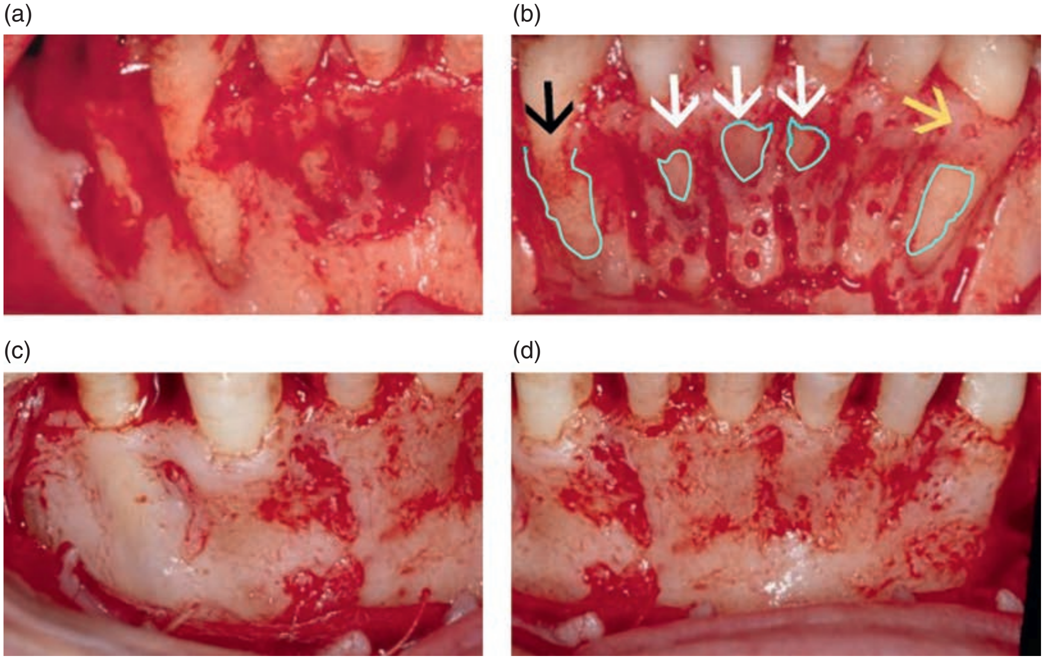

Figure 1.24 This image illustrates how copious bone can be created *de novo* with the PAOO protocol even where stem cells are not employed. In (a) the fenestrations are obscured by blood but are artfully displayed in (b). The bony deficiencies of the original phenotype are dramatically eliminated and replaced by *thick, healthy and normal alveolus bone* in the newly engineered phenotype. (c, d). Images courtesy of Dr. M. Thomas Wilcko, Erie, Pennsylvania, USA.

particularly onerous because they are self-perpetuating with a net tissue loss. First, let us consider the most serious, periodontitis. For 50 years, we have known that irreversible crestal attachment (bone) loss may occur in orthodontically treated adolescents.

In the classic study by Zachrisson and Alnaes (1974), the article states "The orthodontic patients demonstrated slightly, but significantly more loss of attachment clinically than did the reference subjects." We object to this interpretation of empirical data. The mean loss was 0.41 mm which seems insignificant. But the devil lies in the statistical details. The report by Zachrisson and Alnaes *pooled data*. A closer look at the Zachrisson and Alnaes data demonstrate that a cohort of individual patients did indeed suffer clinically significant attachment loss. Data pooling is misleading to conscientious clinicians. It obscures important findings in a sea of numbers. The error of pooling lets the authors to emphasize an arithmetic mean, a descriptive statistic important to discern general trends when standard deviations are also reported. But an arithmetic mean can be an intellectual trap to practical clinicians who treat individual patients (A jocular adage in statistical classes is that a man can drown in a lake that only *averages* 2 ft deep if he just steps into one small spot that is 20 ft deep). The objective of microbial management is to create a microecological niche in which commensal organisms can thrive. So, a clinically relevant measure of lesion occurrence is *not* the midpoint on a Gaussian frequency distribution. Rather, a measure of *range* demonstrates a worst-case scenario for the *individuals* we treat. We treat individuals not arithmetic means. More academic efforts should be directed toward the *infection* that we orthodontists are not taught to see, often ignore, yet always create. The individualized creation of a commensal niche can be achieved with astute patient collaboration.

However, the modern abandonment of orthodontic bands, with inevitable "overhangs," in favor of bonded brackets is a very good start. Beyond that improvement, all too often only a perfunctory acknowledgment of the infection problem is made with a token admonition to "brush better." The problems are more serious in adult patients and very problematic when surgery is involved. So, clinicians should be periodontally vigilant and, solicit ancillary supportive care.

As orthodontists, we operate in a bacterial stew of pathogens and commensal microorganisms. Yet, very little bacteriology is taught in orthodontic training programs. This is unfortunate because the bacterial biofilm in which pathogens thrive is ubiquitous. So, its effect on treatment must be acknowledged and constantly modulated. Knowing how a bacterial niche is created around orthodontic appliances gives us insight into many side effects. It must be noted that these infection side effects are predictable in the aggregate but not necessarily foreseeable in the particular. Thus, a kind of universal precaution must always be maintained, and every patient must be informed *individually* of untoward bacterial events.

Oral Hygiene – A Pragmatic Imperative

Periodontal biofilm accumulation is one of the biggest threats to OTE success. Yet, many orthodontists do not realize that *aggressive mechanical removal* is best for patients. This fault derives from a popular canard that aggressive brushing is harmful. But that idea cannot be supported by compelling empirical data. Indeed, biofilm eliminated around fixed brackets by mechanical means should be as aggressive as possible with an antiseptic-laden (e.g., chlorhexidine or Listerine®) brush. The bristles must be pushed into the gingival sulcus (Figure 1.25). The best protocol is brushing with an "Enhanced-Bass technique" (Bass, 1957), using an end-tuft brush (ETB). But the Bass technique is not effective unless 2–3 lbs. of therapeutic force is placed on the tip of the bristles (Figure 1.28). Therefore, we recommend a "definitive" application of the Bass principles but *enhanced* with contemporary knowledge and a strong antiseptic, not flavored toothpaste.

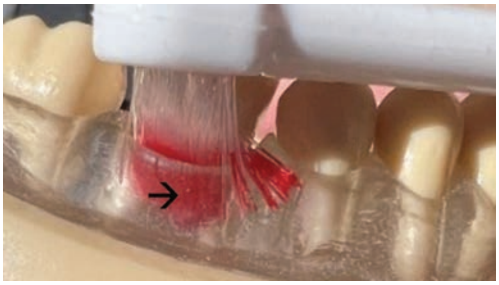

Figure 1.25 An end-tuft toothbrush (ETB) and the enhanced Bass brushing techniques, with 2–3 lbs. of force is necessary to safely remove subgingival bacterial biofilm effectively, improve gingival tissue tonus with dense fibrous connective tissue and create a surface of protective keratinized epithelium at the gingival margin. For best results employ a small circular or scrubbing motion to insert the bristles over the friable attached gingiva, onto the biofilm-covered cervical third of the crown, and into the infected gingival sulcus (pocket). Bleeding with this method ensures that venous congestion is relieved so oxygenated arterial blood can freely flow into the infected tissue. With this technique, the clinical infection will be eliminated with increased gingival tonus and orthokeratinized epithelialization so gingival health will be observed in 7–10 days.

Initially, if there is already a gingival infection the brushing technique will elicit copious bleeding and pain. This is a disincentive to effectively debride the infected gingival tissue. To an uninstructed patient bleeding suggests iatrogenic damage. This of course is untrue. So, patients must be reassured. One effective way of enhancing compliance is to reassure the patient that bleeding is an indication of improvement not damage. An analogy to "draining a swamp" works well.

A typical dialogue between a doctor and the patient to enhance compliance and understanding may hypothetically sound like this:

Compliance Dialogue

PATIENT: This brushing method causes gums bleeding; isn't that bad?

DOCTOR: No. Bleeding on brushing is the first step to oral health. This kind of voluntary bleeding is a sign of improving health. This bleeding is (endotoxin) "contaminated" blood. And it comes from the infected tissue like the skin bleeds around a dirty splinter. It is not the brush doing damage. It is the brush *improving* health. It bleeds the same way a contaminated stagnant swamp is drained to improve water quality.

Swamps smell bad because fluid circulation is stagnant and they are also infected (technically, infested). Toxic *venous congestion* (infection-edema) is eliminated by bleeding. Brushing properly takes out the poison (endotoxin) in the gingiva and allows fresh "oxygen-filled blood" to enter the gums and heal the tissue. Each day will elicit less bleeding. Therefore, each day you must brush the gumline harder and harder, like an athlete in training, until no more poisoned blood can be drained out. Then the infection (for all practical purposes) will BE GONE.

Not brushing vigorously because it bleeds is like stopping exercise because it makes your musclES HURT.

A well-regarded laboratory study by Loe (2000) has shown that an insignificant surface component of enamel might be removed because of brushing, but there appeared to be no major danger of extensive abrasions of the enamel. A popular canard suggests that brushing with great force can damage dental enamel or "cause" gingival recession. This represents a misinterpretation of literature. Considering the Brinell hardness of enamel, it also seems intuitively logical. Moreover, in our experience, any purported injury to marginal gingiva is doubtful, considering the attached gingiva is composed of dense fibrous connective tissue covered with a thick *keratinized* stratified squamous epithelium (Figure 1.26).

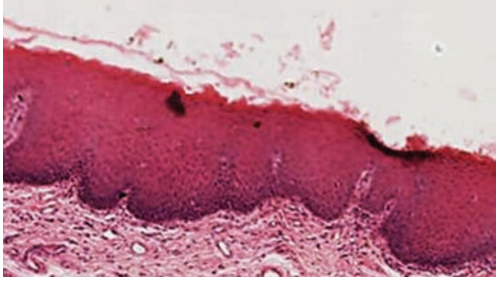

Figure 1.26 Keratinized-stratified squamous epithelium of the marginal gingiva is capable of enduring great mechanical stimulation. This gingival tissue in states of health displays firm tonus and a well-developed *stratum corneum* which is capable of absorbing 2–3 lbs. of brushing force. *Source:* Albrecht *et al.* (2020)/MDPI/CC BY 4.0.

This epithelium can create a dense and thick *stratum corneum* only when mechanically stimulated by aggressive home care. This histological description of masticatory mucosa could easily describe the palm of a human hand or the ventral (plantar) surface of the foot. Each is covered with the same keratinized stratified squamous epithelium (Figure 1.26). So, why should the gingiva be treated differently? We propose that the so-called gingival recession is merely the cardinal sign of a local exsanguination of contaminated venous congestion, a hallmark of chronic infection. Thus, any initial transient bleeding with this technique is salutary and *exactly* the phenomenon seen when any infected wound is debrided in a hospital emergency room.

Enhanced Bass Technique

One popular but discredited technique is called a *Bass-Roll* technique, this is not to be confused with an *enhanced Bass technique*. The former is unnecessarily complicated and too gentle for effective biofilm removal and wound debridement.

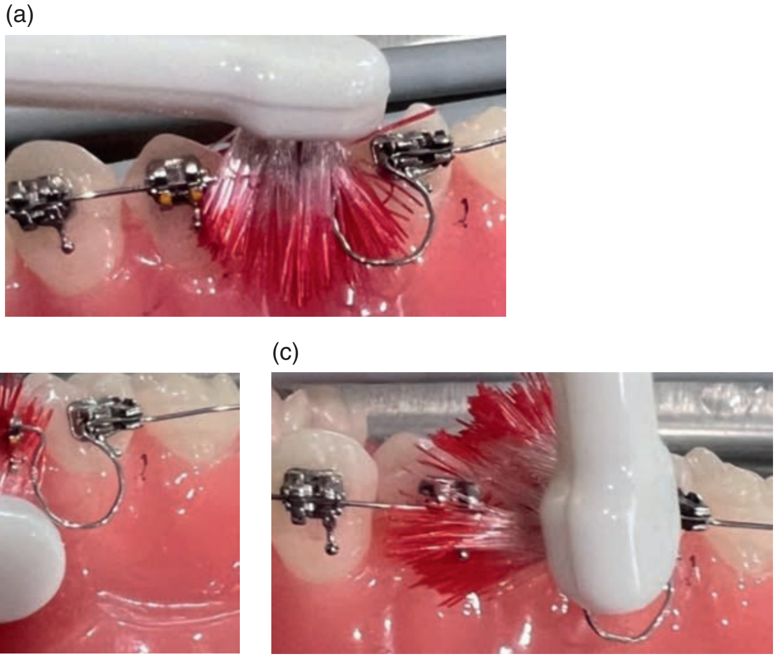

Figure 1.27 The effective debridement maintenance consists of three parts. (a) The standard end-tuft brush (ETB) is employed with an "enhanced Bass technique" to debride the occlusal third of the crown and occlusal edge of the bracket; (b) the ETB is positioned at the gingival margin with bristles directed occlusally to debride the gingival edge of the bracket, (c) the brush is held perpendicular to the bracket with the bristles jammed interproximally and into the gingival sulcus with a circular or scrubbing motion. These brushing motions contrary to some popular but erroneous advice should *not* be done gently. The motion should be *maximum comfortable force*, as would an athlete training, ultimately working up to 2–3 lbs.

Our method considers prior methods too effete to be effective with orthodontic patients. Our method aims not at removing plaque but rather acknowledging infection of the gingiva and attempting classical techniques of *wound debridement* and fortifying *stratum corneum*. With confidence and empirical evidence, this method not only relieves venous congestion and endotoxin pathoses but also stimulates orthokeratinization of the gingival margin, thus demonstrating superior outcomes compared to even the much-venerated Bass technique. The "enhanced Bass technique" is as follows:

First, an ETB bristles must be soaked in strong antiseptic. Then, it should be angled approximately 45° apically from the horizontal plane of the alveolus and pushed 1–2 mm into the gingival sulcus. This is done gently at first then incrementally stronger, as athletes gradually increase their training stressors. Ultimately, as the bleeding dissipates the gingival tone becomes more resilient. A vibrating or scrubbing motion flattens the bristles into the sulcus with 2–3 lbs. of force. Second, the brush in placed *coronal to the brackets* (Figure 1.27b) and 45° from the horizontal axis, with as much aggressive vibrational/scrubbing motion as needed to flatten the bristles (Figure 1.28) into the interproximal contact point. Third, flatten the brush perpendicular to the tooth surface to ensure the antiseptic-laden bristles reach deeply into interproximal areas and debride the gingival surface of the boded brackets (Figure 1.27c).

Debridement should be completed with a Platypus® floss holder.[13] This device should be carried with the patient and used habitually

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[13] Platypusco.com.

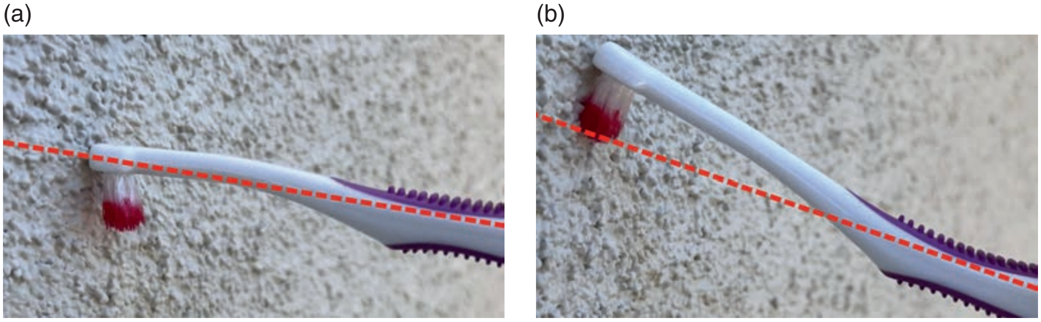

Figure 1.28 Properly loaded, the ETB (from Sunstar Americas, Inc. Schaumburg, IL, United States 60195) requires 2–3 lbs. of force for maximum efficiency. (a) Note how the long axis of the brush aligns with the *non-functional* end of the bristle when gentle force is used. (b) When therapeutic force is applied the force is transferred to the *functional* end of the brush. This is generally how hand instruments are designed, and why the ETB is so effective around orthodontic brackets.

many times throughout the day. The floss should also be pushed to the floor of the gingival sulcus with a coronal-apical/rotating motion (Figure 1.29). Other brands do not reach the bottom of the sulcus and thus provide only illusory protection.

Considering the keratinized potential of the masticatory mucosa, there is little to fear with aggressive brushing with the enhanced Bass technique described herein. Indeed, this mucosa *invites mechanical stimuli* to achieve the tonus and optimal fulfillment of its phenotypic potential.

Effective Oral Health Techniques

The need to keratinize masticatory tissue has been recognized for over 50 years (Weinmann and Meyer, 1959). Only aggressive mechanical stimulation can do this. The orthodontist is often put into the position of an athletic coach attempting to motivate a reluctant and slothful recruit. Repeating the valid notion of "infection," rather than the prosaic word "inflammation," will help. Indeed, this home care regimen creates a commensal niche and a healthy, resilient periodontal environment in 7–10 days. Any recession noted after achieving periodontal health is *not iatrogenic*. It is probably the disclosure of previously undiagnosed loss of attachment apparatus which was obscured by gingival edema.

A Heretical Dehiscence Theory

One dramatic complication has been placed at the feet of orthodontists unfairly. That is gingival recession (bony and soft tissue dehiscence). This damage is obvious and can be alarming. This is one example of a self-perpetuating bony pathosis, referred to in orthodontic parlance by the unfortunate term "runner." But this phenomenon is often mischaracterized as an ill-effect of orthodontic therapy which pushes a tooth outside of its "alveolar housing." This is a mistaken notion. Djeu *et al*. (2002) among others have shown that there is no correlation between specific types of OTM and gingival recession while others consider such movement a mucogingival threat. It is at least a contestable issue according to Professor Melsen. So, any discussion of corticotomy-like procedure involves the issue of mucogingival stress, bony dehiscence, and gingival recession.

An indeterminate variable may be operating in the studies of gingival recession and orthodontic biomechanics. This indeterminate is the quality of the bacterial biofilm accumulation. The biofilm can change qualitatively and harbor bacteria that release endotoxins. Endotoxins, also called lipopolysaccharide (LPS), are integral parts of the cell membranes in gram-negative bacteria. Upon cell lysis, they are released into the periodontium with very destructive effects mimicking a classic Arthus

Figure 1.29 Conducted properly this flossing method with a Platypus® Orthodontic Flosser (platypusco.com) combined with an enhanced Bass brushing technique, should allow any orthodontic patient to achieve a bleeding index score of 10% or less. The orthodontic flosser should be used assiduously in a complex rotational-coronal-apical oscillation *to the depth of the gingival sulcus* repeatedly during the day. And twice per day it should be used with an end-tufted brush. *Source:* Sunstar Americas, Inc.

reaction. One of the most pertinent effects of LPS is the *inhibition of fibroplasia*. As teeth move, the periodontal ligament moves with the tooth and the gingival unit sometimes everts to expose the gingival sulcus (Figure 1.30a–c).

This eversion, or prolapse, is recognized clinically as the so-called "The Red Patch of Atherton." Close examination of the gingival margin will disclose that it is often accompanied by exposure of the root surface (Figures 1.30a–c).

The gingival unit generally reestablishes its normal architecture when the tooth stops moving but that requires a concomitant fibrogenesis commensurate with the movement. And, in the case of an infected gingiva, this is not histologically possible. *LPS inhibits compensatory fibroplasia*. Periodontists have known this for nearly 50 years (Aleo *et al*., 1974). This recession phenomenon is complicated if chewing food traumatically injures a fragile and infected labial gingival margin. This causes a very fast "unzipping" or acute apical movement of the labial gingival margin, incapable of fibroplasia commensurate with tooth movement. This is what causes a sudden "runner." The author being trained in both periodontology and orthodontics has never experienced acute gingival recession in patients over 40 years of practice. This is because the author insists his patients achieve a gingival bleeding index of less than 10%. From a liability perspective, it should be mentioned at the beginning of

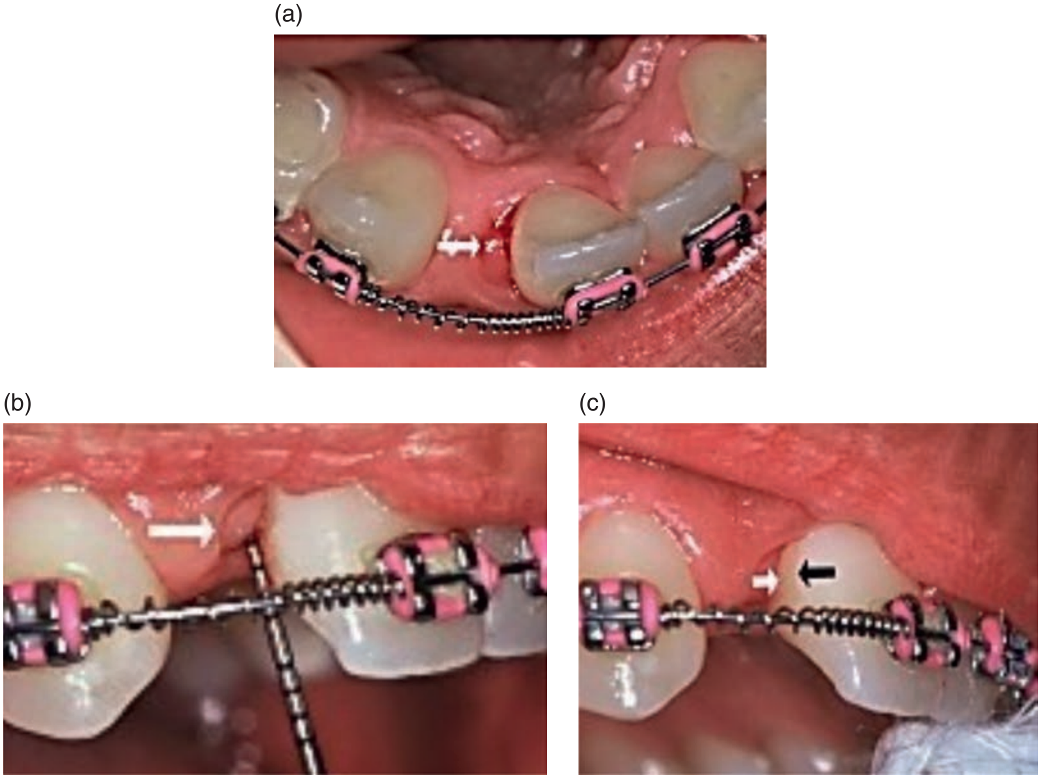

Figure 1.30 (a) The Red Patch of Atherton (arrow) represents a sulcular *eversion* or *prolapse*. It occurs when gingival regenerative capacity is not able to coordinate with needs of a moving tooth. (b) The distance between the tooth and the arrow is the *everted* or *prolapsed* lining of an erstwhile gingival sulcus. This tissue will, in healthy environments differentiate into keratinized epithelium and marginal gingiva. (c) The space between the white and black arrows is exposed root. If infection does not inhibit local fibroplasia the “lesion” is benign and reversible. This is an area of future attachment loss in the presence of infection. Biofilm endotoxins inhibit the compensatory fibrogenesis needed to accommodate the moving tooth.

treatment that bacteria can cause sudden “runners” and when the unfortunate event occurs patients will immediately know it was caused by their own noncompliance. Acute gingival recession in fixed-appliance cases is *not* caused by orthodontic activations. *Recession is caused by bacterial infections*. Orthodontists should no longer “wear a hair shirt” over this issue.

John Stuart Mill’s succinct analysis of causation (Churchill, 1990) is appropriate in this context. Although OTM may be, in some cases, a *necessary cause* (contributory factor) since it appears in fixed appliance therapy. But orthodontic movement is neither a *proximate cause* nor a *sine qua non*. Fact: orthodontic activation is not a *sufficient cause of recession*. The categorical assertion that “orthodontic tooth movement ‘causes’ gingival recession” is without compelling scientific basis and assumes the status of a clinical wives’ tale. The take-home message is this: practicing corticotomy-facilitated orthodontic therapy without some management and disclosure of the risk of infection is tantamount to practicing in a wooden structure without a fire extinguisher.

How Fast is “Fast”? – A 4-Day Treatment

The series of images in Figure 1.30a–f document a four-day alignment of lower anterior teeth that had relapsed in a slight Class II deep bite case. The patient was only available for care for only two weeks and demanded that the treatment employ accelerated orthodontic techniques.

The brackets were bonded to all mandibular anterior teeth and an .018” initial nickel-titanium round wire was engaged fully into the edgewise, Roth prescription brackets. A full-thickness labial flap was reflected to the mucogingival junction revealing the anterior labial bony cortex. This was perforated with punctate and linear decortication with a number two stainless steel irrigated bur. On the lingual surface only transmucosal perforations (TMP) were made deep into the medullary bone 4–8 mm. The labial flap was replaced to the cemento-enamel junction (CEJ) and secured with a braided silk 3–0 continuous suture.

The patient was told to return to the office as soon as the teeth were aligned to his satisfaction. The patient reported all the teeth were aligned in 4 days. The alignment was retained with a clear aligner and the patient was told to report to a local orthodontist when he returned to his home city.

This is not the only time the author has employed such rapid care with full biological and ethical impunity. Further the unique biological attributes of each patients suggests that some patients will need more time. But the 4-day regiment has actually been exceeded in other cases where complete alignment was achieved in one day using twice-daily adjustments in a previously prepared osteopenic bone.

Only time will tell if this rapid care, sans root resorption, pain, or periodontal attachment loss, will become *de rigueur* internationally. But this testimony demonstrates that engineering the alveolus bone prior to orthodontic tooth movement results in a kind of 21st century treatment for our patients which is faster, safer, and better than traditional 20th century care. (Figure 1.31).

Conclusions

This treatise purposely evokes controversial issues in a historical and philosophical context to give the thoughtful clinician pause for reflection in a meaningful dialectic. The synthesis, borne of controversy and its consequent explication of important nuance, continues today through the *Sturm und Drang* of daily practice. When confronted, it is the stuff of progress; when ignored, it invites intellectual corruption. That difference is decided by the earnest professional men and women, elevating daily observations and opinions to that higher level of intellectual abstraction where universal truth abides. What is heartening to any progressive orthodontist is that this moderate and collegial dissonance can deliver practical clinical outcomes and enriching professional insights.

With the artists’ intuitions and the scientist’s cold, redoubtable truths, it is the *clinician* as the front-line soldier who must sort out all the proverbial wheat from the sophistic chaff, all on behalf of the patient. And, as a clinical calling, all our intellectual imperatives must lie within legal (Garner, 2021) and ethical (Mouradian *et al.*, 1999) guidelines, compatible with an ever-changing culture. That challenge is our mission, our duty, and our privilege which only the devoted academic Man embraces.

It is incumbent on all students of dentofacial orthopedics to actualize their full potential if these innovative techniques are to be realized. As specialists, we need to continually strive for perfection knowing full well that we will never achieve it. But challenge is why good students become scientists. Art, in contrast, while endlessly enchanting, can all too often parade as pseudo-intellectualism. Our science must always reign sovereign over our art.

The artist’s mind runs free but undisciplined. Then, like a random walk, it ultimately leads us nowhere but where we started. Proffering an enriching grace to the human condition, the artists brush, drawn untrue over the face of youth is hard to erase. By contrast, the disciplined journey through the rigors of logical and scientific scrutiny leads to greater certitude, better health, and predictable success.

True, it cannot be denied that at times many clinical imperatives of scientific truth require an artistic embellishment. This is indeed

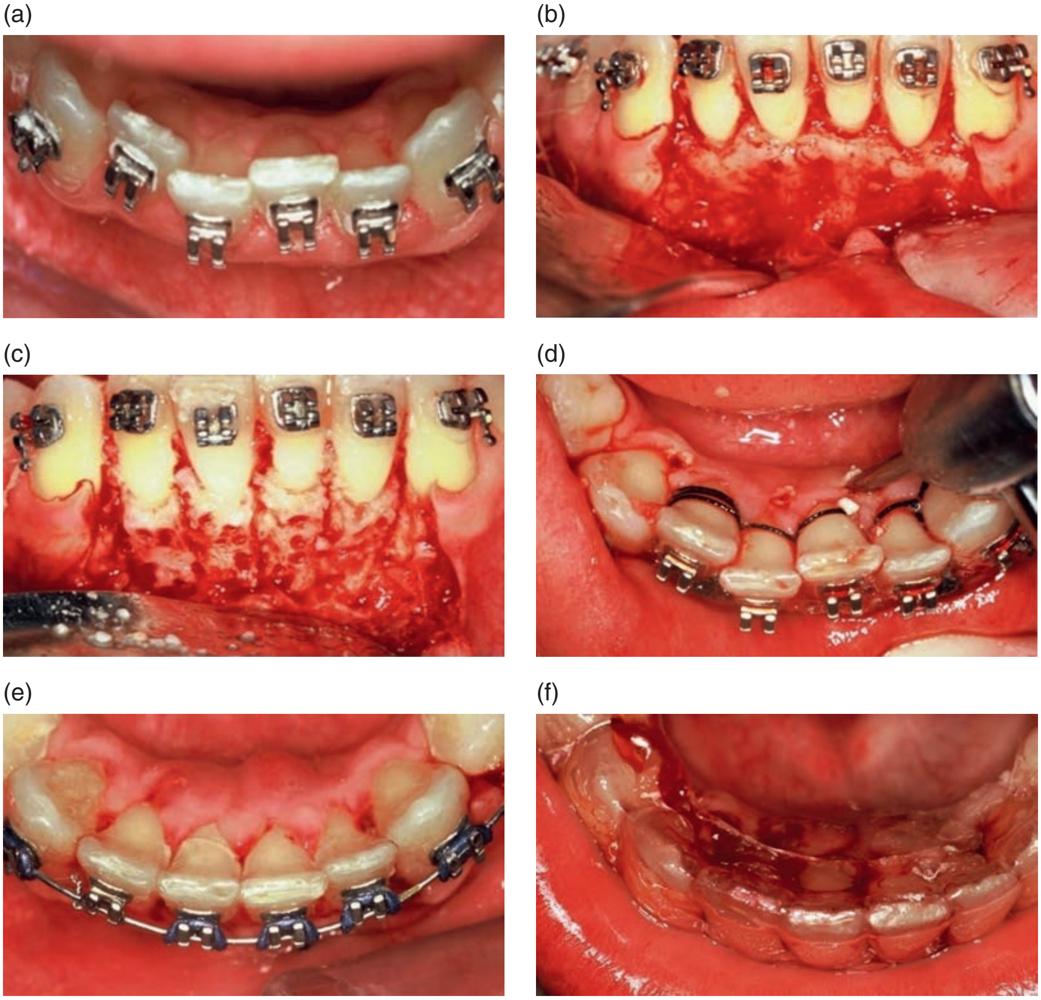

Figure 1.31 (a) This patient presented with moderate mandibular arch length deficiency (ALD) and only had 4 weeks in town for the treatment. The therapy was a standard selective alveolar decortication (SAD) because the surgeon palpated sufficient bone on the labial alveolus. The lingual alveolus was treated with TMP to avoid lingual flap reflection, a site of significant complications necessitating long-term follow-up. (b) The reflected mucoperiosteal flap revealed sufficient bone for selective alveolar decortication. (c) Clusters of hemorrhagic perforations were rendered under local anesthetic and without pharmaceutical sedation. Note how some perforations have crossed onto the alveolus osseous crest. This is a mistake but fortunately, the patient healed without gingival recession. This was due to his young age, 21. Had this been an older patient it is likely recession would have appeared as a sequela. For nonesthetic areas recession may have no clinical significance other than providing better embrasures to facilitate daily oral hygiene. In the esthetic zone, such violation of crestal alveolar bone would constitute a most unfortunate complication. (d) The labial flap is secured at its original cementoenamel junction with a continuous (sling) suture. This is 3-0 silk, but resorbable 2-0 suture is better because it saves time and discomfort upon removal. Lingual osteopenia is induced with a Number 2 copiously irrigated round bur for transmucosal perforation abbreviated TMP (Murphy, 2006). Slight penetration into the root surface can ensure ankylosis in cases of refractory relapse. The ankylosed lower incisors will form a stable template for maxillary arch stability. Contemporary standards may demand the use of sterile saline irrigation and an electrical torque handpiece. (e) Alignment of the mandibular incisors was achieved in 4 days. The patient was followed for 3 weeks to ensure maturation. (f) A vacuum-formed clear aligner, prepared with a cad–cam algorithm, can serve as an immediate retention device, but a bonded retainer is recommended. A 4-days treatment duration is not unusual for such a localized malocclusion. The limiting factor is wound maturation and suture removal. In the future, TMP would well suffice for cases like this to achieve the same clinical outcome, without the morbidity of labial flap reflection.

evident in the literature on orthodontic-driven corticotomy procedures. Still, it is within the firmament of science where fragile individual expressions of clinical art bloom under the aegis of predictable competence. Thus, in the grander schemes of the dental specialties, at the very frontiers of stem cell tissue design, we posit that all SFOT enrich and fortify our mission, in the dawn of oral tissue engineering.

Complementing the art of traditional biomechanics with OTE, is a clarion call to progress, not a distracting siren song. SFOT and all its subsequent iterations are destined neither to founder on the shoals of fatuous novelty nor fall ensnared in clinical disappointment. The history of tissue engineering shows starkly that the spirit of tissue management is not tethered to an obscure laboratory benchtop nor the exclusive domain of any one cloistered scientist.

The bridge between clinical orthopedics pioneered by Gavriil Abramovich Ilizarov, who improved the function of deformed legs, and the orthodontic tissue engineers, who improve facial esthetics, is a short one. And, it rests on a firm foundation of 21st-century science. Orthodontic alveolus tissue engineering employs the very same scientific principles employed by Professor Ilizarov. Enrichment of these ideas lies in the other chapters of this book, as our esteemed and resolute colleagues take us on a journey toward an exciting future. And that personal future – liberated from artistic dogma or the minions of corporate leviathans, untethered from a mindless morass of autocratic statists, and freed from oppressive pseudoscience – renders, a bountiful province for each of us to assiduously pursue a better path. The rhetorical question that this sojourn entreats is: "Will you?"

~ Quo Vadis? ~

Afterword for Academic Leaders

Twentieth-century orthodontic educators have the comfortable option of continuing the standard artistic model of orthodontic tissue dynamics, essentially 1901–1911 dogma. But mindlessly parroting historical habits is fraught with significant risks to clinical identity and patient safety. The fate of the new generation of orthodontists can lie within a more difficult but spectacular vision, one of biological engineering that transcends the venerable art of wire bending. On a practical level, traditional wire bending art, in the age of evidence-based dentistry, stem cell regeneration, and ex vivo organ farming, may fade into an interesting anachronism, as straight wire biomechanics becomes commoditized in the hands of less trained individuals. The orthodontic specialty is haunted by corporate usurpations, misappropriations, and commercial entrepreneurs who justify retail mediocrity as merely "good enough." While the naïvely trusting public rests securely in the belief that modernist devotion to excellence prevails, the hapless patient can drift into a morass of mediocrity, postmodern apostasies, and mega-corporate usurpations.

Tissue engineering, in contrast, protects excellence because it does not lend itself to commoditization. Therefore, new, earnest, and trusting orthodontists, heirs apparent, and champions of the specialty have an existential choice upon graduation. Will they surrender their historically inviolable authority to corporate minions, distributing a commodity of short-order smiles, with plebian artisans, who mass produce mediocre art? Or will they choose to become applied scientists, tissue engineers, thinking independently to bring the best science to each individual patient? The challengers to specialty excellence are not without merit; they serve a manifest need or social desire. But these competing institutions need austere leadership with neither animus nor capitulation. Excellence is what patients expect and integrity is what they desire. For children and other consuming novitiates, we are manifest fiduciaries.

As this brief history lesson has demonstrated, time carries us to new vistas often only dreamed about by previous generations. Sometimes, these vistas are presaged in other specialty literature, other intellectual disciplines, or ironically, even from nonacademic sources. It is only the dedicated scholar who will pick up such nuances. The future arrives at our doorstep whether we like it or not. The challenge to the professional educator is to separate transient fashion from epochal change and pursue a new course to better horizons.

Orthodontic-driven corticotomy procedures subsumed by the enduring promise of OTE are not going away. The pregnant question is whether curricular innovations will go along with it. Only faculty dedicated to individual quality and lofty idealism can make that happen. Defining new frontiers has always been the credo for the orthodontic specialty but that legacy often surrenders to stale bureaucratic inertia, tenured complacence, timid inaction, or, sadly, even subversive hostility. Progress will endure only by younger generation of orthodontists who wish to supplement the mantle of clinical artist with the science of dentofacial orthopedics. This "NewThink," like the existential choice of personal optimism, can define both the specific nature of each case and the specialty in general.

Unfortunately, for decades progressive orthodontists who understand where science is leading us have tolerated reactionary elements in the American orthodontic community engaging in political subterfuge that limited the acceptance of OTE by published innuendo, spurious threats of litigation, and solipsistic reasoning (Matthews 2013; Buschang 2012) that strained the bounds of rational skepticism. But a plethora of independently replicated studies over the last twenty years from international stalwarts has ignored American retrogression and documented the advanced science of OTE through a blossoming development that benefits orthodontists, patients, and timeless imperatives to achieve a noble excellence.

Fortunately for the science, American bias is weakening in the global community and the tide of academic apathy and recalcitrance seems to be ebbing. For example, in 2023 The World Federation of Orthodontists published guidelines for postgraduate orthodontic education (Ono 2023) that include both "accelerated orthodontic tooth movement and bone healing" and "periodontal and orthodontic interrelationships".

Thus, any further dismissal of these integral clinical sciences, it may be argued, represents a disregard for patient choice, emerging consensus, and scholastic excellence. So, in the end one must ask, what will the academic community do with these stunning new data that OTE pioneers have given us?

~ Quo Vadis ~

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