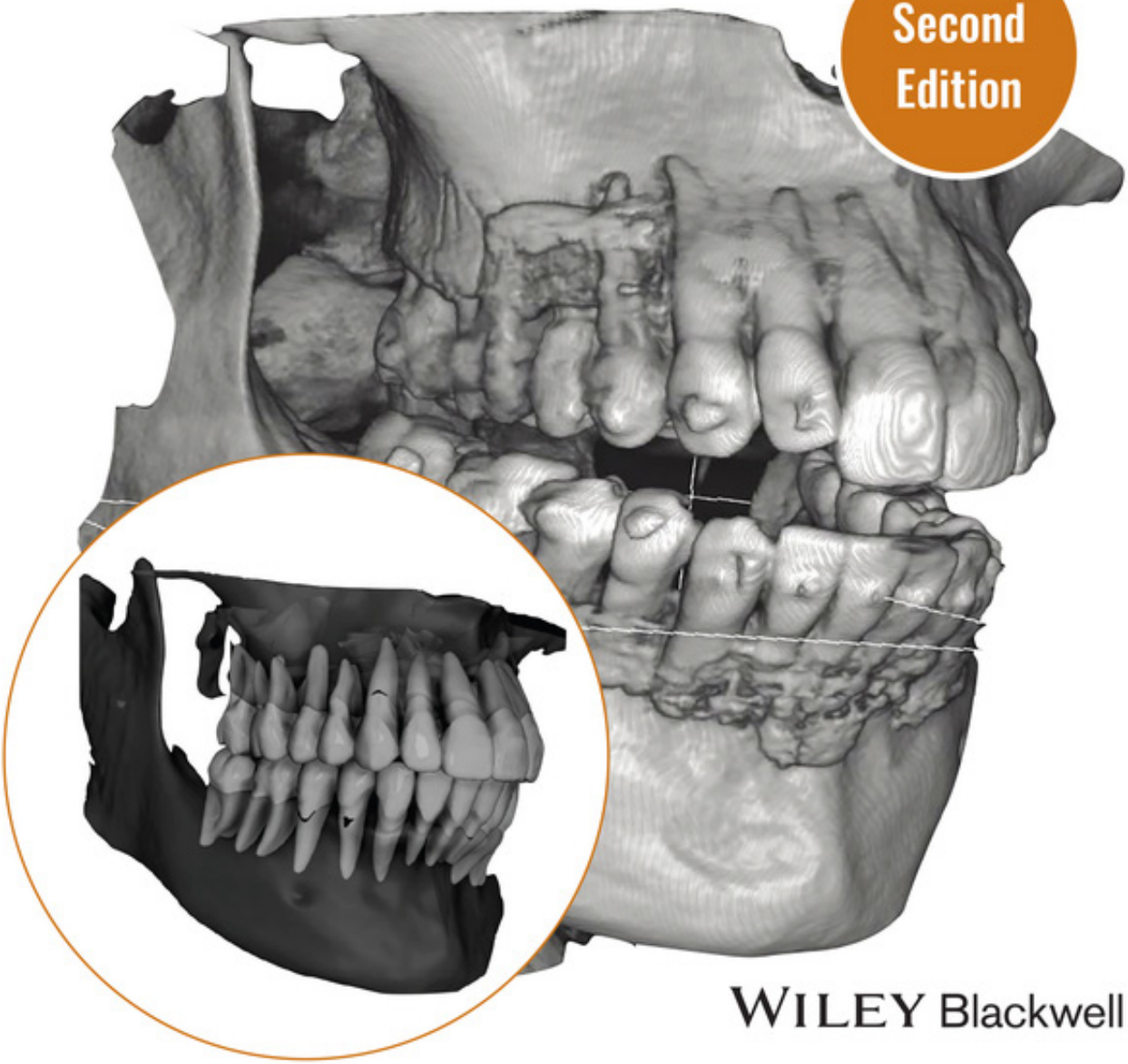
Orthodontically Driven Osteogenesis

Edited by Federico Brugnami
Alfonso Caiazzo • Simonetta Meuli

Second Edition



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Federico Brugnami
To Giulia, my daughter and center of gravity

Alfonso Caiazzo
To my parents, my inspiration

Simonetta Meuli
To my sons, Giovanni and Paolo

Contents

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Introduction

Improving orthodontic treatments by minimizing side effects and broadening their range has been a top priority for clinicians and researchers. Corticotomy, an established technique, has recently gained increased attention. However, confusion and misinterpretation surrounding the term, its application, and its benefits have led to global horizontal (pioneers worldwide) and low vertical diffusion (members of the same society). This underscores the need for a dedicated book on corticotomy or Periodontally Accelerated Osteogenic Orthodontics®. The introduction to the first edition, written 10 years ago, remains relevant today. Despite yearly evidence demonstrating the procedure's effectiveness and safety, orthodontic attention remains unchanged, shifting focus to 3D Digital Planning and Clear Aligners. The preferred terms used to describe the procedures have also evolved, with corticotomy-facilitated orthodontics (CFO), accelerated osteogenic orthodontics (AOO), and selective alveolar decortication gaining preference. Orthodontically driven osteogenesis (ODO), or the ability to grow new bone, is now favored over regenerative. The osteogenic potential, combined with bone grafting, can expand basal bone. This leads to two positive effects: less extraction of healthy premolars in growing patients and a more robust periodontium that prevents recessions during orthodontic movement. This concept initiates tissue engineering in orthodontics, where orthodontists and dental surgeons should define themselves as dentoalveolar orthopedists and embrace this new treatment philosophy. This approach maintains the promise of a faster orthodontic treatment and expands the limits of a safer one. It also offers a sound alternative to more invasive procedures, such as orthognathic procedures and can even modify the lower third of the face. This is impressive for a localized periodontal surgery when correctly combined with orthodontic treatment.

Orthodontically Driven Osteogenesis and 3D Digital Planning: New vs Innovation

As per the principles of innovation and change, "technology transfer" encompasses all activities that result in any user adoption of a new product or procedure. The term "new" here denotes any improvement over existing technologies or processes rather than a recent invention. Technology transfer necessitates active interaction between technology sponsors and users, which leads to actual innovation. It is crucial to differentiate between innovators or early adopters and late adopters. Innovators are individuals or groups willing to take risks by adopting new methods, products, or practices not widely used. They provide practical evidence that an innovation works, which is vital for later adopters.

Conversely, diffusion refers to the spread of an idea, method, practice, or product throughout a

social system. There are various kinds of technology transfer, including horizontal transfer (the movement of information on technology between innovators within an organization or between similar organizations) and vertical transfer (the movement of information on technology from innovators to late adopters of an organization or system of organizations).

ODO has been increasingly successful worldwide, resulting in extensive horizontal diffusion. However, paradoxically, the highest percentage of orthodontists in any developed country will either ignore or be skeptical of ODO and fail to present it to their patients as a viable and valuable alternative due to a lack of vertical diffusion. There are different reasons to explain this difference in diffusion, but it is important to note that natural resistance to change is just one factor.

1) The innovation is not disseminated. Given that the "innovation" is truly innovative, one of the most important driving factors is the economical one. For example, most of the innovations in dentistry in the last 30 years, from implants to membranes to clear aligners or straight wire, have been "encouraged" by suppliers and manufacturers. It is the same as in the pharmaceutical business: any revolutionary drug to cure a rare syndrome would suffer a difference in diffusion compared to Viagra® or Biposphonates or Statins.
2) The innovation is disseminated to the wrong people. The information is not referred to the proper person or somehow gets lost on the way.
3) The innovation is not understood by the potential user.

Most of the time, this is created by a superficial knowledge of the technique or misinterpretation, lack of homogenous terminology, and underestimation of potential benefits.

3D digital planning may be the correct driver for the diffusion of ODO: It becomes more difficult to ignore when the roots extend beyond the original bony envelope.

Federico Brugnami, Alfonso Caiazzo,
and Simonetta Meuli

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

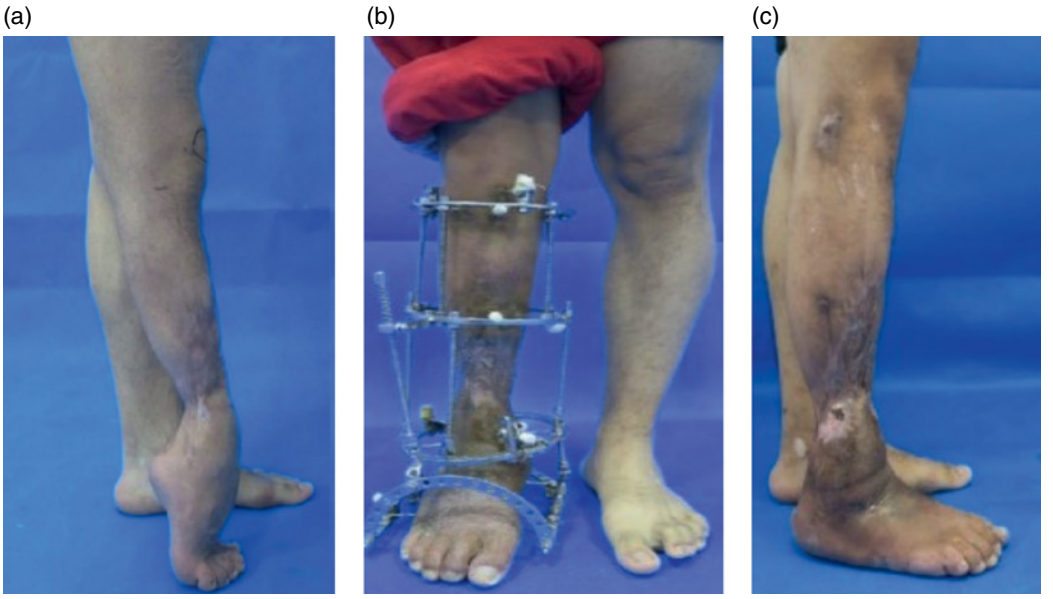

Figure 1.1 This treatment demonstrates what can be done to correct a deformed long bone. The same principle used by Gavriil Ilizarov can be applied to the alveolus bone in correcting dentoalveolar deformities. The leg deformity in (a) represents a deformed bone. (b) The Ilizarov orthopedic device with 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 checkups 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

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 re-instate 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

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,

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

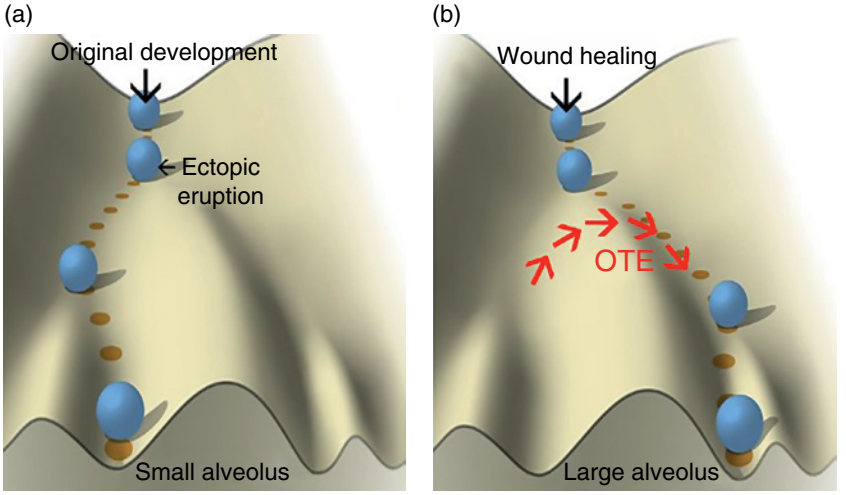

Figure 1.2 Phenotype development and recapitulating regional ontogeny in the alveolus bone is demonstrated, yet modified from Waddington's epigenetic landscape. The original landscape on the left is changed to demonstrate how SFOT and TMP can cause such an epigenetic perturbation that sufficient drive 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

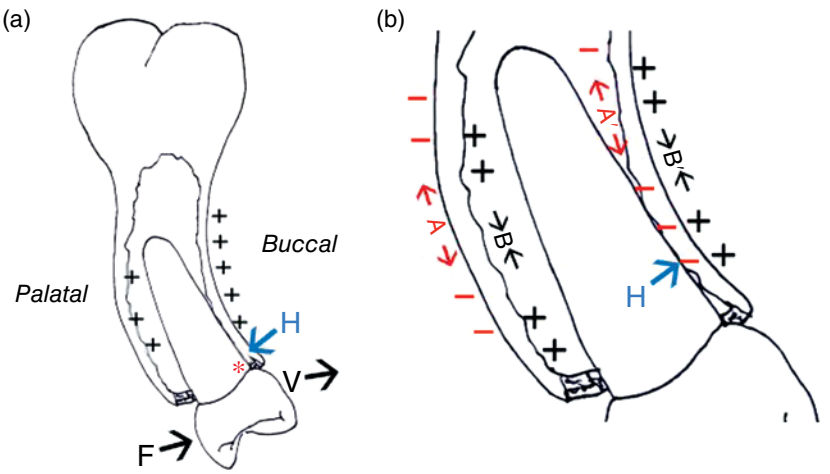

Figure 1.3 This figure is intended to demonstrate the entire load dissipation when a force is applied to a tooth. The traditional "pressure-tension" concept is so elementary that it is misleading and archaic. The figure demonstrates shear compression and shear tensions on the alveolus bone and explains 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