1 Corticotomy-facilitated orthodontics: Clarion call or siren song

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PREFACE

All Men by nature desire knowledge.
Aristotle

This chapter attempts to create an intellectual matrix within which other contributors writing about orthodontically driven corticotomies – also known as surgically facilitated orthodontic therapy (SFOT) – find both justification and inspiration with a modicum of practicality. The corticotomy, a selective alveolus decortication (SAD) of the alveolus bone, is but one in a family of related procedures encompassed by the inchoate field of SFOT. This treatise, by the very nature of the subject, focuses more on science than orthodontic art. And that science is orthodontic (bone) tissue engineering (OTE).

Yet, the emphasis on bone engineering during orthodontic tooth movement (OTM) promises much more than an alternative protocol or new clinical gadgetry. This chapter, in the context of an historical review, presents an evolution (and a clash) of ideas to reveal universal biologic principles. It is these principles, these transcendent truths, that should be applied to particular clinical events in a meaningful and rewarding manner. The student of SFOT should not indulge in mindless dedication to one technical recipe without understanding the specific biologic mechanisms and therapeutic objectives that define it.

The ideas and procedures of SFOT herein are increasingly being employed with great success worldwide despite the natural impediments of healthy skepticism and unintentional misrepresentation. Importantly, this global popularity is forging a new identity for those who wish to embrace it. Twenty-first-century orthodontists, periodontists and other surgeons are becoming

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international citizens endowed with skills and intellects of global scientists, forming a mastermind that is liberated from "brick and mortar", national, or even regional biases. We comment on that emergent event as doctors who have participated in a nascent science; we witnessed its birth, watched it develop, and remain ever fascinated by it. The contents of this book lend credence to that new identity and the authors personify the spirit of free inquiry that sustains it. Yet, in our zeal to share knowledge, we posit most humbly that we are merely the messengers.

INTRODUCTION

Conceptual issues

The title of this chapter is not a question; it is an existential choice. Because the history of the corticotomy presents thematic questions much more profound than where one should make surgical cuts, some explanation of this chapter's syntactical style is in order. The historical journey of the orthodontic specialty reflects a similar kind of thematic development replete with controversy. Throughout that rocky sojourn, two contentious themes have always emerged. 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 perspectives of the world. So conflict between these two worldviews is actually quite illusory. It is resolved only by realizing that arts and sciences are merely tools, intellectual instruments with which we achieve a nobler mission: our humanitarian endeavor of caring for others. Still, the two classic perspectives always prevail and must be constantly rebalanced: humanistic art as the ends, science as the means.

The second theme, a perennial conflict between extraction and non-extraction protocols, is more philosophically relevant to our topic. One of the great advantages of SFOT is that alveolar bone can be reshaped to accommodate an idealized dental arch rather than modifying a dentition to "inferior bone." An historical drag on this progressive trajectory is the assumption that the alveolus bone is immutable. It is not; the alveolus bone is remarkably malleable.

So, in a way, the new realization that the alveolus bone is malleable and the ability to "build a better bone" renders the extraction–expansion debate somewhat moot. With a "new biology" of orthodontics this historical debate has been rendered simplistic and false, just as epigenetics has rendered the nature–nurture debate an anachronistic dichotomy in the face of evolutionary sciences.

Our historical review cannot dictate where art ends and science begins in the mind of each orthodontist, for as every flower is beautiful, yet every flower is unique. And the sensitive orthodontist takes each individually unique "flower" to full bloom in its own season using both art and science. Likewise, one cannot dictate to every orthodontist exactly when extractions in particular should or should not be prescribed. One can only disclose a wider scope of therapeutic options, to achieve high-quality care. And, to many, quality is an event; namely, the coincidence of doctor talent with patient expectations in a universe of humanistic but rational achievement. This is the tacit mission of this textbook and its selfless contributors.

When dealing with facial esthetics the artistic imperative is undeniable and the decision to extract or not to extract reflects individual interpretations of timeless principles. Most art is intuitive. Yet even art – namely, impressions, culturally influenced in the aggregate and subjectively sensed in the individual – is not totally beyond the reach of scientific scrutiny. And, for better or worse, scientific scrutiny must always be the abiding companion of the 21st-century doctor. This is because contemporary practice, whether engaged with biological principles or indulged in psychosocial imperatives, operates in a postmodern world that demands demonstrable scientific proofs where we find them or (at least) compelling biological rationales where

we can divine them. History reveals the former and justifies the latter.

In this chapter, our methods are innovative, and admittedly somewhat polemical. We do not merely report a litany of events and experimental results. We cleave existing basic science to pertinent clinical data and synthesize them with traditional protocols. This hopefully will fortify what is done right by explaining it and provide alternatives to what can go wrong by explicating errors from their historical context.

When innovative science is seen through the lens of historical context, two important revelations occur. First, sophisticated insights of nuance are clarified (e.g., bony block movement versus enhanced physiology); and second, some new ideas are revealed simply as "old wine in new bottles" (normal healing, the regional acceleratory phenomenon – see 1983: Frost and his regional acceleratory phenomenon). This chapter will undoubtedly serve some old wine, but that insight does not diminish its worth. The historical context merely legitimizes the insights as more salient and timeless.

Through the gauntlets of criticism and the civil internecine bickering that often characterizes our specialties, it is curious indeed how truth emerges. Yet it is important to note that an assiduous intellectual analysis, emancipated from the strictures of dogma, and inspired by intrepid pioneers who have preceded us, is what sets the tone for this chapter and perhaps even the textbook itself. Query: is it nobler to suffer the indignities of dogmatic tradition or bear the yoke of exciting innovation? The former is safe, but the latter is tantalizing since it unravels the nettlesome enigmas of biology.

We must choose the latter despite the fact that unraveling mysteries is politically and philosophically risky when it exposes hues of uncomfortable truth. But the explication of truth is our deontological duty, because we have the power to control the welfare of other human beings, and that duty imposes a fiduciary standard more exalted than the "treatment to the norm (average)." Axiomatic to all clinical endeavors is the view that treating patients is a privilege, not a right, and a privileged position demands excellence, not mediocrity.

Dedication

John Donne reminds us that "no man is an island, entire of itself," and this chapter is a collaborative exemplar of that reality. Yet, the exciting frontiers of oral tissue engineering herein belong to neither our venerable teachers alone nor the seasoned clinicians who wrote this chapter. Rather, the future and our efforts are dedicated to those who will enjoy a longer tenure of equity in the specialty than we. This chapter is dedicated to them: the young idealists still seeking a place in the pantheon of clinical science.
Arete

THE NATURE OF THE PHENOMENON: INTUITED EARLY, DEFINED LATE

Diction and definitions

For the purposes of expediency and ease of reading, certain terms will depart their strict scientific definitions to be used in a liberal sense. The terms "corticotomy" and "SAD" will be used synonymously, and "mobilization" will be interchangeable with "luxation," meaning the physical jarring, fracturing, or cracking of bone. Surgically facilitated orthodontic therapy means any cutting of tissue that makes orthodontic treatment work better or faster. Other terms will submit to strict definition.

From osteotomy to corticotomy to tissue engineering

When reviewing the history of corticotomies one discovers that it originated in attempts to minimize the harsh side effects and risks of

segmental osteotomy. And this 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 of a section of bone the way one might break a twig from the branch of a tree. Thus, "mobilization" is a kind of purposeful fracturing of bone, sometimes literally done with a mallet and chisel to move physical parts, whereas a corticotomy is limited to the initial incision to modify physiology without luxation or fracture. When studying SFOT one must keep in mind the fundamental effects and esoteric mechanisms that facilitate the phenomena.

These effects, "observed" in the mind of the surgeon during the operation, occur subclinically at the tissue and cell levels. They are less clearly defined than clinic-level gross anatomy, a level to which most orthodontists are accustomed. Therefore, new modes of thinking must occur that could not have been appreciated by the specialty's earlier advocates. However, these histological mechanisms may have been singularly intuited by John Nutting Farrer (1839–1913) as early as 1888. He was referring to orthodontic tissue effects from a "whole alveolus bone" perspective when he wrote (emphasis added):

> 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.

Histophysiology of orthodontic-driven corticotomy

The "whole-bone perspective" is a new way of looking at alveolar bone reactions to orthodontic forces that goes beyond the narrow perspective of the periodontal ligament or a focus on the midpalatal sutures. This "NewThink" attempts to preclude retruded profile risks of extraction therapy. Williams and Murphy (2008) documented, with unequivocal biopsy images, that lingual forces can stimulate labial subperiosteal (compensatory) osteogenesis by showing samples of labial woven bone where the alveolus was expanded slowly from the lingual aspect. It should be noted, however, that any claim of permanent bone alteration with Williams and Murphy's appliances or surgical phenotype reengineering cannot be made before 3–4 years into the retention stage when the calcification is complete to an osseous "steady state" (dynamic equilibrium). Inherent in Williams and Murphy's philosophy is the assumption that emerging esthetic standards are shifting toward "full facial" esthetics quite different from the classic retruded profile of Apollo Belvedere (Angle's esthetic standard). This philosophy is not only compelling because of his biopsy evidence of alveolus development, but also because of its natural appeal to good common sense.[1]

The osteogenic effects demonstrated by Williams and Murphy (2008) in the alveolar subperiosteal cortices in nonsurgical cases capture exactly the histophysiology of corticotomy surgeries. Surgery simply elicits the phenomenon more dramatically and faster. The theoretical concept had been alluded to previously and was most recently expanded in the excellent textbook by Melsen (2012), where it refers to a "…change in surface curvature of the alveolar walls." All contemporary orthodontists should read this most enlightened summary of basic alveolar osteology to fully understand bone strain in all patients (Verna and Melsen, 2012). This "whole-bone" perspective posits the alveolus bone, cf. alveolar "process," as a separate operative organ independent of its subjacent corpus. As the whole bone is orthodontically bent, each osteon is deformed. The "peri-orthodontic hypothesis" (Murphy, 2006) contends that this bends protein molecules and DNA, opening obscure binding sites

on important molecules to elicit an epigenetic perturbation and redesigning the morphogenesis to a novel phenotype unique in alveolus in shape, mass, and volume. The value of this new perspective is that it conforms well with contemporary basic biological sciences, particularly molecular biology and epigenetics.

In this regard, alveolar subperiosteal tissue and periodontal ligament act no differently than the periosteum and endosteum respectively in any long bone (Figure 1.1).

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, we believe 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, without surgery, cannot overcome the natural tissue "canalization" that resists phenotype change (Waddington, 1957; Siegal and Bergman, 2002; Slack, 2002; Stearns, 2002).

Cell-level orthodontics

Bone cells, and homologues in other tissues as well, sense changes in their mechanical environments, internally throughout the cytoskeleton and externally through focal adhesions to the extracellular matrix (Benjamin and Hillen, 2003; Murphy 2006; Verna and Melsen, 2012). 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

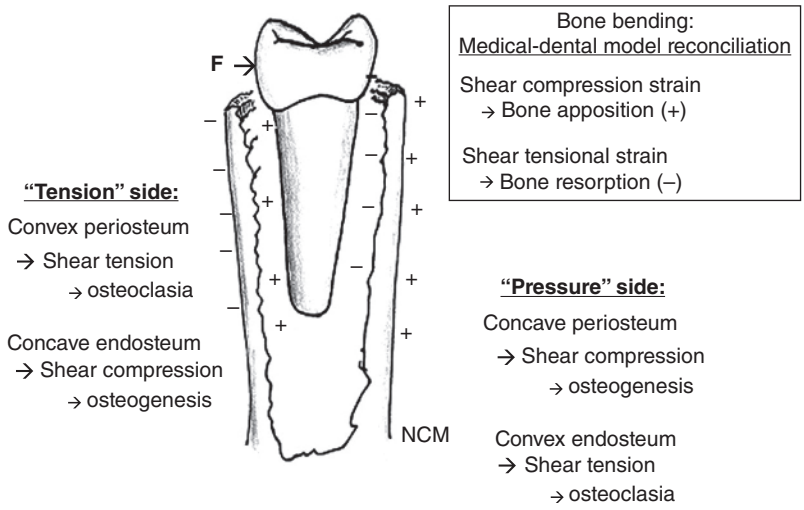


Figure 1.1 It is important to realize that SAD and particularly PAOO/AOO can change the configuration of the alveolus bone regardless of the form of the underlying maxillary or mandibular corpus. This apparently occurs by subperiosteal appositional osteogenesis stimulated by shear compression and shear tension on the facial and lingual cortices. In this respect, the periodontal ligament acts like endosteum in long bones. This realization resolves the ostensible conflict with the medical orthopedic claims that pressure is osteogenic and tension has osteoclastic effects, in contrast to the traditional orthodontic pressure–tension hypothesis that has been criticized so much in recent literature. This perspective does not deny that ischemic necrosis occurs in the periodontal ligament, but merely expands the biological concept of orthodontic histology beyond the ligament. This resorption on the so-called "pressure" side may be more related to osteogenic shear tension (–) in the cribriform plate similar to long bone homologues. On the so-called "tension" side, an increase in concavity of the cribriform plate is evident causing shear compression (+), which is osteoclastic in long bones.

the introduction of tissue engineering concepts and a revival of corticotomy-facilitated orthodontics, a new interest in cell- and tissue-level phenomena has appeared in the dawn of the 21st century.

Induced mechanical stimuli not only change the internal cytoskeleton but – by epigenetic perturbations – can determine internal stereobiochemistry and ultimate morphogenesis. To the extent that *wound healing recapitulates regional ontogeny*, orthodontic modulation of the healing bone wound can engineer a new phenotype ideally suited for an ideal dental alignment and dental arch juxtaposition even to the point of modifying the need for orthognathic surgery. The alveolus bone, which lives, thrives and dies by virtue of its functional matrix (Moss, 1997), the dental roots, are especially responsive to therapeutic intervention in this regard because of behavioral imperatives identified by Wolff's law and Frost's "mechanostat" model (Frost, 1983).

What skeletal muscle can do to bone morphogenesis at the gross anatomical level is similar to the effects of microstrain at the cell/tissue, whether that be hypertrophy, hyperplasia, or atrophy, thus demonstrating that engineering bone morphogenesis is a threshold phenomenon; that is, too much or too little is dysfunctional. It should be remembered that the influences of mechanical stimuli at the cell and tissue levels, mechanobiology, lie not only the domain of bone alone. Indeed, the even pathoses of atherosclerotic cardiovascular disease are directly related to mechanobiological changes in vessel walls. With modern analytic methodologies and a burgeoning body of science, too extensive for the scope of this writing,[2] responses to all tissue can now be studied and actually modulated, be they integumental or neuronal, mucosal or bony. This is the essence of tissue engineering science. Thus, orthodontic scientists have a legitimate equity claim in mechanobiological fathoms as well. So, there is no reason they should not be involved considering the critical importance of their domain, the face of a human child.

The study of cell/tissue-level orthodontic therapy, especially the nature of genetic expression evident in healing bone wounds, suggests that orthodontic relapse can be seen as a simple reversion to original phenotype, regardless of the method used. That is why some SFOT has been proven to be a popular – in some cases manifestly superior (Dosanjh *et al.*, 2006a,b; Nazarov *et al.*, 2006; Walker *et al.*, 2006a,b) – and professionally acceptable adjunct to traditional orthodontic therapy. The evidence of efficacy that this innovation enjoys lends both clinical quality and stability to OTM, justifying it as a reasonable therapeutic enhancement. There are advantages and disadvantages with both conventional OTM and SFOT, and it is only fair to patients that they be made aware of all treatment alternatives. At the clinical level, SAD is termed the Periodontally Accelerated Osteogenic Orthodontics™ (PAOO) technique or the Accelerated Osteogenic Orthodontics™ (AOO)[3] technique only when a bone graft is added, and these two terms can be used interchangeably. The lead author prefers to use PAOO when there is periodontal involvement and AOO when the periodontium is healthy.

Experience suggests that, in most cases, demineralized human bone graft or viable stem cell (allograft) therapy (SCT) should provide a predictable outcome. A non-surgical derivative, trans-mucosal perforation (TMP), (Murphy, 2006) can be employed when flap surgery is not indicated in small areas with excess bony support.

A HISTORY OF THE ORTHODONTIC-DRIVEN CORTICOTOMY (OVERVIEW)

Origin of the concept

Cano *et al.* (2012), as with other authors, generally attribute the first published surgical method to facilitate orthodontic therapy to Cunningham (around 1894) after his lecture in Chicago the previous year. While having

some rudimentary characteristics in common with modern corticotomies, close scrutiny of Cunningham's SFOT procedure suggests it was really a luxated segmental osteotomy. Cunningham's singular goal of making teeth move faster has since evolved to more global objectives, and variations on the corticotomy theme have spawned interesting incarnations throughout the 20th and 21st centuries in many different countries and cultures.

These variants evolved in a progression of surgical refinements designed to (a) accelerate OTM, (b) limit the quantity and pathologic potential of the inevitable bacterial load, (c) enhance stability, and (d) reduce the morbidity of orthognathic alternatives. As Cunningham's crude luxating osteotomy evolved, the term "corticotomy" emerged in the clinical lexicon with its approximate and more disciplined synonym, SAD. So both terms may be used as roughly synonymous for practical purposes.

But it should be noted that SAD with OTM will not grow new bone mass. In fact, in an adult, steady-state alveolus treatment may ostensibly slightly reduce alveolar bone mass. This is described in the non-surgical orthodontic literature as "moving bone out of the alveolar housing." So applying Cunningham's derivatives indiscriminately may indeed result in a net loss of supporting bone. This dilemma was solved by altering phenotype and creating additional bone *de novo* (Figure 1.2). Developing bone *de novo* has graced orthodontics exclusively through the prodigious efforts of many doctors in the Wilckodontics research groups, which are represented academically at Case Western Reserve University (Cleveland, OH, USA) by the second author. When grafted demineralized bone matrix (DBM)[4] (circa 1998) and viable cell allografts entered the SAD protocol, the thresholds of bone tissue engineering (Murphy, 2006) and SCT (Murphy *et al.*, 2012) were breached. This subsequently defined the dentoalveolar surgeon and orthodontist as partners in surgical dentoalveolar orthopedics and alveolar osteology.

New ideas often do not fit easily into old paradigms (Kuhn, 2012), so a new *Weltanschauung*,[5] (Freud, 1990) coined "NewThink", must be embraced to mark a clear distinction between the philosophy behind new orthodontic-driven corticotomy protocols (Pirsig's dynamic quality) and traditional orthodontic art of wire and plastic bending (Persig's static quality[6]).

The SFOT we describe here purposely executes OTM through a healing bone wound or bone graft eliciting a purposely delayed wound maturation. This occurs by perpetuating a natural bone "callus" or osteopenia until all the teeth are ideally aligned, coordinated, and detailed. This kind of surgery is decidedly *not* merely a variation of a basic surgical theme of the manner in "rearranging anatomical *parts*" like so many Lego® children's toys. Parts rearrangement is the stuff of orthognathic osteotomies. In stark contrast, the corticotomy-facilitated therapy does not create anatomical fragments or separate "parts." *Corticotomies re-engineer physiology.* Specifically SFOT, SAD, PAOO/AOO and TMP seek to *re-engineer* epigenetic potential in both the basic physiology of healing and ultimate morphogenesis at the molecular level of DNA and (endogenous and grafted) stem cells.

Early concepts: German pioneers

While Cunningham's procedure seemed bold to many American orthodontists, it soon became popular in the German scientific community. Cohn-Stock (1921), citing "Angle's method," removed the palatal bone near the maxillary teeth to facilitate retrusion of single or multiple teeth, and a host of German *Zahnartzen* followed his lead. Later, Skogsborg (1926) divided the interdental bone, with a procedure he called "septotomy," and a decade later Ascher (1947) published a similar procedure, claiming that it reduced treatment duration by 20–25%.

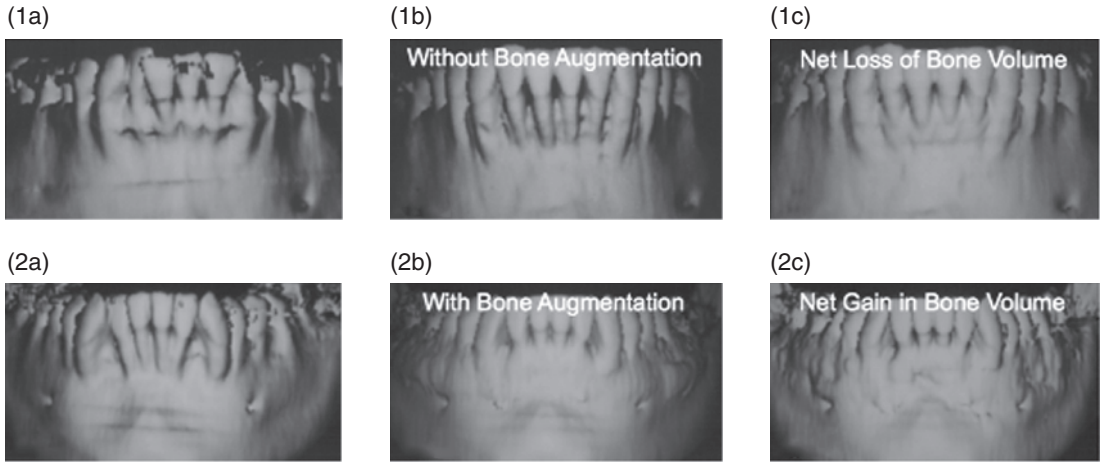


Figure 1.2 (1a) Comparison of SAD with (PAOO/AOO) and without a bone graft demonstrating the necessity for grafting when insufficient bony support is evident in adults. The figure shows the pre-treatment high-resolution computerized tomography (CT) scan (accurate to 0.2 mm) of the lower arch of a female, age 39, prior to having circumscribing corticotomy cuts performed both labially and lingually around the six lower anterior teeth. Note the arch length deficiency (overlap crowding), the pronounced crestal glabella, and the distance between the crest of the alveolus and the corresponding cemento-enamel junctions (CEJs). Clinically, the circumscribing corticotomy cuts resulted in the appearance of outlined "blocks of bone" connected by medullary bone. The total treatment time for this case was 4 months and 2 weeks with eight adjustments appointments. (1b) At 1 month retention the integrity of the outlined "blocks" of bone appears to have been completely lost and the layer of bone over the labial root surfaces appears to have vanished. In reality, this layer of bone has undergone demineralization as the result of a normal osteopenic state (RAP); the soft tissue matrix of the bone remains but is not visible radiographically. This is why radiographic assessments of expansion cases before 3–4 years in retention, while interesting in the short term, are premature for final policy conclusions. This demineralized matrix was carried into position with the root surfaces (bone matrix transportation). (1c) This shows the high-resolution CT scan at 2years and 8 months retention. Note that the layer of bone over the root surfaces has only partially reappeared due to the remineralization of the soft tissue matrix.

This suggests that there may have been a *net loss of bone volume* in this adult. In adolescents this is not seen. Owing to a greater regenerative potential there seems to be a complete regeneration of bone after SAD. (2a) This shows a high-resolution CT scan of the lower arch of a male, age 23, prior to circumscribing corticotomy cuts being performed both buccally and lingually around all of the lower teeth with a large bone graft placed over the corticotomized bone. Note the paucity of bone over the buccal root surfaces. The total treatment time was 6 months and 2 weeks with 12 adjustment appointments. (2b) At 3 months retention the labial root surfaces are now covered with an intact layer of newly engineered phenotype appropriate for the new position of tooth roots (the functional matrix of the bone).The pre-existing paucity of bone over the lingual root surfaces has been corrected in the same manner so that the roots of the teeth are now "sandwiched" between intact layers of bone both buccally and lingually. There has been a *net increase in bone volume*. (2c) At 2 years and 8 months retention the increase in the alveolar volume has been maintained. These data argue for PAOO/AOO in non-growing orthodontic patients where dental arch expansion is considered.

As good as it may appear, the scientific literature of the 20th century seems to have missed the central purpose of SFOT, SAD and PAOO/AOO, and TMP. This illustrates a social phenomenon where older, more experienced but doctrinaire, clinicians see innovation not in the context it promises, but rather in the context of the *status quo*. This is an unfortunate but common event seen best in retrospect. This bias and the lack of modern biological standards is the reason why some literature of this period is merely anecdotal, dismissive and often

patently incorrect. Yet, ironically, this body of data is still cited as authoritative and used to justify specious criticisms of 21st-century dentoalveolar surgery.

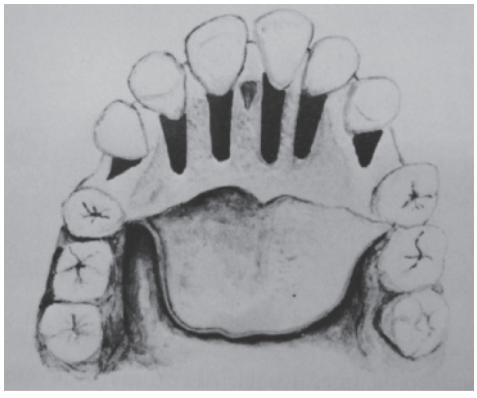
1931: Bichlmayr's breakthrough – A wedge resection

After Cunningham's osteotomy publication, a series of German papers sustained the notion that outpatient surgery could be beneficial to orthodontic patients. Ironically, the original presentations of rudimentary SFOT at the turn of the 20th century languished in dusty journals and were not widely discussed in America for over half a century. However, the concepts blossomed in Europe.

Then, just before World War II, Bichlmayr (1931) described a very practical surgical procedure for patients older than 16 years to accelerate tooth movement and reduce relapse of maxillary protrusion. This was employed with canine retraction and first bicuspid extraction, by "excoriating" cortical plates of the palatal and crestal alveolus, and cortices of the extraction sites. Later, Neumann (1955), who divided the inter-radicular bone and ablated a wedge of bone palatal to the incisors meant to be retracted, would be most laudatory of Bichlmayr's work. But this praise was to be proven faint.

Important to note is that Bichlmayr probably excised significant amounts of medullary bone with his procedure. He redefined orthognathic surgery by reclassifying it into two categories: "major" (total or segmental maxillary and mandibular correction) or "minor" (interdental osteotomy or corticotomy), and was the first to described the corticotomy procedure to close diastemata (Figure 1.3). Bichlmayr's extensive wedge-shaped bone resection (*Keilfoermige Resektion*) was more extensive than the punctate and linear patterns presently employed. The latter seem more discrete and somewhat sophisticated, but the fundamentals of induced osteopenia and recalcification in retention are the same. If protracted decalcification is desired or

(a)



(b)

Figure 1.3 (a) Bichlmayr's representation of palatal and distal decortication. Shaded wedge-shaped areas diagram a wedge-resection (*Keilfoermige Resektion*) of alveolar cortices and probably significant medullary bone. (b) Buccal areas subjected to posterior alveolus expansion. In view of more modern revelations about the physiology of SAD it seems that Bichlmayr's aggressiveness, though done with clinical impunity, may have been somewhat superfluous. Considering the concern for buccal dehiscence and gingival recession associated with posterior dental arch expansion, a bone graft buccal to Bichlmayr's posterior corticotomies (b) may be prudent for non-growing patients.

if the degree of tooth movement is onerous (e.g., mass posterior bodily vectors), then Bichlmayr's extensive decortication is appropriate.

However, in the context of movement alone, if a simple labial tipping of mandibular incisors is desired, then a more conservative procedure, even as minor as TMPs, may work where sufficient alveolar bone is present. However, this should not be done where there is a question of bony support. Bone paucity dictates that PAOO/AOO is clearly indicated. This is why Bichlmayr's procedure is limited; not every oral site has the abundant bone of the maxillary palate. It is this wide spectrum of procedures that are presently defining the nascent clinical subspecialty of OTE.

The discerning principle of orthodontic-driven corticotomy procedures is this: the degree and duration of the necessary osteopenia is directly commensurate with the degree of induced surgical trauma, and proportional to the amount of bone density through which the teeth are moved; that is, a lot of denser bone means more decortication is needed for long tooth movement distance. It takes just as long for five 2 mm linear decortications to heal as it does two 5 mm linear decortications. Given that the comparative healing time is the same, where the sufficiency of SAD is in doubt there is little justification for timid decortication. Later, near mid-century, one can better appreciate Bichlmayr's contribution to 20th-century orthodontics with the publication of Köle (1959), who derived his work from many previous German publications, but particularly that of Bichlmayr.

1959: Köle's American debut

The seminal American work belongs to Köle because he wrote the first English-language paper describing a practical decortication of the alveolus bone to facilitate OTM. With some notable refinements, this is the basic technique that is employed today by those who promote the integration of orthodontic therapy and periodontal surgery. The Köle surgery was limited to the cortex of the dental alveolus, but subapical decortication was embellished by extending buccal and lingual cuts into the spongiosa until they communicated through the subapical medullary bone. Bucco-lingual communication is now considered unnecessarily morbid and eschewed by later SAD and PAOO/AOO protocols.

When Köle popularized corticotomy in the English literature he also promoted the so-called "bony block" hypothesis, an explanation later abandoned as the underlying physiology of SFOT became more clearly defined. He also reported buccal corticotomy in posterior inferior sectors to correct molar linguo-version and facilitate orthodontic expansion. Here, we see an emergence of physiology engineering theory as a conceptual replacement for the mechanical rearrangement models. He relied on the reduction of cortical resistance and tried to preserve the vascular supply from the trabecular bone to the teeth. Some years later this vascular issue was the focus of criticism by Bell and Levy (Verna and Melsen, 2012).

Special consideration should be made when studying this milestone contribution of Dr Köle in 1959. Where Bichlmayr indicated that he was essentially just making room for the roots by removing a physical impediment, Köle emphasized a subtle but key element. He left a thin layer of bone over the root surface in the direction of the intended tooth movement. This aspect of the root, the surface exposed to the resistance of tissue or bone, is called the root "enface" (Murphy *et al.*, 1982). This is the area of microanatomy that we think travels with the root as a kind of "demineralized root–bone matrix transportation" tissue complex.

Köle's methods were less ablative than Bichlmayr's except for through-and-through osteotomies (cuts) made apical to the root tips of the teeth and next to the extraction sites. Köle's observations led him to surmise that the roots of the teeth were not moving through the bone, but rather the bone was moving *with* the roots of the teeth. With his technique Köle claimed to be able to complete most major movements in

adults in 6–12 weeks. To illustrate how far Köle's concept has traveled, the modern incarnation of this idea can tip lower incisor teeth labially out of crowding in 4 days (Figure 1.4).

Unfortunately, Köle's interpretation of the mineralized medullary (spongiosa) bone moving with the roots of the teeth was incorrect. But in 1959 the correct scientific explanation for

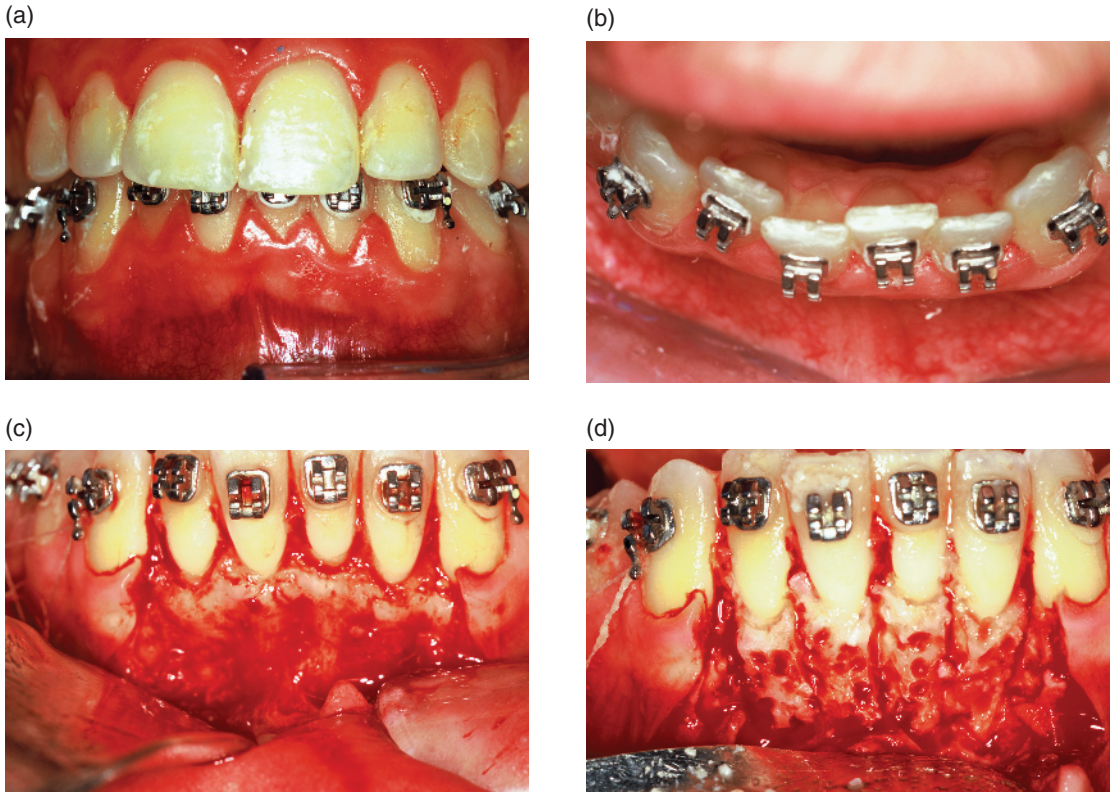


Figure 1.4 The contemporary method of SAD is represented in this 23-year-old Caucasian male college student. (a) His clinical appearance, where his orthodontic treatment relapsed after losing his retainer. He presented with a chief complaint of "crooked teeth." Oral examination revealed an Angle's class I occlusion with ostensible lower incisor arch length deficiency. This is a misinterpretation, because the so-called "crowding" is actually "pseudo-crowding" due to a return of the deep bite and the restriction of space for lower incisors as they approach the maxillary incisors' cervical lingual surface. Note the relapsing dental deep bite, a musculo-skeletal phenomenon.

The patient needed treatment to be completed within a month. A full-thickness mucoperiosteal flap (c) was reflected and linear and punctate decortications (d) on the abundant labial cortex were combined with lingual TMP (trans-mucosal perforations) (e) to elicit a regional acceleratory phenomenon (RAP). A lingual hemi-circumferential fiberotomy (CSF) completed the surgical treatment. Minor interproximal enamel reduction was performed and a week later the patient presented with lower incisors well aligned (f). The treatment was maintained with a vacuum-formed plastic retainer (g). The patient reported that, "The teeth were straight in 4 days but I had to wait a week for an appointment." Such rapid tooth movement is not uncommon when simple labial tipping is facilitated by interproximal enamel reduction (stripping) and a corticotomy.

This case illustrates how a panoply of SAD procedures (PAOO/AOO, TMP, CSF, etc.) can be employed according to each individual's needs and desires. Corticotomies cannot be legitimately rendered without specific characterization of each patient's needs and preferences, an individual imperative often lost among policy-level autocrats, doctrinaire ideologues, and corporate utilization review committees who are unduly enamored by Gaussian means.

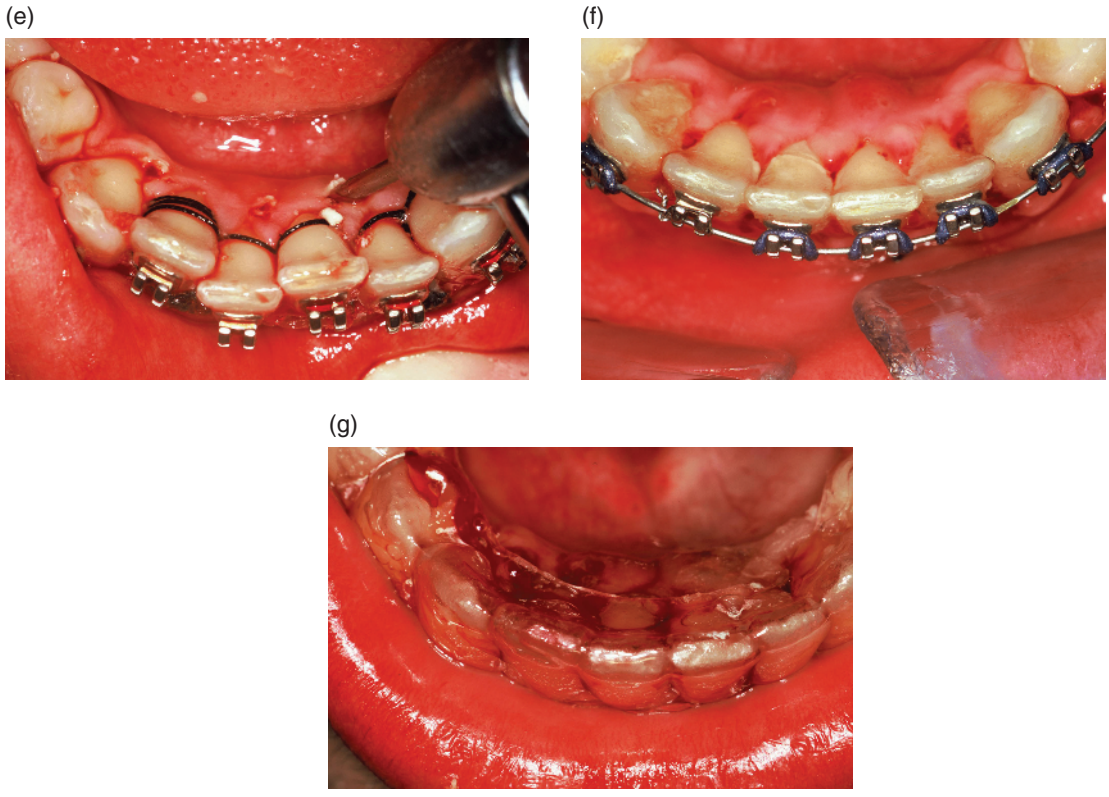


Figure 1.4 (*Continued*)

what was occurring subsequent to surgery did not yet exist. Unfortunately, this dissociation between Köle's clinical perspective and scientific reality led to confusion and contradictory rationales for over three decades. As a result of this misconception, corticotomy-based surgeries evolved mostly into buccal and lingual cuts circumscribing the entire root with little regard for the facilitating physiology. Although Köle removed the buccal and lingual plates of bone at extraction sites, there was little evidence he fathomed the principle element in altering OTM. What is critical is this: bone volume must be reduced adjacent to the root surface at the root enface; that is, *in the direction the tooth is moving*. This was not emphasized for many decades, and thus stigmatized SFOT until this misconception was cleared up by the computerized tomography (CT) research by Professor Wilcko. Additionally, research at Loma Linda University began to bring into question the long-held notion of "bony block" movement (Anholm *et al.*, 1986; Hoff, 1986; Tetz, 1986; Ryenarson, 1987; Gantes *et al.*, 1990; Khng, 1993).

Compared with the more aggressive osteotomy protocols at the time, there was relatively little morbidity in Bichlmayr's and Köle's surgeries because luxation was not employed and the blood supply was not jeopardized. For those who subscribed to "mobilization by luxation," their patients will generally suffer more, with little or no added value. Although it would certainly be preferable to have thicker layers of bone encasing the roots of the teeth in retention, this comes later when bone grafts (PAOO/AOO) were added to corticotomy protocols.

Eventually, advances in the basic sciences would help to explain the lingering confusion and give more credence to the work of both Köle and Bichlmayr. But that would not occur until 1983 and did not reach most of the American dental profession until 2001.

1931–1965: moving parts or modulating physiology?

Kretz (1931), a contemporary of Bichlmayr, described a procedure similar to Cunningham's, creating, in effect, a therapeutic fracture of the anterior alveolus. His aggressive manipulation of bone and preoccupation with the mechanical movement of "parts" continued the kind of "Newtonian bias" orthodontics as a pure mechanical art that still haunts SFOT today and eclipses any appreciation of how physiologic alterations may be modulated. The naiveté of the surgeons using these procedures belies the insights into wound-healing sciences. And this bias, to fracture bone and then rearrange it, is evident even in Reichenbach's (1965) contribution over three decades later, which also expressed concern for iatrogenic periodontal damage.

Regarding this concern, one must certainly be adroit with a scalpel to avoid any ancillary periodontal damage. However, the benefits of SAD, PAOO/AOO, and TMP, for anyone who understands the pathogenesis of periodontitis, far outweigh any perceived risks. A number of cases over the last two decades have demonstrated that, aside from occult endodontic infection, periodontitis is not an absolute contraindication to SFOT. After flap reflection and SAD, a debrided lesion at the grafting site is no more unhealthy than that of a young child. In fact, the tissue surrounding such a debrided lesion is surfeit with a regenerative physiology and growth factors that actually facilitate phenotype changes and stem cell osteogenesis. Therefore, no resective periodontal surgery or initial therapy needs to be performed as a separate procedure before SFOT because the latter subsumes the former as long as the roots of the teeth are fully debrided and general principles of periodontal therapy are respected.

Although seemingly naive in periodontal science, Reichenbach was very wise in concern for alveolar blood supply, because the aggressive surgery described in the article certainly poses risks. In contrast, the very purpose of a refined SAD technique is to minimize that risk and maintain as much bone vitality as possible. This is done by limiting the surgical intervention to the cortex only; this *enhances* vascularity from the spongiosa. These simple caveats will ensure that Reichenbach's concerns are honored. Unfortunately, without an empirical basis derived from controlled clinical studies, laboratory data, or conceptual sophistication (epigenetics) available, Reichenbach's misapprehensions tend to be perpetuated.

1983: Frost and his regional acceleratory phenomenon

In the late 20th-century medical orthopedic literature a new concept was emerging that would ultimately be wed to the corticotomy rationale. Frost (1983) began to educate clinicians about two biological events seen with long bone fractures, which he called the systemic phenomenon and (what concerns us) the Regional Acceleratory Phenomena, RAP. This localized trauma-induced essentially a localized trauma-induced transient osteopenia was touted as an original insight and the definitive operational entity in bone fracture healing. The concept is largely attributed to Frost, despite the fact that Kolář *et al.* (1965) introduced many RAP effects as early as 1965.

Frost elaborated on the concept as an acceleration of the multiple stages of natural wound healing in the long bones. He explained that RAP begins within a couple of days of the fracture (osseous wounding), usually peaks at 1–2 months, and may take 6–24 months to subside. In SFOT, OTM and TMPs can perpetuate the osteopenic state beyond 2 months. RAP

provides for a dramatic acceleration in bone turnover and, in earlier stages, an increase in the number of osteoclasts that produce a significant, but transient, osteopenia, a sort of benign osteoporosis. This is a condition similar to what would be observed in hyper-parathyroidism or a simple fracture, but in the case of selective decortication (SAD) no abnormal metabolism is involved and the effect is both transient and therapeutic.

LATE MODERN PERSPECTIVES (1959–2000): CONSTRUCTIVE CONTROVERSY

1972: Bell and Levy

Bell and Levy (1972), in keeping with the *Volkgeist* of the times, published a more cynical interpretation of SFOT. Their iconoclastic approach to certain salient issues is the kind of controversy that vexes the neophyte surgeon and orthodontic students but ultimately, by intellectual conflict, results in synthesis and clarity in scientific thought. Theirs was one of many articles in about one decade that provide depth to the subject but also muddied the water by conflating simple corticotomies with osteotomy-like protocols.

This conflation obscured the important distinction between superficial cuts into the cortex of bone and deep cuts further into the spongiosa. Deep cuts indeed pose a greater risk of compromising blood supply according to animal studies, but luxated alveolar segments pose a more serious threat to the patient. In the later half of the 20th century it was unclear how deep the cuts needed to be in order to facilitate OTM and whether luxation (mobilization) was really necessary. Mobilization is a vague and variable term used as a synonym of luxation, the forcible loosening of the dentoalveolar unit by controlled fracture of the bone. The question mobilization begs is this: is mobilization necessary to facilitate tooth movement? As we will see, the answer is probably not.

The Bell and Levy work was the first experimental animal study of 4 *Macaca mulatta* monkeys, but they used the term corticotomy as a misnomer. They described a model of simultaneous mucoperiosteal flap reflection, and interdental corticotomies with *mobilization* of all "dento-osseous segments" which, they claimed, markedly compromised the blood supply to the anterior teeth. Specifically, they described "… immediate repositioning of one- and two-tooth dento-osseous segments." They ablated excessive interradicular bone with a fissure bur, noting (emphasis added) that it should be done "…after the dental-osseous segments have been *made freely movable with an osteotome*" and warn that "Great care must be exercised to maintain a palatal soft tissue pedicle to the *mobilized segments*…"

Their histological study showed the risk of this type of procedure (full mucoperiosteal detachment plus deep cutting of medullar bone) to the vascularity of a dental pulp and surrounding medullar bone. They demonstrated distinct avascular zones that progressively recovered 3 weeks after surgery, except for the central incisor.

Referencing Köle, the authors reiterated that "…dentoalveolar segments can be readily moved bodily…" It is important to note that this "bony block" perspective is exactly the kind of misinterpretation that confuses sophisticated corticotomy with luxated osteotomies. The prejudice is noted further in the Bell and Levy article when the authors claim the rationale for "corticotomy" is vague and that circulation based on "pedicled cortical bone to a relatively small amount of spongy alveolar bone would *presumably* imperil circulation to the mobilized dental segment" (emphasis added).

Bell and Levy express legitimate concerns for safety with the mobilized osteotomies, but

transferring that concern for corticotomies is illogical and untenable. It is ironic that the authors also claim that, theoretically, the corticotomy could "… have a destructive effect on the periodontium." By its very definition the corticotomy part of their protocol refers to the *cortex*, and only an indiscrete surgeon could jeopardize the periodontal anatomy. Even where inadvertent periodontal tissues are breached, the regenerative potential of the organ – in the absence of infection – is robustly regenerative. In the modern definition of corticotomy the periodontal anatomy is explicitly excluded for SAD, PAOO/AOO, and TMP. When thus protected it is precisely the root–periodontal ligament–demineralized cribriform plate complex that seems to move, rendering luxation and mobilization absolutely unnecessary. Bell and Levy, it seems, criticized a clinical straw man.

What is actually surprising in their article is the degree of normal healing given the ablative nature of their surgery. They note, "postoperative healing in all animals was uncomplicated." Histological evaluation demonstrated avascular zones at the site of decortications, but all the other vasculature maintained its normal course. The authors claimed that the roots were "traumatized" but failed to elaborate on the specific sense of the term. All surgeries "traumatize" to some degree, and with corticotomies that is precisely the therapeutic objective; that is, controlled therapeutic "trauma." Technically and biologically speaking, a therapeutic "trauma" is the same physical mechanism that normally strengthens muscle tone in weightlifters and elicits healing in wound debridement.

At 3 weeks the bony cuts showed noticeable evidence of repair. This observation gives evidence of normal clinical behavior. It is significant because with any OTM, a 2–3-week post-surgical latent period is encountered after which the OTM is accelerated. By 9 weeks the authors noted "complete healing in the posterior corticotomy sites." In their discussion the surgeons make a most revealing observation about the cause of "excessive tissue damage". They stated,

> ….immediate mobilization of each dental alveolar segment probably compounded the injury by producing complete interdental osteotomies in certain areas.

We would contend that the mobilization of the segments was not merely a compounding factor but indeed the *sine qua non* of any serious injury and that SAD *per se* will produce normal tissue healing after measured precise, discretely targeted, and conservative cuts limited to bone cortices. This contention is based largely on the work of later research (Twaddle, 2001; Fulk, 2002; Hajji, 2002; Machado, 2002a,b; Kasewicz *et al.*, 2004; Skountrianos *et al.*, 2004; Ahlawat *et al.*, 2006; Dosanjh *et al.*, 2006a,b; Nazarov *et al.*, 2006; Oliveira *et al.*, 2006; Walker *et al.*, 2006a,b), which investigated both the clinical behavior and the tissue science in much greater depth. Particularly impressive in this paroxysm of scholastic excellence was the study of the rat model (Ferguson *et al.*, 2006; Sebaoun *et al.*, 2006, 2008), an even more difficult specimen to work with than the rhesus monkey. These researchers showed histological sections that clearly demonstrate normal tissue changes with corticotomy without luxation or deep cuts into the spongiosa.

Bell and Levy, from a clinical perspective, may indeed have genuinely worried about irreversible damage. That is understandable and laudable. However, they did not document permanent damage; and since ischemia or any kind of normal healing can be seen as temporarily "destructive," it appears that the authors also committed a logical error common to many clinical investigators. They pathologized normal variations. Without replicable empirical data, fully displayed, clinical impressions, for better or worse, must be taken as less compelling than "hard science."

Wherever this error is committed it overstates authors' cases and undermines their credibility. The wise writer knows that very few universals can be legitimately defended in clinical biology. It seems that the Bell and Levy article merely achieved a consecration of bias and documentation that any overly aggressive surgery is risky. To their credit, however, the authors discussed the thoughtful idea of a two-staged procedure reflecting the palatal flap and labial flaps 5 weeks apart. Moreover, it must be acknowledged that Bell and Levy made a significant contribution about how alveolar bone heals, despite their vague and confusing overstatements about what should be done.

1975: Düker's redemption

After, Bell and Levy's article demonstrated luxation and questioned whether the risk was worth the benefit, other exceptional articles on corticotomy were published that presented a solution to this quandary with lucid simplicity. Düker (1975) replicated Köle's work more exactly in dogs, moved an incisor segment 4 mm in 8–20 days, and concluded that neither the periodontal attachment nor the pulps of teeth demonstrated significant injury. In fact, he stated correctly but vaguely that, "…weakening in the bone by surgery and consequent orthodontic treatment reduces these dangers."

The authors seem to have grasped the central concept of SFOT but expressed the rationale too ineloquently for immediate implementation. This limited scope still haunts the literature today, since many commentators, often uninitiated, feel a surgically induced and transient osteopenia is too effete and temporary for practical use. This myopic perspective misapprehends a critical central element of SFOT; that is, to induce constant internal bone strain by efficient, judicious tooth movement designed to perpetuate the osteopenia state.

Düker published his elegant study of the corticotomy in six male beagle dogs by using injections of "plastoid" (presumably a viscous polymer) into the gingival vasculature (Figure 1.5). He demonstrated that little to no changes occurred after corticotomy with the Köle style. Rearrangement of the teeth within a short time after corticotomy damaged neither the pulp nor the periodontal ligament. Yet, he supported the idea of preserving the marginal crest bone in relation to interdental cuts. He also proposed that linear cuts should always leave at least 2 mm of the alveolar crestal bone untouched.

The author warned rather naively that there may be an increased risk of periodontal damage

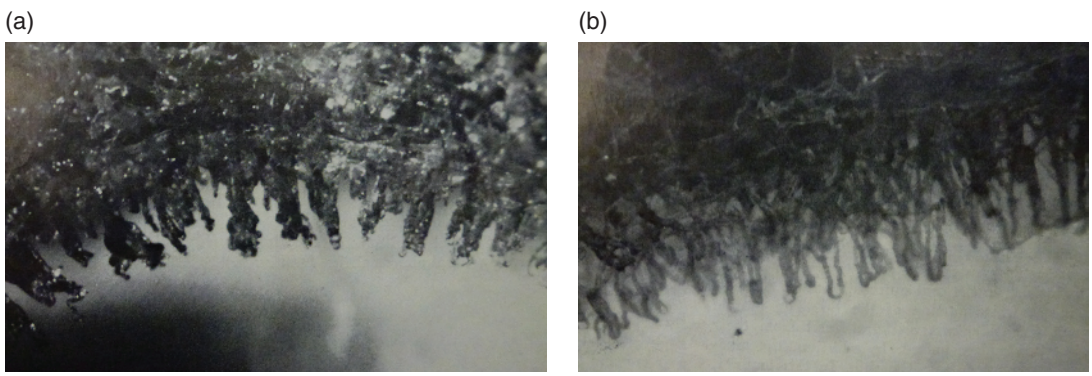


Figure 1.5 Marginal gingival vascular "agglomeration" (loosely organized masses) of capillaries (a) are demonstrated keeping the structural integrity intact with only mild atrophic appearance (b), which could easily be a processing artifact caused by a drop in blood pressure when the animals were exsanguinated upon sacrifice. *Source:* Düker (1975). Reproduced with permission of Elsevier.

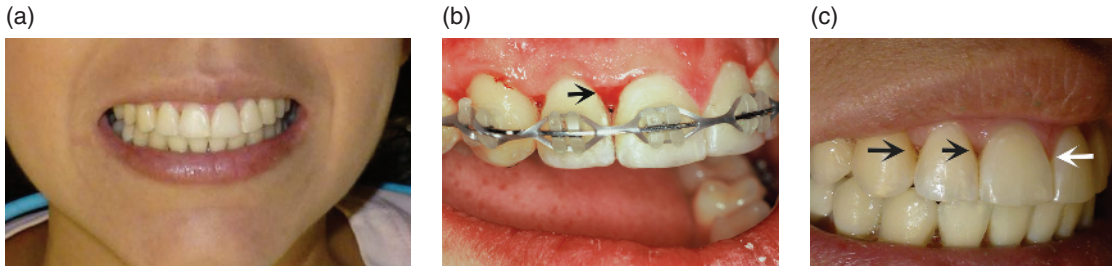


Figure 1.6 Despite an excellent 9-month class II treatment outcome (a) this patient objected to the open embrasures (b,c) at the lateral incisor (black arrows) contrasting with the central incisors' embrasure (white arrow) filled with healthy gingival tissue. The embrasure opening was caused by dehydration of a mucoperiosteal flap during a protracted surgical procedure. Good surgery is "swift, sure, and clean." Indications of gingival slough were evident in the first post-operative appointment (arrow in b).

in cases in which the interradicular space is less than 2 mm. This paean to the obvious is germane where one fears damage to root surfaces but cannot be extrapolated too far. Given contemporary knowledge about infectious periodontitis, SAD and PAOO both include the debridement of infectious accretions during the surgical procedure and therefore constitute a kind of surgical periodontal debridement themselves. This is both harmless and helpful. While surgical misadventures can adversely affect the patients' periodontal health, infection mismanagement is a greater threat to the integrity of the periodontal organ than the cuts of a skilled surgeon as long as the periodontal anatomy is preserved. The simple mucoperiosteal flap surgery itself can have salutary effects, in that the singular reflection alters redox potential of the bacterial niche, making it relatively unsupportive of anaerobic infection.

In summary, Düker's article is instructive in three major ways. First, he efficiently dismissed fears of vascular damage with credible photographic evidence. Second, citing previously underreported German authors, he intimated that American literature was deficient, a subtle criticism with which we agree. Third, he noted that the corticotomies were made short of the marginal alveolus and attributes the benign side effects of corticotomy to this forbearance. It is this last notation that should be addressed more deeply. In our experience, carrying the corticotomy to the marginal crest of the alveolus is acceptable if no physical ablation of the periodontal ligament is made and the periodontal anatomy is otherwise healthy. Nonetheless, this is a legitimately debatable issue.

Sometimes, cases of post-operative recession and papillary atrophy create unaesthetic open labial embrasures (Figure 1.6). However, wherever periodontal structures are intact and healthy tissue appears after periodontal flap reflection, inattentive authors may commit a *post hoc* fallacy,[7] inaccurately attributing the "black triangle" appearance of open embrasures to the surgery rather than pre-existing periodontal attachment loss obscured by arch length deficiencies. In contrast to exceptional cases such as the case in Figure 1.6, the underlying cause of spontaneous open embrasures is usually preexisting and undiagnosed marginal periodontal disease, not the surgery itself. When periodontal disease is present and the surgeon wishes to avoid opening labial embrasures, the sub-crestal limits of the interproximal linear decortication should be maintained in the mass of attached gingiva apical to the osseous crest (Figure 1.7). Full-thickness flaps can be elevated after a sub marginal incision that preserves the alveolar crest and interdental papillae. Where no periodontal attachment loss

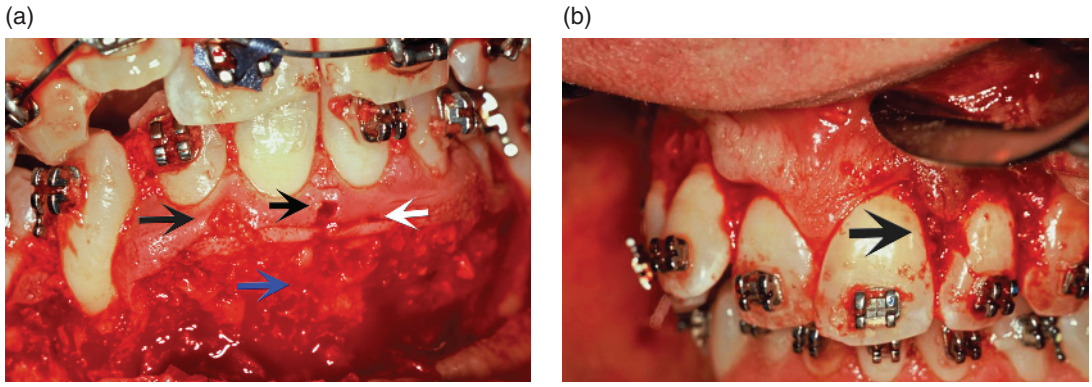


Figure 1.7 Demonstration of how contemporary alveolar tissue engineering can employ a constellation of techniques to facilitate "smile engineering" by applying TMPs (black arrows), sub-marginal initial incisions (white arrow) to preserve the structural integrity of the alveolar crest, and aggressive linear and punctate decortication (blue arrow). Each technique can contribute to a composite, wholely *orchestrated* manipulation of alveolus bone physiology inducing a transient therapeutic osteopenia with stem cell "targetting". Allogeneic stem cell grafting, and alveolus mass augmentation (PAOO/AOO) for use with accelerated and traditional but efficient biomechanical protocols.

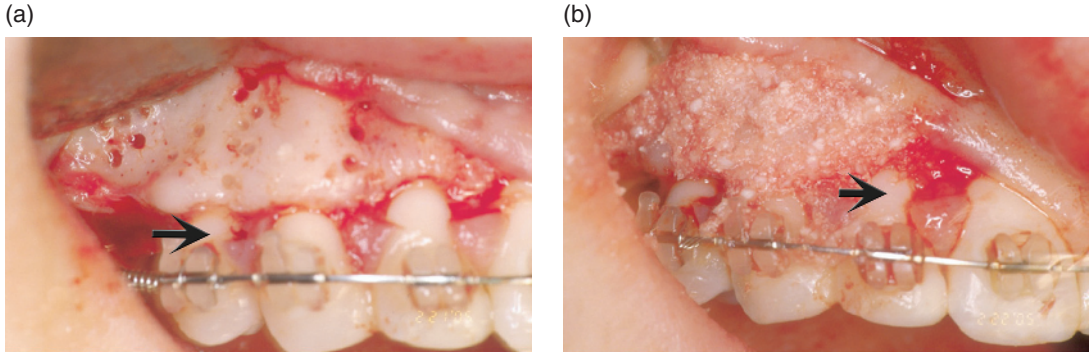


Figure 1.8 Where crestal alveolar bone is to be avoided, a sub-marginal initial incision can be made as indicated to keep the crestal bone and interdental papilla (arrows) untouched. Contrast this timid decortication with the aggressive decortications of 1.7(a). The degree of osteopenia and OTM acceleration is directly commensurate with the degree of decortication, and experience will allow an accurate, albeit subjective, sense of "how much is enough." When in doubt, there is little justification for timidity. *Source:* Mihram and Murphy (2008). Reproduced with permission of Elsevier.

is evident, the surgeon can reflect flaps with impunity in the labial esthetic zone as long as the supporting lingual half of the lingual papilla remains intact.

However, where periodontitis is active and esthetics is not an issue, opening embrasures actually indicates a rational, prudent treatment plan and should be encouraged by combining traditional ostectomy and osteoplasty with the linear decortication. Thus, the admonition that SAD and AOO should not be performed in the presence of "infection" is imprecise and misleading; submarginal incisions bypass crestal defects (Figure 1.8).

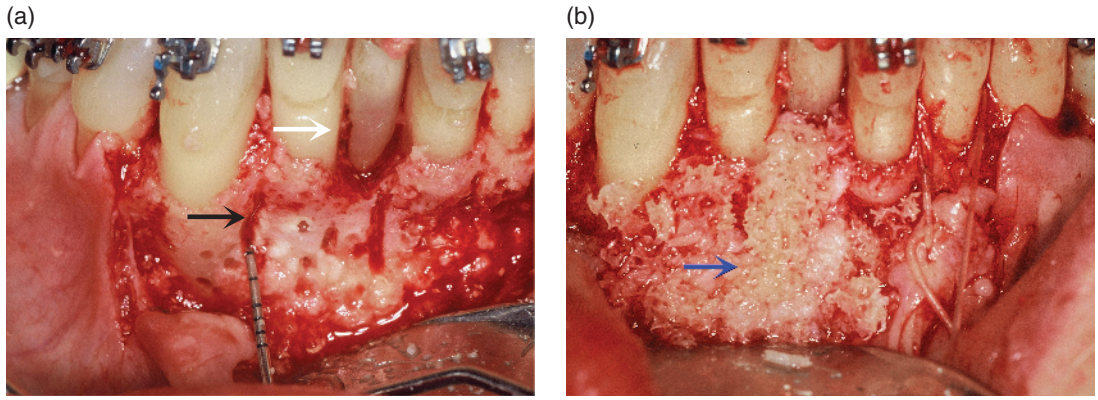


Figure 1.9 Simultaneous treatments of an infrabony defect and PAOO. (a) A periodontal defect (white arrow) after linear and punctate decortication for accelerated OTM. Note, depth of decortication (black arrow) approximates 3 mm the thickness of the labial cortex. Decortication was designed to simultaneously liberate endogenous stem cells from the medullary bone and induce a transient osteopenia to accelerate the rate of tooth movement. This demonstrates that PAOO can be combined with conventional periodontal surgical pocket reduction and that bone grafting can serve the dual purpose of creating a new bony phenotype and regenerating new periodontal attachment. *Source:* UniversityExperts.com. Used with permission. (b) The use of exogenous Stem Cell Therapy in conjunction with PAOO. Blue arrow indicates viable allogeneic stem cell graft. *Source:* UniversityExperts.com. Used with permission.

Certainly when bone grafts are used, in either PAOO or AOO, no active periapical lesions or untreated periodontitis should be present as the bone grafts or stem cells are placed. But when flaps are reflected for orthodontic purposes and the opportunity presents for traditional marginal alveolar bone management (infrabony pocket decortication, ostectomy, osteoplasty, or bone grafting,), pocket reduction and regeneration should be included in the surgical treatment plan (Figure 1.9).

To neglect this important service would constitute an error of omission and a logical *non sequitur*. Given that full disclosure of pathosis is *de rigueur* (and a legal imperative) in any healthcare venue, it can even be considered ethically untenable to overlook periodontal health. Ignoring periodontitis renders lesions vulnerable to exacerbation during accelerated orthodontic therapy because moving teeth in the presence of active periodontitis can pump supragingival pathogens subgingivally, accelerating the permanent loss of supporting periodontal attachment and creating problematic vertical defects.

Despite the subapical through-and-through transverse cut in the alveolus, no pulpal or alveolar necrosis was evident in Düker's model (Figure 1.10). The distinguishing characteristic in Düker's procedure was the absence of luxation (mobilization). So the results of Düker's beagle dog study sufficiently confirm that luxation provides little advantage to SFOT and only invites, as correctly feared, an untreatable ischemic necrosis.

1978: Generson – The open bite

Three years after Düker published his article, Generson *et al.* (1978) applied corticotomy to the treatment of apertognathia (anterior open bite). The authors' concern for the possibility of compromising the blood supply to the teeth was settled when the authors make a point that "the bony cuts were made only through the cortex." So, articles on simple decortications of the alveolus begin, with Generson *et al.*, to

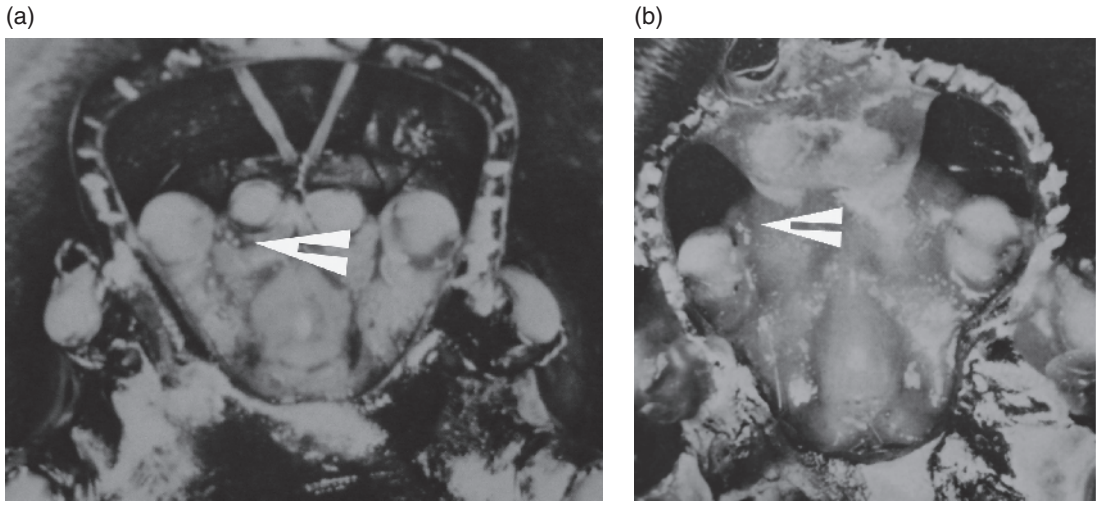


Figure 1.10 The arrows indicate the wake of tooth movement in the adult male beagle dogs before (a) and after (b) SFOT. This successfully accelerated movement suggests that a pedicle-based osteotomy (which risks both pulpal and alveolar necrosis) is unnecessary for SFOT, thus allaying the fears expressed by Bell and Levy (1972). The figures (a) and (b) suggest that a tipping motion resulted in 4 mm movement in 8–20 days. *Source:* Düker (1975). Reproduced with permission of Elsevier.

suggest that through-and-through osteotomies connecting linear decortications, like luxations, are superfluous.

Generson *et al.* applied the decortication concept and initiated orthodontic force 3 days after surgery. This is significant, because some authorities generally recommend a 2-week hiatus between surgery and the initiation of tooth movement. In contrast, the senior author has been initiating tooth movement with fixed brackets and a 0.018″ or round nickel–titanium archwire immediately after tying the last suture.

What is more significant in Generson *et al.*'s work is their description of the open bite. Again, we see a conflation of concepts where Generson *et al.* stated that their Case 1 exhibited "…no skeletal abnormality." Apertognathia is a dento-skeletal abnormality that may or may not reflect a similar spatial abnormality in the skeletal corpus subjacent to the alveolus bone. However, the surgeons did not present a cephalometric tracing or numerical parameters defining the underlying dysplasia of the skeletal corpus. An insufficient overbite is called "dental open bite" and may or may not be combined with aberrant overjet, and a dentoalveolar open bite can appear with a normal mandibular plane angle. Consequently, the reader must presume that the patient presented with the kind of dento-alveolar anterior open bite often caused by thumbsucking during the transitional dentition.

This contrasts with a *skeletal* open bite caused by an excess posterior maxillary vertical dimension. This is measured by the mandibular plane angle which is formed by the inferior border of the mandible *vis à vis* a Frankfort horizontal reference plane. As a rough guideline, skeletal open bites and skeletal deep bites begin with great deviations of the angle above and below 26° respectively.

The excess vertical dimension of the posterior maxilla, caused either by aberrant craniofacial growth or extrusion of the maxillary molars, causes a posterior–inferior autorotation of the mandible at the temporomandular joint and often requires orthognathic surgical correction.

The dental open bite, in contrast, is quite amenable to anterior alveolus elongation by SAD as long as the inferior elongation, a kind of distraction osteogenesis of the maxillary anterior alveolus, does not create an unaesthetic gingival display.

The distinction between a "skeletal open bite" and "dental open bite" is critical, and it is unfortunate that Generson *et al.* did not even produce a facial photograph for their article. In "dolichocephalic open bites" characteristic of a "long face syndrome," indiscriminate use of a corticotomy to elongate the maxillary alveolus can be just as ill-coceived as when orthognathic surgery is used for simple dental open bites. Both facial forms can produce unaesthetic results, pejoratively referred to as a "gummy smile." So, cephalometric analysis and photographic documentation are critical in these cases.

The authors published a post-operative photograph of the treated apertognathia, but the outcome displays an insufficient residual overbite and overjet. This documents the notorious potential for "vertical problems" (skeletal deep bites and open bites) to relapse.

Many fundamental musculo-skeletal deformities are clearly beyond the reach of dentoalveolar SAD. So, it may be unrealistic to expect corticotomy procedures to correct some skeletal dysplasias, and yet an alteration of the alveolus form may indeed provide sufficient clinical camouflage for them.

It would be helpful if a twin study were possible to directly compare the treatment of apertognathia with selective decortication versus LeForte procedures. In the absence of a comparative study it seems the corticotomy, while displaying some relapse potential when unretained, offers less biological cost (surgical morbidity) with presumably equally satisfactory results. So it seems that in some cases of apertognathia the corticotomy may be the first treatment of choice. However, in any case, a cephalometric analysis is still *de rigueur* when counseling the patient for any SFOT that might alter the vertical dimension of occlusion.

Interesting in this regard is the publication by Oliveira *et al.* (2006). In this article the authors describe a case of an apparent *skeletal* open bite that was treated to an excellent clinical outcome in only 4 months using corticotomy without orthognathic surgery. This outcome, which would normally involve major in-patient surgery, confirms that any proposal for orthognathic surgery may need to consider corticotomy-type surgery as a singular alternative or at least an adjunctive therapy. Relevant to this point, 2 years later Generson *et al.* ended their article with the statement that, indeed, "…corticotomy has a place in the armamentarium of the orthognathic surgeon."

1976: Merrill and Pedersen

Merrill and Pedersen (1976) resurrected the role of iconoclast when they investigated SFOT further for "immediate repositioning" of "dental-osseous elements." Ironically, after claiming to document the safety of the osteotomy and immediate repositioning of the dentoalveolar complex, the authors said that some unspecified complications had occurred, but were not sufficient to condemn the procedures. Based on our experience, these complications may well have been papillary slough, ecchymosis, or pain, all the hallmark of limited experience, naive clinical management, and serendipity.

Even more controversially, they stated that a corticotomy "…has not proved to be a successful method… in our hands…," adding that "… resistance by cortical bone has little to do with the reaction of teeth to force…" and "…little if any time is saved when classical corticotomy is used…" This intemperate conclusion stands in sharp contrast to recent controlled studies, by orthodontists and periodontists, that have demonstrated stunning efficacy, comfort, and safety with SAD, PAOO/AOO, TMP, and SCT. So, in the long history of literature on this subject, to some extent the Merrill and Pedersen article must be considered an aberration.

Conceptually, the most common cause of such tepid endorsements or unsubstantiated statement of difficulties is a failure to understand either the malleability of the alveolus or the need for managed strain in the spongiosa. This strain must be episodically induced with bi-weekly clinical adjustments in order to perpetuate the therapeutic osteopenic state. The strain must not induce resonance in the bone. That would cause premature recalcification, a return to steady-state physiology, and complicate the therapy. The well-designed perpetuation of decalcification produces a bone state similar to the relatively decalcified status of a malunion or a therapeutic site in long bone distraction osteogenesis.

When the RAP is thus perpetuated, the orthodontic clinician has sufficient time to adapt to interruptions and inefficiencies in their clinical biomechanics treatment. Yet even when the RAP dissipates (usually due to poor patient compliance or outright patient negligence) it may be resurrected with periodic and benign TMP of the alveolus (refer to Figure 1.4e). Thus, an optimal osteopenia, engineered intelligently, can be maintained almost indefinitely. Even though Merrill and Pedersen offer no one-to-one comparisons or quantitative data to substantiate their claim, relying solely on the disclaimer that results were limited "…in our hands…," they suggested a meaningful alternative.

Repeating the beliefs of Bell and Levy, they posit that two surgeries could be performed sequentially, first on the buccal aspect and then on the lingual side of the alveolus to provide collateral circulation for each surgery. A vascular anastomosis was proposed as the theoretical connection from the contra-lateral mucoperiosteal tissue that was not reflected. They also suggested that a 0.25 mm thin blade is preferred to a Stryker saw (0.80 mm) if safety for the adjacent roots is considered. We propose that a high-speed irrigated no. 2 or no. 4 round bur is the instrument of choice for precision and control (see Figure 1.4d).

In defense of Merrill and Pedersen, SAD limited to the labial alveolar cortex is a reasonable variant where the surgeon may wish to facilitate simple labial movement and wants to maintain a copious blood supply from the lingual aspect. Bear in mind, however, that the facilitating osteopenia is commensurate with the degree of therapeutic surgical "trauma" to the alveolus, and reflection of both a facial and lingual mucoperiosteal flap – even for simple labial movement – may contribute greater stability via more dissipated decalcification. Still, since the relative merits of a two-staged protocol are largely a matter of a surgeon's style, it would be too presumptuous to endorse or condemn such a procedure categorically.

1987–1974 Rynearson's revelation, Yaffe's physiology

Rynearson (1988) made a meaningful contribution to dentoalveolar surgery that even he may not have appreciated. He tested Köle's hypothesis that, under orthodontic force, teeth in a corticotomy-treated segment move as a tooth–bone unit. Utilizing implanted radiopaque pins and bone labeling, he found no evidence of movement of the cortical plates and concluded that the corticotomy procedure, albeit efficacious, did not effect a mechanical movement of a tooth–bone unit, but rather elicited a facilitation of normal physiologic tooth movement metabolism. The fallacy of "bony block" movement was finally beginning to be challenged, at least for space-closing procedures. However, the exact cell- and molecular-level mechanism responsible for the facilitated tooth movement was still an enigma.

Seven years later Yaffe *et al.* (1994) added a very important clue to unraveling that mystery by reporting a robust RAP response in the jaw bone of rats to a simple mucoperiosteal flap reflection. They documented not only massive decalcification of the alveolar bone, but also a widening of the periodontal ligament space. The demineralized zone was observed as early as 10 days, and the alveolar bone returned to

control levels 120 days after surgery. The authors also suggested that RAP might be responsible for tooth mobility and bony dehiscence formation where the bone was thin. Although Yaffe *et al.*'s research did not include tooth movement, in terms of our understanding today this is an ideal explanation for facilitated tooth movement. That is to say, a thin layer of bone, ahead of the movement vector, will demineralize, leaving the soft tissue matrix of the cribriform bone to be carried with the root surface into the desired positioning. The term used for this phenomenon is *bone matrix transportation*.

1985: Mostafa – Limited objective therapy

Mostafa *et al.* (1985) diagrammed a surgical–orthodontic technique to treat overerupted maxillary molars showing that corticotomy could be used for one or two tooth segments. It was a Köle-like decortication localized to the alveolus of one tooth, an extruded molar.

They reported a survey of 15 patients, noting that only the cortex was incised with a surgical bur and osteotome. No explanation was made about the specific nature of the surgery. Further, no statistical analysis or even photographs were presented. It was noteworthy, however, that the authors found a single-tooth procedure helpful. As discussed below, the same issue was debated between Kim *et al.* (2009) and Murphy (2010) as late as 2010 and is helpful as preprosthetic SAD.

1987: Goldson and Reck

Goldson and Reck (1987) reported a similar surgical–orthodontic treatment of malpositioned cuspids. They reported on the use of a bur and osteotome combination to completely separate the dentoalveolar segment through both the buccal cortex and medullary bone. A blood supply from the collateral sources in the adjacent mucoperiosteum was apparently sufficient for this procedure, which went deeper than today's SAD. Although one may need to induce a thorough osteopenia, there are reasonable limits. For example, osteopenia usually needs to be induced only within 2–3 mm of the teeth to be moved.

Suya and the Asian connection

Suya (1991) stimulated significant academic interest in Asia (Chung *et al.*, 2001; Hwang and Lee, 2001; Kim and Tae, 2003) for nearly two decades.

As doctors collaborated in the USA, Chung *et al.* (2001) in Asia also reported a decortication-assisted orthodontic method. Also, Hwang and Lee (2001) introduced a technique for intrusion of overerupted molars, using a combination of decortication and magnets. Kim and Tae (2003) moved teeth facilitated by decortication, referring again to the phenomenon as "distraction osteogenesis," and citing it as a "new paradigm in orthodontics." They removed part of the cortical bone, which resulted in "a speedy rate" compared with "conventional" OTM. They noted that intrusive movements were without side effects, such as root resorption or periodontal breakdown, and reaffirmed other colleagues' observations that corticotomy procedures were actually clinically superior to conventional orthodontic methods.

Park *et al.* (2006) and Kim *et al.* (2009) reported an interesting technique that is often contrasted with flap reflection methods. Although it does not allow the surgeon to visualize periodontal pathosis, and may indeed exacerbate pre-existing lesions if not executed with precision, it is noteworthy. These talented clinicians successfully used a method of surgical incision called "corticision," wherein a reinforced scalpel is used as a thin chisel to separate the interproximal cortices trans-mucosally,

without a surgical flap reflection. This transmucosal incisional manipulation, similar in effect to TMP of alveolar bone, minimizes morbidity but may fail to recruit significant RAP, which occurs simply with mucoperiosteal flap reflection as reported by Yaffe *et al.* in 1994. Also, the corticision is designed merely to accelerate tooth movement, not to redesign underlying bony phenotype. Nonetheless, used prudently in cases without periodontal problems, the corticision appears to have earned a place in clinical practice as a legitimate and meaningful SAD modification.

Suya's paper reintroduced a refinement of Köle's work into the American mind with a report on "corticotomy-facilitated orthodontics." His contentions were well received because it demonstrated that conservative intervention could yield dramatic results, and he substantiated that opinion by reporting his experiences in over hundreds of patients. Importantly, he also did not connect the buccal and labial incisions, like Köle, but relied purely on linear interproximal decortication. Although Suya contended that the facilitated tooth movement was the result of "bony block" movement, we now know that the style of decortication, divots, lines, or other patterns is irrelevant. Only the sum total of therapeutic trauma is significant. Suya's refinement of Köle's methods has essentially set the standard for decortication procedures that followed in the post-modern era.

1986: Anholm and the Loma Linda investigation

Following communications with prior visionaries, periodontists and orthodontists collaborated in the first major university studies of the phenomenon at Loma Linda University in 1986 (Anholm *et al.*, 1986; Hoff, 1986; Tetz, 1986; Rynearson, 1987; Gantes *et al.*, 1990; Khng, 1993), and some elaboration of their work is fitting.

Preeminent in this collaboration is the work of Anholm *et al.* (1986). Sobered by minor attachment loss, the orthodontists on the team were cautious in their praise of corticotomy-facilitated treatment in a male patient. While the attachment loss was not clinically significant, it is often noted in some cases, but not related directly to SAD or PAOO/AOO, as a proximal association. Some ostensible attachment loss will sometimes occur if the periodontal (mucoperiosteal) flap is reflected for too long, and thus dehydrated (see Figure 1.6(b)). Another source of error is the failure to completely debride the surgical site of infective detritus. The surgery should be "sterile, sure, and swift." Most other examples of presumptive attachment loss are usually due to undiagnosed previous periodontitis. This misinterpretation of side effects is common in poorly educated orthodontists, who overemphasize the art of clinical biomechanics to the detriment of life science, and argues that better education in principles of periodontal science and bacteriology should be incorporated in orthodontic educational curricula.

1990: Gantes et al. To pool or not to pool?

The issues of root resorption and potential periodontal damage were substantially dismissed in an excellent article by Gantes *et al.* (1990). They treated five patients, 21–32 years old, with Suya's protocol and a removal of cortices adjacent to an extraction site, and the original intent of the research was only to measure attachment level changes. They observed accelerated OTM, some mild root resorption, but no significant loss of attachment or loss of root vitality. The issue of root resorption was subsequently dismissed by later controlled studies, which finally revealed that SAD and PAOO/AOO, done correctly, indeed produced *less* root resorption than conventional nonsurgical protocols. By this time it was well understood that a regional osteopenia offers less physical resistance to root movement.

The Gantes *et al.* article can be disappointing to neophyte orthodontists and surgeons because they reported that, even when the buc-

cal and lingual cortical plates were removed at the extraction sites, the mean overall treatment times were still only 14.8 months in comparison with 28.3 months for the control group. The authors then questioned the practicality of the corticotomy because of increased complexity, the frequency of patient visits, and chair-time being approximately the same as conventional orthodontic treatment. So the authors seriously saw very little practical benefit to the patient.

One can agree with this assessment if surgery is eschewed by a patient who has no concerns for the protracted therapy time in conventional orthodontic protocols. However, the increasing risk of root resorption and bacterial load virulence has always loomed as a dark shadow over conventional therapy, and SFOT offers a reasonable solution to this threat. So, despite doctor disdain, the corticotomy in this regard is done for patient safety not doctor convenience. Moreover, the ostensible lack of significant time savings in Gantes *et al.*'s work appears to be a misinterpretation when one takes a closer look at these raw data. This deep analysis also reveals a common but startling hidden trend in commercial-based literature and a more profound schism of epistemological perspective in our profession.

It is said that "The devil is in the details", and the article by Gantes *et al.* provides a good example of that pithy truth. Overall, the authors' "data pooling" may represent an accurate, albeit highly diverse, sample of "what walks into the office," but it obscures a very important fact. Assuming *arguendo* the questionable axiom that the authors' five patients accurately reflect the nature of the patient population, then indiscriminate application of corticotomy would indeed render little practical advantage. But, as applied scientists, orthodontists must be more discriminating and identify clinically significant subsets of the general heterogeneous population.

So, setting aside the data of "outlier" of patient no. 1 as nonrepresentative, it is entirely legitimate to interpret the Gantes *et al.* clinical observations in an entirely different manner. When the extreme data are dropped as nonrepresentative, a new picture emerges (Table 1.1)

Table 1.1 Reanalysis of the Gantes *et al.* (1990) data.

Patient type		Treatment time (months)		Time saved (months)	Reduction (%)
		With corticotomy	Without corticotomy		
1.	Brachy[a,b]	20	24	4	16.7
2.	Brachy	Indeterminate data			
3.	Brachy	11	28	17	60
4.	Bracy	12	35	23	65
5.	Meso	16	16	26	38

Source: Gantes *et al.* (1990). Used with permission.
The table shows that, in general, we can expect in most cases of SFOT to reduce treatment time by 40–60%. This is not true if the outlier, patient number 1, is included in pooled data. The decision to drop outliers from data is a controversial heuristic issue in quantitative research, but is often easier for seasoned clinicians. The decision determined by researchers familiar with the subject being studied and determining if such an outlier represents central tendency of the sample universe of data.
[a]Data for patient number 1 can legitimately be discarded as outliers.
[b]Brachycephalic refers to a "low (small) mandibular plane angle facial phenotype, skeletal dysplasia). Brachycephalic class II, division 2 malocclusions belong to a class by themselves. They are musculo-skeletal phenomena, a subset of malocclusion not within the universe of dentoalveolar pathosis and should not be conjoined with the standard universe of patients seeking therapy. This "data pooling" is a very common experimental design problem even in National Institutes of Health studies of arteriosclerotic cardiovascular disease and cancer research.

that indicates the corticotomy can be of significant advantage to doctors and patients alike. The existential question is, will the orthodontist choose to discriminate subsets?

Each philosophical approach – (a) pooling to reflect a heterogeneous clinical universe or (b) discriminating subset analysis to more precisely predict our clinical outcomes – has its proponents. The first is less accurate but convenient; the latter is precise but arduous. Arguing against discrimination one can say that all decisions are practically 50/50, and neither patients nor doctors alike can make such fine probabilistic discriminations. (So why try?) This is the rationale of the orthodontist-as-artist. The other perspective is that of the orthodontist-as-scientist, presuming that a reductionist approach will indeed allow more robust forecasting of an empirical event in fields of future uncertainly; that is, precise prediction.

This chapter argues for discrimination because such an analytical approach has characterized the orthodontic field since Angle discriminated three basic malocclusion classes in the sagittal plane. Beyond tradition, however, it is wise to discriminate subclasses of data to explicate nuance that otherwise might compromise the quality of clinical outcomes. Finally, reductionist thought is what scientists do, and thus justifies itself. Yet, in the entrepreneurial class of clinicians in open societies the choice is ultimately a matter of independent and often arbitrary professional standards. Those who choose subset discrimination are in good company for a number of reasons that involve a branch of scientific epistemology too tedious to argue here. Suffice it to say that one is compelled to agree with Popper (2002) on falsification as the most exalted test of universal truth, not independent corroboration or, worse, collaboration.

The Gantes *et al.* article still remains as a milestone in the evolution of corticotomy development, even though the outlier case may have been caused by inefficient biomechanics, effete surgery, complicating medical issues, or idiosyncratic individual biodiversity. There is still a great deal of variance of style among surgical procedures, and contemporary protocols are still developing. So, some "failure" is always predictable. It is worth noting that a 95% confidence interval in biological research is merely another way of saying that one time in 20 (5%) we are wrong. This is why the research of Gantes *et al.* was valuable: it lent a note of caution, and that alone is clinically significant.

Their research also revealed a significant deficiency in the conceptual organization of orthodontically driven corticotomies in that generation. This deficiency, of course, has been addressed and rectified in subsequent publications. Worldwide, clinicians and academic researchers have found that the corticotomy works quite well where its limitations, so well defined by the excellent scholarship of Loma Linda University, are realized and avoided.

A twentieth-century summary

In retrospect, from the work of Bichlmayr and Köle 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 10 months except in cases of severe class III skeletal dysplasias.

The majority of treatment times range from 6 to 8 months with class I and mild class II malocclusions. Independent clinicians have documented that severe posterior crossbites and anterior open bites may be treated successfully

in about 10–12 months. With the use of *orthopedic* forces (e.g., jack screws), space closing alone could be accomplished in 3–4 weeks, and with orthodontic forces one should reasonably expect a treatment duration of 10–12 weeks.

As the century closed, the specialty of dentoalveolar surgical orthodontics was reaching the end of clinical art and the beginning of a long scientific journey. Scientific specialists remained reasonably skeptical but undaunted because, with the consistently gratifying clinical results around a shrinking world, their collective confidence continued to grow.

TWENTY-FIRST-CENTURY PIONEERS

The entry of academics

By 2007, collaboration among clinicians and researchers at Case Western Reserve University and St, Louis and Boston universities (Twaddle, 2001; Hajji, 2002; Fulk, 2002; Machado *et al.*, 2002a,b; Kasewicz *et al.*, 2004; Skountrianos *et al.*, 2004; Ahlawat *et al.*, 2006; Dosanjh *et al.*, 2006a,b; Ferguson *et al.*, 2006; Nazarov *et al.*, 2006; Oliveira *et al.*, 2006; Sebaoun *et al.*, 2006, 2008; Walker *et al.*, 2006a,b) resulted in significant documentation of the SAD efficacy with disciplined university-level studies. Clinical researchers resolved once and for all much of the contention among the earlier clinicians by subjecting the 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 it demands. At the time, these early 21st-century studies of SAD and PAOO were unparalleled; in retrospect their findings seem epochal.

PAOO/AOO and tissue engineering

Wilcko and co-workers (Wilcko *et al.*, 2001, 2003, 2008, 2009a,b, 2012) described an innovative strategy of combining the corticotomy with alveolar grafting in a technique referred to as the AOO technique or the PAOO technique. Initially, these protocols combined fixed orthodontic appliances, labial and palatal/lingual corticotomies, bone-thinning ostectomies at extractions sites and other selected areas, and particulate bone grafting materials. The introduction of PAOO/AOO marked a particular quantum leap into 21st-century tissue engineering and the tantalizing dynamics of biological nonlinear complexity (see Bak, P in Recommended reading). This is because these techniques engineer novel genetic expression and morphogenesis[8] that seems to account for the stability of the outcome.

Tooth movement with the PAOO/AOO techniques was typically initiated sometime during the week preceding the surgery and every 2 weeks thereafter by activation of the orthodontic appliance. The resultant the creation of bone *de novo* was a revelation. Prior to this observation it was considered axiomatic that one "…cannot grow bone on a flat surface." Yet PAOO/AOO did just that.

The first publication by Wilcko and co-workers was preceded by 8 years of research and development on humans, which began by performing corticotomy surgeries without bone grafting on both young adolescents and adults. Prior to this research, corticotomy-based surgeries were only recommended for individuals 18 years of age or older following the cessation of growth. As a part of their research protocols, high-resolution hospital-based surface CT scans were performed on their patients both pre- and post-treatment.

The high-resolution surface CT scans showed what appeared to be a demineralization–remineralization process at work. The Drs Wilcko

first suggested that this was consistent with Frost's (1983) RAP. The most rapid movement was most highly correlated with the thinnest volumes of bone attached to the periodontal ligament (attachment apparatus). This is what inspired the hypothesis that a demineralized matrix was seemingly transported with the root surface and available for remineralization following the cessation of tooth movement. The remineralization process, when completed in the adolescent, seemed to reestablish the original bony (native) architecture usually within 2–3 years. In the adult, however, the remineralization process, when complete, seemed to result in a net loss in bone volume and mass if particulate bone grafts were not included in the surgical procedure (PAOO/AOO).

Wilcko and co-workers have repeatedly referred to this phenomenon as "bone matrix transportation," and it is that phenomenon that effects a consistent 300–400% increase in tooth movement rate when combined with efficient biomechanical protocols. These evidence-based studies replicated those of Köle and Bichlmayr and substantiated the merits of SFOT with more sophistication than the prior clinical reports achieved by trial, error, and speculation.

It is important to note that if a tooth is being moved through a large volume of bone it is likely that only about a 30% increase in movement efficiency will be realized. This is consistent with the many findings of others throughout the last two decades. For example, even when RAP is employed, the bodily translation of a tooth through the long axis of the alveolus will typically only allow for about a millimeter of movement per month when no significant entry into interproximal medullary bone is made (Iino *et al.*, 2006).

What the corticotomy-derived procedures are not

The PAOO technique[9] was proven to be a very efficient orthodontic protocol with a predictable and safe in-office surgical component. It was demonstrated in many independent cases that patient care could be completed in one-third to one-fourth the time required for traditional orthodontics, and borderline orthognathic cases could be improved or even precluded entirely. It is important to note that, through the research, two salient functions of PAOO/AOO were repeatedly emphasized. The newest developments of SFOT continue to define new horizons for orthodontists, periodontists, and oral/maxillofacial surgeons alike, but they are *not* variants of orthognathic surgery. The latter is a rearrangement of basal bone parts rather than a *re-engineering of alveolus bone physiology* with endogenous biochemical and exogenous load applications. Yet, this modest manipulation of the alveolus bone with PAOO/AOO and other surgical interventions serves two basic functions: (a) facilitated tooth movement and (b) an increase in the alveolar volume. Alluded to above, this dualism deserves some elaboration.

Facilitated tooth movement

The facilitated tooth movement can be maximized as RAP provides a dramatic decrease in bone density (mass per unit volume). SAD must maintain a thin layer of bone over the root enface, the surface in the direction of the intended tooth movement. This thin layer of bone can readily undergo amplified demineralization as a result of a surgically stimulated RAP, and the resulting soft demineralized tissue matrix of the bone carried with the root surfaces (demineralized *bone matrix transportation*) can remineralize during retention. This remineralization process is fairly complete in the adolescent, but only partially complete in the adult. That is to say, in the absence of alveolar augmentation there may be a net loss in bone volume in the adult that justifies the bone graft with DBM or stem cells (see Figure 1.2) allografts to compensate for matrix deficiency

(e.g., iatrogenic gingival recession) during mechanotherapy.

Increasing the alveolar volume

The increased alveolar volume is accomplished by placing a relatively large mass of resorbable particulate bone grafting material or stem cell allograft between an intact elevated periosteum and the opposing denuded alveolus. The main intent is to increase the likelihood of having the roots of the teeth "sandwiched" between intact buccal and lingual plates of bone. Maintaining the continuity of the periosteum is critical in maximizing the volume of this new bone. Pre-existing dehiscences and fenestrations can be eliminated (Figure 1.11), but only where

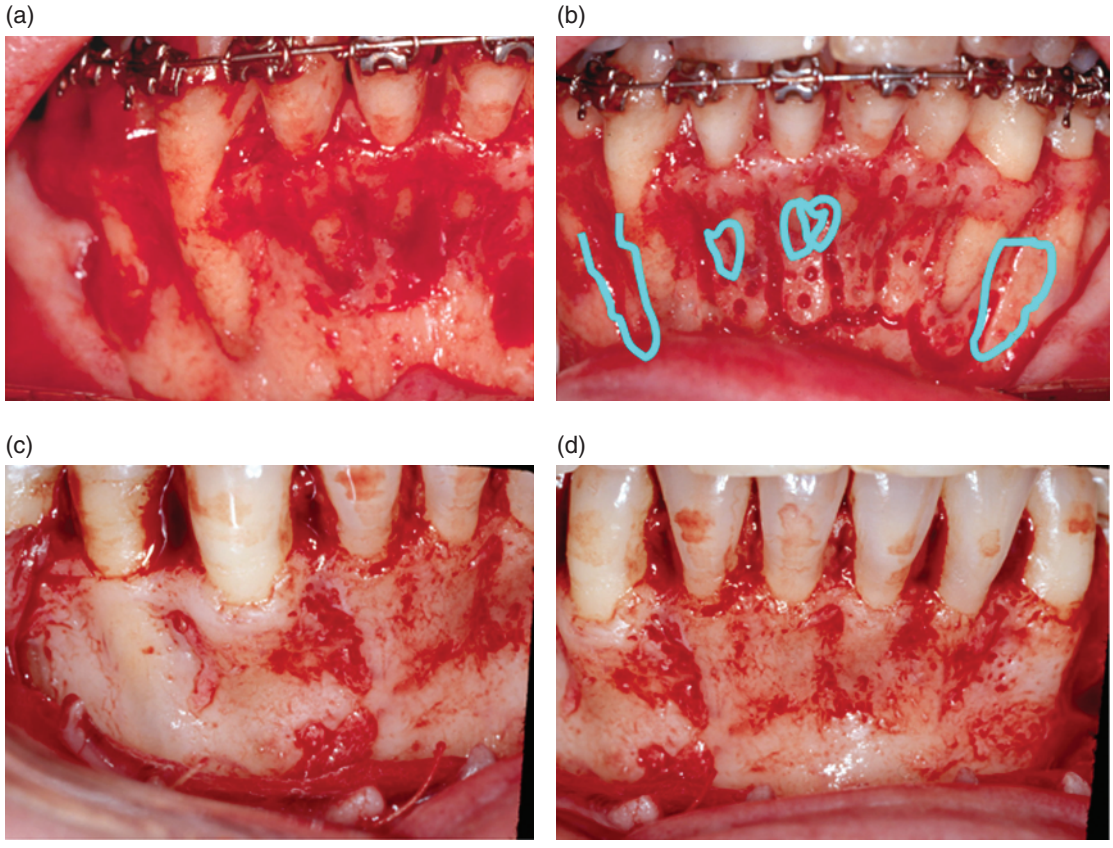


Figure 1.11 Demonstration that PAOO/AOO can create healthy alveolar bone in a steady state that lasts indefinitely. The bony dehiscence on the patient lower right canine in (a) and the fenestrations, outlined with light blue in (b) are sufficiently ensconced in bone even after 8 years (c,d). This is understandable since the procedure takes advantage of epigenetic restructuring of morphogenesis during healing by using the roots of the teeth as the functional matrix (see Moss (1997)) for new phenotype formation. This exemplifies how healthy, stable alveolus bone can be created *de novo* with the PAOO/AOO protocol. Previous to this demonstration it was believed by many periodontists that alveolar bone could not be "grown on a flat surface." This opinion is generally correct and precludes complete alveolus regeneration in the absence of orthodontic-induced strain. The combination of bone grafting, decortication, and induced strain suffice to overcome epigenetic buffering. Courtesy of Wilckodontics, Inc.

there is still vital root surface apical to the epithelial attachment (junctional epithelium). The ability to increase the alveolar volume makes it feasible to dramatically increase the envelope of motion, minimize gingival recession, reduce relapse, reduce the need for tooth extractions, minimize the risks of some orthognathic surgery, soften the acute angle of the labiomental fold, and reshape facial appearance ("face morphing") as an applied epigenetic event (Hochedlinger and Plath, 2009).

Special elaboration should be made about PAOO/AOO. In the growing child or adolescent the robust regenerative potential is evidenced by copious bony support of the teeth even in arch length deficiencies. Yet, in the older adolescent and adults there is often a net tissue loss when SAD is performed without bone graft supplementation. Figure 1.2 illustrates this phenomenon most clearly. Figure 1.2-1a demonstrates a pre-treatment high-resolution CT scan (accurate to 0.2 mm) of the lower arch of a female, age 39, prior to having circumscribing corticotomy cuts performed both labially and lingually around the six lower anterior teeth. Note the arch length deficiency (overlap crowding), the pronounced crestal glabella, and the distance between the crest of the alveolus and the corresponding cemento-enamel junctions. Clinically, the circumscribing corticotomy cuts resulted in the appearance of outlined "blocks of bone" connected by medullary bone.

The total treatment time for this case was 4 months and 2 weeks with eight adjustment appointments. Figure 1.2-1b shows that, at 1 month retention, the integrity of the outlined blocks of bone appears to have been completely lost and the layer of bone over the labial root surfaces appears to have vanished. In reality this layer of bone has undergone demineralization as the result of the normal osteopenic state (RAP); the soft tissue matrix of the bone remains but is not visible radiographically. This demineralized matrix was carried into position with the root surfaces (bone matrix transportation). Figure 1.2-1c shows the high-resolution CT scan at 2 years and 8 months retention. Note that the layer of bone over the root surfaces has only partially reappeared due to the remineralization of the soft tissue matrix. This suggests that there has been a *net loss of bone volume* in this adult. In adolescents this is not seen. Owing to a greater regenerative potential, there seems to be a complete regeneration of bone after SAD.

Figure 1.2-2a, again a high-resolution CT scan of the lower arch, this time of a male, age 23, prior to circumscribing corticotomy cuts being performed both buccally and lingually around all of the lower teeth with a large bone graft placed over the corticotomized bone. Note the paucity of bone over the buccal root surfaces. The total treatment time was 6 months and 2 weeks with 12 adjustment appointments. At 3 months retention Figure 1.2-2b shows the labial root surfaces are now covered with an intact layer of newly engineered phenotype appropriate for the new position of tooth roots (the functional matrix of the bone).The preexisting paucity of bone over the lingual root surfaces has been corrected in the same manner so that the roots of the teeth are now "sandwiched" between intact layers of bone both buccally and lingually. There has been a *net increase in bone volume*. At 2 years and 8 months retention, Figure 1.2-2c shows the increase in the alveolar volume has been maintained.

In the years around 2006 the Ferguson research group made great strides in documenting the science behind corticotomy procedures, which should inspire others. They characterized the RAP as amplified metabolic modeling of the alveolus adjacent to the SAD. The intensified anabolic activity in the rat appears to be increased by 150% at 3 weeks. This increase represents about a two- to threefold greater anabolic modeling activity in the spongiosa compared with same-animal contralateral controls. This same year, Sebaoun *et al.* (2008) reported a 200% multiple of spongiosa catabolic activity and a 400% increase in osteoblastic

activity at 3 weeks in the rat model. It was concluded that this effect represents the normal physiologic mechanism at the molecular level which quite adequately explains rapid OTM.

This emphasis on biochemical analysis evokes speculation about possible pharmaceutical manipulations to elicit so-called "optimal bone response." Optimal response is now the natural complement of any dialogue about optimal force. The fact that it may be done with pharmaceutical manipulation in situ is not only intriguing, but also, by reports of the Ferguson research group,[10] both proven in principle and justified by evidence.

The researchers of SAD, PAOO/AOO, TMP, and various other corticotomy incarnations have shown that it is important to maintain a strict, technique-sensitive protocol if one wishes to avoid a wide variance in clinical outcomes. While variance is inevitable, and indeed the fountainhead of creative progress, variance and occasional untoward events can be minimized by strict adherence to protocols that have been proven to protect patients and doctors. Undocumented facsimiles may not work as well as the evidence-based original described herein as it has evolved and is taught at university-standard institutions.

The post-modern consortium of dental scientists has convincingly demonstrated the very limited distance to which RAP extends from the point of surgical entry (approximately 2–3 mm in conservative surgery). Another advantage that analytical science lends to orthodontics is the concept of "enhanced relative anchorage." This is a term reserved for the development of differential anchorage advantages incurred when the treated segment of the dentition is rendered more mobile, thus enhancing the established anchorage units, *relatively*.

Following the realization that the facilitated tooth movement was an amplification of normal physiologic tooth movement metabolism, researchers attempted to rapidly close bicuspid extraction spaces without removing the interradicular bone around the extraction socket (buccal and lingual cortices). This attempt met relative failure, demonstrating that the cortices were indeed a source of anchorage strain and tooth movement resistance. The space closure alone was requiring nearly 7 months, and the overall treatments took over 1 year to complete.

What had begun at American universities as treating moderate malocclusion faster has blossomed within two decades into predictable protocols for the successful treatment of very complicated cases and ushered in the compelling science of tissue engineering and SCT. The treatment planning can at first seem daunting, and sometimes requires different sets of diagnostic parameters (even to the threshold of life itself (Gibson *et al.*, 2010), and, indeed, even new ways of thinking. But with a much better understanding of alveolar bone osteology, the new depth of orthodontic science has expanded the horizons of both orthodontics and periodontics and fostered their development into sophisticated multidisciplinary specialty options.

ORTHO-INFECTION HYPOTHESIS FOR GINGIVAL RECESSION

A credible discussion of any clinical practice is painfully incomplete without addressing the potential for irreversible tissue damage by oral infection. The fact that therapy may be accelerated also implies that the emergence of pernicious side effects may also be accelerated. Periodontal infections are endemic, and we operate in a bacterial stew of pathogens and commensal microorganisms. The bacterial biofilm in which they thrive can be modified, but this habitat cannot be eliminated. So, its effect on treatment must be acknowledged and constantly modulated. Knowing how the bacterial niche created by orthodontic appliances interacts with orthodontic therapy gives us insight into side effects that are predictable in the aggregate but *not necessarily foreseeable in the*

particular. Thus, a kind of universally precautionary protocol must always be maintained, and every patient must be informed of untoward bacterial events.

Usually, the orthodontist cannot act without some significant compromise of the supporting structures; periodontal impunity is the exception, not the rule. For example, Waldrop (2008) has pointed out that over 50% of adolescents may have permanent damage to soft gingival tissue, and we have known for nearly four decades that about 10% will demonstrate permanent damage to the alveolus bone (attachment apparatus) (Zachrisson and Alnaes, 1973). Permanent damage to the soft tissues (hyperplasia) is easily rectified by periodontal flap surgery. But when gingivitis progresses to periodontitis, with the loss of the first bone cell, the tissue dynamic is no longer a steady state; it is progressive and self-perpetuating.

However, one area of tissue dynamics has been placed at the feet of orthodontists unfairly. Gingival recession (bony and soft tissue dehiscence) is one example of a self-perpetuating bony pathosis, referred to in orthodontic parlance by the unfortunate term "runner." But this particular phenomenon is mischaracterized as an effect of orthodontic therapy which pushes a tooth outside of its "alveolar housing." This is a mistaken notion. Djeu *et al.* (2002), among others (Artun and Krogstad, 1987; Wennstrom *et al.*, 1987; Ruf *et al.*, 1998; Artun and Grobety, 2001), showed that there is no correlation between specific types of OTM and gingival recession, while others consider such movement a mucogingival threat (Dorfman, 1978; Hollender *et al.*, 1980; Genco, 1996), or at least a contestable issue (Melsen, 2012). So any discussion of corticotomy-like procedure involves the issue of mucogingival stress, bony dehiscences, and gingival recession.

A thorough review of the scientific literature suggests that an indeterminate variable in many of the studies of gingival recession and OTM is the quantity and quality of bacterial biofilm (plaque) accumulations and the host's local/systemic resistance. So, from an etiological and logical epistemological perspective indeed, a strong case can be made that the proximate cause of "runners" may not even involve OTM at all. Aleo *et al.* (1974) noted that bacteria endotoxins can inhibit fibroplasia. So, one may hypothesize that, when tooth movement requires fibroplasia to adjust phenotype for compensatory gingival adaptation, the compensatory soft tissue generation can be inhibited by patient noncompliance. Where OTM is increasingly viewed as a commodity the risk to patient welfare is increased by the contributory negligence of the dental "consumer."

Thus, although gingival recession cannot be correlated with OTM, the attachment loss it reflects can indeed be directly attributed to patient negligence. So any "cause" by orthodontists can only be attributed to acts of behavioral omission, not acts of therapeutic commission. In other words, OTM may be, in some cases, a necessary cause (contributory factor), but it appears as neither a proximate cause nor a *sine qua non* in all cases. The categorical assertion that "orthodontics tooth movement 'causes' gingival recession" is without compelling scientific basis and assumes the status of a clinical wives' tale. A new, better substantiated, and logical orthodontic-infection hypothesis posits that "runners are caused by germs." 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.

CONCLUSIONS

This treatise purposely evokes controversial issues in a historical context to give the thoughtful clinician pause for reflection in a meaningful dialectic. The dialectical progression,

borne of controversy and its consequent explication of important nuance, continue today through the *Sturm und Drang* of daily practice. When kept legitimate it is the stuff of progress; when abused 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 scientists' cold, redoubtable truths, it is the clinician as the frontline soldier who must sort out all the proverbial wheat from the sophistic chaff, all on behalf of the patient. That is our mission, our duty, and our privilege.

It is incumbent on all students of dentofacial orthopedics to actualize their full potential if this technique is to be realized. As specialists, we need to continually strive for perfection knowing full well that we will never achieve it. To quote Professor Colin Richman, "The PAOO™ technique is not for unschooled amateurs."[11] But challenge is why good students become scientists.

Art, while endlessly enchanting, can all too often parade as pseudo-intellectualism. 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 artist's brush, especially drawn untrue over the face of youth, cannot be erased if wrong. By contrast, the disciplined journey through the rigors of logical and scientific scrutiny leads to greater certitude, health, and predictable success. For the certitude we seek, science must be universal and timeless, reassuring us that, whenever we employ it, we may find a safe and steady path, literally into the human genome itself.[12]

True, it cannot be denied that at times many clinical imperatives of scientific truth require an artistic embellishment. This is indeed evident in the literature of 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, we posit that all SFOT will be enriched and fortified, in the dawn of oral tissue engineering. Rathbun's and Gantes' (personal communication, 2013) cautions in this regard are particularly sage. In retrospect, they remind us that treatments of young patients and short durations make careful treatment planning a critical clinical imperative; there is no substitute for assiduous scholarship. This is why constant tooth movement or the liberal use of periodic TMP is important, and an adroit orchestration of many modalities is needed to define the master clinician.

This, we contend, complementing the art of traditional biomechanics, is a clarion call to progress, not a siren song. SFOT protocols are destined neither to founder on the shoals of fatuous novelty nor fall ensnared in clinical disappointment. They shall find enrichment through the following chapters as our esteemed and dedicated colleagues take us on a journey toward an exciting future. And that personal future, liberated from the minions of corporate leviathans, untethered from the mindless morass of autocratic statists, renders individually and collectively a bountiful province for each of us to define in our clinical practice. The rhetorical question that this sojourn entreats is: "Will we choose to?"

Quo Vadis

AN AFTERWORD FOR ACADEMIC LEADERS

Twentieth-century orthodontist educators have the comfortable option of continuing the standard model of orthodontic tissue dynamics, essentially a 1901–1911 dogma, but that is

fraught with significant risks to clinical identity and patient safety. The fate of the new generation of orthodontists can lie within a greater 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, may fade into an interesting anachronism as straight-wire biomechanics becomes commoditized in the hands of nonspecialists.

Tissue engineering, in contrast, does not lend itself to commoditization. Therefore, new orthodontists, heirs apparent and champions of the specialty, have an existential choice upon graduation from their training: will they become corporate minions, distributing a mere commodity of short-order smiles, plebian artisans, or applied tissue engineers, thinking independently to bring the best science to each individual patient.

As this brief history lesson has demonstrated, time carries us to new vistas often only dreamed about or hinted at by previous generations. Sometimes these vistas are presaged in other specialty literature or in discussions of other subjects, or ironically even from nonacademic sources. It is only the thorough scholar who will pick up such nuances. Then, the future arrives at our doorstep whether we like it or not. The challenge is to separate transient fashion from tidal change.

Orthodontic-driven corticotomy procedures subsumed by the entire promise of SFOT is tissue engineering and it is not going away; the question is whether curricular innovations will go along with it. Defining new frontiers has always been the credo for the orthodontic specialty, but that legacy will endure only by the younger generation of orthodontists who wish to supplement the mantle of clinical artist with surgical 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.

Carpe diem!

Notes

1 "Give the adolescent an adult smile he can grow into, not an adolescent smile he will grow out of" (Williams MO, personal communication, 2013).
2 See Recommended reading.
3 Both the AOO technique and the PAOO technique are patented and trademarked by Wilckodontics, Inc. Erie, PA, USA. The acronym PAOO is generally used by the lead author when AOO candidates present periodontal issues.
4 Also known as demineralized freeze-dried bone allograft.
5 *Weltanschauung* (worldview) is a term used by educational psychologists and refers to the phenomenon by which the psyche interprets and adapts to a novel environment. Freud (1990: 195) said it is "…an intellectual construction which gives a unified solution of all the problems of our existence in virtue of a comprehensive hypothesis, a construction, therefore, in which no question is left open and in which everything in which we are interested finds a place." This plays an important part in the acculturation process for SFOT, which some academic institutions are precluded from inculcating due to intransigent socio-political commitments.
6 See Pirsig R in Recommended reading.
7 *Post hoc ergo propter hoc* (after this therefore because of this).
8 Wound healing recapitulates regional ontogeny (Murphy, 2006).
9 Both the AOO technique and the PAOO technique are patented and trademarked by Wilckodontics, Inc. Erie, PA, USA. The acronym PAOO is generally used by the lead author when AOO candidates present periodontal issues.
10 Donald J Ferguson DMD, MSD and his academic protégés made significant contributions to accelerated OTM and the nascent science of oral tissue engineering from his research facilities at St Louis University and

Boston University. After this prodigious independent corroboration he was appointed Dean of the European University College of the United Arab Emirates.

11 Assistant Clinical Professor, Georgia Health Sciences University, Augusta, GA, USA. Personal communication, 2013.

12 23andme, Inc. Mountain View, CA USA, www.23andme.com.

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