Beyond the Ligament:©

A "Whole Bone" Approach to Dentofacial Orthopedics and Falsification of Universal Alveolar Immutability

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by

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INTRODUCTION

Clinical tissue engineering in dentofacial orthopedics is in its infancy; most research presently is in vitro and pharmaceutical. But the conceptual stage is set for in vivo bone tissue engineering by regenerative procedures in periodontology* and modern medical orthopedics. The conceptual basis however cannot be fortified by traditional orthodontic tooth movement (OTM) biology which focuses solely on the periodontal ligament as the operant organ. It is forces acting beyond the ligament which may be significant determinants of the alveolus and the consequent dentofacial form which lives, thrives and dies by the grace of dental root positions. (Moss, 1987) Specifically forces eliciting an osteogenic threshold of ~1,500-3,000 microstrain (according to Professors Frost and Jee (Frost, 2004)) may stimulate appositional bone growth.

A morphotype which nature initially "intended" before perverse environmental perturbations distorted its form may be "recaptured" from an environmentally developed malocclusion. Of course this must occur within the range of physiologic potential but where etiologic agents such as chronic mouth breathing or premature tooth loss have caused abnormal distortion, that potential range is often quite obvious. Therefore, at the risk of overstating the theory of "alveolus development", this paper presents a modest synthesis of contemporary theories in cell biology to explain ostensible osteogenic activity, and alveolar phenotype alterations by ultra-low orthopedic force or by moving roots through a healing bone graft.

Dentofacial orthopedic physiology of the alveolus does not deny the relevance of periodontal ligament phenomena but merely goes *beyond the ligament* to analyze the alveolar response to orthopedic force from a "whole bone" perspective. This "whole bone" paradigm is an important complement to the classic pressure-tension model because it lends a consistency with medical orthopedic and contemporary osteology literature. As Baumrind (1969) has so eloquently proposed in the past, the periodontal ligament is best characterized, not as a pressure-tension sling, but rather as a contained viscoelastic gel where forces are distributed in all directions. Dental osteology which supports the pressure-tension model conflicts not only with

logic but also with medical long bone osteology as well, *viz.* pressure on long bones stimulates osteogenesis yet orthodontic pressure in the periodontal ligament is considered a stimulus for resorption.

We propose, not as a contentious polemic but rather in the tradition of the Western dialectic, which this conflict may have derived from an ill-advised emphasis on the pressure-tension mechanism of the periodontal ligament (PDL). The original PDL model proposed by Schwarz and Oppenheim the 1930's and expanded by Reitan in the middle of the 20th Century should be reconsidered in terms of 21st Century science. This paper aims to illustrate the practical application of alternative theories which help reconcile the inherent contradictions of the PDL model with case studies of surgical and non-surgical therapeutics that support the thesis. Presently, *in vitro* analyses of force distribution are being pursued by talented student research teams at ULCA.

Not wishing to compound the semantic ambiguities in this science we use the words "remodel" and "model" indiscriminately to refer to architectural changes secondary to therapeutic mechanical stimuli. Finer distinctions between the terms is important but beyond the scope of this discussion. A clear contrast is made however among rapid palatal expansion (RPE) of the maxilla, *orthodontic* dental arch expansion and *orthopedics development* of the bony alveolus with direct continuous modulated force application. Since wound healing recapitulates regional ontogeny* on a molecular level, regional alveolar ontogeny may be engineered in a similar manner whether the wound is surgical or simply a microfracture "healing" of the alveolar osteon (Haversian system). Thus, the alveolus is proposed herein as a separate ontogenic entity, capable of singular active biological response to loading irrespective of the subjacent maxilla or the subsumed dental matrix. In prior publication the "maxillary expansion" is used in all three contexts without clear differentiation. This is unfortunate yet ubiquitous in the literature (McNamara, 1999) and it would be most fortunate indeed for students and other neophytes in craniofacial orthopedics if more nuanced distinctions could be articulated. Case reports and

* See: Murphy NC, 2006

histological data demonstrate that both surgical and non-surgical tissue engineering can not only produces manifestly stable alterations of alveolus form but also reflect abundant clinical evidence of molecular phenomena discovered by clinicians and researchers in the latter decades of the 20th Century. These observations are not intended to prove universality but rather build on a rich theoretical heritage which considers corroborative evidence, Gaussian statistical validation and even independent replication less robust evidence for "laws" of nature that falsification. Thus the aim of this paper, its null hypothesis so to speak is an attempt to falsify the common presumption of alveolar immutably. By doing so it should liberate the ingenuous efforts of individual practitioners from both the strictures that deny the "art" of individual facial esthetics and a kind of "tyranny of the mean" which naïve collectivist thinking parades as the highest form of evidence-based quality parameter when treating individuals and their unique requests, preferences and biological imperatives. (See: Johnston, 1990, 1999, Ilizarov 1969, 1989, Popper, 2002)

Case Report #1 E.R.

E. R., a non-compliant 18 year old Hispanic-American male presented with hemorrhagic hyperplastic gingivitis and incipient periodontitis after a protracted period of inadequate oral hygiene. Approximately 15 months earlier a Max 2000® palatal appliance was placed to treat a posterior cross bite as the anterior arch length deficiency was addressed by an 0.018 nickel titanium round wire in labial 0.022" slot brackets. This arch wire was replaced with a 0.018" stainless steel arch wire but no activation was made in the anterior or posterior sextants. The Max 2000® orthopedic expansion appliance was not activated upon insertion because the mechanism is a self-limiting continuous release of two transverse nickel titanium springs embedded in two acrylic panels that active 150 grams each. (Fig. 1).

The bands on first molars and first bicuspids are for retention only. The active force solely lies on the palatal alveolus. No arch wire adjustment or palatal appliance adjustments were made for one year. Fifteen months after the appliances were placed they were all removed and the patient was treated with periodontal flap surgery to regain periodontal and gingival health.

During the surgery a biopsy specimen was taken from the labial alveolar crest of the maxillary right first bicuspid. (Figs. 2, 3) and sent to a UCLA oral pathologist for a blinded microscopic examination. An image of the specimen was then sent to the Medical College of Georgia for fractal analysis, a biomathematical parameter for bone remodeling.

Histological Analysis:

The specimen demonstrates young bone (yellow arrow) with conventional H & E stain. (Fig.4) The same histological specimen is also examined under polarized light demonstrates a "woven bone" pattern (Fig.5). The appearance of "woven bone" and confirmation of fractal patterns in the specimen (Fig. 6) are both is important because they suggest a pattern of immature bone remodeling; preexisting bone presumably would demonstrate a mature "lamellar" pattern. Mechanical loading is thought to an increase fractal dimension at a bone interface which reflects mechanisms of cell- mediated remodeling presumably within regional deformations of 2,000-3,000 microstrain. These changes in fractal dimension appear to be proportional to loading and are thought to provide a new parameter for force determination in orthodontic tooth movement. Fig 6 demonstrates that an increase in fractal dimension is indeed present in the biopsy specimen.

DISCUSSION

Since no active adjustments were made by a clinician during that time the Max 2000® appliance in Case #1 worked, it seems to have acted as a kind of "osteogenic machine", altering the alveolus form without producing any expected bony or soft tissue dehiscence. Indeed the bone biopsy (Figure 5) actually demonstrated woven bone characteristic of active remodeling. This case study suggests that continuous light mechanical loads directly on the palatal alveolus may preserve labial bone as a tooth-alveolus complex "moves" labially through remodeling "drift". (See: Enlow D and Hans M, 1996). Some theorists speculate that the alveolus is im-

mutable and expansion of the dental arch inevitably produces bony dehiscences or fenestrations. This case ostensibly suggests that bone may remodel to preclude these complications if two conditions are met: (1), the movement occurs without active periodontitis and (2) the internal strain falls within a threshold remodeling range.

Because the bone volume (BV) to total tissue volume (TTV) ratio is reduced from a normal 60% to ~30% (Ferguson, 2007) by surgical perturbation we conjecture that surgically-facilitated OTM accelerates bone remodeling in a similar and normal but amplified manner. There is no scientific evidence that pathological elements are operative or that the modeling elicited either by bone grafts or threshold bone flexing (strain) is qualitatively different from standard bone remodeling concepts. Surgery merely adds speed, convenience, stability and a mitigated bacterial challenge.

Some experts claim surgically-facilitated OTM e.g. selective alveolar decortication (SAD) may be contraindicated in patients presenting with periodontitis. (Ferguson, 2007). It is important to note that this may be a relative contraindication, not absolute. Case by case determination of indications and contraindications are often patient-specific and preference driven; while some risk-averse patients may elect to forgo surgical orthopedic care others can legitimately employ the SAD cost effectively and with clinical impunity.

The Max 2000® Case #1 also illustrates a paradoxical but important event where bone remodeling occurred even in a field of infection. The reasons that infection has not complicated the remodeling are that the force magnitude was low and unidirectional, not oscillating ("jiggling") as is generally evident in cases of occlusal trauma. In Case #1 the

infection qualitatively defined gingivitis, not periodontitis; the distinction between these entities is critical for the uninitiated clinician but often problematic to diagnose. (Longhurst, 1980). Thus, instead of moving teeth through the alveolus, it appears that palatal panels moved the whole alveolus, remodeling labial bone instead of moving roots beyond the alveolar envelope risking bony dehiscence and gingival recession ("stripping").

Conventional "Wisdom" and Innovation

Conventional wisdom based on work by Egelking and Zachrisson (1982) and others (Thilander, et.al.1983) suggests that the labial alveolus is immutable and labial movement causes bony dehiscence. However, contrasting data in reports by Lindskog-Stokland and Wennström, et. al. and others (Hom, 1984, Wilcko 2001, Melsen, 2006) suggest that the alveolar "envelope" or limits of alveolar housing may be more malleable than previously believed. The phenomenon of "phenotypic plasticity" explains this well. This plasticity is a well accepted concept in the field of developmental biology and is manifest only by various environmental or epigenetic perturbations. (See Waddington, Fig. 7) In disease the perturbation may be infection, or pathological trauma. Therapeutic intervention may also be considered an epigenetic perturbation where chemical effects or orthopaedic force are sufficient to overcome buffering tendencies (canalization) that secure ontogeny on a fixed trajectory. (See: Fig.6 and Waddington, 1954, Siegal and Bergman, 2002) The difference between pathologic form and therapy is the ability to control and predict treatment outcome. This refined conceptualization of alveolus physiology appears to be increasingly appealing to the enlightened clinicians and researchers alike.

This control is evident in the effect of the spring loaded Series 2000® appliances, presented here both grossly and microscopically. Yet the manipulation of bony form is not the monopoly of dentofacial orthopedists. The histological actions of the Max 2000® appliance mimics the principles applied in the innovative philosophy of Professor Ponseti's (1997) treatment of *talipes equinovarus* (club foot) in that it attempts to redirect a pathologic growth trajectory toward a physiologic course Since facial morphotype evolves even throughout adulthood (Behrents, 1986) the Max 2000 appliance may be a more benign alternative to surgically assisted rapid palatal expansion (SARPE) a traditional mechanical treatment that attempts to rearrange physical *components* instead of recruiting and engineering biological *forces*.

The presumed immutability of the alveolus has been historically is problematic in that it can reduce treatment options to extraction or orthognathic surgery, conventional protocols which juxtapose "parts" instead of engineering physiologic dynamics. In this regard any concept which emphasizes mechanical solutions can truncate intellectual growth and eclipse emerging biological imperatives. This is a needless and unfortunate limitation because many patients disdain major surgical alternatives Moreover, the studies of Little et. al. (1990) indicate that routine extraction therapy to camouflage dysmorphic skeletal elements is neither a panacea nor guarantor of stability.

Molecular Biology

A further understanding of the molecular basis for alveolus physiology may take us closer to predictable modification of form by surgical, non-surgical or pharmaceutical means. For now, these cases prove the principle that immutability of alveolar bone is not universal and alveolar surgery or continuous ultra-low force magnitude may indeed be an appropriate starting point from which orthognathic surgery or extraction may be deferred

as a reasonable second choice or "fail-safe" tactic. This is justified because since the time of Wolff and Roux bone has been considered a dynamic tissue adapting it form to its environment (form follows function). For the modern dentofacial orthodontist, Moss elaborates that "roots are the functional matrix of the alveolar bone". * More recently, Professors Pavalko and Bidwell (2002) and Maniotis, with Ingber (1997) join other mechanobiologists in suggesting that, on tissue, cellular and molecular levels, biochemical osteogenic functions such as morphogenesis can follow altered cellular form. Thus, form follows function and function follows form.

The Utah Paradigm in Dentofacial Orthopedics

The Utah Paradigm of bone physiology is also an instrumental element in explaining a "whole bone" approach to alveolar bone modeling. It proposes that a tissue-level entity (termed the "mechanostat") is a definitive but neglected functional determinate of bone physiology in steady state homeostasis or remodeling. Structurally a basic multicellular unit (BMU) is a collection of regional osteons that act as the bone analogue of the nephron. What Frost (2004) called the "nephron equivalent" is completely compatible with the principles of Wolff, Moss and contemporary cell biologists. This makes Frost Jee concepts superior metaphors for the alveolus that deserve more attention as a functioning component in orthodontic therapy. We propose that the woven bone patterns seen in the presented cases exemplify these concepts. Melsen (2006) has also acknowledged that the concepts deserve greater research scrutiny and reminds us that the Utah Paradigm has appeared in the orthodontic literature over 40 years ago. (Epker and Frost, 1965).

*Personal communication, 2005

The "whole bone" paradigm essentially presents orthodontics from the bottom-up, focusing on "optimal response" of alveolar bone instead of a bottom-down analysis of "optimal force". This is important because extraction in the prepubertal patient sacrifices developmental potential as it ablates part of a functional matrix critical to future facial development. The problem for the injudicious clinician is that this pernicious sequella may not be evident for decades, a prevalence which only longitudinal twin studies can explicate clearly. If alveolar development can call upon a strong foundation in basic osteology the orthodontist is liberated from outdated strictures of traditional thought which accepts pernicious side effects by default. New ways of thinking of course often cause some consternation and angst among those comfortable with the *status quo* but change is integral to the very fabric of science, an intellectual discipline some suggest is painfully lacking in the orthodontic art. (Johnston, 1990, 1999)

Where alveolus development can help define eruption trajectories the treated adolescent may present a smile that is seemingly disproportionately large for the immature face. However, Figure 11 demonstrates how a smile that defines the face of youth also fits esthetically well with the more mature adult face. Regardless of historical guidelines we believe that the emerging esthetic standard for a so-called "full" smile must be recognized and justified scientifically. The figure and the histological documentation of supporting bone remodeling echoes the maxim of Williams that facial orthopedists should "…create an adult smile which the adolescent can grow into, not an adolescent smile the adult grows out of. *

* Michael O. Williams, DDS, MS Gulfport, Mississippi, inventor of the Series 2000® appliances

On a molecular biologic basis explanation for the Max 2000 effects may be found in the science of mechanobiology. The literature on cell activity in fields of tensional stress may alter both extracellular and intracellular chemical activity by amplifying actions of membrane ion channels and secondary messenger cascades which can "reprogram" genetic expression.

Since morphogenesis is a transcription event of the nucleus dentofacial orthopedics qualifies as a "genetically engineered" phenomenon employing intracellular mechanisms well known to molecular biologists. Protein chemistry reminds us that changing the shape or conformation of important cytosol proteins alters their reactivity in a way that adding radicals to an inorganic molecule alters chemical behavior. Moreover some researchers have identified complex proteins (mechanosomes) which transduce mechanical data directly to the DNA to alter genetic expression. If the movement of the alveolus directly or indirectly through root movement can be seen as a flexure of the whole (alveolus) bone, it is no great logical leap to understand that "bending bone bends DNA" indeed. (See: Fig 8) In fact, on an intracellular level mechanobiologists have demonstrated in tissue culture that distortion of the cytoskeleton releases calcium ions and nitric oxide (NO), both well known morphogens.

TREATMENT TIMING,

It is important to understand that the scientific literature contains compelling and ample justification for redirecting growth trajectories in prepubescent humans. Therefore, the best time to treat the growing child with this self-limiting "machine" is measured in dental age not chronology. Generally, optimal effects are elicited during the transitional or mixed dentition. At this time phenotype changes dramatically. PAOO™ presents no age preference or restriction since surgical healing *per se* reverts all tissue to a kind of "neonatal" state.

Arch development from the palatal aspect may be employed simultaneously with conventional labial therapy. However, singular use of the Series 2000® appliance sans labial archwires reduces the risk of bracket breakage, pernicious increases in pathogenic bacterial biofilm (dental plaque) load and addresses orthopedic problems directly instead of camouflaging dysmorphic alveolar form with altered tooth positions.

GENETIC EXPRESSION AND PHENOTYPIC PLASTICITY

It is also important to note that phenotype is not a one-to-one manifestation of genotype. While genotype has often been defined as a "blueprint" for tissue form, this allusion is simplistic to the point of gross misrepresentation and leads to frankly erroneous thinking. In fact the genotype is more akin to an "instruction manual" directing tissue development to a myriad of forms depending on the degree of phenotypic plasticity inherent n the biological system. Phenotypic plasticity is the "property of a given genotype to produce *different phenotypes* in response to distinct environmental conditions" *" (emphasis added) Pathosis and clinical therapy both qualify as determinate environmental conditions.

In the case presented the "pull" of the labial arch wire is purely orthodontic. This "environmental condition (perturbation) is qualitatively distinct from the orthopedic "push" of the acrylic panels in the Max 2000 appliance. In the case of the PAOO™ procedures the surgical trauma itself and the actions of growth factors in the allograft qualify as environmental conditions sufficient to overcome canalization (buffering) of the developmental path to render a given clinical result, (alveolar bone de novo). Thus, it is quite logical and consistent with molecular biology and cellular genetics that different

* See Pigliucci, 2001

qualitative and quantitative perturbations would necessarily elicit different phenotypic clinical outcomes.

CONCLUSIONS

We have proposed that the ultra-light, self limiting force of the embedded nickel titanium springs in the appliance has activated a bone "mechanostat" phenomenon described by Melsen, (Cacciafesta, 2006) the Frost-Jee collaboration (2002) and by others (Pavalko, 2002, Chung 2003) in variable contexts. The appearance of woven bone in this case histological section and analysis of the fractal pattern therein suggest that remodeling of the alveolar bone was indeed occurring when the sample was taken.

This serendipitous observation gives serious pause to claims about the behavior of bone around teeth which are moved orthodontically. Specifically its immutability and the tendency of dental arch "expansion," to cause gingival recession must be reconsidered in light of this clinical demonstration. "Arch Expansion" is a dangerously vague term subsuming, flattening the Curve of Wilson, separating the maxillary suture, flaring incisors labially to original positions, tipping or bodily movement of molars buccally or labially or in our case, physiologically recapturing the alveolar phenotype and engineering it de novo. However it is defined, expansion may indeed, in a narrow epistemological sense, "cause" bony dehiscence sometimes. However this prediction can neither be defended with categorically certitude nor in any individual case, described as a foreseeable, inevitable event. Indeed the incidence is best characterized as infrequent, rare and easily remediable. A meaningful correlation between labial movement and gingival dehiscence is simply "not there". (Djue, 2002)

The practical asset this appliance has is that it acts as an "orthopedic machine" eliciting "alveolus development" in an effort to obviate bony dehiscence and periodic adjustment pain associated with other RPE appliances. The problems with such appliances derive from their reliance upon mechanical ratcheting instead of biological engineering. In that respect the PAOO™ and Series 2000® orthopedic appliances are "not your father's orthodontics".

The rationale for their clinical efficacy should at least dispel any fear or loathing that theory in the orthodontic specialty is moribund or dead. The Series 2000® appliances and surgical orthopedic innovations of Wilcko-Ferguson may well emerge as progressive ideas whose time has come in the "Century of the Biologist. With the presentation of these cases the burden to disprove universality now lies with academics and intellectual clinicians, who, through further research may confirm, refine falsify (Popper, 1971, 1992) or reject the legitimacy of these conjectures. Only time will tell how prescient these proposals will prove to be.

Meanwhile, the fact remains: clinical evidence of phenotypic changes and labial alveolus remodeling have been associated with PAOO™ surgery and the Max 2000® appliance, clinical phenomena philosophically consistent with a "whole bone" approach to dentofacial orthopedics.

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Appendix I: Tissue Engineering: The Non-surgical Alternative.

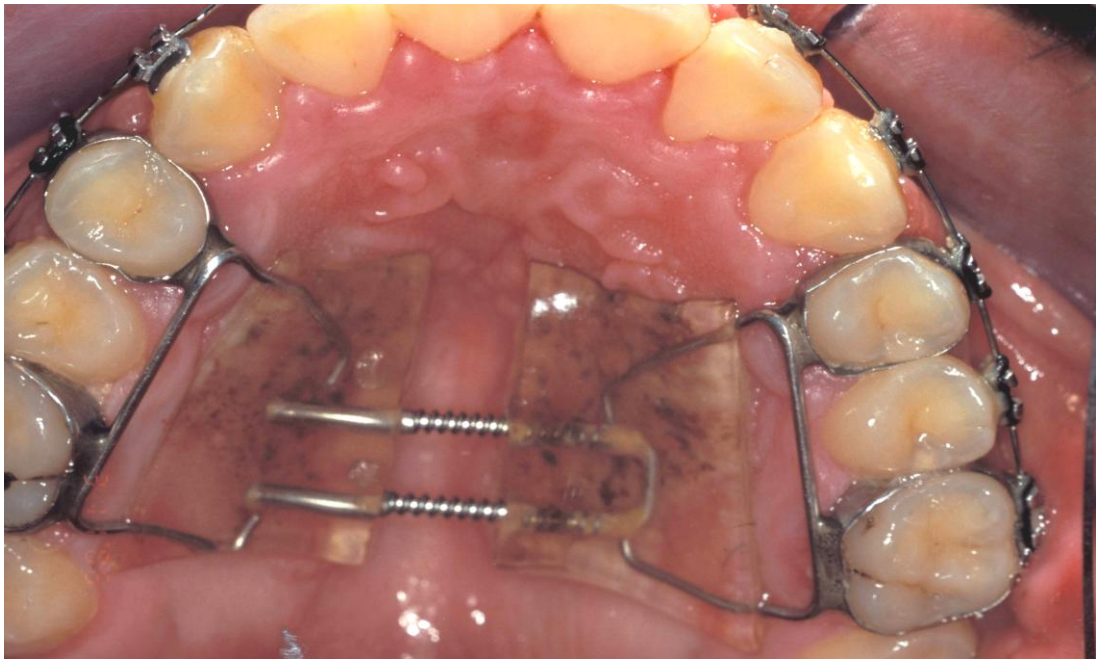


Figure 1 Max 2000® appliance applied ultra-light pressure to palatal alveolus. No attempt at maxillary expansion is made beyond the biologic signals transferred to the buccal plate through the alveolus spongiosa. The bands on teeth are for retention only and the buccal arch wire is passive. (Orthodontist: Neal C. Murphy, DDS, MS, Oxnard-Ventura, California, USA) (Max 2000® is a registered trademark of Dr. Michael O. Williams, Gulfport Mississippi.

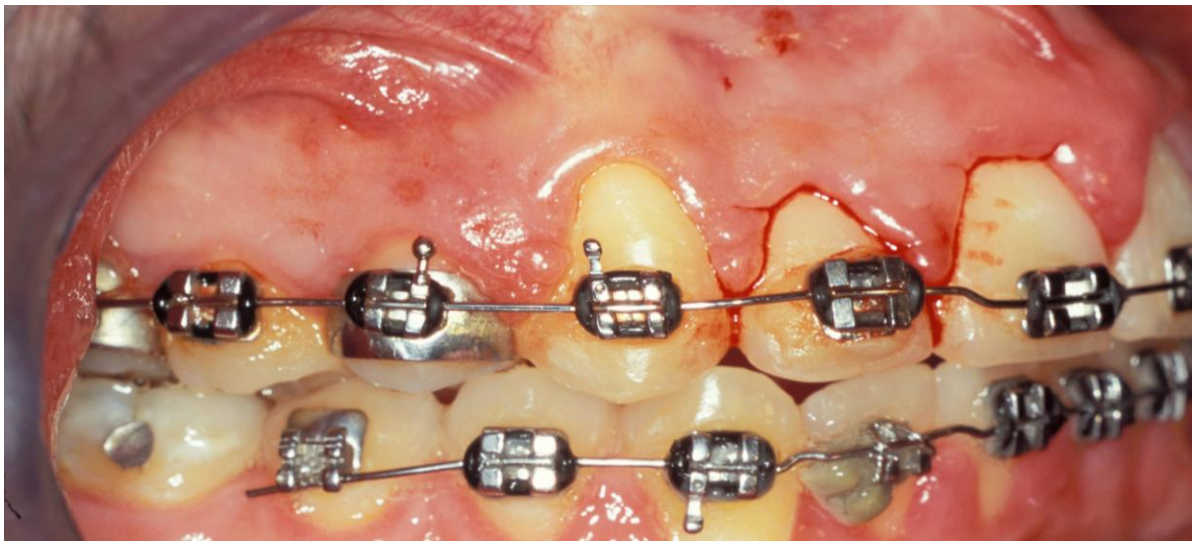


Figure 2 Case #1 E.R. Noncompliant 18 y.o. Hispanic male. Arch wire is passive.

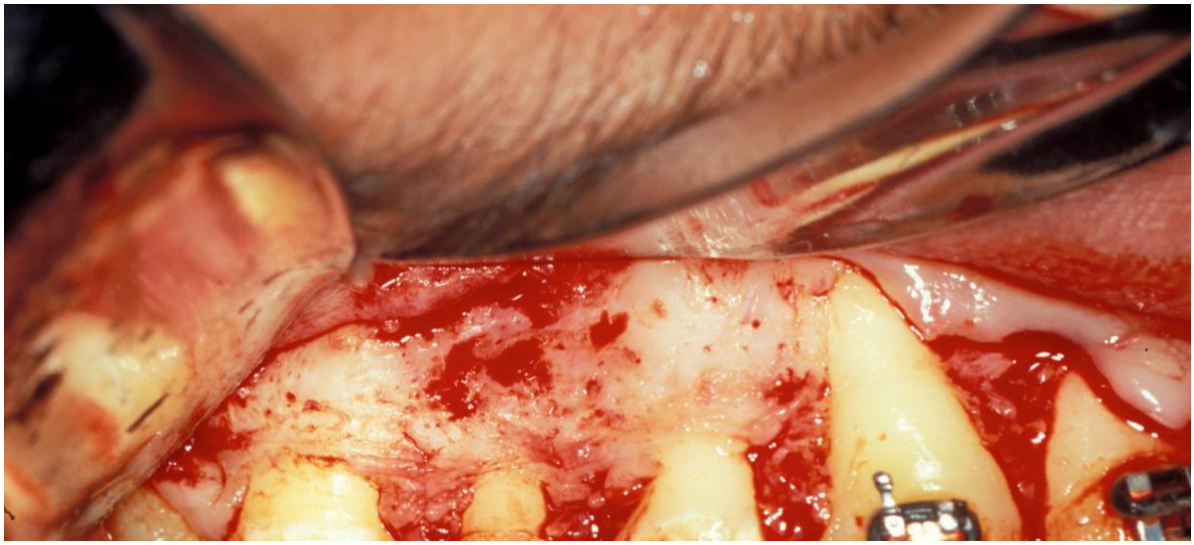


Figure 3 Case #1 E.R. Periodontal surgery reveals buccal bone where conjecturally palatal alveolus forces were transferred to buccal cortical plate, flexing the bone to stimulate osteogenesis in areas of periosteal compression. Note marked dehiscence where labial arch wire "pulled teeth beyond phenotypic range.
(Periodontal Surgeon: Dr. Neal C. Murphy)

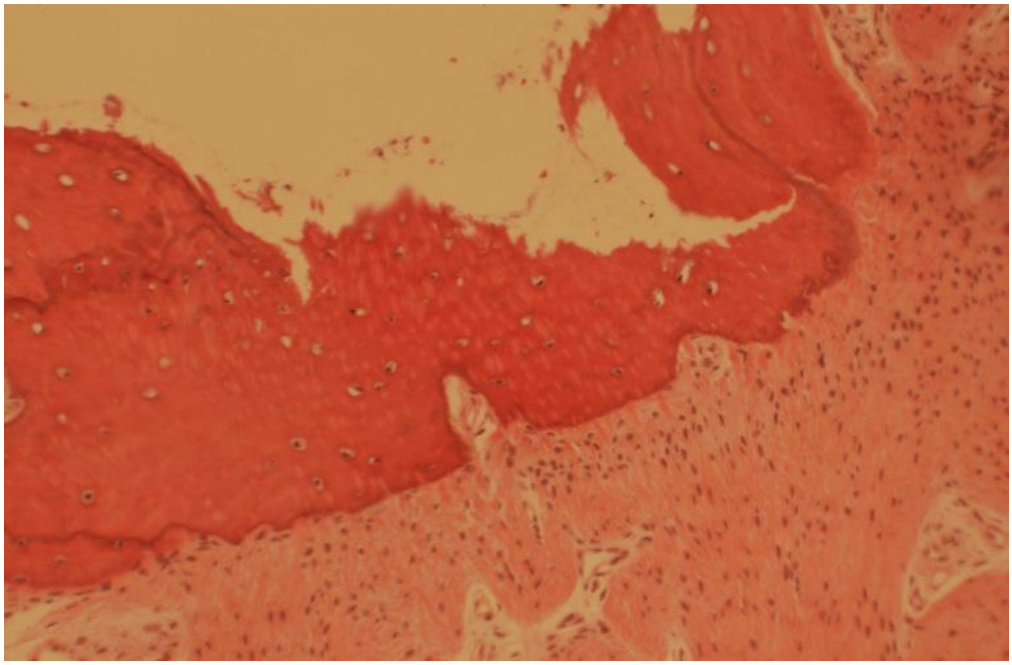


Figure 4 Routine H&E histological section at buccal aspect of tooth #5, labial to Max 2000® palatal alveolus development appliance. Note absence of lamellar pattern characteristic of mature bone.

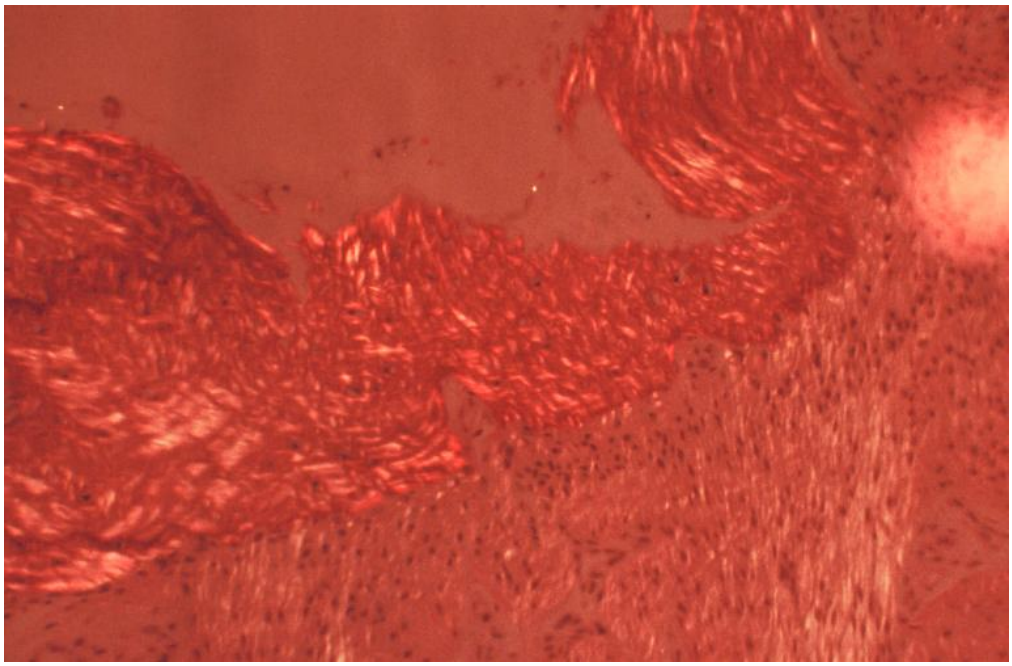
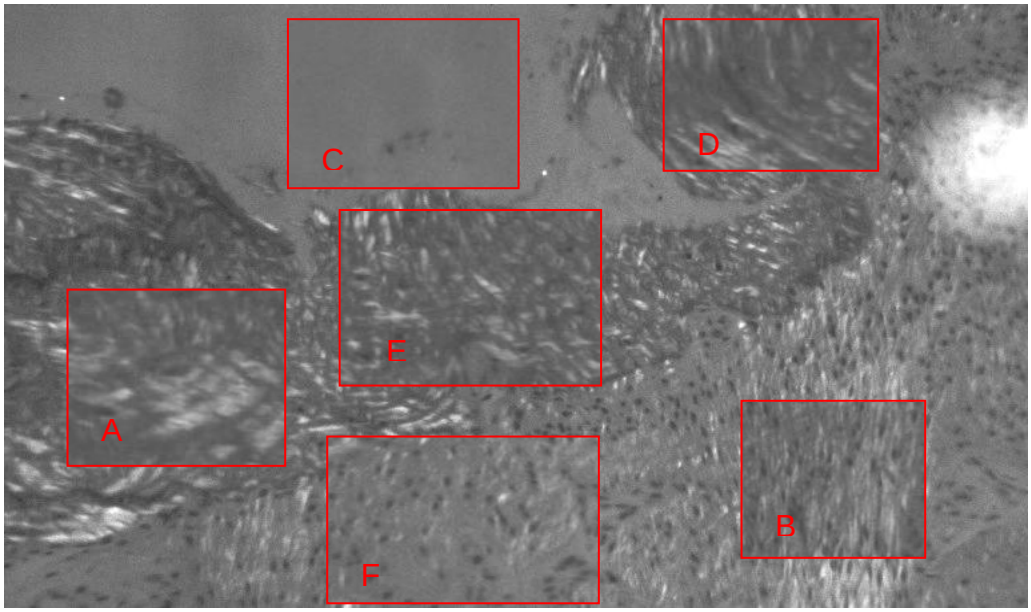


Figure 5 Polarized light section of specimen above. Note “woven bone” pattern characteristic of immature bone and regional osteogenesis.



Area	Fractal Dimension	SD
A	1.18002	0.009829
B	1.14476	0.023592
C	1.44143	0.00472
D	0.97931	0.010024
E	0.94117	0.009662
F	1.07864	0.003869

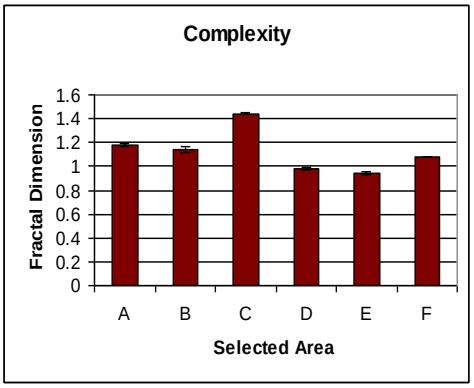


Figure 6 Fractal Analysis demonstrating bone remodeling. (Collegial gratitude is extended to James Borke, PhD, Medical College of Georgia for his consultation and the fractal analysis.)

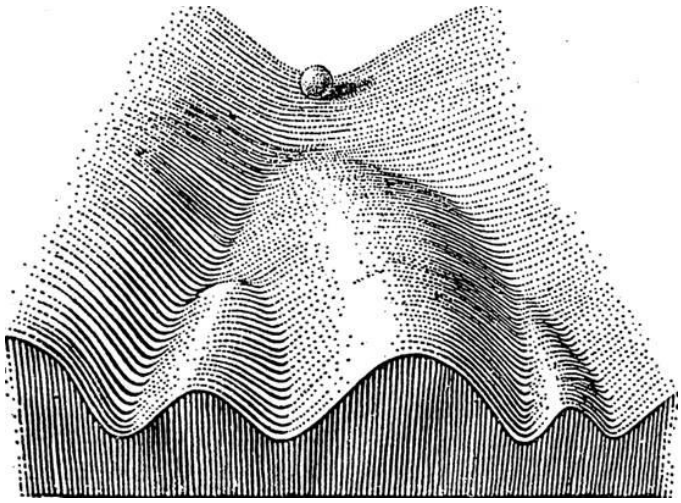


Figure 7 Waddington's Epigenetic Landscape metaphorically illustrates the fate of tissue development as a series of alternate ("canalized")fates buffered by variable heights of ridges that represent "environmental perturbations" altering the progress of a rolling ball the "genetic component" of morphogenesis. The height of each canalized path represents a kind of "energy of activation" threshold needed to alter the canalized" developmental path. Genetic expression interacts with and indeed is defined by environmental (epigenetic) influences. Genetic expression is not represented by a fixed phenotypic form directed by genotype. Thus the question is not, "…nature or nurture?", but rather "nature and nurture interacting together to manifest variable phenotypes. Thus, genotype is not so much a "blueprint" of phenotype but rather an "instruction manual" on how tissue should "act" in variable environments to manifest various phenotypic potentials.

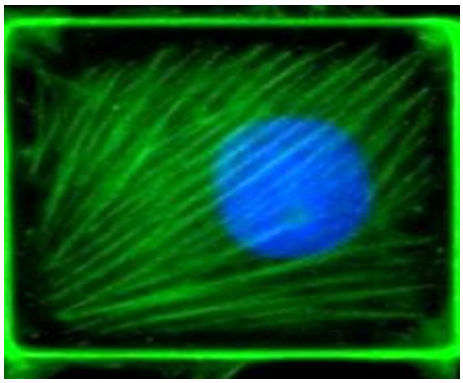


Figure 8 Demonstration of cytoskeleton deformation *in vitro*. Ho: this phenomenon in alveolar bone *in vivo* facilitates altered genetic expression as optimal response" is elicited by ultra-light orthopedic forces. Bending bone bends DNA and intracellular proteins altering genetic expression to change regional phenotype. With apologies to Wolff and his famous law, (Form follows function") it seems that on an intracellular level, "Function follows form". Green: actin filaments, Blue: nucleus (See Pavalko, 2002 and Pigliucci, 2001)

Appendix II: Tissue Engineering: The Surgical Alternative

PAOO™ 3x - 4x faster movement of teeth, less root resorption and better quality than conventional orthodontics, more stable because regional phenotype is altered by manipulation of genetic expression.

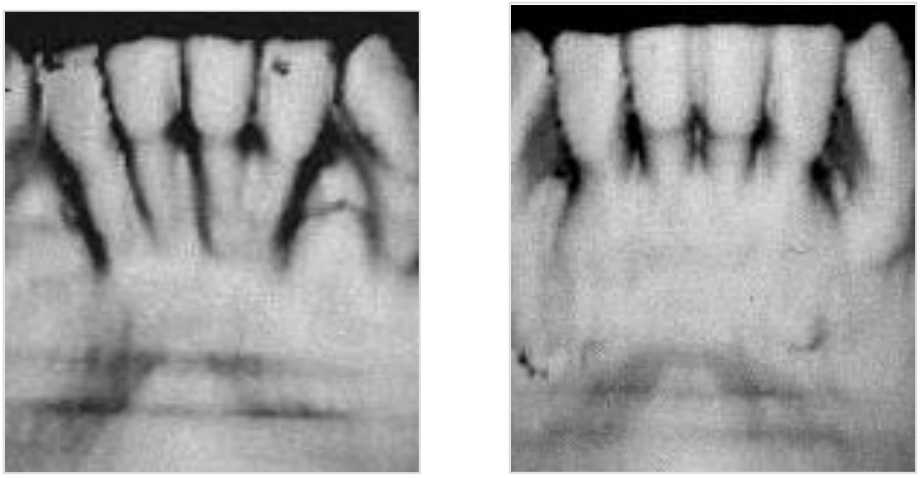


Figure 9 "Bone Morphing" altered regional phenotype after lower incisors were moved labially through a periodontal bone graft. Left is *before* orthodontic treatment; Right is *after* treatment. Most orthodontists would guess the reverse to be true but this reflects the popular conception that phenotype is immutable. The alteration of alveolar phenotype was possible because *bone healing recapitulates regional ontogeny.* (See Murphy, 2006)
(Surgeon: Dr. M. Thomas Wilcko, Orthodontist: William M. Wilcko, Erie, PA, USA, used with permission)

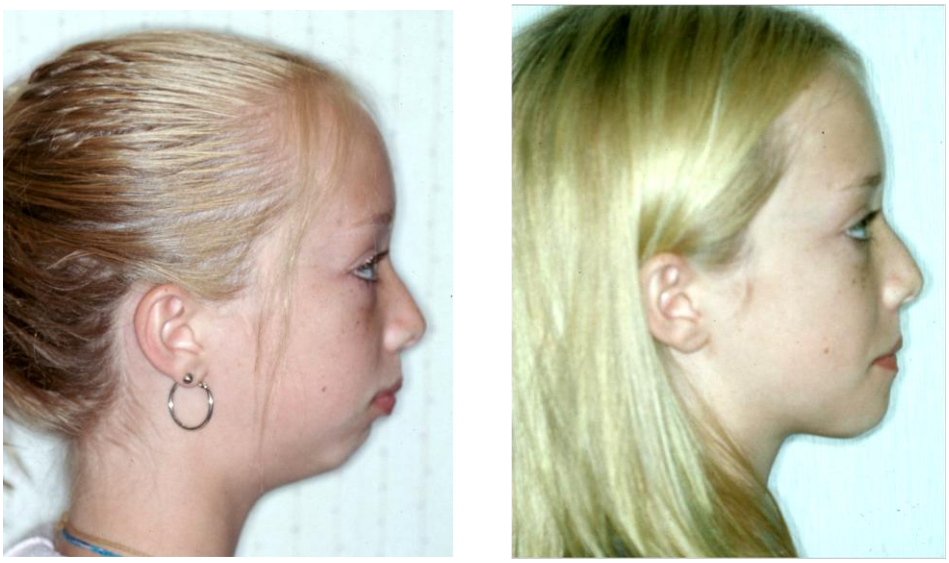


Figure 10 “Face Morphing” Note before and after facial esthetics when orthodontic forces are applied to bone as teeth roots (alveolus functional matrix) are moved through a healing bone graft. No orthognathic surgery was rendered in this case.
Surgeon: Dr. M. Thomas Wilcko, Orthodontist: William M. Wilcko, Erie, PA, USA, used with permission

Figure 11 Demonstrates the importance of developing a “large” smile that the adolescent can ‘grow into” rather than a “small adolescent smile that the patient grows out of”./ The adolescent, seemingly disproportionate smile in youth is also compatible with the enlarged face. The broad smile defines beauty in both age cohorts and reflects the prevailing 21st Century archetype for facial esthetics.

(Photos and quote compliments of Dr. Michael O. Williams, Gulfport Mississippi, USA inventor of the Series 2000® dentofacial orthopedic appliances. Used with permission. See: www.gulfcoastorthodontics.com)