To: All First-Year Residents

From: L. Johnston

Re: "Facial Growth" 603

Date: December 13, 2001

2002, Tuesday, 10-12, B312B

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Organizational details are subject to modification as the course unfolds; however, as things now stand, I plan to give a midterm and a final. The midterm will deal with the prenatal molecular biological material; the final, the postnatal. The two examinations will be of equal weight. In the past, the midterm has employed essay questions; the final, a mixture of objective and essay questions. This year, the schedule argues that the reverse will be true. The course will be graded A, B, C, etc.

December 12, 2001
Facial Growth 603

Assignment 1—Cranial Vault and Cranial Base

For: January 29, 2002

Some of you may plan to attend the AAO’s "International Symposium on Early Orthodontic Treatment" soon to be held in Phoenix (Feb. 8–10). Central to a discussion of early treatment is the topic of "growth modification." Scrutiny of the AAO Early Treatment program, however, reveals that little attention is to be paid to the mechanisms of facial growth. Why? Probably because there is no "market" for this type of information. Instead, it is taken as an article of faith that our appliances—perhaps because we refer to them as "functionals,"—can grow faces. Can it really be so easy? I think that if you are going to grow faces, the least you can do is pay your dues by investing some time examining the biology of facial growth.

This is the first of several annotated reading lists. My comments are designed to assist you in digesting the papers. Accordingly, I expect you to prepare for class by reading both the comments in the reading lists and then the papers, themselves. Should it become apparent that a class has been unable to prepare adequately, I will reschedule the meeting, rather than soldier on alone.

Facial growth courses in dentistry/orthodontics commonly deal with the grosser aspects of facial development—what forces cause the cranial vault to expand, what pushes the mandible downward and forward, etc. In other words, we usually study facial growth at a level that seems comparable to that of contemporary clinical intervention ("orthopedic" forces, distraction osteogenesis, "functional appliances," etc.) and the effects we hope to produce. In 2001, however, there is little in the way of demand for this type of information—it is expensive, time-consuming, and occasionally even bad for business. As students of orthodontics and pediatric dentistry, however, we are obliged to do better—to be more skeptical, to be better informed, to be rational. Unfortunately, we occasionally will have to make do with elderly (but classic) papers. Nowhere is this more obvious than in the study of the . . .

CRANIAL VAULT AND CRANIAL BASE
Cranial Vault

It was once thought that bone growth—both transformative and translatory—could be explained solely in terms of the genetic control of deposition and resorption. Although these surface changes are, in the last analysis, the material cause (in the sense of Aristotelian causation, q.v.) of the growth of a bone, it is difficult for us to comprehend or interact with a genome that could specify in advance the exact mission of every osteoclast and osteoblast. Instead, we now assume that it is the bone's local environment that somehow controls surface deposition and resorption. In the end, however, this formulation may prove to be just about as complicated as the purely genetic "explanation" of facial growth we are trying to avoid. In any event, a survey of the control of bone growth seems a logical place to start:

In an attempt to simplify things, craniofacial biologists commonly seek an understanding of the factors responsible for gross translation in space. This approach assumes that the surface changes are a secondary response to the new physical and functional environment encountered by the bone as its position changes.

*Fibrous sutures (syndesmoses)* were the first structures whose growth was thought to generate the "tissue separating" forces thought to be responsible for translatory craniofacial bone-growth:


As you read this paper, reflect on the significance/meaning of the book's title. With respect to the assigned pages, please note that hypotheses involving the generation of force by the proliferation of sutural connective tissue require a considerable suspension of disbelief: can bone form (rather than resorb) in the face of the pressures supposedly generated by proliferating sutures? The key demonstration that sutures may not constitute "growth centers" (as opposed to "growth sites") for vault growth came from Melvin Moss' Ph.D. dissertation, in which he argued persuasively that it is the growth of the brain that is the primary cause of vault enlargement. For a classic and influential summary (indeed, according to the editors of Current Contents, one of the most frequently cited papers in all of science), see:


Agreement with Moss' explanation of vault growth (and, by extension, orbital growth) is now almost universal. Perhaps as a result, he has attempted to apply this same mechanism (the expansion of a central, expanding mass) to all regions of the head. Because the various versions of this "functional matrix hypothesis" constitute an important force in contemporary craniofacial biology, now is as good a time as any to become familiar with Moss' concepts and his terminology (a confusing concatenation of assuasive neologisms):


In considering Moss’ ideas, be sure to consider carefully the concrete details: what is a matrix? What is a periosteal matrix? A capsular matrix? A microskeletal unit? A macroskeletal unit?

**Cranial Base**

Although most workers no longer believe that fibrous sutures can function as growth centers, many—perhaps most—believe that the interstitial of *cartilaginous* sutures (*synchondroses*) is a major factor in the elongation of the cranial base:
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Scott, J.H.: The cranial base. Am. J. Phys. Anthropol. (n.s.), 16:319-48, 1958. [Read the first part carefully; however, you may if you wish merely skim pp. 333-46; however, read the last paragraph or so—Scott was a man of considerable intellect and sensitivity; I was his student 40 years ago and can attest to the quality of his mind.]

Hypotheses concerning the importance of primary hyaline cartilage to the growth of the cranial base follow a line of development that will be repeated for each region of the head:

1. The interstitial expansion of primary cartilage generates force and does not depend on the environment (i.e., it will show "donor specific" growth when placed in a "neutral" environment); synchondrose are primary cartilage; therefore . . . —


2. Synchondrosal destruction alters the elongation of the cranial base—


Moss, however, has hypothesized that all cartilage growth—even that of epiphyses—is regulated by environmental influences. Thus, he would argue that papain and radiation and surgery merely interfere with the ability of cartilage to respond to its "capsular matrix," in this instance, the growth of the brain. We will run into this argument again and again. For now, consider the evidence and begin to decide who is right and why. . . by 1/29/02.

Finally, I ask that each of you read and digest the papers on your own. No abstracts; no dividing up the readings. I will expect you to be able to provide details of the assigned readings when called upon in class. Do not fall behind; this course gets no easier the second time around.
As with the cranial vault, midfacial growth was once "explained" solely on the basis of surface changes. Although deposition and resorption are ultimately responsible for the growth of the facial skeleton, craniofacial biologists have sought refuge in the assumption that these surface changes are a secondary response to "environmental" changes accompanying bodily midfacial translation caused by the forceful proliferation of tissues in key areas of the face ("growth centers"). The first of these simplifying "theories" was the so-called "pterygoid buttress hypothesis" (PBH):

Many, however, were uncomfortable with the idea that bone can generate enough force to produce bodily translation of maxilla, palatine bones etc. (i.e., they didn't believe that bone could withstand the resulting pressure). In any event, the anatomical assumptions of the PBH proved to be wrong—-in children, the tuberosity doesn't lie immediately in front of the pterygoid plates. In line with current thinking re vault growth, researchers then turned their attention to the proliferation of fibrous sutures (the "four paired, parallel suture theory"):


From the standpoint of science, this was an excellent hypothesis: it could be tested. Unfortunately, it just didn't work out. Sutural ablation had no obvious impact (see, for example, Sarnat, 1991, p.)
At about this time, a Northern Irish anatomist (and dentist and physician) resurrected a venerable 19th century hypothesis that ascribed a central role to the nasal septum. In its original form, the vomer was thought to be the key; in Scott’s version, it was the cartilage of the septum:


This, too, was a good hypothesis; moreover, it appears to have survived the most obvious test, septal ablation. The following papers summarize a variety of experiments designed to examine the mechanism of midfacial growth:


The contention that anything—sutures, cartilage, or whatever—can push bones around without causing resorption is of some concern; however, it may be that the septum pulls the midface forward (but perhaps not downward) in space:


Moss and Salentijn, however, disagree. Examine once again “The primary role of functional matrices in facial growth” from the last reading list. Needless to say, there have been numerous attempts to subject their ideas to experimental verification. Rather than assign one of each kind of study, the following turgid summary commonly inflicted on orthodontic and pediatric dental residents throughout the world should more than suffice:

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1Do not infer that the midfacial sutures are of no significance. They are important sites of adjustment and, as such, are the basis for much of our “orthopedic” therapy with protraction headgear and face masks.

2But is ablation a good test? Give it some thought.

3In case you think that master’s theses are mere “exercises” that have no impact, check the Literature Cited in this paper. Also, please note that it was Nick Palmer’s thesis that paved the way for this bit of research.
MANDIBLE

In both the 1999 Moyers Symposium, Growth Modification: What Works, What Doesn’t, and Why and in the 2001 Moyers Symposium, Orthodontics in Four Dimensions, there was an extensive discussion of "growth modification," most commonly mandibular growth modification. It is interesting to note, therefore, that the mechanisms by which this modification takes place received only scant, wary attention. The apparent "disconnect" between theory and practice makes for fascinating reading and a measure of sober reflection. During the course of your study, you should take time to ask yourself how orthodontic treatments designed to "grow faces" are supposed to work. Absent any rational answer, you would be well advised to proceed with caution . . . .

Based on vital dyes and ingenious experimentation, the pattern of surface transformations ultimately responsible for the growth of the mandible is well known and has been well known for about two centuries. From time to time, these data are repackaged and refined; however, the general picture remains the same. The most recent reincarnation of this genre involves painstaking microscopic analysis, rather than vital dyes. It might be subtitled, "1001 things you can do with osteoblasts and osteoclasts"\(^1\):


As has been noted in earlier discussions, it commonly is assumed that these surface changes are, at least in part, a secondary response to a changing interaction with the local environment brought about by the mandible’s translatory growth. In line with this assumption, it was once believed that this bodily movement is caused by the forceful growth—both interstitial and appositional—of the cartilage of the mandibular condyle (the "condylar epiphysis"):


The types of experiments designed to test this hypothesis

\(^1\)I do not expect you to remember all the details. I do, however, expect you to: 1) be familiar with the repertoire of effects that surface changes can have, 2) understand the "expanding V" concept (and figure out that it is just a description of the only way a hard substance can "grow," and 3) have a rough idea of the sites of mandibular deposition and resorption.
are similar to those employed to test the significance of other putative "growth centers." For example, consider the significance of the condyle as revealed by its surgical removal:


It is interesting to note that many workers have performed condylectomies, and all have seen about the same outcome; the only difference is in their interpretation of the data—is the glass half full or half empty? As might be expected, Moss weighs in with the most extreme interpretation:


Think carefully about the results of this paper (it is discussed in some detail in Jaundiced Eye from the previous list, q.v.\(^2\)). Firstly, is the growth depicted in Moss & Rankow really "normal"? Secondly, even if condyles normally are responsible for pushing the mandible downward and forward, would it be reasonable to expect a complete lack of mandibular translatory change/growth in their absence? **Be prepared to discuss these questions.** At any rate, Moss inferred from this datum (note the use of the singular) the concept that the expansion of the "oropharyngeal functioning space" is responsible for mandibular translatory growth:


Stop for a moment and ask yourself a simple question: what is the physical mechanism by which "functioning spaces" expand capsules and, in the process, produce bodily translation of the bones of the face? Look up the word teleology. **Be prepared to discuss this question.**

Some feel that neither the classical Charles\(^3\)/Sicher hypothesis nor the newer functional matrix hypothesis (in any of its many versions) is consistent with the available data. For example, from the first reading list we learned that Copray, Jansen, and Duterloo (1986) employed organ culture to see whether

\(^2\)Note: q.v. means "which see." In other words, I’m asking you to go back and look at this paper in the context of mandibular growth.

\(^3\)The idea of a condylar epiphysis was introduced by S.W. Charles in 1925 (Brit. Dent. J., 46:845-55).
or not various "growth" cartilages, including that of the mandibular condyle, have an intrinsic, forceful growth potential. Based on their data, does the condyle qualify as a "growth center"? Review this paper and be prepared to discuss this question.

Although they may have little concern and/or appreciation for the mechanisms involved, it is clear that contemporary advocates of "functional" appliances feel that the condyles are in some way crucial to the overall growth of the mandible. Thus, although it is no longer fashionable to speak of a "condylar epiphysis," considerable time and effort are expended in the demonstration of condylar plasticity by those who claim the ability to modify the growth of the mandible. To this end, Petrovic has devised an influential "cybernetic" hypothesis that serves as the theoretical basis for much of our contemporary functional appliance therapy (incremental activation, "phantom activator" effect, etc.). The following reference is very long; however, if you ignore his tables and attend instead to the figures (especially Fig. 7, p. 145) and the text, your burden will be eased. Your goal is to understand Petrovic's concept of the role of lateral pterygoid traction on mandibular (condylar?) growth. Please note that Petrovic's group uses the term "passive" to mean that the appliance exerts no force. Today we might reverse the terminology--active for an appliance that makes the patient move his/her mandible (as with, say, an activator) and passive if the appliance does all/most/some of the work (e.g., a Herbst).


-----------About to here on February 12-----------

Although left conspicuously unsaid, it is clear that Petrovic thinks/thought that the condyle is in some way the key to mandibular growth. Unfortunately, however, in the absence of a demand for proof, functional-appliance devotees are exasperatingly vague concerning the contribution of the condyle to the growth of the mandible as a whole. It is an orthodontic version

4A phrase made respectable in the USA by Moss' use of the terms "function" and "functional" in his various hypotheses.

5A way never to be wrong is to avoid the issue. Note, for example, that Enlow almost never goes beyond description. He is, therefore, the modern incarnation of Hunter and Brash (people who described growth sites by way of vital dyes). Questions about mechanisms commonly are dismissed as inappropriate or worse.
of "don't ask; don't tell." For an overview of a number of elderly studies designed to test and discuss the various competing hypotheses, see:


The above-mentioned studies argue that condylar growth is both important and modifiable (slightly in terms of amount; greatly in terms of direction). Based on these papers, do you think it appropriate to classify the Herbst appliance as a "functional" appliance? Does it seem to have a mode of action that has anything in common with that of the activator family of appliances? Be prepared to discuss these questions. In the final analysis, whatever the mode of action, it is appropriate to consider the long-term significance of the "extra" growth that is said to result from functional-appliance treatments. Does it have ponderable long-term clinical significance? Can it be seen as a substitute for surgery? Will it reduce the need for bicuspid extraction? Be prepared to discuss these questions. Consider the following, the first of which is a landmark paper:


In the midst of all the controversy (Yes, there is controversy!), it is easy to lose sight of the fact that many of the world's greatest authorities on functional appliances (e.g., people like Wieslander and Panchez, both of whom have a considerable vested interest in a positive outcome) have been unable to demonstrate a long-term mandibular growth effect. Others (e.g., Bimler, Harvold, and perhaps Woodside) have not tried, apparently because they do not believe that functional appliances can grow mandibles. As you consider this question, ask yourself whether it is important that the extra mandibular "growth" commonly seen during the "functional" phase of treatment be permanent (i.e., real) or whether a temporary acceleration would be good enough.

"At least I think it is a landmark paper: 1) The World's senior active functional appliance/Herbst clinician reported that he could not detect a long-term increase in mandibular length and 2) in the process, he called to our attention past conclusions--optimistic, influential conclusions--that proved to be in error.
Indeed, ask yourself whether a temporary acceleration has to be anything more than a just a transient “Sunday bite.” Be prepared to discuss this question. The following may help:


Envoi: Give these readings careful consideration and remember them when confronted with the necessity of obtaining informed consent for early treatment. The first person to be informed is you. Caveat emptor and lector.
"Prediction is very difficult, especially with respect to the future."

-Anon.

This reading list is designed to introduce the gross pattern of facial growth. It does not deal with mechanisms, either gross or cellular. For example, regardless of mechanism (septum, expanding spaces, sutures, etc.), what changes can we expect in the absence of treatment? With treatment? Accordingly, this may be the most immediately important discussion in the entire course.

At the start of the 20th Century, orthodontists thought that they could grow optimal faces by the simple expedient of creating a Normal occlusion. Perhaps as a result, orthodontists once expressed at least a mild interest in "Science"—biology, physiology, embryology, etc., largely because they thought that orthodontics could have a profound effect on the face. The "bone-growing" era came to an abrupt end, however, when the results of the first longitudinal cephalometric studies were published. Firstly, there proved to be nowhere near the individual variation over time that orthodontists had expected. Secondly, six months or so after birth, facial growth proved to be "linear," a finding that was equated with "unchanging." As a result, many rushed to the conclusion that the facial pattern is established early, never changes, and, based on faulty superimposition methods, can't be changed. To examine the source of this influential idée fixe, skim the following, paying particular attention to pages 226-59:


Figs. 17 and 18 and some 9th grade algebra (graphing of functions) should prove useful in evaluating the inference that linear growth implies constant proportion. I will expect informed participation in the development of this point. Secondly, when Brodie's group (Brodie, Downs, Goldstein, and Meyer, 1938) examined Class I, II, and III nonextraction treatment, they were drawn to the conclusion that orthodontics has little if any effect on the growth of the face ("Actual bone changes accompanying orthodontic management seem to be restricted to the alveolar process"). Indeed, it was even suggested that molars can't be moved distally, a claim that has taken on a life of its own and which was discussed in the Summer cephalometrics course (consult your notes). The outcome of these two reports (not to mention Broadbent's early papers) was the so-called "pattern concept," the widely held view that facial form does not change and cannot be changed. As enunciated in an influential review by Brodie (1946):

The growth of the pattern is proportional. This means that the disharmony is present from before birth; it gets neither better nor worse. It cannot be changed by treatment. The teeth and the alveolar processes constitute the only area of the face where change may be expected or induced.

\[\text{Note: in Angle's terminology, Class I is a malocclusion.}\]
The impact of the Pattern Concept was profound. As noted by Brodie in 1950 (Angle Orthod., 20:21-38):

When the staff at Illinois in 1937, [sic—this means that the comma is a mistake by Brodie in the original manuscript, rather than a mistake of mine in copying it] presented its findings on the results of treatment we were all shocked by the revelation that we were influencing only alveolar bone. There was a universal attitude of despair. Drilled in a concept that was as tangible as the occlusion of the teeth, it came as a terrible disillusionment to discover that the concept was not tenable. It was of the nature of childhood’s chagrin and disappointment upon finding that there is no Santa Claus.

Reproachful eyes were cast on the teachers who had perpetrated the fraud, and without more ado the rush was on to discover how the prophets of the new school were answering their problems. The rush had all of the characteristics of any stampede. There was no glancing back, no weighing of values. Facts and tenets learned by hard experience, all that had come to be accepted over the years, were ignored. Almost over night there was a reversion to the stage of tooth alignment with no thought given to occlusion, to muscle balance, to type or to tissue response. These were matters to be left to the theorists who had caused all the misunderstanding in the first place. Let them continue to play with them if they wanted.

A half century later, whenever you hear someone say that a headgear holds the molars while the maxilla grows downward and forward, you are witnessing the intellectual constraints of the Pattern Concept in action. Indeed, if nothing changes and nothing can be changed, many respected clinicians have concluded, “forget growth; treat the teeth” . . . and then charge double for adults. What is there about being young that makes a malocclusion easier to correct?

First and foremost, children grow. Contrary to what Brodie said, the normal (average, usual, probable, most likely, expected, mean, etc.) pattern does feature changes and these changes appear to be of potential benefit in the treatment of Class II malocclusions. Contrary to what you might expect or may have read (“Them that has, gets”), these changes are seen both in Class I and Class II patients:


Note the general nature of the changes. What is the morphological impact of the average pattern of growth? Are “good” changes seen only in “good” faces? (Read Lande, pp. 86-89, carefully; also read Johnston, 1986, pp 115-9 in the second section of the present list; remember these data when it comes time to discuss prediction.) Although patients commonly show a favorable pattern of change², many influential orthodon

²Recently I was asked by AJO/DO to referee a paper that was highly critical of the pitchfork analysis because of its reliance on midfacial, rather than cranial-base, superimposition. I recommended that the paper be cleaned up a bit and then published. Apparently the author declined, because the paper actually appeared later in another journal—unchanged (Männchen, R: Europ. J. Orthod., 23:1-14, 2001).
tic "leaders" argue that "growth" has no clinical significance because it will not in and of itself correct a malocclusion. To evaluate this argument, you need to learn more about the normal rotational pattern of jaw growth and its effect on the occlusion:


While we are at it, we might as well re-visit Steiner's "incisor compromises," which, in the presence of a "Class I" molar relationship, emerge as a natural dentoalveolar responses to various maxillomandibular relationships:


Gordon Gecko (Michael Douglas) said "Greed is good." From the standpoint of orthodontics and the Class II malocclusion, I will argue that growth is good. Accordingly, can you see how the usual pattern of growth might be helpful, even though in the absence of treatment it would leave a malocclusion largely unchanged? Unfortunately, not all patients (both Class I and II) grow "normally"—some do better, some do worse. Accordingly, it might be useful to be able to predict the individual pattern of facial growth:


Questions that will be asked of you in class: What is the basis of Ricketts' predictions? How are the various elements to be combined to yield a prediction? How accurate are his predictions? Did he say? How accurate would you have to be? Be prepared to respond to these questions.

In the years since Ricketts first publications, cephalometric programs (e.g., dentofacial Planner) have taken the heavy labor out of generating a so-called "VTO." Sad to say, the advent of the PC and thirty years of experience argue that an individualized prediction scheme (any method that would enable you to predict a different pattern for two subjects of the same age and sex) has not yet been demonstrated and may in fact be impossible. Infer the bases for this assertion of impossibility from the following turgid little opus:

Johnston, L.E.: A statistical evaluation of cephalometric prediction.

note that, if treatment produces a clockwise midfacial rotation that exceeds the usual counterclockwise rotation, this differential would lead to a positive apical-base change. Accordingly, you might be wise to assume that the pattern is favorable, but perhaps not quite as favorable as would be implied by and inferred from my paper.

3A vague term that presumably refers to the change in pattern that growth produces.

4As you think about prediction, what would you predict, given a perfect prediction scheme? I doubt that this question has ever been given much thought.

Nearly a quarter century later, orthodontic alchemists are still trying to divine signals from noise.\(^5\) Even prediction schemes that seem good, fail when put to the acid test of validation:


So much for Science! Given that science does not seem to be as clinically useful as was once hoped and assumed, many orthodontists--"Once burned, twice shy"--have decided to go off on their own. Experience, after all, is supposed to be the best teacher. As we will see, clinical intuition fares even worse.

-------------------To about here for 4/9/02-------------------

"Orthodontics is a 6 mm specialty"
-M.L. Moss

Many clinicians, for example, are uncomfortable with and mistrustful of statistical analyses. They assert that common sense and clinical experience can succeed where "pointy-headed" academics fail:


In the real world, if an automobile doesn't run, people don't buy it.\(^6\) In the world of orthodontics, however, if a clinical stratagem doesn't work, the patient will probably still get a result that is "good enough" to allow the orthodontist to survive in practice. Thus, although many clinicians think they can tell which patients are going to grow well, reality testing is rarely an issue, especially now that the personal computer can be used to generate inexpensive, impressive, four-color, but generally useless, predictions.

Because of our inability to predict individual variation combined with the residual impact of the Pattern Concept, people tend to forget that the usual pattern is favorable (perhaps 9 times in 10). Accordingly, it can be argued that, because the by-products of the usual pattern of growth are favorable, it would suffice to be able to predict the timing of growth:


\(^5\)It is, perhaps, significant that Roth is a great believer in prediction, specifically, the so-called Visualized Treatment Objective (VTO).

\(^6\)They also don't buy it if the manufacturer advertises that it doesn't run, as did General Motors when they introduced the Nova (no va) to México.
But why bother to predict growth if it can be changed therapeutically? As you no doubt know, functional and orthopedic appliances currently constitute a major “growth industry” in orthodontics. The following review paper (assigned previously in another course) should serve as an introduction to the interaction between “treatment” and the usual pattern of facial growth:


In reviewing the foregoing, you must decide under what conditions it would be fair to compare treatment effects produced by, say, non-extraction edgewise and activators; by extraction and non-extraction edgewise; etc. (Susceptibility bias is the key phrase.) For the purposes of comparison, how would you improve on this admittedly crudely designed survey? Fortunately, it is useful merely to know the effects of each treatment within the patients on whom it is normally used.

Once again, it must be emphasized that, because of our inability to predict individual increments of change, orthodontists commonly forget that there are average changes produced both by growth and by treatment that affect almost every patient to some degree. It is, therefore, a great mistake to plan treatment (e.g. bicuspid extraction) without taking these expected (anticipated, probable, usual, average, etc.) changes into account. To see more of what the “normal” pattern of facial growth can do, briefly review the model proposed in the reading list for the mandible:


It has been said that science progresses from superstition to description to control (“control” in the sense of being able to predict and/or manipulate future events and outcomes). Based on what you’ve learned thus far, where on this progression do you think we stand with respect to facial growth? Do we walk the walk as well as we talk the talk? Do we understand “facial growth” well enough to control it? Does it matter, given that nobody ever starved because they used a defective appliance?

The recent AAO Symposium on Early Treatment attracted about 10% of America’s orthodontists. The various participants were asked to summarize their remarks for the American Journal of Orthodontics and Dentofacial Orthopedics. My summary took the form of a series of questions that would seem an appropriate way to end this assignment: