American Journal of ORTHODONTICS

Volume 69, *Number* 5, **May, 1976**

ORIGINAL ARTICLES

All in the family: Use of familial information in orthodontic diagnosis, case assessment, and treatment planning

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Orthodontics has never been a quiet, settled specialty, but the current controversy, touching as it does upon the very scientific bases of orthodontic diagnosis, case assessment, and treatment planning—not to mention a number of complex ethical issues—has commanded an *unusual* amount of attention, both at the meetings of our societies and in the pages of our journals. Computers,[1] large data bases,[2] and service agencies[3] are used to provide the clinician with norms, standards, growth projections and even appropriate treatment strategems. *Morphometrics, computerized cephalometrics,* and *clinical deviations* are all catchwords and phrases coined, in keeping with the best of advertising tradition, to divert attention from the intrinsic values of the product and focus on the packaging.

In selling the concept of a "norm," for example, it is suggested that data generated by a computer is *a fortiori* scientifically acceptable. What remains unspoken is the tacit assumption that the extent of the individual patient's departure from such norms is a reliable indication of his growth potential. Some have, of course, recognized—and rejected—this assumption. The result of the ensuing dialogue has been a bipolarization of opinion among orthodontists—

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This study was supported, in part, by National Institute of Dental Research Grant SR01-DE 03013-06.

the "computerniks" on the one hand and, at the other end of the spectrum, a group we may call the "artisans"—and an almost unbelievable amount of time and space has been devoted to publicizing the interchanges accompanying this confrontation.

It is not our intention to provide a detailed bibliographic history of these skirmishes. The extent of the chasm may be readily appreciated by reading Hixon[4] and Ricketts[5] or, for the younger genré, the well-timed, sophisticated and witty, modernized versions of the same material.[6-8] Rather, it is the purpose of the present article to challenge the premises upon which this debate is based. We inquire: *Are we—as a profession—asking the right question?* More explicitly, *Is our preoccupation with norms computed from large data bases (computerized or not!) justified?*

It is our contention that the answer to this question is in the negative; that the computer is but a calculational tool and the mere fact that it *can* accommodate large data bases has tended to mask the more fundamental question of just what of clinical import these data bases may provide; that population norms are, at best, of some anthropometric interest and, at worst, misleading as rigid, fixed treatment goals; and that the family provides the clinician with the most reliable indication of his patient's growth potential. In short, when one is considering orthodontic diagnosis, case assessment, and treatment planning, "It's all in the family."

Population norms

In a relatively homogeneous society, population norms or standards derived by the use of suitable sampling techniques might provide the clinician with a template against which his individual patient's growth record could be assessed and projected. Even given this ideal situation, however, such standards have severe limitations. These limitations stem from the fact that standards invariably represent mean (average) values. Whether or not these mean values are provided with accompanying measures of variability, such as standard deviations, standard errors, percentile values, etc., they say little, if anything, about any one individual drawn from the population from which these standards were computed.[9]

When using a set of standards, one is comparing the subject under consideration with some *standard population,* and the usefulness of such a procedure clearly depends upon the suitability of the population used in constructing the standards as a reference group.[10] While there are obvious drawbacks in using, for example, standards based on an American Caucasian sample for assessing the growth potential of American Negroes,[11] more subtle interpersonal differences (for example, in ethnicity[12]) severely limit the usefulness of this approach in our heterogeneous society. This was put well by Moss[13] during a discussion of growth prediction in a Conference on Cranio-Facial Growth in Man:

> I have heard the word "norm" and "normal" used several times this morning. I would like to know specifically about the State of Michigan: What norms do you have to predict the growth of the population in the City of Detroit from the age of 10 to 15? In a heterogeneous population of a major city, a child comes in of varying

> background, of varying economic and ethnic and genetic components. I would like to know what norms I should use for this child. If somebody has them, I would like to know about them.

Silence. Admissions that "This logical question of what we do mean by norms has not been approached satisfactorily by anybody . . . " and "I think there are . . . things that we do not want to do, such as talk about norms. Prediction is a very individual thing and is far removed from concepts of norms." There was no response to Moss's challenge. While a careful reading of the entire proceedings of this session is certainly recommended to put these comments into perspective, two aspects of Moss's inquiry are worthy of special attention in the context of the present discussion.

The first is that the answer to the question of the existence of *appropriate* norms in this situation is probably still in the negative—despite the amount of time and effort that have been brought to bear on the computerization of large data bases which, theoretically at least, should have been able to provide us with the required means and standard deviations. Indeed, almost as if in the automatic response we have come to expect from a technology in search of a question, the Center for Human Growth and Development at the University of Michigan in Ann Arbor recently produced an *Atlas of Craniofacial Growth.*[14] And this was done despite the fact that the Dental Research Institute at The University of Michigan, also in Ann Arbor, already houses one of the largest craniofacial growth studies in the world—boasting of sample sizes that result in the standard errors of the estimators of these norms being smaller than the errors with which human beings are capable of measuring the basic input data.

This monograph provides, by sex and the chronologic age groups from 6 to 16, the means and standard deviations of some 188 craniofacial measurements, each of which, we are assured, was made, on each of the individuals comprising the sample and on each of the occasions of measurement, correct to 0.002 inches by a system called TRIDEA. While these results have the virtue of at least being computed on the basis of a sample from what might be considered the "appropriate" population (Ann Arbor is less than 50 miles from Detroit, while the data at the Dental Research Institute were derived primarily from the children in Philadelphia), we are afraid that Professor Moss's question must still echo, unanswered, throughout the orthodontic offices in the City of Detroit. And in Ann Arbor. And even in Philadelphia.

In order to see why this is so, we turn now to the second aspect of Moss's statement that is especially pertinent to our discussion. This is that Moss, quite properly from the point of view of the practicing orthodontist, has tied the utility of such norms to *prediction*—prediction in the sense of establishing a regression function, or growth curve, of the form $y = f(t)$, where y denotes the craniofacial dimension we wish to predict and t the age of the individual in question, which would be used to compute the "standard" or "normal" or typical" value of y, given the value of t. How are such normative values established? The usual way is, for each age level, to compute the mean and variance of a (large) number of such measurements $y_1(t)$, $y_2(t)$, . . . , $y_n(t)$,

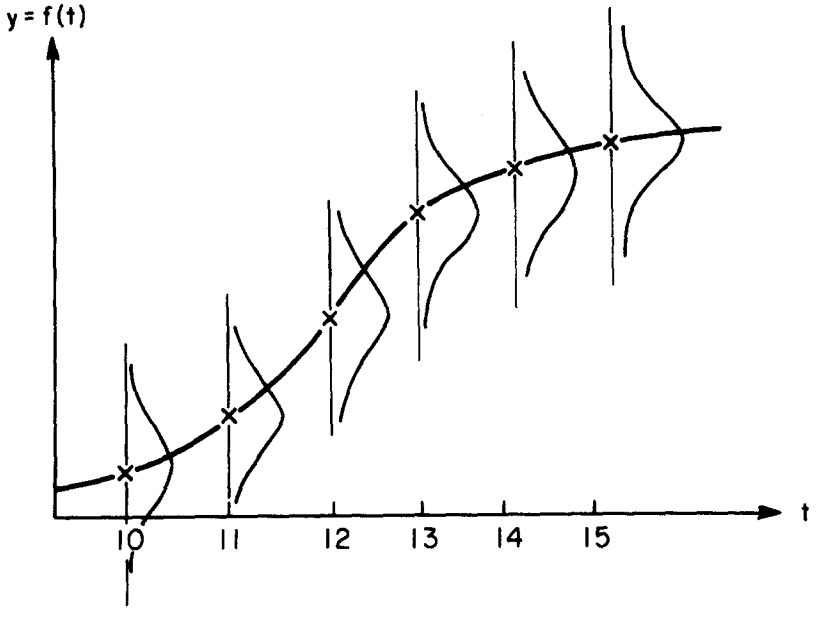

Fig. 1. Representation of a growth curve, y = f(t), showing the mean values of y from 10 to 15 years of age and the variability of individuals about these mean values.

say $\bar{y}(t)$, and $s_y^2(t)$. These may then be represented graphically as shown in Fig. 1.

Here, for any fixed value of t, such as, t = 12, $\bar{y}(12)$ is plotted as a point along the growth curve and the spread of the distribution of the observations about that point (shown roughly in the shape of a Gaussian distribution) is a reflection of the value of $s_y^2(12)$; that is, the variability of the measurements $y_1(12)$, $y_2(12)$, . . . $y_n(12)$ collected on children of t = 12 years of age. The accuracy of any predictions based on this scheme, then, depends on the values of the $s_y^2(t)$ which, as Moss points out, are likely to be large. The use of large samples allows us to estimate accurately the true *mean* values $\bar{y}(t)$, with standard error $s_y(t)/\sqrt{n}$, but *individual* growth predictions must still be made in the face of the $s_y^2(t)$.

While some reduction in this variability may be realized by employing skeletal or dental age instead of chronologic age to index development and/or producing separate growth curves for certain relatively homogeneous subgroups of the sample under consideration,[15] it is our contention that the American population is simply too heterogeneous for this strategy, at least as currently applied, to be successful. Our review of the literature has failed to identify any reductions of this type which render the $s_y^2(t)$ small enough to produce prediction equations of sufficient accuracy to be of any real *clinical* value.

While we must agree that chronologic age is a "somewhat sterile index for developmental theorizing"[16] and that other explications of the time continuum are required if we are to capture the essence of what we mean by

"biologic" time, the current state of the art precludes direct clinical application of the results that have been achieved in this area. Even less progress has been made in the careful definition and utilization of relatively homogeneous, clinically meaningful subgroups.[17] Indeed, examples abound in which the sample under consideration is not even partitioned according to the most basic of interpersonal differences (for example, sex).

Perhaps one of the reasons for the paucity of studies of this type is, as noted by Healy,[10] that when the population is subdivided according to "too many" relevant factors "large samples from such groups may be hard to come by," but this returns us to the point made earlier, namely, that large samples *per se* are less important than identifying the *appropriate* reference group. If we can for the moment ignore the "accuracy" which is supposed to accrue whenever large numbers of individuals are included in statistical investigations, we might push this line of reasoning to the logical conclusion and suggest that the problem of prediction will find its solution only when we are willing to eschew the "lore of large numbers" in favor of concentrating on more homogeneous, albeit smaller, groups of individuals, namely, *the family.*

The basic idea here is to realize, following Kempthorne,[18] that the problem of developing a good prediction system is really the problem of "classifying individuals into groups by one means or another, such that . . . the population can be partitioned into genetic classes with different norms of growth," and then of recognizing the family as the largest such class that can be identified in our heterogenous society. The "melting pot" structure of the American population precludes the use of larger groups as the basis for individual predictions, because larger groups exhibit "interaction between individuals within groups"[18]; that is, the groups are too heterogeneous to support significant—and useful—correlations between their members for use as the basis of a prediction system. While, on the other hand, one must face the possibility that "every genotype has its own particular norm of growth in a particular environment"[18]—a situation in which *all* methods of growth prediction based on the incorporation of extrapersonal data are rendered useless by definition—it is our contention that in our heterogeneous society our only reasonable chance of *increasing* the accuracy with which we can predict individual growth over and above that attainable from data gathered from the patient himself (who remains our *primary* source of information) is through the incorporation of familial data. Some support for this contention and an indication of how one may actually *use* familial data in practice are outlined in the following sections.

The use of familial information

Once the sex of a newborn child has been determined, perhaps the most common question put to the parents of the child by relatives and friends is: "Who does he (or she) take after?" The very frequency of this question attests to a kind of "common knowledge" empirical validity of the important role that heredity plays in determining the morphology (among other things) of the new individual, particularly with respect to the facial contours. As noted by Krogman,[19] "This joyful summing-up of whom the babe resembles is a recogni-

tion of the fact that physical traits are inherited, that they have a genetic basis, and that they are passed on by the maternal family-line and paternal family-line." Today the anxious search by doting parents for resemblances to loved ones has been replaced by a more scientific approach,[20] and a large number of studies have pointed to the *existence* of a genetic influence in dentofacial morphology and the etiology of malocclusion.[27]

Concentrating on the etiology of malocclusion, there are cases on the one hand where the fact and mode of inheritance are relatively simple and straightforward—as, for example, in craniofacial dystosis (Crouzon's syndrome), Hurler's syndrome, and certain of the autosomal trisomies.[20] There are the cases, on the other hand, in which the environment seems to be solely responsible —as, for example, when oral habits such as thumb-sucking or tongue-thrusting may be implicated within a homogeneous population of Class I individuals.[22] In between we have the great majority of cases in which there is a complex interplay between heredity and environment. In this last situation, the very complexity of the problem has resulted in considerable confusion regarding the mode of transmission of the traits responsible for the development of malocclusion and, consequently, has retarded the effective utilization of familial information in orthodontic practice.

To cite but one example, the inheritance of the Class III malocclusion has been variously attributed to a single Mendelian dominant gene, an irregularly dominant gene, a dominant gene with incomplete penetrance, an autosomal recessive gene, and a polygenic threshold model,[23] that is, a model in which susceptibility to the trait has a normal (Gaussian) distribution, but the population is divided into "normal" and "abnormal" types by a threshold value such that phenotypic expression of the trait does not occur unless the summated effect of the genes involved exceeds this value. Confusion even existed as to whether or not the inheritance of malocclusion was sex linked: While Litton and colleagues[23] presented evidence against a sex-linked mode of inheritance, Gorlin and his associates[24] noted an increase in both mandibular and maxillary prognathism with the addition of each X chromosome as one proceeds from the XO (Turner's) through the XXXXY syndromes.

It was against this background of obvious clinical importance on the one hand and complexity accompanied by a conflicting literature on the other that, some years ago, the Department of Orthodontics at the University of Michigan embarked on a series of family studies designed to identify the mode or modes of genic transmission of malocclusion. While current opinion at that time seemed to favor the notion that most of the characters relating to craniofacial growth and development are polygenic and continuously variable,[25] that is, that the phenotypic expression of these characters is determined by the summated effect of a large number of genes, data supporting this model were still fragmentary and the conflicting reports in the literature, alluded to above, simply did not allow the effective utilization of familial information in orthodontic practice. If we are to begin to incorporate such information into the clinical decision-making process, we must first identify the mechanisms governing the genic transmission of those traits which influence dentofacial morphology.

This, then, was the common aim of the Michigan family studies. The result was strong support for the hypothesis that the dentofacial traits of most interest to the orthodontist in assessing dentofacial morphology and planning the treatment of malocclusion depend on the combined action of genes at many different loci rather than on any single allele, that is, that *the inheritance of malocclusion is best explained by a polygenic (multifactorial) mode of genic transmission.*

The evidence for this assertion is sketched in the following section. We conclude this section by noting that, in the context of growth prediction, and given this mode of inheritance, the use of familial information *can* effectively reduce the variability, $s_y^2(t)$, which obtains whenever only population data are used to predict the value of $y(t)$ for any given individual in that population. This follows from the fact that, in the case of autosomal polygenic inheritance with no dominance and no environmental component of variation, correlation coefficients computed for each of the family member pairs (father/son, father/daughter, mother/son, mother/daughter, brother/brother, brother/sister, and sister/sister) have an expected value $r = 0.50$.[26]

Thus, the variability of the trait under consideration can be reduced by $r^2 = 25$ per cent by using the value of this measurement as observed in one of the proband's family members in a simple linear regression equation of the form

$$y(t) = a + bx$$

where x denotes the value of the measurement obtained from the family member and a and b are constants, determined by least-squares from familial data, which minimize the error of prediction.[27] Of course, if the use of a single relative can effect a meaningful reduction in $s_y^2(t)$, one would expect to do even better by using the entire family. This may be accomplished by use of a multiple regression equation relating the measurement in question ($y(t)$) to the value of this measurement observed in family members $\{x_1, x_2, \ldots, x_p\}$ by an equation of the form

$$y(t) = a_0 + a_1x_1 + a_2x_2 + \ldots a_px_p,$$

a direct extension of the simple linear regression equation discussed above.

In this situation the reduction in variation is measured by R^2, where R is the multiple correlation coefficient between $y(t)$ and $\{x_1, x_2, \ldots, x_p\}$, and significant reductions of this type for a number of clinically important dentofacial measurements have been reported.[27] We might note here that while all the family members contain information of potential value in this context, it is true that, beyond a certain point, adding additional familial information into the multiple linear regression equation will produce only slight increases in R.[2] A kind of "law of diminishing marginal returns" sets in due to the fact that relatives of the proband are, of course, related to one another and hence *their* dentofacial measurements are correlated. This means that some of the information contained by, say, a sister of the proband will have already been included if information from a brother of the proband has previously been assessed by means of R.[2]

The amount of *new* (nonredundant) information added by inclusion of the value of the relevant variable for the sister in this case is given by the *partial correlation coefficient*

$$r_{P,S/B} = \frac{r_{P,S} - r_{P,B}\, r_{B,S}}{\sqrt{1-r_{B,S}^2}\sqrt{1-r_{P,B}^2}} = 0.333$$

since all of the simple correlations on the right hand side of the above equation have the value r = 0.50. Here $r_{P,S/B}$ is read "the partial correlation between the proband and sister *given* the value of the relevant variable for the brother (B)," and this is reduced from the nominal value of $r_{P,S} = 0.50$ since the brother has already been included in the equation and $r_{B,S} = 0.50$. In any event, the above considerations imply that *if the polygenic model for the inheritance of dentofacial morphology is correct,* substantial reductions in the $s_y^2(t)$ may be realized by the incorporation of familial information. We turn now to the evidence in favor of this model.

Polygenic inheritance

A number of methods have been developed to demonstrate polygenic, or multifactorial, inheritance. The simplest of these, and the one we have used most frequently in our studies of the genetics of dentofacial morphology and growth, is the *method of resemblance between relatives.* This method is essentially the demonstration that the more closely related two individuals are, the more they resemble one another with respect to the trait or traits in question. A convenient measure of the resemblance between relatives is the simple Pearsonian product-moment correlation coefficient which, under the hypothesis of polygenic inheritance and the assumption of random mating, was shown by Fisher[26] to be the same as the proportion of genes these relatives have in common.

Thus, in particular, given intrafamilial data, one can examine the polygenic model by testing whether the observed value of r computed between family members agrees with its expected value in the context of this model. This expected value, as noted earlier, is r = 0.50 for each of the family pairs (father/son, father/daughter, mother/son, mother/daughter, brother/brother, brother/sister, and sister/sister), provided only that certain assumptions concerning the system of mating and the interaction of the genes which additively determine the trait under consideration are satisfied. The following excerpt from Tanner[28] spells out these assumptions and some points of caution which must be faced when applying this method:

> A word of warning must be inserted here, however, about the interpretation of correlation coefficients in multifactorial characters. Human biometrical genetics is an intensely complicated field, from which many of us have returned (even with our accompanying mathematicians) empty-handed and with burned fingers. It is still not always realized that traits even with complete and quite simple genetic determination can nevertheless give any degree of parent-offspring and sib-sib correlation from 0.5 to zero depending on the number of genes showing dominance in the heterozygote, and the frequency with which each is present in the population. A character depending on a number of dominant genes whose recessive alleles are rare

> may give a parent-offspring correlation as low as 0.2, even without invoking genic interaction or any environmental effect. Conversely, assortative mating raises the coefficient above 0.5. Considerable caution is needed, therefore, in interpreting these correlations unless supporting information is available.

While we would agree with the general tone of this statement, it *is* possible to test the reasonableness of the required assumptions and, as will be pointed out later, to bring other information to bear in support of that provided by simple correlation coefficients. With regard to the assumptions underlying the method, assortative mating can be ruled out if the father/mother correlations are close to zero, and, since the expected value of r under models of inheritance including sex linkage, dominance, and assortative mating are known, tests of simple hypotheses concerning these correlation coefficients can be used to test the polygenic model against these other possibilities.

For example, in the case of autosomal complete dominance.[29] the father/son, father/daughter, mother/son, and mother/daughter correlations all have expected the value of 0.33, while each of the sib/sib correlations has an expected value of 0.42. This pattern of expected values can be compared with the r = 0.50 expected under the polygenic model for each of these combinations, thus providing the required test for dominance. While some have balked at the notion that studying each of these combinations is little more than a game—or, at best, a thinly disguised ruse for the mass production of thesis projects[30] —it is difficult to see why such objections should be raised when one considers the potential pitfalls elucidated by Tanner and, even more fundamentally, the importance of validating *any* model proposed for use in the prediction of growth. In any event, the polygenic nature of the inheritance of a number of important human traits has been established on the basis of the observed values of intrafamilial correlation coefficients, and the present discussion is meant to document the extension of these results to those dentofacial traits of most interest to the orthodontist so that we can stop arguing and get to the important business of utilizing *all* the information relevant to the prediction of individual growth in practice.

An example of the kind of evidence that is available is given in Fig. 2. Here both familial (brother/sister) and nonfamilial correlation coefficients are computed for twenty-one dentofacial measurements. It is seen that the familial correlations are consistently higher than the nonfamilial and that the familial correlations agree quite well with the value of r = 0.50 expected under the polygenic model of inheritance for these variables. Other combinations of family-member pairs, both for families presenting normal occlusions and within families containing a Class II, Division 1 proband, were also studied[27, 31, 32] and produced analogous results strongly supporting the polygenic model for the inheritance of dentofacial morphology generally and, in particular, for the inheritance of the Class II, Division 1 malocclusion. We might also mention that the nonfamilial correlation coefficients shown in Fig. 2 provide yet another indication of the inadequacy of population norms in the prediction of dentofacial growth. Even though the nonrelated pairs were matched for age

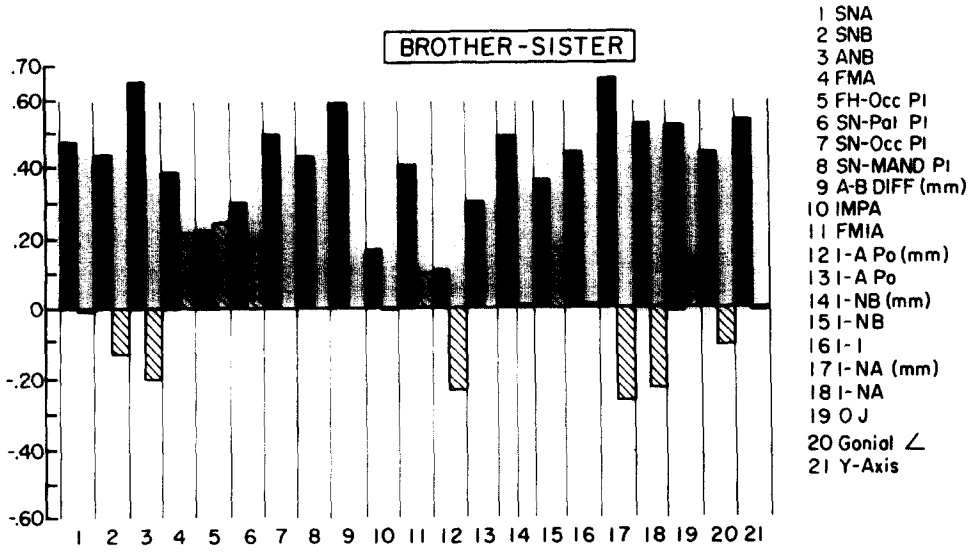

Fig. 2. Familial (solid bars) and nonfamilial (cross-hatched bars) correlation coefficients for twenty-one dentofacial measurements computed for brother/sister family pairs and a control sample of unrelated individuals.

and sex, there is but a negligible correlation between dentofacial measurements made on nonrelated individuals.

In yet another study of the inheritance of dentofacial morphology, the Class III malocclusion was also considered.[33] This, in addition to the computation of intrafamilial correlation coefficients, included a pedigree analysis, the pedigrees containing information on some 4,729 individuals. Here again it was found that the polygenic model best fit the observations. However, because of the infrequency with which the orthodontist is faced with the treatment of a Class III malocclusion—and the distinct possibility that different modes of transmission exist in different families or different populations[23]—the remainder of this article is limited to the use of familial information in the case of a Class II malocclusion, by far the orthodontist's most frequently encountered problem. We mention only that, even here, families tend to resemble one another much more than they do the "average population" in the sense that it is possible to discriminate between the ostensibly normal siblings of Class II and Class III patients.[34] Normal sibs of Class II patients have definite, albeit subclinical, Class II tendencies, while the normal sibs of Class III patients present tendencies in the opposite direction.

Clinical applications

Focusing now on the Class II malocclusion, it should be mentioned that the studies discussed above were prompted by a series of somewhat more practically oriented investigations the results of which, taken in conjunction with the later findings concerning the specific mode of inheritance of the condition, have direct clinical application. Harris[35] showed, on the basis of his studies of the complete

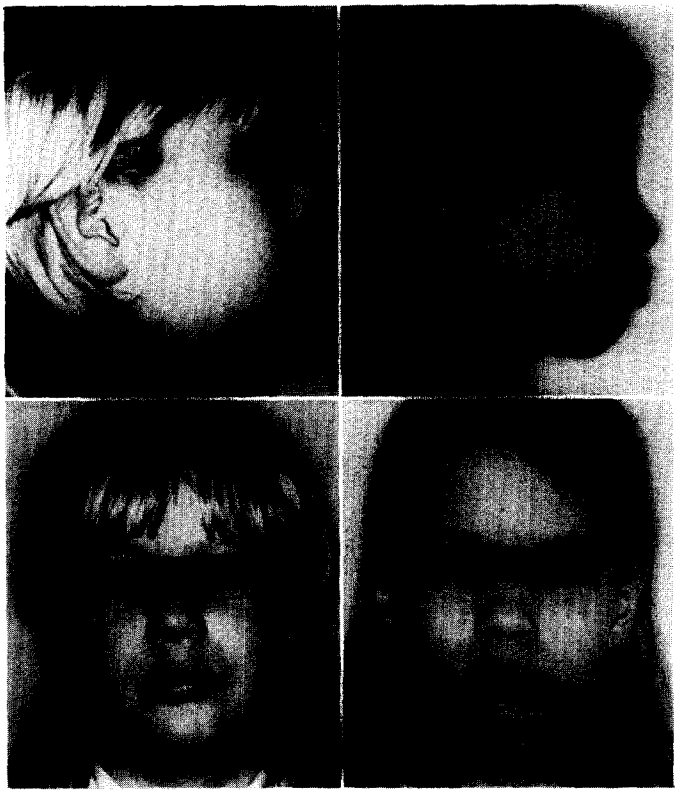

Fig. 3. Lateral and frontal photographs of a Class II boy (left) and of his older sister (right) when she as of the same age and not yet under orthodontic treatment.

family series of the University of Michigan Elementary School Growth Study, that (1) the long-term longitudinal cast and cephalometric records clearly support Brodie's[36] oringinal contention that an individual's craniofacial growth pattern is set early in life and persists, and that within a given family it is possible to distinguish between patients with favorable, or unfavorable, growth potentials by examining the severity of malocclusion presented by other members of the families.

While the subsequent studies discussed in the previous sections corroborated these findings and are useful in explaining *why* these phenomena occur, the most important clinical implications have nothing to do with complex mathematical manipulations, computers, correlation coefficients, and/or multiple linear regression equations. The important thing to realize is that most patients do not spontaneously shift the direction of their individual growth patterns and that this direction may be confirmed through the examination of the immediate family. While some fairly obtuse methods may on occasion be employed to demonstrate these facts, much simpler methods can make the point with almost equal force.

For example, a laboratory exercise used in the Orthodontics Department at The University of Michigan consists of sorting out a number of sets of dental

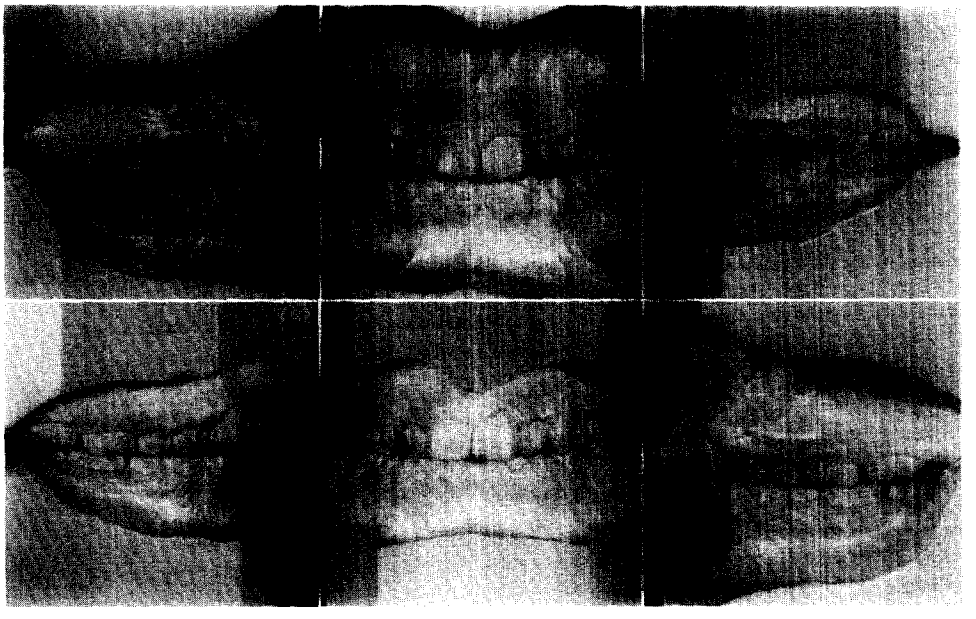

Fig. 4. Lateral and frontal views of dental models of the same Class II boy (top) and his sister (bottom) as in Fig. 3.

casts representing the early mixed-dentition stage and another when the permanent dentition has been completed (the children having received no orthodontic treatment). When all of the casts are mixed together, the dental student has no problem in correctly pairing the casts of each child taken at the mixed-dentition stage with the same child's casts collected in adulthood. This demonstrates both large interpersonal variation and the relative constancy of intrapersonal dentofacial form. More to the point, a patient with a Class I molar relationship rarely becomes a Class II patient unless there is an environmental cause, such as thumb-sucking or a premature loss of the deciduous dentition. The Class II patient likewise remains amazingly constant in molar relationship, overbite, and overjet.

And this constancy, this similarity of dentofacial form, is often shared with the other members of the immediate family. This may be illustrated by inspecting the relationships extant between just one possible family combination—the brother and sister shown in Fig. 3. (For some more complete family portraits making the same point, see the article by Brown.[21])

The boy is currently under treatment at the University of Michigan's Orthodontic clinic, and his older sister is a former patient in the clinic. All of the illustrative material in this section was gathered at the time the individuals involved presented for treatment (before any treatment was initiated) and it is seen that, although the boy is some 5 years younger than his sister, he represents the same sort of problem that was originally presented by her. The dental casts made from these individuals (Fig. 4) were measured to determine the maximum mesio distal and buccolingual dimensions of each tooth, as well as

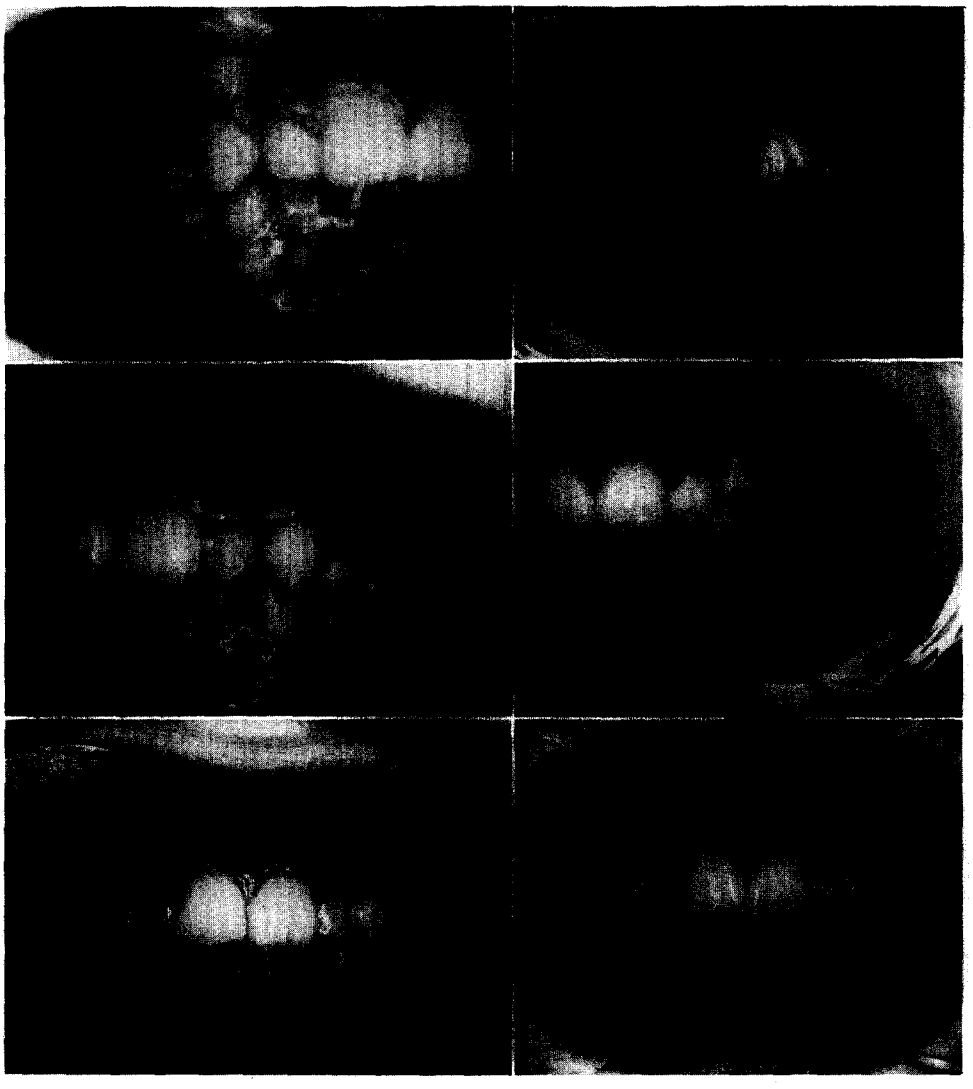

Fig. 5. Intraoral photographs of the same Class II boy (left) and his sister (right) as in Figs. 3 and 4.

parameters expressing similarities in arch form. Obvious similarities exist, as is apparent even from the intraoral photographs shown in Fig. 5. Skeletal similarities are equally obvious. The lateral cephalometric films (Fig. 6) were processed by the method of Walker and Kowalski,[37] which allows the user to obtain the values of any of the cephalometric measurements definable in the context of this two-dimensional coordinate system, and these were used, along with data obtained from other families, to compute intrafamilial correlation coefficients like those shown in Fig. 2.

To reiterate a point made earlier, one need not rely on correlation coefficients to establish the potential usefulness of intrafamilial information. The similarity

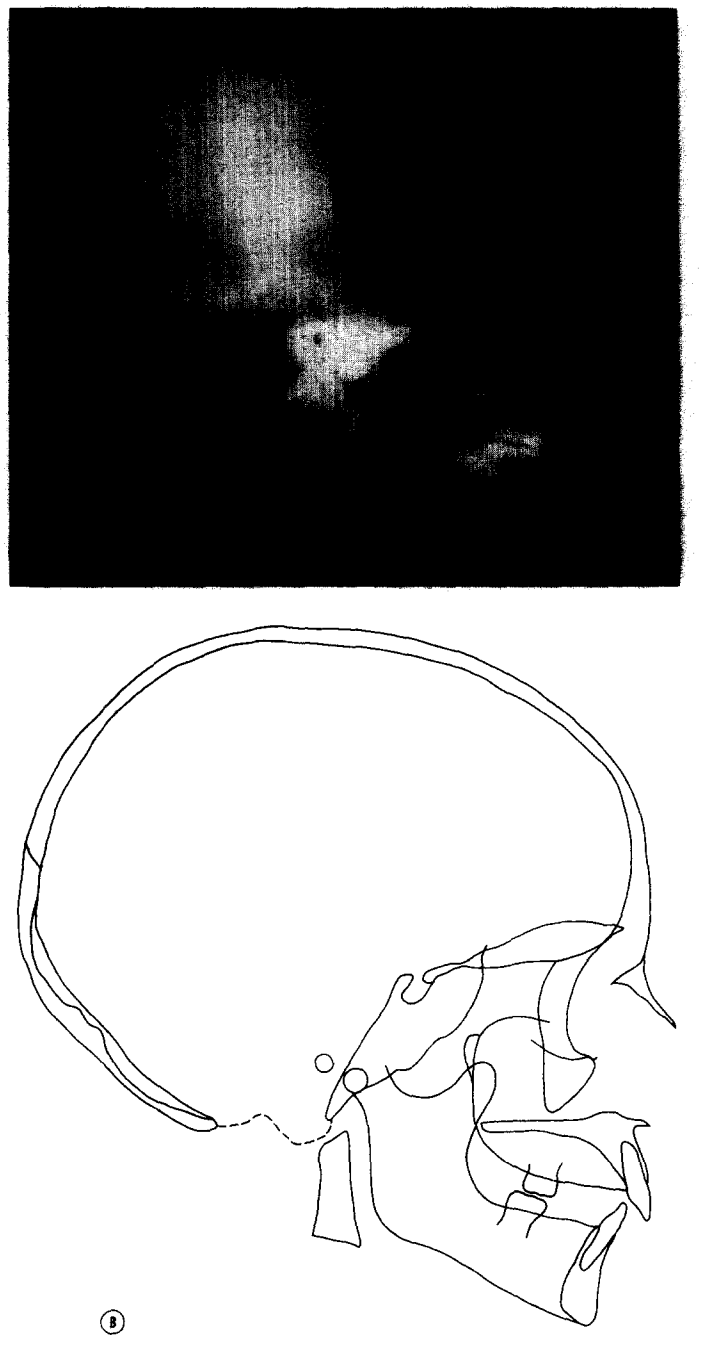

Fig. 6. Lateral cephalograms and tracings of the same Class II boy (left) and his sister (right) as in Figs. 3, 4, and 5.

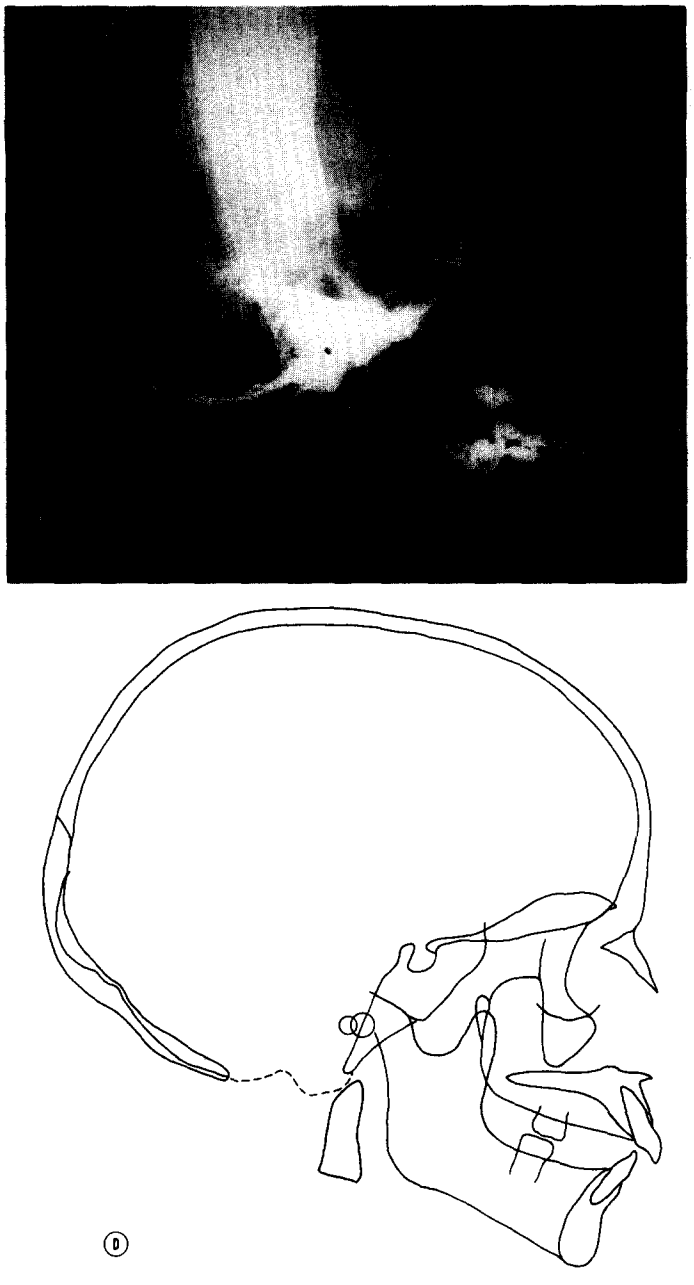

Fig. 6 (Cont'd). For legend, see opposite page.

of the patient to his sister is immediately apparent upon comparison of their photographs, their dental casts, their intraoral photographs, and their cephalograms. And it is precisely this similarity—which, interestingly, has been demonstrated to hold, at least up to the age of puberty, irrespective of sex—that we are suggesting the orthodontist exploit when assessing his patient's growth

potential. What it comes down to is the simple recognition of the fact that a patient with a moderate Class II malocclusion may be expected to have a favorable growth pattern when the other members of his family exhibit dentofacial forms within the range from moderate Class II to Class I molar relationships.

On the other hand, if this *same* patient had family members with severe skeletal Class II malocclusions, the clinician would be prudent in the expectation that his patient might well grow in a less than favorable way. While it is obvious that not all such decisions are as clear-cut as that illustrated above—a given family may present a number of gradations in dentofacial form, ranging from severe to moderate to ideal occlusions—it *is* rare to find a patient who differs greatly from the rest of his family, and this information can, and should, be taken into account when one is assessing a case.

What, then, of the "artisans" and the "computerniks"? We take the position that while certain of our conclusions have been prompted by studies which have relied extensively on computerized technology, the *clinical applications* of these findings have nothing to do with large data bases, computerization, and/or contacting a service agency. Just the patient, together with his family, a study of those facial dimensions *in these individuals* which the orthodontist has found to be most useful, and a treatment plan custom tailored to the patient in the light of *pertinent* information connecting the extent of the problem that he presents to the possibilities and limitations for treatment imposed by his genetic endowment will suffice.

It's all in the family.

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(Duval's) method and reasons for extraction were as follows: "Those which are most frequently out of their places are the canine of the upper and lower jaws, the deformity which results from this deviation might promptly determine us to extract them if we were not aware that they are less susceptible of caries than the small grinders which are in contact with them, and therefore, we ought to sacrifice the latter, and preserve the canine, which are more visible when a person laughs or speaks; for although at first they may be far from the place which they ought to occupy, they will arrange themselves more readily when the obstacle is removed, especially if they are often pressed with the finger according to the advice of Celsus, a celebrated physician of the Augustine Age. We should also take away one of the small grinders if it be irregular which is, however, more rare, and less urgent with regard to the appearance."

Duval was perhaps the first to note the importance of the relation of upper and lower jaws. "It is not sufficient that the teeth are properly arranged, by the side of each other, those of the upper jaw have a special connection with those of the lower, the least deviation from which diminishes the beauty of the appearance, frequently renders their functions laborious, and may often tend to their mutual destruction. Thus the superior incisors pass over the inferior and imitate in their action the cutting of a pair of scissors; in general the more parallel they are when brought into contact, the more they communicate to the face the character of beauty. (J. R. Duval: Des accidents de l'extraction des dents, Paris, 1802, 96 pp. In Weinberger, B. W.: The History of Orthodontia, International Journal of Orthodontia, predecessor of the American Journal of Orthodontics 2: 271, 1916.)