American
Journal of Orthodontics
and Oral Surgery

VOL. 28 OCTOBER, 1942 No. 10

Original Articles

FAMILIAL FACTORS IN DIAGNOSIS, TREATMENT, AND PROGNOSIS OF DENTOFACIAL DISTURBANCES*

GEORGE R. MOORE, D.D.S., M.S., AND BYRON O. HUGHES, PH.D.
ANN ARBOR, MICH.

THE PROBLEM

THE problems of this paper are: (1) to ascertain what features of the dentition and supporting structures are transmitted by the genetic mechanism; (2) to determine the *modus operandi* of these genetic attributes in the production of dentofacial disturbances; and (3) to evaluate their role in diagnosis, treatment, and prognosis of these disturbances. It is impossible, for the moment at least, to conduct experimental genetics on humans in the same sense we do on animals. However, human beings mate extensively and intensively enough that conditions equivalent to experimental animal genetics are obtained, provided controlled techniques for their study can be established. These controls are mainly empirical and statistical. This fact makes the problem of data collection and analysis difficult, but, even so, it does not invalidate the proposition that human genetics can be studied and that conclusions can be drawn.

Although nongenetic factors are important in the formation of facial structures, these are beyond the consideration of this paper. It is an error to presume that genetic factors can ever exist independent of environment or to insist that environmental influences cannot affect or even seriously modify a

From the Department of Orthodontics, University of Michigan.

*Research Project R54 of the Horace H. Rackham School of Graduate Studies, University of Michigan, Ann Arbor, Mich. A Preliminary Report. Sections of this paper have been read before the following groups: The New York Society of Orthodontists, January, 1940; The Great Lakes Society of Orthodontists, November, 1940 and November, 1941; The American Association for the Advancement of Science, Section H, December, 1940; The American Association of Physical Anthropologists, April, 1941; The Detroit Dental Clinic Club, January, 1942. In addition it was given as a series of lectures to postgraduate groups in dentistry under the auspices of the W. K. Kellogg Foundation Institute of Graduate and Postgraduate Dentistry at the University of Michigan, Ann Arbor, Mich., during the years 1939-1940 and 1940-1941.

structure of genetic origin. The intentional omission of a discussion of nurtural factors which also may be involved does not imply they are regarded as unimportant nor does it imply that subsequent work should omit a serious consideration of their interactive roles.

A review of our early data revealed to us that we were guilty of the common error of regarding certain broad classes of malocclusion as entities. For example, we were inclined to accept the occurrence of such anomalies as Class II malocclusion in parents and offspring as being significant, even suggestive of the operation of genetic factors. It was not long, however, until we noted that in families possessing Class II malocclusion, these Class II malocclusions might each exhibit a different set of physical attributes. One might be the result of a combination of normal maxilla, normal mandibular body, and short mandibular rami; another might be the result of deficient maxilla, deficient mandibular body, and short mandibular rami; still another might be the result of normal maxilla, normal mandibular rami, and short mandibular body. It began to appear reasonable that malocclusion or normal occlusion, as such, need not be directly inherited since evidence appeared to support the belief that the dentofacial complex is made up of numerous genetic attributes. It became apparent that if we were to be able to demonstrate how dentofacial anomalies fit into the genetic picture, it would be necessary for us to observe in detail the morphologic traits of the entire dentofacial complex.

For this reason, about four years ago we refined our method of approach to the problem of testing heredity's role in malocclusion by formalizing, systematizing, and particularizing the collection of data.

THE LITERATURE

Papers published in dental periodicals within the last fifteen years furnish adequate evidence that the genetics of the dentofacial structures is regarded as increasingly important in etiology, diagnosis, and treatment of dental disturbances. However, a stock-taking of the literature reveals the major obstacle to an understanding of the importance of hereditary traits to be a lack of adequate data upon which to formulate hypotheses and to draw conclusions. Following are representative comments. Many others could be cited following the same line of thought. Guyer[1] in 1924 summarizes the status of available evidence:

> After trying to get together the available facts regarding the part played by heredity in dental and facial abnormalities, I find myself in much the frame of mind of the quarrelsome negro described in one of the late Ambassador Page's letters. This negro tried in vain to exasperate a fellow negro into a fight. In spite of the storming and swearing of the first negro the other remained stubbornly silent. Finally negro Number 1 burst out with, "Look here, you kinky-headed, flat-nosed, slab-footed nigger, I warns you 'fore God, don't you keep givin' me none o' your damned silence."
>
> Like the vexed negro, I have found silence—or in other words the absence of reliable data—a great source of irritation. Opinions in this field, I find in abundance, but clear-cut unequivocal evidence, backed up by specific cases, is scarce. I have no doubt that some of the opinion in question is correct; nevertheless, it cannot be accepted as scientific truth until the facts upon which it is based are brought forward and subjected to the searchlight of modern genetics.

In 1927 Phillips[2] presents the same argument:

> Of heredity, and by it I refer to the transmutation of type only, we know so little, that I will not attempt to say anything, other than to point to certain striking characteristics of the enamel of the teeth, in which heredity, I think, plays an important part.

The same opinion is iterated by Brash[3] in 1929:

> There comes indeed a point in history of any subject, in which much of the discussion has been of a speculative nature, when it is essential to clear the ground; to examine critically general conclusions which are currently repeated; to probe the basis of facile hypothesis; and face with frankness the sometimes not very welcome fact that speculation, though it may on occasion anticipate discovery, is no substitute for inquiry.
>
> The reading of many papers and a sifting out of opinions and counter-opinions have been, I may be allowed to remark, very stimulating to the critical faculty, and have left a very clear impression on my mind that very little positive evidence has ever been adduced in support of any of the many supposed causes of irregularity and malocclusion. I have indeed found so little of positive knowledge and so much speculation that it has been clear to me, ever since I began to contemplate a discussion of the subject, that it would be necessary to review every aspect of it on fundamental principles and with strict reference to evidence.
>
> It is equally clear, so far as satisfactory conclusions on the inheritance of irregularities and malocclusions of the teeth are concerned, that the necessary data are simply not available.

Charles[4] in 1931 said:

> The relative positions of heredity and environment in growth are still the subject of considerable difference of opinion, principally, I think, because so very little is known about them.

Johnson (1933)[5] indicates evidence of heredity in occlusion to be available, enabling one to formulate some broad generalizations. These factors are of rather indeterminate specificity, however. Crouch (1935)[6] believes, "Heredity doubtless plays an important role in predetermining malocclusions or tendencies toward such malocclusions," but does not state what these hereditary factors are.

Casto[7] (1935) believes:

> The effect of heredity in producing malformation of the jaws and malocclusions of the teeth has been a controversial question for many years. There is, moreover, no present indication that the question will ever be definitely settled.

The textbook *Practical Orthodontia* by Dewey (revised in 1935 by Anderson) fails to reveal specific knowledge of the heredity of traits in the dentofacial complex to be known. As a matter of fact it lists as congenital causes some traits which are believed to be hereditary by other authors.

Goldstein and Stanton[8] (1936) are not specific and say:

> The factor of heredity, however, is undoubtedly the direct cause of malocclusion in specific instances, and, as Brash points out, adequate investigation in this field is lacking, and is much needed.

The same year Sly[9] writes:

> There is probably no more widely discussed or more controversial question than that which concerns the role of heredity in the causation of malocclusion; and while the present knowledge of the subject is insufficient to permit of its final solution, nevertheless some expression of opinion regarding it is essential to any consideration of the biologic factor in orthodontia.

The report of Johnson[10] on the results of cross breeding contrasted types of dogs at Cornell Experimental Farm provides us with the type of well-documented information vital to the understanding of the role of genetics in occlusion and necessary to the formulation of operative procedures which recognize the importance of etiology in the outlining of treatment policies for individual cases. Although this work was done on dogs Johnson's statement is to the point and merits consideration.

> It is true that in skull types and dental occlusion the human being is quite different from the dog. The application of the results of animal experimentation to human requires bridging a gap which to some orthodontists raises grave doubts. Be this as it may, the principles of genetics, so far as we know them today, apply to all living organisms. The nature of chromosomal distribution, the appearance of dominants and recessives, and the problem of mutant factors have been studied in plants and animals of various kinds, and they reveal the same basic phenomena. Man is no exception. Moreover, until proof to the contrary can be adduced, it is safe to assume that the biologic relationship of the teeth to their supporting structures is much the same in man as in the dog.

A review of the literature dealing with the inheritance of traits of the dentofacial complex and of the role of inheritance in etiology, diagnosis, and treatment of dental disturbances leaves the authors in substantial agreement with the writers previously quoted. Many of the articles dealing with the role of heredity in occlusion have been based upon empirical and logical argument and have been inadequately documented in their presentation of supporting evidence. It has been assumed that forces which operate in the biologic world are equally operative in the human world, and, the supporting evidence from experimental genetics is adequate to warrant the belief that genetic factors are both extensive and of basic importance in any form having the property of life. There is no question at this point of the operation of heredity in man. That such genetic factors operate, and extensively, is now established with reasonable scientific probability. Consequently, the problem of further research is to isolate specific attributes, to determine their mode of transmission, to acquire knowledge concerning their development, and to understand how they are affected by the environment in which they must exist. To attack this problem successfully, the first requisite is adequate data.

Although a review of the literature reveals data inadequacy with reference to the broader problems of occlusion, there are many individual attributes in the dentofacial complex whose genetics is moderately well known. For the most part these traits are special characters pertaining to the teeth themselves

and tend to be anomalous, such as absence of certain teeth, absence of certain groupings of teeth, absence of all teeth of one denomination, absence or deficient roots of teeth, diminutive maxillary incisors, noneruption of maxillary canines, hypoplasia of enamel, microdontia, incisor teeth erupted at birth, supernumerary teeth, susceptibility to dental caries, and so on. There are over a hundred references in the literature to the heredity of characters such as these and, on the whole, the details of transmission are fairly well understood. For the most part, however, the characters mentioned above are conspicuous, aberrant, and poorly represented in the population at large. In addition, although these traits are important in the etiology, diagnosis, and treatment of malocclusions which they engender or of which they may be a part, they do not make up the bulk of malocclusions and certainly the majority of treatment cases do not involve factors which can be attributed to these anomalous traits. As a matter of fact, a review of the literature would warrant an expression of the opinion that unless a character is conspicuous and unusual it is likely to be regarded as nonhereditary. In other words, there seems to be a feeling that the normal is immune to genetic laws; this obviously is absurd.

To date, most of the genetic work in orthodontics has been done by studying family pedigrees or by detailed examination of identical twins, comparing them with siblings. The former method lends itself admirably to the study of the conspicuous and anomalous traits; this largely because one can assume that the information presented by the untrained observer is satisfactorily accurate. Probably inaccuracy of observation rather than lack of family pedigree material has made conclusions on the more general problems of occlusion of doubtful validity. People have observed and have commented upon the fact that children resemble parents or earlier progenitors with reference to the face as a whole and there has been a belief that if the parents were normal or the familial line "untainted" with reference to almost any attribute the children would be more likely to be normal than if abnormalities or "taints" appeared within a family. These observations undoubtedly have foundation in fact, and implicit in the conclusions drawn from them is the assumption that genetic factors are operative. However, the majority of conclusions drawn from this type of unformulated observation have been extremely varied and, for the most part, erroneous. Consequently, they have had but little practical value to the orthodontic profession.

Weinberger[11] (1926) lists some of these generalizations:

> Perhaps the most common of generalizations obtained from data of the above-mentioned kinds is the belief that a child inherits his traits or features from some specific member of the family. For example, there has been a wide-spread belief that (1) children resemble their mothers more than they do their fathers and that the mother due to the fact that she nurtures a child during the fetal period, thus contributes much more strongly to the heredity of the child than does the father; (2) that the males show predominant resemblance to the mothers while the father contributes the majority of hereditary items to the females; (3) children resemble grandparents more than parents

> —here again is the belief that the maternal grandparent contributes more strongly to the heredity of the male grandchildren, while the paternal grandparent contributes more strongly to the heredity of the female grandchildren; (4) that heredity is most clearly shown in resemblance between children and parental siblings. In other words, heredity is presumed to operate in family lines with different progenitors or ancestors occupying positions of primary importance. Most of these ideas have largely disappeared from the field at the present time; however, there is one current belief that still has a number of followers, that is the belief that mothers contribute more strongly to the inheritance of children than do fathers.
>
> Another wide-spread belief apparently obtaining from this same type of uniformulized and noncritically tested evidence has been the hypothesis that hereditary malocclusions are more difficult, if not impossible, to treat. As early as 1864 Cartwright states that of two types of underhung jaws one condition is fortuitous and is amenable to mechanical treatment and the other condition is hereditary or congenital and cannot be remedied.

Furthermore, the continuance of controversy regarding the relative importance of heredity or environment in the production of malocclusion can be attributed to the continued reliance of professional men upon this type of evidence and upon their willingness to maintain argument without critically examining the relevancy of fact to opinion. Since this point of view has been extant for well over fifty years in orthodontics and since it has seriously hindered the advancement of understanding, we must again emphasize the primary problem to be a careful collection of material upon which to exercise analytical and critical acumen before presenting conclusions.

METHOD

For many years we have been recording in the orthodontic clinic at the University of Michigan as much data concerning parents and siblings of our patients as we could conveniently obtain, but never with much system. Our observations were concerned mainly with special characters such as those previously mentioned, and our notes on dentofacial anomalies were gross in respect to the anomaly as a whole rather than minute in respect to those component parts whose individual characteristics made harmonious occlusal relation impossible. An evaluation of these unsystematized data clearly demonstrated to us the need for a more detailed examination of the face and its component parts and emphasized the importance of systematic observation and recording if our materials were to be of value for analysis and comparison.

Many of the facial features, which seemed to us important, were not amenable to measurement. These were observed and recorded in a graded series.* Hooton[12] defends this method, "Morphological features which can be observed and described but cannot be measured are probably of greater anthropological significance than diameters and indices."

*The evaluation of detailed attributes of the dentofacial complex must be the work of orthodontists since clinical significance of dentofacial features can be properly appreciated only by men who have made a special study of them. Because orthodontists, as a rule, lack expert knowledge of genetic methodology and biometrical analysis, precise and effective presentation can best be secured by collaboration.

Examination Technique

After many months of preliminary record taking, the examination sheet shown in Figs. 1 and 2 was evolved.

Heading numbers one to four on our examination form refer to excesses or deficiencies in anteroposterior dimensions of the midfacial, maxillary alveolar, mandibular alveolar, and chin regions, respectively (Figs. 3 and 4). All four are taken from a profile view and at first it is surprising to note how

UNIVERSITY OF MICHIGAN
Department of Orthodontics

PLACE ..... OBSERVER ..... RECORDER ..... DATE ..... NO. .....

NAME ..... DATE OF BIRTH ..... SEX .....

KINSHIP WITH .....

1 Displacement Mid-facial Ant. & Post.	2 Displacement Max. Alv. Ant. & Post.	3 Displacement Mand. Alv. Ant. & Post.	4 Displacement Chin Ant. & Post.	5 Inclination Ant. & Post. Max. Teeth	6 Asymmetry Chin Disp.	7 Asymmetry Nose Disp.	8 Asymmetry Orbital Point Upwd. Disp.	9 Asymmetry Malar Bone Post. Disp.	10 Asymmetry Palate Rotary Disp.
1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.
2 Post. ?	2 Post. ?	2 Post. ?	2 Post. ?	2 Post. +	2 Rt. ?	2 Rt. ?	2 Rt. ?	2 Rt. ?	2 Rt. ?
3 Post. +	3 Post. +	3 Post. +	3 Post. +	3 Post. ++	3 Rt. +	3 Rt. +	3 Rt. +	3 Rt. +	3 Rt. +
4 Post. ++	4 Post. ++	4 Post. ++	4 Post. ++	4 Ant. +	4 Lt. ?	4 Lt. ?	4 Lt. ?	4 Lt. ?	4 Lt. ?
5 Ant. ?	5 Ant. ?	5 Ant. ?	5 Ant. ?	5 Ant. ++	5 Lt. +	5 Lt. +	5 Lt. +	5 Lt. +	5 Lt. +
6 Ant. +	6 Ant. +	6 Ant. +	6 Ant. +	Mand. Teeth	Mand. Angle	Malar Bones	Malar Bone		Mand. Angle
7 Ant. ++	7 Ant. ++	7 Ant. ++	7 Ant. ++	7 Abs.	Lat. Disp.	Lat. Disp.	Height		Rotary Disp.
				8 Post. +	7 Abs.	7 Abs.	7 Abs.		7 Abs.
				9 Post. ++	8 Rt. ?	8 Rt. ?	8 Rt. ?		8 Rt. ?
				10 Ant. +	9 Rt. +	9 Rt. +	9 Rt. +		9 Rt. +
				11 Ant. ++	10 Lt. ?	10 Lt. ?	10 Lt. ?		10 Lt. ?
					11 Lt. +	11 Lt. +	11 Lt. +		11 Lt. +

11 Asymmetry Mand. Ramus Height	12 Asymmetry Mand. Dent. Height	13 Absolute Size Maxilla	14 Absolute Size Ramus	15 Absolute Size Molar Bone	16 Obtusity or Acuteness of Angle	17 Arch Form	18 Palate Height	19 Oral Hygiene	20 Dec. Retained in Adult Dent.
1 Abs.	1 Abs.	1 Sm.	1 Sm.	1 Sm.	1 Abs.	1 Taper	1 Sm.	1 Good	1 None
2 Rt. ?	2 Rt. ?	2 +	2 +	2 +	2 Ob. ?	2 Trapez.	2 +	2 Fair	2 1-3
3 Rt. +	3 Rt. +	3 ++	3 ++	3 ++	3 Ob. +	3 U	3 ++	3 Poor	3 4-x
4 Lt. ?	4 Lt. ?	Mandible	Body	Angle Btwn.	4 Ob. ++	4 Square	Width	Caries	
5 Lt. +	5 Lt. +	8 +	8 +	Max. Occl &	5 Ac. ?	5 Ovoid	7 Sm.	Susceptibility	Dec. Retained
Mand. Body	Max. Dent.	9 ++	9 ++	Frank Planes	6 Ac. +	6 Hyperb.	8 +	7 Abs.	Too Long
Length	Height			7 Sm.	7 Ac. ++	Mand.	9 ++	8 +	7 None
7 Abs.	7 Abs.			8 +		7 Taper		9 ++	8 1-3
8 Rt. ?	8 Rt. ?			9 ++		8 Trapez.			9 4-x
9 Rt. +	9 Rt. +					9 U			
10 Lt. ?	10 Lt. ?					10 Square			
11 Lt. +	11 Lt. +					11 Ovoid			
						12 Hyperb.			

21 Unerupted Teeth Inc. Impacs.	22 Partial Impactions	23 Complete Impactions	24 Teeth Beginning Eruption Thru Mucosa	25 Premature Loss	26 Space Closure	27 Pontics	28 Congenital Absence	29 Supernum. Teeth	30 Malformed Teeth
Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.
1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None
2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3
3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x
Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.
7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None
8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3
9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x

Form 5766 4-41 IM

Fig. 1.

31 Hypoplasia of Enamel	32 Crowding & Spacing Up. Ants.	33 Crowding & Spacing Up. Buc. Teeth	34 Rotated Teeth (++ only)	35 Depth of bite	36 Anteriors in X-bite	37 Rt. Buccal Teeth in X-bite	38 Lt. Buccal Teeth in X-bite	39 Buccal Quadrant Completely Ling. to Opponent
Decid.	1 Abs.	1 Abs.	Decid.	Decid.	Decid.	Decid.	Decid.	1 Abs.
1 None	2 Cr. +	2 Cr. +	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	2 Rt. max.
2 1-3	3 Cr. ++	3 Cr. ++	2 Pres.	2 Deep +	2 Pres.	2 Pres.	2 Pres.	3 Lt. max.
3 4-x	4 Sp. +	4 Sp. +	Perm.	3 Deep ++	Perm.	Perm.	Perm.	4 Rt. mand.
Perm.	5 Sp. ++	5 Sp. ++	7 Abs.	4 Deep +++	7 Abs.	7 Abs.	7 Abs.	5 Lt. mand.
7 None	Lo. Ants.	Lo. Buc. Teeth	8 Pres.	5 Open +	8 Pres.	8 Pres.	8 Pres.	Completely Buccal
8 1-3	7 Abs.	7 Abs.		6 Open ++				7 Abs.
9 4-x	8 Cr. +	8 Cr. +		Perm.				8 Rt. max.
	9 Cr. ++	9 Cr. ++		7 Abs.				9 Lt. max.
	10 Sp. +	10 Sp. +		8 Deep +				10 Rt. mand.
	11 Sp. ++	11 Sp. ++		9 Deep ++				11 Lt. mand.
				10 Deep +++				
				11 Open +				
				12 Open ++				

40 Occlusion	41 Class I (Angle)	42 Class II (Angle)	43 Class III (Angle)	44 Oral Habits
1 Normal	1 Abs.	1 Abs.	1 Abs.	1 Abs.
2 Border Line	2 Growth case	2 Div. 1 Bilat. (Typ.)	2 Bilateral	2 Th. Suck.
3 Abnormal	3 Non-growth case	3 Div. 1 Bilat. (Atyp.)	3 Unilateral	3 Fi. Suck.
	4 Max. mesial drift	4 Div. 1 Unilateral	4 Mutilated	4 Tg. Suck.
	5 Mand. mesial drift	5 Div. 2 Bilat. (Typ.)		5 Ck. Suck.
	6 Mutilated	6 Div. 2 Bilat. (Atyp.)		6 Ck. Bite
	7 ..... (other)	7 Div. 2 Unilateral		7 U. Ip. Bite
		8 Mutilated		8 L. Ip. Bite
				9 Nail Bite
				10 Nite Gr.
				11 Mouth Br.
				12 ..... (other)

45 Disposition of Case	46 Disposition of Case	47 Treatment Indicated	49 Records Available	50	51
1 No attention indicated	1 Assign to Grad. for study	1 Comprehensive	1 Operative	1	1
2 Observation indicated	2 Assign to Grad. for obs.	2 Compromise	2 Anthropometric	2	2
3 Treatment indicated	3 Assign to Grad. for treatment	3 Palliative	3 Radiographic, complete	3	3
4 Not admitted here	4 Assign to Grad. for ..... (other)	4 Preventive	4 Radiographic, partial	4	4
5 Place on waiting list with encouragement	5 Referred to ..... (own dentist)	5 ..... (other)	5 Photographic, oriented facial	5	5
6 Place on waiting list without encouragement	6 Referred to ..... (own orthodontist)	48 Case Closed	6 Photographic, unoriented facial	6	6
7 Assign to Soph. for observation	7 Referred to .... (other dept's in D. S.)		7 Photographic, intraoral	7	7
8 Assign to Jr. for observation			8 ..... (other)	8	8
9 Assign to Jr. for treatment				9	9
10 Assign to Sr. for observation				10	10
11 Assign to Sr. for treatment				11	11
12 ..... (other)				12	12

Remarks:

Fig. 2.

independent of each other these four levels of the face appear to be. Mid-face could be termed maxillary apical base and is meant to include maxillary (except alveolar processes), nasal, and zygomatic regions.

Column number five refers to inclination of the maxillary and mandibular anterior teeth regardless of their anteroposterior positions in the skull, and again judgment is based on knowledge of the average condition at any stage of development.

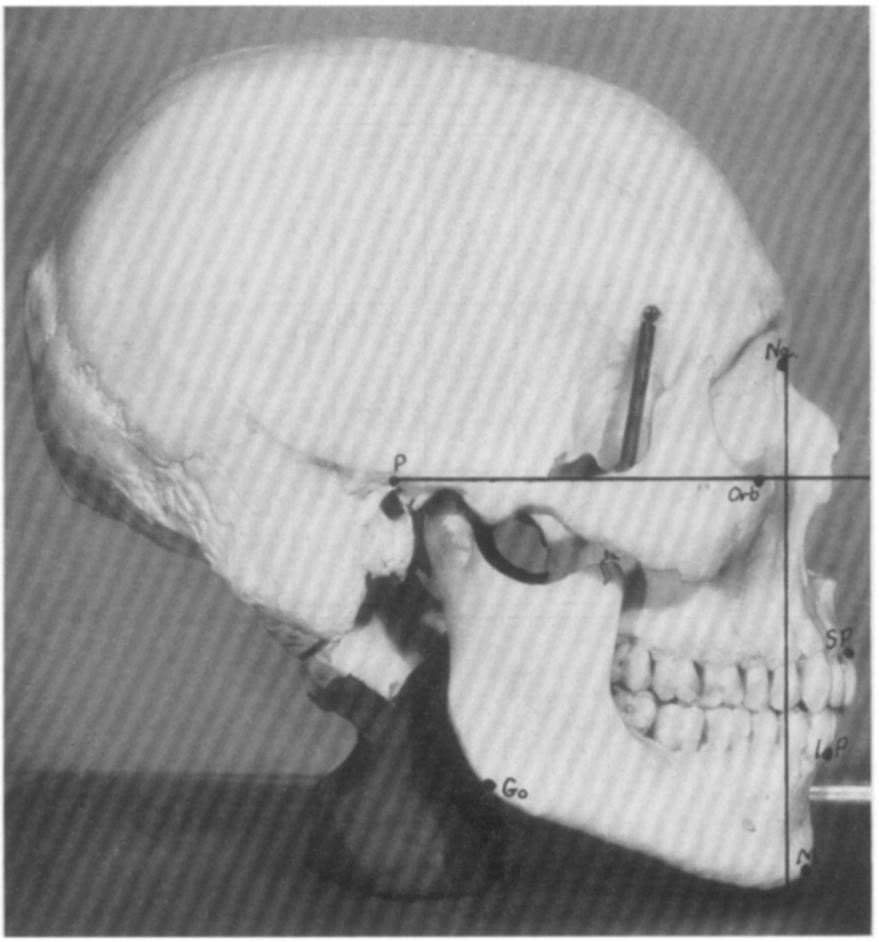

Fig. 3.

Figs. 3 and 4.—*Na.*, Nasion. *Orb.*, Orbital point. *P.*, Porion. *SP.*, Superior Prosthion. *IP.*, Inferior Prosthion. *Go.*, Gonion. The plane formed by the orbital and porion points is the Frankfort horizontal plane. The plane dropped from nasion perpendicular to the Frankfort plane is used for evaluating anterior and posterior displacements. Note in the figure that the maxillary apical base, the maxillary alveolus, the maxillary and mandibular anterior teeth, the mandibular alveolus and the mandibular body are all displaced considerably forward of the plane dropped from Nasion.

Thirteen categories on the record sheet refer to facial asymmetries. Attention has been called to these asymmetries; they have been measured often in the past by anthropologists using dry specimens. It is almost as easy to detect them on living as on dead material. Columns six to twelve, inclusive, record the thirteen different facial asymmetries (Figs. 5 and 6). Four are *lateral*

displacements: chin, mandibular angle, nose, and malar bone. Two are rotary displacements: palate and mandibular angle. Five refer to greater dimensions on the one side than on the other: malar bone, mandibular ramus, maxillary dental height, mandibular dental height, and mandibular body length. One records a higher level on one side: orbital point upward displacement. Finally, one records a more posterior relation of one side: malar bone posterior displacement.

Lateral displacements, orbital point upward displacement, and the various height differences are detected from a front view of the patient's face as he sits in the dental chair.

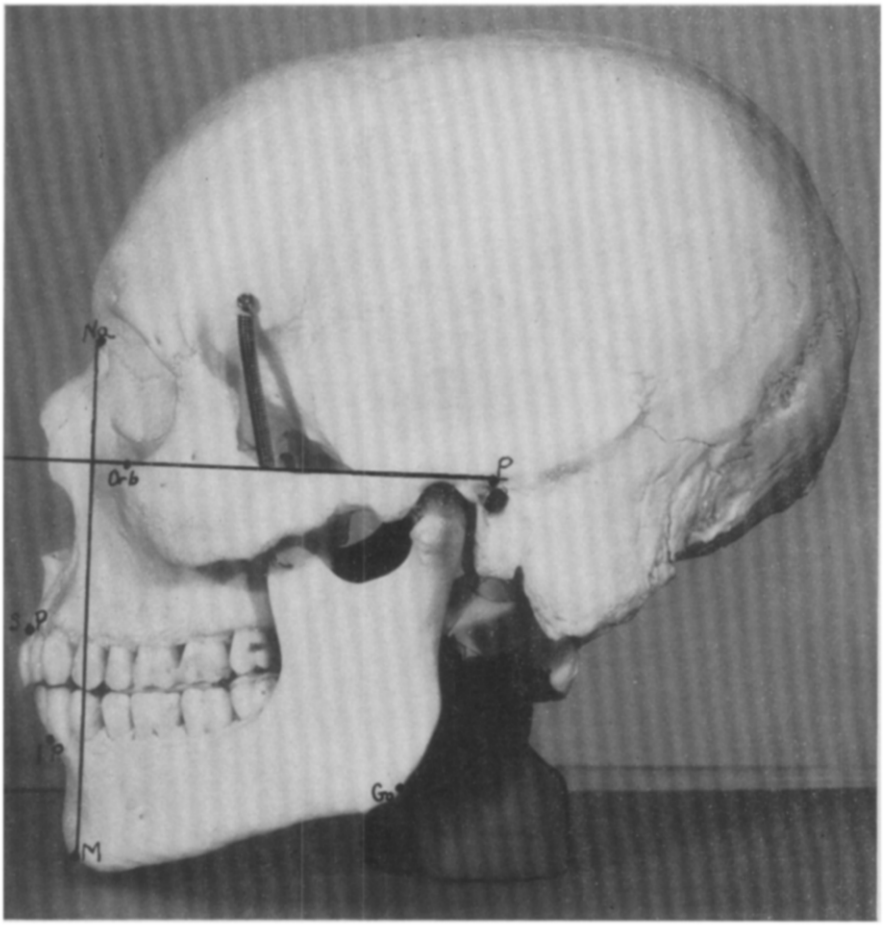

Fig. 4.

Usually palpation is necessary in order to evaluate asymmetry in height of malar bones and mandibular rami. Maxillary and mandibular dental height asymmetries are also detected by palpation. The points taken are occlusal plane to orbits for maxillary dental height and occlusal plane to inferior border of mandible for mandibular dental height.

Asymmetries of malar bone posterior displacement, mandibular angle rotary displacement, and mandibular body length are seen best by tipping the chair

back and viewing the head from below. Rotary asymmetry of the palate is present if a plane passing vertically through the posterior half of the raphe forms an angle other than 90 degrees with the orbital plane. This can be determined only by viewing the palate from directly below with the patient's mouth widely opened.

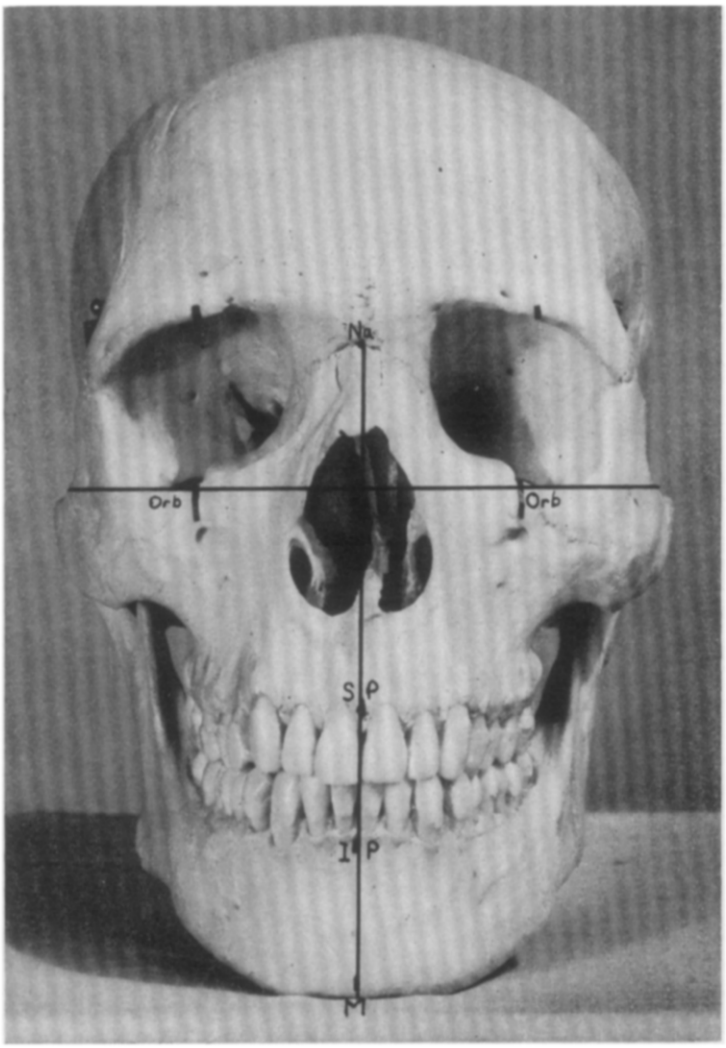

Fig. 5.—Lateral displacements are evaluated from the midfacial plane *Na—SP*. Very few lateral displacements are noticeable in this skull. Note that the plane *Na—SP* extends downward to include the midpoints of the mandible, *IP*. and *M*. Vertical asymmetries are evaluated from the orbital plane (*Orb—Orb*) and from the Frankfort horizontal plane. Here, again, few asymmetries are evident.

Headings 13, 14, 15, 16, 18, and 35 refer to absolute size of maxilla, mandible, rami, body, and malar bone; to angle between maxillary occlusal plane and Frankfort horizontal plane; to degree of obtusity or acuity of mandibular angle; to palate height; to palate width; and to depth of bite. Here, average for developmental stage of the individual under examination must form the basis of comparison in each of these.

Categories such as arch form, deciduous teeth retained, impactions, congenital absence, supernumerary teeth, hypoplasia of enamel, crowding, spacing, types of occlusion and malocclusion, and oral habits appear under twenty-six different headings. Observations in this entire group are absolute in that they are purely descriptive of the condition and are not dependent upon developmental stage.

The balance of the card is arranged for clinical and clerical convenience in our particular institution.

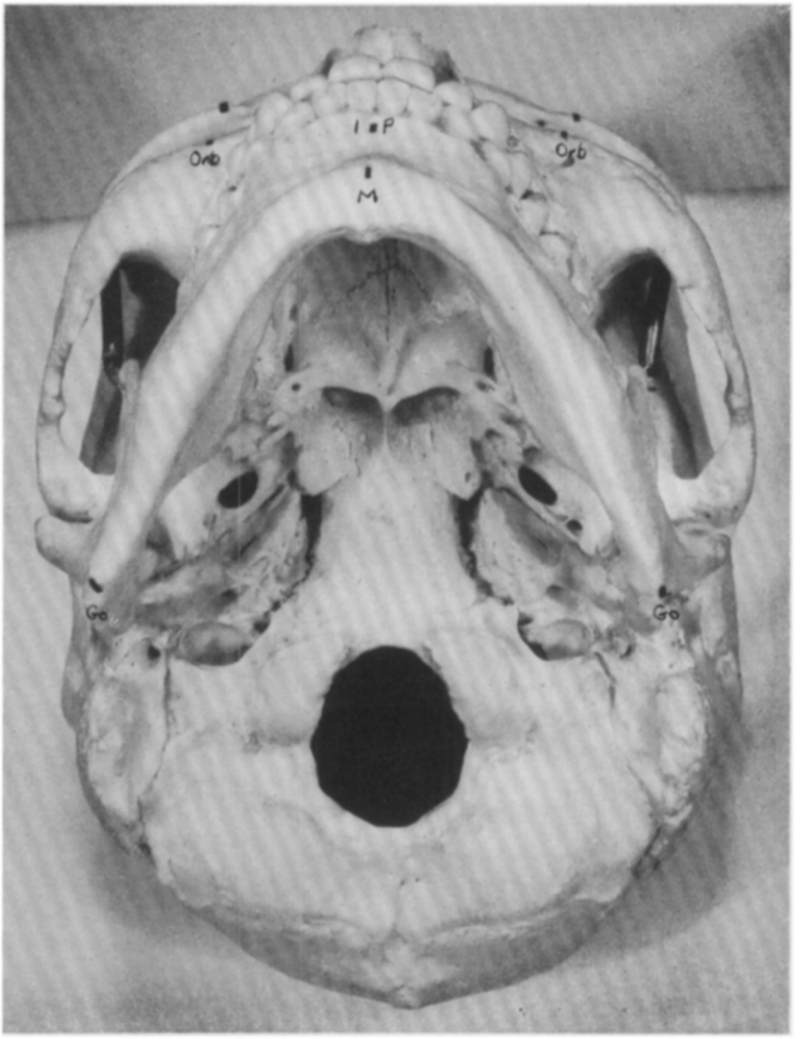

Fig. 6.—The patient is placed in approximately this position for appraising rotary and lateral displacements of the mandible. In this skull the mandible appears to be rotated slightly to the right. The degree of rotation is slight and probably not enough to be detected on the living.

THE SAMPLE

The data for this study include 265 individuals grouped in 78 families; 130 are children and 135 are parents. Dental and anthropometric records were secured on each individual. In addition supporting x-ray, photographic, and

clinical operative records were obtained on those individuals who were admitted to the clinic for treatment.

The population sample is selected and is not presumed to be representative of the population as a whole in the community from which the clinic draws its patients. At least one child in each family was brought to the orthodontic clinic because the parents were concerned over a real or apparent dentofacial disorder. Although this selection fails to give us much evidence of total population, it does give the very important professional advantage of being representative of a group that seeks orthodontic advice and treatment. This is the group which primarily concerns us in problems of etiology, diagnosis, and treatment.

AGE AND SEX

The male parents number 57, present an average age of 41.9 years, and an age range of 31 to 61 years; 73.3 per cent are between 35 and 45 years old; consequently the male parents may be classified as a middle-aged group. Female parents number 78, have an average age of 38.9 years and an age range of 29 to 55 years. Among females age is more variable than among males: 61.4 per cent are between 35 and 45 years, 24.3 per cent are under 35, and 14.3 per cent are over 45 years of age.

It is reasonable to assume that the longer environmental forces operate the more likely are nonhereditary factors to be interposed in the dentition and supporting structures. Some of these nonhereditary factors and their consequent effects are relatively easy to observe, others are exceedingly detailed and complicated. In all cases we have been careful to record the extent to which, in our opinion, these modifying traits have contributed to the dentofacial disturbance.

Three males (35, 43, 61 yr.) and 6 females (43, 44, 44, 47, 49, 55 yr.) present full upper and lower dentures and 3 females (37, 42, 44 yr.) have complete upper and partial lower dentures. One of these latter appeared to have been Class II: no classification of occlusion was made in the other cases.

Five males (8.8 per cent) and 8 females (10.3 per cent) are classified as normal. Class I malocclusions are shown in 36 males (63.2 per cent) and 42 females (53.8 per cent); mutilation of one kind or another has contributed to 22 of the male and to 29 of the female Class I's. The higher incidence of mutilation among the female Class I's appears to be significant. Males present 10 (17.5 per cent) Class II malocclusions and mutilation is a contributing factor in 5 of the cases. Females show 17 (21.8 per cent) Class II malocclusions with mutilation contributing to 6 of the cases. Class III malocclusions are limited in both sexes, 2 males and 2 females; mutilation is involved in 1 male case.

Among the parents, with the exception of those presenting full or partial dentures, the average age for the nonmutilated group is 2.7 years less than for those who show mutilation. This difference while small is significant in view of the fact that the majority of parents fall between 33 and 45 years of age.

The problem of time is as important among the children as it is among parents. Here the reason is different. From the point of view of development heredity becomes a part of a process that extends at least from conception to maturity. Most, if not all, hereditary features undergo vast change between

inception and maturity and it frequently is very difficult to describe the end product in terms of its appearance during the process of development. Time and space do not warrant further elaboration of this thesis. We want to emphasize, however, that the developmental process and heredity are integral.

In order to obtain an overview of some of the changes associated with the developmental process we have arranged the children in four groups. A further refinement will eventually be desirable but for the moment the following arrangement, although arbitrary, is convenient:

Group I. A full complement of deciduous teeth present with no permanent teeth erupted through the mucosa.

Group II. Begins with the eruption through the mucosa of any of the following teeth (a) $\frac{621\ /126}{621/\ 126}$ and terminates with the eruption through the mucosa of any of these teeth (b) $\frac{7543\ /3457}{7543/\ 3457}$.

Group III. Begins with (b) above and extends to eruption through mucosa of $\frac{8\ /8}{8/\ 8}$.

Group IV. Complete young adult dentition fully erupted.

The chronologic age in months sorted on the dentition stages is recorded in Table I. The age ranges included in each dentition stage are, on the whole, satisfactory for present purposes. Females show a somewhat greater developmental precocity than do males. This is a usual sex difference.

TABLE I

DENTITION STAGES AND CHRONOLOGIC AGE IN MONTHS

DENTITION STAGE	MALE			FEMALE		
	NO.	RANGE	MEAN	NO.	RANGE	MEAN
I	6	36- 64	50.3	4+1*	15- 51	36.8
II	23	72-168†	99.3	19	60-127	95.9
III‡	38	111-240	149.3	36	108-274	152.4
IV	2	262-288	275.0	1	------	254.0
Total	69	36-288	127.7	61	15-274	127.0

*In the case which is 15 months of age only $\frac{ba\ /a}{ba/\ a}$ are erupted.

†The case with age 168 months in stage II shows many teeth congenitally absent; excepting this the range is 72 to 132 months.

‡The average age for males with $\frac{7\text{-}1\ /1\text{-}7}{7\text{-}1/\ 1\text{-}7}$ erupted but no eruption of $\frac{8\ /8}{8/\ 8}$ is 138.0 months; for females 132.1; the numbers are 30 and 21, respectively. The remaining 8 males average 191.5 months and the 15 females 180.7 months.

The relation between occlusion and dentition stage (Table II) is not clear. There is the suggestion that malocclusion is less easily defined in stages I and early II than in the later divisions, and the implication is that some of the cases classified as normal in these periods would later be recorded as abnormal.

Table III presents the distribution of maxillary and mandibular arch forms according to dentition stage. The greater emphasis of ovoid and U-shaped arches in stages I and II clearly is evident. The tendency for the maxillary arch to emphasize tapering form and for the mandibular arch to show trapezoidal form is interesting. This is especially clear in females. Dentition stage IV is numerically too inadequate to be meaningful. Although the tendency

TABLE II

DENTITION STAGES AND OCCLUSION

DENTITION STAGE	MALE				FEMALE			
	NORMAL	I	II	III	NORMAL	I	II	III
I	4	1	1	0	1	3	1	0
II	3	6	10	4	2	8	8	1
III	3	21	13	1	3	19	11	3
IV	0	1	0	0	0	1	0	0
Totals	10	29	24	5	6	31	20	4

for ovoid and U-shaped arch forms to concentrate in stages I and II is clear, another fact is equally striking; children showing U-shaped and ovoid arch forms are found in 24 families; of these 24 families one, the other, or both parents present ovoid or U-shaped arches in 13 instances. Consequently the hereditary implications cannot be denied even at the early age levels.

TABLE III

DENTITION STAGES AND ARCH FORM

MALES						FEMALES					
A. MAXILLARY ARCH						A. MAXILLARY ARCH					
	TAPER	TRAPEZOID	OVOID	U-SHAPE	SQUARE		TAPER	TRAPEZOID	OVOID	U-SHAPE	SQUARE
I	1	0	4	1	0	I	0	1	2	1	0
II	9	3	4	6	0	II	5	5	4	2	0
III	11	10	4	5	1	III	17	11	4	1	0
IV	0	0	1	0	0	IV	0	0	0	1	0
Totals	21	13	13	12	1	Totals	22	17	10	5	0
B. MANDIBULAR ARCH						B. MANDIBULAR ARCH					
I	1	1	3	1	0	I	0	1	2	1	0
II	9	7	3	2	1	II	2	12	0	2	0
III	10	14	2	4	1	III	10	19	2	2	0
IV	0	0	1	0	0	IV	0	0	0	1	0
Totals	20	22	9	7	2	Totals	12	32	4	6	0

It is apparent that observations upon crowding do not attain a great deal of significance until the permanent dentition is moderately advanced in eruption (Table IV). The excess of mandibular over maxillary crowding is evident in both sexes.

TABLE IV

DENTITION STAGES AND CROWDING AND SPACING OF ANTERIORS

CROWDING AND SPACING DENTITION	MALES			FEMALES		
	SPACING	ABSENT	CROWDING	SPACING	ABSENT	CROWDING
A. MAXILLARY						
I	4	2	0	4	1	0
II	10	6	5	5	8	6
III	10	12	16	9	11	14
IV	1	1	0	1	0	0
Totals	25	21	21	19	20	20
B. MANDIBULAR						
I	4	2	0	3	2	0
II	1	10	10	2	10	7
III	3	13	22	4	12	19
IV	0	2	0	0	0	1
Totals	8	27	32	9	24	27

Our data fail to reveal that observations upon displacements (see Fig. 1) and asymmetries are much affected by age. Observations upon absolute size are based upon norms; hence while a great deal of development is shown in these factors, the concept of age is implicit in the size recorded. Age trends are but slightly reflected in our observations on obtusity or acuteness of mandibular angle and in depth of bite. These findings are rather contrary to expectation.

In summary then it is apparent that occlusion, arch form, and crowding and spacing of anteriors change considerably with age. The evidence for age effect upon other attributes is negative rather than conclusive in our present data.

GENETIC TRIANGLES

Before proceeding to the detailed analysis of the material it will be well to give a brief overview of the total data in order to provide a reference frame for particular features later on. The basis of this total analysis is the genetic triangle consisting of one mother and one father for each child. Thus statistically the number of parents will always be twice the number of children, or the number of female parents will equal the number of male parents and the number of male parents will equal the number of children. For purposes of this summary overview we have utilized observations upon displacements, inclination of maxillary and mandibular teeth, asymmetries, absolute size, angle between maxillary occlusal and Frankfort planes, obtusity or acuteness of mandibular angle, arch form, congenital absence of teeth, supernumerary teeth, malformed teeth, hypoplasia of enamel, crowding and spacing of teeth, depth of bite, crossbites, and classification of occlusion. It has seemed desirable to omit other observations listed in the recording blank (Figs. 1 and 2). The number of observations available in the genetic triangle analysis is recorded in Table V.

TABLE V

OBSERVATIONS IN GENETIC TRIANGLE ANALYSIS

NO. OF OBSERVATIONS	NO. OF FAMILIES	NO. OF CHILDREN	TOTAL NO. OBSERVATIONS
46	14	27	1242
36	28	45	1620
35	1	6	210
34	1	1	34
33	1	1	33
32	3	5	160
25	1	1	25
24	1	1	24
23	1	1	23
21	1	1	21
19	2	4	76
14	1	1	14
11	1	1	11
Totals	56	95	3493

The number of same observations made on all members of the family is shown in column 1; columns 2 and 3 record the number of families and the contained number of children, respectively; column 4 presents the total num-

ber of observations that may be arranged into genetic triangles. Thus we have 56 families with 95 children with the same observations available for all members of the family. This gives us a total of 3,493 observed features contained in 95 genetic triangles. We now wish (1) to contrast resemblance and not resemblance between parents, (2) to contrast resemblance and not resemblance between each child and each parent, (3) to contrast resemblance and not resemblance between each child and his two parents, when: (a) male parent and female parent are alike for the attribute, and (b) male parent and female parent are unlike for the attribute. Under the latter category some questions appear important:

1. Does the child resemble the male parent?
2. Does the child resemble the female parent?
3. Is the child intermediate between the two parents?
4. Does the child clearly show a feature that is vaguely presented in both parents? in the male parent? in the female parent?
5. Is the child unlike both parents?

In order to obtain a partial answer to these questions we legitimately may use the genetic triangle which statistically gives us 95 units with each one treated as a separate family even though the actuality is 95 children contained in 56 families. The first portion of the analysis is dependent upon two variables only; they can be the same or different. The degree of sameness or of differences does not concern us here. For example if we have recorded asymmetry of mandibular ramus height absent in mother, in father, and in child any relationship between the three members of the genetic triangle with reference to this feature must statistically be *same* or *resemblance* relationships. On the other hand if we have recorded the feature as absent in the father, questionably present on the right side in the mother, and questionably present on the left side in the child all relations in this genetic triangle must become *different* or *not resemblance* relationships. With only two choices available it is probable that random or chance factors will operate to produce 50 per cent of one variable and 50 per cent of the other;* consequently we become interested in percentage concentrations greater than 50 for interpretation. The results of this analysis are reproduced in Table VI. It is to be noted first that the amount of likeness between father and mother is 14.50 per cent in excess of the expected percentage. Many reasons, such as, general racial or national similarity, common circumstances in culture and environment, and so on, might be adduced to explain this excess. More important here is a purely negative answer. At least we cannot explain the excess in terms of genetics of the immediate family. The total amount of resemblance between father and child and between mother and child is somewhat higher than between father and mother but on the whole the difference (+3.08 per cent average) is not striking. There is little evidence to support the belief that children tend to resemble one parent more than they do the other; the percentage difference 0.93 is insignificant.

*Actually this is an oversimplification of the probabilities involved, but is deemed adequate for present purposes. For further discussion see Yule, G. Udney, *An Introduction to the Theory of Statistics,* Charles Griffin and Co., London, 1929, ed. 9, p. 33.

TABLE VI

RESEMBLANCE AND NONRESEMBLANCE

CATEGORY	%
1. Father like mother	64.50
2. Father unlike mother	35.50
3. Father like child	68.04
4. Father unlike child	31.96
5. Mother like child	67.11
6. Mother unlike child	32.89
7. Father and mother alike	
(a) child like parents	85.04
(b) child unlike parents	14.96
1. same feature opposite side	2.31
2. same feature emphasized	4.83
3. clearly different	7.82
8. Father and mother unlike	
(a) child like father	37.18
(b) child like mother	34.52
(c) child intermediate	16.94
(d) child different	11.36
1. clearly different	8.95
2. doubtfully different	2.41

The further analysis of resemblance between parents and child when mother and father are alike is shown in item 7 of the Table. The figures are striking; 85 per cent clearly are alike and only 8 per cent are clearly different. We do not interpret these figures to mean that 92 per cent of the traits we have examined in the dentofacial complex are to be ascribed to the hereditary mechanism, but we believe that they indicate genetic factors to be extensively operative.

The analysis of resemblance of child to parent or parents when father and mother are unlike is contained under item 8. Clear-cut differences not amenable to genetic interpretation obtain in 8.95 per cent of the features and, although the distribution of figures is somewhat different, the interpretation given for item 7 is generally substantiated. The percentage of intermediacy is unexpectedly low.

The presentation of the total data analyzed in terms of genetic triangles is intended as a brief overview. Rather extensive operation of hereditary factors in the dentofacial complex appears to be indicated. The further presentation is concerned with the analysis of specific features.

DETAILED ATTRIBUTES

All of the tables are arranged so that different parental combinations are shown in the vertical column at the left; the corresponding distribution of child traits is recorded in the respective horizontal lines. The number of families and the number of children involved for each parental combination are listed in the last two columns. The total distribution of child traits is shown in the last line.

ASYMMETRIES

The distribution of asymmetries is recorded in Table VII. All asymmetries are grouped into the categories present (p) and absent (a); specific types of asymmetry are not shown.

TABLE VII

ASYMMETRIES

PARENTS	CHILDREN PRESENT NO.	CHILDREN PRESENT %	CHILDREN ABSENT NO.	CHILDREN ABSENT %	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma Fa	2	14.3	12	86.7	9	14
Ma Fp	7	50.0	7	50.0	10	14
Mp Fa	8	28.6	20	71.4	15	28
Mp Fp	21	55.3	17	44.7	20	38
Ma Fu	7	33.3	14	66.7	12	21
Mp Fu	7	63.6	4	36.4	8	11
Total	52	41.3	74	58.7	74	126
Unobserved					4	4
Total					78	130

Observations were made on 74 families; these include 130 parents and 126 children. In 20 of these families, with 32 children, the father was unobserved (u).

One or more asymmetry is present in 41.3 per cent of children and 56.9 per cent of parents while 58.7 per cent of children and 43.1 per cent of parents show absence of facial asymmetry. In the parent groupings, however, 72 per cent show one, the other, or both parents with asymmetries and 28 per cent with an absence of asymmetry.

The detailed distribution reveals the number of asymmetries in children to rise markedly as parents show asymmetry. If we contrast the percentage of children showing asymmetry when both parents have an absence of asymmetry with those in which one, the other, or both parents present asymmetries, we find an average excess of 35.1 per cent attributable to the asymmetry of the parents. There is only one bracket in which a strong tendency for the operation of hereditary factors is contraindicated (mother present, father absent). Here we find 71.4 per cent of children showing asymmetries absent and 28.6 per cent showing asymmetries present. In all other instances the number of asymmetries is three to four times as great among children with asymmetrical parents as it is among children whose parents present an absence of asymmetry.

ABSOLUTE SIZE OF MAXILLA

Observations were made on 73 families and 124 children. The total distribution of absolute size in children, on the whole, is markedly symmetrical and highly concentrated in the category + (medium) size. There is a slightly higher percentage of deficient maxillas than of excess maxillas (9.7 per cent – 5.6 per cent). The difference is of doubtful statistical significance.

The part figures indicate a tendency of children to follow deficiency or excess in absolute size of maxilla if deficiency or excess is shown by parents. However, there are only two parental combinations in which the numbers are sufficiently large to warrant any conclusion: 1. Both parents + (medium); here there are 36 families with 64 children, and 82.8 per cent of the children are medium, 12.5 per cent are deficient (sm), and 4.7 per cent are excessive ++ in size. The tendency toward deficiency rather than excess is probably significant. 2. Mother medium, father unobserved; there are 17 families involving 26 children. The distribution is symmetrical and highly concentrated (92.4 per cent) in the medium category.

TABLE VIII

ABSOLUTE SIZE OF MAXILLA

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	0	0.0	1	100.0	0	0.0	1	1
M+ F+	8	12.5	53	82.8	3	4.7	36	64
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	2	25.0	6	75.0	0	0.0	4	8
M+ Fsm	0	0.0	3	100.0	0	0.0	2	3
M+ F++	0	0.0	7	87.5	1	12.5	5	8
M++ F+	0	0.0	5	83.3	1	16.7	4	6
Msm Fu	1	25.0	2	50.0	1	25.0	2	4
M+ Fu	1	3.8	24	92.4	1	3.8	17	26
M++ Fu	0	0.0	2	100.0	0	0.0	1	2
Total	12	9.7	105	84.7	7	5.6	73	124
Unobserved							5	6
Total							78	130

In two subgroups the mother presents a deficiency and the father is either medium or unobserved; the children present a 25.0 per cent deficiency. In 9 families involving 14 children, one parent presents an excess and the other is medium; 12 of the 14 children are medium; 2 present excesses, and none show deficiency.

The evidence for inheritance of deficiency or excess in the absolute size of the maxilla is not clear cut. There is, however, a tendency for children to follow the parental pattern. Since the majority of parents are medium (82.5 per cent), as regards absolute size of maxilla, it is to be presumed that most children would likewise be medium if hereditary factors were operative. The percentages are almost the same; 84.7 per cent of children are medium. Although there is question regarding inheritance of deficiency or excess, there seems little doubt that children will have medium size of maxilla if parents likewise have medium maxillary size.

ABSOLUTE SIZE OF MANDIBLE

Observations on the absolute size of the mandible were made on 73 families involving 124 children. The majority of children (75.0 per cent) and parents (74.6 per cent) are medium; deficiency is fairly common in children (23.4 per cent) and less frequent in parents (15.9 per cent), and excess is rare (1.6 per cent) in children and infrequent (9.5 per cent) in parents. The detailed distribution reveals a marked tendency for deficiency in mandibular size to occur in children when one or both parents present deficiency. In three of the four categories in which a parent presents deficiency, children likewise show deficiencies ranging between 40 per cent and 60 per cent. Equally noticeable, however, is the fact that deficiencies in children are not at all uncommon when both parents are medium or when one parent presents an excess and the other is medium. The percentage of excesses is too limited to warrant any conclusion. The negative evidence indicates that there is little tendency for mandibular excess to be transmitted.

Although there is a tendency of parental deficiencies to be transmitted to children, there is evidence that deficiency in the absolute size of the mandible

TABLE IX

ABSOLUTE SIZE OF MANDIBLE

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	3	60.0	2	40.0	0	0.0	3	5
M+ F+	10	23.8	32	76.2	0	0.0	31	42
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	4	40.0	6	60.0	0	0.0	4	10
M+ Fsm	3	18.8	13	81.2	0	0.0	5	16
M+ F++	2	22.2	7	78.8	0	0.0	6	9
M++ F+	0	0.0	8	100.0	0	0.0	3	8
Msm Fu	6	50.0	6	50.0	0	0.0	5	12
M+ Fu	1	5.3	17	89.4	1	5.3	14	19
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	29	23.4	93	75.0	2	1.6	73	124
Unobserved							5	6
Total							78	130

frequently obtains in children when contraindicated by parents. Aside from this piling up of deficiencies in children whose parents do not show deficiency, the evidence in general supports the conclusion that size factors are transmitted, at least in part, by the hereditary mechanism.

ABSOLUTE SIZE OF THE RAMUS

Observations were made upon 73 families involving 124 children. Ramus deficiencies are found in 50.8 per cent of cases and medium size of ramus obtains in 47.6 per cent. Excessive ramus size like excessive size of mandible is rare (1.6 per cent).

TABLE X

ABSOLUTE SIZE OF RAMUS

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	9	69.2	4	30.8	0	0.0	8	13
M+ F+	8	32.0	17	68.0	0	0.0	16	25
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	20	64.5	11	35.5	0	0.0	18	31
M+ Fsm	8	47.0	9	53.0	0	0.0	7	17
M+ F++	1	33.3	2	66.7	0	0.0	2	3
Msm F++	1	100.0	0	0.0	0	0.0	1	1
Msm Fu	11	78.6	3	21.4	0	0.0	8	14
M+ Fu	5	29.4	11	64.7	1	5.9	11	17
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	63	50.8	59	47.6	2	1.6	73	124
Unobserved							5	6
Total							78	130

The detailed figures show clearly a pronounced tendency for parents to transmit deficiency of ramus size to children. In the 5 parental combinations in which one, the other, or both parents have deficient rami, the children present deficiencies varying between 47.0 per cent and 100.0 per cent; in only one of the brackets does the percentage fall below 65.0. Of the remaining

parental combinations in which both parents are medium or one or both parents present an excess, the children show approximately 30.0 per cent deficiency and only 2 show excesses.

There is little doubt that hereditary factors are operative in the production of deficiency in the absolute size of the rami. There also is indicated a tendency toward deficiency beyond hereditary expectation.

ABSOLUTE SIZE OF BODY

Observations were made upon 73 families involving 124 children. The total distribution shows 76.6 per cent medium, 15.3 per cent deficient, and 8.1 per cent excess. The detailed distribution (Table XI) indicates clearly that absolute size of the body conforms to the parental pattern. The combination M+ F++ is an exception: 36.4 per cent of children show deficiency, 54.5 per cent are +, and 9.1 per cent present excess. There is a tendency for medium parents to produce deficiency more frequently than excess.

The size of mandibular body in children is dependent, to a considerable extent, on the size of the mandibular body in parents.

TABLE XI

ABSOLUTE SIZE OF BODY

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	2	100.0	0	0.0	0	0.0	1	2
M+ F+	6	9.7	55	88.7	1	1.6	35	62
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	0	0.0	1	100.0	0	0.0	1	1
M+ Fsm	0	0.0	4	100.0	0	0.0	3	4
M+ F++	4	36.4	6	54.5	1	9.1	8	11
M++ F+	0	0.0	6	60.0	4	40.0	4	10
Msm Fu	6	85.7	1	14.3	0	0.0	3	7
M+ Fu	1	4.2	20	83.3	3	12.5	16	24
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	19	15.3	95	76.6	10	8.1	73	124
Unobserved							5	6
Total							78	130

ABSOLUTE SIZE OF MALAR BONE

Observations were made on 73 families with 124 children. Here there are 87 per cent of children who are medium, 8 per cent who are deficient, and 5 per cent who have excess size of malar bones. The detailed distribution, with one exception (M+ F++), shows a pronounced tendency for children to follow the parental pattern.

ABSOLUTE SIZE OF PALATE HEIGHT

Observations were made on 69 families including 119 children. The majority of parents (78.2 per cent) and children (80.7 per cent) present medium height of palate, low palates are found in 0.8 per cent of parents and 4.2 per cent of children, and high palates appear in 21.0 per cent of parents and 15.1

TABLE XII

ABSOLUTE SIZE OF MALAR BONE

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	0	0.0	1	100.0	0	0.0	1	1
M+ F+	2	3.7	51	94.4	1	1.9	31	54
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	3	75.0	1	25.0	0	0.0	2	4
M+ Fsm	1	10.0	9	90.0	0	0.0	5	10
M+ F++	2	15.4	9	69.2	2	15.4	8	13
M++ F+	0	0.0	5	83.3	1	16.7	3	6
M++ Fsm	0	0.0	2	100.0	0	0.0	2	2
Msm Fu	1	25.0	3	75.0	0	0.0	2	4
M+ Fu	1	3.8	24	92.4	1	3.8	17	26
M++ Fu	0	0.0	1	50.0	1	50.0	1	2
Total	10	8.1	108	87.1	6	4.8	73	124
Unobserved							5	6
Total							78	130

per cent of children. The detailed distribution clearly shows children with high palates to have one, the other, or both parents with excessive height of palate (Table XIII). The number of parents and children with low palates is too small to warrant conclusion.

TABLE XIII

ABSOLUTE SIZE OF PALATE HEIGHT

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
M+ F+	1	2.0	40	93.0	2	4.7	26	43
M++ F++	0	0.0	6	60.0	4	40.0	5	10
M+ Fsm	0	0.0	2	100.0	0	0.0	1	2
M+ F++	1	4.5	17	77.3	4	18.2	12	22
M++ F+	1	7.7	8	61.5	4	30.8	7	13
M+ Fu	2	8.7	18	78.3	3	13.0	14	23
M++ Fu	0	0.0	5	83.3	1	16.7	4	6
Total	5	4.2	96	80.7	18	15.1	69	119
Unobserved							9	11
Total							78	130

Hereditary factors are operative in the production of normal and excessive height of palate.

ABSOLUTE SIZE OF PALATE WIDTH

Observations were made upon 67 families involving 117 children. The majority of children (65.8 per cent) are medium in width, approximately one-fourth (23.9 per cent) are deficient, and the remainder (10.3 per cent) show excessive width. The parents present more medium (76.7 per cent) and fewer deficient (16.4 per cent) and excess (6.9 per cent) palate widths than children. The subgroup figures indicate, on the whole, deficiency and excess to be found when indicated by parent combination; both parents medium (M+ F+) is a notable exception with 29.8 per cent of children presenting deficiency.

Although the evidence is not clear cut, there is indication, on the whole, that hereditary factors play an important role in the width of palate.

TABLE XIV

ABSOLUTE SIZE OF PALATE WIDTH

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	0	0.0	3	75.0	1	25.0	2	4
M+ F+	14	29.8	30	63.8	3	6.4	28	47
Msm F+	6	35.3	9	52.9	2	11.8	9	17
M+ Fsm	5	71.4	2	28.6	0	0.0	3	7
M+ F++	0	0.0	5	71.4	2	28.6	4	7
M++ F+	0	0.0	5	83.3	1	16.7	3	6
Msm Fu	3	42.9	3	42.9	1	14.2	3	7
M+ Fu	0	0.0	19	90.5	2	9.5	14	21
M++ Fu	0	0.0	1	100.0	0	0.0	1	1
Total	28	23.9	77	65.8	12	10.3	67	117
Unobserved							11	13
Total							78	130

OBTUSITY OF ANGLE

Observations on the obtusity and acuity of the mandibular angle were made on 70 families involving 120 children. The total figures show that a normal degree of obtusity (obtusity absent, obtusity?) is found in 56 per cent of cases and that somewhat marked angular obtusity (+, ++) is found in 43 per cent of the cases. Acuity of the mandibular angle is rare in children, being found in only one of the cases (0.8 per cent).

The detailed distribution reveals clearly that angular obtusity, on the whole, is found in children when one, the other, or both parents present this feature, and that absence of obtusity in children follows the parental pattern (Table XV).

The shape of the mandibular angle is dependent primarily upon hereditary factors.

MAXILLARY ARCH FORM

Observations were made upon 54 families involving 99 children. The total distribution reveals that tapering and trapezoidal arch forms are predominant in children (62.5 per cent), and that these forms are also most common in parents—42 of the 54 families present one, the other, or both parents with a tapering or trapezoidal maxillary arch. Ovoid arches are found in 21.2 per cent of children, U-shaped arches are found in 15.1 per cent, and square arches are rare. The percentage of ovoid arch forms is rather low considering the fact that several children present complete or almost complete deciduous dentitions.

The detailed distribution reveals a strong hereditary tendency in the inheritance of arch form. In addition to the tendency for children to follow the general parental pattern of arch form, there is a rather marked tendency for the maxillary arch to show constriction (tapering and trapezoidal forms) with a greater frequency than would be expected from the various parental combinations.

The shape of the maxillary arch is strongly conditioned by hereditary factors and constriction appears more frequently than expected.

TABLE XV

OBTUSITY OF ANGLE

PARENTS		CHILDREN SHOWING ABSENCE		OBTUSITY ?		OBTUSITY +		OBTUSITY ++		ACUITY ?		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Ma	Fa	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	3	3
M ?	Fa	2	20.0	8	80.0	0	0.0	0	0.0	0	0.0	5	10
Ma	F ?	3	50.0	3	50.0	0	0.0	0	0.0	0	0.0	3	6
M +	F +	0	0.0	1	8.3	10	83.3	1	8.3	0	0.0	8	12
M +	Fa	4	40.0	3	30.0	3	30.0	0	0.0	0	0.0	6	10
Ma	F +	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	1	1
M ++	F ++	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M ++	Fa	3	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	3
M +	F ?	1	50.0	0	0.0	1	50.0	0	0.0	0	0.0	1	2
M ?	F +	3	25.0	1	8.3	8	66.7	0	0.0	0	0.0	7	12
M ++	F ?	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
M ?	F ++	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M ++	F +	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Ma	Fu	6	42.9	4	28.6	3	21.4	1	7.1	0	0.0	8	14
M ?	Fu	0	0.0	2	50.0	2	50.0	0	0.0	0	0.0	4	4
M +	Fu	1	7.1	4	28.6	9	64.3	0	0.0	0	0.0	8	14
Mac +	Fac +	0	0.0	1	50.0	1	50.0	0	0.0	0	0.0	1	2
Mac ++	Fac ++	1	50.0	0	0.0	0	0.0	0	0.0	1	50.0	1	2
Mob +	Fac +	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mac ?	Fob ?	1	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1
M ?	F ?	0	0.0	7	43.7	9	56.3	0	0.0	0	0.0	7	16
Total		30	25.0	37	30.8	49	40.8	3	2.5	1	0.8	70	120
Unobserved												8	10
Total												78	130

TABLE XVI

MAXILLARY ARCH FORM

PARENTS		CHILDREN SHOWING TAPERING NO.	%	TRAPEZOIDAL NO.	%	U-SHAPED NO.	%	SQUARE NO.	%	OVOID NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Mta	Fta	3	42.8	3	42.8	0	0.0	0	0.0	1	14.2	5	7
Mtr	Ftr	5	35.7	4	28.6	2	14.3	0	0.0	3	21.4	9	14
Mu	Fu	1	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fov	1	14.3	1	14.3	1	14.3	0	0.0	4	57.1	3	7
Mtr	Fta	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mov	Fta	0	0.0	0	0.0	0	0.0	0	0.0	1	100.0	1	1
Mta	Ftr	8	47.1	4	23.5	2	11.8	0	0.0	3	17.6	8	17
Mu	Ftr	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	2	3
Mov	Ftr	4	66.7	1	16.7	1	16.7	0	0.0	0	0.0	2	6
Mta	Fu	0	0.0	3	60.0	2	40.0	0	0.0	0	0.0	2	5
Mtr	Fu	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	1	1
Mov	Fu	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Mu	Fov	1	50.0	0	0.0	0	0.0	0	0.0	1	50.0	2	2
Mov	Fsq	0	0.0	1	50.0	0	0.0	0	0.0	1	50.0	1	2
Mta	Fov	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mtr	Fov	0	0.0	0	0.0	1	50.0	0	0.0	1	50.0	1	2
Mta	Fun	4	44.4	0	0.0	1	11.1	0	0.0	4	44.4	3	9
Mtr	Fun	2	22.2	6	66.7	0	0.0	0	0.0	1	11.1	7	9
Mu	Fun	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
Mov	Fun	2	40.0	0	0.0	2	40.0	0	0.0	1	20.0	2	5
Total		38	38.3	24	24.2	15	15.1	1	1.0	21	21.2	54	99
Unobserved												24	31
Total												78	130

TABLE XVII

MANDIBULAR ARCH FORM

PARENTS		CHILDREN SHOWING TAPERING		TRAPEZOIDAL		U-SHAPED		SQUARE		OVOID		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Mta	Fta	4	44.4	4	44.4	1	11.1	0	0.0	0	0.0	4	9
Mtr	Ftr	7	28.0	12	48.0	2	8.0	1	4.0	3	12.0	13	25
Mu	Fu	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fov	0	0.0	0	0.0	2	50.0	0	0.0	2	50.0	2	4
Mta	Ftr	2	20.0	8	80.0	0	0.0	0	0.0	0	0.0	6	10
Mu	Ftr	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	2	3
Mov	Ftr	0	0.0	3	100.0	0	0.0	0	0.0	0	0.0	2	3
Mov	Fta	1	33.3	1	33.3	0	0.0	0	0.0	1	33.3	1	3
Mtr	Fta	2	40.0	2	40.0	0	0.0	0	0.0	1	20.0	4	5
Mu	Fov	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
Mov	Fsq	0	0.0	2	100.0	0	0.0	0	0.0	0	0.0	1	2
Mtr	Fu	0	0.0	3	75.0	1	25.0	0	0.0	0	0.0	2	4
Mov	Fu	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Mtr	Fov	0	0.0	2	100.0	0	0.0	0	0.0	0	0.0	1	2
Mta	Fun	2	28.6	1	14.3	1	14.3	0	0.0	3	42.8	2	7
Mtr	Fun	3	23.1	8	61.5	0	0.0	0	0.0	2	15.4	9	13
Mu	Fun	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fun	0	0.0	1	33.3	1	33.3	0	0.0	1	33.3	1	3
Total		24	24.2	50	50.5	11	11.1	1	1.0	13	13.1	54	99
Unobserved												24	31
Total												78	130

MANDIBULAR ARCH FORM

Observations were made upon 54 families with 99 children. Tapering and trapezoidal arches are predominant (74.7 per cent), square arches are rare (1.0 per cent), hyperbolic arches are unrepresented, U-shaped arches (11.1 per cent) and ovoid arch forms (13.1 per cent) are moderately frequent (Table XVII).

As in maxillary arch form, there is a strong tendency for the mandibular arch form to follow an hereditary pattern; the dominance of constricted arch forms (tapering and trapezoidal) is indicated.

Arch form is largely hereditary and dominance of constricted over non-constricted types is indicated.

CONGENITAL ABSENCE OF TEETH

Observations were made upon 67 families involving 113 children. The majority of parents and children observed do not have congenitally absent teeth. A sizable percentage (15.0 per cent), however, do present congenital absence of teeth.

The detailed distribution table according to parental pattern clearly shows the hereditary nature of congenital absence of teeth.

TABLE XVIII

CONGENITAL ABSENCE OF TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	4	5.6	67	94.4	42	71
Mp	Fp	7	63.6	4	36.4	6	11
Ma	Fp	2	66.7	1	33.3	2	3
Ma	Fu	1	4.5	21	95.5	13	22
Mp	Fu	3	50.0	3	50.0	4	6
Total		17	15.0	96	85.0	67	113
Unobserved						11	17
Total						78	130

SUPERNUMERARY TEETH

Observations were made upon 66 families including 111 children. The total distribution shows supernumerary teeth largely are absent in the parents and children of this series. Although the evidence is too limited to permit conclusion, the operation of hereditary factors is suggested.

TABLE XIX

SUPERNUMERARY TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	1	1.3	77	98.7	47	78
Ma	Fp	1	20.0	4	80.0	2	5
Ma	Fu	1	3.6	27	96.4	17	28
Total		3	2.7	108	97.3	66	111
Unobserved						12	19
Total						78	130

MALFORMED TEETH

Observations were made upon 67 families with 113 children. Malformed teeth are uncommon both in the parents and in the children constituting this series. What evidence there is does not reveal the operation of hereditary factors.

TABLE XX

MALFORMED TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	5	6.2	76	93.8	48	81
Ma	Fp	0	0.0	2	100.0	1	2
Mp	Fp	0	0.0	2	100.0	1	2
Ma	Fu	1	3.6	27	96.4	17	28
Total		6	5.3	107	94.7	67	113
Unobserved						11	17
Total						78	130

HYPOPLASIA OF ENAMEL

Observations were made upon 67 families and 113 children. Hypoplasia of enamel is uncommon both in parents and in children. There is no evidence, although the data are very limited, pointing toward the inheritance of enamel hypoplasia.

TABLE XXI

HYPOPLASIA OF ENAMEL

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	5	5.9	80	94.1	50	85
Ma	Fu	0	0.0	27	100.0	16	27
Ma	Fu	0	0.0	1	100.0	1	1
Total		5	4.4	108	95.6	67	113
Unobserved						11	17
Total						78	130

CROWDING AND SPACING OF MAXILLARY ANTERIORS

Observations were made upon 64 families with 109 children. Spacing, crowding, and absence of either spacing or crowding are equally distributed in the total group of children. Absence of crowding or spacing is found in 61.8 per cent of the parents, crowding in 26.4 per cent, and spacing in 11.8 per cent. The detailed distribution indicates that the children, on the whole, follow the parental pattern (Table XXII). Crowding or spacing of maxillary anteriors seems to be dependent upon a number of factors, as both crowding and spacing appear in moderately large percentages (25.0 per cent, 33.3 per cent) when neither parent shows crowding or spacing. Likewise, the distribution of crowding and spacing in children when one, the other, or both parents show crowding or spacing or some combination of these two, indicates multiple factors to be involved.

TABLE XXII

CROWDING AND SPACING OF MAXILLARY ANTERIORS

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	8	33.3	10	41.7	6	25.0	17	24
Mp	Fp	0	0.0	3	50.0	3	50.0	4	6
Ma	Fp	3	15.0	6	30.0	11	55.0	10	20
Mp	Fa	1	12.5	2	25.0	5	62.5	5	8
Msp	Fa	8	57.1	5	35.7	1	7.1	5	14
Ma	Fsp	3	50.0	2	33.3	1	16.7	4	6
Msp	Fsp	0	0.0	0	0.0	1	100.0	1	1
Ma	Fu	10	52.6	5	26.3	4	21.1	10	19
Mcr	Fu	2	22.2	3	33.3	4	44.4	6	9
Msp	Fu	1	50.0	0	0.0	1	50.0	2	2
Total		36	33.0	36	33.0	37	33.9	64	109
Unobserved								14	21
Total								78	130

Hereditary factors (probably multiple) are operative in producing crowding or spacing of maxillary anterior teeth. The factor of age, as previously shown, is also involved.

CROWDING AND SPACING OF MANDIBULAR ANTERIORS

Observations were made upon 67 families involving 114 children. Crowding of mandibular anteriors among children occurs more frequently (47.4 per cent), than spacing (10.5 per cent), or absence of crowding or spacing (42.1 per cent). Parents present a slightly higher percentage of crowding (49.1 per cent), a lower incidence of spacing (2.5 per cent), and a greater amount of absence of crowding or spacing (48.3 per cent) than children. The differences are of doubtful significance. Children follow the parental pattern to a considerable extent. Crowding of mandibular anteriors is somewhat more frequent than would be expected from the parental combination. Possibly partial dominance of factors controlling crowding is involved. Multiple factors may be operative.

TABLE XXIII

CROWDING AND SPACING OF MANDIBULAR ANTERIORS

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	3	15.8	11	57.9	5	26.3	14	19
Mp	Fp	3	8.6	12	34.3	20	57.1	15	35
Ma	Fp	0	0.0	6	37.5	10	62.5	10	16
Mp	Fa	1	9.1	4	36.4	6	54.5	8	11
Msp	Fa	0	0.0	1	100.0	0	0.0	1	1
Msp	Fcr	0	0.0	0	0.0	2	100.0	1	2
Ma	Fu	4	22.2	10	55.6	4	22.2	10	18
Mcr	Fu	0	0.0	4	40.0	6	60.0	7	10
Msp	Fu	1	50.0	0	0.0	1	50.0	1	2
Total		12	10.5	48	42.1	54	47.4	67	114
Unobserved								11	16
Total								78	130

The hereditary mechanism operates, at least in part, to produce crowding or spacing of mandibular anterior teeth. Partial dominance or multiple factors may be involved.

CROWDING AND SPACING OF MAXILLARY BUCCAL TEETH (TABLE XXIV)

Observations were made upon 61 families involving 105 children. Crowding of the maxillary buccal teeth is relatively rare in children and in parents. The total distribution for parents and children is symmetrical and highly concentrated in the absent category. There is little indication that heredity operates in crowding and spacing of maxillary buccal teeth, except for negative evidence in the parental combinations mother absent, father absent and mother absent, and father unobserved.

The numbers in the other categories are too limited to warrant any conclusion, but the trend is away from the operation of hereditary factors.

TABLE XXIV

CROWDING AND SPACING OF MAXILLARY BUCCAL TEETH

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	2	3.1	62	96.8	0	0.0	36	64
Mp	Fp	0	0.0	2	100.0	0	0.0	1	2
Ma	Fp	1	25.0	3	75.0	0	0.0	3	4
Mp	Fa	0	0.0	4	80.0	1	20.0	3	5
Ma	Fu	0	0.0	26	96.3	1	3.7	16	27
Mcr	Fu	0	0.0	3	100.0	0	0.0	2	3
Total		3	2.8	100	95.2	2	2.0	61	105
Unobserved								17	25
Total								78	130

CROWDING AND SPACING OF MANDIBULAR BUCCAL TEETH (TABLE XXV)

Observations were made upon 63 families involving 107 children. Crowding or spacing of mandibular buccal teeth are relatively rare, both in children (12.9 per cent) and in parents (4.6 per cent). The detailed figures do not reveal the operation of hereditary factors as regards crowding or spacing of mandibular buccal teeth.

TABLE XXV

CROWDING AND SPACING OF MANDIBULAR BUCCAL TEETH

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	3	4.2	64	88.9	5	6.9	41	72
Ma	Fp	0	0.0	2	100.0	0	0.0	2	2
Mp	Fa	0	0.0	3	75.0	1	25.0	2	4
Ma	Fun	2	7.1	23	82.1	3	10.7	17	28
Mcr	Fun	0	0.0	2	100.0	0	0.0	1	2
Total		5	4.6	94	87.0	9	8.3	63	108
Unobserved								15	22
Total								78	130

ROTATED TEETH

Observations were made upon 65 families with 111 children. Markedly rotated teeth are found in approximately half both of parents (50.9 per cent)

and of children (47.4 per cent). The detailed distribution clearly demonstrates the operation of hereditary factors, apparently single genes segregating normally.

TABLE XXVI

ROTATED TEETH

PARENTS		CHILDREN PRESENCE NO.	%	ABSENCE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma	Fa	4	21.1	15	78.9	14	19
Mp	Fp	25	80.6	6	19.4	15	31
Ma	Fp	8	44.4	10	55.6	11	18
Mp	Fa	8	50.0	8	50.0	9	16
Ma	Fu	1	7.1	13	92.9	8	14
Mp	Fu	7	53.8	6	46.2	8	13
Total		53	47.7	58	52.3	65	111
Unobserved						13	19
Total						78	130

DEPTH OF BITE

Observations were made upon 62 families including 105 children. Pronounced to extreme (++, +++) depth of bite is found in 62.8 per cent of children and in 46.7 per cent of parents. Extreme depth of bite is more frequent in children (15.2 per cent) than in parents (11.2 per cent). Absent and medium depth of bite occur in 30.4 per cent of children and in 52.3 per cent of parents. Open-bite is rare in parents (0.9 per cent) and infrequent in children (6.7 per cent). The detailed distribution (Table XXVII) fails to show the operation of hereditary factors in the production of open-bite; evidence, however, is very limited but does show clearly that deep bites are considerably dependent upon the presence of deep bites in one, the other, or both parents. In general, it seems that children show a somewhat more pronounced depth of bite than would be expected from the parental combination.

ANTERIORS IN CROSSBITE

Observations were made upon 65 families involving 109 children. Anterior teeth in crossbite are found in 14.7 per cent of children and in 9.8 per cent of parents. The operation of hereditary factors in the production of anteriors in crossbite, on the whole, is contraindicated by the detailed distribution.

OTHER CROSSBITES

Observations were made upon 65 families involving 108 children. The total distribution shows 13 per cent of the children present other crossbites, and 87 per cent show other crossbites absent. The detailed distribution fails to show the operation of hereditary factors, with reference to buccal teeth in crossbite (Table XXIX).

OCCLUSION

Observations were made upon 66 families involving 110 children. The total distribution, as would be expected from the type of sample, shows the majority of children to have some type of malocclusion. Of the children, 49.1

TABLE XXVII

DEPTH OF BITE

PARENTS		CHILDREN SHOWING ABSENCE NO.	%	MEDIUM NO.	%	PRONOUNCED NO.	%	EXTREME NO.	%	OPEN + NO.	%	OPEN ++ NO.	%	NO. OF FAMILIES	NO. OF CHILDREN
Ma	Fa	0	0.0	0	0.0	1	50.0	0	0.0	1	50.0	0	0.0	2	2
M +	F +	0	0.0	1	9.1	7	63.6	2	18.2	1	9.1	0	0.0	5	11
M ++	F ++	4	21.0	4	21.0	9	47.4	1	5.3	1	5.3	0	0.0	10	19
M +++	F +++	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Ma	F +	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	0	0.0	2	3
M +	Fa	1	25.0	2	50.0	1	25.0	0	0.0	0	0.0	0	0.0	4	4
M ++	F +	0	0.0	1	12.5	4	50.0	3	37.5	0	0.0	0	0.0	6	8
M +	F ++	0	0.0	1	16.7	2	33.3	2	33.3	0	0.0	1	16.7	4	6
Ma	F ++	4	57.1	1	14.3	0	0.0	2	28.6	0	0.0	0	0.0	4	7
Ma	F +++	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
M +	F +++	0	0.0	0	0.0	1	50.0	1	50.0	0	0.0	0	0.0	1	2
M +++	F +	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M +++	Fa	0	0.0	3	50.0	2	33.3	1	16.7	0	0.0	0	0.0	1	6
M ++	F +++	1	20.0	0	0.0	2	40.0	2	40.0	0	0.0	0	0.0	3	5
Ma	Fun	2	66.7	0	0.0	0	0.0	0	0.0	1	33.3	0	0.0	2	3
M +	Fun	1	7.7	3	23.1	8	61.5	0	0.0	1	7.7	0	0.0	10	13
M ++	Fun	0	0.0	1	16.6	5	83.3	0	0.0	0	0.0	0	0.0	3	6
M +++	Fun	0	0.0	0	0.0	4	80.0	1	20.0	0	0.0	0	0.0	1	5
Mop +	Fun	0	0.0	1	50.0	0	0.0	0	0.0	1	50.0	0	0.0	1	2
Total		14	13.3	18	17.1	50	47.6	16	15.2	6	5.7	1	1.0	62	105
Unobserved														16	25
Total														78	130

TABLE XXVIII

ANTERIORS IN CROSSBITE

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	10	15.2	56	84.8	38	66
Ma	Fp	1	14.3	6	85.7	5	7
Mp	Fa	2	33.3	4	66.7	3	6
Mp	Fp	0	0.0	1	100.0	1	1
Ma	Fu	3	11.1	24	88.9	17	27
Mp	Fu	0	0.0	2	100.0	1	2
Total		16	14.7	93	85.3	65	109
Unobserved						13	21
Total						78	130

TABLE XXIX

OTHER CROSSBITES

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	9	15.5	49	84.5	33	58
Ma	Fp	1	12.5	7	87.5	6	8
Mp	Fa	1	9.1	10	90.9	6	11
Mp	Fp	0	0.0	2	100.0	1	2
Ma	Fu	3	12.0	22	88.0	17	25
Mp	Fu	0	0.0	4	100.0	2	4
Total		14	13.0	94	87.0	65	108
Unobserved						13	22
Total						78	130

per cent show Class I, 32.7 per cent show Class II, 6.4 per cent show Class III, and 11.8 per cent of the children are normal or borderline normal. The majority of parents (64.8 per cent) are Class I. Here the malocclusion is associated with mutilation in many instances. Class II malocclusions rank second among parents (21.3 per cent), normal occlusion obtains in 9.8 per cent and Class III is found in 4.1 per cent of parents.

The detailed distribution indicates clearly that hereditary factors are involved in the production of malocclusion, although the patterning according to occlusion type is not clear. It is to be noted that the majority of those children showing normal occlusions in this series, present at least one parent with a normal occlusion and with the other parent tending to show a Class I malocclusion.

Children in this series present a high incidence of malocclusion. This is to be expected since the basis of selection of the sample is the presence of a malocclusion in at least one child in each family represented. There is evidently an operation of hereditary factors in the production of malocclusion but the patterning is not clear. It is more probable that occlusion is dependent upon many traits, each of which is dependent to a greater or lesser degree upon hereditary factors as indicated in the previous sections of the discussion.

DIAGNOSIS AND TREATMENT

Two points have been brought out in the analysis which warrant further consideration from the standpoint of the bearing they have on diagnosis and

TABLE XXX

OCCLUSION

PARENTS		CHILDREN SHOWING NORMALCY NO.	%	CHILDREN SHOWING CLASS I NO.	%	CHILDREN SHOWING CLASS II NO.	%	CHILDREN SHOWING CLASS III NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Mn	Fn	0	0.0	0	0.0	0	0.0	1	100.0	1	1
Mn	FI	1	11.1	5	55.6	3	33.3	0	0.0	6	9
MI	Fn	1	50.0	1	50.0	0	0.0	0	0.0	1	2
MI	FI	2	5.6	24	66.7	9	25.0	1	2.8	22	36
MI	FII	2	20.0	4	40.0	4	40.0	0	0.0	5	10
MII	Fn	1	25.0	0	0.0	3	75.0	0	0.0	3	4
MII	FI	0	0.0	4	57.1	3	42.9	0	0.0	2	7
MII	FII	0	0.0	4	80.0	1	20.0	0	0.0	4	5
MI	FIII	0	0.0	0	0.0	0	0.0	1	100.0	1	1
MII	FIII	0	0.0	0	0.0	1	100.0	0	0.0	1	1
MIII	FI	2	66.7	0	0.0	0	0.0	1	33.3	1	3
MIII	FIII	0	0.0	0	0.0	1	100.0	0	0.0	1	1
Mn	Fun	0	0.0	0	0.0	0	0.0	1	100.0	1	1
MI	Fun	1	6.3	9	56.3	5	31.3	1	6.3	11	16
MII	Fun	3	23.1	3	23.1	6	46.2	1	7.7	6	13
Total		13	11.8	54	49.1	36	32.7	7	6.4	66	110
Unobserved										12	20
Total										78	130

treatment. The first of these is the extensive operation of heredity in the production of features in the dentofacial complex. If we assume—and we seem obliged to do so—that an individual is developing along the lines of his biologic expectation when he gives full expression to the hereditary circumstances that are initially responsible for him, we must recognize that many undesirable traits biologically are fully normal. We can no longer retain the point of view, "Nature never makes a mistake," with the subsequent assignment of all irregularities and anomalies to nurtural, developmental, or mechanical interferences with nature. And we must recognize that orthodontic procedure will be outlined on a policy of judicious interference with nature as frequently as it will be formulated on a basis of cooperation with nature. Probably the most important contribution hereditary knowledge makes to diagnosis is in the outlining of expectation for the growing child, especially in the bracket of attributes which are markedly influenced by age. It is pertinent to cite some examples. The arch form of the young child with a deciduous dentition characteristically is ovoid both above and below. There is some variability in the deciduous arch form and we find a few cases of markedly tapering arches as well as a few examples of hyperbolic arches. The majority, however, are more or less ovoid. While we have reasonable evidence that the tapering deciduous arch predicts a probably more extremely tapered adult arch, we can know little what the deciduous ovoid arch predicts without recourse to other evidence. We may do two things. The first is accurate: wait until the adult arch form has been observed before describing it. This ordinarily means that we must wait until the child is from 10 to 13 years old before we can make accurate appraisal. The second method is to estimate what the arch form will be through use of associated facts which permit useful generalizations. In arch form heredity provides this body of facts. We note that arch form is strongly dependent upon hereditary circumstances with a general dominance of narrowed or constricted arches

over broad ones. Thus if we know whether one, the other, or both parents present constricted arches, we are provided with a considerable body of evidence about the probable course of development in the child. In a like manner the other hereditary evidence in the dentofacial complex, as size and placement of the various parts of the mandible and maxilla, depth of bite, anterior crowding of teeth, etc., serves further to define the probable course of development of the child. We believe precision in estimate of growth sequences and adult configurations in the dentofacial complex to be fundamental in etiology and diagnosis of malocclusion. Further it appears axiomatic to us that a treatment procedure should be based upon etiologic and diagnostic evidence of the individual case.

The second point to be considered is the nature of a malocclusion or, better, of occlusion. Occlusion cannot be classified as an attribute; it is a configuration of many attributes. At the moment, we are not in a position to give an estimate of the number of attributes in the dentofacial complex. We know there are many rather than few and that the number of combinatorial groupings approaches astronomical figures. We estimate that half, or more, of the twenty-four pairs of human chromosomes are represented in the area with the resultant probability of a high degree of attribute independence. This occlusion, by definition a relationship phenomenon, is established largely by the probability circumstances which juxtapose the attributes of the dentofacial complex with one another. The size of the teeth may or may not conform to the amount of containing alveolus. The alveoli present but low order covariation with their supporting maxillary and mandibular bases. The mandible is far from an integrated whole; its several parts—condylar and coronoid processes, ascending rami, angles, body, alveolar process, and individual teeth —show a fairly high degree of independent variation. This same type of part independence is evident in the maxillary complex, and certainly, the maxillary and mandibular complexes are independent of one another.

The variations within and between the several parts of the dentofacial complex have a strong foundation in heredity; consequently an understanding of the operation of these phenomena is important in outlining treatment procedures. Somewhat as an aside may be added that nongenetic variability frequently is present. There is no doubt that the continued practice of oral habits, as thumb-sucking or cheek-biting, or mutilation of the dentition through extensive operation of caries or premature loss of teeth is orthodontically undesirable. It would be folly of the highest order to omit a consideration of environmental or nurtural circumstances in seeking facts about occlusion. Nor can one view without alarm the widespread laissez-faire attitude that the dentition and supporting structures are self-readjusting to all kinds of variations which are imposed by nonhereditary forces. Our sample fails to provide much evidence for the incidence of environmental variations of the above kinds for the total population. In our group hereditary variations are far more frequent than nonhereditary ones and the bulk of treatment cases have their primary etiology in attributes of the former type. These are particularly striking in Class II and Class III cases of all kinds and in Class I cases involving constriction in the anterior part of the arch and crowding of maxillary and mandibular anterior teeth.

The literature has made frequent reference to the proposition that a malocclusion which had a genetic foundation was not amenable to treatment or, if it were, the treatment would be more difficult and the frequency of relapse higher than if nongenetic factors only were responsible for the malocclusion. The proposition has some truth in it but on the whole, it is not accurate enough to warrant an unqualified recommendation to the orthodontic profession. Any structure may be modified within greater or lesser limits and difficulty usually is encountered when there is an attempt to exceed these limits. In the dentofacial complex there is considerable difference in the modification limits of the various parts. The individual teeth present probably the narrowest limits; their size, shape, number, and rate of growth are almost fixed. The size of the tooth can be changed by destroying parts of it or by restorative processes of various kinds. Caries activity can destroy all or part of a tooth. The tooth can be returned to its original status or to a different status by many kinds of restorative work. A peg lateral may be enlarged by a jacket crown. The number of teeth present may be changed by extraction or by addition of artificial teeth. At present we seem to be able very little either to inhibit or to accelerate the rate of growth. We see then that while we may produce many modifications upon the dentition through surgical techniques of one kind or another, there is little we can do to produce modification of the individual tooth *so long as it is left intact*. Thus most orthodontic change must be about the teeth and not within them. Other areas in the dentofacial complex likewise have narrow limits within which modification may be instituted without resorting to surgical practice. Little can be done to change the size, shape, and configuration of the mandibular condylar and coronoid processes, the rami, the angles, or the body. Almost equally resistant to change is the complex of structure that constitutes the maxillary apical base. So far as we know we can do little either to stimulate or to retard its growth.

The two parts which offer the widest latitude for modification are the maxillary and mandibular alveolar areas. Genetic phenomena appear to be responsible for the amount of alveolar bone to be provided and the rate, through growth, at which it will be provided. Whether we can stimulate a greater amount to be grown than this remains to be demonstrated. We think, probably not!

We pose then the proposition that effective orthodontic work of all kinds may be done within the limits set by the amount of alveolar bone. An appraisal of the amount available at a given time can be made by direct examination and a conservatively accurate estimate of the amount yet to be obtained though growth can be ascertained by recourse to hereditary evidence.

From the standpoint of treatment hereditary evidence serves to outline further the technique to be used. Some of these treatments may be termed comprehensive and others compromise in accordance with usage already established in orthodontics. In some of the compromise cases, especially those involving extraction of teeth or resection of the mandibular rami, knowledge of heredity is very useful. Fewer departures from standard practice are found, perhaps, in the comprehensive types of treatment. Here information concerning heredity functions primarily to guide expectation of what growth will accomplish.

Finally this is a preliminary report on our findings to date (April, 1942). None of the problems initially posed have been solved in the detail we believe essential for the profession. We know, at least, that hereditary factors are present and important in the dentofacial complex. We have found that knowledge of heredity is useful in guiding the operator in orthodontic practice.

Subsequent work will present case reports in which information concerning heredity is used as a basis for treatment and will present more complete and more precise information on several details brought out in the body of the paper.

REFERENCES

1. Guyer, M. F.: Abnormalities of Teeth and Jaws From the Standpoint of Inheritance, INT. J. ORTHODONTIA AND ORAL SURG. 10: 323, 1924.
2. Phillips, George P.: Probable Primary Factors Controlling Occlusion, INT. J. ORTHODONTIA AND ORAL SURG. 13: 379, 1927.
3. Brash, J. C.: The Etiology of Irregularity and Malocclusion of the Teeth, The Dental Board of the United Kingdom, 1929.
4. Charles, S. Wilson: Environment, Heredity and Malocclusion, INT. J. ORTHODONTIA AND ORAL SURG. 17: 566, 1931.
5. Johnson, A. LeRoy: Studies in Genetics of Interest to the Orthodontist, INT. J. ORTHODONTIA 19: 273, 1933.
6. Crouch, S. Stuart: Building Efficient Masticatory Machines, INT. J. ORTHODONTIA 21: 248, 1935.
7. Casto, Frank M.: Orthodontia and the General Practitioner, INT. J. ORTHODONTIA 21: 803, 1935.
8. Goldstein, Marcus S., and Stanton, Frederick L.: Various Types of Occlusion and Amounts of Overbite in Normal and Abnormal Occlusion Between Two and Twelve Years, INT. J. ORTHODONTIA AND ORAL SURG. 22: 549, 1936.
9. Sly, Walter J.: The Biologic Phase of Orthodontia, INT. J. ORTHODONTIA AND ORAL SURG. 22: 691, 1936.
10. Johnson, A. LeRoy: The Constitutional Factor in Skull Form and Dental Occlusion, AM. J. ORTHODONTICS AND ORAL SURG. 26: 627, 1940.
11. Weinberger, Bernhard Wolf: Orthodontics—An Historical Review of Its Origin and Evolution, St. Louis, 1926, The C. V. Mosby Co., Vol. 1, page 396.
12. Hooton, E. A.: Indians of Pecos, New Haven, 1930, page 80.