

# The Angle Orthodontist

VOL. X

OCTOBER, 1940

No. 4

## Growth Theory and Orthodontic Practice \*

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SOME MONTHS ago I was discussing with my students in Anthropology the various approaches to the study of problems of human growth and development. We finally evolved the following, a bit on the facetious side perhaps, but none the less indicative of a really complex situation:

Growth was conceived by an anatomist, born to a biologist, delivered by a physician, left on a chemist's doorstep, and adopted by a physiologist. At an early age she eloped with a statistician, divorced him for a psychologist, and is now being ardently wooed, alternately, by an endocrinologist, a pediatrician, a physical anthropologist, a physicist, a biochemist and a mathematician. A short while ago there was some talk of a eugenicist, and only last week a newcomer, looking suspiciously like an orthodontist, was seen loitering in the vicinity.

If growth be anatomical, biological, chemical, physiological, endocrinological, biochemical, mathematical we must somehow attempt to analyze basic principles of value to the practice of orthodontia—and that is what this paper aims to do: first, to outline certain general principles of growth; second, to discuss orthodontia in terms of dento-facial growth and development. Theory first, then practice.

According to Pearl (1933), after Child, the general vertebrate plan of structure has a primary axis which is longitudinal, with cephalic and caudal ends. There are two secondary axes at right angles to each other: a dorso-ventral and a lateral; both are at right angles to the longitudinal axis, so that growth is in three planes. The external form of the vertebrate organism—the somatology or habitus—is therefore based on the relative and proportional growth in *each* of these three axes. Balance and symmetry are thus inherent in the basic vertebrate growth pattern. We may analyze the three axes even further: growth in bulk is achieved in all three dimensions; growth in shape or proportions proceeds differentially in the first *or* in the second or third dimensions. It is not mere bulk, therefore, that growth aims at, but the harmonious interplay of component dimensions. In other words, growth is not uniform, but differential or proportional.

\* This paper is a composite of addresses before the Twelfth Biennial Meeting of the Edward H. Angle Society of Orthodontia, October 13, 1939 and the Chicago Association of Orthodontists, September 30, 1940.

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The achievement of growth is, according to Cohen (1940), determined by two major reactions, the one governing proportionate growth in all physical dimensions, the other proportionate growth in purely length or circumferential dimensions. Individual differences in the external measurements of the body are due to a process making for general magnitude and a process making for proportion in areas or parts of the body. There are, in short, genetic factors which determine growth or size of the body in all of its dimensions: vertical, horizontal, sagittal; over and above these are genetic factors tending to excess or differential growth longitudinally or circumferentially. The first set of genetic factors is general, the second specific; statistically the former set accounts for from two to three times the amount of variance in growth, compared to the latter. We may raise the question if it is necessary to postulate two sets of genetic factors. May there not be but one set with, however, rate genes for each organ, or complex of organs, each with a specific functional life span? These may well cluster in two sets—a general and a specific—but in final analysis there may be reference to only one basic growth impetus. Notwithstanding Nature's aim at symmetry general growth is variable, due largely to ontogenetic environmental vicissitudes. This does not mean disharmony in a given pattern, but merely variability of patterns and in short, accounts for two things: individual differences and the transmission of these individual patterns through heredity.

Further support to this conclusion is lent by Grüneberg's (1937) study of the grey-lethal mouse. In this mutant the fur lacks pigment, no tooth ever erupts, the skeleton lacks all secondary resorption processes, and there is generally incomplete calcification of bone and teeth. The size and shape of an individual tooth is limited and influenced by that of the socket at the time the tooth becomes enclosed in the socket: teeth which still have to grow are deformed; teeth whose growth is completed develop normally. We must now revise our old idea of cause-and-effect sequence in tooth eruption. No longer is it: stimulus (pressure)—response (resorption); but now it is: stimulus (pressure)—hereditary basis for response—response (resorption). The idea is now not growth *per se*, but growth in obedience to genetic pattern and genetic potential.

We have come this far: there is a basic vertebrate plan of bodily organization which is symmetrical and proportionate; growth is in three planes, aiming at bulk, but within this bulk at linearity and laterality (transverse and sagittal), so that individual differences in form are achieved; these individual differences in form are transmitted in heredity as discrete patterns, so that while there may be variation in patterns as such there is little or no variation normally within the pattern, for it has a germinal or genetic basis. Stated in more general terms, we mean, simply, that there is no variation in the phylogenetic pattern—the basic or inherent growth force—but only in the ontogenetic unfolding—the individual sequence, mercilessly exposed and subject to environmental impacts.

The next step in our analysis is the consideration of the *human* pattern of growth. This is done in the accompanying table on periodicity in human growth, compiled from a number of writers. There is some disagreement as to period-divisions, but there is a basic concordance in that all recognize

the unevenness of form or outline growth pattern, as distinct from mere additive increment. Time has no constant value in growth and development of the child: there are "fat" (fill-out) and "lean" (spring-up) years, spurts of rapid growth, phases of slow progress. In their consideration of stature Simmons and Todd (1938) do not find marked fluctuations, but Berkson (1930), upon closely analyzing Woodbury's data found for both height and weight a parabolic curve with the convexity upward, showing a seasonal wave sweeping along each general curve-trend.

It is inescapable that the growth pattern of general body size and pro-

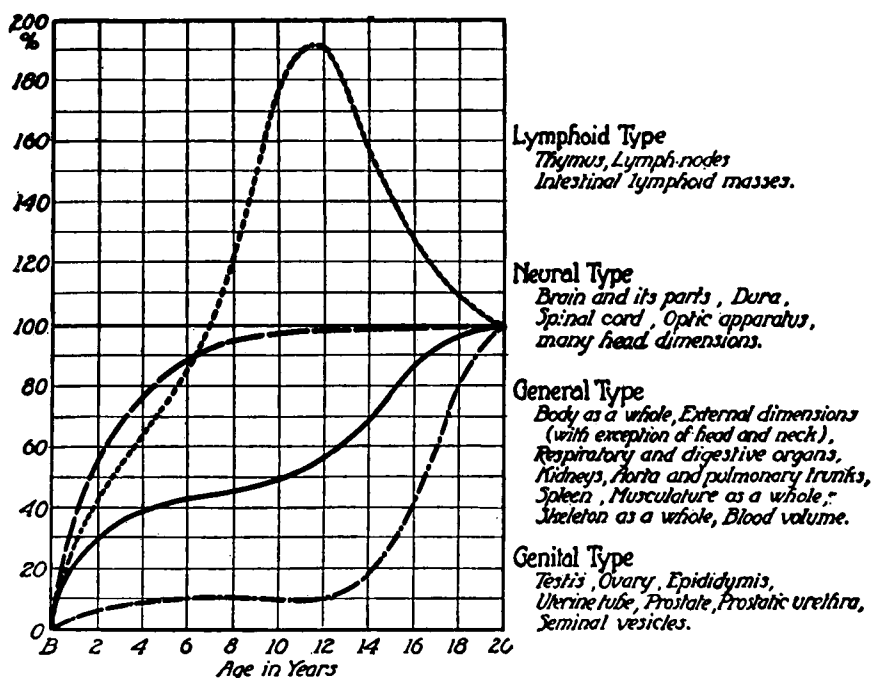


Fig. 1.—A graph showing the major types of postnatal growth of the various parts and organs of the body. The several curves are drawn to a common scale by computing their values at successive ages in terms of their total postnatal increments (to twenty years). (Illustration from Scammon, R. *Measurement of Man*, Rochester, Univ. of Minn. Press, 1930, page 193.)

portions in man demonstrates some sort of periodicity, of interplay between mass and parts, between longitudinal and lateral increments. The end-product of the pattern is, of course, one of integration; the periodicity is not irregular, it is rhythmical, alternate so that harmony and symmetry are ultimately achieved, assuming no growth interference. There is one rule in growth to remember: perfection may be the standard, but adequacy is the goal. This sounds like a compromise or "appeasement," but in truth it is little more than a recognition of the fact that our knowledge of human growth is still imperfect to the point that we are glad to settle for functional usefulness.

We now go a step further in the analysis of human growth, turning to Scammon's (1930) differentiation of growth-types in the human body (see

Fig. 1). The curves in this figure are drawn to a common scale by computing their values at successive ages in percentage terms of their total postnatal increments, up to 20 years. There are four major types of growth-curve: *lymphoid* (thymus, lymph-nodes, intestinal lymphoid masses), *neural* (brain and its parts, dura, spinal cord, optic apparatus, dimensions of head and face), *general body* (body as a whole, external dimensions except head, face and neck, respiratory and digestive organs, kidneys, aorta and pulmonary trunks, spleen, musculature as a whole, skeleton as a whole, blood volume), and *genital* (testis, ovary, epididymis, uterine tube, prostate, urethra, seminal vesicles, secondary sex characters generally). The curve of lymphoid growth accelerates rapidly up to about 12 years, then decelerates until the adult value is reached at 20 years; the curve for neuro-cranial growth accelerates even more rapidly, up to about 4-6 years, and from then on the curve is almost asymptotic; the curve for general body growth accelerates to about 4 years, is quiet until about 12 years, and then accelerates until about 18 years (actually this is the "spring-up" and "fill-out" interplay noted earlier); the curve for genital growth is relatively quiet until about 12 years, when pubertal endocrine influences cause a sudden and marked acceleration.

Once more let us assess our progress to this point: in addition to basic vertebrate plan and to hereditary transmission we have added the concepts of rhythmic interplay of growth periods, and types of growth within the organism. We have brought growth theory from a general vertebrate to a specific human pattern, demonstrating that an orderly variability is expected in terms of individual variation. We are now ready to fit these concepts—all or part of them—into considerations of orthodontic practice.

We may accept, without further discussion, the fact that Man, as a vertebrate, conforms in all major respects to the basic plan of vertebrate organization outlined by Child and Pearl. The fact that in Man the cephalic end is so profoundly specialized complicates but does not distort the picture.

The observations by Cohen regarding the two major genetic growth reactions find response in Brodie's (1940) and Schour's (1940) recent studies of the human cranio-facial growth pattern. It will be remembered that Cohen suggested that there were general and specific genetic factors. Brodie and Schour point out that, on the basis of alizarination, there is early general bone deposition and subsequent local activity of varying intensity. We may speak, therefore, of *generalized* growth and *localized sites* of growth, the one probably referable to increase in size, the other to the changes in proportion that are typical of maturity. Here, too, the cranio-facial pattern accords with basic genetic theory. We would emphasize again that generalized and localized growth are merely different phases of a single growth impulse, but with different gradients plotted against time.

We have, in our analysis of dental development and facial expansion, assumed rather a direct relationship between the two, often stating that if the teeth all erupt correctly in time and sequence facial growth is normal, and vice versa. Recently Speidel (1939) reported on three children whose endocrine balance and familio-hereditary history was quite normal, yet in whom there were marked dento-facial aberrances. In a 5-year-old girl dental development was normal yet facial dimensions were 11-23 mm. smaller than

Hellman's standards for Stage II A; a 14 yrs., 8 mos. girl with normal dental development showed facial dimension 16-26 mm. smaller than Hellman's IV A; an 8 yrs., 11 mos. boy with defective dental development had facial dimensions equal to or larger than Hellman's III A. Apart from the problems of comparability and of family-line size, the fact remains that here, in individual instances, dento-facial imbalance is asymmetrical, i.e., there is a normal dental development picture associated with abnormal facial dimensions, and vice versa. It seems to me that here Grüneberg's observations are apropos, viz., that simple cause-and-effect conclusions are no longer tenable. I am not suggesting, of course, that these cases represent mutants, but I do agree with Speidel that we must assume a greater independence of dental development and eruption and facial growth than heretofore accepted. This dissociation is logical for eruption is the more easily modifiable by environmental factors.

The problem of heredity in dento-facial relationships is a difficult one. We have first the suggestion of Schultz (1925) that in evolution teeth and dental arch have decreased in size disproportionately, so that relatively large teeth in a relatively small jaw are possibly an indication of evolutionary trend. We know, further, that suppression of the upper I 2 is a Mendelian autosomal dominant, that progenia is apparently dominant and "dwarf mandibles" recessive, that broad faces and palates dominate over narrow faces and palates. These are heritable, transmissible entities. I have yet to see, however, any really acceptable evidence of the transmission from either parent of unit characters that in their unique combination are disharmonious, e.g., narrow palate from one parent, short from the other. Fleming (1939) in a few isolated instances reports disharmonious dento-facial relationships in race-mixtures of Negroes, Chinese, Malay, White and the backcrosses of their blends. In no such case, either in a family-line or in a race-mixture, has it been proven beyond doubt that the inheritance of discrete unit characters has been *solely* responsible for the asymmetry. Rubrecht (1939) has taken a step in the right direction in his study of the hereditary transmission of jaw anomalies, pointing out that shape and size are "in great measure determined by heredity," and that "consequently endognathism and exognathism are also, to a great extent, dependent on heredity." It would be folly, therefore, to eliminate familial patterns from consideration; too many practicing orthodontists see in the child's mouth a reflection of a similar condition in Mother or Father. What I do insist on, however, is that as yet we do not know enough about human dento-facial heredity in terms of symmetry or asymmetry, correct occlusion or malocclusion, as determined by harmonious or disharmonious transmission of unit traits. We do have important leads, however, in Johnson's (1940) report on the cross of dogs in Stockard's laboratory. Johnson observes:

The independence in genetic constitution of the maxillary structures from the mandible and the independence of the teeth from both is demonstrated . . . clearly. . . . Structural disharmony, even to the extent of grotesqueness, may be an expression of the very nature of the organism itself.

The importance of underlying genetic constitution must not be minimized.

We come now to the problem of periodicity in dento-facial growth. In the first instance it must be noted that we are concerned with growth in three

planes: height, which is really aligned with the cephalo-caudal longitudinal axis; width, transversely at right angles to that axis; length or depth, sagittally at right angles to that axis. The height dimension is in the primary vertebrate axis, width and length in the secondary vertebrate axes. More than this, the dimensions are absolutely unequal: width is greatest, height next, depth least; this inequality is in part compensated by growth velocities in order of magnitude of depth, height, width. The broad, moderately high shallow face of the child becomes the deeper, moderately high, relatively narrow face of the adult. If we analyze general facial expansion there is a very rapid prenatal acceleration which carries over postnatally until 5 years,

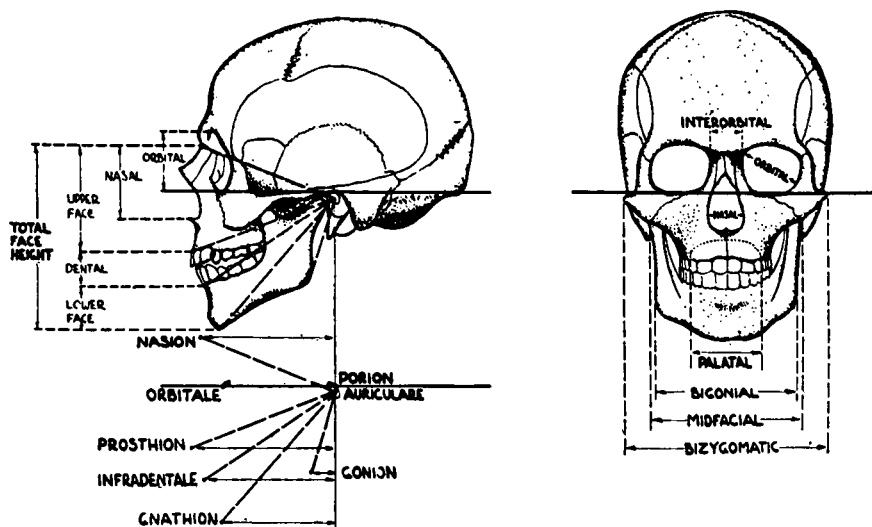


Fig. 2.—The measurement of facial size in height, breadth, and length. Length is shown measured radially from auriculare or projected from the vertical through the porion. (Illustration from Krogman, W. M., *Facing Facts of Face Growth*, Am. J. Orthodont. and Oral Surg. Vol. 25, p. 725, Aug. 1939.)

especially between birth and 2 years; there is a generally continuous retardation until 13 years, a pubertal acceleration at 13-15 years, and retardation to virtual cessation at 21 years. Specific facial expansion may be analyzed: for height there are periods of acceleration birth—1 year, 3-4 years, 7-11 years, 16-19 years; for face length birth—1 year, 4-7 years, 16-19 years; face breadth is slow up to 6 years, but rapid 6-12 years and 16-19 years, the latter especially marked in males. Growth in the face is, veritably, increase in dimension, but unequal in proportionate rate.

The inequalities of facial growth are not alone in general form, but in individual parts or sites as well. Height growth is in upper face (orbito-nasal), mid-face (naso-alveolar), and lower face (dento-mandibular); width growth is mid-line (orbito-nasal and palatal) and lateral (bizygomatic and bigonial); depth growth is cranio-facial (adjustment at hafting zone, especially the sphenoid complex) and facial (dento-palatal). Add to these incre-

mental phenomena those of positional adjustment and angular relationship and the picture becomes increasingly complex. It is no wonder when a "normal" pattern must be a balance of diverse and complex elements, that combinations occur from time to time that demand remedial attention.

In Fig. 2 is a graphic representation of the standard measurements of the facial skeleton (see Krogman, 1939). Height dimensions are for total face from *nasion*, mid-line junction of nasal suture with naso-frontal suture, to *gnathion*, mid-line point on mandible where anterior border curves into inferior border; upper face height is from *nasion* to *alveolar point*, mid-line point on septum between upper central incisors; lower face height is from *gnathion* to *intradentale*, mid-line point on septum between lower central incisors; dental height is from *alveolar point* to *intradentale*. Breadth dimensions are bizygomatic, between right and left *zygion*, most lateral points on the zygomatic arches; midfacial, between right and left *zygomaxillare*, lowest point on the anterior aspect of the zygomaxillary suture; mandibular breadth, between right and left *gonion*, point on the angle of the jaw determined by intersection of plane of lower border of body and plane of posterior border of ascending ramus; palatal breadth is taken at level of upper second deciduous or permanent molar, either on the lingual or the buccal side. Length or depth dimensions are taken from the region of the ear-hole, either radially to *nasion*, *prothion*, etc., or projected distance of these points from a perpendicular to the Frankfort Horizontal (plane through lower margin of left orbit and upper margin right and left ear-holes) erected at *porion*, most lateral point on the roof of the external auditory meatus.

A word of caution is in order: the definitions above given are only general. The location of points on the skull is a very precise technic; their transference to the living is just as precise and even more difficult. The role of anthropometry in orthodontia is an important one, but not a simple one. It is a useful tool only in the degree that it is carefully applied and conservatively interpreted.

Brodie (1940) states that the morpho-genetic pattern of the head is established by the third postnatal month and that "once attained it does not change." This conclusion introduces us to our next theme, viz., the relation of facial growth to growth type-pattern. Let us look at the following figures which I have calculated from Hellman (1927):

Age	Face Height (Na-Gn)		Face Width (Zy-Zy)		Face Depth (Go-Gn)	
	mm.	% Ad.	mm.	% Ad.	mm.	% Ad.
Birth	M 47.0	38.3	78.0	57.7	—	—
	F 46.9	40.5	76.2	58.0	—	—
2 years	M 83.1	67.7	111.4	79.5	75.0	75.8
	F 79.3	58.5	109.1	82.9	75.8	82.4
5 years	M 96.1	78.6	117.1	83.8	79.9	80.7
	F 91.6	78.7	113.3	86.4	77.9	84.7

These figures are instructive from several viewpoints: first, the amount of facial growth achieved at birth and by the fifth year; second, the unequal

rate in the component parts, with the inequality smoothed out, as it were, during the growth process. At birth 39% of height, 57% of width, measured against adult size, have been achieved. In the first five years 78% of height, 85% of width, 82% of depth have been achieved (these are average for the sexes). The importance here is not so much an initial inequality and its subsequent reduction, but the fact that after five years of age only 15-20% of growth increments remain as avenues of possible readjustment. Further, we illustrate once more the tremendous rate of increase by generalized growth and the slower rate of change in proportion by localized growth. In both periods growth gradients effect changes in proportion, true, but the re-modeling is later and slower.

In this connection, the tables given below, calculated from Goldstein (1936) are of equal importance, in that they take us beyond the five-year

PERCENTAGE INCREMENTS OF FACIAL HEIGHT

Age	Total (Na-Gn)		Upper (Na-Pr)		Lower (Inf-Gn)	
	% Inc.	% Ad.	% Inc.	% Ad.	% Inc.	% Ad.
4-5	19.7	76.8	30.4	80.2	20.2	79.0
6-7	17.1	81.5	20.0	85.6	4.4	80.2
8-9	13.0	85.0	11.3	88.6	3.8	81.2
10-11	10.7	87.9	11.3	91.6	13.0	84.6
12-13	10.8	90.9	5.5	93.1	22.7	90.6
14-15	18.7	96.0	22.8	99.2	22.3	96.5
16-17	5.3	97.5	3.1	100.0	10.7	99.3

PERCENTAGE INCREMENTS OF FACIAL WIDTH

Age	Bizygomatic (Zy-Zy)		Bigonial (Go-Go)		Nasal	
	% Inc.	% Ad.	% Inc.	% Ad.	% Inc.	% Ad.
4-5	25.1	82.1	22.4	80.3	15.5	77.5
6-7	15.5	85.8	11.1	83.0	11.2	80.5
8-9	14.4	89.3	18.0	87.3	16.4	84.8
10-11	10.5	91.8	17.0	91.4	12.2	88.1
12-13	10.4	94.3	11.3	94.1	12.0	91.3
14-15	7.8	96.2	9.0	96.3	19.5	96.5
16-17	10.4	98.7	8.3	98.3	9.3	99.0

PERCENTAGE INCREMENTS OF FACIAL DEPTH

Age	Auriculo-nasion		Auriculo-prosth.		Auriculo-gnath.	
	% Inc.	% Ad.	% Inc.	% Inc.	% Inc.	% Ad.
4-5	12.7	83.5	14.1	80.5	21.1	74.9
6-7	14.5	86.2	20.9	85.3	16.4	80.1
8-9	14.5	88.9	8.2	87.1	9.3	83.1
10-11	19.3	92.5	18.4	91.3	13.8	87.5
12-13	8.2	94.1	2.5	91.8	1.2	87.8
14-15	24.5	98.7	15.5	95.4	13.7	92.2
16-17	7.1	100.0	9.7	97.6	12.5	96.2



stage. In parallel columns, the percentage annual increment is compared with percentage growth toward the adult size.

In every one of the foregoing dimensions 90% or more of final adult size (general growth) has been attained by the age of twelve years—a fact which demonstrates only 10-15% of local growth in the seven-year interval of five to twelve years. Once more the relative velocity of the first five years is emphasized.

These general observations are affirmed by the intensive and extensive studies on cranio-facial growth reported by Davenport (1940), who considers his data from every possible angle: general group average, sex differences, and variations introduced by familial background, racial participation, socio-economic status, and individual difference. No matter how the data are considered an early basic velocity is fundamental.

As a general rule the rate of growth in facial height, width, and depth is fairly uniform in the five-to-twelve interval, gradually decelerating until by the age of fifteen a balance is reached. The 5% of growth remaining is quite evenly distributed. The relatively late lower facial height (mandibular) growth is of note; it is sex-linked, more emphasized in males.

The evidence from the studies of Hellman and Goldstein is substantiated by the work of Brodie (1940) and Young (1937), although the age-periods are not directly comparable. For upper face height (nasion to anterior nasal spine) Brodie reported 27.6 mm. at three months, 44.4 mm. at 8 years; the three-month measurement was 62.2% of the 8-year dimension. From Young's data I have prepared the following table:

Dimension	Male			Female		
	2-3 yrs.	12-13 yrs.	% at 2-3 yrs.	2-3 yrs.	12-13 yrs.	% at 2-3 yrs.
<i>Length</i>						
Transmeatal axes-nasion	78.5 mm.	90.7 mm.	86%	77.0 mm.	89.0 mm.	86%
<i>Height</i>						
Nasion-subnasal point	83.1 mm.	103.2 mm.	80%	79.3 mm.	101.8 mm.	78%
<i>Breadth</i>						
Bizygomatic	111.4 mm.	125.6 mm.	88%	109.1 mm.	123.6 mm.	88%

The several dimensions at 2-3 years are all over 80% of the values achieved at 12-13 years. The growth increments in the two sexes are identical save for the height dimension, which may represent an absolute size difference. This is seen when at 2-3 years L, H, B is calculated for female in per cent of male: 98%, 95%, 98% respectively; at 12-13 years the same calculation gives 98% for each dimension. There has been a slight differential height gain in favor of the female.

Further discussion would only belabor our point: that facial growth in width, height, depth belongs to the *neural* mode of growth—it belongs to head, even though cranial growth has a greater velocity and terminates earlier than facial growth.

We come now to a consideration of some of the activating factors which may guide the face in its developmental career. One of the most obvious factors is that of the general constitutional welfare of the child, a

theme so well elaborated by Todd and his associates that detail is unnecessary here. One quotation by Todd (1932) will suffice:

In infancy, when the constitution is most susceptible to disturbance, it is the face rather than any other part of the body which registers permanent damage through interruption of facial growth, a damage which shows little tendency to repair and is forever afterwards merely compensated by structural modification.

We may well ask why the face is so hard hit. The answer perhaps may be that it is a very complex structure serving several highly specialized functions. This does not include the most important reason, viz., that it is peculiarly vulnerable by virtue of its "time specificity," inherent in its rapid rate of growth. Constitutional impairment may be and generally is reflected in the skeletal development of the child. As a result, not only are growth increments reduced or inhibited, but efforts at restorative treatment are frustrated. The demineralized bones of the jaws, already deficient in normal size, are unable to retain corrective impulses transmitted to them by appliances. Goldstein and Stanton (1936) give ample proof of this in their thorough studies of tooth movement, for it is precisely in jaws already maloccluded by virtue of unequal growth that individual adjustment in position is the most haphazard.

Still another phase of constitutional inadequacy is to be found in large, fleshy adenoid masses and congested turbinates, structures which may interfere with the freedom of the respiratory passages but do not necessarily disturb the growth pattern. This is not the entire picture, however, for facial type enters in. In the broad, low face growth begins earlier and has a more rapid rate over a shorter period of time; just the reverse is true of the narrow, high face. It is the latter which possesses the so-called "adenoid facies"; it is in this face that an otherwise normal adenoid mass may be a relative obstruction. It is only when the impeded nasopharyngeal passages lead to improper mouth habits that corrective dentistry need enter.

This is not, however, the entire picture, for nasopharyngeal disturbances are often attributable to a fundamental imbalance in cranio-facial relationship, i.e., an inadequate forward drift of the mask to open up nasopharynx. The work of Rosenberger (1934) has demonstrated the importance of this forward growth in the early years when he concludes that "already by the age of five years the pattern of upper facial development is established. Defects of growth originate, therefore, in early childhood. . . ."

The effects of the endocrinopathies in facial growth have been ably summarized by Schour and Massler (1940). In hypopituitarism eruption is markedly retarded; vertical height of jaws and face and arch length are inhibited so that teeth are crowded and submerged; paranasal sinuses are small. In hyperpituitarism eruption is accelerated; vertical height of face increases and the arch is longer; the teeth are not larger; paranasal sinuses are large. In hypogonadism eruption may be retarded; the rate of endomembranous bone formation is increased so that jaws and sinuses are larger. In hypothyroidism eruption is retarded, though less than in hypopituitarism; alveolar bone growth is retarded so that arch is narrow, teeth are submerged; paranasal sinuses are small. In hyperthyroidism eruption may be slightly precocious; effect on facial growth is not clear. In hypo-

and hyperthyroidism effects on eruption and facial growth are obscure, save that in the latter condition there is resorption of the alveolar bone.

Obviously the time factor is of the utmost importance, particularly with reference to before or after six years of age, as pointed out by Schour and Massler. Before six years is the period of very active generalized bone growth: periosteal, endosteal, sutural. Here the impact of systemic upset will result in general hypo- or hyperplasia. After six years is the period of localized growth; here local hypo- or hyperplasia will result. In the first period cranio-facial proportions change but little; in the second period there are marked changes. Not only time *per se*, but duration and vigor of onslaught are further factors to be considered.

We must proceed with caution for specificity of glandular function in the development of the dentition at a time when even pluriglandular effects are imperfectly known is apt to be premature. The endocrines are of the utmost importance but they must be interpreted carefully.

Orthodontia, if we may make bold to reduce the science to a single aim, has for its major purpose symmetry—*functional* symmetry to be sure, but still that harmonious interplay of component parts that makes for balance in the human dentition and face. This functional symmetry, this balance, this harmonious dento-facial relationship, we refer to as “normal,” thereby tacitly accepting a certain intra- and interdental relationship that can be precisely defined in terms of the relation of one tooth to another and the interlocking interplay of cuspidal elements. There is, for the orthodontist, a standard, a norm, a dento-facial harmony which “works right” and “looks right.” It matters not how the norm be set up: functionally, esthetically, etiologically, anatomically, biometrically—we are still concerned with definition. Brodie, Schour and Massler (1940) are on the right track when they recognize three major types of growth aberrancy: (1) hypoplastic, generalized arrest with no distortion of pattern; (2) hyperplastic, generalized accretion with no distortion of pattern; (3) dysplastic, congenital defect or accident affecting particular growth site, with some distortion of pattern. Obviously each of these, with the exception of the fortuitous accident, probably stems back to endocrine imbalance. The important point for the orthodontist, however, is that the deviation in these cases is usually so great as to be easily discernible, readily measurable.

I have said now what I started out to say, that growth and development operate *with* the orthodontist, not against him. Some who read this article may feel that it is all theory, no practice. In part that may be true; the writer is a student of growth, not an orthodontist. He can but offer to orthodontia a tool to be used, a guide to be followed. Dento-facial development conforms in every respect to the biological laws of all organic growth. At every turn in the path of development there are mile-posts so that he who follows the path may read. If this paper has offered but *one* mile-post then indeed growth theory has been translated into orthodontic practice.

### Summary

1. General vertebrate growth has a primary caudocephalo longitudinal axis, with two secondary axes at right angles, a dorso-ventral (sagittal) and a lateral (transverse).

2. Growth is governed by two groups of genetic factors, the one presiding over general size the other over specific proportion; these groups are probably clusters within one major set of factors. Differential variance in the two groups makes for variation in individuals but does not disturb pattern.

3. There is a definite human pattern of growth involving periodicity or rhythmic alteration, always aiming, however, at ultimate proportional and harmonious integration.

4. In the human body there are four major modes of growth, each with its own gradient and pattern: lymphoid, neural, general body, genital.

5. Human growth complies with the general vertebrate plan and is subservient to genetic factors, though with respect to the latter not enough is known concerning the heredity of unit characters in teeth and face.

6. Facial growth, in its incremental and proportional development, follows in general the periodic and rhythmic pattern of the entire body. Specifically it is closest to the neural mode.

7. Constitutional and endocrine factors influence pattern and type. Precise definition of cause and effect is reasonably possible, but more information is needed with specific reference to pluriglandular interrelationships.

8. It is concluded, since dental development and facial growth are so intimately a part of biological processes with known laws, that growth theory is a valuable tool in orthodontic practice.

I wish to record my sincere appreciation to Drs. Brodie, Massler and Schour for the friendship which prompted a critical and very helpful

PERIODICITY IN HUMAN GROWTH

Author	Years	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	
Montbeillard (Height only)	Rapid increase, birth-3 yrs.				Slow, uniform, constant. 3-12½ years									Rapid increase, 12½-15 yrs.			Terminal period of gradual deceleration, 15 yrs. and on					
Vierordt	Childhood, 1-7 years							Boyhood, 8 to 14 years							Adolescent, 14 years and on							
	Suckling birth-1 yr.		Deciduous teeth, 1-7 years					Permanent teeth, 7 years and on.														
Stratz	Suckling birth-1½ yrs.		Neutral child age, 1½ to 7 years					Bisexual child, 7 to 15 years							Puberty, 15 years and to maturity.							
			1st fill-out, 1½ to 4 yrs.		1st stretch 4-7 yrs.			2nd fill-out, 7 to 10 yrs.			Second stretch, 10 to 15 years											
Weissenberg	1st fill-out, birth to 3 years				1st stretch, 3-6 yrs.			Leisurely growth, ♂ 7-11 years ♀ 7-9 years				2nd stretch, ♂ 11-17 years ♀ 9-14 years				Leisurely growth, ♂ to 25 years ♀ to 18-20 yrs.						
Martin	Rapid growth, birth to six years							Slow growth, ♂ 6-12 yrs. ♀ 6-10 yrs.				Rapid growth, ♂ 16-18 yrs. ♀ 14-15 yrs.				Slow growth, ♂ to 25 years ♀ to 18-20 yrs.						
Friedenthal	Suckling, birth-1 yr.		Childhood, 1 to 12 years										Ripening period 12-18 years				Sub-Adult, 18 yrs. and on					
Von Pfaundler	Latent growth B-9 mo.		Rapid growth: play age					Slow stretch: school age					Relative fill-out: ripening									
----- no precise boundaries set here -----																						
H. A. Harri.	1st springing up, B-1 yr.		1st fill-out 1-5 yrs.			2nd spring- ing up, 5-7 yrs.		2nd fill-out, 7-12 yrs.				3rd spring- ing up 12-14 years		3rd fill-out, 14 to 18+ years								

reading of this paper in manuscript. I have taken the liberty of incorporating their several suggestions without other than this general acknowledgment.

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### Bibliography

- BERKSON, J. "Evidence of a Seasonal Cycle in Human Growth," *Hum. Biol.* 2:523-38, Dec. 1930.
- BRODIE, A. G. "On the Growth Pattern of the Human Head from the Third Month to the Eighth Year of Life," Ph.D. Thesis, Graduate School, University of Illinois. (Unpublished.)
- COHEN, J. I. "Are Differences in Size between Parts of the Body due to General or Specific Factors?" *Proc. Nat. Acad. Sci.* 26:524-26, Aug. 1940.
- DAVENPORT, C. B. "Post-natal Development of the Head," *Proc. Am. Philos. Soc.* 83:1-215, Jan. 1940.
- FLEMING, R. M. "Physical Heredity in Human Hybrids," *Ann. Eug.* 9:55-81, Jan. 1939.
- GOLDSTEIN, M. S. "Changes in Dimensions and Form of the Face and Head with Age," *Am. J. Phys. Anthropol.* 22:37-89, Jan. 1936.
- GOLDSTEIN, M. S. and STANTON, F. L. "Antero-posterior Movements of Teeth between Two and Ten Years," *Hum. Biol.* 8:161-97, May, 1936.
- GRÜNEBERG, H. "The Relations of Endogenous and Exogenous Factors in Bone and Tooth Development: the Teeth of the Grey-lethal Mouse," *J. Anat.* 71:236-44, Feb. 1937.
- HELLMAN, M. "Changes in the Human Face Brought about by Development," *Internat. J. Orthodont., Oral Surg. and Radiol.* 13:475-518, June, 1927.
- JOHNSON, A. LEROY, "The Constitutional Factor in Skull Form and Dental-occlusion," *Am. J. Orthodont. and Oral Surg.* 26:627-63, July, 1940.
- KROGMAN, W. M. "Facing Facts of Face Growth," *Am. J. Orthodont. and Oral Surg.* 25:724-31, Aug. 1939.
- PEARL, R. *Constitution and Health*, London, Kegan, Paul, 1933.
- ROSENBERGER, H. C. "Growth and Development of the Naso-respiratory Area in Childhood," *Ann. Otol., Rhinol. and Laryng.* 43:495-512, June, 1934.
- RUBRECHT, O. "A Study of the Heredity of the Anomalies of the Jaws," *Am. J. Orthodont. and Oral Surg.* 25:751-79, Aug. 1939.
- SCAMMON, R. *Measurement of Man*, Rochester, Univ. of Minnesota Press, 1930, pp. 173-215.
- SCHOUR, I. and MASSLER, M. "Effect of the Endocrines on the Teeth, Jaws and Facial Skeleton," *Proc. Dent. Celeb.* Baltimore, Waverly Press, March, 1940, pp. 145-56.
- SCHULTZ, A. H. "Studies on the Evolution of Human Teeth," *D. Cosmos*, 67:935-47 and 1053-63, Oct.-Nov. 1925.
- SIMMONS, K. and TODD, T. W. "Growth of Well Children: Analysis of Stature and Weight, Three Months to Thirteen Years," *Growth*, 2:93-134, Feb. 1938.
- SPEIDEL, T. D. and HIGLEY, L. B. "Dental Development and Facial Growth" (abst.) *J. D. Res.* 18:241-2, March, 1939.
- TODD, T. W. "Hereditary and Environmental Factors in Facial Development," *Internat. J. Orthodont., Oral Surg. and Radiol.* 18:799-808, Aug. 1932.
- YOUNG, M. "Normal Facial Growth in Children," *J. Anat.* 71:458-70, April, 1937.