Prenatal Ordering and Postnatal Sequence in Dental Development

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The chronology of human development involves complex systems of sequence or order that can scarcely be hinted at in the standard texts. Anatomy students learn one order of ossification for the carpal bones, beginning with the capitate and hamate, and proceeding through the triquetral, lunate, and the multangulars. Dental students learn the median order of tooth eruption as first molar (M1), central incisor (I1), lateral incisor (I2), first premolar (P1), canine (C), second premolar (P2), second molar (M2), and third molar (M3). Students of embryology learn the sequence of development of the deciduous teeth in simple mesial to distal sequence, from the deciduous central incisor (i1) to the deciduous second molar (m2) (A through E), and this is as far as most texts currently go.

Such orders as these are, of course, species specific. The carpal bones do not ossify in exactly the same order in *Macaca,* and the carpus is more complex in *Macaca* and *Hylobates* than in man. The order of permanent tooth eruption shows a progressive change through the primates, from a probable M1-M2-M3-I1-I2-P1-P2-C order, to that in recent man. The order of epiphyseal union is notably different in the macaques and langurs from that in man today.[1]

The changes in relative growth of component organs and tissues, which bring the embryo to adult proportions, are together grouped under the label morphogenesis, and together describe how the human embryo becomes, recognizably, a human adult. The uniquenesses of sequence or order of events in developmental timing do not as yet have a comparable name, although chronogenesis is one appropriate label. The order of events and relative timing in man are uniquely human. The remarkable relative delay in the second and third molars, and delayed carpal development relative to dental development, are both part of the sequential process whereby man is developmentally differentiated from monkeys.

Intraspecific Sequence Variability

Not less intriguing, although less known, are variations in developmental sequence or order within the species. A single bone, such as the triquetral, may be 3rd among 28 postnatal hand centers to ossify, or as late as 24th.[2] A cluster of ossification centers of the wrist may be uniquely late, on a family-line basis.[2,3] Different ossification orders may appear in different populations, say in Ecuador, or in Hong Kong, or in Louisiana "blacks" or Negroes.[4]

For the teeth, the range of actual eruption sequences is large (Table 1). I1 may precede M1. P2 and M2 play sequential tag with each other. The permanent canines may be early in the sequence, an ultra-human feature, or late in eruption order, making some individual human beings more primate than hominid in this particular respect.[5] For eight teeth in each quadrant, there are at least 20 known eruption orders, and for seven carpal bones, there are between 12 and 18 known orders of calcification. Even the deciduous teeth participate in sequence differences, of which the m2-c/c-m2 (E-C/C-E) sequence polymorphism is perhaps the best known, both for eruption, and (as shown in radiographs) for crown calcification.[6]

Far from being fixed, the order of developmental events in man exhibits considerable variety, and it is to this variety that we give attention. In so doing we shift our approach from the compilation of sequences of orders based on means or medians, to

TABLE 1
PERMANENT TOOTH ERUPTION SEQUENCES

Hurme (1949)	Means	M1-I1-I2-P1-P2-C-M2-M3 (maxillary) M1-I1-I2-C-P1-P2-M2-M3 (mandibular)
Schultz (1944)		(M1-I1)-I2-(P1-C-P2)-M2-M3
Schultz (1960)		(I1-M1)-I2-(P1-C-P2)-M2-M3
Koski and Garn (1957)		(M1-I1)-I2-(P1-C-P2-M2)-M3
Garn and Lewis (1963)		(M1-I1)-I2-(P1-C-P2-M2)-M3 or (M1-I1-I2-P1-C-P2-M2)-M3

Data from Garn and Lewis.[5]

those observed on an individual basis, whether in embryos studied histologically, in infants studied radiographically, or in children or adolescents, using both casts and oral examinations. Particularly in children with single gene substitutions or in reduplications or deletions of sex chromosomes or autosomes, the order of developmental events may differ dramatically. Altering the chromosomal complement of man from 46 to 45, 47, 48, 49 or 50, in effect simulates new species, new designs for our analytic procedures to encompass.

Sequence Polymorphisms in Man

The textbook eruption sequence for man (M1-I1-I2-P1-C-P2-M2-M3) is only an approximation.[1,5] I2 may precede I1, M2 may precede or follow P2, and C is highly variable, to the extent that it may even precede P1 and (theoretically) may even follow M3. We acknowledge sequential variability by bracketing teeth (P2-M2) to indicate sequence polymorphism, and as we know more about sequential variability, the brackets become wider and wider. The eruption order in the maxillary teeth is not exactly the same as that in the mandibular teeth. The eruption order in females is not exactly the same as that in males, particularly with reference to M2 and C, since C is more often later than M2 in the male, an old primate characteristic.

The mandibular teeth tend to precede their maxillary opponents in the anterior region[7,8] (mandibular I_2 precedes maxillary I^2), yet tend to follow their maxillary opponents in the posterior teeth (maxillary M^2 precedes mandibular M_2). There is a regular gradient in permanent tooth eruption, from comparative precedence of the mandibular teeth anteriorly, to precedence of the maxillary teeth posteriorly (Table 2).

In addition to clinically discovered tooth eruption, tooth formation as seen in radiographs shows that an M2, P2 order of formation may become, in the same individual, a P2, M2 order of eruption[9–11] (Table 3). If attention is shifted from the permanent teeth to their deciduous predecessors, new sequence polymorphisms are shown, and it is learned that young females are not necessarily advanced over young males.

A tidy system of developmental relationships exists, both in tooth size and in tooth development. Comparable teeth (isomeres) on each side of the arch are highly correlated, both sizewise and timewise. Opponents, although in separate arches, are only slightly less so. Adjacent teeth in the same quadrant are slightly less correlated than opponents, in both size and development. Regardless of morphological class, greater distance between teeth in a quadrant

TABLE 2
DECLINING GRADIENT OF MANDIBULAR PRECEDENCE IN PERMANENT TOOTH ERUPTION

	Pima Indians		Quechua Indians	
Permanent Tooth	Mandible First	Maxilla First	Mandible First	Maxilla First
I1	278	5	45	0
I2	229	4	44	2
C	154	9	26	6
P1	42	80	16	22
P2	46	46	18	17
M1	20	11	17	4
M2	42	14	14	6

Note: For data see references 7 and 8.

TABLE 3
CHANGING DEVELOPMENTAL SEQUENCES DURING DENTAL DEVELOPMENT

Stage of Development	No. of Cases	Sequence (%)		
		P2-M2	(P2-M2)	M2-P2
Beginning calcification	178	33	51	16
Beginning root formation	151	74	21	5
Apical closure	24	71	29	0
Alveolar eruption	56	34	29	38

Data from Garn, Koski, and Lewis.[10]

lowers the temporal correlations between them.[12]

Systematic Variability in the Permanent Teeth

Despite complexities and variations among 52 calcified structures (both deciduous and permanent) that are arranged closely and symmetrically in two pairs of supporting bones, three generalizations can be made.

There is a gradient of mandibular to maxillary precedence in the permanent teeth; permanent incisors are ahead of and permanent molars behind, their maxillary opponents.

There are sequence polymorphisms confirmed in family-line analysis and demonstrable in population comparisons, that show that M2 and P2 and M2 and C, and m2 and the deciduous canine (c) vary in their relative order of precedence.

There are sex differences in absolute and relative timing. The permanent teeth of females are developmentally ahead of the permanent teeth of males,[13] and sequence polymorphisms (P2-M2/M2-P2, M2-C/C-M2 and m2-c/c-m2) are not comparably frequent in the two sexes.[5,14]

Other aspects of dental variability, such as the greater relative variability of the XX, consistent with the diploid number of X chromosomes, side-to-side differences (bilateral asymmetry), and interclass and intraclass correlations will not be discussed here. With trisomy G (specifically 47 trisomy G) and trisomy X, all aspects of dental variability are increased (Table 4). The increase of variability is consistent with the hypothesis of a dosage effect.[15] Theoretically, mosaics such as the XO/XX and the XO/XY should include greater dimensional and bilateral variability, and many asymmetries in the dental development of abnormal karyotypes may stem from tissue mosaicism.

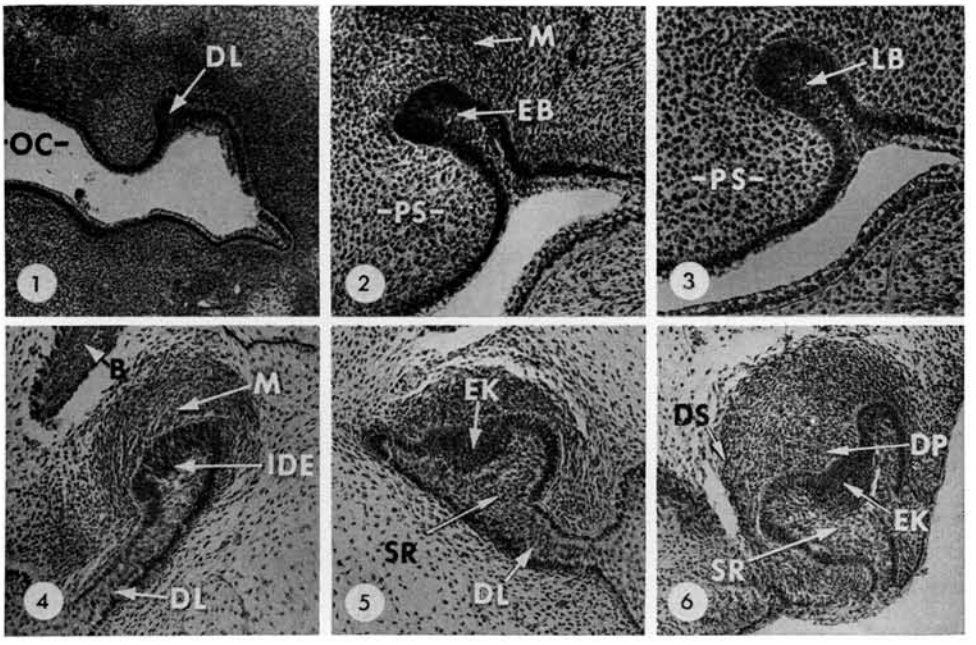
Prenatal Origins

In contrast to the extensive study of postnatal dental variability and its regular and lawful course, little work has been done on prenatal dental variability. Dental embryology has been mostly a statement of the order of stages, their dimensional range,

TABLE 4
INCREASED BILATERAL ASYMMETRY IN TRISOMY G

Tooth	19 Males		28 Females	
	Mongoloid	Normal*	Mongoloid	Normal*
Maxillary				
I1	0.440	0.275	0.310	0.191
I2	0.664	0.268	0.871	0.276
C	0.308	0.240	0.250	0.190
P1	0.426	0.190	0.480	0.178
P2	0.394	0.218	0.376	0.210
M1	0.447	0.260	0.673	0.261
M2	0.621	0.309	0.549	0.415
Mandibular				
I1	0.271	0.147	0.577	0.159
I2	0.303	0.151	0.225	0.152
C	0.251	0.174	0.233	0.195
P1	0.346	0.180	0.240	0.168
P2	0.562	0.200	0.456	0.190
M1	0.410	0.236	0.461	0.307
M2	0.966	0.340	0.731	0.352

* Root mean square crown-size asymmetry in 47 trisomy G and normal controls. For details see reference 15.



First six stages of prenatal tooth development used in present study. *1,* Dental lamina stage (*DL*): early appearance of primary epithelial vagination at margin of oral cavity (*OC*). *2,* Early bud stage (*EB*): proliferation and further ingrowth of oral epithelium to form early bud (*EB*) at margin of palatal shelf (*PS*). Note beginning condensation of mesenchyme (*M*) surrounding early bud. *3,* Late bud stage (*LB*): increased cellular proliferation of bud with continuing absence of flat-based area of inner dental epithelium. *4,* Early cap stage: base of inner dental epithelium (*IDE*) is flat, condensation of early mesenchyme (*M*) continues, and formation of jaw bone (*B*) is well advanced. *5,* Intermediate cap stage: enamel knot (*EK*) is formed superior to area of stellate reticulum (*SR*) with inner dental epithelium pronounced and entire dental organ attached to stalklike dental lamina (*DL*). *6,* Late cap stage: continued ingrowth of inner dental epithelium, adjacent enamel knot (*EK*), stellate reticulum (*SR*), and dental sac (*DS*) surrounding dental papilla (*DP*).

and the approximate developmental horizon. Within the limits set by available fetal specimens of known sex and approximate gestation age, some information has been obtained.

Some dental variability in postnatal life must have its beginnings in prenatal existence. Not all postnatal differences in sequence, mandibular precedence, and absolute and relative crown size, can possibly originate exclusively in the postnatal period. This is especially true of dental polymorphisms of genetic origin, particularly differences in sequence and timing related to agenesis, karyotypic abnormalities such as 47 trisomy G, and variations in number of sex chromosomes from XO through XXXXXY.

There is indirect evidence of prenatal variability in several situations: delayed onset of cusp calcification in prospectively studied children with later verified agenesis of one or more permanent teeth; systematic sex differences in the frequency of agenesis; incisor fusions in both deciduous and permanent teeth; systematic size reduction of the permanent teeth in 47 trisomy G and many of the cleft palate syndromes. It is difficult to imagine development retardations or delays in deciduous incisor formation (as seen in radiographs of premature neonates) as not having prenatal antecedents.

The exploration of prenatal variability demands its own design with the use of postnatal models as descriptive models for departure. There also is a need for sufficient numbers of fetal specimens at each developmental horizon, with careful division by sex. Although this is commonplace in post-

natal studies, it is less frequent in studies of those of tender developmental age. Data print-outs that can be reexamined for extreme or implausible values are essential. They reduce the chances of reporting a recording or transcription error as a novel prenatal dental polymorphism, or reporting sequence polymorphisms in grossly abnormal specimens.

Analyzing Prenatal Dental Variability

For exploration of prenatal dental development variability, specimens in the 15 to 58 mm crown-rump range within the first trimester horizon were used. Those with gross or microscopic abnormalities were excluded, although only a comprehensive tissue culture program would eliminate all possibility of chromosomal abnormality. An attempt was made to obtain a numerically equal series of first trimester males and females for initial comparison, but a forced program of sexual equality was avoided. The approach was conventional in that it recognized stages of dental development for each of 20 deciduous teeth (Illustration) but less conventional in that it used punchcards and computer programs to reduce and store more than 8,000 bits of developmental data from microscopic examinations.

Prenatal Variability

Prenatal dental variability was found in 15 to 58 mm specimens, in many instances remarkably parallel to models derived from postnatal studies, and in others, unexpectedly deviating from them.

In the first trimester horizon there was consistent male dental advancement, as opposed to the postnatal rule of female advancement. Considered tooth by tooth, and each jaw separately, male fetuses were dentally advanced over their female counterparts (Table 5). For constant crown-rump length, for all deciduous teeth, A through E, males are advanced. Individually studied females were longer for equivalent tooth stages, or for mean dental stage.[16] These results were not surprising

TABLE 5
MATCHED-PAIR CONFIRMATION OF MALE DENTAL ADVANCEMENT IN 27 PAIRS OF FIRST TRIMESTER EMBRYOS

Pair No.	Crown-Rump Length (mm)		Summed Tooth Stages for 20 Teeth		
	Male	Female	Male	Female	Difference M-F*
1	15	16	22	18	4
2	15	16	22	6	16
3	15	15	22	12	10
4	15	16	22	16	6
5	19	20	28	40	−12
6	19	20	28	38	−10
7	21	21	60	37	23
8	21	20	60	41	19
9	22	21	60	41	19
10	22	22	60	40	20
11	22	21	52	32	20
12	22	22	52	41	11
13	23	23	56	40	16
14	23	23	56	40	16
15	23	23	60	43	17
16	25	24	103	36	67
17	25	24	85	36	49
18	26	25	81	48	33
19	26	25	67	48	19
20	27	28	68	68	0
21	27	28	101	68	33
22	28	29	82	59	23
23	31	30	91	104	−13
24	31	30	91	90	1
25	33	32	98	108	−10
26	35	36	90	90	0
27	36	36	105	90	15
Mean	24.0	23.9	63.8	49.3	14.5

* Sex difference highly significant by both parametric and nonparametric test.

because one of the authors had demonstrated previously male advancement in palatal closure.[17] The lack of apparent sexual dimorphism in early postnatal tooth eruption was expected.[18] Males are decidedly ahead of females in early dentofacial development, and then at some point in prenatal time, the female advances. This trend possibly represents delayed influence of the second X chromosome at the tissue level, and suggests a dosage effect in later prenatal and postnatal dental development.

Prenatal Dental Polymorphisms

Since, postnatally, anterior mandibular teeth are systematically ahead of their maxillary opponents, it is not surprising that the same trend is found in the 15 to 58 mm crown-rump range. This is a dental development feature for which prenatal origins may be demonstrated. However, the regularity of the mandibular-maxillary precedence and the mesial to distal gradient is surprising.[19] With small number of subjects and technical limitations, it was not expected that the declining mandibular-maxillary gradient from i1 (A) through to m2 (E) would be so well established (Table 6).

Predictions of the extent of numerous prenatal dental sequence polymorphisms had not been possible.[20] Previous reports had suggested a simple mesial to distal, front to back, incisor to deciduous molar (A to E), order of prenatal dental development. True, in our postnatal studies in the USA and Central and South America, we had observed departures from the i1-i2-c-m1-m2 eruption order, as in the rhesus monkey and other species of *Macaca* as well. However, the fact that the limited number of fetal specimens showed a series of development orders (Table 7), gave proof to the hypothesis that prenatal dental development variability is the origin of many sequence polymorphisms seen clinically and radiographically in postnatal life.

TABLE 6
THE MESIAL TO DISTAL GRADIENT OF MANDIBULAR PRECEDENCE IN EARLY PRENATAL DENTAL DEVELOPMENT

Deciduous Tooth	No. of Sides	Mandibular Precedence No.	Mandibular Precedence %
i1	104	23	22
i2	104	27	26
c	104	17	16
m1	104	13	13
m2	104	9	9

Note: Compare with Table 2.

TABLE 7
TOOTH SEQUENCE POLYMORPHISMS IN EARLY PRENATAL DEVELOPMENT

Prenatal Formation Sequence	Males No. Sides	Males %	Females No. Sides	Females %
i1-i2-c-m1-m2	65	74	92	77
i1-i2-c-m2-m1	0	0	1	1
i1-i2-m1-c-m2	0	0	3	3
i1-i2-m1-m2-c	1	1	0	0
i1-i2-m2-c-m1	0	0	1	1
i1-c-i2-m1-m2	3	3	0	0
i1-c-m1-i2-m2	2	2	1	1
i1-m1-i2-c-m2	1	1	0	0
i2-i1-c-m1-m2	1	1	2	2
i2-c-i1-m1-m2	7	8	5	4
i2-c-m1-i1-m2	0	0	3	3
i2-m1-i1-c-m2	0	0	1	1
c-i1-i2-m1-m2	4	5	6	5
c-i1-m1-m2-i2	0	0	1	1
c-m1-i1-i2-m2	1	1	2	2
m1-i1-i2-c-m2	1	1	0	0
m1-m2-i1-i2-c	1	1	1	1
m2-i1-i2-c-m1	1	1	1	1

Field Effects

A number of field effects can be demonstrated in postnatal dental development (calcification, root elongation, movement and eruption, and mesiodistal crown size). The mandible and the maxilla are partially independent dental fields; teeth in opposition constitute a second set of developmental and dimensional fields. Adjacent teeth, even of different morphological classes and sizes, are local fields, as shown by correlations. Distance is clearly a field, as shown by higher communalities for adjacent teeth, and systematically lower communalities for those more removed.

Previously discovered developmental and dimensional fields of postnatal dental development and permanent tooth crown size seem to be foreshadowed by the behavior of deciduous teeth in the first trimester of gestation. Systematically, isomeres show slightly higher developmental correlation than do mandibular and maxillary opponents (Table 8). Generally, isomeres show

TABLE 8
COMPARISON OF INTRAJAW AND INTERJAW PRENATAL DEVELOPMENTAL CORRELATIONS

	Males		Females	
Tooth	Isomere	Opponent	Isomere	Opponent
i1	0.996	0.960	0.985	0.975
i2	0.997	0.888	0.978	0.925
c	0.981	0.950	0.987	0.970
m1	0.970	0.928	0.991	0.972
m2	0.917	0.897	0.986	0.978

Note: Isomere correlations systematically exceed opponent correlations by 0.034.

the highest developmental correlations, opponents show the next highest developmental correlations, and adjacent teeth show lesser correlations. In fact, distance may be regarded as a field (or field as a distance phenomenon) in prenatal dental development, because greater anatomic distance between teeth lowers the developmental correlations among them.[21] As shown in Table 9, adjacent teeth exhibit the highest mutual intercorrelations, and in both jaws and both sexes, these decline with the number of intervening teeth. There is some evidence of the second X chromosome at this early development horizon, with intra-girl communalities higher than intra-boy communalities for every tooth, and for the development correlation network as a whole.

A Look to the Future

Pilot findings are valuable to the extent that postnatal dental variability has obvious prenatal parallels. The mandibular-maxillary precedence gradient is not a postnatal phenomenon alone, and sequence polymorphism is as prevalent in the 2 to 5 cm fetus as in the adolescent boy or girl of 160 to 180 cm. Postnatal dental variability appears to have demonstrable prenatal origins, at least in part.

TABLE 9
DECLINING DEVELOPMENTAL COMMUNALITY WITH INCREASING ANATOMICAL DISTANCE

No. Intervening Teeth	No. of Correlations	Mean r*		
		Males	Females	Combined
Maxillary				
0	4	0.903	0.952	0.932
1	3	0.834	0.919	0.883
2	2	0.735	0.901	0.837
3	1	0.740	0.883	0.824
Mandibular				
0	4	0.917	0.943	0.931
1	3	0.831	0.887	0.862
2	2	0.774	0.831	0.804
3	1	0.701	0.862	0.795

Note: Consistently higher female developmental communalities average 0.085.
* Mean value of r from the z transform of r

The 15 to 58 mm crown-rump range is a small part of the total 3,200 gm full-term neonate. The authors have not explored the prenatal origins of permanent tooth sequence variability, or used 16- to 20-week therapeutic abortions. There have not been explorations of prenatal agenesis and its relationship to early developmental timing, odontogenesis in fetuses with verified C, D, and G group chromosomal abnormalities, or prenatal hypodontia.

It cannot yet be proved that a given prenatal dental polymorphism would have comparable postnatal parallels. However logical this assumption seems when prenatal and postnatal data are in close agreement, the comparison of dead fetuses with living children does not have the advantage of longitudinal study. The solution seems to be study of the older siblings of fetuses, using pilot models previously applied to living children. In the children it was shown that siblings were similar in particular permanent tooth sequence polymorphisms. The same model may apply both prospectively and retrospectively in comparing fetuses with their living brothers and sisters.

Perhaps nutrition affects the order of prenatal dental development, and yields an atypical order in placental insufficiency, maternal protein-calorie malnutrition, or both. Teeth studied postnatally appear to be refractory to many minor nutritional differences, but may be vulnerable to protein quality or placental sufficiency at critical periods in their early development.

These human studies projected forward into postnatal timing and backward into the prenatal period, offer human models and human problems for experimental animal research. The prospects are perhaps less for rodents lacking deciduous parallels, and more for the smaller primates we can rear and study. Even for rodents, conveniently born at fetal levels (by human standards) and subject to deliberate exposure to teratogenic influences, our human data provide useful experimental models.

Recognition of the fact that some models of postnatal dental variability (including sequence polymorphisms) have rather close

prenatal parallels, creates three additional tasks. The first objective is to design experimental approaches that will show whether a given prenatal development sequence will continue into postnatal dental development. The second is to design experimental approaches that will show whether, and to what extent, sequence can be altered and variability increased by interfering with the course of prenatal dental development. The third, a combination of the first and second, is to determine whether, and to what extent, early disturbances of chronogenesis may manifest themselves: as atypical postnatal orders, increased development variability in deciduous and permanent tooth eruption, and agenesis, with associated development delay, crown-size reduction, and reduction of morphologic complexity.

After these investigations, it will be possible to begin to understand the extent to which postnatal dental variability has prenatal origins, and what mechanisms are involved.

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References

1. SCHULTZ, A.H.: Age Changes and Variability in Gibbons, *Amer J Phys Anthrop* **2:**1-129, 1944.
2. GARN, S.M., and ROHMANN, C.G.: Variability in the Order of Ossification of the Bony Centers of the Hand and Wrist, *Amer J Phys Anthrop* **18:**219-230, 1960.
3. GARN, S.M., and SHAMIR, Z.: *Methods for Research in Human Growth,* Springfield, Ill: Charles C Thomas, 1958.
4. GARN, S.M.; ROHMANN, C.G.; and SILVERMAN, F.N.: Radiographic Standards for Postnatal Ossification and Tooth Calcification, *Med Radiog Photog* **43:**45-66, 1967.
5. GARN, S.M., and LEWIS, A.B.: Phylogenetic and Intra-Specific Variations in Tooth Sequence Polymorphism, in BROTHWELL, D.R. (ed): *Dental Anthropology,* New York: Pergamon Press, 1963.
6. MEREDITH, H.V.: Order and Age of Eruption for the Deciduous Dentition, *J Dent Res* **25:**43-66, 1946.
7. ISRAEL, H.; BENITEZ, R.F.; and GARN, S.M.: Eruption Sequence Polymorphisms of Maxillary and Mandibular Opponents in the Dentition of Ecuadorian Mestizos, *Arch Oral Biol* **13:**239-241, 1968.
8. ISRAEL, H.; DAHLBERG, A.A.; GARN, S.M.; and KEREWSKY, R.S.: Relative Eruption Precedence of Mandibular and Maxillary Teeth in Two Populations, *J Dent Res* **46:** 456, 1967.
9. KOSKI, K., and GARN, S.M.: Tooth Eruption Sequence in Fossil and Modern Man, *Amer J Phys Anthrop* **15:**469-488, 1957.
10. GARN, S.M.; KOSKI, K.; and LEWIS, A.B.: Problems in Determining the Tooth Eruption Sequence in Fossil and Modern Man, *Amer J Phys Anthrop* **15:**313-331, 1957.
11. GARN, S.M., and LEWIS, A.B.: Relationship Between the Sequence of Calcification and the Sequence of Eruption of the Mandibular Molar and Premolar Teeth, *J Dent Res* **36:**992-995, 1957.
12. GARN, S.M. and LEWIS, A.B.: Unpublished results.
13. GARN, S.M.; LEWIS, A.B.; KOSKI, K.; and POLACHECK, D.L.: The Sex Difference in Tooth Calcification, *J Dent Res* **38:**561-567, 1958.
14. GARN, S.M.; LEWIS, A.B.; and VICINUS, J.H.: Third Molar Polymorphism and Its Significance to Dental Genetics, *J Dent Res* **42:**1344-1363, 1963.
15. GARN, S.M.; COHEN, M.M.; and GECIAUSKAS, M.A.: Increased Crown-Size Asymmetry in Trisomy G, *J Dent Res* **49:**465, 1970.
16. BURDI, A.R.; GARN, S.M.; and MILLER, R.L.: Developmental Advancement of the Male Dentition in the First Trimester, *J Dent Res* **49:**889, 1970.
17. BURDI, A.R., and SILVEY, R.G.: Sexual Differences in Closure of the Human Palate, abstracted, *IADR Program and Abstracts of Papers,* No. 155, 1969.
18. MEREDITH, H.V.: A Synopsis of Dental Development in Childhood, *J School Health* **36:**503-507, 1966.
19. BURDI, A.R.; GARN, S.M.; and MILLER, R.L.: Mesiodistal Gradient of Mandibular Precedence in the Developing Dentition, *J Dent Res* **49:**644, 1970.
20. BURDI, A.R.; GARN, S.M.; MILLER, R.L.; and NAGY, J.M.: Sequence Polymorphism in Prenatal Dental Development, *J Dent Res* **49:**1554, 1970.
21. GARN, S.M.; BURDI, A.R.; and NAGY, J.M.: A Distance Gradient in Prenatal Dental Development, *J Dent Res* **50:**785, 1971.