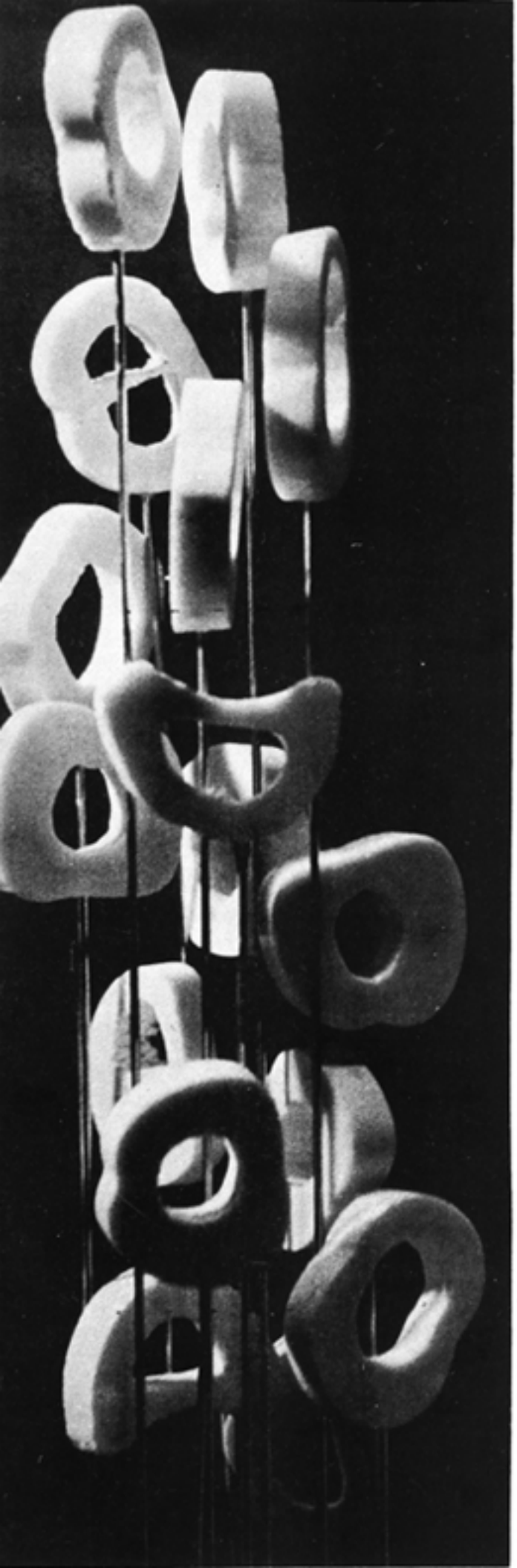

The EARLIER GAIN and the LATER LOSS of CORTICAL BONE

In Nutritional Perspective

STANLEY M. GARN, Ph.D.

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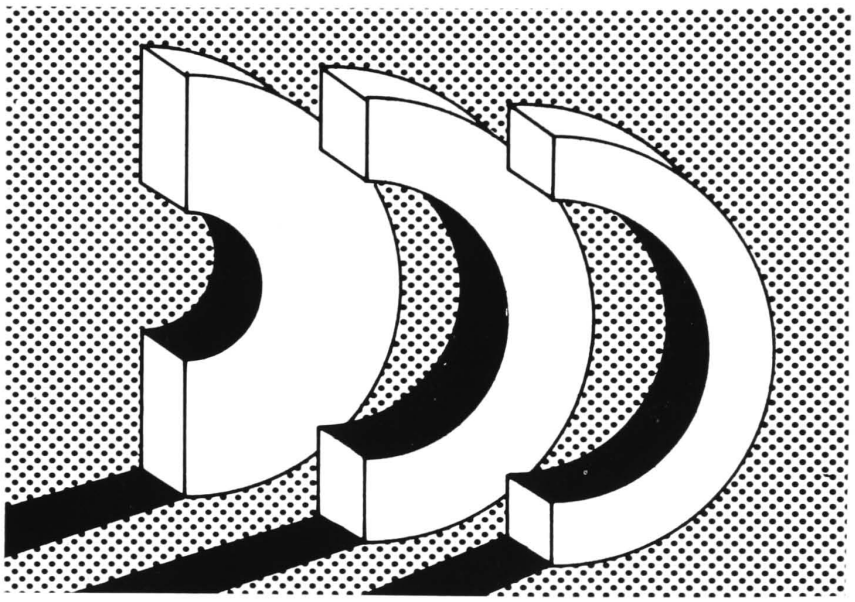

Adult female bone loss in isometric projection. Left, 2nd metacarpal midshaft section at thirty years. Center, the metacarpal midshaft section at eighty years showing marked endosteal resorption. Right, an example of extreme bone loss in a seventy-seven-year-old subject. Drawings to scale, as shown.

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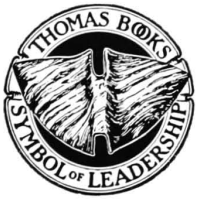

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This report of research is dedicated to the memory of two men: **Fuller Albright,** who turned my attention from the outside of bones to their insides, and **Richard Follis, Jr.,** who suggested that I investigate changes at both bone surfaces simultaneously.

PREFACE

BONE LOSS IS UNQUESTIONABLY the most common disability of later life. It carries with it the increasing probabilty of bone fracture. As we now know, bone loss respects neither sex nor race nor geographical area, and it is partial neither to cortical bone and the long or tubular bones nor to cancellous bone and the round bones and vertebrae.

Ten years ago we began a long-term comprehensive study to determine not only the course of adult bone loss but also the earlier course of bone gain. We selected for analysis tubular bones (and more specifically cortical bone) which we had earlier shown to be susceptible to usefully precise *in vivo* measurement. We wanted to work with the living primarily, and we wanted to cover the entire span of life—from birth through the ninth decade.

We began our studies in the United States, using both cross-sectional and longitudinal (serial) radiographs of clinically normal participants in voluntary programs. Soon we were involved with populations from Central America, then South America, and with skeletalized material from an anatomical collection, and—in the course of time—with survey radiographs from twelve states across the nation.

Concerned with the nutritional hypothesis, we made maximum use of replicate seven-day dietary records, supplemented by in depth recall interviews, with particular attention to protein and calcium—including extradietary sources of calcium. As our approaches showed clinical promise, we began to study endocrinopathies, growth failures, and subjects with malnutrition and malabsorption states. We added chromosomal abnormalities, including an extensive series of Down's syndrome, verified by cytogenetic studies.

Now, over 25,000 radiographs later, we have a comprehensive description of how cortical bone is gained and lost at both bone surfaces. We have found that the subperiosteal surface has at least three distinct phases of gain, and the endosteal surface has an alternating series of loss, gain and loss. We have shown continuing adult gain at the subperiosteal surface, extended gain (from adolescence through the fourth decade) at the endosteal surface, and older adult loss at the endosteal surface in both sexes. We have found family line and population differences in the amount of bone change at each surface, but without exception, international agreement on adult bone loss in both men and women.

We discovered that we were not alone in our objectives, only in our design for lifelong descriptions. We were paced by Dr. Christopher Nordin in

Leeds, and Dr. Richmond Smith, Jr., in Detroit. We learned from the Meemas in Toronto and from Dr. James Arnold in Kansas City. Our work has been followed and extended in Japan, Switzerland, and Holland.

We cannot show an effect of dietary fluoride at 1 ppm on bone loss, nor yet of estrogens, or such adult activities as gymnastics or skiing, but we can raise doubts as to the value of a high calcium intake, a high phosphorus-calcium ratio, or a restricted acid-residue diet. We can show a phase of transient bone loss in all infants; we do document the effects of female castration, the benefits of larger body size (that leads to less bone loss), and the generalization that bone is the best defense against later bone loss.

Not all of our data are described here, nor all of our publications reviewed, nor in detail all those of the many others who, like us, seek the answer to adult bone loss in the knowledge of how cortical bone is gained and how it is lost at each of its two active surfaces. Not all of our comparisons of Negro, Mexican-American, Navajo, or Chinese and Japanese bone changes are detailed, nor yet all we have learned in the Holt-Oram syndrome, in cerebral giants, or in the mandible or the skull.

This book is primarily concerned with the early gain and later loss of cortical bone, viewing its two active surfaces separately.

S.M.G.

ACKNOWLEDGMENTS

Research Support

THE RESEARCH DESCRIBED in this book was supported, in the order given, by a grant from the Nutrition Study Section, Division of Research Grants, United States Public Health Service, Grant AM-08255 from the National Institutes of Health, Contract PH-43-65-1006 (Central American Nutrition Survey) and Contract HSM-110-69-22 (National Nutrition Survey) with the Nutrition Program, Regional Medical Program Service, Health Services and Mental Health Administration; and Grant AM 13378, constituting the terminal year of AM-03816; as well as by earlier support from the Fels Fund of Philadelphia, and including other work accomplished on DE-01294 and Conference Grant A-3890.

Associates

Christobel G. Rohmann, Eleanor M. Pao, and Betty L. Wagner were associated with the studies in southwestern Ohio and in the Central American data analysis. Dr. A. Roberto Frisancho has been directly responsible for the data analysis and field contacts in the National Nutrition Survey in con nection with HSM-110-69-22. Dr. Cyrus W. Stimson is responsible for the studies on trisomy G at the Plymouth State Home, Northville, Michigan; and Dr. John C. Gall, for studies on his Holt-Oram kindred. Dr. John P. Dorst has cooperated in these studies, providing information on bone changes in congenital heart disease. Much valuable assistance was provided by Dr. Frederic N. Silverman of the Children's Hospital, Cincinnati, and since 1968, by Dr. Andrew K. Poznanski, Department of Radiology, University of Michigan, School of Medicine, and by Dr. Arthur B. French.

Other Colleagues

We are indebted to Dr. Christopher Nordin, at Leeds, for access to his published and unpublished data from Scotland, Finland, and other places; to Dr. Anne P. Forbes for radiographs of older XO subjects; to Dr. Richmond Smith, Jr., for much clinical material; and to Dr. Robert Blizzard, Jr., for endocrinopathies and growth abnormalities.

Dr. Richmond Smith, Jr., and Drs. Eric and Sylvia Meema, as well as Dr. Christopher Nordin, have lent their knowledge and thinking. Dr. Mildred Trotter, Washington University School of Medicine, made available hundreds of skeletalized individuals for radiography. Drs. Donald Whedon,

Mark Hegsted, and James S. Arnold made their thinking and findings available, as have Dr. Boy Frame, of Detroit, and Dr. Meinhard Robinow, of the Yellow Springs Clinic. Radiographs and data are individually indicated.

The Book Itself

The manuscript, tables, legends, and bibliography were prepared by Shirley M. Garrett; many of the tables were completed by Jerrold M. Nagy; later computer analysis was directed by Mr. Richard L. Miller at the computer facility of the Center for Human Growth and Development at the University of Michigan.

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Chapter 1

THE SURFACE-SPECIFIC NATURE OF CORTICAL CHANGE

A RADIOGRAPH OF A TUBULAR BONE in a sufficiently standardized projection reveals the subperiosteal surfaces and the medullary cavity which may then be measured as the total subperiosteal diameter (T) and the med ullary cavity width (M). Such measurements are highly replicable on a single radiograph ($r \simeq 0.99$), on a set of radiographs ($r \simeq 0.98$), and over long periods of time ($r > 0.95$). By subtraction of M from T, cortical thickness (C) is then calculated, and the final replicability of C approximates that of T and M.

If a cylinder of material with a linear absorption coefficient similar to bone—such as aluminum—is then substituted for a tubular bone; T, M, and C measured on the radiograph not only agree with the dimensions of the cylinder (after correction for radiographic enlargement) but calculations of *areas* of T, M, and C from the radiographs agree with direct measurements and calculations of areas from the cylinder itself.

T corresponds to the total subperiosteal diameter, and increases in T in either longitudinal (individual) or cross sectional (group) radiographs measure linear subperiosteal apposition.

M corresponds to the medullary cavity width and changes in M meter endosteal resorption or endosteal apposition.

C, that is T-M, corresponds to the summed medial and lateral wall thicknesses. Changes in C may be due to increases in T, or to increases or de creases in M; that is, endosteal resorption or endosteal apposition, in that order.

If C is taken as a percentage of T, either as a linear measurement or as a set of areas, then a new set of data becomes disclosed. In essence, the ratio of C to T is a simple measure of bone density. Further, expressed as an area (in mm^2), if the area of C is 50 percent of the area of T, then the bone section in question has exactly half the physical density of a solid cylinder of tissue cortex of the same diameter T.

Our knowledge of the growth and the loss of cortical bone is to the larg est extent in the changing proportions of T and M, and therefore C. T in creases throughout life, as will be shown. M at first increases, and then

---

Author's Note: See Barnett and Nordin (1961); Bonnaid (1968a, 1968b); Garn, Nolan and Rohmann (1964); Garn, Feutz, Colbert and Wagner (1966); and the Appendix for measurements of T, M and C, and for T-M-C intercorrelations.

from puberty to the fourth decade, it decreases. C gains in thickness and area through the fourth decade and then reverses. Bone loss in adult life is in essence a decrease in C, due to an age associated nonlinear increase in M, but compensated in part by a slow linear increase in T.

Our primary interest here is in cortical bone, that is C. But C is the difference between T and M. At times growth of T outpaces that of M and so C increases. At times M outpaces T in expansion and so C decreases. In the later years, subperiosteal apposition increases C at one bone surface, while endosteal resorption simultaneously reduces C at the other surface. Thus, while we are concerned with cortical bone and its gain and loss, we are necessarily most concerned with the surface-specific changes, both subperiosteal, as reflected by T; and endosteal, as metered by M (Fig. 1).

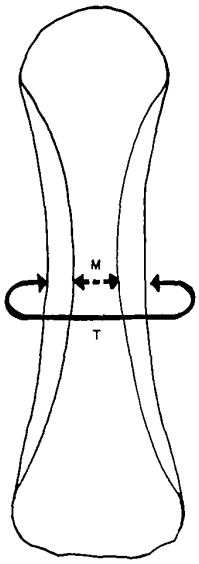

FIGURE 1. Diagrammatic representation of tubular bone, showing midshaft measurements of T and M. Using 0.05 mm readout Helios caliper and expressing cortex (C) as T-M, measurement accuracy is then maximized. For other techniques see Barnett and Nordin, 1961; Bonnard 1968a, 1968b; Kimura and Hattori, 1968; and Adams, Davies, and Sweetnam, 1969, as well as Virtama and Helelä, 1969.

CORRESPONDENCE WITH BONE DENSITOMETRY

Both film-type bone radiogrammetric densitometry and caliper radiogrammetry make use of the same bone shadow. Both the trace path in bone densitometry and the measurement site in caliper radiogrammetry are selected on the basis of visual examination, avoiding morphologic variations, cortical defects, fracture sites, et cetera.

Data for T, M, and therefore, C are inherent in the conventional densi-

tometric trace from the Joyce, Loebl® or other microdensitometer. Taking the M shaped or hat-shaped trace of the bone shadow of a tubular bone, the total width of T corresponds to the width of the trace above the background, M is the central area of increased density, and the two cortical walls are the sides of the M in the trace. In fact, T and M can be measured directly on the densitometric trace, with the ×10 enlargement of the ratio arm increasing apparent measuring accuracy. Comparison of measurements made on the trace and those measured directly on the film show the natural tendency in direct film shadow measurement to overmeasure T, to undermeasure M, and therefore to overestimate C (Fig. 2).

In both microdensitometry and caliper radiogrammetric mensuration, exposure quality is important and for reasons that a reference wedge cannot correct. If film density within the bone shadow region falls below a useful level, the width of the marrow cavity cannot be distinguished from the cortical walls at the endosteal surface. At levels much below an optical density of 1.0, this is true for radiographic microdensitometry (assuming a fog level of 0.3 0.6). Under these circumstances, total bone density is underestimated. In caliper measurement, at low levels of density, the width of the marrow cavity is underestimated, and cortical thickness is overestimated. Loss of film quality below an optimum level of density produces errors by either approach, but in opposite directions insofar as total bone mass is concerned.

While for a tubular bone following the cylindrical bone model, film-type microdensitometry and caliper radiogrammetry can yield corresponding results, attention is ordinarily directed to disparate problems. In microdensitometry, attention is given to the mass of bone and mass relative to area, without attention to anatomical detail. In radiogrammetric measurements of T, M, and C, attention is given to the subperiosteal and endosteal surfaces, and therefore to T and M but without attention to the bone quality (Q) represented by T-M.

If nonscreen film is used, either of the conventional or rapid processing type (RP), then either microdensitometry, caliper measurement or both, can be accomplished on the same bone shadow. With screen-type, film, only micrometry is now practicable, since the film density: bone-absorption relationships are altered by the screen characteristics and the sensitivity of the film to actinic radiation. Thus, for the vast majority of clinical radiographs and for those taken in nutritional surveys, the micrometer caliper measurement of T and M is practicable and radiogrammetric microdensitometry is not.

CHOICE OF BONE AND SITE

Radiogrammetric studies of changes in cortical thickness; as derived (by subtraction) from the total subperiosteal diameter (T) and the medullary

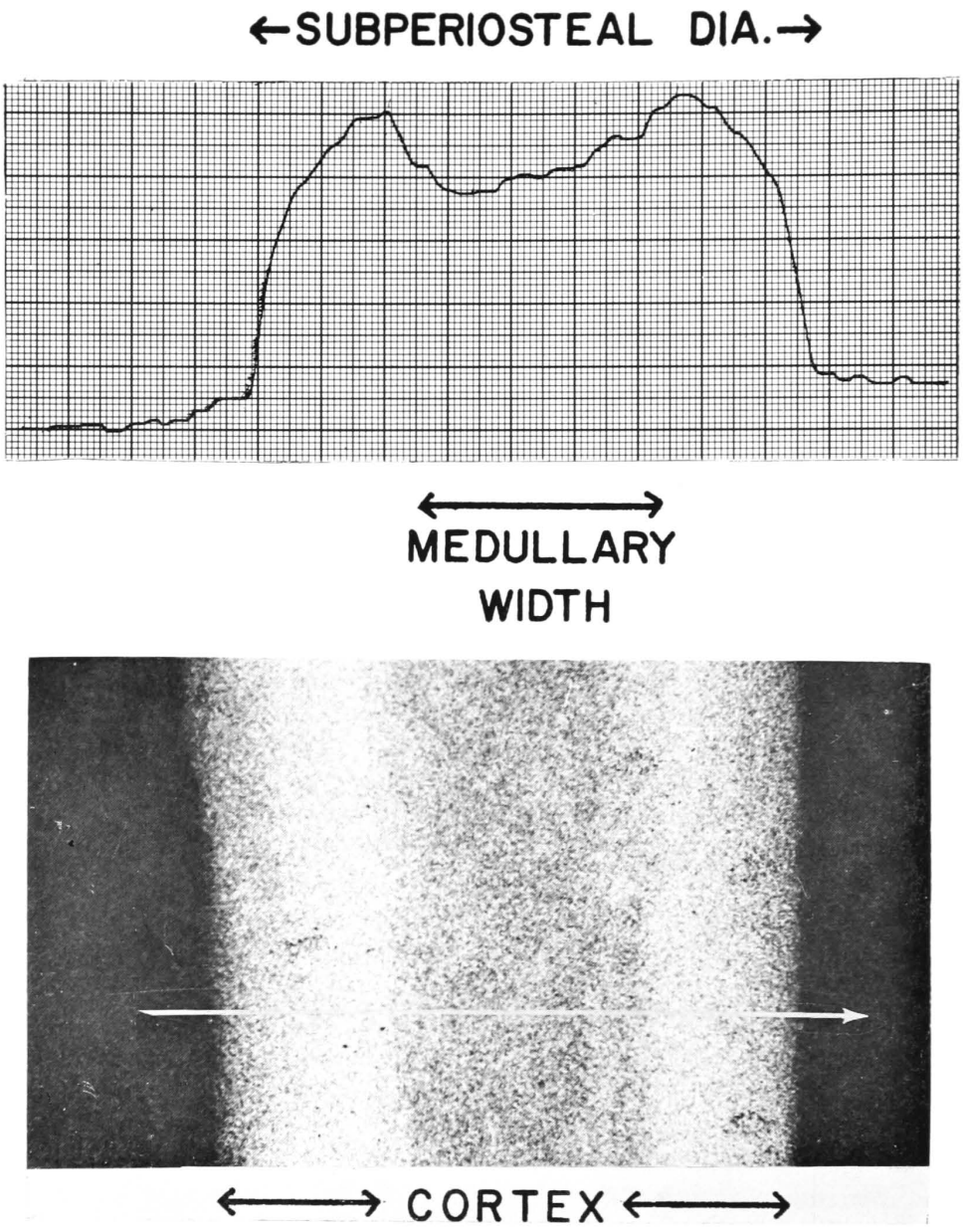

FIGURE 2. Comparison of microdensitometric trace (above) and radiographic image (below) of 2nd metacarpal traced at midshaft with the Joyce, Loebl recording microdensitometer. Measurements T and M can be made on the microdensitometric trace (Cf. Garn, Feutz, Colbert, and Wagner, 1966, Figures 2 and 3 and Table II).

cavity width (M), are most applicable where the tubular bone model holds. If the bone section approaches circularity and the medullary cavity is reasonably circular and concentric, then calculations of the subperiosteal area (or bone envelope), the medullary area, and the cortical area can be made from T and M. Furthermore, the percentage of cortical bone in the cross section can easily be calculated, using a programmable desk calculator, or from punch-cards with a larger computer. Alternatively, emphasis can be given to subperiosteal apposition or to endosteal apposition resorption or both.

The necessary measurements of T and M can be made on radiographs of many tubular bones—the femur, the tibia, the radius and ulna, and the metacarpals and metatarsals. We have made measurements on all of these bones *in vivo* over a wide age range from infancy through old age, on hundreds of fetal bones (provided by Dr. Mildred Trotter), and on skeletalized adult material, both fresh and archaeological. Applications to archaeological material and to skeletal collections are obvious.

But not all bones meet the cylindrical model well. At midshaft, the adult femur includes the linea aspera, so that calculations of subperiosteal area and cortical area inadequately describe the bone in cross section if only the anteroposterior projection is employed. The tibia, which we have studied extensively, has a complex triangular section at midshaft for which empirical formulae are best used if more than T, M and C (cortex) are desired. The radius and ulna present similar problems. For these reasons, the 2nd metacarpal holds obvious advantages. Though T and M can be measured on most tubular bones and for some studies the femur must be employed, most of the data in this book relate to the 2nd metacarpal.

Further, the cylindrical model assumes that all or most of the tissue bone is contained within the boundaries set by T and M. This is true for most longer tubular bones at midshaft and has been verified experimentally for the 2nd metacarpal, using dried bones before and after sectioning. It is ob viously not true for the phalanges, and it is obviously not true for all tubular bones. The distal radius and ulna is a complex of cancellous and cortical bone, as is true for the femur, the tibia, and others, particularly in age and in the aging female.

Morphological variations also provide limitations. The 3rd, 4th, and 5th metacarpals may be reduced in length in many chromosomal and genetic abnormalities. These variations limit the use of the 3rd-5th metacarpals just as brachymesophalangia and clinodactyly preclude the use of the middle segment of the 5th metacarpal in both film and nonfilm densitometry. The distal ulna is highly subject to variation, and to some extent the distal radius is too.

For the 2nd metacarpal and the femur, midshaft measurements of T and

M are easily made with maximum replicability, an inordinate advantage when thousands of films are to be analyzed. The measurement of T and M at minimum shaft width in the 2nd metacarpal yields results closely similar to those at midshaft, as Richmond Smith, Jr., has extensively shown; his data and ours neatly agree. For the tibia, however, the measurement of both T and M at minimum shaft width is more reproducible than at a mid-shaft location and reveals age changes more effectively, and with higher correlations with other sites in the same subjects.

RADIOGRAMMETRIC REPLICABILITY AND RELIABILITY

As with all measuring techniques, radiogrammetric replicability and reliability depend upon the size of the measurement, readout capability, and the training or "practice" effect. The smaller the measurement, the greater the measuring error relative to that measurement (percent error); the smaller the readout capability of the instrument used, the smaller the measuring error, the less training and practice the greater the (random) measuring error and in many cases, the systematic measuring error. In radiogrammetric measurements, variations in tube-to-film distance and in positioning also contribute errors, in some cases substantial. Morphologic variation is also a source of measuring imperfection. (Fig. 3).

To a large extent, radiogrammetric replicability and reliability can be improved by careful attention to standardizing the tube-to film distance. In many of our studies this source of error has been eliminated by a fixed tube head. Positioning errors can be minimized by avoiding medial and lateral rotation in the case of the femur or tibia, or by selection of metacarpal measurements where rotation is effectively eliminated when the hand is positioned flat on the plate and the radius and ulna are maintained axial to the hand. Morphological variations can be minimized as a source of error by selection of least variable tubular bones and measurement sites (see above.)

The use of a pinpoint micrometer caliper with 0.05 mm readout capability, such as the Helios dial reading caliper,* with careful attention to calibration, yields an R.M.S. measuring error of 0.10 to 0.15 mm. This is effectively ±10 percent of the smallest medullary cavity widths commonly encountered in the second metacarpal and ±5 to ±2 percent of the more usual range in this bone (3 5 mm). It amounts to as little as ±1 percent of the total subperiosteal diameter (T) in adults. The magnitude of the readout error dictates the measurements T (total) and M (medullary) rather

*This dial reading caliper can be converted into a transducer caliper and with the circuitry provided by the GMDRS or other circuitry can yield both card punch and typewriter output (cf. Garn, S.M.; Helmrich, R.H., and Lewis, A.B.: Transducer caliper with readout capability for odontometry. *Journal of Dental Research, 46*:306, 1967).

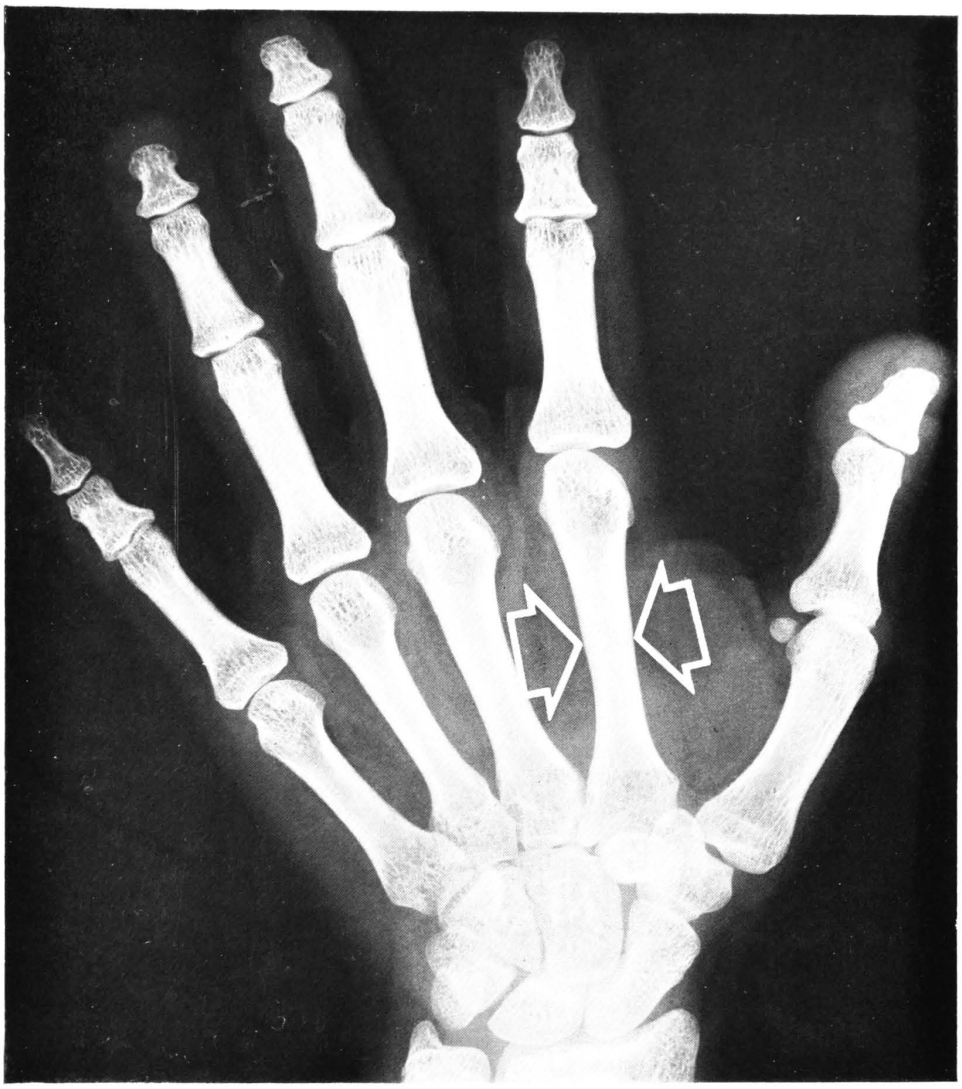

FIGURE 3. Location of midshaft measurement on 2nd metacarpal of subject with brachymesophalangia 2 and 5, reductions of metacarpals 4 and 5, and reduction of distal segments of 1, 3, and 4. As shown, the 2nd metacarpal is least subject to morphological variation (see also Fig. 30).

than the separate measurement of the medial and lateral cortical walls where T is below 15 mm.

With the second metacarpal measurements at midshaft, *intra*observer correlations rarely fall below 0.90 at the beginning of the practice period and rise to 0.98-0.99 with practice. At this point, *inter*observer correlations are of the same order of magnitude. Under these circumstances, changes of

the order of +0.3 mm may be meaningful in individuals and much smaller average changes in groups.

Short-term serial reliability of the measurements averages close to 0.98 for sets of radiographs taken six months apart and corresponds to a time-to-time measuring error of circa 5 percent. This appears to be the practical limit of the technique for radiographs of the second metacarpal, in the hands of experienced measurers (Table I).

TABLE I
SHORT TERM AND LONG TERM INTRAOBSERVER AND INTEROBSERVER RELIABILITY OF T AND C

Measurement	*Observer*	*No.*	*Correlation*
	INTRAOBSERVER RELIABILITY		
Cortex	P.N., Jr.	20	0.98
Cortex	E.D.A.	20	0.95
	INTEROBSERVER RELIABILITY		
Cortex	P.N. E.D.A.	20	>0.99
Cortex	P.N. E.D.A.	20	>0.99
	INTEROBSERVER RELIABILITY		
Cortex	E.H.—M.L.	86	>0.98
Cortex	E.H.—M.L.	25	0.98
	LONG-TERM RELIABILITY*		
Total sub periosteal (f)	P.N.	25	0.97
Total sub periosteal (m)	P.N.	24	0.97

*Radiographs taken an average of fifteen years apart (cf. Adams, Davies, and Sweetnam, 1969 whose measurements were made by reading off distances on pointed divider onto ruler and reducing all measurements by 0.5 mm. Also, Garn, Feutz, Colbert, and Wagner, 1966).

CHOICE OF MEASUREMENTS TO REPORT

The raw measurements in these studies are effectively two, the total subperiosteal diameter (T) and the medullary cavity width (M). The simplest computational measurement is then cortex (C) which is simply T-M. Early in our studies we primarily reported C, that is T M, which is in many ways satisfactory. C (cortex) measures the decrease in cortical thickness in subjects over forty, and C compares cortical thickness in Japanese and Negroes, in men and women, and in childhood and age.

But C is not enough. As our studies progressed we discovered that T (total subperiosteal diameter) increased with increasing or advancing age; T increased even as C decreased. There was merit in reporting T and then calculating how much the loss of C was actually compensated by increase in T. For some purposes, as in the analysis of continuing subperiosteal apposition throughout life, the total measurement T is the measurement to report.

We soon gave separate attention to M as well. Medullary cavity width increases to the early teens, it decreases thereafter, and then, by the mid-thirties, it begins to increase again. In adulthood and in childhood alike, the

major action may be in medullary cavity width. Hence changes in M may be essential to report, apart from T and C.

Size is a factor, and the amount of bone relative to size is a size-related factor. Dividing cortex (T M) by the total (T), Nordin introduced an index or "score" that describes how much of the total width is cortex. This score or index of Nordin's $\frac{T\text{-}M}{T}$ is a valuable description even though it is essentially two-dimensional.

T (total) is further described (for tubular bones) as the total subperiosteal area $\pi\left(\frac{T}{2}\right)^2$, which simplifies down to 0.785 T^2. This describes the total envelope of a tubular bone in cross section, which envelope increases with advancing age.

M (medullary width) is also best described as an area which, like T, is simplified to 0.785 M^2. Changes in the smaller diameter of M must be compared to changes in the larger diameter of T. A small increase in T (the larger diameter) may negate a larger linear increase in M (the smaller diameter) in terms of areas of bone.

In cross section the area of C is, of course, the difference between the total subperiosteal area (0.785 T^2) and that of the medullary area (0.785 M^2) simplified down to 0.785 (T^2-M^2). This simplification allows a desk computer program for cortical area, which may increase in the early stages of endosteal resorption as T outpaces M in areal changes but not in the later stages of endosteal bone loss when the area of M increases far more rapidly than the area of T (Fig. 4).

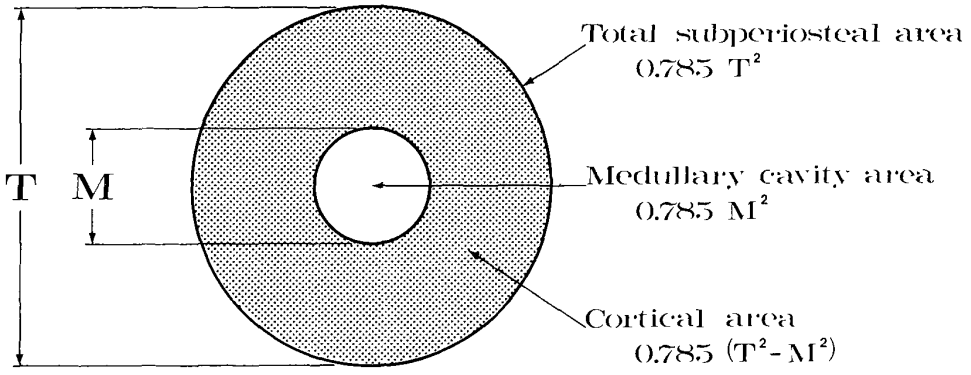

FIGURE 4. Representations of total subperiosteal area, medullary area, and cortical area as derived from T and M. For many purposes, expression of T, M, and C as areas or as unit volumes best describes dimensional changes of tubular bones.

So T merits reporting for its purpose, M for its purpose, and C for its own purpose. C as a percentage of T represents the amount of cortical bone

in the flat cross section, and the other measure given, subperiosteal *area,* cortical *area* and percent cortical *area* are cross sectional area measurements. They are, then:

1. Total subperiosteal diameter	T
2. Medullary cavity diameter	M
3. Cortical thickness (T M)	C
4. Percent cortex $\frac{T-M}{T}$ or	$\frac{C}{T} \times 100$
5. Total subperiosteal area	$0.785\ T^2$*
6. Medullary area	$0.785\ M^2$*
7. Cortical area	$0.785\ (T^2-M^2)$ *
8. Percent cortical area	$\frac{0.785\ (T^2-M^2)}{0.785\ T^2}$ * $.100$
which simplifies to	$100\left(\frac{T^2-M^2}{T^2}\right)$*

For the tibia, with a triangular rather than a circular cross section, and assuming equilateral proportions:

9. Total subperiosteal area	$\frac{\sqrt{3}}{4}\ T^2$
10. Medullary area	$\frac{\sqrt{3}}{4}\ M^2$
11. Cortical area	$\frac{\sqrt{3}}{4}\ (T^2\ M^2)$
and	
12. Percent cortical area	$100.\ \frac{\frac{\sqrt{3}}{4}\ T^2 - \frac{\sqrt{3}}{4}\ M^2}{}$
which simplifies to	$\frac{\sqrt{3}}{4} T^2$
as in 8 above.	$100\left(\frac{T^2-M^2}{T^2}\right)$

*Simplified formulae. Compare with Exton Smith *et al.* (1969), p. 1153. Their formula is identical with 7 above.

Where the tibia is not an equilateral triangle, the following empirical formulae have been developed, using serial sawn tibial sections at minimum medullary width (Garn and Wagner, 1969, p. 145):

13. Cortical area $T^2\text{-}M^2$ where $0.37 > \frac{T\ M}{T} > 0.33$

and/or

Cortical area $0.75(T^2\text{-}M^2)$ where $0.33 > \frac{T\text{-}M}{M}$

For 9 through 12., $\frac{\sqrt{3}}{4}$ simplifies to 0.43.

Chapter II

CHANGES AT THE SUBPERIOSTEAL SURFACE

AT A LEVEL APPOXIMATING the true growth center of tubular bones, changes at the subperiosteal surface throughout life are ordinarily positive, apposition rather than resorption. At levels nearer the areas of transverse subperiosteal remodeling, the growth picture is more complex—involving a phase of apposition, a resorptive phase and then apposition again. For complete safety in measuring subperiosteal apposition throughout life, without any possible complication of subperiosteal resorption, one might concentrate on cases of Pyle's disease (in which superiosteal resorption does not occur) and where the narrowest width of T corresponds to the level of the original center of growth. One might alternatively measure only at a level close to the nutrient foramen (when radiographically visible), or at a level dictated by available knowledge of proximal and distal growth rates, or exclusively in serial longitudinal radiographs using natural bone markers to approximate the center of moment of growth (Garn, Silverman, Hertzog and Rohmann, 1968, Figs. 22-25). However, in practice, measurements of the midshaft site provide useful information on changes in T for many bones and correspond closely to measurements made at the original growth center, while for other bones the level of minimum diameter of T or M may be employed.

The fact is that T behaves in all important respects like the "sexual" growth curve of Scammon in which there is first a postnatal phase of rapid apposition, second a phase of juvenile growth, third an adolescent phase or steroid-mediated growth spurt and, following an asymptote, what would at first appear to be a steady state (see Table II). However, T does not terminate its growth at age twenty-one or even thirty (as does stature when fol lowed in longitudinal perspective). Rather, T grows on and on, albeit slowly, adding perhaps 2 percent from age thirty to age eighty.

Measuring T simply does some violence to changes in shape, as in recovery from bowing. Measuring T in one dimension ignores "cortical drift" (in Enlow's terminology), where, as we have shown, lateral surface apposition exceeds medial surface resorption in the tibia and in the femur so that the entire bone moves in a lateral direction during growth (Garn, Silverman, Hertzog, and Rohmann, 1968, Figs. 26 28). Axial, or rather an

Author's note: This chapter includes data on continuing expansion of total subperiosteal area, as originally suggested by Dr. Richmond Smith, Jr., and contains references to continuing bone growth in the skull as shown by Dr. Harry Israel and more recently extended by Dr. W. Stuart Hunter.

TABLE II
TOTAL SUBPERIOSTEAL DIAMETER (T) IN OHIO WHITES

Age	*Males*		*Females*		*Percent Sexual Dimorphism*†
	Mean	*S.D.*	*Mean*	*S.D.*	
1	4.51	0.33	4.37	0.36	3
2	5.12	0.45	4.94	0.47	4
4	5.54	0.49	5.39	0.49	3
6	6.07	0.54	5.77	0.53	5
8	6.60	0.54	6.28	0.59	5
10	7.17	0.59	6.81	0.64	5
12	7.75	0.64	7.42	0.70	4
14	8.56	0.77	7.80	0.63	10
16	9.11	0.73	7.81	0.62	17
18	9.30	0.70	7.91	0.66	18
22	9.44	0.57	7.96	0.51	19
30	9.40	0.65	7.93	0.81	19
40	9.34	0.68	8.06	0.69	16
50	9.49	0.79	7.93	0.49	20
60	9.68	0.66	8.09	0.47	20
70	9.37*	0.77	8.34	0.70	12*
80	9.07*	0.51	8.29*	0.61	9*

*N below 25

†$100 \left(\frac{M}{F} - 1.00\right)$

gular, remodeling is also ignored in the simple measurement of T, though we have considered angular axial remodeling elsewhere (Garn, Goodspeed, and Hertzog, 1969). But if T is viewed purely in reference to the bone and changes in T are taken to represent net appositional changes at midshaft, this aspect of surface-specific change is in accord with the static bone picture and illustrates one aspect of its complex dynamics.

SUBPERIOSTEAL GROWTH IN INFANCY AND CHILDHOOD

Subperiosteal apposition during infancy and childhood resembles, in pattern, axial growth during infancy and childhood in that there is an early period of rapid increase and a later period of more moderate increase, continuing for a prolonged period of time until the onset of the steroid-mediated growth spurt. In many ways this pattern represents two distinct growth phases, the first an extension of late prenatal growth and the second constituting childhood growth per se.

Rapid early rates of subperiosteal metacarpal apposition are particularly well documented in the Central American data and indicate that the short period of most rapid appositional growth (at a rate equal to 2.00 mm yr) is terminated by the middle of the first year, and the rate declines to approximately 0.5 mm yr through the second year. After a tremendous relative rate of enlargement, therefore, the subperiosteal surface settles down to a lesser rate (with a transient hiatus during the second half of the first year of life), as is shown in Table III.