Geometric Similarity in Allometric Growth: A Contribution to the Problem of Scaling in the Evolution of Size

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GEOMETRIC SIMILARITY IN ALLOMETRIC GROWTH: A CONTRIBUTION TO THE PROBLEM OF SCALING IN THE EVOLUTION OF SIZE

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I. INTRODUCTION: OF BRAINS AND BODIES

"Since men have intelligence," said Anthony (1938, p. 18), "it is natural that they should try to assess the degree of their superiority over other animals in this respect."[1] It was apparent to Cuvier that neither absolute nor relative brain weight would measure this advantage—lest a whale on the one hand or a shrew on the other be regarded as the paragon of intellect. Brain weight is correlated with body weight, and the relation is not linear; small animals have relatively larger brains. Yet, if plots of brain versus body had the same slope for all mammals, then the superiority of man could be measured by his large y-intercept (and the heaviest brain for his body weight that it implied; see fig. 1). Thus, there arose the idea that the brain might be partitioned into two factors, one dependent upon size and one independent of it—and that the slope of a proper growth equation would represent the first, its y-intercept the second. Manouvrier (1885) tried a linear model, with little success. In 1891, Snell first fit the power function

$$y = bx^a, \tag{1}$$

which, in its logarithmic transformation,

$$\log y = a(\log x) + \log b, \tag{2}$$

plots as a straight line with slope a and y-intercept of $\log b$ (with $y =$ brain weight and $x =$ body weight). Since Snell related brain weight to metabolism and M. Rubner had equated metabolism to body surface, Snell expected all curves of brain weight versus body weight to plot with a slope of 2/3. The ratio of y-intercepts for any two lines will then equal the ratio of brain weights at *any* body weight; therefore, Snell argued, the coefficient b represents the part of brain weight that is independent of body weight. And the higher the value of b, the more "cephalized" the animal. Snell named b the "psychic factor" and a the "somatic exponent," believing that he had separated the two components of brain weight.

Eugen Dubois, discoverer of *Homo erectus* in Java, extended Snell's work. He retained the idea of constant slope but reduced Snell's value

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[1] This and all other translations from French and German sources are my own.
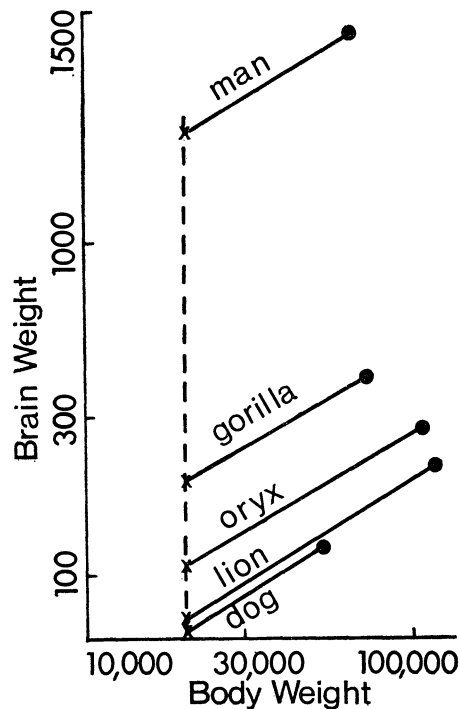
FIG. 1.—Dubois's (1897) method for measuring "degree of cephalization." Abscissa represents body weight in grams plotted on a logarithmic scale; ordinate is brain weight in grams, also on a logarithmic scale. Each point represents a single adult or the mean of several adult animals. A line of slope 0.56 is passed through each point. At any common x-value (dotted line of figure at arbitrary x), relative y-values measure degree of cephalization. Since relative y-values are constant when all lines have the same slope, any x-value can be used; therefore, the y-intercept of this log-log plot ($x = 1$, not shown on this plot) measures the "degree of cephalization." Data from Dubois (1897).

from 2/3 to .56, claiming that the surfaces most influencing brain weight belonged to sensory organs and that eyes (the most important of these) decreased in size relative to body weight far more rapidly than the general body surface (1897, p. 21). He retained Snell's .66 for bats with poor sight and for certain series of small mammals, apparently realizing (as indeed is true) that the rate of relative decrease in eye size is not constant but rather increases greatly in larger mammals. The edifice of his next 30 years was built on the constancy of .56; that conclusion was based on seven pairs of points for seven pairs of "related" species that differed greatly in size (1897, p. 21). The conclusion was self-perpetuating, since Dubois then calculated his y-intercepts from *single* points, assuming the constancy of .56 a priori.

Dubois (1922, 1928) built his famous theory of brain evolution on a belief that evolutionary increase in b occurred in steps of a geometric progression with base 2. Thus, he reasoned, the brain evolves by a doubling of neurons

early in embryology; (the change is reflected only in the increase of size-independent b; the slope remains constant). Brummelkamp (1940, p. 55), a disciple of Dubois, proclaimed: "This leapwise increase in nuclear volume of cortex is the essential moment in the evolution of the vertebrates. . . . This nucleus mass may be considered as the point of application by which Nature drives the vertebrate to still higher levels of evolution." Thus, the coefficient of the power function became the basis of a theory, wondrous in its implications though fragile in its foundations, that depended on the notion that b measured a size-independent component of brain weight.

The weaknesses should have been evident from the start. Dubois's interpretation of b requires absolute constancy of slope; yet he had recognized exceptions to $r = .56$ in his original paper. Dubois's curves were based on adults of related species. Lapicque (1907a, 1907b) plotted adults within species and claimed a "universal" value—"la loi intérieure de l'espèce"—of $r = .25$. This constancy, of course, was as fallacious as Dubois's, but at least it cast doubt on the generality of .56. After numerous criticisms (Anthony 1938; Sholl 1948; Wirz 1950; Jerison 1955; Pirlot 1969), Dubois's theory has passed to oblivion. The reasons are many, but most prominent by far is the fact that interspecific slopes are not constant for all groups. The values hover about .66—and I cannot doubt that there must be some relationship to surfaces in this—but they vary greatly. Intraspecific values are lower (limited growth of nondividing neurons and independent variation in body weight due to nutrition), but they vary just as much.

Snell's power function was extended by Huxley and Teissier in the 1920s and 1930s to the wide range of phenomena that encompass differential or allometric growth. The interpretation of α as the ratio of specific growth rates of y/x has been accepted by all, but the meaning of the coefficient b has generated a large and inconclusive literature (reviewed in White and Gould 1965). Moreover, its interpretation as a size-independent factor has persisted with surprising popularity and stubbornness, even though its warrant had disappeared with the collapse of Dubois's theory.

Interpretation of b as a size-independent factor can be dismissed most easily for regressions that differ in α. This view requires that b and α be independent of one another, but the two parameters are related for mathematical reasons (White and Gould 1965; Mayrat 1966). Yet size independence as a general property of b has been maintained by Oboussier and Schliemann (1967, p. 464) and Zehner (1967, p. 3).

For those numerous cases in which we compare regressions with the same α, the interpretation of b as size independent does not, at first sight, seem incorrect. For in two regressions of equal α, the ratio of b-values equals the ratio of y-values *at any* x (fig. 1). This interpretation has been maintained recently by Röhrs (1958, p. 288), Frick (1961, p. 140), Stork (1968, p. 77), and by Klemmt (1960, p. 273), who says of b: "It encompasses the totality of all factors that influence organ weight independently of the body weight." Indeed, the relative difference in shape between any two

curves (fig. 1) appears at the smallest size common to both regressions and remains constant for all later sizes. But the fallacy is that these are not ontogenetic curves: each represents the static allometry of a series of adults (see Cock 1966 on types of data). There is no reason whatever for thinking that such a static curve should coincide with the ontogenetic curve of a large member of the series. Is there any other meaning that could be given to differences in b for static curves of equal α? Figure 2 shows such an interpretation. The hypothetical curve at T_1 is drawn through three co-linear points representing average adults of related species. Suppose that each animal then evolved to the same multiple of its original size at time T_2 and that, in so doing, its brain increased in constant proportion to body weight (i.e., with $\alpha = 1$ for phylogenetic allometry in each of the three lines). The curve for time T_2 would still have the same slope as T_1, but its b-value would be higher—and that higher value would be a result of *in-creasing size* in each lineage.

In theory, then, shifts in b may reflect size differences between regressions. But does this interpretation ever hold in fact? The answer is that it does, and it must. Bauchot and Stephan (1964) studied intra- and inter-specific brain-body relations in insectivores. Static curves for adults of a single species average .23 for α; α is .64 for the interspecific regression of mean adults. If α is set at .23 for each intraspecific curve and if a line of that slope is drawn through the joint mean of each species, the resulting set of b-values are, with one exception, correlated with the mean size of the species (fig. 3). And this must be so if the joint means are to lie on an interspecific regression of $\alpha = .64$.

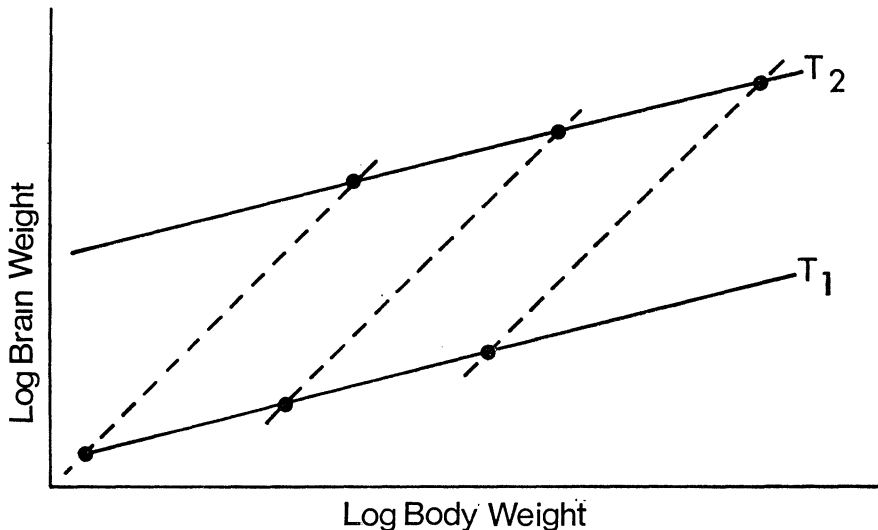

FIG. 2.—A shift of regression lines on logarithmic scales (change of inter-cept; no change of slope) produced by size increase of adults rather than by reorganization of proportions at the outset of allometric growth. See text for details.
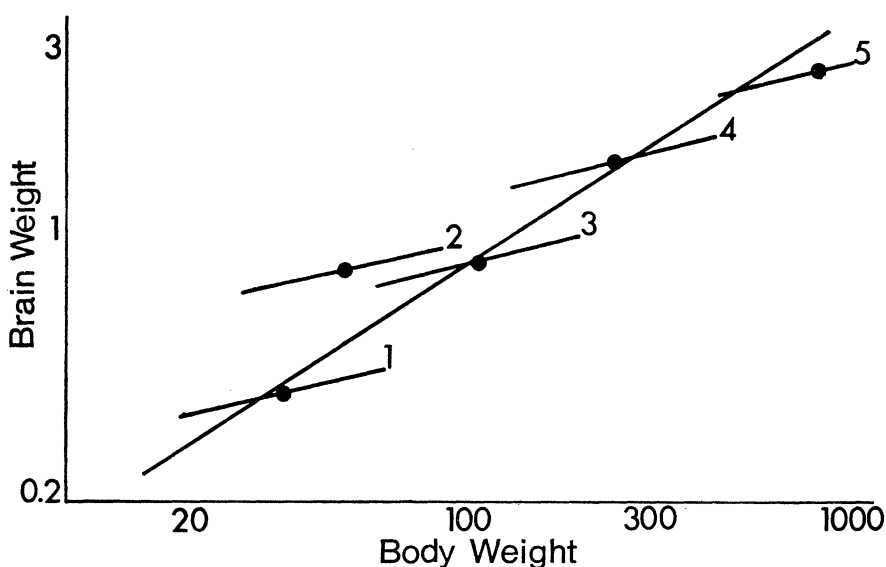
FIG. 3.—Intra- and interspecific brain-body curves for insectivores from Madagascar (from Bauchot and Stephan 1964), logarithmic scales. Intraspecific curves are set at their average α-value of 0.23 (points represent the joint means); α is 0.64 for the interspecific curve. For this situation to obtain (inter α > intra α), b-values *must* generally increase for larger animals; b will be a measure of size, not of "level of cephalization." *1, Suncus murinus; 2, Nesogale talazaci; 3, Hemicentetes semispinosus; 4, Setifer setosus; 5, Tenrec ecaudatus.* Weight given in grams.

Allometric equations apply only to the range of data that they fit; yet they are usually written as if they applied to an infinite range of x. We are so used to writing them in this general form that we have neglected to compare the size ranges of the several lines in a series and have thereby missed the significance of many shifts in b—we have not understood that they are size required. I recognized this by accident. In scanning all series of constant α that I knew in the literature, I noticed the consistent pattern depicted in figure 4: when $α < 1$, higher b-values are associated with larger sizes; when $α > 1$, higher b-values characterize smaller sizes. Values of α that differ strongly from 1 are almost always size limiting (Gould 1966a, 1966b) because extrapolation to a much-widened size range produces such drastic and rapid changes in shape. If the ontogenetic or static α for each of several related species is constrained to be nearly constant and much less than 1, if the species differ in adult size, and if each must display the same range of proportions and attain the same final shape, then higher b-values for successively larger species must characterize the series. Likewise, among several ontogenetic or static lines of constant $α > 1$, larger species must have lower b-values to develop the same range of proportions and final shape (fig. 4; remember that lines of $α = 1$ join points of constant shape). I can imagine no other explanation for this consistent pattern than that larger species must often attain ranges in shape similar to those of smaller relatives.
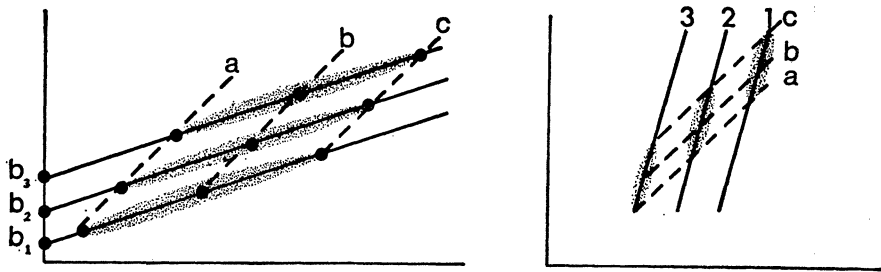
FIG. 4.—The correlation of b and size in systems of constant α. In each case, the three regressions represent changes in size with *no change of shape* (i.e., dotted line a of slope $\alpha = 1$ joins the smallest animals of each regression, line b the joint means, and line c the largest animals; on log-log plots, lines of $\alpha = 1$ join points of equal shape). Abscissa and ordinate are log body weight and log brain weight, respectively, as before. Case 1, if α (*solid lines*) is equal and less than 1 for all regressions, then *larger* animals must lie on regressions with higher b-values ($b_3 > b_2 > b_1$) if constant shape is maintained. Case 2, if α is equal and greater than 1 for all regressions (*solid lines* 1, 2, and 3), then *smaller* animals must lie on regressions with higher b-values ($b_3 > b_2 > b_1$).

Kurtén (1954) first argued, for a specific case, that a shift in allometry lines had occurred to preserve shape at larger sizes. Meunier (1959*a*, 1959*b*), the only other author who has recognized the size dependence of such shifts, refers to them as ''Transpositionsallometrie''—''a size dependency of higher ordering'' (1959*b*, p. 346). ''Transposition,'' he states (1959*b*, p. 353–354), ''is the ordering system of size ranges in which intraspecific allometries lead to functional [*lebensfähigen*] forms.'' Yet, despite this insight, Meunier does not realize that b measures his size dependency, for he is still tied to the view that it ''encompasses all influences that do not depend upon size'' (1959*b*, p. 351).

Misinterpretation of b extends far beyond the false idea of size independence; in fact, there are national schools of error. Size independence is stressed by German authors; English writers have generally followed Huxley's view that b ''has no biological or general significance'' (1950, p. 465; see also his excellent book [1932]). Since b is the value of y at $x = 1$, and since $x = 1$ often represents a distant extrapolation from the size range of data, Needham (1950, p. 553), Schadé (1959, p. 162), Mitra (1958, p. 999), and others have pronounced it devoid of biological meaning. But $x = 1$ should be seen as a convenience for calculation; b is part of a relation that is valid in a certain size interval; if the y-value for an extrapolated $x = 1$ lies in the realm of biological absurdity, a claim for b's importance is not upset thereby.

II. DEVELOPMENT OF A MEASURE OF SIZE DIFFERENCE FOR CONSTANT SHAPE

I shall now show that b can be manipulated to yield a measure of the relative difference in size at which one regression has the same shape as another, that is, of how much larger an animal on one regression must be

than that on another to have the same y/x ratio in any bivariate relation of constant α for both curves. Thus, b is a scale factor; it shifts as the arbitrary unit of measurement changes and when the size of objects, but not their range of shapes, is altered. In other words, whenever we modify size and *only* size, b and *only* b changes. Thus, this measure tells us whether a change in b represents a shift of scale alone. If the actual difference in the size of animals on two regressions equals this measure of the difference in size for constant shape, then a change in scale alone has produced the shift in b, for the animals on the two curves are constituted in geometric similarity. The following argument is a simplified version of that presented in White and Gould (1965).

Consider two regressions of constant $\alpha \neq 1$. (When $\alpha = 1$, all points on any one curve and no corresponding points of two curves have the same shape, since y/x is a constant for each curve.) For any point on regression 2, there is one and only one point of the same shape on regression 1 such that:

$$\frac{y_1}{x_1} = \frac{y_2}{x_2}, \tag{3}$$

and

$$\frac{y_1}{y_2} = \frac{x_1}{x_2}. \tag{4}$$

At these points, the two regression equations are

$$y_1 = b_1 x_1^a \quad \text{and} \tag{5}$$
$$y_2 = b_2 x_2^a.$$

Therefore,

$$\frac{b_1}{b_2} = \frac{y_1/x_1^a}{y_2/x_2^a}, \tag{6}$$

and from (4)

$$\frac{b_1}{b_2} = \frac{x_1/x_1^a}{x_2/x_2^a}, \tag{7}$$

or

$$\frac{b_1}{b_2} = \left(\frac{x_1}{x_2}\right)^{1-a} \tag{8}$$

Now x_1/x_2 is the desired quantity—the relative difference in size at which shape on the two regressions is the same. Let us call this quantity s. Finally,

$$s = \left(\frac{b_1}{b_2}\right)^{1/1-a} \tag{9}$$

Since α is constant, s, the size difference for constant shape, depends upon the ratio of b-values.

In the next two sections, I shall present two types of examples in which the calculation of s from a ratio of b-values can help to illuminate a prob-

lem. In the first type, we make no hypothesis about geometric similarity of animals on two regressions, but merely maintain that s provides a useful comparison of two curves. I shall argue that s is sometimes more appropriate or enlightening than the usual comparison of b-values. In the second type, I am testing the hypothesis that a shift in b has occurred in order to maintain geometric similarity between animals in different size ranges.

III. THE SIMILARITY CRITERION s AS A FIGURE FOR THE USEFUL COMPARISON OF TWO REGRESSIONS

a) Evolution of the Brain

Jerison (1961) plotted static curves for the brain-body relation in adult Eocene, Oligocene, and Recent mammals. He obtained the following regressions:

$$y = .026x^{.657} \text{ for Eocene mammals,}$$
$$y = .055x^{.655} \text{ for Oligocene mammals, and} \qquad (10)$$
$$y = .115x^{.664} \text{ for Recent mammals.}$$

The standard comparison for, say, Eocene and Recent mammals would be made by dividing the two b-values and stating that the brain of an average Recent mammal is 4.4 times as heavy as that of an average Eocene mammal at any body weight. But the similarity criterion, s, provides another comparison. From (10)

$$s = \left(\frac{.115}{.026}\right)^{1/(1-.66)} = (4.42)^{2.94} = 64. \qquad (11)$$

Thus, an average Recent mammal is 64 times as heavy as an average Eocene mammal of the same brain-weight/body-weight ratio. Since weight scales as l^3, the average Recent mammal is four times as long as the average Eocene mammal with the same relative brain weight.

Both of these comparisons are "correct," but which is more enlightening? This depends on how the mammalian brain increased in phylogeny, a question that cannot be answered from these static curves. If Dubois was right after all and the brain increased by a reorganization of proportions in the developing embryo, then the ratio of b-values is an appropriate comparison, since it emphasizes the constant difference in relative brain weight at any postnatal size. If, however, the brain evolved by maintaining a constant ratio with body weight during phylogenetic size increase (the simplified situation of fig. 2), then s is a more meaningful comparison because it measures that increase in size. (It is unlikely in the extreme, of course, that such a multifaceted phenomenon as brain evolution proceeded according to any model as simple as these.)

Although comparisons by the b-ratio and s criterion are both "correct," this does not label them as neutral, commensurate, and purely descriptive

schemas. Both contain an implicit preference for a certain theory of brain evolution. I am not arguing for the theory more congenial to the s comparison, but merely pointing out that our previous reliance on b-ratio comparisons has directed our thinking along certain unsubstantiated lines.

b) Acceleration and Paedomorphosis in the Evolution of Fossil Invertebrates

Newell (1949) studied the relationship of suture length to conch diameter in an evolutionary series of Paleozoic ammonoids. He plotted the inferred ontogeny of five genera in the series and found a nearly constant α ranging from 1.20 to 1.28; b-values increase continually with time. Since $\alpha > 1$, descendants reach any given suture-length/conch-diameter ratio at a smaller size than ancestors. We have a case of acceleration in development, or recapitulation. Although Newell recognized this (1949, p. 115), he used only the b-ratio comparison and obtained no measure for the extent of acceleration. That measure is provided directly by the similarity criterion s. For the relationship between *Uddenites* ($\alpha = 1.23$) and its descendant *Medlicottia* ($\alpha = 1.24$), for example, using an $\bar{\alpha}$ of 1.235 and estimating b_1/b_2 from Newell's graph (his reported b-values are incorrect):

$$s = \left(\frac{15}{22}\right)^{1/(1-1.235)} = .67^{-4.2553} = 5.50. \tag{12}$$

Thus, *Medlicottia* reaches the same suture-length/conch-diameter ratio as its ancestor at a conch diameter 5.5 times as small.

Hallam (1968) studied the evolution of *Gryphaea* through Liassic deposits of England. Although discussion still proceeds on whether this oyster increased the tightness of its coil during the lowermost Lias, Hallam has shown that the overall trend throughout the lower and middle Lias was toward decreased coiling. He finds that length/height is a good measure of coiling: the greater the relative height, the tighter the coil. Ontogenetic plots for log length versus log height display, for almost all samples, a very small range of α from .77 to .82; that is, coiling increases during ontogeny. But geologically younger samples show progressively increasing values of b. Using the b-ratio criterion, these younger samples decrease their coiling because they have, at any common size, a greater length/height ratio. But this may not be the appropriate contrast. *Gryphaea* also increases in size during phylogeny. If increase in size equaled the value of s, then, in one important sense, there would be no decrease of coiling at all; adults would have the same shape at their progressively larger size. Lower length/height at any common size would constitute a false comparison; for that, common size would represent a different developmental stage in each sample. But if the rate of size increase were smaller than s, Hallam would be upheld because adult descendants would not proceed far enough along their ontogenetic curve to attain the shape of adult ancestors; their final shape would be that of a juvenile stage of ancestral ontogeny, that is, less

tightly coiled. The difference between s and the actual size increase would measure the extent of this paedomorphosis. This indeed is the case. If we take the two end points of the sequence—*G. arcuata obliquata* and its ultimate descendant *G. gigantea*, both at $\alpha = .800$—s is 7.42 while the actual ratio of maximal sizes is 1.76.

c) *Metabolism*

For most animal groups, log-log plots of basal metabolism versus weight have a slope near .66 (usually slightly higher); some relation between metabolism and body surfaces is strongly implied (Kleiber 1961; Gould 1966*b*, p. 613–615). While α varies little in these plots, b ranges widely among groups. For example, Dawson and Hulbert (1969) present the following generalized curves (static, interspecific) for marsupials and eutherians:

$$M = 12.5W^{.74} \text{ for marsupials and}$$
$$M = 16.4W^{.75} \text{ (from Kleiber) for eutherians.} \tag{13}$$

These authors use the b-ratio comparison in concluding that marsupial metabolism is about 30% below the eutherian rate at any body size (1969, p. 383). I regard this comparison as appropriate. We can also compute s and state that an average eutherian is 2.9 times as heavy as an average marsupial of the same metabolism/body-weight ratio. Likewise, Lasiewski and Dawson (1967) cite identical $\alpha = .72$, but unequal b-values, for passerine and nonpasserine birds (static, interspecific plots; Zar [1968] has challenged the curve-fitting technique of these authors). By the b-ratio comparison, the metabolic rate of an average passerine is 1.62 that of an average nonpasserine at any body weight; by the s criterion, the average passerine is 5.66 times heavier than an average nonpasserine of the same metabolism/body-weight ratio.

IV. THE SIMILARITY CRITERION s AS A TEST FOR GEOMETRIC SIMILARITY BETWEEN ANIMALS ON DIFFERENT REGRESSIONS

In this section, I use s to test a hypothesis: that the magnitude of a transposition between regressions of constant α is such that animals in a new size range remain geometrically similar to related forms in the original range.

a) *The Comparison of Closely Related Species*

Meunier (1959*a*, 1959*b*) developed his concept of transpositional allometry in studying gulls of the genus *Larus*. Plotting the cube root of weight versus vertebral column length for adults in each of five species, Meunier found little variation in α; but the lines were transposed, one over the other, with the higher b-values belonging to species of larger body
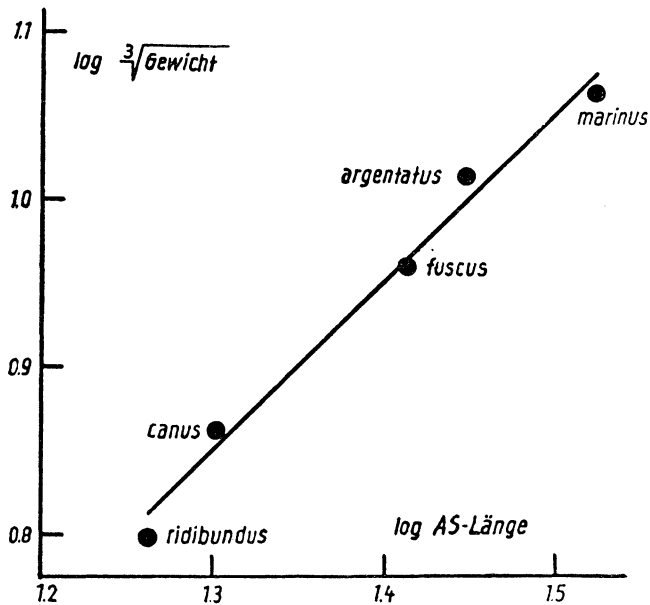
FIG. 5.—The maintenance of geometric similarity. Line of $\alpha = 1.003$ fitted to the joint means for cube root of weight versus vertebral column length in five species of gulls. Within each species, α is about 0.75. From Meunier (1959b).

size. If a single line is fitted to the joint means of the five species (fig. 5), the interspecific α is 1.003 (intraspecific α averages 0.75). Thus, as a result of transposition, larger species are geometrically similar to smaller ones for this relation. This should be reflected in a correspondence between the value of s and the actual ratio of mean sizes. Indeed, for the two end points of the series, L. ridibundus and L. marinus at $\alpha = 0.75$,[2] s is 1.90 while the ratio of mean x-values is 1.88.

Röhrs (1958) studied the brain-body relation in Felis caracal and the larger cat, F. lynx. For intraspecific plots of adults, α is .226 for both species, but F. lynx has a higher b-value than F. caracal, the ratio of b-values being 1.163. By his allegiance to the fallacious notion of b's size independence, Röhrs is compelled to offer the following interpretation:

Caracal skulls of the same size as lynx skulls have an absolutely smaller brain volume [true, so far, of course]. Therefore the differences in proportion between the skulls of the caracal and lynx are independent of their size. . . . From our previous knowledge of

[2] I have, in this and other cases, used, as a common α-value, the mean between regressions that exhibit small differences in this parameter. When I have done this, I have always recalculated b for each line by passing a line of average α through the joint mean. If I could not determine the joint mean, I abandoned the calculation. This must be done because the relation between b and α is so strong, especially when $x = 1$ is far from the actual range of data (White and Gould 1965). Large errors would enter if s were computed from a ratio of b-values, each determined from a different α, even though that difference be small.

the evolution of the brain, we know that phylogenetic increase in cranial capacity often occurs by transpositions [*sprunghafte Vergrösserungen*—literally "saltational increases"] that are independent of body size. [1958, p. 286–288]

But comparison at the same size is inappropriate, for *F. lynx* is larger than *F. caracal*; thus, we compare a small caracal with nothing, a large caracal with a small lynx, and a large lynx with nothing. In fact, there has been no relative increase of brain size at all, for the lynx, at its larger size, is a scaled-up replica of the caracal for this relation:

$$s = 1.163^{1/(1-.226)} = 1.163^{1.276} = 1.21, \tag{14}$$

while the ratio of mean body weights for the two species is 1.16. Note that this geometric scale-up corresponds with the unorthodox interpretation of brain evolution that would vindicate the *s* comparison for Jerison's data mentioned earlier.

Bährens (1960) provides a similar case for endocranial volume versus skull length in the mustelid carnivores *Mustela vison* and *Martes martes*. At a common α of .44, the larger *M. martes* has a higher *b*-value; *s* is 1.30; the ratio of mean skull lengths is 1.25.

Zollitsch (1969) stated, as the main conclusion of his comparison between the skulls of *Canis lupus* and *C. aureus:* "a jackal's skull is differently proportioned than a hypothetical wolf's skull of the same size. . . . Therefore a jackal can in no way be viewed as a wolf that decreased in size" (p. 181). Again the comparison is a false one. Zollitsch has proved that you cannot make a jackal's skull by extrapolating down the static intraspecific regressions of wolf characters to a jackal's size. But we know that extrapolation of strong allometry to new sizes quickly produces inadaptive proportions. There are several meaningful senses of "scaling" down or up; the notion should not be restricted to the extrapolation of ontogenetic or static regressions to new sizes. *Geometric* scaling occurs when actual size differences equal calculated *s*-values.

An inarticulated premise pervades most of our literature on this subject: that scaling up or down an ontogenetic curve is somehow "simpler" and less likely to require genetic modification than any other mode of size change. In fact, a transposition, in which *s* equals the extent of initial size change, requires only that an animal begin allometric growth with the same proportions as its prototype, but at a different size (fig. 4). I shall argue below, in discussing local races, that this mode of size change is as "simple" as any other.

Zollitsch's conclusion does not exclude the possibility that a jackal's skull may, for certain relationships, be a geometrically scaled-down model of a wolf's skull. Indeed, it is. This is clear from the many plots of constant α that Zollitsch draws: when allometry is positive, *b* for jackal skulls is higher than that for wolf skulls; when allometry is negative, wolf skulls have the higher *b*-values. Consider the dimensions of the fourth upper premolar, the important carnassial tooth. For a plot of its length versus basal length of the skull, α is 1.9 for both species; *b* is higher for jackal skulls, as a hypoth-

esis of geometric similarity requires. Yet, although there is no overlap in basal length between species, Zollitsch (p. 172) interprets the situation as follows: "On the average, a jackal's upper carnassial tooth is, independent of skull size, 49.3% longer than that of a wolf." But the value of s for these two regressions is 1.56, while the ratio of mean basal lengths for the two species is 1.51. An average wolf's skull has a carnassial of the same relative length as does the average jackal's skull. The same is true for carnassial width versus basal length, where s is 1.47. For carnassial dimensions, an average wolf's skull is a scaled-up geometric replica of an average jackal's skull. Geometric similarity is preserved by a transposition of allometry lines expressed as a shift in b-values.

As a final example, I may cite Bohlken's (1967) comparison of the American bison with its slightly smaller European counterpart. Bohlken makes all his contrasts in the traditional way: for systems of constant $α$, he states (p. 88, for example) that differences in b are independent of size because they signify a constant ratio between y-values of two regressions at any x. In fact, many of the differences in b are size dependent because they allow the American bison to maintain the same shape as the European bison at its larger size. The ratio of mean basal skull lengths for the two species is 1.06; s is near this value for many relations with basal length: 1.04 for infraorbital width, 1.07 for occipital height, and 1.05 for circumference of the horn. In several ways, the skull of an American bison is a geometrically scaled-up model of its European prototype.

b) Evolutionary Sequences

Kurtén (1955) studied the relationship between paracone height and crown length in upper first molars of the Pleistocene cave bear, *Ursus spelaeus*, and its close relative, the smaller, modern brown bear, *U. arctos*. Allometry is positive and of almost equal intensity in both species: mean $α$ is 1.47 (fig. 6). Yet, as we have come to expect when $α > 1$, the line for the larger cave bear is transposed below that for the brown bear. In formulating an explanation for this shift, Kurtén considers the consequences of extrapolating one curve to the size range of the other species: "Imagine the allometric pattern of *U. arctos* projected into the larger size of *U. spelaeus*. The result would be a very hypsodont tooth. . . . The first molar would then jut out of the tooth row and probably inconvenience its bearer" (1955, p. 114). In fact, Kurtén's data imply strong selection to maintain a constant molar shape at very different sizes. For six populations, mean hypsodonty indices (100 × paracone height/crown length) vary only between 38 and 43 (1955, p. 116, table 2)—"a surprising constancy," Kurtén notes, "despite marked differences in gross size" (p. 115). Shape is preserved by a shift in b-values, and s should equal the actual ratio of mean sizes. For the two populations of figure 6, s is 1.50 while the ratio of mean sizes (1955, p. 116, table 3) is 1.42.

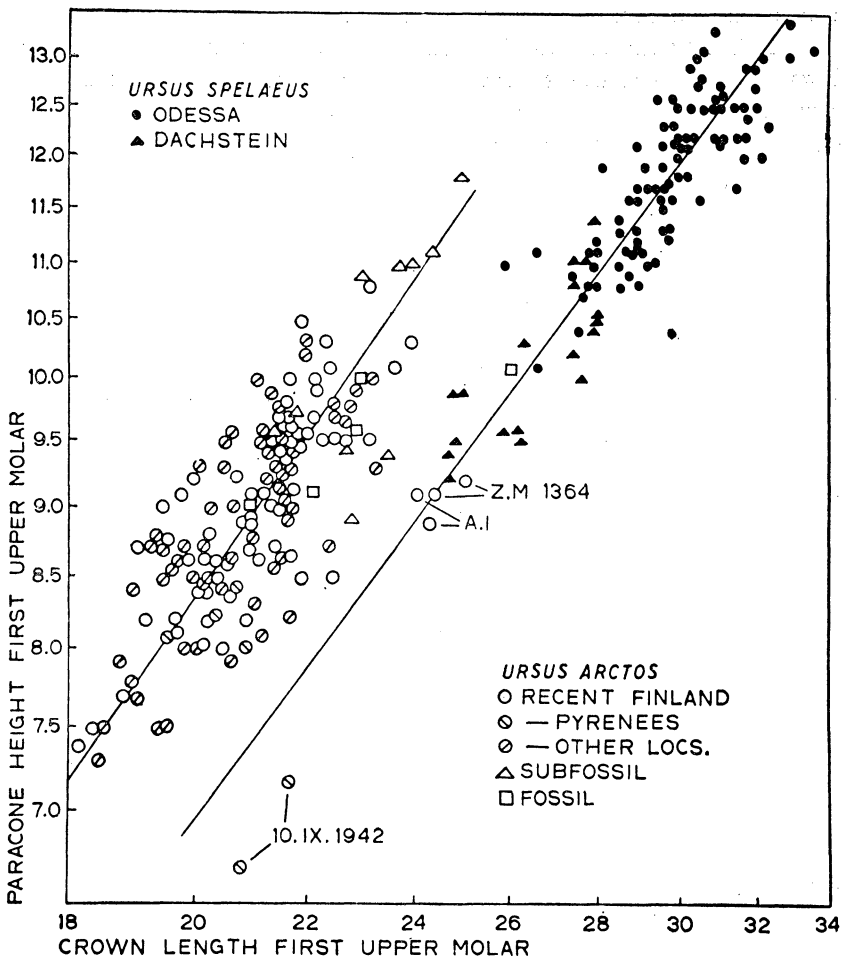The great debate on *Gryphaea* has been plagued, since its inception in

FIG. 6.—A transposition in the evolution of bears. Both cave bears (*Ursus spelaeus*) and brown bears (*U. arctos*) have the same average shape for paracone height/crown length of the first upper molar. Since intraspecific α > 1, the line for the larger cave bear must be transposed below that of the brown bear to preserve a constant shape. From Kurtén (1955).

the 1920s, with biometric errors arising from the improper treatment of size. All admit that this Jurassic oyster increased in size during the deposition of lowermost Liassic strata in England. The major argument is whether it increased the intensity of its coiling as well. (Hallam's earlier-mentioned work on the later evolution of *Gryphaea* is not involved in this case [1968].) I have reviewed elsewhere the various contentions and the methodological errors upon which they were based (Gould 1970 and in press). In 1965, Burnaby made the surprising claim that *Gryphaea* had decreased the intensity of its coil from the *Angulata* to the *Gmuendense* zone. For a growth series in each sample, he plotted left (coiled) versus right (flat) valve length. The intensity of coiling increases in ontogeny;

the power function applies with a value of α that Burnaby took as a constant 1.766 in all samples. Burnaby's claim of decreased coiling in phylogeny is based upon a systematic decrease in *b* with time: at any given size, descendant populations have a lower coiled-valve-length/flat-valve-length ratio than their ancestors. This ratio is a fair measure of coiling in *Gryphaea,* but phylogenetic size increase throws doubt upon such a comparison, for it leads to the contrast of an ancestral adult with a juvenile descendant of the same size (see analogous cases in Zollitsch's canids and Röhrs's felids, discussed above). Size increases, *b* decreases, and α < 1; we have all the conditions for geometric scale-up (i.e., for no change in shape for comparisons at similar developmental stages). Mean *b* for four *Angulata* (ancestral) populations is .237, while mean *b* for two *Gmuendense* populations is .210; therefore, *s* is 1.17. We cannot compare this with the ratio of mean *x,* since these are ontogenetic plots, but taking Hallam's data (1959) for *maximal* sizes in the two zones, the *x*-ratio of largest specimens is 1.21. The large, descendant *Gryphaea* of the lowermost Lias is a geometrically scaled-up replica of a smaller ancestral *Gryphaea;* allometric lines shift in phylogeny in order to maintain *Gryphaea* at the same shape with increasing adult sizes.

c) Domestication and Local Races

Walton and Hammond (1938) performed breeding experiments between Shetland ponies and Shire horses. Plotting forelimb length versus body length for ontogenies of parents and offspring, they claimed (but did not calculate) a common α for all regressions (in the neighborhood of 0.8) and increasing *b*-values for the following series of four plots: pure Shetland, Shire father and Shetland mother, Shetland father and Shire mother, and pure Shire. This is also the sequence of final adult size. Walton and Hammond (1938, p. 329) give the usual interpretation: "This implies that the pure Shetland is relatively shorter in the limb than the pure Shires and the crosses are intermediate and that this is true at any body size." But again, this compares an adult Shetland with a juvenile Shire, and we know that young horses have relatively long legs. The average body-length/forelimb-length ratio for adult Shetlands is 1.395 (at mean length of 195 cm) and 1.36 (at body length 318 cm) for the single Shire adult that they cite. The *b* increases in order to preserve a constant shape at similar developmental stages in forms of different sizes.

Teissier (1936) discusses plots of claw length versus body length for two local races of the lobster *Homarus americanus.* Before maturity, both samples lie on the same regression with a slope of 1. At maturity the slope changes to 1.55 for each species; but this occurs at 22–25 cm body length in one sample and at 33–35 cm in the other (fig. 7). Since α is 1 for juvenile growth, both populations reach maturity with the same shape for this relation; *s,* as computed from the two regressions for mature lobsters, must equal the ratio of sizes between the two populations at the onset of maturity.
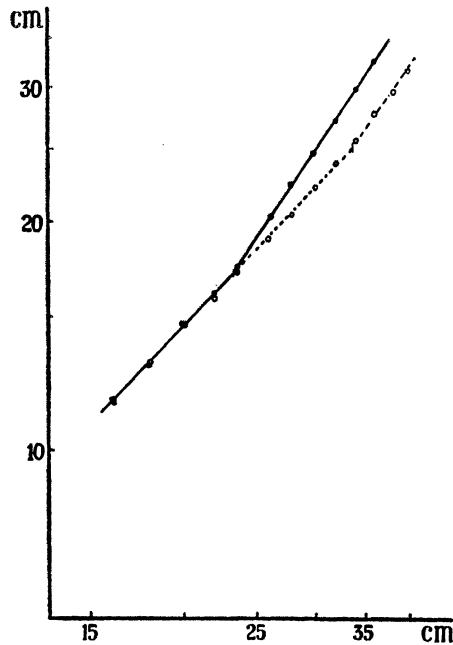
FIG. 7.—Claw length (*ordinate*) versus body length (*abscissa*) for two local races (*points* and *circles*) of the lobster *Homarus americanus*. Ontogenetic plots; α changes from 1 to 1.55 at maturity, but maturity occurs at different sizes, producing a transposition. Modified from Teissier (1936).

Of this type of growth, Teissier aptly remarks (1936, p. 631) : ''The differences between local races of lobsters are not due to differences in the nature or the intensity of growth, but to the fact that the same series of processes takes place later in certain races than in others.'' I had this example in mind when I stated earlier that a transposition may represent as ''simple'' a change in growth as the extrapolation of an ontogenetic curve to new sizes; for, in this case, one population begins allometric growth with the same shape, but at a larger size, than the other. That is the only difference. Thereafter, each stage is reached by one curve at a correspondingly larger size, equal to s, than the other. Many multivariate studies of growth have distinguished size and shape vectors as mathematically independent (that these are ''real'' biological entities and not mere formal abstractions is unproved, but at least not implausible). Reyment (1961, 1966) has argued that size vectors are more easily altered in evolution than shape vectors. Shifts in b that change size and preserve shape may reflect minor genetic differences or phenotypic responses to varied environments. I doubt that any more is involved in the Shetland-Shire case. Here the sequence of increasing b parallels that of size at birth. If all these horses, whatever their size, are born with approximately the same proportions and attain similar shapes as adults, then their allometric relations must form a set of transposed regressions.

Bohlken (1964) compared wild and domestic populations in five bovid species. I shall cite just one among many cases of geometric scaling: the relationship between cheek width and basal length in *Bos* (*Bibos*) *gaurus*. For wild and domestic forms, α is constant at 1.61. Bohlken's *b*-values are incorrect; they were recalculated to pass through the joint means at his given α. For the two regressions, *s* is 1.09; the ratio of mean basal lengths is 1.08. Moreover, geometric similarity is preserved throughout the size range, for the ratio of smallest basal lengths is 1.10, while that of largest basal lengths is 1.08.

Throughout the 1920s, Nomura and his colleagues made a series of meticulous studies on local variation in relative growth of molluscan shells. Finding little variation in α within a species, they used its average value to compute *b* for all regressions. Their data abound in cases of geometric scaling that they did not recognize. For the periwinkle *Littorina sitchana* from Asadokoro and Yunoshima at α = .74 for height versus width (Nomura 1926), *b*-values are 1.419 and 1.332, respectively, and *s* is 1.28 for these ontogenetic plots, while the ratio of maximum shell sizes is 1.29. For the snail *Purpura* from Oshima-Watanoha and Bentenjima at α = .92 for height versus width (Nomura 1928), *b*-values are .697 and .684, respectively, and *s* is 1.28, while the ratio of maximum shell sizes is 1.30. Nomura tried to give *b* a general meaning as a "local constant." Although incorrect as a universal interpretation, the term is apt for these cases; *b* seems to vary in response to local conditions that influence size but not shape.

V. DISCUSSION: THE IMPORTANCE OF GEOMETRIC SIMILARITY IN EVOLUTION

I have shown, in the last section, that allometric plots of related animals are often transposed as adaptations to maintain geometric similarity (constant proportionality of parts) over a wide range of size. If this claim contains any element of significance or surprise, it must be shown that preservation of shape is not the anticipated result of change in size. We might be tempted to consider geometric similarity as "no change," as, therefore, the easiest way (in a formal sense at least) of altering size. Yet this is not so for two major reasons:

1. The most direct path to increased size—the extrapolation of ontogeny —does not produce geometric similarity, for most individual growth is replete with strong allometry in many of its features.

2. If geometric similarity is not the most direct way, is it at least the best way? It is not, because large animals must change their shape in order to function as well as smaller animals built upon the same plan (a consequence, for the most part, of declining surface/volume ratios at increased sizes [Thompson 1942; Gould 1966*b*]). Thus, I conclude that geometric similarity is a problem, not an expectation.

I shall try to resolve this problem in two ways: by showing that proportioned change in size can be accomplished easily (in a genetic and developmental sense) and by arguing that it might be advantageous to grow this

way (as an adaptation). Some clues to the first point can be gleaned from the vast literature on dwarfing, both racial and individual, in animals and man. There are many kinds of dwarfs and more names by an order of magnitude to describe them. Three general types (each with a variety of genetic bases) seem most common. Achondroplastic dwarfs have very short limbs with normal heads and trunks (Stephens 1943; Stevenson 1957); the condition is usually inherited as a simple Mendelian dominant. Infantilized dwarfs retain juvenile proportions and often do not reach sexual maturity (Snell 1929; Smith and MacDowell 1930; McKusick and Rimoin 1967). In a third, and broader, category lie the proportioned dwarfs—miniaturized replicas of normal forms. Races of proportioned dwarfs—and mutated individuals in normal populations have been described in a wide variety of animals, both living and fossil (Mead, Gregory, and Regan 1942; McKusick 1955; Gates 1958, 1961; and several articles of the symposium edited by Weninger 1954). In man, proportioned dwarfs include hypopituitary ateliotics within normal populations (Andersen 1966; Dupertius 1945; McKusick 1955; McKusick and Rimoin 1967) and several "pygmy" races and tribes. Debate has raged over the type and genetic basis of dwarfing in these "pygmies." The true pygmies, the Ituri of the African Congo (Gusinde 1955), have been likened both to achondroplastic (Gates 1958) and to infantilized (Hohenegger 1954) dwarfs, but most anthropologists speak of them as fairly well-proportioned (Adé 1954). Few doubt that dwarfing among the Ituri occurred via a major mutation (Fischer 1950, 1954), but dwarfing among other peoples has been ascribed to poor conditions of life (Gusinde 1956) and to gradual selection within the polygenic series controlling general body size (Fischer 1950). Some outstanding cases of proportioned giantism have also been described in man; see Schlaginhaufen's (1959) 25 years of observation on a man 254 cm tall.

Two points are, I think, relevant to our argument:

1. The genetic basis of proportioned dwarfism is often simple and monofactorial. Mead et al. (1942) and Johnson, Harshfied, and McCone (1950) traced proportioned dwarfism in beef cattle to a simple autosomal recessive; the same is indicated for human ateliotics (McKusick 1955; see McKusick et al. [1965] on dwarfing among the Amish, and McKusick and Rimoin [1967] on the pedigree of General Tom Thumb).

2. Its developmental basis is, likewise, often simple. Ateliotic dwarfs in normal populations owe their condition to hypopituitarism, that is, to undersecretion of human growth hormone (HGH) (see Korner [1965] on the action of HGH and Glick et al. [1965] on factors that affect its secretion). Rimoin et al. (1968) have recently found inactive HGH in the Ituri pygmies. Oversecretion of HGH produces well-proportioned giantism if accomplished before closure of the epiphyses (and disproportionate acromegaly if after [Andersen 1966]).

My previous analysis of geometric similarity was based upon allometric transpositions. Is the genetic basis of any transposition known? Is it simple? Sinnott and Dunn (1935) have shown that such differences in b arise from

single gene effects in gourds and peppers. Cock (1966) has reanalyzed previous data on the creeper gene in fowl, an autosomal semidominant (lethal in homozygous form) that produces chondrodystrophic syndromes (reduced length of limb bones). Plotting tarsometatarsal length versus body weight for prenatal growth from 7 days to hatching, Cock showed that the regression for creeper embryos was transposed below that for normals; the slopes of the two lines did not differ significantly. The developmental basis of creeper, though unknown, is certainly established during the first 7 days of life. Perhaps the formation of the tarsometatarsus within the limb bud is merely delayed in time relative to normals; this would be sufficient to produce the transposition. It is intriguing, if only analogical, that key features in proportioned dwarfs are often delayed in time of appearance (sexual maturity [McKusick 1955; Andersen 1966, p. 208–209] and pubertal growth spurt [McKusick and Rimoin 1967] in human ateliotics and ossification centers of limb bones in dwarf rabbits [Crary and Sawin 1949]).

Thus, in conclusion:

1. Proportioned dwarfs and giants often owe their condition to single gene changes that control simple hormonal mechanisms.

2. Allometric transpositions may result from single mutations.

3. Geometric similarity in dwarfs and giants might arise from the simple acceleration or delay in ontogenetic time of appearance of a feature relative to body weight (fig. 8). Such a change in time will produce a transposition.

The major theme underlying this whole discussion is an old one: the interaction of growth and development. Falkner (1966, p. xv) has written:
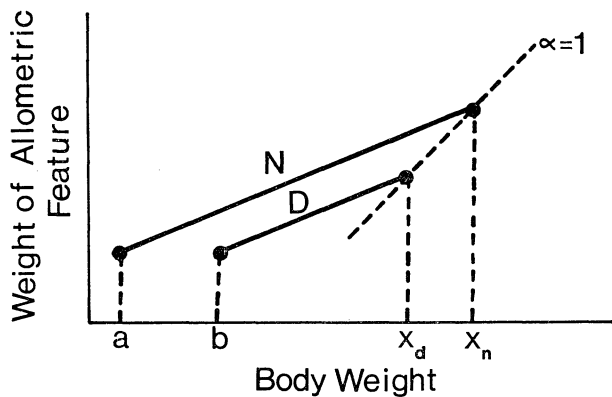

FIG. 8.—Production of proportioned dwarf by simple delay in time of appearance in development of a feature; ontogenetic plots; a = body size at appearance of allometric feature for a normal animal (regression N); b = body size for appearance of the same feature in a proportioned dwarf (regression D); x_n and x_d are the final sizes of normals and dwarfs, respectively. Both x_n and x_d have the same shape (y/x, since both terminal points lie on a line of $\alpha = 1$).

We have been taught that growth refers to the multiplication of cells and to size changes, while development concerns the maturation of such structures and their concomitant functions. Today we realize that the whole process—call it what you will—is extremely complex and a mass of processes interacting upon each other. Because, in addition, there is no dividing line between the two terms, it seems desirable to drop an unnecessary and rather false division and use but one of the two good words.

I believe that this is an oversimplification and that the two processes, while highly intercorrelated, are not completely interdependent. It is the conclusion both of some modern multivariate studies (Reyment 1961, 1966) and of all the old phylogenetic arguments collectively grouped under the concept of heterochrony (de Beer 1958) that, under certain circumstances, the usual correlation of growth and development, of size and shape, can be unlocked. Transpositions that have as their adaptive significance the preservation of geometric similarity at new sizes express this uncoupling of the usual correlation between size and shape, for they permit size to change alone. The possibility of this uncoupling has several consequences for macroevolution, two of which are important to this paper:

1. It permits rapid evolution of size. A strong and sudden selective pressure for size change will require a rapid response. Simple genetic and hormonal changes can unlock the usual correlations of ontogeny and produce an immediate and major change in size alone. Szarski (1964) has argued that large changes in size usually precede the compensatory changes in shape that mechanical analysis would predict. The large and rapid fluctuations in size that mark the adaptation of many vertebrates to Pleistocene cycles of glacial advance and retreat often occurred without change of shape (Kurtén 1968; Stephenson 1963).

2. It can produce an evolutionary improvement in shape. This statement is enigmatic. How can no change of shape be an improvement in shape? It can because the maintenance of shape at increased size does not yield an array of forms that are mechanically similar; mechanical similarity requires allometric growth (Gould 1966b). Static α-values for brain-body plots usually lie near .66 (Jerison, in press). If we take this α-value as the criterion of mechanical similarity, then large animals functioning as well as smaller ones will have relatively smaller brains. Now, if geometric similarity ($\alpha = 1$) is maintained during the phylogenetic size increase of a lineage, the large descendant will harbor a brain far larger than that predicted on mechanical grounds (size increase at $\alpha = .66$). So we end with what the Pirate King called "a most ingenious paradox": that phylogenetic improvement in function can arise from an evolution in size that includes no change of shape.

VI. SUMMARY

The coefficient b of the power function $y = bx^{\alpha}$ has long been misinterpreted as a measure of size-independent differences between regressions. Just the opposite is true; b is a scale factor that expresses differences in

size between comparable animals of the same shape on two or more regressions of constant α. When α is invariant for two regressions, a similarity criterion s can be extracted from the two b-values ($s = [b_1/b_2]^{1/(1-\alpha)}$); s measures the relative difference in size at which animals on the two curves have the same shape. If this calculated difference equals the observed difference in size, then the transposition (shift of regression line without change of slope) occurred in order to maintain geometric similarity in a new size range.

I present examples of geometric similarity via transposition for body shape in gulls, brain weight in felids and primates, tooth shape in canids, skull form in bovids, the evolution of *Gryphaea*, the growth of horses, and differences between local races of lobsters and molluscs.

The literature on dwarfism in humans and animals shows that proportioned change in size can have a simple genetic and developmental basis. As a mode of size change, geometric similarity may be important in macroevolution because: (1) it allows size to change rapidly by uncoupling the usual correlation of growth and development and (2) it can produce a phylogenetic increase in effective organ size when the expected correlation of that organ with body size is negatively allometric.

LITERATURE CITED

Adé, B. 1954. Le nanisme racial: essai d'interprétation des facteurs constitutifs de la morphologie du Pygmée africain. Arch. suisses anthropol. gén. 19:1–18.

Andersen, H. 1966. The influence of hormones on human development, p. 184–221. *In* F. Falkner [ed.], Human development. Saunders, Philadelphia.

Anthony, R. 1938. Essai de recherche d'une expression anatomique approximative du degré d'organisation cérébrale autre que le poids de l'encéphale comparé au poids du corps. Bull. mém. soc. anthropol. Paris 9. 8:17–67.

Bährens, D. 1960. Über den Formwandel des Mustelidenschädels. Morphol. Jahrb. 101: 279–369.

Bauchot, R., and H. Stephan. 1964. Le poids encéphalique chez les insectivores malagaches. Acta Zool. 45:63–75.

Bohlken, H. 1964. Vergleichende Untersuchungen an den Schädeln wilder und domestizierter Rinder. Z. wiss. Zool. 170:324–418.

———. 1967. Beitrag zur Systematik der rezenten Formen der Gattung *Bison* H. Smith 1827. Z. zool. Syst. Evolutionsforsch. 1:54–110.

Brummelkamp, R. 1940. Brainweight and bodysize (a study of the cephalization problem). Verhandelingen ned. Akad. Wetensch., vol. 39, no. 5. 57 p.

Burnaby, T. P. 1965. Reversed coiling trend in *Gryphaea arcuata*. Geol. J. 4:257–278.

Cock, A. G. 1966. Genetical aspects of metrical growth and form in animals. Quart. Rev. Biol. 41:131–190.

Crary, D. D., and P. B. Sawin. 1949. Morphogenetic studies in the rabbit. VI. Genetic factors influencing the ossification patterns of the limbs. Genetics 34:508–523.

Dawson, T. J., and A. S. Hulbert. 1969. Standard energy metabolism of marsupials. Nature 221:383.

DeBeer, G. 1958. Embryos and ancestors. Clarendon, Oxford. 197 p.

Dubois, E. 1897. Ueber die Abhängigkeit des Hirngewichtes von der Körpergrösse bei den Säugetieren. Arch. Anthropol. 25:1–28.

———. 1922. Phylogenetic and ontogenetic increase of the volume of the brain in Vertebrata. Proc. Koninklijke Akad. Wetensch. Amsterdam, vol. 25.

———. 1928. The law of the necessary phylogenetic perfection of the psychoencephalon. Proc. Koninklijke Akad. Wetensch. Amsterdam, vol. 31.

Dupertius, C. Wesley. 1945. The size and proportions of adult midgets. Amer. J. Phys. Anthropol. 3:111–125.

Falkner, F. 1966. Human development. Saunders, Philadelphia. 644 p.

Fischer, E. 1950. Über die Entstehung der Pygmäen. Z. Morphol. Anthropol. 42:149–167.

———. 1954. Die genetische Seite des Pygmäenproblems. Mitteilungen Anthropol. Ges. Wien 83:107–118.

Frick, H. 1961. Allometrische Untersuchungen an inneren Organen von Säugetieren als Beitrag zur ''neuen Systematik.'' Z. Säugetierkunde 26:138–142.

Gates, R. Ruggles. 1958. The African pygmies. Acta Genet. Med. Gemmellol. 7:159–218.

———. 1961. The Melanesian dwarf tribe of Aiome, New Guinea. Acta Genet. Med. Gemmellol. 10:277–311.

Glick, S. M., J. Roth, R. S. Yalow, and S. A. Berson. 1965. The regulation of growth hormone secretion. Recent Progress Hormone Res. 21:241–270.

Gould, S. J. 1966a. Allometry in Pleistocene land snails from Bermuda: the influence of size upon shape. J. Paleontol. 40:1131–1141.

———. 1966b. Allometry and size in ontogeny and phylogeny. Biol. Rev. Cambridge Phil. Soc. 41:587–640.

———. 1970. Evolutionary paleontology and the science of form. Earth-Sci. Rev. 6:77–119.

———. Allometric fallacies and the evolution of *Gryphaea*. J. Paleontol. (in press).

Gusinde, M. 1955. Pygmies and pygmoids: Twides of tropical Africa. Anthropol. Quart. 28:3–61.

———. 1956. The Yupa Indians in western Venezuela. Amer. Phil. Soc., Proc. 100:197–219.

Hallam, A. 1959. On the supposed evolution of *Gryphaea* in the Lias. Geol. Mag. 96:99–108.

———. 1968. Morphology, palaeoecology, and evolution of the genus *Gryphaea* in the British Lias. Roy. Soc. (London), Phil. Trans., B. 254:91–128.

Hohenegger, M. 1954. Der Zwergwuchs bei Ituri-Pygmäen und Negrito. Mitteilungen Anthropol. Ges. Wien 83:123–128.

Huxley, J. S. 1932. Problems of relative growth. Methuen, London. 276 p.

———. 1950. Relative growth and form transformation. Roy. Soc. (London), Proc., B. 137:465–469.

Jerison, H. J. 1955. Brain to body ratios and the evolution of intelligence. Science 121:447–449.

———. 1961. Quantitative analysis of evolution of the brain in mammals. Science 133:1012–1014.

———. 1971. Brain evolution: new light on old principles. Science (in press).

Johnson, S. L., G. S. Harshfied, and W. McCone. 1950. Dwarfism: an hereditary defect in beef cattle. J. Hered. 41:177–181.

Kleiber, M. 1961. The fire of life. Wiley, New York. 454 p.

Klemmt, L. 1960. Quantitative Untersuchungen an *Apodemus sylvaticus* (Linnaeus 1758). Zool. Anzeiger 165:249–275.

Korner, A. 1965. Growth hormone control of biosynthesis of protein and ribonucleic acid. Recent Progress Hormone Res. 21:205–236.

Kurtén, B. 1954. Observations on allometry in mammalian dentitions: its interpretation and evolutionary significance. Acta Zool. Fennica 85:1–13.

———. 1955. Contribution to the history of a mutation during 1,000,000 years. Evolution 9:107–118.

———. 1968. Pleistocene mammals of Europe. Aldine, Chicago. 317 p.

Lapicque, L. 1907a. Tableau général des poids somatique et encéphalique dans les espèces animales. Bull. mém. soc. anthropol. Paris 8:248–262.

———. 1907b. Le poids encéphalique en fonction du poids corporel entre individus d'une même espèce. Bull. mém. soc. anthropol. Paris 8:313–345.

Lasiewski, R. C., and W. R. Dawson. 1967. A re-examination of the relation between standard metabolic rate and body weight in birds. Condor 69:13–23.

McKusick, V. A. 1955. Primordial dwarfism and ectopia lentis. Amer. J. Human Genet. 7:189–198.

McKusick, V. A., R. Eldridge, J. A. Hostetler, U. Ruanguit, and J. A. Egeland. 1965. Dwarfism in the Amish. Bull. Johns Hopkins Hosp. 116:285–326.

McKusick, V. A., and D. L. Rimoin. 1967. General Tom Thumb and other midgets. Sci. Amer. 217:102–110.

Manouvrier, L. 1885. Sur l'interprétation de la quantité dans l'encéphale et dans le cerveau en particulier. Mém. soc. anthropol. Paris, 2d ser., vol. 3.

Mayrat, A. 1966. Les variations des taux d'allométrie et des indices d'origine dans une série, et leurs relations supposées. Compt. rend. soc. biol. 160:470–471.

Mead, S. W., P. W. Gregory, and W. M. Regan. 1942. Proportionate dwarfism in Jersey cows. J. Hered. 33:411–416.

Meunier, K. 1959a. Die Allometrie des Vogelflügels. Z. wiss. Zool. 161:444–482.

———. 1959b. Die Grössenabhängigkeit der Körperform bei Vögeln. Z. wiss. Zool. 162:328–355.

Mitra, K. C. 1958. Variation in *Goniorhynchia boueti* from Normandy and Dorset. J. Paleontol. 32:992–1006.

Needham, J. 1942. Biochemistry and morphogenesis. Cambridge Univ. Press. Cambridge. 785 p.

Newell, N. D. 1949. Phyletic size increase, an important trend illustrated by fossil invertebrates. Evolution 3:103–124.

Nomura, E. 1926. Further studies on the applicability of $a = kb^x$ in expressing the growth relations in molluscan shells. Sci. Rep. Tohoku Imp. Univ. Biol. 2:63–84.

———. 1928. On the local variation in some littoral gastropods. Sci. Rep. Tohoku Imp. Univ. Biol. 3:249–258.

Oboussier, H., and H. Schliemann. 1967. Hirn-Körpergewichtsbeziehungen bei Boviden. Mitteilungen Hamburg Zool. Mus. Inst. 64:464–471.

Pirlot, P. 1969. L'exposant phylogénique de Snell-Dubois chez les chiroptères. Biométrie-praximétrie 10:22–37.

Reyment, R. A. 1961. Quadrivariate principal component analysis of *Globigerina yeguaensis*. Stockholm Contrib. Geol. 8:17–26.

———. 1966. Studies on Nigerian Upper Cretaceous and Lower Tertiary ostracoda. 3. Stratigraphical, paleoecological, and biometrical conclusions. Stockholm Contrib. Geol. 14:1–151.

Rimoin, D. L., T. J. Merimee, D. Rabinowitz, V. A. McKusick, and L. L. Cavalli-Sforza. 1968. Growth hormone in African pygmies. Lancet 1:596.

Röhrs, M. 1958. Allometrische Studien in ihrer Bedeutung für Evolutionsforschung und Systematik. Zool. Anzeiger 160:276–294.

Schadé, J. P. 1959. Differential growth of nerve cells in cerebral cortex. Growth 23: 159–168.

Schlaginhaufen, O. 1959. Mitteilungen über einen Riesen. Bull. Schweiz. Ges. Anthropol. Ethnol. 35:23–37.

Scholl, D. A. 1948. The quantitative investigation of the vertebrate brain and the applicability of allometric formulae to its study. Roy. Soc. (London), Proc., B. 135: 243–258.

Sinnott, E. W., and L. C. Dunn. 1935. The effect of genes on the development of size and form. Biol. Rev. 10:123–151.

Smith, P. E., and E. C. MacDowell. 1930. An hereditary anterior-pituitary deficiency in the mouse. Anat. Rec. 46:249–257.

Snell, G. O. 1929. Dwarf, a new Mendelian recessive character in the house mouse. Proc. Nat. Acad. Sci. 15:733–734.

Snell, O. 1891. Das Gewicht des Gehirnes und des Hirnmantels der Säugetiere in Beziehung zu deren geistigen Fähigkeiten. Sitzungsberichte Ges. Morphol. Physiol. München 7:90–94.

Stephens, F. E. 1943. An achondroplastic mutation and the nature of its inheritance. J. Hered. 34:229–235.

Stephenson, N. G. 1963. Growth gradients among fossil monotremes and marsupials. Palaeontology 6:615–624.

Stevenson, A. C. 1957. Achondroplasia: an account of the condition in Northern Ireland. Amer. J. Human Genet. 9:81–91.

Stork, H.-J. 1968. Morphologische Untersuchungen an Drosseln: eine Analyse von Anpassungsstrukturen im Körperbau von sechs europäischen Arten der Gattung *Turdus* L. Z. wiss. Zool. 178:73–185.

Szarski, H. 1964. The structure of respiratory organs in relation to body size in Amphibia. Evolution 18:118–126.

Teissier, G. 1936. Croissance comparée des formes locales d'une même espèce. Mém. Mus. roy. d'hist. natur. Belg. 3:627–634.

Thompson, D'Arcy. 1942. On growth and form. Cambridge Univ. Press, Cambridge. 1,116 p.

Walton, A., and J. Hammond. 1938. The maternal effects on growth and conformation in Shire horse–Shetland pony crosses. Roy. Soc. (London), Proc. 125:311–335.

Weninger, M. [ed.]. 1954. Gedanken zum Problem des Zwergwuchses. Mitteilungen Anthropol. Ges. Wien 83:97–130.

White, J. F., and S. J. Gould. 1965. Interpretation of the coefficient in the allometric equation. Amer. Natur. 99:5–18.

Wirz, K. 1950. Zur quantitativen Bestimmung der Rangordnung bei Säugetieren. Acta Anat. 9:134–196.

Zar, J. H. 1968. Calculation and miscalculation of the allometric equation as a model in biological data. Bioscience 18:1118–1120.

Zehner, I. 1967. Über den Einfluss veränderter Umwelt (Freigehege) auf das Herzgewicht der Albinomaus. Zool. Anzeiger 178:1–17.

Zollitsch, H. 1969. Metrische Untersuchungen an Schädeln adulter Wildwölfe und Goldschakale. Zool. Anzeiger 182:153–182.