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Review Article

THE EVOLUTION OF THE PRIMATE BRAIN: SOME ASPECTS OF QUANTITATIVE RELATIONS

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INTRODUCTION AND SCOPE

Two aspects of neurology have particular significance for the anthropologist's area of study: palaeoneurology and comparative neurology. The former studies the changes in structure as they appear in the fossil record, while the latter attempts to better understand the nature of extant forms through study of structure and function. The former is necessarily limited to surface features. While this distinction has been stressed a number of times by Edinger[58-60], it is apparent that the two aspects are not exclusive of one another but are necessary complements in following the evolution of behavior. A study of extant brains of monkeys, apes, and men is not a direct study of the evolution of man, but of the end result of three lines of evolution in the primate order. They do not chronicle the actual evolutionary changes of the organ in any one group. Given a fossil record of a line of evolution where the number of remains is great, such as in the horse family, a study such as Edinger's[58] indicates the solid position which the palaeoneurologist can take. It is no secret that such a magnificent array of material does not exist for the primates. Aside from this limitation one other deserves particular emphasis. The study of primate endocasts, particularly in the great apes and hominids, gives exceedingly little information about evolutionary changes and permits little basis for suggesting hypotheses concerning the events of man's evolution. Not only are the visible markings on the endocasts extremely variable in both relief and form, but their relationship to variables of behavior allow only for the most molar correlations. The extreme variability of cranial capacity is well appreciated, and its correlation with any sort of behavioral attribute is notoriously low. Brilliance may be exhibited by men whose cranial capacity differs as much as 1000-1100 ml, a magnitude which is as much or more than the known increase in this parameter from say, *Australopithecus* to modern *Homo sapiens*, *i.e.*, the whole of hominid evolution. The most obvious draw-back to the study of fossil endocasts is that it tells one nothing about the internal structure of the nervous system, and a reliance on the one parameter of cranial capacity passes over many aspects of re-

organization of the total brain. It will become obvious in this review that any such comparison of forms on this basis is essentially a comparison of unequal units at a variety of levels. While it is definitely true that the amount of brain increased and was attended by increasing behavioral adaptability with respect to such rubrics as 'intelligence', 'foresight', 'memory', etc., it is to be emphasized that reorganization along sensory, motor, and integrative modes were also taking place. These are not likely to be understood from the study of endocranial casts. For this appreciation, a study of extant forms bearing some degree of similitude to our models of primate evolution must be undertaken.

The main theme of this paper should now be apparent. It is the study of reorganization of the primate brain during evolution. Ideally, a study of the evidence for reorganization would involve analysing all lines of evidence which relate to the total life-ways of the organisms under consideration. Changes in sensory and motor modalities; the changes in higher functions often referred to as 'intelligence' (memory, foresight, insight, complexity of operations that are intrinsic), the relation of the 'emotional sphere' to the total interaction of the organism with its environment, social and physical, external and internal, would also be studied. Such an endeavor would not only be a study of the total brain but would also include all aspects of its behavior from both a field and laboratory viewpoint. Clearly, such a study is matter for a lifetime, if not many.

This review will deal primarily with the reorganization of the cerebral cortex, that part of the brain which has been the focus of so much attention in attempting to understand the nature of man as well as his evolution. A review of the data will be presented to indicate that reorganization has taken place at several levels, and that comparisons of mass give little understanding of those aspects of hominid behavior that have been subject to natural selection.

More specifically, data relating to quantitative aspects of reorganization will be examined. The first section of this paper will examine basic considerations of cerebral mass, and indicate the limits of this approach. In addition, a brief examination of reorganization of the subcortical components of the primate brain will be given. This is undertaken to indicate that shifts in sensorimotor behavior patterns have been attended by shifts in the subcortical structures involved.

The second section, which forms the main body of this review, is devoted to analyses of the structure of the cerebral cortex. This section is intended to make as salient as possible the notion of reorganization at a series of molar and molecular levels. It will show the directions of reorganization, and provide data that support the assertion that a comparison of mass is not a comparison of equal units.

To facilitate discussion, three main areas of reorganization are recognized. The aim of the first section, as stated above, will consider brain mass and the limitations of this approach. The second section on reorganization will treat two aspects of behavior as if they were clearly separate, analytically. The first aspect will relate to what is regarded as difference of degree but not kind, and the second with that which is regarded as specifically human, *i.e.*, qualitatively different. This conceptualization has the danger of being arbitrary, but has been chosen as a device or artifice to facilitate

discussion, definition of problems, and provide a framework for further conceptual sharpening and experimentation.

It is impossible to examine the totality of information regarding neurological structure and behavior. The treatment to be provided in the following pages is therefore purposefully selective and oriented toward quantitative aspects of reorganization. Such selectivity precludes any general theory of man's place in nature, or a detailed theory of hominid evolution. In fact, little attempt is made to *prove* any particular assertion or theory.

This point needs more explicit statement since it is often overlooked that a valid portion of scientific method is criticism, examination of logical relata, and erection of hypotheses to both organize data and suggest lines of research. It is admitted early in this paper that the hypotheses and suggestions made are speculative, and that the neurological evidence for them is very sparse. Care has been exercised, however, to make explicit the assumptions upon which the hypotheses are built. This is absolutely necessary in view of the fact that the evidence for behavioral evolution is essentially indirect.

Before proceeding to the presentation of the evidence for reorganization, it would be well to state clearly the assumptions to be held regarding the nature of the evidence and the problems examined. (1) It is assumed that no general theory of man's nature or his evolution can be based on the study of the nervous system alone, nor on the rest of his morphology. Primates interact in social environments, and the structure of the individual animals and the social environments have changed in time, and are both constraints upon one another in any description of a total system. (2) It is assumed, however, that the nervous system does bear some relation to the total range of the organism's activity, both extrinsically and intrinsically, and that furthermore, certain structures or configurations of structures have clearer relationships to certain behaviors than others. (3) That reorganization of subsystems of nervous structure have some degree of correlation with subsystems within a total behavioral matrix. It is not the purpose of this paper to describe these in any exact sense. (4) It will be assumed that in primate evolution three broad stages were realized, corresponding in very broad terms to the levels of organization shown by extant monkeys, apes, and man. (5) It is further assumed that while it is true that extant brains are the results of separate lines of evolution in monkeys, apes and man, they correspond closely enough in general structure to those of past forms to allow their use in elucidating reorganization of structure from a catarrhine monkey, to pongid, to man. (6) It is assumed that the 'mind-body' problem is a pseudo-problem, the result of symbol manipulation, and that 'mind' is the activity of the brain. It is assumed that primates have no immaterial souls, and that there is no need to call upon vitalistic explanations.

EVIDENCE FOR REORGANIZATION

General

This section will present evidence that the brain as a whole has changed through

time, and that these changes have been in conjunction with the changes of life-ways among the primates. Such an exposition, although brief, will help to give some perspective to what is meant by reorganization, and will serve as a useful setting for the later evidence regarding the cerebral cortex. First, however, the basic issue of mass will be explored.

Conceptually, the idea of reorganization in the primate brain during evolution is not original to this paper. Implicit in the studies of such workers as Campbell, Brodmann, Economo, the Vogt's, Kleist and others connected with the elucidation of cytoarchitectonics was the idea of increasing differentiation and addition of functions, mirrored by changes in cortical pattern. The series of charts and tables in Tilney's[179] two volumes bear evidence of his realization of reorganization, particularly at the subcortical level. G. E. Smith's emphasis on the frontal lobe and on the predominance of the role of vision in primate evolution gives further testimony to such an appreciation. One might mention as well the popular notion of 'corticalization' ('telencephalization') or the increasing dependence of function on the cerebral cortex. This concept was certainly present by Gall's time, and was elaborated by Hughlings Jackson, by Fulton and Keller[67] with respect to the sign of Babinski, by Dusser De Barenne[50] on the effects of decorticalization in different primates, and recently affirmed by Noback and Moscovitz[126]. On the other hand, many studies have tended to emphasize so strongly the mass aspects of the brain in terms of milliliters, that reorganization principles have received more in the way of lip service than analysis. The numerous writings of Eugene DuBois, in which ideas of brain-body weight relationships were expanded and formalized to indicate natural laws of dependence, have led to many contributions in which the search for parameters for mass additions of neurons, either in steps of 2 (DuBois) or $\sqrt{2}$ (Brummelkamp) have been argued, pro and con. (See DuBois[17,18]; Brummelkamp[27]; Bok[7]; Bonin[8]; Count[37]; Sholl[162]. This is not an exhaustive list.) In addition, various relationships have been established in the primate order, *e.g.*, Harmon's[83, 85] work showing that the relationship between brain weight and cortex weight is linear when plotted on a log-log graph. Bonin[12] used Brodmann's[24] data to show that the amount of frontal lobe in the primates is proportional to brain weight in a log-log graph with a slope of 1.0. Jerison[98] has shown further that the main increase in cortex has involved areas other than the pre- and postcentral sensorimotor areas, and that 'association' cortex has increased strikingly with respect to mass. The number of workers which accept this emphasis on increase of 'association' area, that is without direct specific thalamic afferents (see Pribram[132,133]), is too large to register here. (This matter of 'association' area increase will be returned to later in the review, particularly in relation to Hebb's[91] interesting 'A S' ratio, and its increase in evolution.)

Certainly the expansion of the parietal and temporal cortex is obvious when comparing gross brains of monkey, ape and man. Most recently, these mathematical relationships between body size, brain weight and cortical volume, have led Jerison[98] to extrapolations of cortical neuron numbers in both extant apes and certain fossil hominids. Tobias[180] has even used Jerison's figures to support certain taxonomic conclusions about the earliest hominid discoveries from Africa. While Jerison's[98]

figures and method will be treated at greater length elsewhere in this review, it should be noted that his figures do not agree with empirical counts made by Shariff[161]. Jerison's mathematical extrapolations rest upon assumptions of cortical volume and neuronal density, and overlook the fact that in primate evolution, the cortex has undergone reorganization. Cobb[34] has recently reviewed the general question of brain size in absolute and relative terms for a variety of animals, but relates behavior at the molar level of 'intelligence' to these crude parameters, although he recognizes a relative increase in 'association area' during primate evolution.

It is not the point of this review to argue any of these relationships here, since many unquestionably hold. Rather, I have given a sampling of certain works which one might argue has tended to emphasize mass relations rather than the significance of restructuring or reorganization. This preoccupation led to the use of these mass aspects as explanations *in themselves* of behavioral differences of quite specific natures, such as 'memory', 'insight', 'forethought', 'symbolization' etc. It should be obvious that such correlations are not causal analyses, and that a parameter such as brain weight in grams, or volume in ml, or area in sq. mm, cannot explain the differences in behavior which are observed. On the other hand, it is futile to overlook the fact that mass increase has occurred during evolution, and that it dovetails nicely within the framework of natural selection and adaptation. A more general and encompassing theory, however, must synthesize the increase in mass with that of reorganization.

Another aspect of the ease with which cathection on brain mass increase takes hold, is the explanation of qualitative differences on the basis of quantitative addition. The most facile expression of this type of transformation from mass increase with respect to the brain and behavior is to be seen in the principle of the cerebral 'rubicon' (Keith[102]; Vallois[185]; and Krantz[106], for example. For criticism, see Dart[42]; and Straus[176]). Here, man's symbolic behavior is taken as a qualitatively different way of interacting with the total environment, with some arbitrary magnitude of mass representing the threshold above which human behavior emerges. There is no really effective argument against this reasoning, since it cannot be disproved. What can be argued, however, is that such an explanation is again a correlation and not a causal analysis. For some level of behavior chosen, there will always be some minimum amount of tissue necessary. The 'rubicon' does not explain the transform, as how it operates, but is instead an assertion of probability. Cases do exist in which the value taken as the rubicon will not be obtained, and yet human behavior is possible (Dart[42]). Or the converse may be cited, where the value is exceeded in an individual and yet nothing other than sheer idiocy is evident. In all fairness to Keith, it should be pointed out that he had in mind a population mean of 750 ml as the 'rubicon' and probably would not assess the status of any individual from its cranial capacity or brain weight. This matter of variability, however, is particularly high among the higher primates (Hagedoorn[77]), and can almost be considered a sort of antithesis which comes out of, and eventually undermines, the strength of the parameter of which it is a part. Again, from the viewpoint of the present framework of genetic theory, such a dependence on mass aspects leads to but one deduction: that the only shift of biochemical constituents of the genes controlling the brain over the last ten or so million years has

involved the speed and duration of mitotic division, perhaps at different sites in the nervous system. It is certainly true that no elements of *kind* are known to exist in man's brain that do not exist in other primates (outside of the arcuate nucleus, perhaps: see Bruce[26]), and that there has undoubtedly been conservatism in genetic entities controlling brain development. Such problems, however, have so little data available that there is no point in attempting further discussion of these considerations. Let it suffice to state the logical deduction from the postulate of mass increase, and note that natural selection is selection ultimately for biochemical constituents and their relationships. It will be argued later that while the units which make up the brain, such as neurons, glia and transmitter substances, are essentially the same, they are not identical in all primates and that these differences must be incorporated *with* notions of mass increase to effect an appreciation of reorganization. (By 'identical', I do not mean biochemically, necessarily, but instead the proportions of components and the complexity of interrelations.) While there is no evidence at present to indicate that the biochemistry differs among primates in relation to such variables as thresholds for excitation, or differences in permeability to the flow of Na^+, K^+, Cl^-, or Ca^{2+} ions through the membranes, or even in nucleotide bases in the nuclear RNA, it hardly seems improbable. However, until such evidence is present it seems best to ignore them throughout the remainder of this paper. Bonin[15] has taken a similar position.

I have given a preliminary treatment of the problem of increase of brain mass here in order to show why my emphasis will rather be placed on reorganization. Later, ideas on brain mass and behavior will be examined in greater detail, in relation to specific ideas on brain evolution which are in the literature. Some examples of reorganization now follow.

Total reorganization — the example of microcephaly

Perhaps one of the most convincing arguments in favor of broad reorganization is provided by a study of microcephaly. In several cases it has been shown that patients with a total brain volume within the upper range of pongid values are capable of behavior patterns that are human rather than pongid, although these patients are usually classed as idiots. Therefore, a brief examination of certain aspects of this condition seems in order, in view of the emphasis placed on the conception of cerebral mass and behavioral capacity. As far as can be ascertained, Dart[42] is the only worker who gives any discussion along these lines, and this from the viewpoint of criticism of a 'cerebral rubicon', rather than emphasis on reorganization. Perhaps the pathological nature of this condition, as well as the limited number of cases in which any speech is involved (von Bonin, personal communication), has not induced the anthropologist to give the condition much consideration. While it must be emphasized that the incidence of this condition is rare, roughly one out of 250,000 (Book *et al.*[18]; Brandon *et al.*[22]; Cowie[38]; Koch[103]; Konai *et al.*[104]), the fact remains that very small brain size within pongid limits permits behavior patterns that in no sense can be viewed as pongid (Yakovlev[195]). This generalization holds, furthermore, whether the carriers are capable of speech or not. While a thorough study of these cases is pro-

bably warranted, it must suffice here to mention but briefly a few cases so that a basis for emphasis on reorganization will be apparent. Of particular interest are those cases usually referred to as 'microcephaly vera', a condition considered attributable to a single recessive gene, in which there is usually no evidence of other brain defects beside microdevelopment of the cerebral hemispheres. Other types, such as 'spuria', may involve secondary complications with the basal ganglia, or cerebellum, or peculiar islands of gray matter may appear in the diencephalon. (See Bailey *et al.*[3]; Brandon *et al.*[22]; Cowie[38]; Greenfield and Wolfsohn[76]; Minkowski[120]; Tredgold[182], for particulars and problems of classification.) In these instances other than microcephaly 'vera', either *postmortem* examination or careful analysis of receptor and motor functioning will usually ascertain secondary involvement.

One of the best descriptions of a case of microcephaly from the behavioral aspect was that given by Korsakov in 1893, recently translated and reprinted in a current journal (Korsakov[105]). The patient was a female named 'Masha', who could speak a great deal, but with little connected content. There was no question that she understood a limited number of commands and questions, and was capable of remembering and discussing things that occurred early in her life, although context seemed senseless. There were, nevertheless, some verbal exchanges that were meaningful. She could distinguish objects and name them, although Korsakov noted an 'extreme poverty of logical associations'. The almost mechanical aspects of her 'intelligence' are brought home with the following example: food on the table stimulated M. to eat, and if the food was removed in the middle of this activity, M. would act as if the meal were over, saying thank you, and would bless herself. If the plate were returned she would start eating again, and follow the same pattern if the sequence were repeated. Apparently this behavior was invariable with several repeats.

Hughes and Grieves[96] have reported a case of three microcephalic brothers in a Bantu tribe, possessing speech capabilities. J. M., 18 years old, had an estimated cranial capacity of 561 ml; T. M., 14 years old, 517 ml; H. M., who was 8 years old, a cranial capacity of 511 ml. These authors described the motor action as in some cases coarse, sluggish, and in the case of H. M., sluggish 'but well coordinated'. There was no question but that intelligence was subnormal.

Keith[101] described a sheep herder who died at 60, after many years of earning wages. The brain of the subject, Joe, weighed 560 g at death. The convolutional pattern, according to Cunningham (quoted in Keith[101]), was simpler than that of a chimpanzee. 'He had command of a considerable number of words and could frame sentences ... his face clearly reflected every emotion in a perfectly human fashion ...'.

Dru-Drury[46] described an adult Bantu woman whose cranial capacity was 340 ml, who was quite capable of doing routine work in an institution, and could make use of a limited number of words.

Tredgold[182] gives some other examples which need not be reported here. His general statement is of interest, however:

'The mental features common to most microcephalics are the absence of sensory defect, a general vivacity, restlessness and muscular activity, a considerable capacity for imitation and, usually, an inability for sustained effort. In their perceptive faculties

these persons often compare favorable with aments of considerably higher intelligence and many of them not only have remarkably good hearing and sight, but extremely quick powers of observation . . . Their power of mimicry is often very marked and this combined with their general alertness, causes them to be amongst the drollest inmates of the imbecile ward.'

Lennenberg[112], based on Seckel's[160] monograph on nannocephalics (pinheaded dwarfs), has shown that these people can converse freely with cranial capacities less than 700 ml. The microcephalic vera cases are somewhat more interesting in that body size is not dwarfed; only the brain is underdeveloped, and yet a number of these examples have shown speech behavior and a repertoire of affective behavior patterns that are typically human.

A capacity of 685 ml has for some time been taken as an upper limit to pongid brain capacity (Schlaginhaufen[158]; Vallois[185]). Recently, Schultz[159] recorded a case of a gorilla having a cranial capacity of 753 ml. Weidenreich[190], Straus[176] and Dart[42] have been adamant in denying a 'rubicon' for attainment to *sapiens* status, and have stressed 'quality' of brain as the more essential aspect than quantity.

These examples of microcephaly make salient the fact that something in the way of a human specificity exists, even when the brain is deficient. With regard to the particular specificity, very little in the way of histological examination has been undertaken, and the data are not exact enough to determine the bases for reorganization. We do not know whether human behavior relies on changes that are in the line of structural differences such as cell density, dendritic branching parameters, glial/neuron ratios, blood supply, expanded association cortex, different metabolic patterns, or an emergent sort of organization resulting from the reorganization of the total brain. This is presently unanswerable. Those who would suggest that the number of nerve cells, once it reaches a certain value, allows for human behavior, must deal with the problem of microcephaly. Here, in addition to loss of nerve cells, a large number of those remaining are morphologically immature (Hammarberg[82]). Since cell counts do not exist for microcephalics or nannocephalics, it is not possible to know whether they have as many neurons as do chimpanzees. It is best to leave this interesting subject for the time, since it will be taken up briefly again in another section, and proceed to other aspects of reorganization.

Subcortical reorganization in primates

The above discussion pointed out that the microcephalic, despite his low brain size, manifests a total behavioral pattern which is human and not pongid. To emphasize that reorganization in the primate brain has been a total process, several aspects of reorganization at the subcortical level will be given.

Tilney's volume, *The Brain from Ape to Man*[179] (see Tilney[178] for a shorter treatment), will be of use since it provides quantitative data relating to subcortical structures in the primates. In addition, this volume formulates Tilney's views relating neurological structure to function within an evolutionary framework. For the present, Tilney's ideas regarding the frontal lobe, or the interpretation of hominid behaviors

based on the ranking of endocranial casts, will be ignored.

That Tilney was particularly impressed with the changes in subcortical organization relative to motor activity is very clear in his conception of 'neokinesis'. Tilney was convinced that the evolution of the human foot, *i.e.*, its specialization, was necessary for the establishment of a higher level of manual differentiation with respect to 'neokinetic' activity in man of the hand.

'Neokinesis' was, in Tilney's view, the development of elaborate, individuated activity related to simultaneous coordination of hand, head and eyes. While the label is perhaps new, and even useful, Smith[169] had earlier stressed somewhat the same aspects, particularly with respect to hand-eye coordination. Likewise, Tilney stressed 'corticalization' or 'telencephalization' with respect to this conception of function.

To illustrate his claims, Tilney used a series of planimetric indices of nervous structures in extant primate forms, and attempted to relate the quantitative differences of the structures to functional behavioral differences. Table I gives a summary of Tilney's measurements. The figures present ratios of the areas of structures to the total cross sectional area taken at various levels throughout the neuraxis. Unfortunately, no data are given regarding the number of specimens measured, and no data exist regarding species variability. This places great limits, of course, on interpreting the quantitative differences with respect to function, but should serve as a beginning guide for further analysis (Table I).

If those structures concerned with the elaboration of fine movements and coordination, that is 'neokinesis', are considered, the increase with ascending phylogenetic status is evident. For example, consider the values in the tables for the following structures: the pyramids, largely made up of corticospinal tracts; the pontile nuclei, concerned with cerebro- (frontal, parietal, temporal) ponto-cerebellar path-

TABLE I

PLANIMETRIC INDICES OF NERVOUS STRUCTURES (TILNEY[170])

	M	*G*	*C*	*O*	*Gi*	*B*	*Ma*	*L*	*T*
Pyramids	0.183	0.161	0.172	0.160	0.138	0.143	0.147	0.110	0.032
Pontile nucleus	0.550	0.480	0.400	0.300	0.200	0.164	0.150	0.055	0.057
Cerebral peduncules	0.321	0.187	0.223	0.110	0.110	0.190	0.169	0.086	0.017
Inferior olive	0.226	0.186	0.174	0.172	0.155	0.125	0.128	0.060	0.042
Dentate nucleus	0.176	0.152	0.136	0.160	0.134	0.165	0.155	0.110	0.059
Red nucleus	0.128	0.096	0.086	0.087	0.051	0.060	0.057	0.012	0.034
Sup. cerebellar peduncules	0.088	0.047	0.047	0.064	0.063	0.044	0.046	0.033	0.032
Infer. colliculi	0.070	0.111	0.132	0.131	0.130	0.155	0.175	0.223	0.337
Sup. colliculi	0.104	0.140	0.125	0.124	0.132	0.123	0.158	0.140	0.230
Burdach's nucleus (cuneate)	0.100	0.081	0.073	0.093	0.068	0.065	0.086	0.049	0.029

Key: M man, G gorilla, C chimp, O orang, Gi gibbon, B baboon, Ma macaque, L lemur, T tarsius.

ways and expansion of the lateral lobes of the cerebellum; the dentate nucleus of the cerebellum; the red nucleus as a way-station for impulses from the cerebellum; the superior cerebellar peduncles containing tracts from (at least in part) the red nucleus; the inferior olivary nucleus, with impulses from the cerebellum, cerebrum, basal ganglia, spinal cord and reticular formation and their correlation. The figures suggest the increasing importance of these structures for higher forms. It is interesting that the olivary nucleus has many folds, and is very distinct in section. Kappers *et al.*[100] suggested that the folding followed the same dynamics involved in the cortex. This development goes along with the development of the dentate nucleus in the cerebellum. The spino-cerebellar fibers mediate tactile and proprioceptive impulses to the inferior olive, and these in turn give rise to the olivo-cerebellar fibers to the cerebellum. Other confirmatory evidence on the increasing fineness of tactile appreciation and control as well as the proprioceptive side, comes from the work of the Scheibels' histological analysis of the inferior olive. Following an idea expressed by Bonin[13], on increasing interneuronal distance allowing for greater degrees of freedom, the Scheibels[151] found a similar trend in the inferior olive, noting that heavy afferent fibers might serve to fractionate the large groups of neurons which would otherwise have a high probability of firing synchronously in large numbers. Parenthetically, it can be added that severity of damage with respect to motor activities following cortical ablations advances with phylogenetic position (corticalization of function). This relates to expansion and distinctness of the above subcortical components and their relationship to the cerebral cortex.

Along with this planimetric increase in structures relating to the general notion of 'neokinesis', the values given for the inferior and superior colliculi decrease ties in again with the notion of increasing 'corticalization of function'. Tilney, as well as Smith, was also impressed with the role of vision in the primates and its importance in evolution. Using longitudinal cross-sectional area of the oculomotor decussation, Tilney found the following indices, which are expressed in terms of percent of area covered by decussating fibers: man 90%, gorilla 88%, chimpanzee 86.5%, orang 71%, gibbon 66%, baboon 79%, macaque 71%, lemur 16%. This convincingly shows that the decussation of the oculomotor nucleus progresses with phylogenetic ascent and is in line with the views of more complete conjugate movements and association of the eyes.

Of interest also is the increasing value for the cuneate nucleus, indicating increased input from the upper extremities, particularly in those forms where manipulation of the distal extremities is advanced. The values for the gracile nucleus do not appear as systematic. Tilney sums up these various indices as follows:

'In its brain each one of the primates below man has, in its turn, borne the impress of the effort to advance the cause of neokinesis. For this reason, if for no other reason, each infrahuman primate gradation is important in the evolutionary picture.' (p. 1043).

In connection with the idea of elaboration and increasing fineness of movement and adjustment, some recent work of Kuypers'[107] is relevant. Kuypers found that in monkeys (rhesus), cortical fibers to the spinal trigeminal were predominantly postcentral projections, whereas the precentral gyrus gave projections primarily to

the lateral tegmentum and motor nuclei. In the chimpanzee, however, the precentral gyrus projects almost entirely to motor nuclei, and it was also observed that the direct cortico-nuclear projections were noticeably higher in the chimpanzee, and even more massive in man, while the projections to the lateral tegmentum decreased.

Yet another aspect of these differences of motor activity may be gained through a brief treatment of some of the data regarding the extrapyramidal system (basal ganglia, corpus striatum). These deep gray nuclei of the telencephalon include the globus pallidum, putamen, caudate nucleus, claustrum, substantia innominata with the associated sublenticular gray, and the amygdaloid complex. The functional aspects of this system, especially in terms of its own interconnections as well as intra-connections with the cortex, thalamus, reticular system, and cerebellum, are far too complex for any review here. (For extended reviews, the reader is referred to Crosby *et al.*[39]; Jung and Hassler[99].) These structures, the amygdaloid possibly excepted, may be regarded as participating in the integration and regulation of motor activity. Jung and Hassler[99] note that patients whose basal ganglia are affected by disease or experimental lesions, '. . . are characterized by one or more of the following: an excess of spontaneous, aimless, and unintentional movements, a lack of associated and synergistic movements, a persistent increase in muscle tone but with no spastic pareses, and absence of essential changes in the reflexes'.

These same authors note the interesting fact, for this theme of reorganization, that there is usually a 'marked divergence between the symptomatology of human extrapyramidal disorders and those experimentally produced in animals . . .' (p. 919). Parkinson's disease is apparently specific to man (although it can be initiated in monkeys with lesions to the extrapyramidal system), and Jung and Hassler assume this to be the result of the 'more differentiated and delicate balance of human motor functions, subserving manual manipulation and regulating upright gait . . . (making them) more liable to disorders resulting in involuntary movements' (p. 919). This last note is of particular interest, for these authors stress that erect posture must be correlated with more elaborate mechanisms of regulation to work against gravity. This point will be returned to later when considering the Australopithecine brain and its capacities (behaviorally) in relation to tool-making and bipedalism.

Quantitative data for the extrapyramidal system are not as dramatic in showing the reorganizational aspects as those from clinical and behavioral studies. Bonin and Shariff[17], and Harmon and Carpenter[86] have provided some data for primates (see also Feremutsch[62]). The authors found that the total volume of the basal ganglia was proportional to the total brain volume, with, however, some differences in the caudate-putamen complex with increasing phylogenetic position. The following table, adapted from their data, illustrates this point. It should be kept in mind, however, that the range of variation is not known, and while these figures are suggestive, the differences noted here may not be significant (Table II).

Bonin and Shariff's[17] data are somewhat different from those of Harmon's and Carpenter's, due in part to the use of different methods of volume calculation and methods of preparation. These are included in Table II and stress the increase of the pallidum with the primates. In supplementation to Harmon and Carpenter,

TABLE II

QUANTITATIVE DATA FOR THE EXTRAPYRAMIDAL SYSTEM (HARMON AND CARPENTER[86]; BONIN AND SHARIFF[17])

	Percent volume of basal ganglia			*Percent volume of striatum*		*Percent of total extrapyramidal system*	
	Caudate	*Putamen*	*Pallidum*	*Caudate*	*Putamen*	*Zona incerta*	*Substantia nigra*
Harmon and Carpenter							
Galago	50.6	38.9	10.5	56.6	43.4		
Macaque	35.6	48.3	16.1	42.3	47.7		
Cebus	32.2	56.0	11.8	36.6	63.4		
Chimpanzee	33.9	56.2	9.9	37.6	62.4		
Man	35.3	44.9	19.8	44.1	55.9		
Bonin and Shariff							
Opossum	66	29.2	5.3	70	30	8.4	7.1
Rat	93		7	–	–	10	5
Tarsius	51.0	36.0	13.0	58.7	41.3	2.9	6.6
Macaque	36.5	46.0	17.5	44.2	55.8	1.1	3.6
Chimpanzee	32.6	53.4	14.9	37.4	62.6	1.1	4.3
Man	38.7	46.4	14.9	45.5	54.5	0.45	5.3

their data show a significant reduction of the zona incerta, particularly for man. Again, however, nothing is given with respect to variation. Talairach *et al.*[177] (quoted in Bonin and Shariff) gave figures showing that the human thalamus varied from 8.82 to 15.5 ml, and Lashley[109] clearly warned that variations in the structures of the brain were high. Thus, the figures in the above table should not be overemphasized. However, it seems safe to conclude that there is reorganization of a broad sort. While Bonin and Shariff view the extrapyramidal system as a 'servomechanism for the cortical control of the final common pathway', it is difficult to carry the analysis given with respect to component reorganization to any sort of microlevel in terms of differing input output feedback relationships for the interspecific differences noted above. Quantitative data at this level of analysis are a necessary requisite for more complete treatments regarding species differences in behavior.

In line with the present emphasis on reorganization and increased fineness of motor control, data presented on the cerebellar nuclei by Solnitzky[171] are of interest. Using planimetric methods to determine relative volumes of the dentate, fastigial, emboliform and globosal nuclei in the primate cerebellum, the following was noted: in lemuroids, the four nuclei are nearly equal, each varying from 20 to 28% of the total nuclear volume. In the higher primates, volume was consistently greater in the dentate and least in the globosal nuclei. The following figures are given in terms of the percent of total cerebellar nuclei taken up by the dentate nucleus: man 75%, chimp 69%, spider monkey 63%, baboon 47% and cercopithecus 34%. The distinct lamination of the dentate was evident in only two species: man and chimpanzee. The decrease in emboliform, globosal and fastigial nuclei correlates with the decrease in importance of equilibratory impulses for orientation.

Another set of interesting correlations has been provided by DuBrul[19]. Working

on the principle that decrease in muscle size was attended by decrease in neural apparatus, DuBrul investigated the mesencephalic nucleus of the fifth cranial nerve in a series of animals. His interest centered on the number of nerve cells related to proprioceptive control of the jaw muscles, since these have strikingly diminished in the course of human evolution. It was DuBrul's conviction that a reduction of cells of this nucleus marked a loss of control on final common pathways such that other influences could have more effect; a sort of competition for space on cell bodies of the final common path. The release of jaws from the task of prehension is tied-in with the increasing dependence of the forelimbs for feeding and tool-making, attended by dependence on the hind limbs for locomotion. His counts of nerve cells in this nucleus gave the following: baboon, 6116; chimpanzee 4800; man 2900. These clearly indicate the progressive decrease in number of cells in the mesencephalic nucleus in correlation with the reduction of muscle mass and grasping functions of the jaws.

DuBrul feels that the shift in adaptive behavior was from the function of jaws in prehension to that of speech. Accepting the notion that a single event entering the brain results in myriad pulse patterns spreading through the brain, as well as reverberating in self-stimulating chains, he views redundancy as having selective value, since it allows for greater chance for innovation in behavior (p. 55).

DuBrul's formulation is essentially the one maintained in this paper. However, the invoking of the 'final common path' at such a frankly economic level abstract is a distraction. Here, DuBrul uses the model of a single motor neuron as the final common path, but tells nothing about its locus, its grouping, and the set of final common pathways attending the particular phylogenetic change. The shift in one component would mean shifts in the level of functions regarding the brain as a system, or innumerable final common pathways. Nevertheless the conceptualization is valuable in making salient the shift of subsystems within an evolutionary framework.

Evidence has thus far been presented for reorganization of the primate brain with respect to motor and sensory systems, where input–output relationships are more evident than those connected with what might be labeled as 'affective' or 'emotional'. It will be of interest to note briefly some data relevant to this category of behavior.

Daitz[41] undertook some measurements of the fiber contents of the fornix system in investigating certain aspects of the 'limbic' system. His analysis showed that a number of fibers of the fornix in the human brain had increased roughly to five times that of the macaque. There were 2,700,000 fibers in the human subcallosal fornix on each side. In comparison to this number, 500,000 in the macaque on each side were estimated. In the hypothalamic part, there were 912,000 fibers for the human, in the macaque, 1,000,000.

Powell *et al.*[131] compared the number of fibers of the fornix just anterior to the mammillary bodies to the cell number in the medial mammillary nucleus. This ratio was about 1 : 2 in the rat; 1 : 1 in the rabbit, cat and monkey; and 2 : 1 in man.

Unfortunately, no figures are available for pongids, so nothing can be said concerning the differences between them and man. Nevertheless, these figures show that systems of neural interaction in relation to 'emotional' behavior have shifted during the course of primate evolution. Interpretation of the significance of the shift

is very difficult, since not only are data lacking which relate to other anatomical ingredients of the 'limbic system', but also the interactions and interrelations within this system, and its relations to other systems. Brady[21], in a recent review of emotional behavior, after a lengthy section covering the work on effects of ablation and stimulation throughout the hypothalamus, concludes that: '. . . primitively organized, relatively undifferentiated patterns of emotional behavior may be elaborated within limited reticular and hypothalamic levels of neural organization'.

Andy and Stephan[1] (see also Stephan and Andy[175a]) have provided quantitative data on the septal nuclei of the primates. They showed that while the percentage of septal volume to total brain volume decreases with phylogenesis, there is an allometric increase in septal volume in absolute terms. In terms of percentage of total brain volume taken up by the septal nuclei, the following were given: insectivores, 1.85%; the tree-shrew, *Tupaia*, 1.08%; cercopithecus, 0.40%; chimpanzee, 0.22%; man, 0.1%.

Heiner[92] provided a review of differences in the diencephalic nuclei in macaque, chimpanzee and man. While no quantitative data were given, his summation is of interest in relation to this theme of reorganization. Heiner[92] noted the following differences: in the hypothalamus, both the tuberis lateralis and tuberis mammillaris increase with phylogenetic ascent. In the thalamus, there was a decrease of the anterior dorsalis nucleus; the centralis lateralis was not as distinct in the chimpanzee as it was in man and macaque; the lateralis dorsomedialis was more differentiated in man; the lateralis dorsalis was the same relative size in macaque, chimpanzee and man; the parafascicularis was larger in the chimpanzee than in the macaque, but small in man.

In general, it can be said that the more complex the nuclear configurations of subcortical centers, the less quantitative data there are. This is particularly true for all the structures subserving emotional attributes of behavior, *i.e.*, the limbic lobe, portions of the thalamus, septum and hypothalamus. In terms of the latter structure, measurements given by Grünthal[76a] related to an index formed by dividing hypothalamic length by cerebrum length. As expected on the basis of the increase of cortex in phylogenesis, the index decreases from monkey to man. Grünthal's figures are as follows: monkeys and gibbon, 0.11 0.12; orangutan and gorilla, 0.104 0.109; chimpanzee and man, 0.08 0.09. Naturally, such figures tell one nothing about possible reorganization of differential nuclear changes within the hypothalamus for different species. Grünthal states that in the hypothalamus and thalamus, the structures are less complex in man than in the apes and that the number of nuclei decreases. This view was criticized in LeGros Clark's[31a] study. Clark found that, in man, there were more extensive distributions of large cells, particularly in the lateral hypothalamic area, more differentiation of the tuberal nucleus, and a diminution of size of the lateral mammillary nucleus, in comparison to apes.

The dorsomedial and anterior nuclei of the thalamus are well known for their mediation of emotional behavior under stimulation. The anterior nuclei have intimate relationship to neocortical, palaeocortical and juxtallocortical systems. Papez[125] formulated a mechanism of emotion which has since borne much fruit for experimental analysis, and stands today as a major framework for ordering data relative to emotional behavior. Papez theorized that emotive processes of cortical origin would be built

up in the hippocampus, hippocampal gyrus, dentate gyrus, and amygdala. This would reach the mammillary bodies by way of the fornix. From these centers, efferents both descend and ascend, the former to lower motor mechanisms at the spinal level, the latter ascending through the mammillo-thalamic tracts to the anterior thalamic nuclei, and then to the cingulate cortex. This basically represents the Papez scheme, and has been altered little since (Green[74]). It is interesting that in connection with the quantitative data presented earlier, Rothfield and Harmon[143] found that placidity resulted from neocortical ablation if the limbic system was not interfered with. They further demonstrated that intercepting the fornix fibers would *lower* rage thresholds in their decorticate preparations. This was found also by Spiegel *et al.*[175] and Green and Arduini[73] observed a sort of hyperactivity in rabbits with fornix sectioning, with a tendency to attack the observer. However, it should be stressed that this is apparently not a constant occurrence. Green[74] lists several studies where no behavioral change was noted after fornix section. They suggest that since the olfactory sense is markedly reduced in the human, the great increase in fornix fibers is related to increase of fibers from expanded neocortical areas.

The very great complexity of the limbic system and the controversial evidence regarding its functioning strongly limit any discussion of the quantitative data, outside of speculation. It may well be that the great increase signifies nothing, or perhaps it relates to enhanced cortical control of emotional behavior, that is, lower and higher thresholds to rage or aggression. Chance[28] has discussed certain of these limbic structures in different primate social groupings.

Summary

Before proceeding to a discussion of the changes in the cortex, it would be well to summarize the above variations and their implications for reorganization. As the data have shown, a consideration of the weights of primate brains for comparative purposes is a comparison of unequal units. The entire brain has undergone changes in directions dependent upon the life-ways of the animals. These differences in reorganization are not reflected in the endocranial casts. The great increase in cerebral cortex through phylogeny has been paralleled by a series of reorganizations in other systems, particularly in the motor sphere. *Pari passu* with these developments, it should be expected that changes in the locomotor musculo-skeletal system would also change, and certainly the fossil record bears some witness to these changes.

Consequently, it seems reasonable to assert that while particular anatomical entities subserve the same functions, quantitative changes in the entities result in changes of the functioning whole. This occurs by a raising and lowering of thresholds for probable interactions with other systems, and or by bringing more or less discriminative control to bear on the major motor activities of the organism. While this section stressing subcortical reorganization is far from being complete either in the sense of dealing with analysis of function or in describing the total anatomy of the brains of various primates, it has aided in underlining the fact that several systems have undergone reorganization toward increased fineness of motor activity. The control and operation of 'emotion' is very complex, and even if quantitative data were

available on the entire 'limbic system', for example, the lack of knowledge of interactions with respect to functioning would make the mere presentation of such quantities almost useless. In this respect it is doubly unfortunate that quantitative (see Feremutsch[62]) data for the thalamus are not available since this system, along with the expansion of the cortex, has undergone change also (Clark and Boggon[32]). The tie-in of these systems with the reticular formation is one attracting much attention; in terms of activation, synchronization, and maintenance of consciousness, as well as control of afferent information, and offers many interesting challenges to those interested in the evolution of man. An analysis of the dynamics of system changes, in terms of altering quantitative relations between excitatory and inhibitory relationships, such as Ashby's[1a] analysis, is impossible for primate evolution, based on present evidence. Enough has been given to suggest a direction which one might turn in the future. Quantification is but an early step.

Cortical reorganization

As the introduction attempted to make clear, mass increase as expressed in terms of cranial capacity provides a minimal means for understanding the relationship between neural structure and behavior within the primate order. The sections relating to subcortical structures and microcephaly have indicated that reorganization at a number of levels is masked when only cranial capacity is considered. This section will extend the analysis of reorganization to the cerebral cortex at a variety of levels.

Due to the complexity of this region of the nervous system and the enormous amount of literature dealing with its anatomy and functioning, it would be impossible to attempt a complete discussion of all aspects of the cerebral cortex. Clearly, some constraints must be placed on the problem. A discussion of cytoarchitectonics would be a thesis in itself; the same could be said for the problem of localization of function. In addition, the anatomical evidence regarding the structure of the primate cerebral cortex is hardly complete, nor is there any actual consensus regarding the nature of the behavioral differences between man and the apes. Symbolic behavior (at least on the extrinsic level—Hallowell[78, 80]; Munn[122]) offers an operational definition of one important aspect of man's behavior, but it is not clear how this relates to current knowledge of comparative neurology. Whether or not it is an emergent property involving a new behavioral process both intrinsically and extrinsically, is an unanswered question. Later in this review assumptions regarding this aspect of human behavior will be considered more extensively, and following a brief analysis of various conceptions regarding the role of the cortex in this behavior, a tentative explorative hypothesis will be offered. For purposes of discussion, the analyses will proceed as if the cortex alone were involved. This approach is admittedly artificial, but is necessitated at this stage of understanding regarding the role subcortical mechanisms play in behavior. The brain functions as a total system, and it is an assumption of this paper that a better understanding of human behavior must eventually result from a systems analysis. A basis for this type of analysis would depend upon a clearer understanding of what quantitative changes have taken place in components such as the

amygdala, hypothalamus, hippocampus, cingulate cortex, specific and non-specific reticular formations, etc. For the present, this paper admits that explanation at several levels is feasible, but will forego the systems approach in favor of a more molecular approach.

Three broad aspects of reorganization of the primate cortex will be examined. Two of these will deal with the notion of complexity of behavior, and will provide evidence for reorganization at two levels: a molar level, based on shifting areal patterns of types of cortex, and a molecular level, dealing with neurohistological reorganization. The third aspect will involve some suggestions regarding the relation of neurohistological structure to extrinsic symbolization, or that which is regarded as an emergent property of man's behavior. By complexity of behavior, this paper refers to those behavioral operations which are continuous with those known in other animals. This concept includes such items as learning-to-learn phenomena, the number of stimulus cues dealt with in discriminative behavior and problem-solving tasks in general. In other words, those behavioral capacities in which man may be superior, but which are matters of degree rather than kind, or quantitative increase with continuity of operation. Again, this approach is somewhat artificial in that it segregates functions which operate as a unit, but without some conceptual distinctions it would be difficult to discuss the several aspects of neuroanatomical evidence in a functional manner. (For an interesting review of this problem see Geertz[70].) It is here viewed as a prelude to a more unified approach in the future. Before proceeding with these levels of analysis, it will be useful to first examine some current ideas regarding neural structure in man's behavior.

Aside from views which regarded the acquisition of a soul as the unique qualitative distinction between man and other animals, most explanations of the past and present have relied on quantitative distinctions to explain man's behavior. In view of the knowledge that man's brain does not contain any different structures from that of an ape or a monkey (Bonin[10,15]), this approach would seem a basic starting line. Whatever qualitative differences there are, they are to be explained on the basis of emergence from the interaction of components in a total system. This does not exclude the possibility that differences in the genotype allow for the new metabolic processes in man that do not exist in the lower primates. However, no direct data exist to substantiate this possibility, and it seems best to ignore it for the present, maintaining an openness to the possibility.

In general, three sorts of explanations which have been offered will be examined. They are: (1) cytoarchitectonic differentiation, (2) relatively large frontal lobe and association areas, (3) addition of great numbers of neurons.

(1) Man's cortex is more finely differentiated cytoarchitectonically, and therefore capable of unique and complex behaviors

This position is perhaps most familiar through the numerous publications of Brodmann, Beck, Campbell, Kleist, the Vogts, and others of the cytoarchitectonic school. The criticisms of Lashley and Clark[110] and the failure of the attempts of Bailey and Bonin[3], Bonin[13], Bonin and Bailey[16] to find clearly demarcated cyto-

architectonic patterns in the cortex, have led to a re-assessment of this old problem. Current opinion views man's cerebral cortex as predominantly homogeneous in structure, rather than greatly differentiated regionally (Bonin and Bailey[16]). This does not assume, however, that all cortical tissue has the same function, as Lashley[108] had earlier thought to be the case. (Here it should be understood that the pre- and postcentral gyri, the striate cortex and that of the superior temporal gyrus concerned with audition are not included. Instead, this view of Lashley's was concerned with the 'intrinsic' association cortex as conceptualized by Rose and Woolsey[140]; Pribram[132,133].) That man has more cortex is beyond doubt, but the older architectonic views which presupposed an increased 'organology' in the case of man are no longer held today. Such views as Bonin's[13] regarding the 'forecasting' function of association cortex, or Pribram's[132-134] views regarding anterior and posterior association cortex with discriminative and ordering functions respectively, have superseded the older views mentioned above and will be treated in more detail in a later section.

(2) The 'hallmark' of man is his relatively large frontal lobe, and/or association cortex

From the early part of the 18th century, two main schools of thought with respect to localization of function have tended to dominate investigations on brain functioning. Gall[69] held to an extreme localization of 'higher mental functions', whereas Flourens (1824; see Boring[18a], pp. 62-67) took the opposite view of total cerebral participation. This paper is not concerned with detailing the genesis of thought regarding this issue or the interplay of interpretations resulting from clinical and experimental studies. (Reviews of these aspects may be found in Meyer[118]; and Willett[191].) Instead, this section will limit discussion to those data purporting to show the relative increase of these areas in the case of man, and the supposition that man's unique behavior somehow depends on these relative increases.

Exactly when the anterior cerebrum became a focus of interest is unclear. Certainly by the time of Gall and Spurzheim, this division of the cerebrum was favored for explaining human behavior. Perhaps the vertical disposition of man's forehead has been a factor since Antiquity, and the degree to which it slopes more backwardly in other primates has led to the concept. The number of neurologists and anthropologists who have asserted the prominence of the frontal lobes in man is far too numerous to list here. (The history of thought on this subject up to the 1920's has been reviewed by Bianchi[6], another frontal lobe devotee.) Most recently, LeGros Clark[31], Cobb[31] and Vallois[186] have asserted that this aspect of cerebral anatomy is one of the main factors in differentiating human from ape behavior. Several authors (Brodmann[21], Boule and Anthony[19], and Weil[191]) have attempted to provide comparative quantitative data on this cerebral division within the primate order. The last two named authors have even attempted to follow its development through hominid evolution by measuring endocasts. Tilney[179] and Schepers[155-157] have carried this line of analysis to extreme lengths, attempting to explain behavior in the Pleistocene and its selection on the basis of area measurements and arc lengths measured on pieces of endocasts or their reconstructions.

Thus Boule and Anthony obtained values of 43.3% of total surface for man,

32.2% in the apes, and 35% for Neandertal Man. These values are still being quoted by LeGros Clark and Vallois (see above). Tilney[179] arrived at the following results: man 47%, gorilla 32%, chimpanzee 33%, baboon 31%. Brodmann[24] presented a series of measurements of the relative surface area of the regio frontalis of the frontal lobe (excluding areas 4 and 6) for a series of primates: man 29%, chimpanzee 16.9%, macaque 11.3%. Unfortunately, Brodmann never published his cytoarchitectonic results for man, and his maps were based on work done on the monkey, transferred to man on the basis of homology. In view of the lack of any evidence for clear-cut cytoarchitectonic differences for subregions of the frontal lobe, the problem of variability and the difficulty of assuming homologies, it seems wise to await further measurements rather than accept these as final. It is of interest that Bonin[12] took Brodmann's figures of surface area of the frontal lobe and plotted these log-log against total surface area. His result was an essentially straight line with a slope of 45°, indicating no relative increase. In fact, the point for man was somewhat below the line. Bonin[15], in agreement with Weidenreich[189], feels that the parietal lobe instead has shown the most relative increase, but no figures are given. Brodmann also pointed this out, but stressed the frontal lobes.

Weil[191], employing a number of base lines, divided the projections of endocasts into a series of triangles and rectangles, measuring these for a number of different primates, including fossil man. Not surprisingly, his results emphasized a great increase in the frontal lobe also. As Weidenreich[189] pointed out, his figures are meaningless since they give only partial areas for the frontal lobe, and are not consistently oriented on the endocasts. This is compounded by the fact that the fissure of Rolando falls in the middle of one of his areas, thus eliminating different portions of the frontal lobe for different casts. Other workers have utilized tape measurements of the distance from the Rolandic fissure to the frontal pole, both at the apex and where the fissure meets the temporal lobe. Cunningham[40], and particularly Connolly[36] have favored this approach. When indices are formed by relating these distances to total cerebral length, differences of the order of 1 out of 50 units appear between apes and man. It is difficult to accept these as indicating significant differences.

Using more direct methods, Brummelkamp[27] attempted to reproduce some of the above figures, using a planimetric projection from cortical slices. His ratios appear as follows: man 1 : 3.79 or 25.4%; chimpanzee 1 : 3.61 or 27.8%; macaque 1 : 3.79 or 26.4%. This method overcomes the problem of arbitrary landmarks, and is probably more accurate. Earlier, Brodmann[24] had used the tissue paper method of covering the cortex and arrived at the following set of figures for the percent of total frontal lobe area: man 33.7%, a microcephalic 34.6%; chimpanzee 30.6%. Using a precipitation method, Leboucq[111] found: chimpanzee 37.5%; orangutan 36.6%; man 36.9%. The differences in absolute values are probably due to different methods. What is of particular interest here, however, is the degree of species difference. The last named authors, using the dry-weight method, provided data regarding the relative volume of the frontal lobes for a few species. Their figures are as follows: newborn infant 1 : 2.53 or 39.6%; adult man 1 : 2.47 or 40.4%; microcephalic 1 : 2.51 or 39.8%; orangutan 1 : 2.45 or 40.7%; chimpanzee, 1 : 2.62 or 38.2%.

The above figures should provide a good basis for doubting the commonly held view that man's frontal lobes have shown a relative increase. Those measurements which are the most direct, that directly measure cortical area or volume rather than endocasts, give little basis for accepting relative increase. Whether or not the figures given by Brodmann for the regio frontalis (and thereby of more interest since they deal with association cortex) are valid, needs further study. However, assuming for the moment that a relative increase in frontal association cortex did take place in the case of man, it still remains unclear as to how this would account for man's behavior in contrast to the ape's, unless it relates to increased complexity rather than something unique. While the clinical aspects of frontal ablations or leucotomies are far from clear (Hebb[90]; Meyer[118]), it cannot be said that humans undergoing these procedures suddenly become simian.

A better case may be made for relative increase of parietal and temporal association cortex in the case of man. Weidenreich[189] pointed this out, but did not provide any figures for this increase. While these increases undoubtedly help to enrich the possibilities of more complex behavior in the human case, it would be somewhat of a retreat to an 'organology' to assert that man's behavior in the unique sense, depends on these masses of tissue. Whether the functioning of this tissue is described as 'forecasting' (Bonin[13]) or in more modern terms, such as Pribram's[132, 134] 'search for information' or 'test-operate' (posterior and anterior intrinsic association cortex, respectively) is not germane to the level of discussion taken here. Hebb's[91] analysis of the slowing down of primary learning with concomitant retention, based on an increased association to sensory projection cortex (the A/S ratio), assumes that additional units enable emergent functioning. Herrick[93] attempted an explanation of human behavior on the basis of association cortex in much the same manner as Hebb, but the functioning he described would appear to be elements which are continuous among the primates. This does not mean that explanations using these morphological units are incapable of explaining human behavior. These explanations provide a major share of explaining the richness of human behavior in terms of complexity of operation. For the present, they do not explain, in their present state, the transform of behavior between man and ape. The next section will attempt to make this observation somewhat clearer.

(3) Man's behavior is an epiphenomenon or emergent, resulting from the addition of great numbers of neurons

Of the three hypotheses discussed, this third requires careful analysis, since it holds forth the most promise as an explanation. In view of the fourfold difference in weight between a chimpanzee's and man's brain, it is a common assumption that man's brain contains at least four times as many neurons. (See Wilkinson[193] for explicit statement. The number of workers who hold to this assumption are too large to reference here. For explicit statement regarding the reduction of ape human differences to a matter of neuron number, see Gerard[70a], p. 361.)

There is evidence to suggest that neuron improvement, better connections and better circuitry has taken place in the course of mammalian evolution. The difficulty

with using additional units as a total explanation resides in its inability to deal with whatever transformations have taken place in behavior. To discount this explanation as holding possibilities, however, would be unnecessarily negative, and it will later be shown that by using an intervening variable, it is possible to relate the numbers of neurons to behavioral phenomena. For the present, this section will explore further the consequences of this position toward understanding 'new dimensions of richness in performance'.

In this connection, it is interesting to recall an early paper by Pitts and McCulloch[117] which suggested calculating the number of possible states with a population of ten billion neurons, each being either on or off. The number was two multiplied by itself ten billion times, or $2^{10^{10}}$. This number refers, of course, only to a double-state neuron, one utilizing the all-or-none model of axon impulses. With subthreshold influences from many dendritic and somatic synapses on each neuron, the number of actual states would be far greater. It will be instructive to return to this figure later, noting for the present the order of possibilities existing. There is little point in arguing the main essentials of Gerard's point, since man surely possesses more units and his behavior is most complex. The essential problem is how far this explanation goes toward dealing with something like symbolic behavior at the extrinsic level, or arbitrary representative factors. That is, how does it explain man's uniqueness?

Rosenblatt[141] has attempted to unite neurological and psychological concepts by basing the latter on a probabilistic calculus. He demonstrated that alteration of connections between three series of neurons (receptor, associative and responsor, the 'perceptron')[6] with varying degrees of cross connections and feedbacks, was able to account for several psychological constructs: discriminating classes of patterns; discriminating arbitrary classes of stimulus patterns or stimulus sequences; solution of generalization patterns; and selective attention to certain stimulus classes were the constructs chosen. His models were based on known histological structure in the cortex, including recurrent axons, axon collaterals, and known physiological properties of the neuron. The complexity of the psychological processes increased step by step with the types of additional connections made, particularly cross connections and feedbacks, and depended on the quantitative arrangements between numerous neurons. The application of cybernetic concepts combined with logical network schemes is fast becoming a field in its own right, and the above example far from exhausts the amount of work going into a common language to describe psychological and neurological functioning. The main reason for including this brief sketch of one example of this trend is to indicate that these attempts have so far not differentiated human from subhuman behavior except at the level of complexity of operation. Part of the reason for this probably lies in the fact that the histological models used to calculate probable psychological processes are certainly common to all primates if not all mammals. Lorente de Nó's[113,114] work based on Golgi-stained materials from the mouse cortex shows that cross connections and feedbacks of the type discussed above exist at this level. Nor is there anything from the psychological constructs mentioned above that could not be found in chimpanzees and perhaps macaques. (For specific tests relating to the above constructs see Harlow[81]; Nissen[123,124];

TABLE III

RATIOS OF CEREBRAL PARAMETERS (FROM SHARIFF[161])

	Brain weight	*Cortex volume*	*No. cortical neurons*
Man/chimp	3.84	2.75	1.25
Chimp/macaque	3.90	4.20	2.20
Man/macaque	15.00	11.60	2.76

and Munn[122].) While it would be dogmatic to assert that these models will not explain symbolic behavior, they so far show most promise in offering a better understanding of increasing complexity of operation, and are similar to Pribram's notions referred to above. It is advantageous to present here the evidence regarding the reality of the large cerebrum which man possesses, and compare it with other primate brains with respect to number of units.

Using a value of 90 g for the macaque brain, and 350 for the chimpanzee, man's brain 1350 g is roughly 15 times as heavy as the macaque's and about 4 times that of the chimpanzee. (See Bonin and Bailey[16].) Using Shariff's[161] values for cortex volumes, the ratios are as follows: man/chimp 2.75; chimp/macaque 4.2; man/macaque 11.6. Taking cell counts throughout the iso-cortex of these primates, Shariff calculated the number of neurons based on neural densities and volume of cortex. Figures are given for both hemispheres in billions– man, 6.9; chimp 5.5; macaque 2.5. The ratios are summarized in Table III.

Actually, as Sholl[166] has pointed out, Shariff's value for the volume of human cortex is somewhat low, but it is safe to conclude that man does not have 4 times as many neurons as the chimpanzee*.

Recalling Pitt's and McCulloch's[117] earlier figures of $2^{10^{10}}$, what is the difference between $2^{6.9 \cdot 10^9}$ and $2^{5.5 \cdot 10^9}$? Does this infinitesimal difference between two infinities decide the difference in behavior between the two forms? $1.5 \cdot 10^9$ neurons is a considerable quantity, and $2^{1.5 \cdot 10^9}$ is perhaps an infinity of its own right, but it is disappointing to find that man has but 1.25 more neurons in his cortex than his lowly cousin. The essential point, however, is the question that such an analysis raises regarding the relation of these macronumbers to actual behavioral processes. One cannot avoid considering differences in neuron number and their related possible states between the famous Anatole France with 1100 ml and Turgeneev with 2200 ml, or the fact that many millions or perhaps billions of neurons are lost in human subjects undergoing radical ablations. (See Brody[25] for data relating to neuron loss with advancing age. Up to a third are lost.)

In this connection it is of interest to consider some aspects of ontogenetic development in the primate order. In early postnatal growth, man's brain almost doubles from 380 to 630 g, and then doubles once more to about 1330 g at age four

* *Note added in proof*: Pakkenberg (*J. comp. Neurol.*, 128 (1966) 17–20) has found that figure in human cortex is $2.6 \cdot 10^9$ neurons when shrinkage is taken into consideration.

(Magoun *et al.*[116]). Further growth is almost nil up to the second decade, amounting to about 120 g. Ape and monkey brains mature much faster. Consequently, there is a prolongation of dependence time for learning in the postnatal life of man, being almost double that of the apes. (Twenty years as against 11 for chimpanzee and 7 for macaque.)

Magoun *et al.*[116] (p. 10) note: 'Maturation of the brain's functional capacity is related more to the increase of its cytological than its gross complexity. These microscopic alterations, in which weight changes are minimal, involve the elaboration of dendritic processes ...; the formation of loop circuits and feedbacks modifying neural input and output, and the capacity, whatever its basis, by which previous activity in the nervous system can later be reinvoked, as memory, to merge with and influence current actions.'

It is tempting to view man's behavioral capacity as due to this lag of ontogenetic development, and to credit his abilities as the result of longer time of plasticity in neural maturation. It would be useless to deny that there must be some measure of truth in such an assumption, and it must not be forgotten that man is an open system, and that the interaction of brain structure with environment is likely to be highly important in the attainment of human abilities. But there would be difficulties in assuming that the above explanation is adequate to explain human behavior. All of the changes enumerated by Magoun *et al.* would be common to any primate cortex, and probably occur at lower phylogenetic levels also. The same histological basis for the actions described above are known for the mouse cortex (see Lorente de Nó above). It is not clear how either 1.25 more neurons or additional maturation time would explain the differences of behavior between man and ape. One point in the above quotation deserves particular emphasis. The growth of the brain postnatally is not a function of more neurons being added, but rather proliferation of processes. Whether the increase of 1000 ml can be explained as entirely due to ramification of dendritic processes is unclear and will be discussed in greater detail later. To what extent other changes such as glial proliferation or vascularization contribute is unknown. Considering the Hayes'[88,89] experience in attempting to raise a chimpanzee at home with their own child, it should be pointed out that the chimpanzee initially outstripped the child in performance, and this with a smaller brain, one with fewer neurons and supposedly fewer connections. The chimpanzee did not acquire words, but even at 6 months the child was *babbling*, and at 1 year or slightly later *could talk*. This argues for a particular kind of ontogenetic specificity differing between ape and man, and which probably involves factors other than neuron number, branching parameters, or the like. Perhaps increased dendritic processes do act to lower the threshold of motor neurons in the human precentral area, allowing for easier spontaneous firing. But this speculation, while interesting, says nothing about whatever possible maturational factors might be operating in the human case.

A brief examination of some aspects of microcephaly will again be pertinent. It is unfortunate that no quantitative work regarding the cerebral cortex is available with respect to neuron number or density and branching parameters, since these would offer a basis of comparison with other primates. A few references are of interest,

and these are best summarized by Benda[5] and Minkowski[119]. In the microcephalic there are large numbers of neurons in the cortex which are morphologically quite immature and which surely do not function as normal. The sketches given by Hammarberg[82] show this clearly. Fois[66] has shown that the EEG records of microcephalics indicate abnormal patterns of firing and synchronization. Although no density figures are available for the microcephalic cortex, it seems difficult to imagine that the value or range of variation would be far outside that for the normal human cortex, although some reports hint that density is greater. If this assumption is granted, then a similar density of cells combined with large numbers of morphologically immature neurons would suggest that the microcephalic might have less functional neurons than a healthy chimpanzee. It is clearly premature on the basis of actual data to assert this to be the case, but it is difficult to see how one might argue the difference between microcephalic and chimpanzee behavior as resting purely on the basis of neuron number. Nor does it seem likely that an expansion of intraparietal values will suffice as an explanation. Many microcephalics with a full complement of this tissue do not possess the faculty of speech, or extrinsic symbolization; yet their affective interactions with others and the environment have a sense of human specificity.

Two other aspects of this problem should be mentioned before concluding this introductory section on cortical reorganization. The first deals with the matter of chemical specificity. Weiss[192] and Sperry[173,174] have shown that different neurons have high degrees of specificity, and that the complex interaction of genotypes at the level of the gene and inducers argues that numerous events during ontogenesis are of importance in neural differentiation. While most studies on differing chemical and electrical sensitivities of neuronal populations have emphasized subcortical structures such as the hypothalamus, nucleus ruber, substantia nigra, etc. the known morphological types in the cortex must have some genetic basis for their different manifestations. Exactly what subtle biochemical differences differentiate stellate from pyramidal types in the neural ectoderm, or the several types of neuroglia, is unknown. It does show that a large number of genetic events take place during ontogenesis, and this argues for a large number of gene loci. Furthermore, if genius has any physiological basis, it would seem impossible to attribute this behavior to additional neurons or cortical tissue. Perhaps Donaldson's[15] suggestion regarding efficient nourishment of nerve cells is worthy of more consideration. There is some evidence to suggest that genetic influences are related to subtle biochemical differences in neuron metabolism. Strain differences in rats exist for biochemical variables where no difference in cortical morphology have yet been demonstrated, and selection for behavioral patterns in maze tests have resulted in strains differing with respect to biochemistry (Tryon[183]; Roderick[139]; see Rosenzweig *et al.*[141] for discussion).

While all this relates logically to our current models of genetic systems, the implications of this with respect to phylogenetic differences is seldom drawn. For the present it must suffice to recognize that neuron chemical specificity, genius, and strain differences, as well as genetic distance in phylogeny, all have a common axis in so far as metabolic processes depend on enzymatic events, which in turn are related

to gene interactions. While these problems have scarcely been touched as far as actual research is concerned, the implications of the logical dependence clearly relate to the problem of behavioral differences within the primate order.

A second aspect deserving attention is the problem of units. It is becoming clear from recent work using electron microscopic methods that the neuron, particularly its perikaryon, comprises but a small portion of cortical tissue (see Hydén[97], for review). Shariff[161] estimated that about 5% of the cortical volume was attributable to nerve cell bodies. Hydén[97] gives estimates of 25% for dendritic processes and about 5% for glial cells, not including their processes. Hydén's numerous works on glial-neuronal interaction during functioning, and Hild's and Tasaki's[94] observation of glial motility and electrical properties is rapidly leading to the view of the glia-neuron as the basic unit, rather than the neuron alone. Galambos'[68] speculation that the glia help lock-in neuronal pulses, perhaps in reverberating cycles, may soon be answered one way or the other. Some of this evidence will be returned to in greater detail later in this paper. For the time, it is sufficient to point out that the conception of what comprises units in neural functioning is changing. This provides an additional basis of support for the argument taken here that number of neurons alone is not sufficient to explain the behavioral differences between species. Other units and their study must be undertaken to understand the evolution of the behavioral differences within the primate order (Holloway[95]).

While this section has admittedly only skimmed the surface, it is hoped that the nature of the problem and some objections to current explanations have been made clearer. The tone of treatment in the past pages has been negative, and little in the way of positive alternatives brought forth. Any alternatives, however, must first be based on new conceptualizations of the problem, and this has required some treatment of past conceptions. From the preceding pages it seems safe to conclude that any explanation will have to involve a number of factors, and that no one particular morphological observation is likely to be satisfactory as an explanation of behavioral differences. A more eclectic approach seems clearly warranted. The concepts entertained in (2) and (3), which related to increased association cortex and increased units are starting points for more elaboration. Before proceeding directly to an attempt at synthesis, it will be convenient to summarize the principal findings of this introductory section.

(a) Gross size of the brain alone does not explain differences of behavior within the primate order. The variation is higher in some cases than the differences supposed to be critical for 'rubicon' models. The attempt is at best a statement of correlation, rather than causal analysis. This parameter overlooks the fact that equal volumes of cortices from different species, or even different individuals of the same species, are not a comparison of equal units.

(b) Cytoarchitectonic differentiation, while at one time a leading concept for explaining species differences is not in line with current evidence from neurohistology.

(c) Frontal lobe differences appear to be slight if they exist at all, within much of the primate order. Increased association cortex, particularly in the parietal and temporal lobes has taken place within the primate order. This fact has not yet provided

a causal explanation, but should be a useful starting point for discussing aspects of increased behavioral complexity in man, rather than emergent properties.

(d) The simple addition of more units, *e.g.* neurons, to explain emergent behavior processes in man is perhaps the most attractive and strongest explanation examined. The difficulty of explaining the nature of the transform still exists. Evidence from interprimate differences in neural units suggests that the differences are not as large as commonly assumed on the basis of cranial capacity or cortex volume. Evidence suggests that the neuron alone is not a profitable unit of study, and that it should be replaced by considerations of the functioning of the neuron-glia unit.

(e) A more eclectic approach should be entertained which will employ as starting points the molar conceptions noted in (c) and (d), and which relate more to problem of increased behavioral complexity rather than emergence.

NEUROLOGICAL EVIDENCE RELATED TO COMPLEXITY OF BEHAVIOR AND NEURAL INTERACTION

As outlined in the Introduction, this section will treat those aspects of the neuroanatomical evidence relating to complexity of behavior. As stated earlier, these behaviors are regarded as continuous, or of degree rather than kind, within most of the mammalian class. Certainly for the primates, it is difficult to assume that at each level the problem-solving processes are qualitatively different. This is not to assert that there is no problem relating to species-specific behavior within the primate order. The past section regarding 'representative factors' has already made clear the position of this review with respect to human behavior as an emergent, and will be taken up in greater detail in the second part of this review. This section, while examining complexity of behavior as if it were something separate from all other aspects of behavior discussed, does not assume that the separation is so clear-cut in reality. The view taken is indeed more holistic, and human behavior is viewed as the outcome of systems-operation, an outcome of dynamic interactions of components. To discuss the aspects of reorganization which have taken place, some analytic conceptualization is necessary as a first step. It is convenient to consider the aspects of reorganization at two levels: molar and molecular.

(1) Molar

This subsection will deal mainly with the quantitative evidence regarding the expansion in the primate brain, of that cortex usually referred to as 'association' cortex. The matter of definition with respect to function is not simple, and ideas regarding this subject have probably had an evolution of their own. Herrick[93] viewed this tissue as a pool of internuncial neurons awaiting input from the sensory projection areas of the postcentral gyrus, elaborating the sensations into ideas. This was achieved through a kind of discharge of reserve energy. This view has been more or less superseded by discoveries which have been the outcome of technological advances in stimulation and recording techniques. Earlier, for example, Lashley[108] had con-

cluded that these areas tended to be homogeneous with respect to function, and that behavioral deficit was a matter of the quantity of tissue ablated. This view contrasts with that of Pribram[133], Chow *et al.*[29], Geschwind[71,72]. More recently, evidence regarding the secondary sensorimotor functioning of these areas has been produced (see Crosby *et al.*[39] for review; Fleming and Crosby[64] and De Jonge and Crosby[43]), and are certain to reorient thinking with respect to the 'associative' functioning of this tissue. For the purpose of this review, it will suffice to define these areas anatomically and proceed to the evidence regarding their distribution in the primates. The most recent definition of 'association cortex' is that provided by Pribram[132,133], based on the older formulation by Rose and Woolsey[140]. In this account, association cortex will be defined as that cortex which has no direct afferent supply from the thalamus, but which is in feedback or loop-circuits with the intrinsic thalamic nuclei. Since the proportion of dorsal to ventral thalamus increases in mass and differentiation in higher vertebrates (see Heiner[92]), discussions are made on the basis of this distinction and its relation to types of input. An external and internal dichotomy is used with respect to dorsal thalamus. The external portion is made up of the ventral posterior (lateral and pulvinar) and the geniculate nuclei. The internal core of the thalamus is divided into anterior, medial and central groups. The external core, *i.e.*, the ventral and geniculate nuclei, receive large 'specific' fibers from somatic, gustatory, auditory and visual systems. In the internal core, the anterior nuclei receive input from the mammillo-thalamic tract (posterior hypothalamus). The central nuclei have as their input the non-specific afferents from the reticular formation and anteromedial hypothalamus. The posterior nuclear group of the external core, and the medial group of the internal core do not receive any major 'extrathalamic' input, and are viewed as 'intrinsic' nuclei. As noted before, there is no quantitative study of these nuclei as far as this author is aware. What does exist relates indirectly, in that certain areas of the cortex are expanded which have an input from intrinsic nuclei (see Feremutsch[62]; Heiner[92]). The following table offers a brief reference summary of the anatomical basis for the past definition and is adapted from Pribram's[132,133] tables.

Non-specific nuclei	*Cortex*
Posterior	Posterior parietal cortex, temporal cortex, and anterior occipital cortex (Posterior association areas)
Anterior	Retrosplenial cingulate cortex, posterior and anterior cingulate cortex
Central	Allo- and juxtallocortex of the limbic portion
Medial	Frontal eugranular isocortex. Frontal association areas
Specific nuclei	
Ventral	Pre- and postcentral cortex. Also dorsal frontal cortex
Geniculate	Posterior supratemporal plane and striate occipital cortex. Koniocortex

Bonin[11] compared the absolute and relative size of the striate cortex in a series of primates, based on earlier measurements by Filimonoff[63]. The figures given below indicate the increase in posterior association cortex, which is related to the posterior non-specific thalamic nuclei (Table IV).

These values are in good agreement with those made by Brodmann[24] who found

TABLE IV

SIZE OF STRIATE CORTEX (AFTER BONIN[11])

Species	*Absolute area (sq. mm)*	*Percent total cortical area*
Man	2613	3.0
Orang	1876	8.5
Cercopithecus	933	10.3
Galago	58	15.1

the striate area to be 10% in apes, 15% in lemurs, and with Economo and Koskinas[57] who found man's value to be about 2–2.5%.

Solnitzky and Harmon[172] attempted to determine relative volumes of the types of cortex in the occipital lobe and their values are given below, in terms of percent of occipital lobe. Area 17 refers to striate cortex proper. Areas 18 and 19 would be posterior association cortex (pulvinar) as noted above (Table V).

TABLE V

RELATIVE VOLUME OF AREAS IN OCCIPITAL LOBE (AFTER SOLNITZKY AND HARMON[172])

Species	*Area 17 (%)*	*Area 18 (%)*	*Area 19 (%)*
Felis	67.3	24.4	8.3
Galago	66	25.4	8.6
Cercopithecus	41.1	32.2	26.7
Orang	39.6	30.5	29.9
Homo	25.4	37.7	36.9

These values show directly the clear-cut decrease of striate area and concomitant increase of posterior association area. While separation between areas 18 and 19 are more difficult on the basis of cytoarchitectonic structure than between area 17 and the rest of the cortex, lumping the two areas together still shows the increase. Bonin[11], taking volumes of striate cortex and comparing it to volume of lateral geniculate found increased ratios up to man, indicating the increased corticalization of function.

More direct evidence for shifts in association cortex may be gained from reference to Shariff's[163] figures on cortex types (Table VI).

These figures are based on one brain for each species, whereas those adapted from Filimonoff[63] in Bonin[11] are for several. The values for eulaminate cortex are equal for man and chimpanzee, which seems opposed to all ideas regarding the increase of association areas in man. It may be that the values would alter considerably if enough specimens were measured. All other opinions (Bonin and Bailey[16]; Bonin[14,15]; Weidenreich[189,190]; Brodmann[21]) have clearly indicated that the association cortex of the parietal and temporal lobes have increased in man. Visual inspection

TABLE VI

VOLUME OF CORTEX IN CU. MM (ONE HEMISPHERE) (AFTER SHARIFF[161])

	Eulaminate frontal, parietal, temporal	*Percent*	*Agranular Area 6*	*Percent*	*Koniocortex*	*Percent*
Man	97,740	84.9	13,850	12	3610	3.1
Chimpanzee	35,600	84.9	5,610	13.5	1750	4.2
Cercopithecus	6,880	69.8	1,560	15.8	1430	14.5
Hapale	650	66.6	166	17.0	160	16.4
Tarsius	225	57.3	60	15.3	108	27.4

of the disposition of the lunate and Rolandic landmarks clearly shows this. Either the figures given by Shariff are incorrect with respect to eulaminate cortex, or the brain shrank differentially, or the specimen was at one end of a distribution and others should be measured. The difficulty probably lies with the particular cortex used for the human example. Sholl[166] has pointed out that the value of 115.2 ml which Shariff gives for the total cortex is probably from an unusual brain, and other measurements would probably tend to raise the volume. Only further data will straighten this out. This work does show, however, extent of motor cortical reorganization, a fact not taken into account by Jerison[98] or Tobias[180].

On the other hand, if use is made of Hebb's[91] suggestion regarding the A/S ratio mentioned earlier, the difference becomes more apparent if the agranular cortex is not considered. In man, the A/S ratio, or eulaminate to koniocortex (*i.e.*, association cortex to sensory projection cortex), is *27.8*. For the chimpanzee, the value is *20.4*, and for the macaque, *4.8*. As was earlier pointed out, this interesting relationship may have application to the hypothesis considered earlier.

If the behavioral processes noted under terms such as symbolization, or extrinsic representative factors are constructs valid for describing certain aspects of human behavior which are species-specific, then Hebb's[91] proposal may be seen as more closely tied-in with behavioral-complexity than emergence. His model does not offer an explanation of the transform, at least as this paper has thus far described the nature of the problem. It should be pointed out, that Hebb's analysis tends to treat the association cortex as a reservoir of neurons intercalated between stimulus and response, without any input other than sensory projection areas or centers. It must be kept in mind that in 1949 the beginning of a more inclusive appreciation of the role of subcortical mechanisms and influences on the cortex via the thalamus was just coming into being (Magoun[115]). As the chart adapted from Pribram[132,133] shows, these association areas have input exclusive of projections from the sensory cortex, and the neurons are not simply passively awaiting information from the sensory projection areas. Activation and inhibition are operating also, and it is probable that the notion of 'set' or 'attention' must be included. Nevertheless, the skeleton of Hebb's proposal seems important with respect to additional complexity, whatever the actual relations of this cortex to subcortical influences, particularly the ascending reticular formation. The above data showed that these areas increase in man, and

perhaps some adaptations of Hebb's proposal, such as given by Pribram[132,133] for posterior and anterior association areas with respect to discrimination and operation will serve as partial explanations at this molar level.

(2) Molecular

The first level, the molar, has dealt with reorganization from the viewpoint of increased association cortex. The second level, the molecular, will now be treated. There is some degree of isomorphism between the two levels. In the first, the addition of more intercalated units was shown. In this second level, it will be shown that increased cell size, decreased density of neurons, and increased dendritic branching contribute to increased complexity. It will be convenient to discuss this evidence under three headings: comparative, ontogenetic, and experimental.

(a) Comparative

Using the older ideas of Nissl[125] and Economo[56] on the increasing intercellular gray matter (neural density) with phylogenetic advance, Shariff[161] has provided empirical data for primate neural densities. His values are given below since they will form a nucleus for further discussion (Table VII). (Figures inside the parentheses are reciprocals of Shariff's figures, and were calculated from a slide rule. They are based on Haug's[87] definition of the gray cell coefficient. The odd value for Hapale is considered by Haug to be the result of a thinner section (5 μ) and will be eliminated from hereon.) As can be seen from the above figures, the density decreases in all areas of the cortex. With respect to average cell size, Shariff[161] gave the following figures (see Table VIII).

TABLE VII

GRAY CELL COEFFICIENTS (NEURAL DENSITIES) (AFTER SHARIFF[161])

	Eulaminate	*Agranular*	*Koniocortex*
Man	0.057 (21.3)	0.045 (22.2)	0.087 (11.5)
Chimpanzee	0.052 (19.3)	0.051 (19.6)	0.107 (9.3)
Cercopithecus	0.082 (12.2)	0.053 (18.9)	0.124 (8.1)
Hapale	0.044 (22.7)	0.056 (17.9)	0.113 (8.9)
Tarsius	0.163 (6.2)	0.091 (11.0)	0.238 (4.2)

TABLE VIII

AVERAGE CELL SIZE (AFTER SHARIFF[161])

	Eulaminate (cu. μ)	*Agranular* (cu. μ)	*Koniocortex* (cu. μ)
Man	1621	2468	895
Chimpanzee	865	1035	616
Cercopithecus	718	855	468
Tarsius	536	580	339

These are average values only. As Haug[87] has pointed out, they tend to oversimplify the matter, since in man at least, the number of cell classes based on size increases. Nevertheless, it is taken as evidence for the statement made earlier with regard to increasing cell size. For other confirmatory evidence that cortical cells are on the average larger in man, see Bonin[9] and Bok[7].

Haug[87] finds that Shariff's data support older arguments that cell size increases while density decreases in phylogeny. Thus the volume is correspondingly made up more by the nervous gray, of neuropil, than nerve cells. Haug's calculations show this relationship very clearly (see Table IX).

TABLE IX

DATA FROM HAUG ON CORTEX VOLUME AND CELL NUMBER

	Vol. cortex in ml	*Relative volume*	*Total No. cells* × 10^9	*Rel. No. cells*	*Vol. cell coeff.*
Man	230.4	293	6.9	22	13.6
Chimpanzee	95.9	108	5.5	18	6.0
Cercopithecus	19.7	25	2.5	8	3.1
Tarsius	0.786	1	0.31	1	1.0

Tarsius is here being used as a standard for relative volume and relative number of cells. The volume cell coefficient expresses the ratio of cortex with respect to volume, to that of cell number. This shows that increased cranial capacity within the primates is more than simple addition of nerve cells, and is instead a reorganization of units. 1 ml of human cortex is not equivalent to 1 ml of chimpanzee cortex, except in terms of volume. In terms of function, such a comparison would not be a comparison of equal units.

It should be made clear that not all workers agree with Shariff's and Haug's interpretation. Tower[181] notes:

'The fact that this expression (density) is valid from the smallest mammalian species to species with brains much larger than man is another indication that intelligence does not bear a simple relationship to neurone density and degree of axodendritic complexity in the cerebral cortex.' (p. 31).

This conclusion of Tower's is based on the fact that density bears a clearer relationship to brain weight than phylogenetic level. To support his contention, Tower investigated three whale brains and one of an elephant. The average density for the whale brains was 6800 neurons/cu. mm, and for the elephant brain, 6900 cu. mm. These results were in very close agreement to the expected values based on a logarithmic formula relating density to brain size. While the first part of Tower's statement regarding the correlation of intelligence and density is probably a more precise view than Haug's or Shariff's, his conclusion regarding axodendritic complexity is possibly premature. Tower's study was done with Nissl-stained material, and data for a series of animals using Golgi Cox methods have yet to be produced. Furthermore, neither Haug nor Shariff have asserted that intelligence bears any simple

relationship to neuron density or axodendritic complexity. On the other hand, primates do have relatively more cerebral cortex than most other mammals, particularly the elephant and whale. In comparing members of an order, such as the primate, it seems reasonable to expect that the relationship which Tower doubts, is more real than in comparing members of widely different orders. Thus Tower's data do not invalidate entirely any correlation between density and intelligence, but show that other factors must also operate. Without knowledge of the dendritic ramification in whale and elephant brains, and their intelligence, it would seem premature to regard Tower's contention as final.

Tower's warning, however, has brought attention to an important point. There are at present very little data to allow a linking between neuron density and dendritic branching in the cortex, although Rensch[137,138] claims the linkage is valid. One reason for this is that Golgi stains for adult animals in a species are not able to impregnate axons and dendrites in any satisfactory manner (Sholl[166]). This state of affairs is particularly true in the case of higher mammals such as the primates. The closest approach made to dealing with a comparative series through Golgi methods was that of Erp Taalman Kip[61] (quoted in Bonin and Bailey[16]). He found that in a series of rodents, the branching of dendrites increased with increasing brain size and decreasing gray cell coefficient. This analysis has not been extended to primates, nor the whale or elephant. Barasa[1] (summarized in Excerpta Medica, 1962) examined twelve species of small mammals, but did not include any primates. He found that the sizes of the cells were only slightly different between species, but that density did not vary according to brain size. In addition, he found that increased parameters of dendritic branching were shown by the larger species.

One possible way of connecting the data on density with that of dendritic branching would be through an extension of Bok's[7] work. Golgi-stained materials would be a necessity. Bok claimed that larger neurons had more dendritic branching. Some of his relationships are of particular interest. Bok found that the cell surface, the perikaryon, was proportional to the nuclear volume. In somatic cells, cell volume is proportional to the nuclear volume, suggesting that neurons have quite different functions and metabolism. The second power of cellular volume is reported as proportional to the third power of nuclear volume. In addition, nuclear volume is proportional to the radius of the 'dendritic tree', the average distance of dendritic terminals from the cell body. Total dendritic length is proportional to the square of nuclear volume. Therefore, Bok concludes that dendritic function is proportional to the first power of nuclear volume, while total dendritic length is proportional to the square of nuclear volume. The axon length is considered by Bok to be proportional to the square of dendritic length.

Unfortunately these beautiful mathematical relationships have not been confirmed by any other independent study. These relationships had been worked out during the 'thirties' by Bok in a large number of papers and are collected together in the above 1959 volume. Sholl[166] has pointed out that these relationships given by Bok are the result of measuring only eight cells in an unnamed part of the cat cortex. More extensive sampling by Sholl did not confirm the relationships described by Bok.

The exact relationship between nuclear volume, perikaryon volume and dendritic parameters is still an open question. If the relationships hold in all species, even at a lower level of correlation than Bok suggests, one would expect the whale brain to contain the most neuropil, since its density is the least. In any event, the general relationship between larger neuron size and more dendritic branching has been confirmed for some species in the above mentioned works (see also Rensch[137,138]), and further illustrations of this relationship will be shown in following pages. Clendinnen and Eayrs[33] have shown this to be the case in the rat cortex (see Eayrs and Goodhead[51] for further demonstration of this relation). Conel's[35] work on human ontogenesis of the cerebral cortex as well as that of Schadé *et al.*[151,152] indicates that dendritic branching increases with the growth of the neuron. The same relation has been shown for the rabbit by Schadé[149], Schadé *et al.*[150]; in the cat by Purpura *et al.*[135], Noback and Purpura[127]; in the guinea pig by Peters and Flexner[130], and for the monkey by Yakovlev[196]. These data will be treated more fully under (b).

The data of Shariff[161] indicated that only the average cell size increased with the higher primates. Bonin[8] found that the gray cell coefficient in the fifth layer of area 4 increased in the primate series (galago 52, macaque 87, chimpanzee 112, man 233). Bonin[9] also found that the ratio of mean nuclear volume of the giant cells with respect to ordinary ones increased with phylogenetic level, or brain size. Without Golgi-stained material it is very difficult to assert that dendritic branching also increases. Indirectly, the evidence from the other sources (above) tends to suggest that this is the case. If this relationship can be demonstrated more exactly for the primates, the increase in cell size, decrease in density, and concomitant increase in dendritic branching, particularly in the motor cortex, would fit well the behavioral evidence relating to finer degrees of motor control, or 'neokinesis' as examined under 'Subcortical Reorganization'. More dendritic branching would mean more synapses on the motor cells, *i.e.*, more modulating influences.

Finally, Sholl[166,167], in a study of mouse, rat, cat and human cortex, found regions which had lower neural densities associated with increased axon numbers. This refers to numbers of axons crossing a standard area, and not to an increase of axons of a nerve cell. Sholl concluded that the efficient operation of the cortex depended on the complexity of axonal connection patterns. In the cat cortex, Sholl[161,166] found that dendrites of neurons formed about 90% of the receptive surface of the neuron. Using Golgi-stained material from the motor and visual areas, he measured perikarya values ranging from 550 cu. μ to 19,620 cu. μ, and total dendritic branching ranging from 18 to 79 μ. Relying on his data from the cat cortex, Sholl concluded that about 5000 neurons would be distributed around the terminal branches of a single primary visual fiber. These data help to provide some indication of the order of complexity of neural connections.

Until Golgi-stained materials are provided for a series of primate cortices, it is only possible to suggest that these relationships hold for this Order also. As far as the density and size of neurons is concerned, the evidence does indicate that these parameters vary in the direction expected on the basis of other work. As will become more evident in a later section regarding these relationships to anthropological notions

of brain evolution, these considerations offer some promise as a means of overcoming past difficulties with respect to cerebral mass. It seems very obvious from the past discussions that a comparison of equal volumes of cortical tissue of different primate species cannot be a comparison of equal units. The implications of this assertion will be more fully treated in another section.

(b) Ontogenetic evidence

The previous section provided indirect and direct evidence for increasing neurohistological complexity in a comparative series. This section will provide further illustrations from developmental studies, to indicate that reorganization takes place in the ontogenetic as well as phylogenetic sense. Another reason for analysing these data is that data exist relating the changes in histological complexity to increases in the complexity of behavior.

Peters and Flexner[130] examined the guinea-pig, and related histological to biochemical changes. They found that the period between the 41st and 45th days of gestation were critical. At this time, the following changes were noted: the first appearance of five layers as in the adult; Nissl bodies appeared in great numbers; great increase in number and size of cell processes; activity of the enzyme adenylpyrophosphatase increased to adult level. Flexner[65] found that movements of the hand, lip, mouth and neck were elicited upon stimulation on the 42nd 46th day. At this time spontaneous electrical activity was recorded from the cortex. Unfortunately, the glia were not studied. Recently, Peters and Bademan[129] investigated the form and growth of stellate cells in the sensorimotor cortex, and found that dendritic segments in the adult were hardly increased over the newborn. This indicates the mature state in which this animal is born. Initial segments varied little between adult and newborn, but more terminal branches did show increased length in the adult, providing more area for synapses. Using Sholl's[166] method of shell perforation, they found that the adult showed more perforations than the newborn at increasing distance from the cell. The difference, however, was not large. The authors then concluded that learning is dependent on pathway selection rather than pathway number. While their conclusion may apply to the guinea-pig, the mature state of this animal at birth must be kept in mind, and the above conclusion should not necessarily be applied to other animals such as the rat or cat.

In addition to Sholl's[166,167] work on the cat cortex, Brizzee and Jacobs[23] have undertaken a study of maturational changes in the motor area of the cortex. They found that neuron density decreased from $2.6 \cdot 10^5$ cu. mm in 100 g animals to $0.68 \cdot 10^5$ cu. mm in the 0.5 kg group, remaining constant until adulthood. As Peters and Flexner had found, the cytoplasmic increase was greater than that of the nucleus. An unexpected finding was that a decrease in mean cell volume, both cytoplasmic and nuclear, occurred in the 2 kg group. They suggest that a decrease in synthetic activity takes place after the growth of cell processes is complete.

Purpura *et al.*[135] studied kittens in the fetal stage up to an age of 6 weeks postnatally. Electrophysiological data correlated with the changes of morphological features. The superficial cortical response, for example, correlated with the maturation

of basilar dendrites. Noback and Purpura[127] found that in the first three postnatal weeks, the dendrites of the cortical neurons increased in length and caliber, and possessed greater degrees of branching and dendritic spines. Voeller *et al.*[187] found, from electron microscopic study of the cat cortex, that higher packing density of cell bodies in fetal and neonatal kittens could be explained by the absence of neuron processes, and possibly neuroglia.

In the rabbit, Schadé and Baxter[150] study volume and surface area changes of cortical neurons. At 10 days, 2.1 % of the cortex was occupied by apical dendrites, with a surface area of $6.0 \cdot 10^7$/sq. μ. The values at 30 days were 6.2 % and $14.2 \cdot 10^7$/sq. μ, and at 300 days were 7.1 % and $15.4 \cdot 10^7$/sq. μ. Similar changes were noted for basilar dendrites, except that the changes were even greater. These morphological changes paralleled the development of surface electrical activity.

Eayrs and Goodhead[54] have applied this method to the rat. The gray cell coefficient decreased most rapidly during the first few postnatal days, then at a slower pace to maturity. These rates varied in different layers. In layer I, which is sparse in neurons, little change was noted. The greatest changes took place in the granular layer where the concentration of neurons is the highest. Table X illustrates the changes of the basilar dendritic branching patterns with time.

TABLE X

NEURONAL PARAMETERS IN DEVELOPING RAT BRAIN (FROM EAYRS AND GOODHEAD[54])

	6 days	*12*	*18*	*24*	*30*	*Adult*
Mean No. dendrites arising from perikaryon (1)	0.6	5.4	5.2	5.3	5.3	5.2
Mean No. branching sites (2)	0.6	4.7	8.3	18.0	19.4	23.7
Mean No. endings (3)	0.6	10.1	13.5	23.3	24.7	28.3
Branching index (1–3)	1.0	1.9	2.6	4.4	4.7	5.5

It might be mentioned however, that this study indicated that the major reduction in cell density occurs *prior* to the period of maximal dendritic growth. What other factors besides neuropil are operating is still an open question.

Schadé and van Groeningen[152] have published quantitative data on the maturation of the middle frontal gyrus in the human cerebral cortex. Table XI indicates the correlation between density decrease and increase of connectivity.

Similar changes were noted for layers IV and V and also for stellate cells. These changes have also been correlated with the development of Nissl bodies and the electroencephalogram.

Based on Sholl's[163,166] work, these authors also found an exponential relationship between dendritic density and distance from the nerve cell to exist for pyramidal cells in the three layers. Since the value of the regression coefficient is a measure of the dendritic field expansion, a reciprocal index 1 K is found, and defined as the 'dendritic field factor' (Dff) which increases with the expansion of the dendritic field. The values of Dff for various species are of interest, since they show in quantita-

TABLE XI

NEURONAL PARAMETERS IN DEVELOPING HUMAN BRAIN (SCHADÉ AND VAN GROENINGEN[152])

	Newborn	*6 months*	*24 months*	*Adult*
Layer III				
Neurons × 10^3 cu. mm (ND)	99.0	30.5	20.1	12.5
Gray/cell coeff. (GCC)	41	53	55	77
Layer IV				
ND	444	151	59.8	35.0
GCC	55	95	180	250
Layer V				
ND	60.5	16.1	8.9	6.0
GCC	36	37	53	66
Layer III (pyramidal cells)				
No. dendrites arising from perikaryon	6.7	6.4	7.9	7.6
No. intersections	5.8	67.7	93.1	195.3
No. branching points	3.1	15.6	16.7	40.8
Est. total dend. length	203μ	2369	3259	6836

tive fashion the increase of dendritic connectivity. In the human, the Dff for layer III in the adult is 128.2, 90.9 in layer IV, and 142.9 in layer V. In the newborn, the corresponding values were 20.8, 10.2, and 45.5. Using Sholl's regression coefficients[163], the authors found a Dff in the cat of 38.5 and 37.0. With Eayrs and Goodhead's[54] data, a Dff for the rat gave a value of 28.6. These results show beyond any reasonable doubt that species differences with respect to dendritic ramification do exist. The parameter increases, if not phylogenetically, at least with brain weight. This point and the data referred to above are of very great interest to the brain size problem mentioned earlier in this paper. The number of neurons in the human neonatal brain are the same as in the adult. If the brain weight is plotted in terms of percent of adult value against time, and the same done for the Dff, it becomes obvious that brain weight or capacity increases at a greater rate than the Dff. This probably represents a number of factors such as glial proliferation, vascular growth, perikaryon growth, and of course, neuropil. The curves are strikingly isomorphic, except for rates.

As has been pointed out earlier, the number of neural units in relative numbers is not strikingly larger than in the apes, the figure being roughly 1.25 times. Yet roughly 1000 ml of brain substance separates the two forms. While no figures exist with respect to branching parameters in apes or monkeys, the data with respect to density suggest that branching parameters would be expected to fall beneath the human value. This relationship has been traced through a variety of mammalian brains, and unless the morphogenetic processes for intermediate members of the primate order are different from all other mammals thus investigated, the probability seems extremely high that the expectation would be met. Essentially, this means that in addition to subcortical reorganization with respect to locomotor and manipulative abilities, the reorganization of neural units to allow for greater gray cell coefficients (*i.e.*, decreas-

ed density, and increased branching parameters) is the internal result of the outward 1000 ml increase in mass noted for hominid evolution. This increase took place mainly in the association areas. Part of this increase would be reflected in glial increase also.

Schadé and van Groeningen[152] conclude their work as follows:

'The postnatal evolution of the middle frontal gyrus proceeds gradually in regard to the histological structure to about 2 years and does not change much to adulthood. This period after birth is devoted largely to the perfection of sensorymotor performance. The period thereafter in which speech, writing, and the intellectual performances develop is not characterized by major changes in the microscopic structure if we take the middle frontal gyrus as a standard for the whole neocortex.' (p. 106).

In addition, then, after the cessation of mitotic division of neurons, the growth of dendritic processes increases but slightly after 24 months. By the age of 2 years, the human is essentially 'programmed' for the full range of complexity of behavior. He is also programmed at this time for whatever it may be that is defined as unique in the way of speech behavior and use of extrinsic symbolization. By 'programmed', it is meant that the structural bases have been laid down by age 2 which permit the development of human abilities throughout life. The human is thus an open system with unique potentialities, and it is the nature of his continuing interaction with the environment, as a system, which leads to the development of human behavior in the familiar sense. This point is of some importance, since what is taken as great increase in the range of behaviors (and their increasing degree of complexity) is not attended by further structural changes *at this level*. In other words, some more molecular level than dendritic branching will have to be investigated to pick up the structural changes which might be concomitant with further ontogenetic development of human abilities. Dendritic spine development within the dendritic plexuses is one possibility (see Conel's volumes I–VII, 1939–1963), but one gathers from Schadé's work that this process is essentially complete when the dendritic plexuses have grown. (With respect to human spines, or bouton terminaux, see Smythies *et al.*[170] for quantitative data. The data exist only for the human as far as I am aware.) Thus some more molecular level must be investigated to determine whatever structural changes there are that follow the ontogenetic development of behavioral complexity. Glial cells are more labile with respect to continuous production, and perhaps have a role in this interplay of structural and behavioral development. At the present it is too early to do other than suggest this as a possibility. On the other hand, the problem is compounded by not knowing the significance of the human code or type of representative factor in this development. Is the learning in animals which are not immature or totally naive (such as adult monkeys in learning-to-learn situations) a matter of the organization of experience rather than structural alteration in dendritic branching parameters? If both factors are important how does one ever *separate* and *measure* these two aspects? This question is of particular importance in the human case, given the known potentialities of extrinsic symbolization (*e.g.*, conceptual thought) for organizing experience, and which allow for long retention and immediate recall.

If the 'emergence' view taken in this review can be accepted, the increase in neuro-

pil is seen as one factor common to a large group of vertebrate species. This section has tried to indicate the evidence for the increasing neurological complexity, at the level of dendritic branching. Further support would be gained by investigating the ontogenetic development of behavioral abilities, particularly in the area of problem-solving. Such a task is beyond the scope of this paper, and the assertion must rest at present with the neurological picture I have presented. The problem of separation and measurement would still exist, however. In the next section, (c), some further data will be presented to merge a little more the neurological and behavioral aspects.

(c) Experimental evidence

Logically, one would expect that increasing neuron number would result in more complex behavior. With the development of endocrinology in the 20th century, it became possible to provide extractions or syntheses of certain hormones. The first attempt to stimulate hyperplasia or increased mitotic activity in nerve cells was undertaken by Rubinstein[146]. This worker used growth hormone with rats, injecting the substance peritoneally, after birth. No effect on brain weight was found. Zamenhof[197-199] pointed out that mitotic division was complete in the rat brain by birth. Using purer forms of somatotrophin, he administered this substance to pregnant rats, and delivered the offspring by caesarian section. An earlier procedure was worked out with tadpoles[198], and it was found that for the entire cerebral hemispheres of this animal, the neuron and glia numbers were higher by 44–126% in injected animals. In 1942, in his extension of this approach to the albino rat, the following results were noted: offspring at birth showed an 18.7% increase in body weight, 36% in cerebral hemisphere weight, 21% in cortical thickness, and 70.4% in cortex volume, all in comparison to controls. Cell density increased 9.27% over the control value, and the increase in % of cell numbers per volume of cortex was 86.5%. All these increases were reported as statistically significant. At maturity, however, the only increases which were statistically significant were for cell density, 14.8–27.6%, and total number of cortical cells, 38.0–40.6%. In females of 124 days of age, a volume increase of 22.5% over that of controls was statistically significant. Several of the experimental and control animals were later tested for psychological performance by Warden *et al.*[188]. No significant changes in maze performance were found. These authors used a 12-cul-de-sac maze, and suggested that a task requiring a higher level of intelligence might better indicate the differences. Of interest was the observation that males, rather than females, needed fewer trials for criterion. It was the female adults which showed more significant changes in cortical structure.

While it may be true that this work raises many more problems than it solves, it does indicate that experimental alteration of neuron number and density is feasible. The increase in density of neurons is a particularly interesting observation, but it is unclear whether neurons and glia were clearly identified. In light of the following sections, it seems hardly expectable that dendritic ramifications occurred for the experimental rats. Zamenhof did not measure neuron size so that it is impossible to suggest hypertrophy in addition to hyperplasia. The former condition, as shown in earlier sections, would suggest increased dendritic branching. Perhaps the

behavioral evidence is best explained by the possibility that increased density actually lowered dendritic branchings, and level of task requirement. Only replicated studies, using Golgi-stained material, will decide this question.

Clendinnen and Eayrs[33] applied purer forms of growth hormone in the same manner as Zamenhof, and achieved results clearly in contrast to this earlier work. Rather than hyperplasia or numerical increase in cells, these authors noted a hypertrophy of neurons. In addition, cortically mediated behavior, as measured by the Hebb-Williams closed-field test, was enhanced, as were certain aspects of electrical activity and dendritic branching. In view of these different results, it will be worthwhile to note some further details. Table XII shows the effects of this treatment with growth hormone.

TABLE XII

EFFECT OF GROWTH HORMONE (CLENDINNEN AND EAYRS[33])
Parentheses refer to number of animals in each group.

Response and unit of measurement	*Normal*	*Exper.*	*Diff.*	*Signif. (P)*
(1) Reflex suspension (sec/day)	8.64 (13)	9.49 (7)	0.85	0.4 0.3
(2) Startle response (mean age first appearance)	12.1 (13)	12.1 (7)		
(3) Righting reaction (mean age first appearance)	16.1 (13)	15.6 (7)	0.5	0.1 0.05
(4) Placing reaction (mean age first appearance)	17.1 (13)	16.3 (7)	0.8	0.05 0.02
(5) Closed-field test (mean errors over twelve problems)	165 (8)	134 (8)	31	0.01 0.001

The tendency for automatic behavior patterns to mature earlier in experimentals is evident, but the most striking advance is in cortically-mediated behavior. The learning curves followed an identical pattern for both groups, and the authors note that a high correlation (r 0.799; P 0.01 0.001) was found in the degree of difficulty presented by test problems both to experimentals and controls. With respect to electrical activity, the dominant frequencies and amplitudes of response to rhythmic photic stimulation did not differ significantly between the two groups of animals. Spike and wave patterns of response to photic stimulation, with a tendency to 'follow' at half the frequency of the applied stimulus, were seen in the records of all experimental animals but only one of the controls.

The histological changes were no less clear-cut and are given below (Table XIII).

Using the previous expression Diff, the normal value is 29.0 and 38.4 for experimentals. When the data for axons and dendrites were combined using Uttley's[181] formula for estimating interaction probabilities, an arbitrary factor of 145 for the controls was increased to 302 for the experimentals. (This estimation is based on the work of Sholl and Uttley[168], and Uttley[181], where it is assumed that units are connected randomly. The probability of a connection with another unit thus falls exponentially as this distance from the unit increases. Eayrs[33] notes that in normal rats from the

TABLE XIII

NEURONAL PARAMETERS IN NORMAL AND EXPERIMENTAL ANIMALS (AFTER CLENDINNEN AND EAYRS[33])

Measurement	*Normals*	*Experimentals*	*P*
Gray cell coefficient			
layer IIIc	7.67	9.18	0.05–0.02
layer Vb	8.39	9.66	0.05–0.02
Cell density (perikarya/sq. mm)			
layer IIIc	1629.3	1522.6	N.S.
layer Vb	866	908	N.S.
Axon density (axons/sq. mm)	13.91	13.43	N.S.
Dendrite number from perikaryon			
layer IIIc	4.8	5.2	0.1–0.05
Total dendrites formed	16.9	21.9	0.05–0.02
Mean dendrites length	67.6	82.3	0.01–0.001
Parameter of decay of dendritic field with increasing distance from perikaryon	$0.00345e^{-0.054}$	$0.00261e^{-0.042}$	

Eayrs and Goodhead[51] study, the factor is small at 12 days of age, but increases 12-fold by the 30th day, and continues into adulthood.)

As is obvious, the above data tie in very well with that given throughout this section on complexity. The increase in gray cell coefficient is correlated with increased perikaryon size (hypertrophy), increased dendritic branching, and increased behavioral adaptability. As for the differences between Zamenhof's and this work, the authors conclude that hormone preparation is the most probable factor, since Zamenhof's earlier preparation contained detectable amounts of adrenocorticotrophins and thyrotrophin. The increased cell density noted by Zamenhof might also be taken to indicate that perikaryon size as well as dendritic branching or neuropil, has decreased, or at least is unaffected. Such an interpretation is consistent with the testing by Warden *et al.*[188].

The second line of experimental restructuring of the finer histology of the cortex is that done during the past decade by Eayrs and his associates on the relationship between thyroidectomy and structure and behavior. In general, the results have been in the opposite direction to those noted above. The following discussion will be taken from two recent reviews (Eayrs[52,53]). Where necessary, other references will be cited.

In rats thyroidectomized at birth, the gray cell coefficient is decreased. Concomitantly, the perikarya are smaller, and the neuropil definitely hypoplasic. (For details, see Eayrs[51].) The dendrites are reduced in number, and do not follow the typical exponential decay with distance, as do the normal controls. This accordingly reduces the probability of interaction between neurons to about 85% of that of normal. The decrease in the factor was from 296 in the normals to about 123 in the experimentals. Bradley *et al.*[20] found that in experimentals the potential changes from EEG recordings were of small amplitude. There was also an absence, up to 24 days, of slow activity and blocking by sensory stimulation. Photic driving was reported as absent. Eayrs feels that these changes must relate to the neuropil, since metabolic tests have not disclosed any other factors as responsible for this retardation.

With respect to behavior, Eayrs and Lishman[55] found that innate responses were retarded. Cortically-mediated behavior showed striking changes. Experimentals habituated more rapidly to new experimental situations, and made significantly more errors in maze-learning experiments. The findings of neurohistological and behavioral aspects fits closely the formulation of Bishop and Clare[30]. These authors regard the neuron as demonstrating *both* graded response activity and all-or-nothing discharges. The former they associate with dendrites and the latter with the activity of the perikarya and axons. The former activity would correspond to slow spontaneous potentials, and the axodendritic stimulation is viewed as modulating the excitation states of the neuron and its response probability to the latter type of all-or-none kind. As was pointed out before, Schadé[149] had found similar evidence for the developing rabbit EEG. Eayrs[53] notes that slow waves appear in the thyroidectomized rats when the connectivity approaches that of the normal rat.

Since all the evidence reviewed this far has successfully related the finer cortical neurohistology to behavioral differences (with respect to complexity), it seems reasonable to conclude that increased gray cell coefficient (decreased density) is associated with increased dendritic ramification, increased connectivity, increasing modulation of graded-type in neurons, and finally, an increased ability for cortically-mediated, adaptive problem-solving behavior. The numerous works of Haug cited in an earlier section relating to neural density in primates, as well as Shariff's work on density and Bonin's analysis of cell size, all lead one to expect the same mechanisms to be operating in the primates. These conceptions thus provide an immediate task for Golgi-staining procedures.

The third and final line of experimental evidence relates to the effects of environmental stimulation upon brain biochemistry, neuroanatomy, and problem-solving behavior. While a series of programs are under investigation in this general area, only one trend is considered here: the relation of environmental complexity and training (ECT) on the cholinesterase acetylcholine (ChE, ACh) activity and brain anatomy in the rat. This section will not treat extensively these developments since the anatomical aspects are just beginning to become clear. For the early history, rationale, and development of hypotheses, see Rosenzweig *et al.*[142, 144].

Broadly, this work is based on the assumption that variation in brain chemistry relates to differences in adaptive behavior. To implement investigation of the problem, different strains of rats have been investigated using the ChE ACh system as the biochemical variable, and the Kretch hypothesis apparatus to test behavioral differences. Tryon[183] had successfully produced different strains of rats selecting for behavioral aptitude, and found that a maze dull and maze bright strain, S_8 and S_1 respectively, could be produced. Roderick[139], using high and low criteria for ChE, bred for the extremes in two strains. Behavioral tests indicated that superior maze performance was found in rats with lower ChE activity. Later hypotheses suggested that ACh and ChE were under different genetic influence, and that the disparity of behavior could be explained on the basis of different ratios of these biochemical indicators. Later tests bore this out. This was based on the assumption that greater functioning at the synapse was correlated with greater release of ACh. Since ChE

hydrolyzes the former transmitter substance, higher amounts of it in proportion to ACh would be expected to have a too rapid breakdown of ACh and therefore result in less effective learning. It was found that the greatest difference in ACh occurred between S_1 and S_3 strains, with ChE activity showing the least difference. On the other hand, the strains produced by Roderick showed little difference with respect to ACh but differed greatly with respect to ChE. In view of these differences between strains with respect to maze ability and brain biochemistry, it was later hypothesized that increasing the animal's experiences would lead to greater synaptic firing, and therefore, greater ChE activity in the brain. Using S_1 and S_3 strains, littermate pairs were randomly selected and placed in one of two environments: ECT and IC. The former signifies 'environmental training and complexity', the latter, 'isolated control'. The former condition consists of providing a series of diverse stimulations ranging from daily handling, communal living, objects and toys to explore, and training in a variety of problem tasks. The latter condition consists of isolation, living in a small cage, no toys or objects to explore, no handling, and no problem-solving tasks. Food and temperature schedules are kept the same for both groups.

Using a measure of ChE activity per unit weight of cortex, it was found that this specific ChE activity decreased in the ECT condition, the opposite of what was expected. However, with respect to subcortical sections, the relation expected did hold. Consequently, cortical/subcortical ratios in ChE activity differentiated the two groups. This set of results was finally explained on the basis that cortex weight in the ECT animals had increased, and that when total, rather than relative or specific ChE activity was considered, the results fitted the hypothesis. The ECT animals showed about a 4% increase in cortical weight, and analyses of variances for littermate pairs showed each difference to be significant at better than the 0.01 level.

Unfortunately, anatomical studies designed to indicate the nature of the increase in cortical weight are currently in progress, and it is too early to be definite regarding the nature of the exact changes. If in fact the ChE activity increases, one would expect this to be the case where more synapses occur. That a relationship between decreased neuronal density and increased dendritic branching and connectivity in the neuropil exists, has been shown in a number of instances throughout this review. Diamond *et al.*[41] have found increases in cortical thickness in the visual and somesthetic cortex. Preliminary histological investigation by myself showed an increased cortical depth of about 6% in ECT animals in the anterior cortex. Diamond *et al.*[41] have also found decreased neuronal density in ECT animals, the difference amounting to about 17%. There were also some changes in glial counts. These changes are most obvious in layers II and III where concentrations of intercellular material, the neuropil, are the highest. Thus the increase in cortical weight can be explained by the increase in cortical depth, giving a decrease in neuronal density. One would expect increased dendritic branching, signifying more synapses and thus more ACh release, to be correlatable with the decrease in neuronal density. Naturally, Golgi Cox staining is necessary to determine this. Tentatively[95b] I have found confirmatory evidence for such an increase. In an examination of the second layer of visual cortex in 15 litter-mate pairs which had undergone ECT and IC training, 11 out of the 15 ECT animals showed increases in

dendritic branching of stellate cells when compared to their IC litter-mates. It must be emphasized that this finding is suggestive only; until larger samples are studied, the issue remains open. In fact, Schadé (personal communication) has not replicated these results, finding no significant differences between ECT and IC animals with respect to dendritic branching. Nevertheless, Diamond *et al.*[44] reported that cortical depths in the visual cortex of ECTs were 6.2% higher than ICs'. Neurons per microscopic field for ECTs were 17% less than for ICs, while mean number of glia per field was only 6.7% less in ECT animals. With a decrease in neuron density and an increase in dendritic branching, it would be expected that glial density would show less relative increase. If neuron density has decreased by 17% and glia only by 6.7%, it stands to reason that the glial/neuron ratio per microscopic field has increased in ECT animals. Thus the tentative support given above is fully concordant with the kind of changes expected on logical and other experimental and ontogenetic evidence.

Before terminating this section on complexity, it is interesting to briefly discuss some miscellaneous observations with respect to this problem. Cajal[136] made the observation from his Golgi studies that the number of Golgi II cells (short axon) increased phylogenetically. Little exists, however, in the way of quantitative data. Mitra[121] counted the relative numbers of neuron types in the cortices of a variety of animals. His results are given in Table XIV in terms of percent of the total number of classified neurons.

TABLE XIV

RELATIVE PROPORTION OF NEURON TYPES (AFTER MITRA[121])

Species	*Pyramidal*	*Stellate*	*Fusiform*
Rabbit visual (adult)	66	31	3
Cat visual (adult)	60	35	5
Monkey visual (adult)	52	45	3
Cat somatosens. (adult)	63	35	2
Monkey motor	74	22	4
Monkey parastriate	66	29	5
Cat motor (adult)	85	9	6
Human prefrontal (adult)	72	26	2
Kitten visual (6 wks.)	66	31	3
Rabbit visual (17 days)	74	23	3
Rabbit visual (10 days)	85	13	2

The above data are not sufficient to prove this assertion, but surely they are suggestive. Sarkisov[117,118] has emphasized increased stellate cell number in the cortex of higher mammals, particularly in the supragranular layers. His findings are descriptive rather than quantitative, however. (See also Herrick[93], Bonin[13,15], Scheibel[153].)

Herrick[93] had concluded: '... the cortex contains innumerable nervous elements with shorter axons (type II neurons .....) which seem structurally not well adapted to participate in the more extensive associational patterns of the usual memory vestiges

but are so situated as to be able to function in a non-specific way by discharging their reserve energy locally into any associational system that at the moment may be activated within their field.' (pp. 317–318).

This statement brings to mind the A/S parameter suggested by Hebb[90], and the point made earlier regarding modulatory influences on the spike impulse of pyramidal neurons resulting in more control of fine movements. In other words, these cells would contribute to increased complexity through additional modulatory influences. This brings once again into focus the problem of increased complexity through additional connectivity and altered memory functioning through more associational units. Herrick was disposed to view the increase of Golgi II cells as acting as a sort of reinforcement for engrams, a view later made more explicit by Hebb. Herrick noted: 'Somewhere in the history of primate evolution, during the course of progressive elaboration of the apparatus of cortical associations, sufficient complexity of tissue and plasticity of organization was obtained to facilitate rapid learning, the retention of memories of single experiences and the abstraction from these of certain features common to all of them, and finally the integration of these common features into symbolic patterns. Symbolic thinking is a new kind of function, though the steps by which it was fashioned can probably be traced, just as we have already succeeded in charting in outline the progressive elaborations of the neurologic mechanisms employed.' (pp. 350–351).

It would be premature to conclude either that the increase in associational cortex and dendritic branching accounts for emergent properties, or, in contrast, that they account only for additional complexity along continuous lines, and that something else provides the basis for truly emergent behavior. (The transition from ape to man.) The assertion of any *either-or* proposition at this stage of knowledge of the nervous system and its functioning would be clearly uncalled for. While it is true that the latter position will be favored in the second part of this review, and the latter section on complexity is interpreted as supporting this position, it must not be overlooked that the possibility remains for a complementation of views rather than an 'either-or' position. The additional units, and increased connectivity might, in addition to providing for more complexity along continuous lines, provide the basis for emergence as well. This latter view would demand some 'rubicon' model, however, since a minimal number of such influences would be necessary to make the transformation into an emergent behavioral process. It will have to be admitted that the problem remains. The proceeding section has merely tried to throw some light on the matter and suggests the kinds of evidence which might relate to the problem. The earlier discussion on microcephaly showed that cerebral mass or association areas did not explain the specifity of behavior, however rudimentary. It is the nature of the specifity which is of most interest. Obviously, it is the full complement of cortical tissue connected with complexity and modulation that permits the full repertoire, the full expression of human behavior. The ability of humans to remain human with extremely large ablations of cortical tissue suggests the relevance of this cortex to complexity operations rather than basic emergent properties. But then again, the human undergoing such operations were humans from the start, and had progressed

for some time as open systems in particular symbolic environments. The ablation data, while suggestive, cannot answer the problem.

As will become more salient in the next article, the distinctions made with respect to these aspects of behavior have heuristic merit, and in addition to the neurological evidence sketched this far, are appropriate for a beginning analysis of the behavioral evidence for hominid evolution. But it must be emphasized again that the discussion given, with respect to complexity, has been suitable only for a general level of conceptual analysis of certain aspects of behavior. It is not intended to stand as a total explanation of *all* aspects of complex human behavior. For this purpose, a systems approach would be more suitable. It would have to be an approach which took up the particular nature of the environments, social codes, motivations and genesis, and like problems. This is clearly beyond the scope of this review.

SUMMARY

To summarize the pertinent findings of this review, it may be said that reorganization of the primate cortex is indicated at a number of levels. Intrinsic thalamic afferents to the cortex increase with increasing association cortex. A series of various indices were given to illustrate this, such as the A/S ratio, and various surface areal and volume measures. At a more molecular level, comparative, ontogenetic and experimental evidence showed that increasing brain size is associated with decreased density, or increased gray cell coefficient, and that this is paralleled by increase in dendritic branching parameters. With the increase in dendritic branching, and thus more synapses through more connectivity, the complexity of behavior was seen to increase. The experimental evidence tried to show this point. Finally, it was suggested that this interpretation based on a conceptual distinction between complexity processes in behavior and that of emergence with respect to the code or arbitrary representative factors, could be integrated with the quantitative-emergence views of Hebb and Herrick, as well as ablation data. While little space was devoted to the neurophysiological aspects, enough had been presented concerning Clare and Bishop's view to find a means of integration of this aspect with the purely anatomical. The following sections will attempt to relate these discussions of reorganization at all the levels discussed to some aspects of the emergent, specifically human kind of behavior. Three hypotheses will suggest relevant neurological mechanisms that might relate to behaviors of kind rather than degree. This will then be followed by a discussion of hominid evolution and the fossil record, and an attempt will be made to relate aspects of this dichotomy to the evolution of man. While the interpretations of this section may be open to question, one fact clearly emerges from all this discussion: a comparison of cerebral units (volumes) is not a comparison of equal units. Endocasts cannot show this. Discussions of the brain in terms of total mass must be replaced by more molecular appreciation of the range of structural and behavior changes for different extant primate species. Quantification is but one necessary early step toward systems synthesis of the naturalistic and anatomical data.

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