# LANGUAGE IN A SOCIOBIOLOGICAL FRAME

Karl H. Pribram

# LANGUAGE IN A SOCIOBIOLOGICAL FRAME*

Karl H. Pribram

*Department of Psychology*
*Stanford University*
*Stanford, California 94305*

## THE STRUCTURE OF LANGUAGE

In an earlier paper published by The New York Academy of Sciences I addressed the contributions that comparative neurobehavioral research had made to delineating the structure of language.[1] That report distinguished two quite separate aspects of animal communication, each of which has at its disposal a different neural mechanism. One form, the *symbolic*, relates the communicative act to the internal state of the animal and is in a sense arbitrary and synthetic with respect to the situation in which it occurs. The other constructs *signs* by differentiating the features distinctive to the situation, thus assigning attributes and characterizing it.

Further, the earlier report developed the thesis originally proposed by Vowles[2,3] and amplified by Beer (Ref. 4 and this volume) that three levels of neural mechanisms could be identified in the production of both sign and symbol. At the deepest level, which is shared by vertebrates and invertebrates alike, presymbolic processes are based on recurring regularities such as those that make up circadian and ultradian rhythms. Rudimentary significations, by contrast, are at this level based on stochastic finite-state Markov-type mechanisms. In vertebrates, presymbolic and presignificant communications are constructed from these primitive operations by phrase structure hierarchies, which in man are subject to still another type of operation leading to the transformational capacities necessary to the construction of linguistic signs and symbols.

## SIGN AND SYMBOL IN HUMAN LANGUAGE

On this occasion I want to concentrate on the relevance of these grammatical distinctions to the origins of human language. The first of a series of questions that stems from the earlier analysis is whether symbolic or signifying processes are more rudimentary and pervasive in the development of man's linguistic abilities. The logical possibilities are, of course, four: Human language is derived from 1) symbolic expression, 2) from assigning features, 3) from both, 4) from neither. Let us explore these alternatives.

The facts supporting biological evolution make it unlikely that human language has sprung *de novo* with no relationship to subhuman primate forms of communication. I shall therefore, as an act of faith, dismiss this alternative and bolster the decision by arguments to be made in favor of the other possibilities.

Human language is nothing if not arbitrary. The sheer variety of languages, the different forms of expression used within a language to convey a meaning, the almost universal use of an alphabet (an arbitrary code), and the variety of alphabets all attest to the fact that the form of human language is symbolic.

Equally apparent is the fact that the content of human language is much more developed and richer in signifying, characterizing the attributes of the environment than in expressions relating the dispositions of the communicants. The likelihood is therefore remote that human language is derived exclusively from either the symbol or the sign aspect of animal communication. Somehow, both have become fused in the process by which man communicates.

The linguistic analysis of language and its development in the human infant make it imperative to view human communication as a fusion of symbolic and significant operations. Man's languages have two primary characteristics: they provide a prolongation of reference[5] and they are productive.[6] Both these characteristics are found in primordial form in the signs and symbols of nonhuman primates, but the extent to which they are developed in man is hardly foreshadowed by these rudiments. Even a retarded child before he is a few years old will spin sentences that are so far beyond the nonhuman level[7] that he can easily be identified as a member of the genus *Homo* by this action alone.

A child begins verbalization, just as does the nonhuman primate, with what are called holophrases: single utterances signifying something or symbolizing some state. Linguists have not classified holophrases in this fashion, but my own observations make clear to me that holophrases are of two kinds: more or less continuous grunts, coos, and explicatives which refer to the baby's internal state, and shorter, repetitive syllablelike sounds often accompanied by directive gestures that indicate something about the world the baby sees, hears, touches, or tastes.

At about the age of two years the holophrases become more precise and their referent more readily distinguishable, until couplings of holophrases occur. Such couplings—and later strings—are also observed in chimpanzee utterances. But the child quickly goes on to make *propositions*, something which, at the time this is written, has not been observed in ape-language.

Propositions or sentences develop around predication. Predication implies another function, that of nominalization or noun-formation. The line of development from sign to nouns as found in human utterances seems to be straightforward enough. Verbs such as *run, catch, flow*, and so on, and other parts of speech such as adjectives and adverbs give somewhat more trouble, until one realizes that they also signify existences and are therefore forms of nominalization. In a sense, such verbs are names for actions rather than things, the adjectives and adverbs are names for attributes—and the difference lies in the number of transformations over which things, attributes, and actions remain invariant.

Predication, however, is premised on more than existence. Predication makes a statement about occurrences, the truth or falsity of a proposition, the rules that proclaim the is-so and the is-not of such occurrences. *Black sand water* is a string of holophrasticlike utterances that a chimpanzee might make pointing to a beach in Hawaii. I should understand him. A child would say, "Look, the sand *is* black *next to* the water." He would be very upset if you explained to him that he is subject to an illusion caued by the heating of the air over an asphalt landing strip. It would be foolhardy to try to communicate the difference between propositions "The sand is black next to water" and "The illusion of water is produced on asphalt by heat" to a chimpanzee.

The point here is that predication is premised on symbolization. Predication is an expression by the human organism that this is the way it is (or is not). According to this view, predication is derived from the ordering of events into a temporal structure (a rule) that groups occurrences in terms of equilibrations and disequilibrations of the brain. Some groupings are right, others wrong. And sometimes the grouping becomes rather complicated before it feels right.

Nominalization derived from signifying (through predication) provides the extended referent in human language: "purple people eaters," for example. Predication derived from symbolizing (and utilizing nomination) provides human language with its productivity: the variations of groupings (purple people or purple eaters?) that can feel right could be almost infinite.

How is this fusion between symbol and sign performed? How do the parts of the brain involved in symbol construction and those involved in the construction of signs come to interact? Is a new cortical formation responsible for the fusion, do corticocortical connections develop that are poorly, or not at all, functional in subhumans, or is the transformation accomplished subcortically? Each of these possibilities has been seriously entertained by brain scientists, and there is some evidence in support of each. Again, let us examine them in turn in order to obtain some grasp regarding where inquiry must go to resolve the problem.

## WHAT IS NEW IN MAN'S BRAIN?

There is little question that in the human cerebral cortex, areas can be found that are either absent in subhumans or present only in rudiment. The large development of frontal cortex in terms of man's vaulting forehead was already noted by the early phrenologists. Equally impressive is the growth of the posterior nonprojection cortex centering on the angular gyrus, the confluence of parieto-, temporal, and occipital cortical formations.[8] Does the quantitative increase in these cortical structures herald the qualitative transformational change expressed as human language?

My answer to this question is a tentative no. I reason as follows: if the cortical growth is *per se* to be responsible for the development of human language, evidence should lead to two major language "centers": one well forward in the frontal cortex, the other in the tissues around the angular gyrus. The evidence for and against a major category of aphasia centered on the third frontal convolution is critical. If Broca's[9,10] aphasia is to be given weight equal to Wernicke's (see Pribram[15]), the idea that new cortical accretions are responsible for human language is tenable. So let us look at the problem handed us by Broca.

All of the evidence[11-15] shows that expressive aphasia does not result from damage as far forward as Broca's part of the third frontal convolution. Further, to fit the facts of a cortical topography peculiar to man, even the third frontal convolution is too ventral and posterior a location for a new language "center" to be developed in tissue not present in subhuman primates. Electrical stimulation of the third frontal convolution, in all primates including man, yields tongue movements. This is not the locus of the new cortical accretion.

The place of the territory around the angular gyrus in the development of human language is not so easily disposed of. Aphasic symptoms, as we have seen, result when the cortex of the angular gyrus is damaged. But again the match is imperfect (see Bogan and Bogan, this volume). All of the evidence points to the posterior part of the superior temporal gyrus as the locus involved in Wernicke's syndrome and holds that Wernicke's is the major, central, or primary aphasia. Also, as in the case of the frontal cortex, although the fit is better, the angular gyrus is not exactly the place of maximum new accretion of cortex in man; it is somewhat too close to the Sylvian fissure to be equated with the considerable anatomical development of intrinsic nonprojection cortex.

These mismatches, although some of them are slight, give me an uneasy feeling when the origin of human language is attributed simply to the growth of new areas in the frontal and posterior intrinsic cortex.

## CONNECTIONS AND DISCONNECTIONS

If not the new areas directly, perhaps their development brings with it new functional pathways that allow symbol and sign aspects of communication to interact. This possibility is detailed in the aphasia literature under the heading of disconnection syndromes most recently advocated by Geschwind.[16] Earlier versions of the disconnection view were voiced by Freud,[17] Liepman,[18] and Dejerene.[19] All these authors adduce specific case histories in support of their suggestion that one or another major pathway is pathologically involved in the production of a language (or language-related) disorder. Unfortunately, to date, comprehensive and quantitative behavioral analyses such as those produced by Bay[20] and Hecaen and Angelergues[21] have not been performed on such patients. Often the anatomical verification of the lesion also leaves a good deal to be desired: multiple damage is often reported when a single focus is held responsible for the disorder; histological serial analysis of the entire brain is seldom performed.

Arraigned against the corticocortical disconnection hypothesis are all of the subhuman primate experimental findings I reviewed in my earlier paper. In the monkey it appears that intrahemispheric corticocortical connections play a minor, if any, role in the organization of the psychological process. But monkeys do not talk the way men do. Is the difference in importance of corticocortical pathways the critical reason why they do not? Or do the corticosubcortical connections shared by all primates, which, up to now, have been ignored, play the critical role?

## THE CENTRENCEPHALIC HYPOTHESIS

Convergence of significant and symbolic processing at some subcortical locus or loci is, on the basis of subhuman evidence, a serious contender as an explanation for the emergence of human language. Subcortical formations are rarely given more than cursory inspection when the brains of aphasics are studied. When the lesion is caused, as it so often is, by disease of the middle cerebral artery, the basal ganglia, parts of the thalamus, and many fiber tracts are affected. Penfield, among others, has opted, on the basis of his experience, for a centrencephalic mechanism in the production of human language (Ref. 13; cf. Robinson, this annal). Careful surgical excisions of cortex so rarely produce lasting changes in man or monkey that one is literally driven to the subcortical formations for an answer to questions of localizing the site of disturbances.

The one exception to this is, of course, Wernicke's zone in the posterior part of the superior temporal gyrus (see Bogen and Bogen, this volume). Here, because neurosurgeons tread with extreme caution, data are hard to come by, but opinion is strong and to the point: in the adult, at least, damage is not to be hazarded.

The centrality of Wernicke's zone and the possibility that subcortical convergences are critical to the production of human language make up the centrencephalic hypothesis. This hypothesis takes strength from the subhuman primate experimental results that show that the nonprojection cortex associated with the auditory mode lies in the midtemporal region;[22] that most likely, this cortex exerts its role in audition through efferents coursing to subcortical stations in the auditory projection systems;[23,24] that removal of this cortex results even in monkeys in the inability to discriminate vowel sounds;[25] and that, contrary to any other cortical removals in subhuman primates, unilateral damage plays havoc with certain types (sequences) of auditory discriminations.[25,26]

The subcortical locus upon which the symbolic and signifying processes can conveniently converge has not yet been established. From the results of experiments on nonhuman primates, however, the basal ganglia and related nuclei in the upper midbrain are the best candidates. These are motor structures involved in producing the muscular settings necessary to action. It should therefore not be too surprising that communicative and linguistic acts also depend on the function of these motor structures.

Thus both the corticocortical disconnection and the centrencephalic hypotheses continue to be tenable though unproven. Techniques to test them are available. Quantitative behavioral evaluations of aphasic patients and serial histological reconstructions of their lesions and resulting degeneration (e.g. retrograde change in the thalamus) will go a long way toward supporting or disproving the disconnection hypothesis. Especially important is a comprehensive evaluation of whether language-related and nonlanguage disturbances are correlated or whether they are separable when a disconnection syndrome is suspected.

With regard to the centrencephalic hypothesis, the current vogue in electrical stimulations of deep brain structure should uncover evidence regarding possible subcortical language mechanisms. Such studies should, over the next decade, provide the necessary crucial facts to test the centrencephalic hypothesis.

### The Inside and the Outside of Language

Finally, the alternative must be entertained that *the convergence of sign and symbol does not take place primarily in the brain at all.* It is possible, and indeed likely, that the auditory mechanism in primates, because of the development of cerebral dominance, has extended to encompass such a large share of the cortical and subcortical process that all but the purely symbolic aspects of human language are accounted for, as suggested by Piere Marie,[27] Bay,[20] and Hecaen and Angelergues[21] (Ciba Foundation Symposium, 1964). A central language processor based on the auditory projection system and its associated cortex would account for the signifying aspects of language. With use of adjacent accessory "poles" in the precentral, parietal, and occipitotemporal cortices, expressive, somatic, and visual communication could be established simply by an overlap of functional areas. The primary auditory projection is ideally placed for such overlap. The derivation of the acoustic system from gill and jaw is reflected in the cortex by the fact that auditory projections encompass both somatic area 11 superiorly and motor face and mouth areas anteriorly.[28] Even when such proximity does not exist, "associations" within the primary projection are present innately or established through learning.[29] Electrocortical evidence obtained in the visual mode shows cells to be present that react to auditory stimulation,[30] to the presence or absence of reinforcement, and to the intention of making a specific movement.[29,31]

The route by which such "associations" are established is unknown, but in the auditory cortex the path need not at least be long.

These considerations, however, apply only to significant communication. How, then, does the symbolic aspect of human language become involved in communication? It is possible that this occurs only through the environment— that there is no corticocortical connection nor subcortical convergence involved in all. When the neural sign system becomes sufficiently powerful (i.e., has sufficient memory and coding capability), it can treat the tokens of expression (of others and of self) as signs, signifying social rather than physical situations. This power, of course, would be immensely enhanced when memory is augmented externally; i.e., when symbols become recorded and treated as signs.

Should this cultural hypothesis of human language development prove to be correct (and I believe there is much to be said for it), the brain problem of human language production would be immensely simplified. First, the fact that aphasics can still express their feelings through gestures, expletives, and "emotional" language and simple song (which elsewhere in this annal have been shown to be related to the limbic forebrain (e.g., Robinson, Myers, this annal) would fit the conception that the human language system is primarily sign-based. Second, the fact that the arbitrariness of human language is so culture-bound would tend to validate the cultural hypothesis. Third, the overwhelming evidence of the central place of Wernicke's syndrome in the aphasic complex would support a sign-based view of the problem. The several dimensions along which the aphasic syndrome may vary would be attributable to invasion of areas adjacent to Wernicke's. The operation of the language-producing mechanism would involve the subcortical connections by way of which Wernicke's area operated on the auditory projection system. Lesions of such connections would impair language processing. Still to be tested is the possibility that more remote corticocortical connections might also be important, especially in rare and unusual syndromes--perhaps even idiosyncratically with patient-to-patient variation dependent on the particular experience of the individual.

According to this view, then, human communication, just like animal communication, is in the first instance bimodal. Only through culture do the symbolic and significant aspects of language become interwoven, and the evidence from aphasia suggests that the interweaving takes place mostly within the neural systems serving communication by signs.

### BRAIN ORGANIZATION AND MEANING

In addition to insights into the development of the grammatical structure of human language, studies of brain function can teach us a good deal about the fundamental issues of the organization of meaning. In Chomsky's terms (this annal), what has been discussed so far concerns the relation between superficial and deeper structure. If the analysis has been correct that this relationship initially devolves on the formation of prelinguistic signs through hierarchical phrase-structuring, the question to be explored is the nature of features and what makes them distinctive.[6,32,33]

Units in the nervous system have been discovered that are sensitive to features of the environment (e.g., Mountcastle;[34] Werner;[35] Evarts;[36] Hubel & Wiesel;[37] Barlow & Hill;[38] Spinelli et al.[39]). Further, these features appear to be organized into different configurations in each of the hemispheres of the human brain. Thus after the age of seven or thereabouts, damage to the right hemisphere of most people impairs primarily spatial relationships, whereas damage to the left hemisphere impairs the linguistic abilities, which are the concern of this paper. Sperry,[40] in an elegant series of experiments, has demonstrated the separateness of these functions in patients whose hemispheres have been severed from one another by sectioning of the major commisure, the corpus collossum, that ordinarily connects them.

But these important contributions also pose problems of interpretation to neurolinguists. Are we to search for a different brain cell for each distinctive feature of language? If so, do these brain cells respond to the feature innately, or do they become responsive only through experience? Further, most of the feature-sensitive units that have been discovered so far deal with the spatial aspects of input. How do such feature-sensitivities relate to linguistic structure in a hemisphere that supposedly does not process these features?

Many of these puzzling problems are resolved if we look at the evidence from a somewhat different theoretical perspective. The ordinary interpretation that feature-sensitive cells in the brain serve as "detectors" for that feature has been found wanting. Thus, for instance, Pollen and Taylor[41] have shown that the output of "complex" cells of the visual cortex (which are assumed to be detectors of lines of specified length and orientation) is not invariant across all transformations of input other than orientation. In fact, changes of luminance, width of line, number of lines and their spacings all influence the cell's output. Thus only a network of neurons could separate their orientation specificity from that to luminance, for example. Several groups of investigators[41-43] have shown that such cells are in fact more accurately stated to be sensitive to spatial frequencies than to lines *per se*, and that it is therefore in error to think of them as simple line "detectors."

The change from sensitivity for lines to one for spatial frequency has major consequences. As discussed elsewhere,[15,44-46] a spatial frequency analysis of light just like the temporal frequency analysis of sound (by the auditory system) is accomplished in the domain of continuous oscillations and not in the digital domain in which present-day computers operate almost exclusively. This shift in emphasis allows alternate hypotheses to be formulated regarding what might distinguish a feature in speech and what the organization of deep structure might look like in the brain.

Phoneticians have in fact already made it clear that the distinctive features of spoken language are most readily analyzed in terms of the wave forms generated by the vocal apparatus—the vocal cords, larynx, oral cavity, tongue, and lips. For example, one recent study was able to decompose speech sounds into some six to eight components by performing a Fourier analysis, taking into account both spatial and temporal relations.[47] And the Haskins group has for years been simulating sounds by using spectral techniques (e.g. Liberman *et al.*[48]).

If, in fact, the distinctive features by which linguistic communications take place are to be identified as wave forms, perhaps the deep structure of such communications is to be found in the wave mechanical domain. The computer, with its programmable digital information-processing capabilities, has been of great service both as a model and in data analysis with regard to syntactic superficial structure. Is there not an information-processing system that can serve with equal value as a model (and perhaps in due time in data analysis) in our search for the deep semantic structure of meaning?

Optical information-processing systems are just beginning to be recognized as useful analogues in studies involving the oscillatory domain. Aside from their image-constructing capacity, they partake in organizations characterized by the distribution of information produced by interference among wave fronts. This distributed aspect of their organization makes them attractive to brain scientists who have been puzzled for years by the apparent distribution of input for storage over reaches of brain surface, rendering their functions resistant to local damage.

Organizations of optical information-processing systems in which information is distributed are called *holograms*.[49,50] The proposal has therefore been made that the spatial and temporal frequency analyses performed by the brain are indicative of a holographiclike brain process.[51,15,46] But it must be borne in mind, of course, that for neural holograms only the organization of the paths taken by light in optical systems serves as the model. The energy involved in neural excitation is electrical, not photic.

The suggestion to be entertained here is therefore that deep structure is, in the final analysis, semantic structure and that semantic structure derives from a distributed neural organization akin to that found in the holograms of optical information-processing systems. Note that deep structure is conceived to be derived

from a holographic organization and is not synonymous with it. Syntactic structures, as delineated in the earlier paper, partition map the holographic-distributed store of information into useful, meaningful organizations.

In biology, this mapping of a distributed, more or less homogeneous matrix into useful hierarchical and heterarchical organizations is commonplace. Thus the morphogenetic field becomes organized into useful structures by the action of inducers that derepress the potentialities of DNA molecules embedded in those fields. Thom[52] has recently developed a topological mathematics to describe the mappings of the morphogenetic field. Applying this to language, each distinctive feature would be occasioned by continuous interactions (i.e., wave forms generated in the vocal apparatus), but the ensuing stabilities, the distinctive features *per se*, would result when the interactions—the relationships—temporarily gel into nonlinearities, a process Thom calls a "catastrophe."

The difference between right and left hemisphere function is ordinarily conceptualized in terms of whether processes leading to image formation or to nonlinear catastrophic processes are emphasized. But more likely a simpler distinction based on sensory-motor (e.g., auditory-verbal vs. visuosomatic) mode is responsible. Reynolds[53,54] has suggested that differential use of the hands by primates has necessitated specialization of function between their cerebral hemispheres (see also Steklis & Harnad, this annal). Abler[55] has suggested further that when such specialization occurs, a problem arises for innervation of midline structures such as the tongue. He has experimentally demonstrated that one innervation (usually the right in right-handed persons) must dominate, or conflicting signals from the two hemispheres disrupt function. In short, once hemispheric specialization has occurred, dominance must follow if the midline structures involved in speech are to function harmoniously. And dominance entails some catastrophic-like "decisional" mechanism which more or less stably "takes over" the innervation of the midline. In discussion, we have even entertained the possibility that the left hemisphere has the edge in achieving dominance because the heart is located on the left side of the body and thus provides some slight advantage to the embryo's circulation. Wada *et al.*[56] (see also LeMay, this annal) have shown that asymmetries in the size of the cortex of the supratemporal plane already exist at birth—although some puzzling sex differences also emerge in these studies: the difference is greater in males. There are, however, as yet only anecdotal data on differences in early cerebral circulation to support such a hypothesis.

## CONCLUSION: RESOLUTION OF THE PARADOX

To summarize these arguments, two views of human language production have been presented. One holds that the symbolization of signs takes place within the brain. The other proposes that symbols must be externalized in culture to be operated upon by the neural process that produces signs. The latter view makes the production of ordinary human language a more or less unitary process and has substantial support. The view that the brain is involved in bringing together sign and symbol depends on the presence of corticocortical connections or corticosubcortical convergences the function of which in language have yet to be firmly established as existing.

I am thoroughly convinced of both views and believe that resolution of the paradox lies in making a distinction between two uses of human language: the communicative and the thoughtful. When we restrict ourselves to testing the communicative capacity of brain-damaged man, we come to an essentially unitary

position on aphasia (cf. Marin & Saffran, this volume). When, however, we ask questions about the *structure* of language disturbed by brain damage, or when we introspect and study our own and other people's verbal reports of introspection, we find a rich internal commerce between sign and symbol that at any moment, at least, needs no external crutch (though such a crutch may well be necessary to the development of this commerce).

I believe that the communicative use of human language is to a considerable extent an elaboration of animal sign communication, a relatively unitary process based on constructions of features from invariances occurring in stochastic events. Such significant communications are dependent on culture for their connectivity with symbolic processes; however, a parallel cultural elaboration of context-dependent symbolic communication based on recurring variances also occurs, and this is manifest through nonverbal (gestural-kinesis) and nonlinguistic (tonal quality, intensity) verbal channels.[57] The significant (cerebral convexity) and symbolic (frontolimbic) communicative processes most likely develop separately and can be maintained separately or brought together through experience and thought. Bateson has, in fact, suggested that in every human communication these two types of processes must be and are attended and that psychopathology results when the significant and symbolic messages continuously conflict[58,59] (see also Tanner, this annal).

Thus the thoughtful, reasoned use of language appears to be structurally multidimensional. The question remains an open one, whether this multidimensionality depends primarily on brain connectivity or whether it is largely the result of cultural factors external to the brain. Evidence in support of both possibilities exists and has been reviewed here.

I have therefore come to the conclusion that man's linguistic complexity must be due to the type of brain connectivity that has made possible the richness of all his culture. Culture is composed—made—by successions of acts. Thus the brain's motor mechanism must be involved with production of culture, and so I look to them for the necessary connectivity. Both the frontolimbic and posterior intrinsic cortex, so highly developed in man, have been shown in nonhuman primates to exert their influence via subcortical motor structures. I thus place my faith in the centrencephalic hypothesis as the most likely to account for the fact that the complexity of language is determined both by man's superior brain and his superior culture (which, in turn, is fashioned by actions guided by his superior brain).

## REFERENCES

1. Pribram, K. H. 1973. The comparative psychology of communication. Ann. N.Y. Acad. Sci. 223: 134–143.
2. Vowles, D. M. 1970. The psychobiology of aggression. Lecture delivered at the University of Edinburgh. University Press. Edinburgh, Scotland.
3. Vowles, D. M. 1970. Neuroethology, evolution and grammar. *In* Development and Evolution of Behavior: Essay in Memory. T. C. Schneirla, Ed. W. H. Freeman and Co. San Francisco, Calif.

4. BEER, G. 1974. Ethology—the Zoologist's Approach to Behavior. Parts 1 and 2. Tuatara. Vol. 2: 170–177; Vol. 12: 16–39.
5. BRONOWSKI, J. 1967. Human and animal languages. *In* To Honor Roman Jakobson, Essays on the Occasion of his Seventieth Birthday. Vol. 1: 374–394. Mouton. Paris, France.
6. JAKOBSON, R. 1956. Two aspects of language and two types of aphasic disturbances. Part II. *In* Fundamentals of Language. R. Jakobson and M. Halle, Eds.: 53–82. Mouton. The Hague, The Netherlands.
7. KLIMA, E. & U. BELLUGI, 1972. The signs of language in child and chimpanzee. Communication and Affect: A Comparative Approach T. Alloway, L. Kramer and P. Ploner, Eds.: 67–96. Academic Press. New York, N.Y.
8. BLUM, J. S., K. L. CHOW & K. H. PRIBRAM. 1950. A behavioral analysis of the organization of the parieto-temporo-preoccipital cortex. J. Comp. Neurol. 93: 53–100.
9. BROCA, P. 1861. Remarques sur le siège de la faculté du langage articulé suivies d'une observation d'aphemie (perté de la parole). Bull. Société Anatomique de Paris 36: 330–357.
10. BROCA, P. 1878. Anatomie comparée des circonvolutions cerebrales. Le grand lobe limbique et la scissure limbique dans la serie des mannifères. Rev. Anthrop. 1: 385–498.
11. LURIA, A. R. 1964. Factors and forms of aphasia. *In* Disorders of Language. A. V. S. de Reuck and M. O'Connor, Eds.: 143–161. Ciba Foundation Symposium. Little Brown and Co. Boston, Mass.
12. LURIA, A. R. 1966. Higher Cortical Functions in Man. Basic Books. New York, N.Y.
13. PENFIELD, W. & L. ROBERTS. 1959. Speech and Brain Mechanisms. Princeton Univ. Press. Princeton, N.J.
14. TEUBER, H. L. 1960. *In* Handbook of Physiology: Neurophysiology, Vol. 3: 1595–1668. J. Field, H. W. Magoun, V. E. Hall, Eds. American Physiological Soc. Washington, D.C.
15. PRIBRAM, K. H. 1971. Languages of the Brain: Experimental Paradoxes and Principles in Neuropsychology. Prentice-Hall, Inc. Englewood Cliffs, N.J.
16. GESCHWIND, N. 1965. Disconnexion syndromes in animals and man. Brain 88: 237–294.
17. FREUD, S. 1953. On Aphasia. Authorized and translated by E. Stengel. International Univ. Press. New York, N.Y.
18. LIEPMAN, H. 1912. Anatomische befunde bia aphasischen und apraktischen. Neurol. Zbl. 31: 1524–1530.
19. DEJERENE, J. 1914. Semiologie des Affections du Systeme Nerveux. Masson et Cie. Paris, France.
20. BAY, E. 1964. Principles of classification and their influence on our concepts of aphasia. *In* Ciba Foundation Symposium on Disorders of Language. A.V.S. de Reuck and M. O'Connor Eds.: 122–142. Little Brown and Co. Boston, Mass.
21. HACAEN, H. & R. ANGELERGUES. 1964. Localization of symptoms in aphasia. *In* Ciba Foundation Symposium on Disorders of Language. A. V. S. de Reuck and M. O'Connor, Eds.: 223–246. Little Brown and Co. Boston, Mass.
22. DEWSON, J. H. III, K. H. PRIBRAM & J. LYNCH. 1969. Effects of ablations of temporal cortex upon speech sound discrimination in the monkey. Exp. Neurol. 24: 591–597.
23. DEWSON, J. H. III, K. W. NOBEL & K. H. PRIBRAM. 1966. Corticofugal influence at cochlear nucleus of the cat: Some effects of ablation of insulartemporal cortex. Brain Res. 2: 151–159.
24. NOBEL, K. W. & J. H. DEWSON III. 1966. A corticofugal projection from insular and temporal cortex to the homolateral inferior colliculus in cat. J. Aud. Res. 6: 67–75.
25. DEWSON, J. H. III & A. COWEY. 1969. Discrimination of auditory sequences by monkeys. Nature 222: 695–697.
26. DEWSON, J. H. III & A. C. BURLINGAME. 1975. Auditory discrimination and recall in monkeys. Science 187: 267–268.
27. MARIE, P. 1926. Trauvaux et Memoires. Vol. 1. Masson. Paris, France.
28. PRIBRAM, K. H., B. S. ROSNER & W. A. ROSENBLITH. 1954. Electrical responses to acoustic clicks in monkey: Extent of neocortex activated. J. Neurophysiol. 17: 336–344.
29. PRIBRAM, K. H., D. N. SPINELLI & M. C. KAMBAK. 1967. Electrocortical correlates of stimulus response and reinforcement. Science 157: 95, 96.
30. SPINELLI, D. N., A. STARR & T. BARRETT. 1968. Auditory specificity in unit recording from cat's visual cortex. Exp. Neurol. 22: 75–84.

31. SPINELLI, D. N. & K. H. PRIBRAM. 1970. Neural correlates of stimulus response and reinforcement. Brain Res. 17: 377-385.
32. JAKOBSON, R. 1964. Towards a linguistic topology of aphasic impairments. In Ciba Foundation Symposium on Disorders of Language. A. V. S. de Reuck and M. O'Connor, Eds.: 21-42. Little Brown and Company. Boston, Mass.
33. JAKOBSON, R. 1966. Linguistic types of aphasia. In Brain Function. Vol. 4. Speech, Language and Communication. E. C. Carterette, Ed.: 67-91. Univ. California Press. Los Angeles, Calif.
34. MOUNTCASTLE, V. B. 1967. Modality and topographic properties of single neurons of cat's somatic sensory cortex. J. Neurophysiol. 20: 408-434.
35. WERNER, G. 1974. Neural information with stimulus feature extractors. In The Neurosciences; Third Study Program. F. O. Schmitt and F. G. Worden Eds.: 171-183. MIT Press. Cambridge, Mass.
36. EVARTS, E. V. 1967. Representation of movements and muscles by pyramidal tract neurons of the precentral motor cortex. In Neurophysiological Basis of Normal and Abnormal Motor Activities. M. D. Yahr and D. P. Purpura, Eds.: 215-254. Raven Press. Hewlett, N.Y.
37. HUBEL, D. H. & T. N. WIESEL. 1962. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J. Physiol. 160: 106-154.
38. BARLOW, H. B. & R. M. HILL. 1963. Selective sensitivity to direction of movement in ganglion cells of the rabbit retina. Science 139: 412-414.
39. SPINELLI, D. N., K. H. PRIBRAM & B. BRIDGEMAN. 1970. Visual receptive field organization of single units in the visual cortex of monkey. Intern. J. Neuroscience 1: 67-74.
40. SPERRY, R. W. 1970. Perception in the absence of the neocortical commissures. In Perception and Its Disorders. Res. Public. ARNMD. Vol. 48: 123-138.
41. POLLEN, D. A. & J. H. TAYLOR. 1974. The striate cortex and the spatial analysis of visual space. In The Neurosciences: Third Study Program. F. O. Schmitt and F. G. Worden, Eds.: 239-247. Cambridge, Massachusetts: MIT Press. Cambridge, Mass.
42. CAMPBELL, F. W. & J. B. ROBSON. 1968. Application of Fourier Analysis to the visibility of gratings. J. Physiol. 197: 551-556.
43. GLEZER, V. D., V. A. IVANOFF & T. A. TSCHERBACH. 1973. Investigation of complex and hypercomplex receptive fields of visual cortex of the cat as spatial frequency filters. Vision Res. 13: 1875-1904.
44. PRIBRAM, K. H. 1974. How is it that sensing so much we can do so little? In The Neurosciences: Third Study Program. F. O. Schmitt and F. G. Worden, Eds.: 249-261. MIT Press. Cambridge, Mass.
45. PRIBRAM, K. H. 1974. Holonomy and structure in the organization of perception. In Proc. Conf. Images, Perception and Knowledge. University of Western Ontario. London, Canada. In press.
46. PRIBRAM, K. H., M. NUWER & R. BARON. 1974. The holographic hypothesis of memory structure in brain function and perception. In Contemporary Developments in Mathematical Psychology. R. C. Atkinson, D. H. Drantz, R. C. Luce, and P. Suppes, Eds.: 416-467. W. H. Freeman and Co. San Francisco, Calif.
47. PORT, R. 1973. Oral presentation on speech mechanisms and Fourier analysis at Univ. Minnesota Conf. Cognition, Perception and Adaptation. Minneapolis, Minn.
48. LIBERMAN, A. M., F. S. COOPER, D. P. SHANKWEILER & M. STUDDERT-KENNEDY. 1969. Perception of the speech code. In Brain and Behavior. Vol. 4. Adaptation. K. H. Pribram, Ed.: 105-148. Penguin Books. Harmondsworth, Middlesex, England.
49. GABOR, D. 1969. Information processing with coherent light. Optica Acts 16: 519-533.
50. STROKE, G. W. 1969. An Introduction to Coherent Optics and Holography. Academic Press. New York, N.Y.
51. PRIBRAM, K. H. 1966. Some dimensions of remembering: Steps toward a neuropsychological model of memory. In Macromolecules and Behavior. J. Gaito, Ed.: 165-187. Academic Press. New York, N.Y.
52. THOM, R. 1972. Stabilite Structurelle et Morphogenese. W. A. Benjamin, Inc. Reading, Mass.
53. REYNOLDS, P. C. 1974. Handedness and the evolution of the primate forelimb. Presented at Conf. Human Brain Function. Neuropsychiatric Institute. U.S.C.A.

54. REYNOLDS, P. C. Play, language and human evolution. *In* Play: Its Evolution and Development. J. Bruner, A. Jolly and K. Sylva, Eds. Penguin Books. London, England.
55. ABLER. Personal communication.
56. WADA, J., L. CLARK & I. HAMM. 1973. Asymmetry of temporal and frontal speech zones. Presented 10th Inter. Congr. Neurology. Barcelona, Spain.
57. BIRDWHISTELL, R. I. 1954. Introduction to Kinesics. Washington, D.C. Department of State. Foreign Service Institute. Reprinted Univ. Louisville. 1954. Louisville, Ky.
58. BATESON, G. 1963. Exchange of information about patterns of human behavior. *In* Information Storage and Neural Control. W. S. Fields and W. Abbott, Eds.: 1-14. Charles C Thomas. Springfield, Ill.
59. BATESON, G. *In* Pragmatics of Human Communication. P. Watzlawick, J. Beavin and D. Jackson, Eds. W. W. Norton & Co. New York, N.Y.