Gradiental Approach to Neocortical Functional Organization*

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ABSTRACT

Examination of neuropsychological syndromes suggests strong congruence between neuroanatomical and functional neocortical metrics. Two cortical syndromes disrupt cognitively close functions if and only if their neuroanatomical territories are close. To capture this relationship, the concept of a cognitive gradient is introduced as a basic unit of macroscopic neocortical brain-behavioral analysis. A cognitive gradient is a continuous distribution of related functions along an axis defined at its extremes by a pair of sensory projection or motor areas. Cortical gradiental structure is viewed as a fundamental symmetric organization on which hemispheric elaborations are superimposed. The gradiental structure of the left hemisphere is presented in detail as an illustration of the concept. It is presumed that a similar set of elaborations can be uncovered for the right hemisphere. The gradiental model implies that at least from the macroscopic standpoint, functional neocortical organization is to a substantial degree continuous, interactive, and emergent, as opposed to mosaic, modular, and prededicated. It is proposed that a shift from latter to former properties signified a major development in the evolution of the brain. Thalamo-cortical integration may represent coexistence of evolutionally early and recent principles of cerebral organization.

I. THE CONCEPT OF A COGNITIVE GRADIENT

Attempts to understand the principles of cerebral localization of functions in man have resulted in two basic approaches. The first approach is aimed at the formulation of fundamental dichotomies (left-right, anterior-posterior) and

emphasizes horizontal aspects of cortical organization (Bogen, 1969; Sperry, 1966). The second approach emphasizes the hierarchic aspect of cerebral organization. The brain is viewed as consisting of "vertically" interrelated structures providing different levels of integration (Bernstein, 1967; Hughlings Jackson, 1931, 1932; Miller, Galanter, & Pribram, 1960).

The purpose of this paper is to introduce another approach to the brain-behavorial relations in the neocortex, the *gradiental approach*. Like the first, dichotomous approach, it emphasizes the horizontal aspect of cerebral organization. It emphasizes, however, its continuous rather than oppositional, discrete aspect. Like the hierarchic approach, the gradiental approach makes substantial use of the concept of the levels of functional integration. It emphasizes, however, relations across the levels rather than the structure of the hierarchy itself, and concentrates on cortico-cortical rather than neuroaxially "vertical" integrations.

It is proposed that the gradiental approach captures an aspect of cortical organization which is as fundamental as the ones reflected in dichotomous and hierarchic approaches. The three approaches are viewed not as mutually exclusive, but as capturing complementary aspects of cerebral organization.

Phenomena of focal brain damage in humans will be reviewed with the attempt to reconstruct the properties of normal organization. Although existing empirical evidence does not permit a complete, detailed substantiation of the gradiental approach at this time, I feel that its introduction is justified at least as a heuristic device.

This paper provides a preliminary introduction of the gradiental approach based solely on clinical neuropsychological evidence. Complete rendition of the subject would require careful neuroanatomical and neurodevelopmental reviews. Such a discussion of the gradiental approach will be the subject of a future publication.

A strong continuity exists between the work of Alexandr Luria and the gradiental model, which I developed while a graduate student of his in the early seventies. The model has been clearly influenced by Luria's (1980) notion of "dynamic functional systems" and his assumption of a three-level functional hierarchy of "primary" sensory projection, "secondary" modality-specific, and "tertiary" associative cortices. The gradiental approach makes use of this assumption.

My work on the gradiental approach was not informed by the current studies in parallel distributed processing (PDP). It grew out of the macroscopic neuropsychological context and with a clinical audience in mind (Goldberg, 1978). Having familiarized myself with the ideas of PDP (McClelland & Rumelhart, 1986; Rumelhart & McClelland, 1986), however, I find a strong affinity between them and the gradiental approach. Although specific parallels between gradiental and PDP concepts run the risk of being superficial (due to the discrepancy between the microscopic level of PDP analysis and the macroscopic level of the gradiental approach), I completely share the hope that

"we will soon see real synergistic interactions between neuropsychology and parallel modeling" (McClelland & Rumelhart, 1986, p.551).

In terms of the scope of phenomena that the gradiental model attempts to explain, it is comparable to the disconnection model of Geschwind (1965) and the modular model of Fodor (1983, 1985), which I consider similar. The gradiental approach, however, offers an account which is opposite to these two models, by emphasizing continuous interactions within the grey matter rather than white matter-mediated communications between distinct, distant structures. As I will argue below, these approaches are not necessarily mutually exclusive, and they may be capturing complementary, coexisting principles of neural organization. The gradiental approach emphasizes those aspects of cortico-cortical interactions which are mediated by short, mostly nonmyelinated fibers, whereas the disconnection model of Geschwind emphasizes interactions mediated by major fasciculi. The exact relative roles of these two types of integration are yet to be determined.

Central to the gradiental approach is the premise of a high degree of consistency between the spatial and functional metrics of the neocortex. By the spatial metric the physical distribution of various cortical regions is meant. By the functional metric I mean a set of "distances" which characterize the relationship between various cortical areas in terms of the cognitive characteristics of the functions mediated by them. It is proposed that an isomorphic or near-isomorphic relationship exists between the two. This means that any two cortical regions are functionally close if and only if they are spatially close.

The notion of a cognitive gradient is only as viable as the premise of cortical spatial-functional isomorphism is correct. A cognitive gradient is an array of functionally distinct cortical regions aligned along the axis defined at its extremes by two areas of primary sensory cortical projections (A,B). It is assumed that the cortical regions along such an axis adhere to the rule of spatial-functional isomorphism: Areas physically closest to sensory projection area A are functionally dominated by that sensory modality. As one moves away from pole A, one encounters areas critical for intermodal integration between modalities A and B. As one proceeds further along the axis toward pole B, one encounters cortical regions dominated by sensory modality B. The schematic, invariant structure of a cognitive gradient is illustrated in Figure 1.

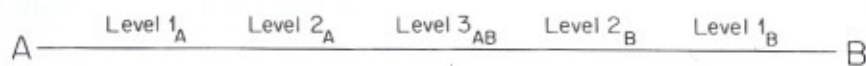A ————— Level 1_A ———— Level 2_A ———— Level 3_{AB} ———— Level 2_B ———— Level 1_B ————— B

Figure 1. Schematic representation of a cognitive gradient.

      A and B - two sensory modalities. Levels 1A and 1B - corresponding cortical primary sensory projection areas. Levels 2A and 2B - corresponding secondary, modality-specific cortical areas. Level 3AB - associative, tertiary cortex.

It is clear that any cognitive gradient thus defined invariably consists of three types of areas as described in hierarchic terms. It by definition includes two primary cortical sensory projection areas at its extremes (level 1). Adjacent to each of them is an area which in hierarchic terms would be labeled a level 2 area, i.e., an area of higher order but still modality-specific integration. Finally, in the center of the gradient a level 3 area is found, which can be characterized as associative, modality-nonspecific. This establishes the relationship between the vertical, hierarchic and the horizontal, gradiental dimensions of cortical localization. The notion of "level" is used here in a strictly cognitive rather than neuroanatomical sense. Although cytoarchitectonic stratification is presumed to roughly parallel the functional stratification of neocortex, the exact relationship between the two taxonomies is not discussed in this paper.

My purpose is not to give an exhaustive acount of neuropsychological phenomena and syndromes, but to introduce a certain principle of cerebral organization. The illustration of this principle is limited in this paper to the left hemisphere. It is assumed that the gradiental principle is equally applicable to the functional organization of the right hemisphere.

II. COGNITIVE GRADIENTS OF POSTERIOR LEFT HEMISPHERE

By the definition of a cognitive gradient, there should be three of them in each of the posterior halves of the two hemispheres: occipito-temporal, occipito-parietal, and temporo-parietal. Symbols O, P, and T will be used to denote the extreme points of the three gradients. The occipito-temporal gradient is defined by the O-T pair; the occipito-parietal gradient by the O-P pair; and the temporo-parietal gradient by the T-P pair. Each gradient has level 1, level 2 and level 3 components. Level 1 components are defined by the three areas of primary sensory cortical projections: the occipital striate Brodmann area 17 (O1), the parietal, postcentral gyrus Brodmann areas 3, 1 and 2 (P1), and the temporal Herschl area, Brodmann areas 41 and 42 (T1), (Carpenter, 1976). These extreme points of the gradients are presumed to be functionally equivalent in the two hemispheres at least at a gross level of analysis (Carpenter, 1976). We will refer to modality-specific level 2 components as O2, P2, and T2; and to associative, level 3 components involved in intermodal integration as OP3, OT3, and PT3. Figure 2 provides a schematic representation of the posterior cortical gradiental structure and the hierarchic composition of each gradient. In this schematic form, the diagram equally applies to either hemisphere.

1. Associative Agnosias
The extreme points of the three left posterior gradients have already been defined, since it is presumed that they are functionally equivalent for the two hemispheres. Secondary, level 2 components of the cognitive gradients of the posterior left hemisphere will now be specified. It is proposed that all level 2

areas of modality-specific higher order perceptual integration in the left hemisphere can be characterized in similar terms. All three are critical for categorical stimulus identification, in other words for recognizing specific exemplars as members of generic categories. When damage occurs to any of these three areas, a particular form of associative, "symbolic" agnosia occurs.

Visual object agnosia (or "associative blindness," or "psychic blindness") was first described by Lissauer (1890) and Freud (1891) and more recently by Albert, Reches, and Silverberg (1975), De Renzi and Spinnler (1966), Hécaen and Albert (1978), Hécaen and Angelergues (1963), Hécaen, Goldblum, Masure, and Ramier (1974), Luria (1980), Rubens and Benson (1971) and Warrington (1975). The syndrome is characterized by the dissociation between an intact visual sensory and perceptual analysis, and an impaired ability to assign the
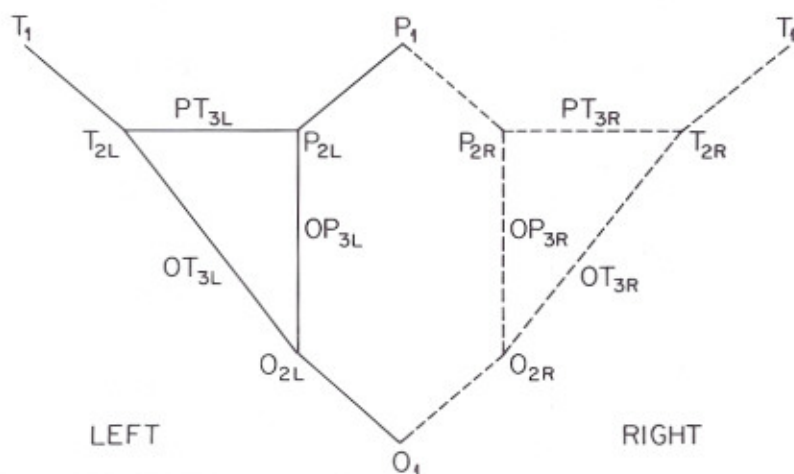

Figure 2. Gradiental structure of posterior cortex.

O1 - primary visual cortex (Brodmann area 17); T1 - primary auditory cortex (Brodmann areas 41 and 42); P1 - primary somatosensory cortex (Brodmann areas 3, 1 and 2). O2 - secondary visual cortex (Brodmann areas 18 and 19); T2 secondary auditory cortex (Brodmann area 22, or posterior portion of the superior temporal gyrus); P2 - secondary somatosensory area (Brodmann areas 5 and 7, or superior parietal lobule). OT3 - associative inferotemporal cortex; PT3 associative cortex (supramarginal gyrus division of the inferior parietal lobule); OP3 - associative cortex (angular gyrus division of the inferior parietal lobule). These abbreviations refer to corresponding areas in the left hemisphere when amended by L, and the right hemisphere when amended by R.

Damage to O2L produces visual object agnosia; OT3L - lexical deficit of Wernicke's type (when more anterior) and anomic aphasia (when more posterior); T2L - acoustic agnosia and semantic associative auditory agnosia; PT3L - semantic aphasia and acalculia of spatial type; P2L - tactile agnosia; OP3L - ideational apraxia and spatial apractagnosia.

visual stimulus to a generic category, to discern its "meaning." Upon visual examination of an object, the patient can accurately describe its sensory and perceptual attributes and even copy it but he is unable to identify it by name, through a functional description, or a pantomime. The deficit is not a linguistic one, since not only the ability to name the object but also the ability to convey its meaning through nonverbal means suffers, and the deficit is limited to visual inputs. When the patient is allowed to touch the object, smell or hear its characteristic odor or sound, the identification becomes possible. It is not, however, an elementary sensory or perceptual deficit, since the perceptual pattern analysis is intact or near-intact.

The neuroanatomy of visual object agnosia has been invariably associated with the periphery of the occipital lobe, close to its border with the temporal and parietal lobes (Albert et al. 1975; Benson, Segarra, & Albert, 1974; Hécaen et al., 1974; Rubens & Benson, 1971; Warrington, 1975). Although some authors emphasize the bilateral nature of the lesion (Albert et al., 1975; Hoff & Poetzl, 1935), most regard the left occipital area the site of the critical lesion (Benson et al., 1974; Hécaen et al., 1974; Lhermitte, Chedru, & Chain, 1973; Nielsen, 1937; Rubens & Benson, 1971; Warrington, 1975).

On the basis of the aforedescribed clinical material, I conclude that the normal function of the left occipital periphery (Brodmann areas 19 and in part 18) on its junction with the temporal and parietal lobes, involves categorical identification of visual percepts which is a form of higher order visual perceptual integration. It corresponds to area O2 in the diagram of the left posterior gradiental structure (Figure 2).

Tactile agnosia (or "pure astereognosia," or "tactile asymbolia") is in a sense, the tactile equivalent of visual object agnosia (Bauer & Rubens, 1985; Hécaen & Albert, 1978; Lhermitte & de Ajuriaguerra, 1938; Luria, 1980; Wernicke, 1894). The ability to identify the object by touch as a member of a meaningful generic category suffers in either hand, although it can be accomplished in other sensory modalities. The ability to describe separate tactile properties of the stimulus is spared.

The anatomy of this form of astereognosia implicates secondary parietal areas of the left hemisphere which correspond to Brodmann areas 5 and 7, the superior parietal lobule (Lhermitte & Ajuriaguerra, 1938; Luria, 1980). On the basis of aforedescribed clinical material, I conclude that the normal function of this area involves categorical identification of tactile and proprioceptive percepts, which is a form of higher order somatosensory perceptual integration. This is area P2 in the schematic representation of the left posterior gradiental structure (Figure 2).

Acoustic agnosia is the term used by Luria (1980) to denote a clinical syndrome similar but not identical to Wernicke's aphasia (Goodglass, 1980, Goodglass & Geschwind, 1976). Acoustic agnosia is a deficit of phonemic hearing in the

absence of elementary hearing loss or primary articulation deficit. It is a categorical disorder, in which the perception of the "physical identity" of the sound may be intact but the ability to classify it as an allophone of a particular phonemic class is impaired (Luria, 1970, 1980). This description is not substantially different from that of Wernicke's aphasia. In explaining the mechanism of paraphasias of Wernicke's type, Goodglass (1980) also proposes the breakdown of phonological retrieval.

The neuroanatomical descriptions of the critical lesions responsible for "acoustic agnosia" and "Wernicke's aphasia" are virtually identical: both implicate the posterior portion of the superior temporal gyrus in the left hemisphere.

A related syndrome of "semantic associative" auditory agnosia has also been described (Faglioni, Spinnler, & Vignolo, 1969; Spinnler & Vignolo, 1966; Vignolo, 1982). It involves an inability to understand the "meaning" of nonverbal sounds and noises, i.e., associate them with correct sources. Purely auditory perceptual aspects of analysis are, however, intact (e.g., the patient's capacity for "same-different" judgement with respect of nonverbal sounds), nor is amusia present. Semantic associative auditory agnosia usually co-occurs with Wernicke's aphasia and the neuroanatomical territories of both conditions are invariably associated with left temporal damage.

It is not clear whether acoustic agnosia/Wernicke's aphasia and semantic associative auditory agnosia represent two entities with different mechanisms but adjacent neuroanatomical territories, or two aspects of the same syndrome with the same underlying mechanism, although the latter appears more likely.

On the basis of aforedescribed clinical material, I conclude that the normal function of the secondary temporal area (posterior portion of the superior temporal gyrus, or Brodmann area 22) of the left hemisphere involves categorical recognition of auditory stimulus patterns, which is a higher-order form of auditory perception. This function is critical both in the linguistic context for phonemic hearing, and in nonlinguistic contexts for the categorization of environmental sounds. The corresponding area is T2 in the schematic representation of the left posterior gradiental structure (Figure 2).

All three secondary modality-specific, level 2 areas of the posterior portion of the left hemisphere share the same fundamenal property. They provide the capacity to identify unique stimulus patterns as members of generic categories. Each of the three areas ensures this in a particular, single sensory modality: visual, somatosensory, or auditory. It is proposed that the categorical processing is the basic function of the posterior portion of the left hemisphere. This point of view is distinctly different from the one regarding language as the fundamental attribute of the left hemisphere. The concept of categorical processing as used here is broader and can be viewed as more fundamental both on developmental and evolutional grounds, than the concept of language. In fact, it can be viewed as the evolutional and cultural precursor of language.

We are now ready to review the associative, level 3 areas and by this complete

the cognitive gradients of the posterior left hemisphere. The neuroanatomical territories of the middle portions of the postulated occipito-parietal and temporo-parietal gradients are adjacent, and together they occupy the territory of the inferior parietal lobule, a major part of the tertiary, associative cortex. It consists of two subdivisions, the supramarginal and the angular gyrus. On strictly geometric grounds the angular gyrus can be assigned to the occipito-parietal gradient and the supramarginal gyrus to the temporo-parietal gradient. Theoretically then, the angular gyrus can be viewed as the associative OP area, and the supramarginal gyrus as the associative TP area.

In reality, however, neuroanatomical descriptions of various neuropsychological syndromes do not clearly distinguish between the two subdivisions of the inferior parietal lobule, possibly due to the close proximity of the two gyri and their relatively small size. Most cortical lesions are too large to be contained within either one of these two areas.

2. Syndromes of the Left Occipito-Parietal Junction

Ideational apraxia. This notion refers to the disintegration of skilled, over-learned, object-oriented movements. Although individual motor components are intact, their integration into coherent motor programs fails (Hécaen & Albert, 1978; Liepmann, 1900, 1908). Liepmann's definition of "ideational apraxia" requires that it occur without associated paresis or ataxia. The deficit is bilateral and general rather than segmental with respect to body parts, but it is produced by a unilateral, circumscribed lesion limited to the posterior parietal and temporo-parietal regions of the left hemisphere (de Ajuriaguerra, Hécaen, & Angelergues, 1960; Hécaen & Albert, 1978; De Renzi, Pieczulo, & Vignolo, 1968); more specifically, the left angular gyrus (Foix, 1916, 1922; Heilman & Rothi, 1985).

Spatial apractagnosia. Luria (1980) uses this term to denote a group of (presumably representing a unitary syndrome) deficits of processing asymmetric, meaningful spatial arrays, such as maps, clocks, letters and digits. The common denominator among the stimuli whose processing is affected in this condition includes their symbolic, representational nature; visual input modality; and asymmetric space being critical in representing the meaning of the symbols. "Spatial apractagnosia" arises following lesions in the occipito-parietal junction, in the area of the angular gyrus.

Other authors consider its different aspects separately. Hécaen and Albert (1978) and Benton (1985) regard the loss of topographical concepts a separate clinical entity, distinct from the disturbance of processing letters and digits, and associated with a bilateral damage to posterior parietal areas.

The other component of "apractagnosia" is known as "alexia with agraphia", or "parietal alexia" (Friedman & Albert, 1985; Hoff, Gloning, & Gloning, 1954; Hermann & Poetzl, 1926). It occurs in the absence of obvious aphasia. "Alexia with agraphia" and the related deficit, "alexia/agraphia for

numbers" are commonly associated with lesions of the left angular gyrus (Benson & Geschwind, 1969; Hécaen, 1967; Henschen, 1925; Levin & Spiers, 1985; Nielsen & Raney, 1938).

The two types of deficits - ideational apraxia and "apractagnosias" (including alexia and agraphia) - usually co-occur (Friedman & Albert, 1985; Luria, 1980). They constitute two clinical forms of the same fundamental deficit: disintegration of the visuo-spatial synthesis with respect to categorical representations. "Apractagnosia" is by definition characterized by the deficit of processing of symbolic, generic representations. Ideational apraxia should be regarded as categorical since the affected "motor engrams" describe motor programs invariant across a variety of specific conditions, applicable to any object of a given class and executable by any limb. An engram of a skilled movement must contain both spatial and sequential information. Its cortical substrate is likely to be distributed between parietal (spatial) and premotor (sequential) regions. Ideational apraxia represents the breakdown of the spatial aspect of the engrams of skilled movements. Its anterior, sequential counterpart will be described later under the name of limb-kinetic apraxia.

On the basis of the aforedescribed clinical material, I conclude that the left parieto-occipital junction (angular gyrus) is critical for intermodal, associative integration between higher order visual and somatosensory/spatial information which is symbolic, representational in nature. This is OP3, the level 3 component of the occipito-parietal gradient. The cognitive properties of this area are in agreement with its neuroanatomical geometry. Both cognitively and anatomically, it is situated between the visual modality-specific and the somatosensory/spatial modality-specific areas. The rule of cognitive-neuroanatomical isomorphism appears to hold along the left occipito-parietal cognitive gradient. It can be otherwise referred to as the visuo-spatial gradient of symbolic representations.

3. Syndromes of the Left Temporo-Parietal Junction

Semantic aphasia. Head (1926) and Luria (1970, 1980) maintain that lesions of the left temporo-parietal areas lead to a deficit of comprehending and expressing asymmetric propositional constructions. These include spatial ("to the right of" vs. "to the left of"), temporal ("before" vs. "after") and other comparative relations ("smaller" vs. "larger", "taller" vs. "shorter"), passive voice and possessive case ("father's brother" vs. "brother's father"). The patient cannot interpret reversible constructions devoid of lexical redundancy. By contrast, phonological and lexical aspects of language are preserved. Luria terms this syndrome "semantic aphasia." It closely resembles the "morphological agrammatism" described by Tissot, Mounin, Lhermitte, & Dordain (1973).

Acalculia. Various types of acalculia have been described. "Acalculia of the spatial type" (Levin & Spiers, 1985; Luria, 1980) is of a particular relevance here. It is characterized by the disintegration of the decimal structure of

numbers and of the appreciation of the spatial asymmetries implicit in this structure. The patients cannot properly align compound multi-digit numbers in written computations or even understand their meaning. It is different from alexia/agraphia for numbers in that the ability to read or write separate digits is preserved. Spatial acalculia is usually associated with the inferoparietal, post-Rolandic lesions (Levin & Spiers, 1985; Luria, 1980).

Luria (1980) proposed that the cognitive mechanism of "semantic aphasia" is related to the disintegration of the spatial basis of relational constructions. This implies that normal cognitive representations of a wide range of relational concepts are spatial or "quasispatial" in nature. This is true, according to Luria, not only for spatial relations proper, but also for temporal, and any quantity relations, as well as for those expressed by passive voice and possessive case.

Likewise, Luria (1980) regards acalculia of the spatial type to be secondary to the disintegration of the spatial basis of numerical concepts. This implies that normal cognitive representations of numbers and numeric operations have a very strong spatial component, a conclusion based on cultural-anthropological and developmental considerations.

This reasoning implies that the syndromes of "semantic aphasia" and "acalculia of the spatial type" represent two manifestations of the same fundamental cognitive deficit: disintegration of the spatial aspect of cognitive representations underlying linguistic and other symbolic (e.g. numeric) codes. Indeed, "semantic aphasia" and acalculia are highly correlated. It is proposed that the cognitive dimension whose disintegration leads to "semantic aphasia" and "spatial acalculia," is that of the interface and intrinsically intermodal integration between auditory-based linguistic (and quasilinguistic) codes and somatosensory-based spatial schemata.

In terms of its cognitive status, the proposed cognitive dimension of "linguo-spatial interface", or "spatial basis for linguistic representations" adheres closely to the properties expected of the middle, level 3 segment of the left temporo-parietal gradient, or area PT in our notation (Figure 2). Disintegration of this cognitive dimension is indeed associated with damage to the left inferoparietal area.

4. Neurolinguistic Digression: Cortical Representation of Lexicon
Before proceeding with the discussion of cortical cognitive gradients, we must discuss cerebral representation of lexical knowledge in general terms. Logically, two extreme possibilities exist: (a) that cerebral representation of lexical knowledge is modular, and separated from the representations of those aspects of physical world which it denotes; (b) that it is distributed and is in close neuroanatomical relationship with cerebral representations of the various aspects of physical world denoted by it.

The assumption of "separateness" of lexical vs. perceptual cerebral represen-tations was implicit in the "disconnection" view of anomia (Geschwind, 1965; 1967; Geschwind & Kaplan, 1962).The alternative idea of the distributed nature

of cerebral representation of lexical knowledge is supported by the findings that semantic knowledge, or the "mental dictionary" itself is impaired in anomias. The "semantic deficit" hypothesis implies that in anomic patients, the hierarchic organization of the *physical world* representations is itself disrupted. Three lines of evidence support this conclusion: that in anomic patients categorization processes are impaired not only with respect to names but also pictures of objects (Grober, Perecman, Kellar, & Brown, 1980; Zurif, Caramazza, Myerson, & Galvin, 1974); that in tasks requiring classification of pictorial representations into functionally defined categories, anomic patients fail to use "semantically guided, perceptual parsing" (Whitehouse, Caramazza, & Zurif, 1978; Caramazza, Berndt, & Brownell, 1982); and that perceptual degradation of pictorial images exacerbates anomia (Benton, Smith, & Lang, 1972; Bisiach, 1966; North, 1971).

This implies that lexical-semantic and perceptual-categorical "dictionaries" are functionally intertwined and involve similar encoding units (Miller & Johnson-Laird, 1976). It is possible that the cerebral representations of the two "dictionaries" are intertwined also neuroanatomically, which implies the distributed nature of cerebral representation of lexicon. Lexical-semantic representations are presumed then to be localized in close proximity to, and possibly even strongly overlap with the areas in which representations of corresponding aspects of the physical world are localized.

Although most representations of things and events are multimodal, some are more dependent on certain modalities than on others. Thus representations of static objects are likely to be encoded with a particular reliance on the visual modality (Beauvois, 1982; Goodglass, Barton, & Kaplan, 1968), and representations of actions are more based on motor images than representations of colors. This means, among other things, that cerebral representations of concrete nouns (the lexical domain which denotes objects) are expected to be in the intimate proximity to the cortical areas involved in visual processing, the occipital cortex, and cerebral representations of concrete verbs in the intimate proximity to the cortical areas involved in the control over motor sequences, the premotor cortex. Likewise, more abstract concepts denoting actions may be represented in close proximity to prefrontal convexity.

Lexical deficit is usually reported in posterior (Wernicke's and anomic) aphasias (Damasio, McKee, & Damasio, 1979; Coughlan & Warrington, 1978; Grober et al., 1980; Whitehouse et al., 1978; Yamadori & Albert, 1973; Zurif et al., 1974). However, these studies utilized only nouns, but not verbs, and cannot therefore assess the possibility of verb-noun, anterior-posterior double dissociation. McCarthy and Warrington (1985) reported a case of extremely impaired comprehension and retrieval of verbs and action names but intact competence for nouns. The case is neuroanatomically uninformative since diffuse cortical atrophy was present. It is important, nevertheless, since it provides clear evidence of a reverse dissociation, lexical deficit for verbs but not nouns.

Only few studies explicitly compared competence for nouns and verbs.

Goodglass, Klein, Carey & Jones (1966) failed to find any difference in verb vs. noun proficiency between anterior and posterior aphasics. Micelli, Mazzuchi, Mann, and Goodglass (1983), however, described a massive omission of verbs but not nouns following a focal lesion in the Broca's area. Furthermore, Micelli, Silner, Villa, and Caramazza (1984), demonstrated a *double dissociation* between verbs vs. nouns, and agrammatic vs. anomic aphasics. In the former, naming actions was more impaired than naming objects. The latter demonstrated the opposite picture. Micelli et al. (1984) propose that at least in part agrammatism in anterior aphasias is due to a selective lexical deficit for verbs. Luria (1980) claims that posterior, temporal-lobe aphasias are characterized by a greater impairment of nouns and to some extent adjectives than of verbs or prepositions; that posterior, parieto-occipital aphasias are predominantly characterized by impairment of prepositions and adverbial clauses; and anterior aphasias (i.e., Broca's and Kleist's) by a particular disintegration of verbs. Brown, Lehman, and Marsh (1980) report noun-verb, anterior-posterior double dissociation in normal subjects. They studied the topography of CNV-like preparatory, slow negative wave during a listening task involving verb- and noun-phrases.It was found that the preparatory wave shifted anteriorly in verb- and posteriorly in noun-phrases.

Warrington (1975) reported a case of anomia due to a posterior lesion characterized by a greater impairment of concrete (i.e., sensory-based) than abstract (i.e., devoid of a distinct sensory image) nouns. Goodglass, Hyde, and Blumstein (1969) reported that highly picturable nouns are particularly impaired in fluent (Wernicke's and anomic) aphasias. Yamadori and Albert (1973) described a patient with a left posterior lesion and anomia for body parts and room objects but not tools (whose representations are likely to have a stronger motor component). His circumlocutions were dominated by action-words, e.g. "to help people walk" for "cane", "to sit on" for "chair."

In anterior, nonfluent aphasias, on the other hand, nominalization of verbs has been noted (Goodglass & Geschwind, 1976; Luria, 1980, Saffran, Schwartz, & Marin, 1980). This phenomenon is complementary to the heavy reliance on verbs in fluent aphasics' circumlocutions. Conspicuous paucity of action words and predicative forms was also found in Kleist's dynamic aphasia due to left frontal damage (Luria, 1970; Luria & Tsvetkova, 1968).

It is proposed that cerebral representation of lexical, semantic knowledge is not compact, but is distributed throughout the neocortex. The distribution of various lexical domains parallels the distribution of cortical representations of corresponding aspects of the physical world denoted by these various lexical domains.

5. Syndromes of the Left Temporo-Occipital Junction

Fluent aphasias. The middle segment of the temporo-occipital cognitive gradient, its level 3, OT portion occupies the associative cortex of the temporal lobe in close proximity to the temporo-occipital junction. Damage to this area

leads to "fluent" aphasias: Wernicke's anomia and in particular anomic aphasia. According to the rule of functional-spatial neocortical isomorphism, the function of this posterior temporal area can be expected to be intermodal and related to the integration of visual and auditory modalities. In the left hemisphere, it is expected to be symbolic, representational in nature.

It has been proposed earlier that lexical deficit in fluent aphasias is specific to the lexical domains which denote those aspects of physical world represented with maximum reliance on the visual modality, and that such selectivity of lexical deficit in posterior aphasias is a reflection of the distributed nature of the cerebral representation of lexicon. I now propose that the normal function of the middle portion of the left occipito-temporal gradient (posterior portion of the temporal lobe at the temporo-occipital junction) is related to those aspects of linguistic representations which denote visually based aspects of physical world. The role of this area consists of auditory-visual integration within the linguistic context.

This completes the review of the left temporo-occipital gradient. As previously described gradients, it abides by the rule of functional-structural isomorphism. Fluent aphasias represent the breakdown of its level 3, associative segment.

6. Summary of Posterior Cognitive Gradients

A distinct triangular cognitive gradiental structure of the posterior portion of the left hemisphere emerges with a continuous distribution of functions along each of its sides: occipito-parietal, occipito-temporal, and temporo-parietal. This description reflects the fundamental property of intermodal interaction along the gradients, the occipito-parietal gradient being the visuo-spatial one, the temporo-parietal the auditory-spatial one, and the occipito-temporal the visual-auditory gradient. This basic gradiental structure is presumed to be symmetric in the two hemispheres. The functional descriptions of the occipito-temporal and occipito-parietal gradients as they pertain to visual pattern recognition are consistent, respectively, with the object ("what", ventral) and spatial ("where", dorsal) visual systems in primates (Mishkin, Ungerleider, & Macko, 1983).

Superimposed upon this basic symmetric gradiental structure are hemispheric elaborations. The left posterior hemisphere is viewed as central to categorical processing, i.e., relies on generic classifications and rules of processing specific items in terms of pre-existing generic categories. When this elaboration is superimposed on the posterior gradients of the left hemisphere, the occipito-temporal, visual-auditory gradient becomes linguo-visual; the temporo-parietal, auditory-spatial gradient becomes linguo-spatial; and the occipito-parietal, visuo-spatial gradient becomes involved in processing representational, symbolic stimuli.

III. ANTERIOR COGNITIVE GRADIENTS

1. Motor-Prefrontal Gradient

Anterior cortex is different from posterior cortex in basic neuroanatomy since it consists of only one lobe. The frontal lobe can also be viewed in terms of a level 1 to level 2 to level 3 progression. I will refer to such progression as a partial gradient. Such organization characterizes every cortical lobe, and a gradient is constructed of a pair of lobe-specific partial gradients which are directed toward each other. The concept of a partial gradient as applied to the frontal cortex is not fundamentally different from that of a cognitive gradient as applied to the posterior cortex, since both are expressions of the principle of cognitive-neuro-anatomical isomorphism.

In the anterior partial gradient, level 1 is represented by motor cortex (Brodmann area 4); level 2 by premotor cortex (Brodmann area 6 and, in part, 8); and level 3 by dorsolateral prefrontal cortex (Brodmann areas 9, 10, and in part, 8). If a hierarchic analogy is to be drawn between posterior and anterior cortical gradiental organizations, motor cortex is comparable to the primary, sensory modality-specific areas; premotor cortex to secondary, modality-specific areas; and prefrontal cortex to tertiary, associative areas of the posterior brain.

The motor-prefrontal partial gradient will be introduced as a basic, hemisphere-invariant organizational property of the frontal lobe.

Motor cortex. Level 1 of the motor-prefrontal gradient consists of the motor cortex (Brodmann area 4) whose organization roughly parallels the organization of the postcentral, somatosensory cortex. It occupies the anterior wall of the central sulcus and adjacent portions of the precentral gyrus (Carpenter, 1976). Both in its somatotopic organization and its function, that of the final motor cortical output station, it is comparable with the primary cortical projection areas of posterior gradients.

Premotor cortex. Level 2 element of the motor-prefrontal gradient consists of premotor cortex (Brodmann area 6 and according to some authors parts of area 8). Unlike motor cortex, lesions of premotor cortex do not lead to permanent paralysis or paresis. Instead, the disintegration of sequential, kinetic organization of complex skilled movements or *ad hoc* motor sequences takes place (Foerster, 1936; Luria, 1980). It may take two forms which are usually combined.

Transition from one motor element to another within the kinetic sequence is impaired, often producing motor perseveration with excessive repetition of the same discrete motor act or maintenance of a posture (Goldberg, 1986; Goldberg & Tucker, 1979; Goldberg & Costa, 1986; Goldberg & Bilder, 1986; Luria, 1965, 1980). Denny-Brown (1958) considered the latter type of perseveration central to frontal-lobe pathology. The resulting disorders are known as "melokinetic"

and "limb-kinetic" apraxias (Fulton, 1937; Hécaen & Albert, 1978; Liepmann, 1920; Heilman & Rothi, 1979; Nielsen, 1946).

On the basis of these clinical observations, various authors concluded that the function of premotor cortex consists of sequential organization of discrete movements into organized, smooth sequential patterns.

Prefrontal (dorsolateral) cortex. The prefrontal cortex has been associated with the highest levels of neurocognitive control. Major differences exist between different aspects of prefrontal cortex and corresponding syndromes (Damasio, 1985; Goldberg, 1985; Luria, 1980; Pribram, 1987). We will concentrate on the dorsomedial prefrontal cortex which lies on the continuation of the motor-prefrontal gradient and constitutes its level 3 component.

Damage to the dorsolateral prefrontal cortex leads to a range of clinical manifestations, among which the following are most prominent: *perseveration* (Goldberg, 1986; Goldberg & Bilder, 1987; Goldberg & Tucker, 1979; Luria, 1980); *aspontaneity*, which may take two forms, inertia of initiation and inertia of termination (Bilder & Goldberg, 1987; Goldberg, 1986; Luria, 1980), *field-dependent behavior* (Goldberg & Costa, 1986; Lhermitte, 1986; Lhermitte, Pillon, & Serdaru, 1986; Luria, 1980); and *imitative ("echo") behaviors* (Luria, 1980). Many of these phenomena follow other focal lesions, but then they are limited to a particular sensory modality, type, or level of processing. Following dorsolateral prefrontal damage, these deficits are supramodal, universal in their impact, and pervasive both horizontally, by affecting virtually every behavioral domain, and vertically, by disrupting virtually every level of neurocognitive control (Goldberg & Bilder, 1987).

Geometrically, dorsolateral prefrontal cortex is an extension of premotor cortex. It is of heuristic value to conceptualize also its function as an extension of and elaboration on that of premotor cortex. Just as premotor cortex is central for sequential motor organization, so is the function of prefrontal cortex central for the sequential organization of behavior at large. The continuity between motor and cognitive controls has been noted by Vygotsky (1934), Bernstein (1947, 1967), and Bruner (1966).

Current attempts to capture the nature of prefrontal function use descriptions like "programming of behavior," "formulation of plans," "sequential organization of behavior," "maintenance of behavior selectivity." It is not clear whether these descriptions allude to a unitary "executive function" or a group of related cognitive controls. In either case, this function or class of functions are closely related to motor controls. Just as a complex motor act requires a precise selection and smooth alignment of motor elements into a co-ordinated sequence, so does a strictly cognitive act require the selection and organization into a co-ordinated pattern of pre-existing cognitive routines, skills, and representations. Dorsolateral prefrontal cortex is critically involved in the *ad hoc* selection, co-ordination and sequential organization of cognitive elements into patterns dictated by the ongoing behavioral context.

The motor-prefrontal gradient can be described in terms of the following continuum: motor control (level 1 motor cortex) - sequential organization of complex motor patterns (level 2 premotor cortex) - hierarchic/sequential organization of any type of behavior (level 3 dorsolateral prefrontal cortex). A clear agreement exists between neuroanatomical and functional continuities also in the anterior brain. The motor-prefrontal gradient is not hemisphere-specific. It reflects the fundamental principle of organization of the anterior cortex as a whole.

2. Left Inferior Motor-Prefrontal Gradient

The hemispheric elaboration of the motor-prefrontal gradient will now be discussed with the focus on the left hemisphere. This continuum is neuroana-tomically defined at its two extremes by the inferior portion of the motor cortex (Brodmann area 4) and the dorsolateral prefrontal area. The inferior portion of the motor cortex is, together with its premotor counterpart, the area of motor control of the oral apparatus. The functions of the dorsolateral prefrontal cortex have been described earlier.

In defining the middle elements of this continuum, we enter the territory whose damage produces Broca's aphasia. Neuroanatomical definitions of Broca's area encompass a premotor (the inferior portion of Brodmann area 6) and an associative component (frontal operculum, Brodmann area 44) (Benson & Geschwind, 1969; Goodglass & Geschwind, 1976; Luria, 1980).

Cognitive descriptions of Broca's aphasia also encompass two distinct components: disintegration of kinetic, sequential organization of articulatory output; and agrammatism. They are usually regarded as two components of the same syndrome with the same substrate. It is possible, however, that a neuroanatomically finer level of analysis would reveal a double dissociation between the two cognitive aspects of Broca's aphasia and the two neuroanatom-ical components of Broca's area. If so, then the co-occurrence of the two aspects of Broca's aphasia is due to the physical adjacency of two distinct cortical territories rather than a unitary, shared cognitive mechanism. Benson (1985), and Hécaen and Albert (1978) allude to the existence of "variants" of Broca's aphasia. The view of Broca's aphasia as "speech apraxia", a disorder of the kinetic, sequential aspects of oral praxis is shared by Luria (1970; 1980); Denny-Brown (1963); Johns and Darley (1970); and Trost and Canter (1974). I propose that this deficit is caused by the damage to the inferior portion of the premotor area (Brodmann area 6) in the left hemisphere. The normal function of this area can be inferred as that of sequential organization of articulatory patterns, a special case of the sequential motor organization mediated by premotor cortex.

Tissot et al. (1973) have proposed that at least two forms of agrammatism exist. One of them, "syntactic agrammatism", is characterised by impaired use of verbs. McCarthy and Warrington (1985) argued that an essentially lexical deficit, i.e., impaired semantic representation of verbs and action names, is at the core of this syndrome. Agrammatism following lesions in the Broca areas

has been emphasized by Goodglass and Geschwind (1976); Hécaen and Albert (1978); and Luria (1970; 1980). I propose that "syntactic agrammatism" is at the core of Broca's agrammatism, and it is caused by damage to the frontal operculum (Brodmann area 44), an associative cortex found on the junction between premotor and prefrontal cortices. The normal function of this area can be understood in light of the previously proposed distributed cerebral linguistic representation. Specifically, it includes the represenation of that semantic domain which denotes actions, that is the predicative aspects of language.

As we proceed along the continuum anteriorly, farther away from the motor and toward the dorsolateral prefrontal extreme, we encounter the territory whose damage produces "frontal dynamic aphasia" (Kleist, 1934; Luria, 1965; 1980). It is located immediately anterior of the frontal operculum, probably involving the area of *pars triangularis*. This syndrome is characterized by the deficit of linguistic behavior rather than of language *per se*. The ability to initiate communications is impaired. Repetition is more intact than spontaneous speech. To the extent that a speech sample can be obtained from the patient, it is likely to be replete with perseveration, thematic rather than linguistic. In a dialogic situation echolalia is present. Phonology, articulation, lexicon, and grammar are spared.

Dynamic aphasia can be best described as an executive deficit without the pervasiveness of the dorsolateral prefrontal syndrome. Kleist (1934) and Luria (1980) referred to this syndrome as the "defect of speech initiative". It selectively affects linguistic behavior and is similar but not identical to "transcortical motor aphasia" and "frontal mutism" (Benson, 1985; Damasio, 1985). It can be concluded that the posterior portion of the prefrontal cortex in the left hemisphere serves the function of executive-linguistic interface. It provides executive control over linguistic behavior.

This completes the review of the left inferior motor-prefrontal partial cognitive gradient. It is viewed as the left hemispheric elaboration on the basic motor-prefrontal partial gradient of the anterior cortex. This elaboration takes the form of the continuum of transitions from motor representation of oral apparatus (level 1 inferior motor cortex), to the control over sequential organization of articulation (level 2 inferior premotor cortex whose damage produces "speech apraxia"), to the representation of predicative aspect of language (level 2-3 frontal operculum whose damage produces "syntactic agrammatism"), to the executive control over linguistic behavior (level 2-3 area of posterior prefrontal cortex whose damage produces frontal dynamic aphasia), to executive control at large (level 3 dorsolateral prefrontal cortex).

3. Summary of Anterior Cortical Cognitive Gradients
We will designate level 1 motor cortex as M1; level 2 premotor cortex as PM2; and level 3 dorsolateral prefrontal cortex as PF3. The M1 - PM2 - PF3 organization is viewed as a fundamental, symmetric description of the frontal lobe. Hemispheric elaborations are introduced upon it in a secondary fashion.

Schematic representation of the gradiental structure of anterior cortex is provided in Figure 3.
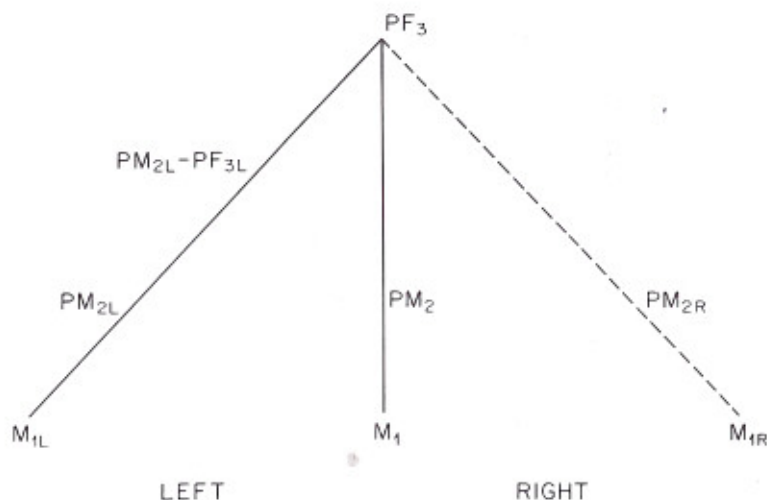

Figure 3. Gradiental structure of anterior cortex. M1 - "primary" motor cortex (Brodmann area 4); PM2 - "secondary" premotor cortex (Brodmann area 6 and in part 8); PM2L - the left inferior portion of premotor cortex; PF3 - dorsolateral prefrontal cortex (Brodmann areas 9, 10 and in part 8); PM2L-PF3L - the left frontal operculum and pars triangularis (Brodmann areas 44, 45). These abbreviations refer to corresponding areas in the left hemisphere when amended by L, and the right hemisphere when amended by R.
Damage to PM2 leads to melokinetic and limb-kinetic apraxias; PF3-executive syndrome; PM2L - speech apraxia; PM2L-PF3L - syntactic agram-matism (when more posterior) and dynamic aphasia (when more anterior).

Like in the case of posterior cortex, a structure of cognitive continua characterizes the left anterior cortex. These are: motor-prefrontal (motor-executive) partial gradient, and inferior motor-prefrontal (linguo-executive) partial gradient. It is presumed that a complementary pattern of elaborations can be uncovered for the right anterior brain.

4. Summary of Cortical Cognitive Gradients
This completes our review of cortical cognitive gradients. These gradients form a diamond-like structure, with a triangular arrangement in every cortical quadrant (Figure 4).
    The general gradiental structure as described above consists of two types of
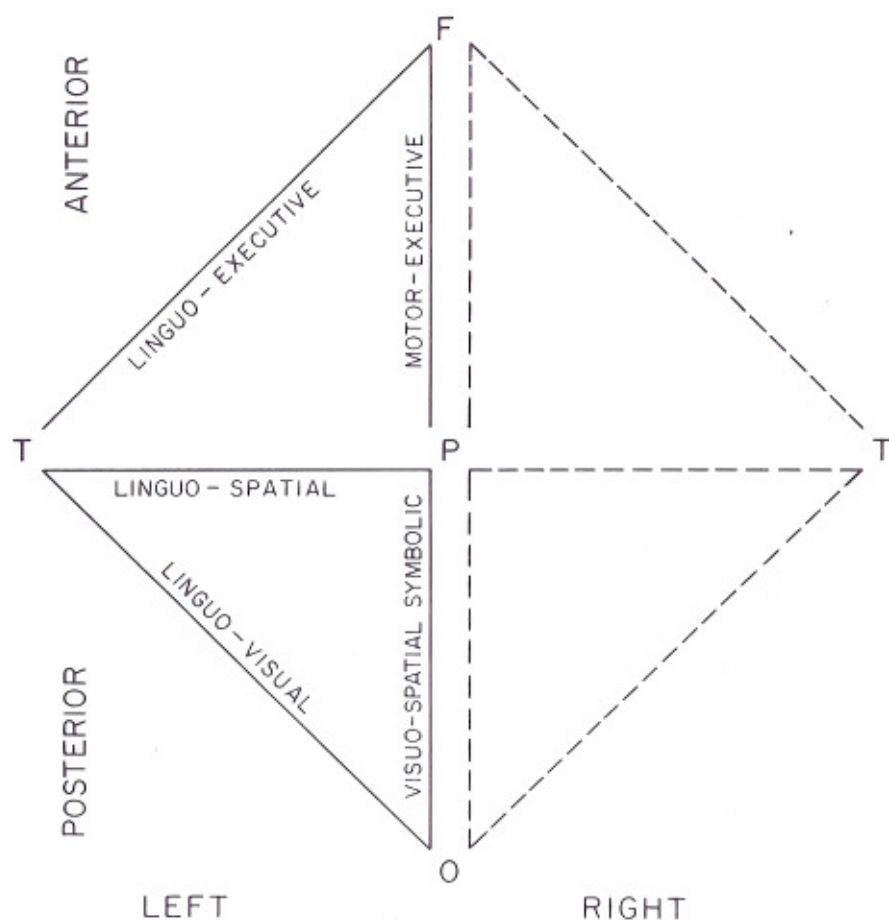
Figure 4. Composite schematic representation of cortical gradiental structure.
O, T, P, and F denote occipital, temporal, parietal, and frontal poles, respectively. Visuo-spatial symbolic, linguo-visual, linguo-spatial, and linguo-executive gradients are left hemispheric elaborations on occipito-parie-tal, occipito-temporal, temporo-parietal, and inferior motor-prefrontal gra-dients, respectively. The motor-executive (motor-prefrontal) gradient is pre-sumed to be symmetrical.

elements: cognitive gradients which characterize the posterior cortex and partial cognitive gradients which characterize the anterior cortex. They both are expressions of the same fundamental principle of brain-behavioral mapping: the principle of cognitive-neuroanatomical isomorphism. A cognitive gradient expresses this principle with respect to the interface between cortical lobes. A partial cognitive gradient expresses this principle within a single cortical lobe. In combination, cognitive gradients define the pattern of the horizontal

neocortical functional organization. This horizontal principle together with the vertical (hierarchic) principle define two aspects of basic (symmetric) cortical organization. A cognitive gradient is the fundamental unit of the horizontal organization just as a hierarchic level is the fundamental unit of the vertical organization. Hemispheric specialization is viewed as a secondary elaboration on the (fundamentally symmetric) horizontal and vertical principles of organization.

IV. THEORETICAL IMPLICATIONS OF THE GRADIENTAL MODEL

1. Mosaic vs. Continuous Principles of Cortical Organization

In the course of the foregoing review the degree of specificity has been introduced which may be perceived as a throwback toward the Gall-style mosaic localization. In fact, exactly the opposite conclusions will be drawn. Two opposing, extreme views on the nature of brain-behavioral relationships are logically possible: that brain-behavioral mapping is essentially mosaic; or that it is essentially continuous.

The mosaic principle permits a random relationship between the neuroanatomical and functional cortical metrics, so that two cortical regions can be neuroanatomically adjacent but functionally distant and *vice versa*. The continuous principle presupposes an orderly relationship between neuroanatomical and functional cortical metrics. In it strongest form, it presupposes neuroanatomical-functional isomorphism. Therefore the existence of cognitive cortical gradients strongly supports the continuous principle of functional cortical organization.

2. Modular vs. Interactive Principles of Cortical Organization

The modular principle implies a system of encapsulated units with interactions limited to input-output relations. Once an input has been received by a module, the processing in it is not affected by external influences until after the output state. The modular principle emphasizes the spatial discreteness of functional units and the temporal discreteness of "bursts" of informational exchange between units (Fodor, 1983, 1985). It can be argued that disconnection models of behavioral neurology (Geschwind, 1965) are also implicitly based on the premise of modularity.

The interactive principle implies a system in which every unit has multiple connections with other units. Informational exchange between units is not limited to input-output relations but occurs continuously. The interactive principle emphasizes spatial continuity between functional units and temporally continuous, uninterrupted information flow between units. Here it may be more appropriate to talk about somewhat arbitrarily specified "locations" in the neural network than "units" in an absolute sense; and to describe information processing in continuous terms of flow than in discrete terms of

stages. The opposite is true for the modular system.

Although the two dichotomies, mosaic vs. continuous, and modular vs. interactive, are related, all four combinations are logically possible. Intuitively, however, some of them are more plausible than others. A mosaic-interactive design of cortical organization is rather impractical, since it implies intensive interactions between neuroanatomically distant regions. A modular-continuous design is redundant, since it presupposes an order (cognitive-neuroanatomical isomorphism) where it is not really required. This reasoning is based on the assumption that in a system composed of insulated processing units (modules) connected by cables, the geometry of modular layout is not critically important. It would be more important in a continuous interaction system. Mosaic-modular and continuous-interactive designs reflect the two more plausible relationships between the two dichotomies in question. This means that the evidence for the continuous principle of cortical organization at the same time provides indirect support for its interactive rather than modular nature. Since the concept of continuous organization developed here is most compelling with respect to the middle, associative segments of the previously discussed cognitive gradients, it can be proposed that the interactive principle is also most applicable to the middle, associative, intermodal aspects of the cognitive cortical gradients.

3. Prededicated vs. Emergent Principles of Cortical Organization

The prededicated principle implies that the functions of cerebral structures are genetically prespecified in a strong way. The emergent principle implies that *a priori* constraints on the functional designations of cerebral structures are weak and the latter assume their function in large measure through self-organizing processes in neural networks.

The two dichotomies, mosaic vs. continuous, and prededicated vs. emergent are not orthogonal, since a neural network which is mosaic and emergent at the same time is a virtual impossibility. This is due to the fact that the mosaic principle of organization permits a random relationship between functional and neuroanatomical "distances." If we believe in the adaptive wisdom of the brain, it is clear that a self-organizing neural network is not likely to develop in this fashion. Instead, it can be expected to follow the principle of spatial economy, which means adherence to the principle of functional-neuroanatomical isomorphism, and a continuous organization.

An argument in favor of a mosaic nature of neocortical functional organization would have supported in the same breath its strong prededication. As it stands, I have argued that it is fundamentally continuous. Although this conclusion does not dictate a particular choice within the prededicated vs. emergent dichotomy in a logical sense, intuitively it favors a substantial role of the *emergent* principle in neocortical functional organization. While realizing that intuitions can be misleading, I will pursue this possibility further and

explore some of its consequences.

If gradiental functional organization is to a large degree the result of self-organizing processes in neural networks, then one must distinguish between its *a priori* and *resultant* aspects. In its specifics, the gradiental scheme presented here reflects *only* the invariant aspects of cortical localization in the adult, mature brain of an individual who is a product of statistically prevalent cultural and experiential circumstances. It represents an asymptotic end-point of the changes of neocortical functional organization throughout childhood and adolescence, which parallel developmental reorganization of cognitive structures.

With respect to the resultant pattern of localization the following question can be asked: Does it exist in the sense that there are distinct cortical areas intrinsically invested with specific functions? Or is our discrete nomenclature of functionally distinct cortical areas only an attempt to force essentially continuous distributions into a finite taxonomy? If so, then our neocortical functional units are nothing more than discrete "flags" to approximate gradients which are essentially continuous and without distinct borders. The cytoarchitectonic and pathway heterogeneity of neocortex is not in doubt, nor is the relevance of its neuroanatomical to its functional heterogeneity. It is proposed, however, that ample room is left for the emergent properties of the neocortex, which arise in accordance with the principle of continuous distributions and are superimposed on the neuroanatomically fixed neocortical characteristics.

It is precisely the assumption of the emergent nature of functional gradients according to the rule of neuroanatomical-cognitive isomorphism, that enables us to regard neuroanatomical and cognitive gradients in an almost interchangeable fashion insofar as the adult, mature pattern of cortical functional organization is considered. We can interpolate the functional description of any arbitrary locus along a continuous cognitive gradient, and it is hoped that the gradiental model will serve as the neuropsychological analogue of the Mendeleev periodic table. This is achieved, paradoxically, through the refutation of the mosaic principle of organization.

Comparison of the Wernicke's and anomic aphasias provides an illustration of "discrete" syndrome descriptions approximating an inherently continuous functional distribution in the posterior portion of the left hemisphere. The former is caused by lesions in the posterior part of the superior temporal gyrus, and is therefore closer to the auditory, temporal end of the left temporo-parietal gradient. Accordingly, it has a strong phonological component. Paraphasias seen in Wernicke's anomia are usually literal, reflecting their phonological basis. Anomic aphasia is associated with more posterior lesions, close to the temporo-occipital junction, in the posterior portion of the middle temporal gyrus. It does not have a distinct phonological component, and appears to be more purely lexical (Goodglass, 1980; Goodglass & Geschwind, 1976).

The transition between "speech apraxia", "syntactic agrammatism", and

"dynamic aphasia" provides a similar illustration of "discrete" syndrome descriptions approximating an inherently continuous functional distribution in the anterior portion of the left hemisphere.

Finally, if the existence of inherently distinct cortical loci could be questioned, so can be the existence of inherently distinct higher order cognitive functions with which these loci are presumably invested. It is possible that our elaborate taxonomies of distinct higher order cognitive functions are but attempts to capture in discrete, finite terms essentially continuous patterns of shades and mixing of a relatively small set of cognitive primitives. This applies also to the notion of "cognitive levels", extensively used in this paper, which represent nothing other than a taxonomic devise. Certain syndromes can be best described as transitional with respect to these levels, reflecting the continuous nature of neocortical functional organization.

The gradiental approach allows one to make distinct predictions regarding the geometry of such neocortical functional distributions. Thus it predicts that a congenitally blind person with a good language facility who learned about physical objects mostly through tactile inputs, will develop anomic aphasia following a stroke along the temporo-parietal and not temporo-occipital gradient, in the supramarginal rather than middle temporal gyrus territory.

4. From Modules to Gradients: An Evolutionary Hypothesis

Examination of the relationship between discrete, modular, prededicated, and continous, interactive, emergent principles of organization is particularly elucidating when thalamo-cortical relationships are considered. The relationship between cortical areas and corresponding specific thalamic nuclei is so close as to approach one-to-one mapping.

What then was the evolutional advantage for a totally new type of cerebral organization, neocortex, to emerge? Why was it not sufficient for a quantitative elaboration on the nuclear, thalamic principle of organization to take place, until the required levels of neural complexity could be attained?

I propose that a general phylogenetic shift had taken place from the emphasis on the modular ganglion to that on the interactive, continuous principle of cerebral organization. The advent of neocortex represents this shift. Thalamus is older than neocortex phylogenetically. Thalamic organization is essentially modular. It consists of separate nuclei, each connected to other structures in a limited input-output fashion. There is very little intrathalamic interactiveness.

Neocortex is a sheet, at least from a macroscopic standpoint (I do not believe that the properties of vertical columnar organization are central to this level of analysis), and its organization is essentially interactive, as this review attempted to show. Probably not all neocortex adheres to the interactive principle of organization to an equal degree. However, to the extent that the modular principle is applicable to some aspects of neocortex, it embraces most readily stimulotopic primary sensory projection areas, considerably less so secondary cortices, and is hardly applicable at all to the associative, tertiary, supramodal

cortices (inferoparietal, inferotemporal and prefrontal). The more phylogeneti-
cally recent a type of neocortex is, the less readily it can be understood in
modular terms and the more readily in interactive terms. The degree of the
"ontological reality" of distinct cortical functional "loci" and of corresponding
cognitive functions as opposed to their inherent fictitiousness (in the sense
discussed in the previous section) probably varies. The more phylogenetically
recent and "interactive" different cortices are, the less "real" are their
functional subdivisions and corresponding "higher order" cognitive constructs.

The transition from modularity and functional prededication to interactive
and emergent principles can be seen as a basic evolutional trend in the
phylogeny of the central nervous system which parallels the quantitative
increase of neural network complexity and ensures more adaptive and flexible
principles of organization. It is almost as if the intellectual evolution of our
ways of thinking about computing, from sequential to parallel distributed,
recapitulated the biological evolution of the first and ultimate computor, the
brain. Yet it is possible and even likely that both principles play a role in the
highly developed brain, and that the wisdom of nature has been in dialectically
combining them instead of completely negating the older principle in favor of
the newer one. Further analysis of thalamo-cortical functional relations will
contribute to a better understanding of the interplay between the modular and
interactive principles of cerebral organization.

Finally, I wish to emphasize that the foregoing analysis and conclusions must
be restricted to the macroscopic level of description of brain-behavioral
relations. I do not believe that these conclusions are automatically applicable to
the more microscopic, cellular level of description, where an inherently modular
and relatively strongly prededicated nature of units such as a neuron with a
receptive field, or a vertical column (Goldman-Rakic, 1984; Hubel & Wiesel,
1962; Mountcastle, 1961) may well be more pronounced.

REFERENCES

Albert, M.L., Reches, A., & Silverberg, R. (1975). Associative visual agnosia without
     alexia.*Neurology, 25*, 322-326.
Bauer, R.M., & Rubens, A.B. (1985). Agnosia. In K.M. Heilman & E. Valenstein (Eds.).
     Clinical neuropsychology (pp.187-242). New York: Oxford University Press.
Beauvois, M.F. (1982). Optic aphasia: A process of interaction between vision and
     language. *Philosophical Transactions of the Royal Society, London, B298*, 35-47.
Benson, F.D. (1985). Aphasia. In K.M. Heilman & E. Valenstein (Eds.). *Clinical
     neuropsychology* (pp.17-48). New York: Oxford University Press.
Benson, D.F., & Geschwind, N. (1969). The alexias. In P.J. Vinken & G.W. Bruyn (Eds.).
     Handbook of clinical neurology: Disorders of speech, perception and symbolic behavior
     (pp. 112-140). New York: Elsevier.
Benson, D.F., Segarra, J., & Albert, M.L. (1974). Visual agnosia prosopagnosia: A
     clinicopathological correlation. *Archives of Neurology, 30*, 307-310.

Benton, A. (1985). Body schema disturbances: Finger agnosia and right-left disorienta-
tion. In K.M. Heilman & E. Valenstein (Eds.). *Clinical neuropsychology* (pp. 141-158).
New York: Oxford University Press.

Benton, A.L., Smith, K.C., & Lang, M. (1972). Stimulus characteristics and object
naming performance in aphasia adults. *Journal of Communicative Disorders, 5,* 19-24.

Bernstein, N.A. (1947). *The construction of movements.* Moscow: Medgiz.

Bernstein, N.A. (1967). *The coordination and regulation of movements.* New York:
Pergamon Press.

Bilder, R.M., & Goldberg, E. (1987). Motor perseveration in schizophrenia. *Archives of
Clinical Neuropsychology, 2,* 195-214.

Bisiach, E. (1966). Perceptual factors in the pathogenesis of anomia. *Cortex, 2,* 90-95.

Bogen, J.E. (1969). The other side of the brain II: An appositional mind. *Bulletin of Los
Angeles Neurological Society, 34,* 135-162.

Brown, W.S., Lehmann, D., & Marsh, J.T. (1980). Linguistic meaning-related differen-
ces in evoked potential topography; English, Swiss-German and Imagined. *Brain and
Language, 11,* 340-353.

Bruner, J.S. (1966). On cognitive growth, I, II. In J.S. Bruner, R.R. Oliver, & P.M.
Greenfield (Eds.). *Studies in cognitive growth: A collaboration at the Center for
Cognitive Studies* (pp. 1-67). New York: John Wiley.

Caramazza, A., Berndt, R.S., & Brownell, H.H. (1982). The semantic deficit hypothesis
of the naming deficit: Perceptual parsing and object classification by aphasic patients.
Brain and Language, 15, 161-189.

Carpenter, M.B. (1976). *Human neuroanatomy.* Baltimore: Williams and Wilkins.

Coughlan, A.K., & Warrington, E. (1978). Word comprehension and word retrieval in
patients with localized cerebral lesions. *Brain, 101,* 163-185.

Damasio, A. (1985). The frontal lobes. In K.M. Heilman & E. Valenstein (Eds.). *Clinical
neuropsychology* (pp. 339-376). New York: Oxford University Press.

Damasio, A.R., McKee, J., & Damasio, H. (1979). Determinants of performance in
color anomia. *Brain and Language, 7,* 74-85.

de Ajuriaguerra, J., Hécaen, H., & Angelergues, S. (1960). Les apraxies cliniques et
latéralization lesionelle. *Revue Neurologique, 102,* 566-594.

Denny-Brown, D. (1958). The nature of apraxia. *Journal of Nervous and Mental
Disorders, 126,* 9-33.

Denny-Brown, D. (1963). The physiological bases of perception and speech. In H.
Halpern (Ed.). *Problems of dynamic neurology* (pp. 1-33). Jerusalem: Jerusalem Post
Press.

De Renzi, E., & Spinnler, H. (1966). Visual recognition in patients with unilateral
cerebral disease. *Journal of Nervous and Mental Disorders, 142,* 513-525.

De Renzi, E., Pieczulo, A.,& Vignolo, L.A. (1968). Ideational apraxia: A quantitative
study. *Neuropsychologia, 6,* 41-52.

Faglioni, P., Spinnler, H.,& Vignolo, L.A. (1969). Contrasting behavior of right and left
hemisphere-damaged patients on a discriminative and a semantic task of auditory
recognition. *Cortex, 5,* 366-389.

Fodor, J.A. (1983). *The modularity of mind.* Cambridge: MIT Press.

Fodor, J.A. (1985). Precis of the Modularity of Mind. *The behavioral and brain sciences,
8,* 1-42.

Foerster, O. (1936). Symptomatologie der Erkankungen des Gehirns. Motorische Felder
und Bahnen. Sensible corticale Felder. In O. Bumke & O. Foerster (Eds.). *Handbuch*

der Neurologie (pp. 154-187). Berlin: Springer.

Foix, C. (1916). Contribution à l'étude de l'apraxie idéomotrice. *Revue Neurologique, 1,* 285-298.

Foix, C. (1922). Sur une variété de troubles bilateraux de la sensibilité par lésion unilaterale du cerveau. *Revue Neurologique, 29,* 322-331.

Freud, S. (1891). *Zur Auffassung der Aphasien.* Leipzig und Wien: Deuticke.

Friedman, R.B.,& Albert, M.L.(1985) Alexia. In K.M. Heilman & E. Valenstein (Ed.). *Clinical neuropsychology* (pp. 49-74). New York: Oxford University Press.

Fulton, J.F. (1937). Forced grasping and groping in relation to the syndrome of the premotor area. *Archives of Neurology and Psychiatry, 31,* 27-47.

Geschwind, N. (1965). Disconnexion syndromes in animals and man, I, II. *Brain, 88,* 237-294; 585-644.

Geschwind, N. (1967). The varieties of naming errors. *Cortex, 3,* 97-112.

Geschwind, N.,& Kaplan, E. (1962). A human cerebral deconnection syndrome. *Neurology, 12,* 675-685.

Goldberg, E. (1978). Brain-behavioral relations in humans: A gradiental approach. Psychiatry Grand Rounds lecture presented at the Cornell College of Medicine.

Goldberg, E. (1985). Amnesia, tardive dysmentia, and frontal lobe disorder in schizophrenia. *Schizophrenia Bulletin, 11,* 255-263.

Goldberg, E. (1986). Varieties of motor perseveration: Comparison of two taxonomies. *Journal of Clinical and Experimental Neuropsychology, 8,* 710-726.

Goldberg, E., & Bilder, R. (1987). Frontal lobes and hierarchic organization of neurocognitive control. In E. Perecman (Ed.). *The frontal lobes revisited* (pp. 159-188). New York: IRBN Press.

Goldberg, E.,& Costa, L.D. (1986). Qualitative indices in neuropsychological assessment: An extension of Luria's approach to executive deficit following prefrontal lesions. In I. Grant & K.M. Adams (Eds.). *Neuropsychological assessment of neuropsychiatric disorders* (pp. 48-64). New York: Oxford University Press.

Goldberg, E., & Tucker, D. (1979). Motor perseveration and long-term memory for visual forms. *Journal of Clinical Neuropsychology, 1,* 273-288.

Goldman-Rakic, P.S. (1984). Modular organization of prefrontal cortex. *Trends in Neuroscience, 7,* 419-424.

Goodglass, H. (1980). Disorders of naming following brain injury. *American Scientist, 68,* 647-655.

Goodglass, H., & Geschwind, N. (1976). Language disorders (Aphasia). In E.C. Carterette & M. Friedman (Eds.). *Handbook of perception* (pp. 234-258). New York: Academic Press.

Goodglass, H., Barton, M., & Kaplan, E.F. (1968). Sensory modality and object-naming in aphasia. *Journal of Speech and Hearing Research, 11,* 488-496.

Goodglass, H., Hyde, M.R., & Blumstein, S. (1969). Frequency, picturability, and availability of nouns in aphasia. *Cortex, 5,* 104-119.

Goodglass, H., Klein, B., Carey, P., & Jones, K. (1966). Specific semantic word categories in aphasia. *Cortex, 2,* 74-89.

Grober, E., Perecman, E., Kellar, L., & Brown, J. (1980). Lexical knowledge in anterior and posterior aphasics. *Brain and Language, 10,* 318-330.

Head, H. (1926). *Aphasia and kindred disorders of speech.* New York: Cambridge University Press.

Hécaen, H. (1967). Approche semiotique des troubles du geste. *Langage, 5,* 67-83.

Hécaen, H., & Albert, M.L. (1978). *Human neuropsychology.* New York: John Wiley.

Hécaen, H., & Angelerques, R. (1963). La cécité psychique. Paris: Masson et Cie.

Hécaen, H., Goldblum, M.C., Masure, M.C., & Ramier, A.M. (1974). Une nouvelle observation d'agnosie d'object. Déficit de l'association, ou de la catégorisation, spécifique de la modalité visuelle? *Neuropsychologia, 12,* 447-464.

Heilman, K.M., & Rothi, L.J.G. (1985). Aphasia. In K.M. Heilman & E. Valenstein (Eds.). *Clinical neuropsychology* (pp. 159-185). New York: Oxford University Press.

Henschen, S.E. (1925). Clinical and anatomical contributions on brain pathology. *Archives of Neurology and Psychiatry, 13,* 226-249.

Hermann, G., & Poetzl, O. (1926). *Über die Agraphie und ihre localdiagnostischen Beziehungen.* Berlin: Karger.

Hoff, H., Gloning, J., & Gloning, K. (1954). Über Alexie. *Wiener Zeitschrift für Nervenheilkunde, 10,* 149-162.

Hoff, H., & Poetzl, O. (1935). Über ein neues parieto-occipitales Syndrom. *Journal der Psychiatrie und Neurologie, 52,* 173-218.

Hubel, D.H., & Wiesel, T.N. (1962). Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *Journal of Physiology, 160,* 106-154.

Jackson, J.H. (1931-1932). *Selected Writings of John Hughlings Jackson.* 2 volumes. London: Hodder & Stoughton.

Johns, D.F., & Darley, F.L. (1970). Phonetic variability in aphasia of speech. *Journal of Speech and Hearing Research, 13,* 556-583.

Kleist, K. (1934). *Gehirnpathologie.* Leipzig: Barth.

Levin, H.S., & Spiers, P.A. (1985). Acalculia. In K.M. Heilman & E. Valenstein (Eds.). *Clinical neuropsychology* (pp. 97-114). New York: Oxford University Press.

Lhermitte, F. (1986). Human autonomy and the frontal lobes II. Patient behavior in complex and social situations. The "environmental dependency syndrome". *Annals of Neurology, 19,* 335-343.

Lhermitte, J., & de Ajuriaguerra, J. (1938). Asymbolie tactile et hallucinations de touches. Etude anatomoclinique. *Revue Neurologique, 19,* 492-495.

Lhermitte, F., Chedru, F., & Chain, F. (1973). A propos d'un cas d'agnosie visuelle. *Revue Neurologique, 128,* 301-322.

Lhermitte, F., Pillon, B., & Serdaru, M. (1986). Human autonomy and the frontal lobes I. Imitation and utilization behavior: A neuropsychological study of 75 patients. *Annals of Neurology, 19,* 326-334.

Liepmann, H. (1900). Das Krankheitsbild der Apraxie. *Monatsschrift für Psychiatrie und Neurologie, 8,* 15-44; 102-132, 182–197.

Liepmann, H. (1908). *Drei Aufsätze aus dem Apraxiegebiet, Vol. 1,* Berlin: Karger.

Liepmann, H. (1920). Apraxia. *Ergebnisse der gesammten Medizin, 1,* 516-543.

Lissauer, H. (1890). Ein Fall von Seelenblindheit nebst einem Beitrag zur Theorie derselben. *Archiv für Psychiatrie und Nervenkrankheiten, 21,* 222-270.

Luria, A.R. (1965). Two kinds of motor perseveration in massive injury of the frontal lobes. *Brain 88,* 1-10.

Luria, A.R. (1970). *Traumatic aphasia.* The Hague: Mouton.

Luria, A.R. (1980). *Higher cortical functions in man.* New York: Basic Books.

Luria, A.R., & Tsvetkova, L.S. (1968). The mechanisms of dynamic aphasia. *Foundations of Language, 4,* 296-307.

McCarthy, R., & Warrington, E.K. (1985). Category specificity in an agrammatic

patient: The relative impairment of verb retrieval and comprehension. *Neuropsychologia, 23,* 709-727.

McClelland, J.L., Rumelhart,D. E., & the PDP research group. (1986). *Parallel distributed processing. Vol. 2: Psychological and biological models.* Cambridge: MIT press.

Micelli, G., Mazzuchi, A., Mann, L., & Goodglass, H. (1983). Contrasting cases of Italian agrammatic aphasia without comprehension disorder. *Brain and Language, 19,* 65-97.

Micelli, G., Silver, M.C., Villa, G., & Caramazza, A. (1984). On the basis for the agrammatic's difficulty in producing main verbs. *Cortex, 20,* 207- 220.

Miller, G.A., Galanter, E., & Pribram, K.H. (1960). *Plans and the structure of behavior.* San Francisco: Holt.

Miller, G.A., & Johnson-Laird, P.N. (1976). *Language and perception.* Cambridge: The Belknap Press of Harvard University Press.

Mishkin, M., Ungerleider, L. G., & Macko, K.A. (1983). Object vision and spatial vision: Two cortical pathways. *Trends in Neurosciences, 6,* 414-417.

Mountcastle, V.B. (1961). Some functional properties of the somatic afferent system. In W.A. Rosenblith (Ed.) *Sensory communication.* (pp. 403-436). Cambridge: MIT Press.

Nielsen, J.M. (1937). Unilateral cerebral dominance as related to mind blindness: Minimal lesion capable of causing visual agnosia for objects. *Archives of Neurology and Psychiatry, 38,* 198-135.

Nielsen, J.M. (1946). *Agnosia, apraxia, aphasia: Their value in cerebral localization.* New York: Hober.

Nielsen, J., & Raney, R. (1938). Symptoms following surgical removal of major (left) angular gyrus. *Bulletin of the Los Angeles Neurological Societies, 3,* 42-46.

North, E. (1971). Effects of stimulus redundancy on naming disorders in aphasia. Ph.D. dissertation. Boston University (Quoted after Goodglass, 1980).

Pribram, K.H. (1987). The subdivisions of frontal cortex revisited. In E. Perecman (Ed.). *The frontal lobes revisited* (pp. 11-40). New York: IRBN Press.

Rubens, A.B., & Benson, D.F. (1971). Associative visual agnosia. *Archives of Neurology, 24,* 305-316.

Rumelhart, D. E., McClelland, J.L., & the PDP research group. (1986). *Parallel Distributed Processing. Vol. 1: Foundations.* Cambridge: MIT press.

Saffran, M.E., Schwartz, M.E., & Marin, O.S.M. (1980). The word order problem in agrammatism. *Brain and Language, 10,* 263-280.

Sperry, R.W. (1966). Brain bisection and counsciousness. In J.Eccles (Ed). *Brain and counscious experience* (pp. 325-354). Wien: Springer-Verlag.

Spinnler, H., & Vignolo, L.A. (1966). Impaired recognition of meaningful sounds in aphasia. *Cortex, 2,* 337-348.

Tissot, R., Mounin, G., Lhermitte, F., & Dordain, G. (1973). *L'Agrammatisme.* Bruxelles: Charles Dessart.

Trost, J.E., & Canter, G.J. (1974). Apraxia of speech in patients with Broca's aphasia: A study of phoneme production accuracy and error patterns. *Brain and Language, 1,* 63-79.

Vignolo, L.A. (1982). Auditory agnosia. *Philosophical Transactions of the Royal Society, B298,* 49-57.

Vygotsky, L.S. (1962). *Thought and language.* Cambridge: MIT Press.

Warrington, E.K. (1975). The selective impairment of semantic memory. *Quarterly*

Journal of Experimental Psychology, 27, 635-657.

Wernicke, C. (1894). *Grundriss der Psychiatrie in klinischen Vorlesungen. Psychophysiologische Einleitung.* Leipzig: G. Thieme.

Whitehouse, P., Caramazza A., & Zuriff, E.B. (1978). Naming in aphasia: Interacting effects of form and functions. *Brain and Language, 6,* 63-74.

Yamadori, A., & Albert, M.L. (1973). Word category aphasia. *Cortex, 9,* 83-89.

Zuriff, E., Caramazza, A., Myerson, R., & Galvin, J. (1974). Semantic feature representation for normal and aphasic language. *Brain and Language, 1,* 167-187.