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Rise and Fall of Modular Orthodoxy*

Elkhonon Goldberg
New York University School of Medicine

ABSTRACT

The premise of cortical modularity is based on strong dissociations caused by focal lesions. These dissociations are rare, and their explanatory power and theoretical importance are vastly overrated. The effects of brain lesions must be considered in their totality, rather than in idiosyncratic selectivity. These effects are more consistent with a continuous, graded functional neocortical geometry, than with a modular neocortex. Distinction must be drawn between strong intrinsic modularity, and weak emergent modularity. Strong intrinsic modularity is more characteristic of the thalamus than of the cortex. The advent of neocortex may have represented an evolutionary escape from strong modularity as the dominant principle of neural organization, and a shift toward a more interactive principle of neural organization dominated by emergent properties. The latter may take the form of weak modularity, reflective of cognitive skill routinization. The extent of weak, emergent modularization may be asymmetric, more pronounced in the left hemisphere, while the right hemisphere is essentially amodular.

STRONG DISSOCIATIONS: ENTHUSIASM BETRAYED

Modularity is defined, in part, as informational encapsulation. This is the condition of brain science, not of the brain itself. The concept of modularity is a peculiar product of that brand of cognitive science, that deals with the effects of brain damage in the context of narrow experimental protocols involving very select patient groups, or none at all. The nature of questions asked then predetermines the nature of answers derived. By phrasing questions in terms of distinct macroscopic operations and restricting the patient groups to those with extreme macroscopic phenomena, one inevitably biases one's perception of the brain in favor of a discontinuous model.

The modularity concept owes its recent revival and prominence to the highly biased way in which cognitive science approaches brain damage. In its search for strong dissociations, cognitive scientists often ignore the fact that such dissociations are rare and may very well represent statistical aberrations.

The far more numerous cases of weak dissociations, which point to the graded nature of functional cortical organization, are discounted as uninformative. Neuropsychological discovery and theory-building has become extremely dependent on the search for "interesting cases", and their automatic theoretical value has become an article of faith. Even when groups of related cases characterized by strong dissociations and, therefore, deemed to be of particular theoretical significance are considered, the epidemiological perspective is often lost. The number of the "mundane" cases that it takes to sieve through, in order to find the precious few "strong dissociations" is ignored.

The theoretical inference based on the cases of strong dissociations, then, becomes circular and solipsistic. The solipsistic nature of such reasoning has been explicitly acknowledged by

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Reprint requests to Elkhonon Goldberg, Department of Psychiatry, New York University Medical Center, 550 First Avenue, New York, NY 10016, USA.
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Fodor (1983, 1985), who claimed that the brain must be modular in order to be knowable. The concept of modularity became so influential precisely because, like every simplistic concept, it has the seductiveness and illusory appeal of instant explainability (by introducing a new module for every new observation). Epistemologically primitive, however, the modular view of the brain fails the first requirement imposed on any explanatory theory, that of information reduction. Like the belief systems of antiquity, it merely re-labels its domain by inventing a separate deity for every thing.

The frequently made claim that even a single strong dissociation proves a theoretical point, is flawed. In symbolic logic, the proof of existence of entity N means precisely that – its existence at least as a single instance ($Ex < X: x = N$). The power of such a proof is limited to the rejection of the claim that N cannot possibly exist ($Ax < X: x = -N$). Proving that N exists is of an undeniable theoretical importance in neuropsychology or any other empirical discipline if the prevailing belief has been that N is impossible. In all other cases such a proof is in and of itself of limited utility. It does not imply the universality of N (i.e., that $Ax < X: x = N$), or even its statistical prevalence (i.e., that for most $x < X: x = N$). The same limitation is inherent in the group studies of strong dissociations, if the instances of such dissociations are taken out of the epidemiological context.

In cognitive neuropsychology, the significance of the proof of existence is often stretched to imply its universality owing to two tacit assumptions: that the variability of lesion anatomy is great, and the variability of premorbid cognitive organization is negligible across individuals. Then the rarity of a particular strong dissociation is attributed to the low probability of the lesion neatly affecting a particular "module" and it alone. Once demonstrated to exist, however, the dissociation is presumed to be equally revealing of the cognitive architecture across all individuals. The combination of a specimen-invariant cognitive architecture and functional neuroanatomy, and a marksman-precision lesion, is the presumed basis of both the existence of a strong dissociation, and of its universality.

The reasoning behind this inference is flawed. It ignores the possibility that the variability in clinical presentations may have nothing to do either with lesion anatomy, or with the principles of cerebral organization. Instead, it may reflect a wide range of premorbid, individual factors that are either completely extraneous to the functional neuroanatomy or are interactive with it in rather complex ways.

While the tacit assumption of lesion variability is self-evident, the tacit assumption of the invariant (across individuals) nature of cognitive architecture and functional neuroanatomy is probably wrong. Functional neuroanatomy has been shown to depend on the individual's experience with the task (Goldberg & Costa, 1981; Ross-Kossak & Turkewitz, 1986). This implies a dynamic, rather than static, nature of functional neuroanatomy both within and across individuals, and this alone may account for tremendous individual differences in the patterns of neurocognitive architectures. The relationship between functional neuroanatomy and sex and handedness may be more profound and complex than previously thought (Goldberg, Podell, Harner, Lovell, & Riggio, 1994).

Finally, a host of premorbid individual idiosyncracies, trivial in retrospect but commonly overlooked in neuropsychological research, may compound the picture. In combination, these factors will make mockery of any neuropsychological inference predicated on the assumption of the invariance of neurocognitive architecture, – and this assumption is central to the premise of the theoretical importance of rare strong dissociations. As a result, rare strong dissociations will be reduced to the level of uninterpretable statistical aberrations.

Consider the following. I am a native Russian-speaker with a reasonable command of English, who came to the United States as a young adult. My proficiency in both languages is extremely state- dependent and replete with strong dissociations. When tired, inebriated, or indisposed, I lose access to concrete lexicon (e.g., household utensils) but not abstract lexicon in English, and to abstract lexicon (e.g., scientific terminology) but not, for the most

part, concrete lexicon in Russian. Certain lexical domains (e.g., names of flowers and fishes) will be found equally severely, and strikingly, impaired in both languages, since I never learned them in either. A good friend of mine, an eminent cross-cultural psychologist from Southern California, is a native English-speaker with a reasonable command of Russian. He describes equally strong state-dependent dissociations, similar in character but not in specifics, in both languages.

Should either of us have the misfortune of suffering a stroke, the cognitive neuropsychological theory may be affected differently, depending on which of us would be examined, and on which side of the Atlantic. "Strong dissociations" will be promptly documented and reported, all due to utterly premorbid, idiosyncratic circumstances of our respective personal histories, absurdly unrelated to anything of neuroscientific consequence.

One might argue that bilingualism is a relatively uncommon condition, but other low-prevalence cognitive factors may play a role in other individuals. In combination, the exceptions may override the rule. The assumption of the invariance of neurocognitive architecture and the resultant functional neuroanatomy can be graphically depicted as a flat cognitive landscape, but this landscape emerges only as an averaging abstraction. Every individual premorbid cognitive landscape is a combination of peaks and valleys, where the disparities in altitudes may be quite dramatic. My near-complete ignorance of fish and flower names in my own native language is the case in point.

The effect of a nonspecific neurological insult on a vastly uneven cognitive landscape can be compared with a flood, which will submerge the valleys but spare the peaks. Graded differences between premorbid competencies will then assume the appearance of "strong dissociations", and the trusting neuropsychologist will be swept by the sea of artifacts.

This goes to show that taking the isolated cases of "strong dissociations" out of their proper epidemiological context is a hazardous activity, wrought with ruinous consequences for the neurocognitive theory. Our search for "interesting cases" at the expense of the mundane ones is like focussing on a few trees and ignoring the forest.

The current infatuation with strong cortical dissociations is not the first such self-inflicted detour that our field had to take. It was preceded by an equally strong infatuation with subcortical dissociations as they pertained to memory and amnesias. This led to two enduring and influential claims: that anterograde amnesia is invariably more severe and more prevalent than retrograde amnesia (Barbizet, 1970; Russell & Nathan, 1946), and that retrograde amnesia invariably affects episodic knowledge but spares semantic knowledge (Kinsbourne & Wood, 1975; Schacter & Tulving, 1982). Both claims inspired far-reaching theoretical speculations regarding the underlying neurocognitive architecture. Both have since been challenged and proven wrong (Barr, Goldberg, Wasserstein, & Novelly, 1990; Butters & Cermak, 1986; DeRenzi, Liotti, & Michelli, 1987; Goldberg et al., 1981; Goldberg & Barr, 1992; Roman-Campos, Poser, & Wood, 1980; Zola-Morgan, Cohen, & Squire, 1983).

This is not to say that all instances of isolated strong dissociations are theoretically useless. This is to say, however, that they must be approached with a degree of weariness, pending the demonstration of their high prevalence in the presence of a particular lesion location, or/and converging evidence from other sources. Isolated demonstrations of strong dissociation should not be treated with an unrestrained enthusiasm as the major tool of neuropsychological discovery and theory-building.

WEAK DISSOCIATIONS AND CONTINUOUS FUNCTIONAL DISTRIBUTIONS

When the effects of focal lesions on cognition are placed in an epidemiologically realistic perspective, a very different type of neurocognitive theory is encouraged, one dominated by the notion of very weak dissociations indicative of continuous functional distributions in the neocortex.

In 1989, I proposed a specific form of this

general family of theories, which I termed at the time "gradiental". Flawed as it may be in its specifics, the approach explicates a set of general assumptions about the neocortical functional geometry, which is finally beginning to gain acceptance among cognitive neuroscientists and neuropsychologists. These assumptions will probably gain in the near future the status of the dominant paradigm of cognitive neuroscience. They will replace the modularity premise or, more likely, will place it into a proper evolutionary perspective. I will briefly review the gradiental approach to neocortical functional organization here, with the eye on the general underlying principles, rather than architectural details.

In a sense, the gradiental model traces its lineage to the work of Alexandr Luria, in particular to his notion of "dynamic functional systems", and his assumption of a three-level functional cortical hierarchy (Luria, 1980). I developed the gradiental model while a graduate student of Luria's in the early 1970s.

The examination of the geometry of known neuropsychological syndromes in their entirety, rather than in arbitrary isolation, leads to the conclusions which are sharply at variance with the modular model. It indicates that neocortical functional architecture is graded rather than modular (Goldberg, 1989, 1990a). The continuous principle of neocortical functional organization reflects strong congruence between the neuroanatomical and functional cortical geometries, which stems from the fundamental observation that two neocortical lesions will disrupt cognitively close processes if, and only if, their neuroanatomical territories are close. In its strong form, this relationship can be described as cognitive/neuroanatomical isomorphism or near-isomorphism.

The reality of the neocortical spatial-functional isomorphism is captured in the gradiental model. A cognitive gradient is a continuous distribution of related functions along an axis defined at its extremes by a pair of sensory and motor projection areas.

The notion of a cognitive gradient is only as viable as the premise of neocortical spatial-functional isomorphism is correct.

The model was originally designed as a way of establishing a Mendeleev table-like, ordered taxonomy of known neuropsychological syndromes, which would permit functional-neuroanatomical interpolation. This, in turn, allows inference about the underlying normal functional geometry, and leads to the conclusion that the known neuropsychological syndromes and the postulated normal cognitive operations are but discrete taxonomic approximations of inherently continuous cortical functional distributions.

The gradiental model considers arrays of functionally distinct neuropsychological syndromes, whose cortical territories are aligned along the gradients defined at its extremes by two areas of primary sensory cortical projections (A, B). Indeed, it can be demonstrated that along each such an axis, damage to the areas physically closest to sensory projection area A will disrupt functions dominated by that sensory modality. As one moves away from pole A, one encounters areas whose damage will disrupt functions critical for intermodal integration between modalities A and B. As one proceeds further along the axis toward pole B, one encounters cortical regions whose damage disrupts functions dominated by sensory modality B.

For taxonomic reasons (rather than out of belief in the intrinsic reality of a discrete classification), I will regard any such cognitive gradient as consisting of three types of areas. First, it includes two primary cortical sensory projection areas at its extremes (level 1). Adjacent to each of them is an area that in hierarchic terms would be labeled a level 2 area, that is modality-specific association cortex. Finally, in the center of the gradient a level 3 area is found, which can be characterized as intermodal association cortex.

Below, I will review a detailed gradiental structure of the posterior portion of the left hemisphere of a right-hander. This is done merely for illustrative purposes. A comparably detailed account of the gradiental structure of other parts of the neocortex is available elsewhere (Goldberg, 1989, 1990).

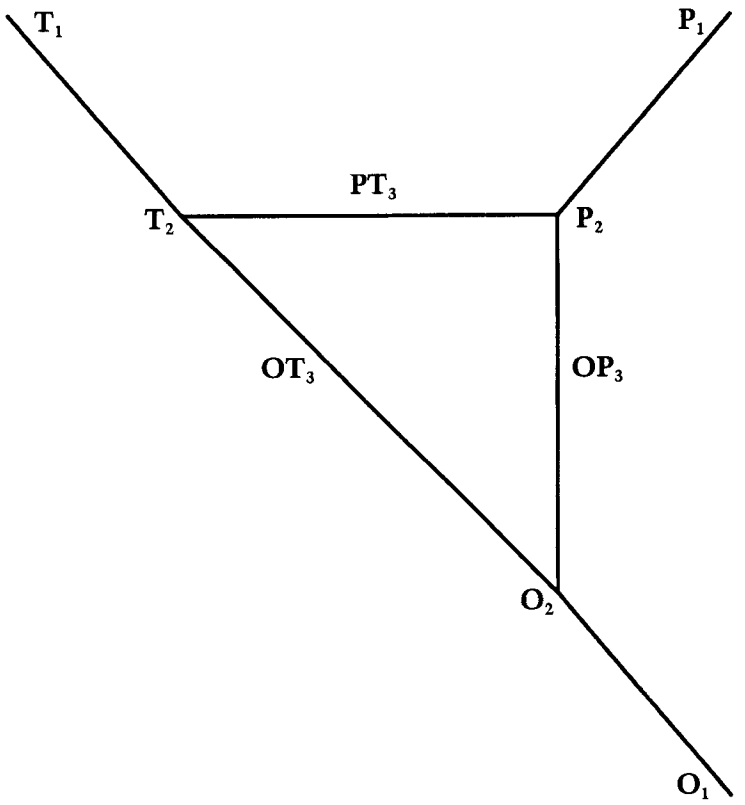

Fig. 1. Gradiental structure of the posterior half of the left hemisphere.
O1 – primary visual cortex (Brodmann area 17); T1 – primary auditory cortex (Brodmann areas 41 and 42); P1 – primary somatosensory cortex (Brodmann areas 3, 1 and 2). O2 – visual association cortex (Brodmann areas 18 and 19); T2 – auditory association cortex (Brodmann area 22, or posterior portion of the superior temporal gyrus); P2 – somatosensory association cortex (Brodmann areas 5 and 7, or superior parietal lobule). OT3 – inferotemporal association cortex; PT3 – supramarginal gyrus/ inferoparietal association cortex; OP3 – angular gyrus/inferoparietal association cortex.
Damage to O2 produces visual object agnosia; to OT3 – anomia of Wernicke's type (when more anterior) and anomic aphasia (when more posterior); to T2 – acoustic agnosia and semantic associative auditory; to PT3 – semantic aphasia and acalculia of spatial type; to P2 – pure astereognosis; and to OP3 – ideational apraxia and spatial apractagnosia.

ILLUSTRATION OF THE CONCEPT: GRADIENTAL STRUCTURE OF THE POSTERIOR LEFT HEMISPHERE

By definition, three cognitive gradients can be discerned 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; occipito-parietal gradient by the O-P pair; and the temporo-parietal gradient by the T-P pair.

The extreme points of the three gradients are defined by pairs of the three areas of primary sensory cortical projections: the occipital striate Brodmann area 17 (01), the parietal, postcentral gyrus Brodmann areas 3, 1, and 2 (P1), and the temporal Heschl area, Brodmann areas 41 and 42 (T1), (Carpenter, 1976).

I will refer to modality-specific level 2 components as O2, P2, and T2; and to intermodal, level 3 components involved in intermodal integration as OP3, OT3, and PT3. Figure 1 provides a schematic representation of the posterior cortical gradiental structure of the left hemisphere.

Associative agnosias and modality-specific association cortices of the left hemisphere

The extreme points of the three left posterior gradients have just been defined. I will now specify level 2 components of the cognitive gradients of the posterior half of the left hemisphere. All level 2 modality-specific association areas in the left hemisphere can be characterized in similar terms. All three are critical for categorical stimulus identification, that is, recognizing specific exemplars as members of generic categories. When damage occurs to any of these three areas, a particular, modality-specific form of associative, "symbolic" agnosia occurs (Goldberg, 1990b).

Visual object agnosia is an extensively studied syndrome, also known as "associative blindness," or "psychic blindness" (Albert, Reches, & Silverberg, 1975; DeRenzi & Spinnler, 1966; Freud, 1891; Hécaen & Albert, 1978; Hécaen & Angelergues, 1963; Hécaen et al., 1974; Lissauer, 1890; Luria, 1980; Rubens & Benson, 1971; Warrington, 1975). The syndrome is characterized by an intact visual sensory and perceptual analysis, and an impaired ability to assign the visual stimulus to a generic category. The patient can accurately describe visual sensory and perceptual attributes of an object, but is unable to identify it by name, through a functional description, or a pantomime. The deficit is not one of naming, and the deficit is limited to visual input. When the patient is allowed to touch, hear, or smell the object, the identification becomes possible.

Visual object agnosia is caused by damage to 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, Goldblum, et al., 1974; Rubens & Benson, 1971; Warrington, 1975). While the bilateral nature of the lesion is sometimes emphasized (Albert et al., 1975; Hoff & Potzl, 1935), the left occipital area is the critical lesion site (Benson et al., 1974; Hécaen, Goldblum, et al., 1974; Lhermitte, Chedru, & Chain, 1973; Nielsen, 1937; Rubens & Benson, 1971; Warrington, 1975).

This clinical material merits the conclusion 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. It corresponds to area O2 in the diagram of the left posterior gradiental structure (Figure 1).

Tactile agnosia, known also as "pure astereognosis," or "tactile asymbolia" is 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, but it can be accomplished in other sensory modalities. The ability to describe elementary tactile properties of the stimulus is spared.

Tactile agnosia is caused by damage to the secondary parietal areas of the left hemisphere, that is, Brodmann areas 5 and 7, or the superior parietal lobule (Lhermitte & de Ajuriaguerra, 1938; Luria, 1980). Therefore, the normal function of this area appears to involve the categorical identification of tactile and proprioceptive percepts. This is area P2 in the schematic representation of the left posterior gradiental structure (Figure 1).

Acoustic agnosia is the term used by Luria (1980) for 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.

The deficit is categorical in nature: 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, which has also been construed as the breakdown of phonological retrieval (Goodglass, 1980). The critical lesions responsible for "acoustic agnosia" and "Wernicke's aphasia" are virtually identical: both involve the posterior portion of the superior temporal gyrus in the left hemisphere.

A related syndrome of "semantic associative" auditory agnosia (Faglioni, Spinnler, & Vignolo, 1969; Spinnler & Vignolo, 1966; Vignolo, 1982) involves an inability to associate nonverbal sounds with their 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 cooccurs with Wernicke's aphasia and their neuroanatomical territories overlap. Acoustic agnosia/Wernicke's aphasia and semantic associative auditory agnosia probably represent two aspects of the same syndrome with the same underlying mechanism caused by left temporal damage.

This clinical material merits the conclusion 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. 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 1).

All three secondary modality-specific, level 2 areas of the posterior portion of the left hemisphere share the same fundamental property. They ensure the capacity for identifying unique stimuli as members of generic categories. Each of the three areas ensures this in a particular, single sensory modality: visual, somatosensory, or auditory. Modality-specific categorical perception appears to be the basic function of the posterior portion of the left hemisphere. This perspective is distinctly different from the one regarding language as the fundamental attribute of the left hemisphere. It has been explored in detail elsewhere (Goldberg, 1990b).

Neurolinguistic digression: Cortical representation of language

Consider two alternative models of the cerebral representation of lexical knowledge: (a) it is modular in nature, and separated from the cerebral representations of those aspects of the physical world which it denotes; (b) it is distributed in close neuroanatomical relationship with cerebral representations of the various aspects of the physical world denoted by it.

Modular separation of lexical versus perceptual cerebral representations was implicit in the "disconnection" models of anomia (Geschwind, 1965, 1967; Geschwind & Kaplan, 1962). The alternative idea of the distributed nature of cerebral representation of lexicon is supported by the findings that semantic knowledge itself is impaired in anomias. This implies that in anomic patients, the representation of the physical world is itself disrupted.

Consistent with this possibility, categorization processes are impaired in anomic patients not only for names, but also for pictorial object representations (Grober, Perecman, Kellar, & Brown, 1980; Zurif, Caramazza, Myerson, & Galvin, 1974). Furthermore, anomic patients fail to use "semantically guided, perceptual parsing" in classifying pictorial representations (Caramazza, Berndt, & Brownell, 1982; Whitehouse, Caramazza, & Zurif, 1978); and perceptual degradation of pictorial images exacerbates anomia (Benton, Smith, & Lang, 1972; Bisiach, 1966; North, 1971).

This implies that lexical and perceptual representations are intertwined and involve similar encoding units (Miller & Johnson-Laird, 1976). It is likely, further, that the cerebral substrates of the two types of representations strongly overlap neuroanatomically. This implies a distributed, as opposed to modular, nature of cortical representation of lexicon. Lexical-semantic representations will then be localized in close proximity to, or even overlap with, the areas in which representations of the 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. In humans, representations of objects are likely to be encoded with a particular reliance on the visual modality (Beauvois, 1982; Goodglass, Burton, & 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) involve the cortical areas central to visual processing; and cerebral representations of concrete verbs involve the premotor cortex.

Furthermore, the representations of certain classes of objects may be dimensionalized along a greater number of sensory modalities than others. The distributed view of the lexicon predicts that this will be reflected in the relative robustness of the corresponding lexical domains. Artificial ("man-made") objects usually serve a function. Therefore, the somatosensory and motor dimensions are integral to their distributed representations. Natural objects by and large are encoded with less, or no, reliance on motor and somatosensory dimensions. This may explain why the names or representations of inanimate objects are less vulnerable in anomias or agnosias, than are those of living things (Hart, Berndt, & Caramazza, 1985; Schecter, 1953; Warrington & McCarthy, 1983; Warrington & Shallice, 1984), since most inanimate objects are man-made, and all living things are natural. This may also explain why color names are particularly vulnerable in anomias (Damasio, McKee, & Damasio, 1979), since colors rely on a single sensory modality, whereas object representations are to various degrees multimodal.

Lexical deficit is common in posterior, Wernicke's, and amnestic, aphasias (Coughlan & Warrington, 1978; Grober et al., 1980; Whitehouse et al., 1978; Yamadori & Albert, 1973; Zurif et al., 1974). In most studies, however, only nouns were used. Only a limited body of data exists regarding the noun-verb dissociation. Goodglass, Klein, Carey, and Jones (1966) did not find any difference in verb versus noun proficiency between anterior and posterior aphasics. Micelli, Marruchi, Mann, and Goodlglass (1983) described a massive omission of verbs but not nouns following a focal lesion in the Broca's area. Micelli, Silver, Villa, and Caramazza (1984) also demonstrated a double dissociation between verbs and nouns, and agrammatic versus anomic aphasics. In agrammatic aphasics, action naming was more impaired was than object naming. The opposite was true in anomic aphasics. Micelli et al. (1984), and McCarthy and Warrington (1985) propose that agrammatism in anterior aphasias is, at least in part, due to a lexical deficit for verbs. According to Luria (1980), temporal lobe aphasias are characterized by a particularly severe impairment of nouns and to some extent adjectives; parieto-occipital aphasias by impairment of prepositions and adverbial clauses; and anterior aphasias (i.e., Broca's and Kleist's) by a particularly severe disintegration of verbs. McCarthy and Warrington (1985) reported a case of impaired comprehension and retrieval of verbs and action names with an intact competence for nouns. Using electrophysiological indices, Brown, Lehman, and Marsh (1980) observed noun-verb, anterior-posterior double dissociation in normal subjects.

Anomia due to posterior lesions may be characterized by a greater impairment of concrete or easily picturable nouns, than abstract (i.e., devoid of a distinct sensory image) nouns (Goodglass, Heyde, & Blumstein, 1969; Warrington, 1975). Yamadori and Albert (1973) described anomia for body parts and room objects but not tools (whose representations are likely to have a stronger motor component), following a left posterior lesion. The circumlocutions in this patient were replete with action-words, for example, "to help people walk" for "cane", "to sit on" for "chair."

Anterior aphasias, on the other hand, are often characterized by the nominalization of verbs (Goodglass & Geschwind, 1976; Luria, 1980, Saffran, Schwartz, & Marin, 1980). Conspicuous paucity of action words is common in Kleist's dynamic aphasia following left frontal damage (Luria, 1970; Luria & Tsvetkova, 1968).

Ample evidence exists that cortical 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 the corresponding aspects of the physical world denoted by these lexical domains. With this in mind, we may proceed with our review of the cortical gradients of the posterior portion of the left hemisphere.

Syndromes of the left temporo-occipital intermodal cortex

"Fluent" aphasias. The central portion of the temporo-occipital cognitive gradient, its level 3 OT portion, comprises the association cortex of the inferior 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. Lexical deficit in fluent aphasias involves particularly the lexical domains which denote those aspects of the physical world represented with maximum reliance on the visual modality. This selectivity of lexical deficit in posterior aphasias is a reflection of the distributed nature of the cortical representation of lexicon.

Consequently, the normal function of the central portion of the left occipito-temporal gradient (posterior portion of the temporal lobe at the temporo-occipital junction) appears to mediate those aspects of linguistic representations which denote visually based aspects of the physical world. The role of this area consists of auditory-visual integration within the linguistic context, which is precisely what the rule of functional-spatial neocortical isomorphism would predict for this cortical region.

I will now review the intermodal, level 3 components of the remaining two cognitive gradients of the posterior left hemisphere. The neuroanatomical territories of the middle portions of the occipito-parietal and temporo-parietal gradients are adjacent. Both are within the territory of the inferior parietal lobule, which consists of two subdivisions, the supramarginal and the angular gyrus. Geometrically, the angular gyrus can be assigned to the occipito-parietal gradient, and the supramarginal gyrus to the temporo-parietal gradient. Then the angular gyrus becomes the intermodal OP area, and the supramarginal gyrus the intermodal TP area.

Syndromes of the left occipito-parietal intermodal cortex

Ideational apraxia refers to the disintegration of skilled, overlearned, object-oriented movements. Individual motor acts may be intact, but their integration into coherent motor patterns fails (Hécaen & Albert, 1978; Liepmann, 1900, 1908). There is no associated paresis or ataxia. The deficit is bilateral and general rather than segmental with respect to body parts, but is produced by a unilateral lesion within the posterior parietal and temporoparietal regions of the left hemisphere (de Ajuriaguerra, Hécaen, & Angelergues, 1960; DeRenzi, Pieczulo, & Vignolo, 1968; Hécaen & Albert, 1978); particularly the left angular gyrus (Foix, 1916; Heilman & Rothi, 1985).

Spatial apractagnosia refers to a group of symptoms, presumably representing a unitary syndrome, affecting visual processing of asymmetric, representational spatial arrays, such as maps, clocks, letters, and digits (Luria, 1980). "Spatial apractagnosia" is caused by lesions in the occipito-parietal junction within the left angular gyrus.

The other component of "apractagnosia" is known as "alexia with agraphia", or "parietal alexia" (Friedman & Albert, 1985; Hermann & Potzl, 1926; Hoff, Gloning, & Gloning, 1954). It may occur in the absence of frank aphasia. "Alexia with agraphia" and the related deficit, "alexia/agraphia for numbers" are caused by lesions of the left angular gyrus (Hécaen, 1967; Henchen, 1925; Levin & Spiers, 1985; Nielsen & Raney, 1938).

Ideational apraxia and "apractagnosias" (including alexia and agraphia) usually co-occur (Friedman & Albert, 1985; Luria, 1980). They represent two forms of the same fundamental deficit: disintegration of processing of representational visuo-spatial arrays. "Apractagnosia" is by definition a deficit of process-

ing of symbolic, generic representations. Ideational apraxia is "categorical" since the affected "motor engrams" are invariant across a variety of specific conditions, applicable to any object of a given class and executable by any limb under any metric circumstances.

This clinical material merits the conclusion that the left parieto-occipital junction/angular gyrus is critical for intermodal integration of higher-order visual and somatosensory/spatial information, which is symbolic, representational in nature. It corresponds to OP3, the level 3 component of the occipito-parietal gradient. The cognitive properties of this area are consistent with its cortical geometry. Both cognitively and anatomically, this area is situated between the visual modality-specific and the somatosensory/spatial modality-specific areas. The rule of cognitive-neuroanatomical isomorphism holds along the left occipito-parietal cognitive gradient. It can be otherwise referred to as the visuospatial gradient of symbolic representations.

Syndromes of the left temporo-parietal intermodal cortex

Semantic aphasia

Lesions of the left temporo-parietal areas impair the comprehension and expression of asymmetric propositional constructions (Head, 1926; Luria, 1970, 1980). Spatial ("to the right of" vs. "to the left of"), temporal ("before" vs. "after") and quantity relations ("smaller" vs. "larger", "taller" vs. "shorter"), passive voice and possessive case ("father's brother" vs. "brother's father") may all be affected. The patient cannot interpret reversible clauses, unless lexical redundancy is present. By contrast, phonological and lexical competence is spared. Termed by Luria "semantic aphasia", this syndrome closely resembles "morphological agrammatism" described by Tissot, Mounin, Lhermitte, and Dordain (1973).

Acalculia of the spatial type is characterized by disintegration of the decimal structure of numbers and appreciation of the spatial asymmetries implicit in this structure (Levin & Spiers, 1985; Luria, 1980). The patients cannot properly align compound multi-digit numbers in written computations or even understand their meaning. Unlike in alexia/agraphia for numbers, the ability to read or write separate digits is preserved. Spatial acalculia is caused by inferoparietal, post-Rolandic lesions (Levin & Spiers, 1985; Luria, 1980).

"Semantic aphasia" may be caused by the disintegration of the spatial basis of relational constructions (Luria, 1980). This implies that normal cognitive representations of all relational concepts are spatial or "quasi-spatial" in nature, regardless of the denotate. Acalculia of the spatial type may also be secondary to the disintegration of the spatial basis of numerical concepts (Luria, 1980). This implies that normal cognitive representations of numbers and numeric operations are substantially spatial in nature, a conclusion born out by cultural-anthropological and developmental evidence as well.

"Semantic aphasia" and "acalculia of the spatial type" are probably two manifestations of the same fundamental cognitive deficit: disintegration of the spatial basis of cognitive representations underlying linguistic and other representational (e.g., numeric) codes. Indeed, "semantic aphasia" and acalculia often co-occur.

The cognitive dimension whose disintegration leads to "semantic aphasia" and "spatial acalculia," is one of the interface and integration of auditory-based linguistic (and quasi-linguistic) codes and somatosensory-based spatial schemata. The cognitive properties of the cortical area in question are consistent with its left temporo-parietal location. Both cognitively and anatomically, this area is situated between the auditory modality-specific and the somatosensory/spatial modality-specific areas. The rule of cognitive-neuroanatomical isomorphism holds along the left temporo-parietal cognitive gradient.

THE NEW PARADIGM

From modular to distributed design

The continuous, distributed nature of functional

cortical geometry and the spatial-functional isomorphism are captured in the distinct triangular gradiental structure of the posterior half of the left hemisphere. The ordering inherent in a strong cognitive/neuroanatomic congruence would be redundant and superfluous in a brain consisting of prededicated modules. By the same token, such congruence is a natural emergent property of a biological self-organizing neural map. Indeed, the emergence of spatial/functional congruence as a result of learning, has been shown to be an important dynamic characteristic of formal self-organizing neural maps (Kohonen, 1989).

The gradiental model argues against a mosaic, modular, and prededicated, and in favor of a continuous, interactive, and emergent nature of neocortical functional organization. It provides the basis for a rational and systematic inquiry into the functional organization of a nonmodular neocortex, since it permits the interpolation of the functions of particular cortical areas even in the absence of direct lesion-based evidence (Goldberg, 1990).

Modularity and evolution

Is modularity in its orthodox version, involving encapsulated and prededicated processing components, a useful model of anything at all? To the extent that it is, it probably reflects the early principles of cerebral organization mediated by the thalamus. A shift from the modular thalamic to an interactive cortical principle of neural organization may have represented a major step in the evolution of the brain (Goldberg, 1989).

The thalamus and the cortex are closely interconnected. The thalamus is often viewed as the precursor of the cortex, containing in a primitive way all of its functions. While functionally close, the thalamus and the cortex differ radically in neuroanatomical structure. The thalamus consists of distinct, structurally encapsulated, sparsely interconnected nuclei. The cortex, to the contrary, is not inherently modular, at least not at the level of gross neuroanatomy. It is a sheet without distinct internal borders, with rich pathways connecting most areas with most others.

If the thalamus is a close functional prototype of the cortex, then what was the evolutionary raison d'être for the emergence of the cortex? What in evolution promoted the introduction of a fundamentally new principle of neural architecture, rather than the refinement of an already existing one? Why was the emergence of a neural sheet, the cortex, adaptively preferable to the elaboration on the thalamic theme – that is, more and bigger nuclei? The question is admittedly a teleological one, but we ask teleological questions all the time in our quest to understand complex systems, biological, economic, and social.

The probable answer is that different neural architectures are optimal for different levels of organizational complexity. Up to a point, modular organization is optimal. With the increasing computational demands, however, the transition toward a heavily interconnected net consisting of a larger number of simpler interactive elements becomes necessary to ensure adaptive success. Throughout evolution, the emphasis has shifted from the brain invested with rigid, fixed functions (thalamus) to the brain capable of flexible adaptation (cortex). The advent of neocortex may have represented an evolutionary repudiation of strong modularity as the dominant principle of neural organization, and a shift toward a more interactive principle of neural organization dominated by emergent properties.

With the advent of the cortex, the thalamus did not disappear, however. Instead, a two-tier neural organization has evolved, with the thalamus surrendering its primacy to the cortex, but still playing a role by imposing weak patterning on the cortical sheet.

Weak modularity as an emergent property

Does cortical functional localization exist in the sense that distinct cortical areas are 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 merely conceptual "histograms" to approximate essentially continuous distributions. The gradiental approach allows one to

make distinct predictions regarding the emergent geometry of such neocortical functional distributions. It predicts, for instance, that a congenitally blind person who learned about physical objects mostly through tactile input, 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.

Furthermore, if the existence of inherently distinct cortical loci is questioned, so can be the existence of inherently distinct higher-order cognitive functions with which these loci are presumably invested.

Is modularity a useful concept for describing neocortical functional architecture at least in some respects? Evidently not, in its extreme, orthodox form. To the extent that it may be at all useful, the notion of weak, emergent modularity must be introduced. Moscovitch and Umiltà (1990) offered an update of the modularity concept, that recognizes the role of the emergent characteristics of the neurocognitive architecture, and acknowledges the relative nature of differences between modules and other computational devices.

If the continuous, gradiental functional organization is the result of self-organizing processes in neural networks, then one must distinguish between its *inherent* and *emergent* aspects. The formation of strongly interactive local neuronal groupings may be an adaptively important emergent property of a net that is initially very weakly prededicated and is characterized by continuous interactions. This would not be incompatible with the continuous model, which distinguishes between the a priori and the resultant aspects of functional cortical organization. Such weak, emergent modules would adhere to the gradiental functional distributions described earlier in this paper.

Emergent modularity has been noted to develop in formal neural nets (Edelman, 1987; Jacobs & Jordan, 1992; Willner, Miranker, & Chien-Ping Lu, 1990). Such a "weak", dynamic modularity will be very different from the "strong", static Fodorian (1983, 1985) modularity, in that the net will be characterized by a constant change of modular "geography", dissipation of old modules, and the emergence of new ones.

The refutation of strong modularity does not necessarily mean refutation of locality, because locality does not necessarily require discrete borders between locations. The existence of cortical functional gradients suggests a major role for local interactions within the grey matter mediated by short, nonmyelinated pathways. The review of cortical neuropsychological syndromes suggests that these pathways in many respects override the effects of longitudinal fasciculi, which interconnect distant regions. These local interactions, however, are characterized by continuous transitions, and by the absence of inherent regionality. Paralleling these conclusions, it has been shown that an architectural bias toward short-range connections is a desirable property of a formal neural net at least in certain computational contexts (Jacobs & Jordan, 1992; Jacobs, Jordan, & Barto, 1991).

The concept of weak, emergent modularity remains a speculative construct awaiting its critical evaluation. Its existence is far from proven. Nonetheless, it is the only tenable modification of the modularity principle, relevant to phylogenetically advanced cortical, as opposed to archaic thalamic, processes.

Emergent modularity and hemispheric specialization

To the extent that weak, emergent modularity is a real phenomenon, it is the consequence, and possibly the mechanism, of the formation and routinization of cognitive skills. This leads to the interesting possibility that the degree of emergent cortical modularity is asymmetric.

The lateralized nature of weak emergent modularity follows from the growing evidence that the novelty-routinization distinction is at the heart of the functional complementarity between the two cerebral hemispheres. The right hemisphere appears to be critical for the initial processing of novel cognitive tasks to which none of the previously formed cognitive representations or strategies can be readily applied. The left hemisphere appears to be critical for processing based on well-formed

representations and strategies (Goldberg, 1990; Goldberg & Costa, 1981; Goldberg, Vaughan, & Gerstman, 1978; Ross-Kossak & Turkewitz, 1986;Tucker & Williamson, 1984).

The process of a cognitive skill acquisition is, then, characterized by a right-to-left shift of the locus of cortical control over the corresponding processes, as they evolve, routinize, and possibly become modularized. Consequently, the functional geometry of the left hemisphere is characterized by weak, emergent modularity, whereas the right hemisphere is by and large amodular. Consistent with this proposition, is the evidence that connectivity via short, nonmyelinated pathways is emphasized in the left hemisphere, and connectivity via long, myelinated pathways in the right (Gur et al., 1980). The right-to-left shift of the locus of cortical control is viewed as a general pattern of cognitive skill acquisition under normal conditions. It should not be confused with the changes of cortical functional distributions as the result of acquired brain damage and subsequent plasticity (Luria, Simernitskaya, & Tubylevich, 1970).

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Today, few scientists will admit to taking the orthodox form of the modularity premise seriously. Yet what makes a concept powerful, is its strong, uncompromised, evangelical kernel, and not its temperate modifications. The concept of modularity became so influential precisely because, like every simplistic concept, it has the seductiveness and illusory appeal of instant explainability (by introducing a new module for every new observation). In the privacy of their crania, so to speak, many scientists probably still fall back on the literal version of modularity as a form of intellectual shorthand. With all the embellishments, disclaimers, and caveats which accompany the modularity claim these days, it remains, unfortunately, the dominant construct of the field.

Modularity theorists sometimes draw the distinction between the architecture of the cognitive system, which comprises the proper domain of the modular theory, and its implementation in the central nervous system. If valid, this distinction would insulate the modularity premise from much of the criticism based on neurobiological and neurological considerations. Yet the assumption that the "cognitive" architecture has an ontological status of its own, independent of the "neural" architecture, is itself under fire. It can be argued that in a neural net, biological and artificial alike, the changing pattern of connections *is* the cognitive architecture. The growing realization of an inherent inseparability of cognition and neural structure is reflected in the advent of the connectionist view of cognition, and of neural net modeling as the tool of *cognitive science.*

In this paper, I have attempted to capture a curious parallel between the biological evolution *of* the brain, and the intellectual evolution of our thinking *about* the brain. Both are characterized by a paradigmatic shift, from modular to interactive. In cognitive neuroscience, the time for this paradigmatic shift has come.

REFERENCES

Albert, M.L., Reches, A., & Silverberg, R. (1975). Associative visual agnosia without alexia. *Neurology, 25,* 322-326.

Barbizet, J. (1970). *Human memory and its pathology,* San Francisco: W. H. Freeman.

Barr, W. B., Goldberg, E., Wasserstein, J., & Novelly, R. A. (1990). Retrograde amnesia following unilateral temporal lobectomy. *Neuropsychologia, 22,* 243-255.

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 of London, B298,* 35-47.

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.

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

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

Butters, N., & Cermak, L. S. (1986). A case study of the forgetting of autobiographical knowledge: Implications for the study of retrograde amnesia. In D. Rubin (Ed.), *Autobiographical memory* (pp. 253-272). New York: Cambridge University Press.

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 & Wilkins.

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

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

de Ajuriaguerra, J., Hécaen, M., & Angelergues, S. (1960). Les apraxies cliniques et lateralization lesionelle. *Revue neurologique, 102*, 566-594.

DeRenzi, E. D., Liotti, M., & Michelli, P. (1987). Semantic amnesia with preservation of autobiographic memory: A case report. *Cortex, 23*, 575-597.

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.

Edelman, G. M. (1987). *Neural darwinism.* New York: Basic Books.

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.

Foix, C. (1916). Contribution a l'etude de l'apraxie ideomotorice. *Revue Neurologique, 1*, 285-298.

Freud, S. (1891). *Zur Auffassung der Aphasien.* Vienna: Deuticke.

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

Geschwind, N. (1965). Disconnection syndromes in animals and man. *Brain, 237*, 585-644.

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

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

Goldberg, E. (1989). Gradiental approach to neocortical functional organization. *Journal of Clinical and Experimental Neuropsychology, 11*, 489-517.

Goldberg, E. (1990a). Higher cortical functions in humans: The gradiental approach. In E. Goldberg (Ed.), *Contemporary neuropsychology and the legacy of Luria* (pp. 229-276). Hillside: Lawrence Erlbaum.

Goldberg, E. (1990b). Associative agnosias and the functions of the left hemisphere. *Journal of Clinical and Experimental Neuropsychology, 12*, 467-484.

Goldberg, E., Antin, S. P., Bilder, R. M., Gerstman, L. J., Hughes, J. E. O., & Mattis, S. (1981). Retrograde amnesia: Possible role of the mesencephalic reticular activation in long-term memory. *Science, 213*, 1392-1394.

Goldberg, E., & Barr, W. B. (1992). Selective knowledge loss in activational and representational amnesias. In L. R. Squire and N. Butters (Eds.), *Neuropsychology of memory*, (2nd ed.) (pp. 72-80). New York: Guilford Press.

Goldberg, E., & Costa, L. D. (1981). Hemisphere differences in the acquisition and use of descriptive systems. *Brain and Language, 14*, 144-173.

Goldberg, E., Podell, K., Harner, R., Lovell, M., & Riggio, S. (1994). Cognitive bias, functional cortical geometry, and the frontal lobes: Laterality, sex, and handedness. *Journal of Cognitive Neuroscience, 6*, 276-296.

Goldberg, E., Vaughan, H. G., & Gerstman, L. J. (1978). Nonverbal descriptive systems and hemispheric asymmetry: Shape versus texture discrimination. *Brain and Language, 5*, 249-257.

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.* New York: Academic Press.

Goodglass, H., Burton, M.J., & 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 the 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.

Gur, R. C., Packer, I. K., Hungerbuhler, J. P., Reivich, M., Obrist, W. D., Amarnek, W. S., & Sackheim, H. A. (1980). Differences in the distribution of gray and white matter in human cerebral hemispheres. *Science, 207*, 1226-1228.

Hart, J., Berndt, R. S., & Caramazza, A. (1985). Category-specific naming deficit following cerebral infarction. *Nature, 316*, 439- 440.

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

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

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

Hécaen, H., & Angelerques, R. (1963). *La Cecite phychique.* Paris: Masson et Cie.

Hécaen, H., Goldblum, M.C., Masure, M.C., et al. (1974). Une nouvelle observation de'agnosie d'object. Deficit de l'association, ou de la categorisation, specifique de la modalite visuelle? *Neuropsychologia, 12*, 447-464.

Heilman, K. M., & Rothi, L. J. G. (1985). Apraxia. 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., & Potzl, O. (1926). *Uber die agraphie und ihre localdiagnostischen beziehungen.* Berlin: Krager.

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

Hoff, H., & Potzl, O. (1935). Uber ein neues parietooccipitaler syndrom. *Journal of Psychiatric Neurology, 52*, 173-218.

Jacobs, R. A., & Jordan, M.I. (1992). Computational consequences of a bias toward short connections. *Journal of Cognitive Neuroscience, 4*, 323-336.

Jacobs, R. A., Jordan, M. I., & Barto, A. G. (1991). Task decomposition through competition in a modular connectionist architecture: The what and where vision tasks. *Cognitive Science, 15*, 219-250.

Kinsbourne, M., & Wood, F. (1975). Short-term memory processes and the amnestic syndrome. In D. Deutsch, & J.A. Deutsch (Eds.), *Short-term memory* (pp. 259-291). New York: Academic Press.

Kohonen, T. (1989). *Self-organization and associative memory*, (3rd ed). New York: Springer-Verlag.

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

Lhermitte, J., & de Ajuriaguerra, J. (1938). Asymbolie tactile et hallucinations du 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.

Liepmann, H. (1900). Das Kranskheitsbild der Apraxie. *Monatsschrifft der Psychiatrie und Neurologie,* 8.

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

Lissauer, H. (1890). Einfall von Sedonblindheit nebst einen Beitrag zur Theorie derselben. *Archiven fur Psychiatrie, 21*, 222-270.

Luria, A.R. (1970). *Traumatic aphasia.* New York: Mouton.

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

Luria, A.R., Simernitskaya, E.G., & Tubylevich, B. (1970). The structure of psychological processes in relationship to cerebral organization. *Neuropsychologia, 8*, 13-19.

Luria, A.R., & Tsvetkova, L.S. (1968). The mechanisms of dynamic aphasia. *Foundations of Language, 4*, 28-43.

McCarthy, R., & Warrington, E.K. (1985). Category specificity in an agrammatic patient: The relative impairment of verb retrieval and comprehension. *Neuropsychologia, 23*, 709-727.

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., & Johnson-Laird, P.N. (1976). *Language and Perception.* Cambridge, MA: The Belknap Press of Harvard University Press.

Moscovitch, M., & Umiltà, C. (1990). Modularity and neuropsychology: Modules and central processes in attention and memory. In M. F. Schwartz (Ed.), *Modular deficits in Alzheimer's disease* (pp. 1-59). Cambridge, MA: MIT Press-Bradford.

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., & 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.* Doctoral dissertation. Boston University (Quoted after Goodglass, 1980).

Roman-Campos, G., Poser, C. M., & Wood, F. B. (1980). Persistent retrograde deficit after transient global amnesia. *Cortex, 16*, 509-518.

Ross-Kossak, P., & Turkewitz, G. (1986). A micro and macro developmental view of the nature of changes in complex information processing: A consideration of changes in hemispheric advantage during familiarization. In R. Bruyer (Ed.), *Neuropsychology of facial expression.* (pp. 52-78). Hillsdale, NJ: Erlbaum.

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

Russell, E. W., & Nathan, P. W. (1946). Traumatic amnesia. *Brain, 69*, 280-300.

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

Schacter, D. L., & Tulving, E. (1982). Memory, amnesia and the episodic-semantic distinction. In R. L. Isaacson, & N. E. Spear (Eds.), *The expression of knowledge* (pp. 35-65). New York: Plenum Press.

Schecter, M. D. (1953). Visual agnosia for animate objects. *Journal of Nervous and Mental Disease*, 341-344.

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

Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. *Psychological Review, 91*, 185-215.

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

Warrington, E.K. (1975). The selective impairment of semantic memory. *Quarterly Journal of Experimental Psychology, 27*, 635-657.

Warrington, E. K., & McCarthy, R. (1983). Category specific access dysphasia. *Brain, 106*, 859-878.

Warrington, E. K., & Shallice, T. (1984). Category specific naming impairments. *Brain, 107*, 829-853.

Wernicke, C. (1894). *Grundriss der psychiatrie. Psychophysiologische Einleitung.*

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

Willner, B. E., Miranker, W. L., & Chien-Ping Lu (1990). *Neural organization of the locomotive oscillator.* IBM Research division: Research Report.

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

Zola-Morgan, S., Cohen, N. J., & Squire, L. R. (1983). Recall of remote episodic knowledge in amnesia. *Neuropsychologia, 21*, 487-500.

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