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The Marin Lab at the Dawn of Cognitive Neuropsychology

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Abstract

This essay discusses the intellectual developments in psychology, linguistics, and behavioral neurology that shaped Oscar Marin's approach to disorders of high cortical function. As Chief of Neurology at Baltimore City Hospitals in the 1970s, Dr Marin teamed with biopsychologist Eleanor Saffran and the author in seminal studies of acquired language disorders (aphasia) centering on core processes of syntax and semantics, and rejecting premature reductionism. The philosophical and methodological principles that motivated these studies are traced through the author's personal recollections and the published writings of the Marin lab. These principles came to be associated with the cognitive neuropsychology school of research and have important linkages to contemporary work in the neuroscience of aphasia and related cognitive disorders.

Keywords

Oscar Marin; aphasia; cognitive neuropsychology; single-case studies; levels of analysis

In 1975, the year I joined Oscar Marin's laboratory at the Johns Hopkins Medical Institutions in Baltimore, Maryland, cognitive psychology was just coming into its own (Miller, Galanter, and Pribram, 1960; Neisser, 1976; Posner, 1973) and the Noam Chomsky-led revolution in language studies was in full swing (Chomsky, 1965; Fodor, Bever, and Garrett, 1974). Oscar had absorbed these exciting developments through his contact with the University of Pennsylvania's Department of Psychology (see Paul Rozin's article in this issue). That was in the years before Oscar moved to Baltimore to take up a position as Associate Professor of Neurology at the Johns Hopkins University School of Medicine and Chief of Neurology at Baltimore City Hospitals. At Baltimore City Hospitals, he established a lab specializing in the cognitive analysis of disorders of high cortical function, and, in particular, aphasia. The work conducted in that laboratory over the next 10 years helped launch the field known today as cognitive neuropsychology.

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THE BEGINNINGS OF COGNITIVE NEUROPSYCHOLOGY

The first and best decision Oscar made in setting up his lab was to hire Dr Eleanor Saffran to run it. Eleanor's PhD was in biopsychology, but it was human cognition that truly excited her imagination. As a postdoctoral fellow at the University of Pennsylvania, she had attended Oscar's case presentations and seminars, and, through them, had become aware of the extraordinary possibilities that neurology offered for teasing apart the functional components of cognition. In Oscar's lab, she set about exploring the myriad presentations of language breakdown manifest in patients referred from the Hopkins neurology clinics.

By the time I joined the lab, with my own freshly minted PhD in biopsychology, Eleanor and Oscar had established an active research program in aphasia and related cognitive disorders (Marin and Saffran, 1975; Saffran and Marin, 1975, 1977; Saffran, Marin, and Yeni-Komshian, 1976).

Boston, not Baltimore, was the geographical heart of aphasia research at the time. Norman Geschwind's highly influential papers on neurological disconnection syndromes rekindled interest the 19th century Broca-Wernicke anatomical model of the aphasias (Geschwind, 1965, 1979). At the Boston Veterans Administration Hospital, the aphasia unit established by Geschwind had grown to become a major center for research and training in aphasia. Under the leadership of Harold Goodglass and Edith Kaplan, the Boston Aphasia Center became renowned for groundbreaking multidisciplinary research on the psycholinguistic description of aphasic syndromes (Goodglass, 1973; Goodglass, Hyde, and Blumstein, 1969; Goodglass, Kaplan, Weintraub, and Ackerman, 1976; Goodglass and Kaplan, 1972).

The approach developed by the Marin lab drew less from the Boston aphasia tradition than from the work of the British neuropsychologists Elizabeth Warrington, Timothy Shallice, Frieda Newcombe, and John Marshall, whose imaginative single-subject investigations were revealing theoretically significant associations and dissociations in cognitive functions (Marshall and Newcombe, 1966, 1973; Shallice and Butterworth, 1977; Shallice and Warrington, 1977; Warrington, 1975; Warrington and Taylor, 1978; see also Albrecht Inhoff's article in this issue of the journal). Such mining of the cognitive symptoms of individual patients appealed to Oscar and played to his clinical strengths.

The clinical neurological exam is an exercise in deductive logic. To diagnose the source of a patient's pain or weakness, the physician engages in a process of hypothesis testing informed by knowledge of the hierarchical organization of the nervous system and how the patient responds to probing questions, sensory and motor system evaluations, and specialized diagnostic tests.

Oscar was a master clinical neurologist, but his area of specialization was disorders of high cortical function, for which nervous system anatomy and function offered few diagnostic guideposts. Fortunately, he was also knowledgeable in music theory, mathematics, linguistics, and, as already noted, psychology, all of which he used to good effect in the clinical setting. Readers familiar with the writings of the neurologist and author Oliver Sacks will appreciate the extraordinary insights that can issue from the examination of a patient by a physician who is also a polymath.

In retrospect, it is obvious how much Oscar's clinical diagnostic approach influenced the style of neuropsychological research that we carried out in his lab. In an overview of the neurobiology of language, written for the New York Academy of Sciences (Marin, 1976), Oscar wrote that clinical neurology:

...looks at the nervous system as a hierarchical organization of interdependent subsystems, many of which can be observed and studied in some lesser or greater degree of isolation. Thus, although we know that the organization of movement involves a series of structures and functional processes, the cerebellar or the cerebral cortical components can be analyzed in relative isolation from the rest.... In the case of language, we believe that the brain deals with the various perceptual, cognitive, and executive tasks, at least to a certain extent, by assigning them to different neural subsystems. If these systems can be disrupted independently by focal brain lesions, the clinical syndromes should provide a general picture of language-brain interactions. [page 904]

In a companion paper (Marin, Saffran, and Schwartz, 1976), we elaborated the assumptions underlying this approach and the implications for the study of normal language function:

[We assume] (1) that the nervous system is organized in terms of functionally meaningful subsystems, and that there is some degree of parallelism between functional organization and anatomical organization; (2) that at least some of these subsystems can be selectively impaired by neurological disease; and (3) that while organic pathology gives rise to a large variety of symptoms that reflect various mechanisms of inhibition, release, isolation, and so on, in no way do they represent the creation of new subsystems; rather, they reflect a reorganization that emphasizes intact subsystems. [page 869]

We should therefore be able to make some inferences about the organization of normal language function from *patterns of functional preservation and impairment*: if process X is intact where process Y is severely compromised or absent, and especially if the converse is found in other patients, there is reason to believe that X and Y reflect different underlying mechanisms in the normal state. At the very least, the resulting matrix of intact and impaired functions should yield a taxonomy of functional subsystems. [page 869]

After contrasting our approach to others that select and group patients based on the lesion site or an aphasia diagnosis (Broca aphasia, etc), we made the argument for the study of individual, "privileged" cases:

It is our contention that at this stage in the endeavor, more useful insights into normal language function will be provided by the detailed consideration of privileged cases, cases where there is still sufficient language to study language behavior, and where there are rather clean dissociations of function.... But we are not suggesting that the single case should be the final testing ground for theory; rather, that it can serve a useful hypothesis-generating function. And some confidence in the results can be sought in the consistency with which cases relate to

one another; put together, they should overlap and interlock to provide, eventually, a comprehensive picture of the language process. [page 871]

These are now familiar as bedrock principles of the cognitive neuropsychology school and of the eponymous journal, founded in 1984 (Caramazza, 1984, 1986; Coltheart, 1984; Saffran, 1982).

THE LEVELS-OF-ANALYSIS PROBLEM

In later years, the cognitive neuropsychology enterprise would come under criticism for focusing on the psychological level at the expense of the brain (Patterson and Plaut, 2009). As practiced in the Marin lab, at least, this was a strategy born of necessity. All 3 of us were neurobiologists at heart, with a shared goal of discovering language-brain relationships. We were keenly aware that we faced a formidable levels-of-analysis gap. In his 1976 paper, Oscar wrote:

It is clear that an adequate neurobiology of language will require the integration of data from disciplines that diverge radically in goals, subject matter, and methodology. A serious problem, then, is the specification of levels of description and units of analysis on both sides of the brain-behavior equation. Is it, for example, reasonable to attempt to correlate a linguistic theory with the microstructure of the brain, or are other levels of analysis more appropriate? [page 900]

He continued with a provisional answer:

In the last two decades a great deal of progress has been made in the description of the language system. Contemporary transformational theories have had a prominent role in this advance. It is important, however, to keep in mind that, to a great extent, this advance has been the result of a deliberate exclusion from the theoretical body of many considerations that, biologically bounded, would have otherwise impeded the degree of formal description that has been required....It is reasonable to expect, therefore, that many of these rules have no direct psychobiological reality and no predictive value for neuronal processing of the same information. [page 901]

[D]emonstration of the psychological reality of the various aspects of the linguistic theory must be recognized as an indispensable prerequisite for the subsequent task of correlating those findings with brain structure and function; it would seem premature, at this stage, to try to plug linguistic theory into the brain. [page 902]

Nor was there a realistic possibility of building up from units of anatomy or physiology:

[I]t is useless to look for physiological units unless we know what, functionally speaking, they are units of. If, for example, we have a system that encodes only the relationships between two stimuli, we would expect it to have very different physiological properties than a system that encodes the stimuli discretely and absolutely. [page 903]

Insightful analyses of the levels of analysis problem would appear soon thereafter. After David Marr famously argued that the brain is a complex information-processing system that

defies understanding at any single level of description (Marr, 1982), neuropsychologists applied this formation to language studies, articulating 4 levels of analysis for brain-language theorizing (Caplan, 1982; Marshall, 1980):

1. A theory of grammar, characterizing what brain mechanisms for language accomplish
2. An account of how language representations are generated and mapped during speaking and listening
3. A taxonomy of the brain's functional mechanisms, ie, its computational building blocks
4. An account of how those functional mechanisms are realized in the information-bearing units of the nervous system, ie, cells, circuits, and networks

The Marin lab accepted this multiple-levels view as basic to the neurobiology of language. In asserting that “the study of the language-brain relationship must begin with language, and not with the brain” (Marin, 1976, page 901), Oscar was taking a pragmatic stand against premature reductionism. What was required was psycholinguistic model building at the aforementioned levels 2 and 3, but not in a way that was isolated from the brain. Shaping such models to be responsive to the characteristics of pathological as well as normal language would help ensure their ultimate biological reality.

SCOPE OF A NEUROBIOLOGY OF LANGUAGE

The simplicity and elegance of the Broca-Wernicke-Geschwind model captured the public imagination, and still does today. Yet it has always attracted criticism for its narrow, associationistic conception of human language. Consistent with his general opposition to reductionistic theorizing, Oscar maintained that it was a fundamental mistake to base a neurobiological theory of human language on anatomical principles. Taking a broader perspective, he wrote (Marin, 1976):

Human language involves complex auditory processes, some of which may be unique, and delicate coordination of the vocal apparatus. Neurology has something to say about the localization of these processes in the brain, although...even this knowledge is scanty and equivocal [references]. But besides these extreme sensory and motor aspects of verbal communication, language also implies a complex set of mental processes: extraction of meaning from words and sentences; recalling verbal symbols from memory; associating verbal symbols with their referents; organizing sentences that convey specific meaning and that follow prescribed syntactic orders and precise phonological rules, and so on....a neurobiology of language that ignores these central processes...is likely to be incomplete. [pages 900–901]

We later fleshed out these ideas in a chapter that traced the evolutionary prerequisites of these central processes, along with the structural and neural adaptations that allowed symbolic speech to develop in the auditory-vocal channel (Marin, Schwartz and Saffran, 1979). In the ensuing years, the Marin lab would focus heavily on the central, symbolic processes that the Broca-Wernicke-Geschwind model left out. Between 1975 and 1980, the

year when Eleanor's and my paths diverged from Oscar's, the 3 of us co-authored what would come to be regarded as seminal papers on semantic dementia (Schwartz, Marin, and Saffran, 1979; Schwartz, Saffran, and Marin, 1980a), semantic mechanisms in reading (Saffran, Bogyo, Schwartz, and Marin, 1980; Saffran, Schwartz, and Marin, 1976), syntactic deficits in production and comprehension (Saffran, Schwartz, and Marin, 1980a; Schwartz, Saffran, and Marin, 1980b), and the dissociation of syntactic and lexical processes in aphasia (Marin, Saffran, and Schwartz, 1976; Saffran, Schwartz, and Marin, 1980b).

I have noted that in these formative years of cognitive neuropsychology, the priority for many of us was to develop an account of the functional architecture of cognition that was responsive to the data from patients, and, specifically, data based on double dissociations in selected patients. I have also noted that, for many of us, this research strategy developed from an awareness of the conceptual and technical limitations of the science of the day.

Fast forward to the present day and the unimagined advances in in vivo neuroimaging, computational models simulating effects of lesions, and institutional infrastructures supporting large-scale patient recruitment, data analysis, and data sharing. The cognitive neuropsychology of today is far more eclectic in its methods. Single-subject investigations are still valued, but model-driven investigations are equally likely to analyze data from carefully selected groups or large series of individual cases (for discussion, see Schwartz and Dell, 2010, and accompanying commentary).

Case series methods have provided useful data for computational models of neuropsychological symptoms and syndromes (Dell, Schwartz, and Martin, 1997; Rogers, Lambon Ralph, Garrard, Bozeat, McClelland, Hodges, and Patterson, 2004; Schwartz, Dell, Martin, Gahl, and Sobel, 2006) and for the large-scale statistical analysis of lesion-symptom relations (Bates, Wilson, Saygin, Dick, Sereno, Knight, and Dronkers, 2003; Butler, Lambon Ralph, and Woollams, 2014; Dronkers, Wilkins, Van Valin, Redfern, and Jaeger, 2004; Mirman, Chen, Zhang, Wang, Faseyitan, Coslett, and Schwartz, 2015; Rudrauf, Mehta, Bruss, Tranel, Damasio, and Grabowski, 2008; Schwartz, Kimberg, Walker, Faseyitan, Brecher, Dell, and Coslett, 2009). Today's neurobiological theories of language benefit from all these advances, and more (Dell, Schwartz, Nozari, Faseyitan, and Coslett, 2013; Hickok and Poeppel, 2004; Ueno, Saito, Rogers, and Lambon Ralph, 2011). Oscar would be pleased, I think, by how far the field has come.

I came of age, professionally speaking, in the Marin lab. Oscar's brilliant, exuberant mentorship inspired a passion for clinical phenomena that has never left me. After a brief spell in academia, I moved to a neurorehabilitation setting to help found an institute devoted to theoretically based clinical research with patients. Hardly a day passes that I don't reflect with gratitude on those exciting, heady years in Baltimore.

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