

Book Review

Tonkonogy, Joseph. *Vascular Aphasia*. Cambridge, MA: MIT Press, 1986.

Reviewed by *Randi C. Martin, Rice University, Houston, TX.*

Researchers interested in cognition are likely to have at least some passing acquaintance with the issues in aphasia. At some point in their education they may have learned about the Wernicke theory of language and the brain, in which the motor representations for words are presumed to be localized in Broca's area and the auditory sensory representations for words are presumed to be localized in Wernicke's area. Those whose interests focus on language may also be familiar with recent approaches to aphasia that attempt to establish deficits in more central cognitive aspects of language such as syntax, semantics, or phonology rather than restricting attention to articulation and perception.

In many respects, the book *Vascular Aphasia* by Joseph Tonkonogy (1986) is in the tradition of the Wernicke approach rather than the more recent cognitive approach. However, the information he presents points out the many inadequacies of the simplistic Wernicke theory. The Wernicke theory holds that Broca's aphasics have a deficit in speech production due to the disruption of motor speech representations, but preserved comprehension because of the sparing of sensory speech representations. The reverse is assumed to be true for Wernicke's aphasics, that is, comprehension is impaired, but production is spared. Tonkonogy presents the clinical syndromes in sufficient detail to make it clear that patients classed as either Broca's or Wernicke's aphasics have both production and comprehension deficits, but that the character of these deficits may differ for the two groups. Several other syndromes are also discussed and the departure of these syndromes from the predictions of the Wernicke model either in terms of the pattern of symptoms or the brain areas affected is made obvious. To support the claims about localization of function, a wealth of data obtained from postmortem examinations as well as from CAT scans is presented.

The most interesting discussion in the book deals with redundancy of representation in the nervous system and the protection that this provides against small lesions. Tonkonogy presents a compromise between the strict localizationist and holistic or equipotential theories of brain function. He argues for what he terms multipotentiality, meaning that different areas are specialized for different functions, but within a given region there are many redundant, parallel channels that carry out the same function. Under this system, destruction of a part of the region does not lead to total failure of the system. He suggests that another means of protecting against system failure might derive from different regions carrying out a function in different ways by responding to different "alphabets" or units of the input. He

suggests as an example that there might be a whole word recognition system that can be used to recognize a small set of common words in addition to a system that recognizes words by first identifying their constituent phonemes. If the phoneme recognition system is damaged, the whole word system could be used to recognize much of everyday conversation.

Much of the localization data he presents supports the contention that small lesions confined to Broca's and Wernicke's areas result in only transient aphasias whereas large lesions including these areas and certain surrounding areas must be sustained to observe the aphasic syndromes that are traditionally called Broca's and Wernicke's aphasia. These data and Tonkonogy's discussion of them are consistent with the parallel-distributed processing models currently being proposed to model brain function. These systems exhibit what is called "graceful degradation," meaning that a breakdown in a small part of the parallel network does not result in complete malfunction but rather a small reduction in performance (Rumelhart & McClelland, 1986). As is apparently true of brain representation of language function, widespread damage to the network must occur for the system to fail completely.

Although the book is of interest because of the detail in the description of the clinical symptoms and the lesion data, a major shortcoming of the book is the absence of a theory of language-brain relationships. The Wernicke theory has survived to the present day despite its poor fit to some of the data because it could elegantly explain the existence of different clinical syndromes rather than simply describe them. In Tonkonogy's approach, syndromes are identified by commonly occurring patterns of symptoms and then these syndromes are correlated with lesion data. The approach is purely descriptive. No attempt is made to specify the mechanisms by which a lesion in a particular area gives rise to the pattern of symptoms.

If one were to take the simple step of assuming that cognitive functions closely associated with the symptoms characterizing a particular syndrome are localized in the damaged areas, numerous problems would arise. For example, the most striking deficit in the syndrome "anomic-sensory" aphasia is the difficulty the patient has in naming objects and pictures. If one were to assume that the ability to name were localized in the posterior temporal regions typically involved in producing this syndrome, one would have to explain why naming is affected in almost all aphasic syndromes. Of course, it is possible that different aspects of naming are affected in different syndromes. For example, the semantic, phonological, and articulatory representations involved in naming might be differentially affected in different syndromes. However, Tonkonogy gives no clue as to whether this might be the case. Also, one might question whether there are other associated symptoms that derive from the same underlying deficit that gives rise to the naming deficit. For example, if the naming deficit derives from a semantic or phono-

logical disruption, one might expect to see difficulties in comprehending names as well as producing them. If the naming difficulty is purely articulatory, comprehension might be preserved. No discussion is directed to the possible linkages that might exist between symptoms because they derive from disruption of the same cognitive mechanism.

Given the usual complexity of the symptom patterns associated with various syndromes, it seems highly unlikely that only a single cognitive mechanism has been disrupted in any syndrome. The fact that certain complexes of syndromes typically arise probably has more to do with patterns of vascularization than with the fact that the symptoms all derive from the disruption of a single cognitive mechanism. That is, given that a particular artery is occluded, it is likely that certain brain areas will be affected. However, these brain areas may subserve many different cognitive functions. It may be difficult if not impossible to discern whether particular regions within the damaged area are specialized for particular functions by examining patients with typical symptom complexes. Rare cases with striking deficits in only one or a few functions would be of more use in determining localization.

The issues raised here with regard to the theoretical inadequacies of the syndrome approach to aphasia are not new, and have been discussed elsewhere by researchers taking a cognitive approach to the study of brain-damaged individuals (Caramazza, 1984). Recently, cognitive psychologists have become interested in using data from brain-damaged individuals as a means of investigating theories of normal cognitive function (e.g., McCloskey, Sokol, & Goodman, 1986). Such research uses the pattern of impaired performance to draw inferences about what cognitive mechanisms have been impaired and which have been spared. These data can be used to establish which cognitive functions are dissociable, or, under certain conditions, establish which cognitive functions are necessarily associated.

Researchers taking this approach have not usually been concerned with identifying the neural substrate underlying particular cognitive functions. Information about the locus of damage may not even be reported. In fact, some researchers have declared the irrelevance of lesion data to their enterprise. Certainly, it is possible to learn about the nature of cognitive deficits in brain-damaged individuals without being concerned about brain-behavior relationships. However, in principle there is no reason why such data about cognitive deficits could not be combined with data on brain damage in order to determine if localization of cognitive function can be established.

The correlational approach undoubtedly serves some useful clinical purposes. In the acute stage of a stroke, for example, a certain pattern of symptoms may indicate a particular medical treatment, or at least guide the clinician to look for lesions in particular brain areas. Also, information may be available about the likely course of recovery for different commonly

occurring syndromes allowing the physician to make a prognosis. Although these and other important practical considerations may justify the need for a syndrome approach, the approach seems unlikely to lead to a theory of language-brain relationships. If readers are looking for such a theory, they will be unable to find it in *Vascular Aphasia*.

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