

Back to the Future: Reclaiming Aphasia from Cognitive Neurolinguistics

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The study of language organization and the brain has in many ways suffered from an embarrassment of riches. Since the early 1960s when the first applications of experimental psychological methods and theory started to appear in studies of aphasic language (e.g., Goodglass & Gleason, 1960), there have been literally thousands of papers published based on the perspectives of cognitive psychology, linguistics, and clinical science. While the methods and logic of experimental psychology were originally adopted to help explain the phenomenology of aphasia, the 1990s brought a radical shift in the relationship between experimental psychology and aphasia; aphasiological phenomena have become part of the “data” to test linguistic or cognitive theory. The result of this paradigmatic shift has been that much of the clinical and biological context of aphasic symptoms has become an epistemological orphan, left behind by the natural focus of normal psychological theory. Though this narrowing of focus and use of modern experimental meth-

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ods have helped our field develop more formal and explicit theories of human language, we will ultimately have to pay the piper and account for the realities of describing aphasic deficits and how they are caused.

One task for the start of the next millennium of aphasia research will be the reclamation of the clinical details of aphasia as a focus of aphasia research. In 1981, we proposed that the then emerging concept of "automatic vs controlled" processing could be used to understand the distinction between Broca's and Wernicke's aphasia (Milberg & Blumstein, 1981). Although over the years we employed this simple theoretical cognitive dichotomy to generate a number of studies extending our original observations (e.g., Milberg, Blumstein, & Dworetzky, 1987), we were always disquieted by the fact that we could not satisfactorily relate the pattern of experimental data to the actual deficits shown by the patients.

We have recently begun work with an extension of our original paradigm that we feel may be a step in the direction of clarifying the relationship between patient's performance on implicit measures of semantic and syntactic knowledge and the clinical characteristics of aphasic language that prompted our interest in the first place. In most priming experiments conducted on patients with aphasia, semantic relationships are explored using pairs of words. These experiments do not reflect the fact that in actual language processing the interpretation of any given word is *ultimately* nuanced by the sentential and extralinguistic context in which the word is produced, as well as the listener's and speaker's expectancies or intentions. Representational schemes for describing lexical/semantic information processing should include mechanisms that allow for the combination or summation of these multiple factors. Summation of activation, both temporal and spatial, is the rule in biological neural networks. A number of models of lexical access use the constructs of a network structure and spreading activation to account for associative and frequency effects in a variety of language tasks (Masson, 1995). In most models those lexical entries that are closely related or share semantic features are stored "more closely together" than those that are not closely related. Summation of activation is implicit in most descriptions of these spreading activation based network models of semantic memory. However, the possibility that summation-like phenomena may occur in priming has only recently begun to be explored in normal adults (Balota & Paul, 1996) and was examined for the first time in aphasic patients in our laboratory (Milberg, Sullivan, & Blumstein, 1998).

Consider the puzzle of the apparent dissociation between linguistic or metalinguistic and on-line semantic facilitation tasks in patients with Wernicke's and Broca's aphasia. Wernicke's aphasics perform poorly on tasks requiring explicit access to semantic information, yet show preserved semantic facilitation when presented with word pairs (Milberg & Blumstein, 1981). The ability to summate contextual, lexical, syntactic, and semantic information may be critical to the performance of semantic judgment tasks and for

lexical access processes supporting semantic production and comprehension (Milberg, Blumstein, Katz, & Gershberg, 1995). Preserved semantic priming may reflect preserved internodal activation (and hence the organization of some part of the linguistic database servicing language performance), while impaired metalinguistic performance and at least some aspects of the clinical symptomatology of Wernicke's patients may reflect deficiencies in the capacity to summate that activation. The "lesion" of Wernicke's aphasia may reflect changes in the decay rate of internodal activation (see also Milberg, Blumstein, Katz, & Gershberg, 1995) or changes in the capacitance or thresholds of the summing processes necessary to access lexical semantic knowledge representations. Maximum summation may even be decreased by factors increasing the correlation between normally independent sources of activation. This would be akin to the phenomenon of leakage or cross-talk in electronic circuits. For example, an increase in gain or the overall level of network activation may under some circumstances increase the likelihood of cross-talk or leakage between inputs that are normally independent.

Broca's patients, in contrast, appear to show less consistent performance on pairwise semantic, syntactic, and even phonologically based priming tasks (Milberg & Blumstein, 1981). These deficits appear to be empirically at least, better correlates of these patients' clinical deficits involving syntactic operations and production. Yet Broca's patients can reliably gain explicit access to the very data they cannot seem to use on-line (Blumstein, Milberg, Dworetzky, Rosen, & Gershberg, 1991; Milberg, Verfaellie, & Bolton, 1993). Broca's patients may suffer from changes in internodal activation but still be able to use informationally redundant contexts, outside the temporal framework of normal production. Hence they may be deficient on tasks requiring nearly reflexive rapid access to linguistic information, a deficiency reflected in their performance on priming tasks, but they show near normal performance when they have ample time to access contextually constrained information, a process more likely dependent on summation of activation.

REFERENCES

- Balota, D. A., & Paul, S. T. 1996. Summation of activation: Evidence from multiple primes that converge and diverge within semantic memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, **22**, 827-845.
- Blumstein, S., Milberg, W. P., Dworetzky, B., Rosen, A., & Gershberg, F. 1991. Syntactic priming effects in aphasia: An investigation of local syntactic dependencies. *Brain and Language*, **40**, 393-421.
- Goodglass, H., & Gleason, J. B. 1960. Agrammatism and inflection morphology in English. *Journal of Speech and Hearing Research*, **3**, 257-267.
- Masson, M. E. J. 1995. A distributed memory model of semantic priming. *Journal of Experimental Psychology*, **21**, 3-23.
- Milberg, W. P., & Blumstein, S. 1981. Lexical decision and aphasia: Evidence for semantic processing. *Brain and Language*, **14**, 371-385.

- Milberg, W. P., Blumstein, S. E., & Dworetzky, B. 1987. Processing of lexical ambiguities in aphasia. *Brain and Language*, **31**, 138–150.
- Milberg, W. P., Blumstein, S. E., Katz, D., & Gershberg, F. 1995. Semantic facilitation in aphasia: Effects of time and expectancy. *Cognitive Neuroscience*, **7**, 33–50.
- Milberg, W. P., Sullivan, K. L., & Blumstein, S. E. 1998. Summation of semantic priming effects in aphasia: Deficits in the integration of activation are related to disorders of language. *Brain and Language*, **65**, 76–78.
- Milberg, W. P., Verfaellie, M., & Bolton, E. 1993. *Summation of semantic priming across weak associates: The whole is greater than the parts*. Paper presented at the Psychonomic Society, Tucson, AZ.