

# Testing predictions of the interactive activation model in recovery from aphasia after treatment

Regina Jokel,\* Elizabeth Rochon, and Carol Leonard

*Graduate Department of Speech-Language Pathology, University of Toronto, 500 University Avenue, Room 1034, Toronto, Ont., Canada M5G 1V7*

Accepted 12 February 2004

## Abstract

This paper presents preliminary results of pre- and post-treatment error analysis from an aphasic patient with anomia. The Interactive Activation (IA) model of word production (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997) is utilized to make predictions about the anticipated changes on a picture naming task and to explain emerging patterns. Error patterns are viewed in light of two putative mechanisms within the IA network, connection strength, and rate of decay. The results suggest that while these mechanisms can successfully account for the magnitude and order of specific errors (e.g., predominance of semantic paraphasias and nonwords followed by formal paraphasias under the conditions of weak connections), the treatment may have influenced the occurrence/absence of some error types.

© 2004 Elsevier Inc. All rights reserved.

## 1. Introduction

Anomia, a difficulty in retrieving words, is one of the most common symptoms in patients with brain damage due to a stroke. Numerous theoretical explanations have been proposed to account for different patterns of anomia errors. One of the most viable accounts is the Interactive Activation model (Dell, Schwartz, Martin, Saffran & Gagnon, 1997), which postulates that the lexical retrieval proceeds in two steps, semantic, and phonological. Activation in the retrieval network cascades in a top-down fashion, while connections are bidirectional and excitatory, allowing for both feedforward and feedback between levels of processing. The degree of activation in the network is a sum of both feedback and feedforward activation that changes over time, and it is relative to the semantic and phonological closeness to the target. The role of feedback within the IA network is to stabilize the pattern of activation over the target nodes and assure that the selected entry is a real word ('lexical bias').

The model has been used to assist in making predictions about normal word access and to provide insights

into the hypothesized mechanisms linked to errors in word selection, which are seen in aphasic speakers (Dell et al., 1997). Two such mechanisms have been suggested; the strength of connections between levels of processing and the decay of the signal traveling between the levels.

Schwartz, Saffran, Bloch, and Dell (1994) utilized the IA model in their study to demonstrate the consequences of the weak connection mechanism in their fluent aphasic patient, FL, who showed high rates of nonwords in spontaneous speech. Under the principles of the IA account, weakening of connection strength reduces the amount of activation that spreads between levels of processing, leading in turn to a decreased amount of feedback. When the system is deprived of feedback, the lexical bias will be replaced by nonwords. An overproduction of nonwords in aphasia was therefore linked to weak connections.

Accelerated decay, on the other hand, was hypothesized to cause semantic errors in single word repetition, an inability to repeat non-words, and the occurrence of formal (phonemic) paraphasias in naming (Martin, Dell, Saffran, & Schwartz, 1994). Using the IA model, (Martin et al., 1994) were able to characterize the deficits of NC, a patient with deep dysphasia. Deep dysphasia is a disorder defined by the inability to repeat non-words, formal paraphasias in naming and spontaneous speech,

\* Corresponding author.

E-mail address: [rjokel@baycrest.org](mailto:rjokel@baycrest.org) (R. Jokel).

and semantic errors in repetition. When the signal within the network decays rapidly, there is not enough activation to complete the feedback loop, therefore selection of a lexical target will be more influenced by the most recently activated level. As both spontaneous speech and picture naming tasks evoke the phonological nodes last, a phonological influence will exert the greatest power on final production. Subsequently, the selected item will most likely be phonologically rather than semantically related to the target. Similarly, when a lexical item is to be repeated, the semantic nodes will be activated last. As the initial activation forwarded from the phonological to the semantic nodes may provide an insufficient boost to select a single lemma, an activated semantic relative may be selected instead. Under conditions of rapid decay, subject to decay severity, the system will not receive confirmatory feedback as to the accuracy of the semantic competitor. This mechanism is thought to cause an inability to repeat nonwords.

In addition, Dell and his colleagues (Dell et al., 1997; Gagnon, Schwartz, Martin, Dell, & Saffran, 1997) showed that simple quantitative changes applied to a normal processing model could account for a variety of naming errors. By modifying a single parameter (i.e., either connection strength or decay rate), they were able to simulate a wide range of errors made by aphasic patients.

This paper presents preliminary error analysis from an aphasic patient who was administered a picture naming task before and after treatment for anomia. To date, the IA model was utilized to examine error patterns at different time points in spontaneous recovery. We wished to examine the potential influence of treatment effects on error pattern. Data presented here is extracted from one of nine patients participating in our project.

## 2. Method

Data presented comes from MB, a 71-year-old male 12 years after stroke, who was randomly assigned to receive a phonologically based treatment for anomia. (Patients in this project received one of three treatment options; phonological, semantic features, and mixed approach.) A set of 175 line drawings of common objects (Philadelphia Naming Test, Roach, Schwartz, Martin, Grenwal, & Brecher, 1996) was presented for naming. MB was asked to provide single word responses (whenever possible). All responses were transcribed, coded, and scored by two independent scorers using the International Phonetic Association rules. The overall inter-judge reliability score for MB was 92.1%. Errors receiving separate coding categories consisted of: (a) formal paraphasias (e.g., *mat* for *cat*), (b) semantic paraphasias, (e.g., *dog* for *cat*), (c) mixed errors (e.g., *rat*

for *cat*), (d) nonwords (e.g., *strek* for *cat*), and (e) unrelated words (e.g., *bench* for *cat*).

## 3. Results

Fig. 1 illustrates the pattern of MB's errors on a pre- and post-treatment picture naming task. Although the results indicate post-treatment improvements (27 errors pre- versus 20 post-treatment), factors other than the overall severity were analyzed in this project.

According to the IA model, it is the order and magnitude of error types that lead to the identification of each of the two postulated mechanisms underlying a naming impairment. Despite the fact that MB's pattern shows no unrelated errors, the order and magnitude of error types appear to be most consistent with reduced connection strength both pre- and post-treatment (see Fig. 1). An intriguing finding post-treatment was the emergence of mixed errors. The IA model applied to recovery without treatment does not predict the occurrence of error types not seen on initial evaluation. Therefore, the emergence of a new error type after treatment may be suggestive of therapeutic influences. The provision of phonologically based treatment in a patient with the predominance of semantic errors at the outset of the experiment, may have lead to the appearance of errors that carry both semantic and phonological resemblance to the target (hence mixed errors in MB's post-treatment profile).

## 4. Conclusions

Findings from this investigation suggest that while the IA model can be successfully utilized in making predictions regarding recovery, the treatment that patients receive may alter the anticipated pattern. Data from a larger sample are currently being analyzed to provide more compelling support for this conclusion. Our findings will also provide directions for explorations in the area of word production that are currently under

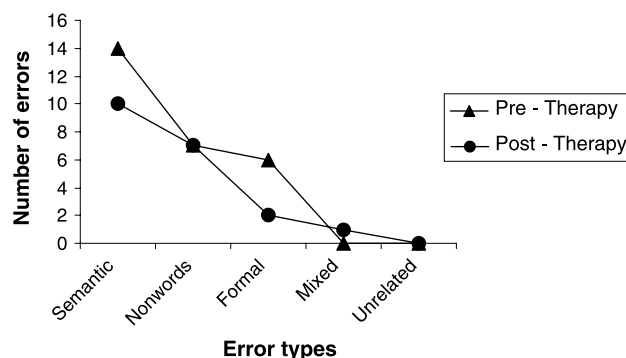


Fig. 1. Naming profile of patient MB pre- and post-treatment.

way, i.e., comparisons of error patterns seen in recovery from naming impairments due to a stroke with those of naming decline seen in primary progressive aphasia, a neurodegenerative disorder.

## References

- Dell, G. S., Schwartz, M. F., Martin, N., Saffran, E. M., & Gagnon, D. A. (1997). Lexical access in aphasic and nonaphasic speakers. *Psychological Review*, *104*, 801–838.
- Gagnon, D. A., Schwartz, M. F., Martin, N., Dell, G. S., & Saffran, E. M. (1997). The origins of formal paraphasias in aphasics' picture naming. *Brain and Language*, *59*, 450–472.
- Martin, N., Dell, G. S., Saffran, E., & Schwartz, M. (1994). Origins of paraphasia in deep dysphasia: Testing the consequences of a decay impairment to an interactive spreading activation model of lexical retrieval. *Brain and Language*, *47*, 609–660.
- Roach, A., Schwartz, M. F., Martin, N., Grenwal, R. S., & Brecher, A. (1996). The Philadelphia naming test. *Clinical Aphasiology*, *24*, 121–133.
- Schwartz, M. F., Saffran, E. M., Bloch, D. E., & Dell, G. S. (1994). Disordered speech production in aphasic and normal speakers. *Brain and Language*, *47*, 52–88.