Simulating Bilingual Aphasia Rehabilitation: Evidence from a computational model

Current research on bilingual aphasia has only begun to inform us about the optimal rehabilitation for bilingual aphasic patients (Edmonds & Kiran, 2006; Roberts & Kiran, 2007) but the literature is still sparse in terms of interpreting the nature of naming impairments in bilingual aphasia. While the influence of theoretical models in guiding assumptions about semantic representation and lexical access in normal bilingual language has generated fairly compelling evidence, the extension of these models to account for language impairment subsequent to brain damage have been less studied due to our limited understanding with respect to the effect of stroke on bilingual aphasia. A potentially innovative solution is to use computational modeling to complement our understanding of how language impairment and recovery occurs in a bilingual individual with aphasia. Over the last twenty years, connectionist and dynamical systems approaches have made remarkable contributions to our understanding of the mechanisms and impairment of language and cognition (Baron, Hanley, Dell, & Kay, 2008; Gary S. Dell, Martin, Saffran, Schwartz, & Gagnon, 2000; G. S. Dell, Schwartz, Martin, Saffran, & Gagnon, 2000; Foygel & Dell, 2000; Plaut, 1996; Schwartz & Brecher, 2000; Welbourne & Lambon Ralph, 2005). For the purpose of understanding bilingual language recovery, they have the distinct advantage incorporating an unbiased and pre-stroke estimation of factors such as AoA and language proficiency on bilingual language impairment and recovery which are assessed in a qualitative and post-morbid fashion and is, at best, a subjective estimate of the pre-stroke language background for each individual. The goal of the present study is to build a model that can simulate language impairments and rehabilitation in patients with bilingual aphasia. Specifically, the model takes into account Age of Acquisition (AoA) and pre-stroke language use in the two languages. This model is subsequently lesioned at specific sites by varying connection strengths between the semantic and phonological networks and then rehabilitated in one language (e.g., either English or Spanish) and the extent of cross language transfer is examined.

Methods
The conceptual architecture of the computational model (DISLEX) described in this paper is based on the model proposed by Kroll and Stewart (1994) because it can account for both AoA and varying levels of proficiency through the differential connection strengths. Three self-organizing maps (semantic, L1 and L2) each with 30x40 neurons were trained simultaneously with the associative connections between each pair of maps that enable network activation to flow between maps, allowing the model to translate between alternative semantic and phonetic representations of a word. All three maps used Gaussian neighborhood functions whose width decreased exponentially (from sigma = 7.0 down to 0.2) over the course of training.

As training progressed, the neighborhood size of each map was slowly decreased, which enabled the maps to develop global structure early and then refine that structure and learn to represent local similarities later in training. This process of gradual focusing allowed the model to account for second language acquisition at different ages: for early AoA, training of both maps started approximately at the same time; for late second

language AoA, only L1 was trained at first, and training of L2 began later. Besides the relative ages of acquisition for L1 and L2, the revised hierarchical model (Kroll & Stewart, 1994) assumes that differences in proficiency between L1 and L2 are the result of relative language dominance, which in turn results from differences in the amounts of exposure to each language. To simulate proficiency, the model was presented with more semantic-phonetic pairs in one language than the other at different times during training; consequently, the phonetic map of one language, as well as its associative connections to other maps, was less developed, resulting in reduced proficiency in that language.

In order to model naming deficits, the lesion was applied to the connections from the semantic map to the phonetic maps. Damage was applied by adding Gaussian noise with $\mu = 0$ to all these connections. The amount of damage (the "lesion strength") in each case was adjusted by changing the σ of the noise between 0 and 1.0 in steps of 0.01. The range of lesion strength was chosen so that the performance dropped to zero for all patients at the maximum level. Then, individual models of premorbid patient performance were used to investigate how damage to the model's lexicon matched actual bilingual aphasia patient naming patterns. Finally, to model rehabilitation, the starting point was set to either a severe impairment in naming (20% or less accuracy) or mild impairment (70% or high naming accuracy). Then the model was retrained using the parameters described above in one of the two languages until performance reached 90% in the trained language.

Results
In order to match the model's performance in both English and Spanish to that of a group of individual normal bilingual human speakers (N = 39) with varying AoA and relative proficiency, the training parameters were set up to match the known ages of acquisition and exposure data as closely as possible for each test case. The model was the extended to simulate a group of bilingual aphasia patients (N = 19), by attempting to replicate the patients' self-reported AoA and pre-stroke performance. For most patients (13/19), the model is able to match the patient data well. In some cases the model either underestimates or over estimates language proficiency reflecting inconsistencies in patient reports of pre-stroke exposure and performance.

Individual models of premorbid patient performance were then used to investigate how damage to the lexicon can lead to the symptoms of bilingual naming impairment. For 16 patients, naming performance in both English and Spanish declined gradually as a function of lesion strength, and three subgroups of patients emerge (see Figure 1). In one subset of patients, different pre-stroke proficiencies, but the same level of noise explains the nature of naming impairment in patients. Therefore, although the damage is symmetric, the result is an asymmetric impairment in the two languages in these patients. In another subset of patients, patients had the same pre-stroke proficiencies but different levels of noise were required to match the naming impairment in the two languages. In this case, the damage is asymmetric, but relative to the starting points (pre-stroke proficiency) the impairment is not always asymmetric in the two languages. Finally, in a third subset of patients, similar pre-stroke proficiencies were associated with similar levels of noise in the two languages. Therefore, the damage is symmetric and this results in a symmetric impairment.

Simulation of rehabilitation component of the project is ongoing. Thus far, we have systematically varied extent of damage (severe impairment vs mild impairment), pre-stroke exposure (high exposure vs low exposure) and language (rehabilitation in English or Spanish) and examined the amount of cross language transfer. As one example (see Figure 2), in a hypothetical "patient" with high Spanish exposure and low English exposure prestroke that are equally severely impaired post stroke, training in English results in cross language transfer to Spanish whereas training Spanish also results in improvements in Spanish (the trained language) and English (untrained language). These preliminary results are consistent with actual patient data reported previously (Edmonds & Kiran, 2006). Additional results and the implications of these rehabilitation results will be discussed.

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Figure 1 shows how the noise lesion at different strengths affects the naming performance of the models of individual patients. The bars on the left side of each plot show the performance of the pre-stroke model and the triangles pointing right indicate the pre-stroke naming performance. Moving to the right in each plot, the lesion strength (the amount of noise) increases. The red and green lines show the resulting naming performance in English and Spanish respectively. The triangles pointing left show the patients' post-stroke naming performance in English and Spanish, i.e. the performance the models need to match in each case.
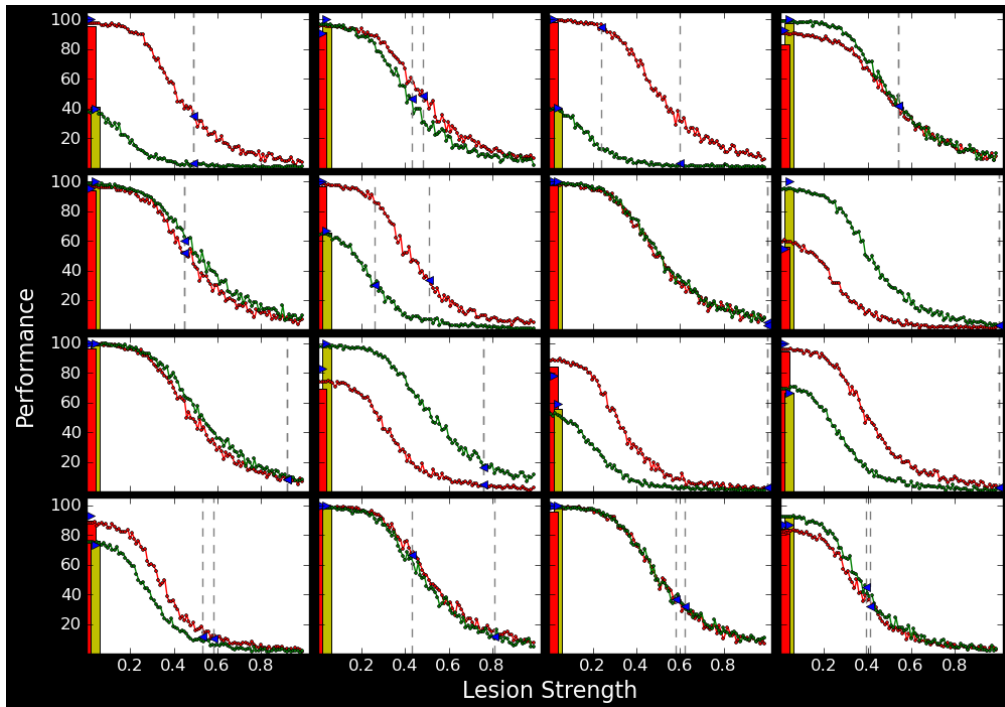
Figure 2 shows rehabilitation results for one hypothetical patient in the model. In this scenario, the model is trained early in Spanish with high exposure and early in English but with low exposure. A hypothetical lesion results in a symmetrically severe (20% or less accuracy) impairment in both languages. When rehabilitation in provided in English, the trained language (English- red line) results in improvements in Spanish (green line). When rehabilitation is provided in Spanish (green line), improvements in English (red line) are also observed. The X axis indicates the number of treatment epochs and the Y axis indicates naming accuracy (in %).