
Platform Session 1: Speech Production

1. Voxel-based Lesion Analysis of Phonological, Lexical, and Syntactic Production Deficits in Post-stroke Aphasia

Faroqi-Shah Y.¹, Kling T.², Solomon J.³, Archibald J.⁴, Park G.⁴, Braun A.⁴

¹ Dept. of Hearing and Speech Sciences, University of Maryland, ² John Hopkins University Hospital, ³ Medical Numerics, ⁴ National Institutes of Health

Individuals with aphasia vary in the extent to which lexical, phonological, and syntactic impairments underlie their language production difficulties. Most aphasic individuals have a combination of these impairments that cannot be always predicted from their aphasia classification. The present study investigated the lesion correlates of lexical, phonological and syntactic production deficits and examined the findings in the context of current understanding of functional roles attributed to these perisylvian regions (Hagoort, 2005).

Methods

Thirty-one pre-morbidly right-handed aphasic individuals with a single left hemisphere ischemic lesion participated in extensive psycholinguistically-motivated language testing. Picture naming, nonword repetition, and sentence elicitation (simple and complex sentences) scores were used as measures of lexical, phonological, and syntactic production respectively. T1 weighted magnetic resonance images (MRI) were obtained using a GE 1.5 Tesla MRI scanner. Spatial normalization, anatomical labeling of brain regions, and statistical operations were conducted using ABLe 2.3 (Solomon, et al., 2007). T-maps illustrating significant voxel-wise relationships between language deficits and the presence or absence of a lesion were generated. Boolean operations were used to create lesion maps unique to (lexical, phonological, syntactic only), and common across various production measures [lexical and phonological and syntactic]; [lexical and phonological not syntactic]; [lexical and syntactic not phonological]; [phonological and syntactic not lexical).

Results and Discussion

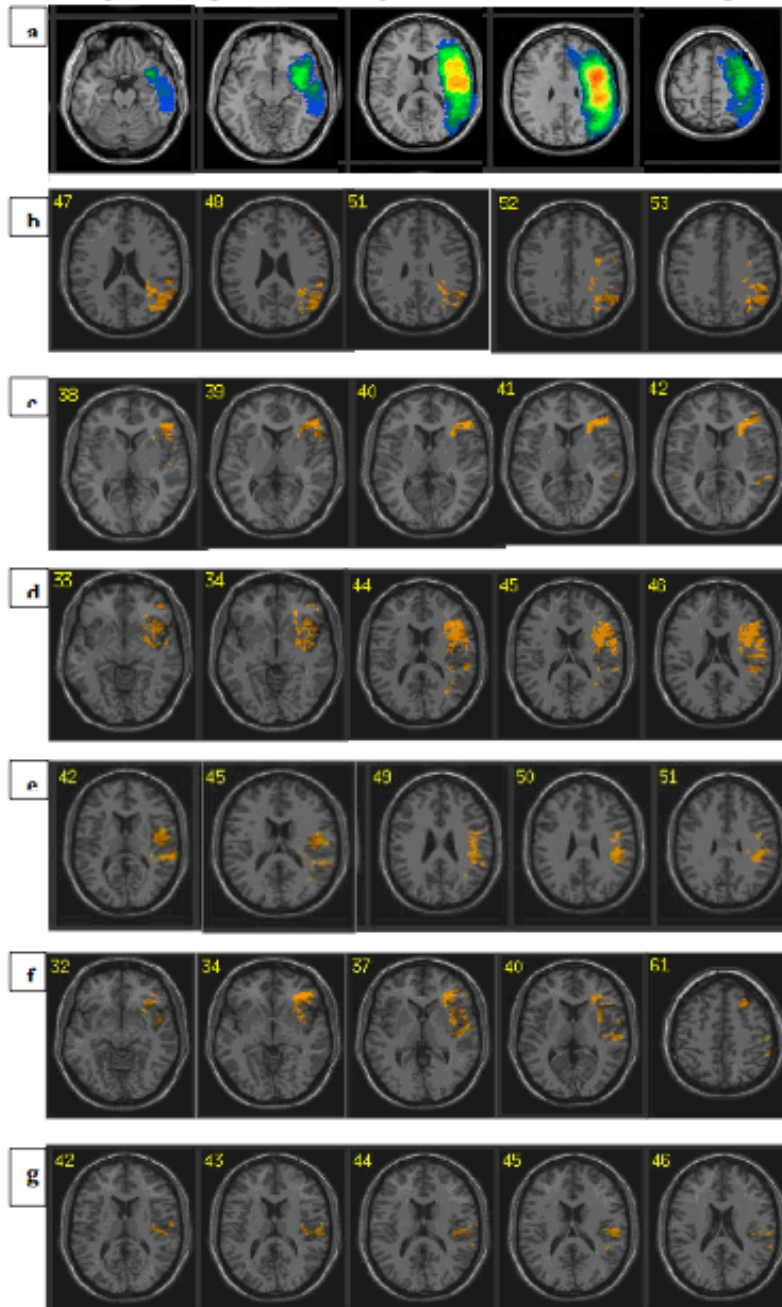
Figure 1 shows the extent of lesions across all participants and representative lesion maps for each comparison. Purely lexical deficits were associated with temporo-parietal lesions (middle temporal gyrus, and posterior planum temporale (PT) extending into the inferior parietal lobule (IPL), and postcentral gyri). Lesions in a restricted portion of the inferior frontal gyrus (IFG, BA 45, 44/6) and rolandic operculum produced phonological deficits. Syntactic deficits were associated with large lesions of the IFG (BA44/45/47), anterior superior temporal gyrus (STG), putamen and anterior insula. Lesions contributing to concurrent syntactic and phonological deficits included IFG, MFG, anterior STG, and anterior insula. Lesions of the superior temporal sulcus, IPL, posterior IFG, and posterior insula resulted in syntactic with lexical deficits. No significant lesions were identified for lexical and phonological deficits combined. Lesions common to all three production deficits included the posterior PT, contiguous insula, and IPL.

Overall, syntactic and phonological deficits were associated with left anterior perisylvian lesions, while lexical deficits were associated with posterior perisylvian lesions. Interestingly, the insula and PT showed a distribution of lesions consistent with this anterior (syntax, phonology)-posterior (lexical) perisylvian trend. The lesion maps are consistent with recent neuroimaging studies of language production, and support the notion that unification operations (syntax and phonology) are mediated by the IFG while retrieval of stored information (lexical-semantic and syntactic rules) is a function of temporo-parietal structures (Hagoort, 2005). However, our lesion data indicate that functional networks for unification-retrieval are distributed more extensively to include the planum temporale, insula and subcortical structures,

References

Solomon, J., Raymont, V., Braun, A., Butman, J., & Grafman, J. (2007). User-friendly software for the analysis of brain lesions (ABLE). *Computer Methods and Programs in Biomedicine*.
Hagoort, P. (2005). On Broca, brain and binding. *Trends in Cognitive Sciences*.

Figure 1. a) Shows the combined lesion of all participants, the colors reflect the number of participants with a lesion in that region: blue=at least 3, green=6, yellow=14, orange=17, red>20), Lesion map of b) only lexical deficits, c) only phonological deficits, d) only syntactic deficits, e) lexical+syntactic deficits combined, f) syntactic+phonological deficits combined, g) all three deficits combined. The slices were selected in figures b) - g) to show the significant lesions and are not contiguous.



Presented by: **Faroqi-Shah, Yasmeen**

2. A Computational Case-Series Approach to Frequency Effects in Aphasic Word Production

Nozari N., Kittredge A., Dell G.

Beckman Institute, University of Illinois at Urbana-Champaign

Introduction

When repeating a word, do we access its lexical representation, or solely its sublexical units? We approached this question by investigating the structural overlap between picture naming and auditory word repetition using Foygel and Dell's (2000) two-step model of lexical access. In this model, naming has two steps: first, meaning is mapped onto a word-form, and second, the word-form is mapped onto its phonemes. For repetition, though, the first step is not necessary. Once the word-form is accessed (through listening), repetition uses the second step of naming (the lexical-route model). Alternatively, the sounds of the word may be directly mapped onto the output phonemes (the nonlexical-route model). A third possibility is to have both routes in place. Such a dual-route system might use both routes on each repetition trial (the summation dual-route model), or only one route on a given trial (the independent dual-route model; figure 1).

These four repetition models differ in their degrees of overlap with the second step of naming. If we can empirically quantify the overlap between naming and repetition, we can pick the correct model. To do this, we investigated the increase in the probability of aphasic subjects' nonword errors in these two tasks as the target's frequency decreases. Frequency has a stronger effect on the second step and nonwords are canonical second-step errors. Therefore, if the frequency effect on nonwords is similarly strong in naming and repetition, the two tasks may share the second step. We tested this hypothesis by comparing aphasic naming and repetition to simulated data from our naming and repetition models.

Methods

59 aphasic patients (from Dell et al., 2007) with minimal input processing deficit were tested on naming and repetition using the Philadelphia Naming Test and the Philadelphia Repetition Test, respectively. Simulations were run using the naming and the four repetition models, with lexical frequency represented in the models' connection weights.

Results

Patients' nonword errors were analyzed using multinomial-hierarchical-logistic-multiple regression, as well as a binomial-crossed-random-effects model. Both naming and repetition showed sizable frequency effects, comparable to one another. The results of the simulations were analyzed using a similar technique. The lexical-route and the summation dual-route models reflected the patients' data pattern best, by showing frequency effects on the probability of producing nonword errors that were at least as large in repetition as they were in naming.

Conclusions

Strong structural overlap between naming and repetition, confirmed by the comparable frequency effects in the two tasks, means that repetition is heavily lexically influenced. Both the lexical-route and the dual-route models simulate the data well and the choice of the model may depend on certain factors, such as accessibility of word meaning.

References

- Dell, G. S., Martin, N., & Schwartz, M. F. (2007). A case-series test of the interactive two-step model of lexical access: Predicting word repetition from picture naming. *Journal of Memory and Language*, 56, 490–520.
- Foygel, D., & Dell, G. S. (2000). Models of impaired lexical access in speech production. *Journal of Memory and Language*, 43, 182–216.

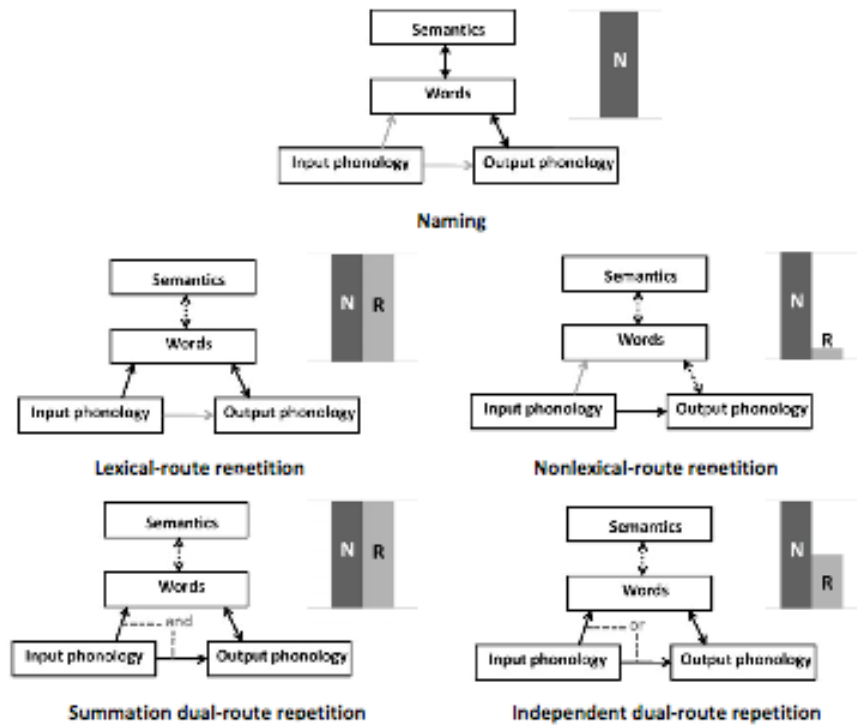


Figure 1- The architecture of the naming and the four repetition models used in the simulations. N= naming; R= repetition; black arrows= active routes; dotted arrows= connections via feedback; gray arrows= inactive routes. The bars next to each model show the predicted pattern of the frequency effect in that model (how much the probability of making a nonword error increases as a function of decreasing target frequency). For repetition models, the predicted effects are graphed next to the predictions of the naming model. Lexical and summation dual-route models predict large frequency effects. The nonlexical-route model predicts a small effect and the independent dual-route model predicts an intermediate effect.

Presented by: **Nozari, Nazbanou**

3. Distinguishing Phonological Errors from Phonetic Errors in Acquired Speech Impairment

Buchwald A. ¹, Rapp B. ², Miozzo M. ³

¹ New York University, ² Johns Hopkins University, ³ Cambridge University and Johns Hopkins University

Distinguishing between errors that arise at phonological or phonetic levels of processing is an important aspect of understanding acquired phonological production impairments. Here we attempt to identify if the errors produced by an individual (DLE) with acquired spoken production impairment originate in phonological processing or in subsequent phonetic and motor-based processes.

Following a left hemisphere infarct affecting the fronto-parietal regions, DLE produces form-based errors in naming and repetition tasks. Interestingly, DLE frequently deletes the initial fricative from fricative-stop (speech → [pitS]) and fricative-nasal (snack → [næk]) consonant clusters. In American English, the [p] in speech is produced without aspiration, thus having a shorter voice onset time (VOT) than the aspirated [p] in peach (and similar to the [b] in beach). Additionally, [n] is significantly longer in knack than in snack. We used these properties of English phonetic

implementation to identify the level of DLE's deficit.

The rationale for the study was as follows. In phonological impairment, we expect the fricative to be deleted prior to generating a phonetic plan, so we predict that the [p] produced in the deletion error (speech → [pitS]) and in the word peach should be similar. In contrast, a 'later' phonetic impairment should yield errors that are consistent with the underlying target cluster, so the unaspirated allophonic variant of [p] should appear in the deletion error speech → [pitS].

To test these predictions DLE was asked to repeat auditorily presented words with fricative-stop (speech) and fricative-nasal (snack) clusters, as well as control words with singleton stops and nasals (peach and beach; knack). In phonologically-based errors, the deleted form for fricative-stops should be more similar in VOT to the voiceless singleton control (peach) than the voiced control (beach), and nasal duration in fricative-nasal targets should be similar to singleton nasals. In contrast, motor-based errors should have appropriate articulatory timings for clusters (i.e., stop VOT closer to voiced controls; nasal duration shorter than singleton nasals).

The results were consistent with a phonological impairment. A VOT analysis revealed that VOT was significantly longer in fricative-stop tokens with deletion errors (speech → [pitS]; 40.9ms) than in responses with voiced control words (beach; 26.3ms; $t(261)=6.30$, $p<.001$). However, deleted tokens trended towards having shorter VOT than the voiceless controls (peach; 45.1ms; $t(309)=1.93$, $p=.051$), indicating a small effect of the underlying target cluster. A second analysis of nasal duration revealed comparable duration for nasals in deletion errors ([n] in snack → [næk]; 50.6ms) and as singletons (knack; 46.9ms; $t(114)=.794$, ns). Taken together, these results indicate that the phonetic characteristics of the deletion errors are consistent with deletion prior to generating a detailed phonetic plan.

These findings are consistent with DLE's/s/-deletion taking place during phonological encoding. We will present other aspects of DLE's production that are also consistent with this conclusion. We will discuss how the data can be accounted for in an interactive speech production framework we will present more general conclusions about factors determining the nature of phonological production errors.

Presented by: **Buchwald, Adam**

4. Syllable Frequency Effect in Progressive Apraxia of Speech: A Case Study

Laganaro M.¹, Bagou O.¹, Croisier M.²

¹ University of Neuchâtel, Switzerland, ² Hôpital neuchâtelois, Switzerland

Introduction

Although apraxia of speech (AoS) has usually been described in degenerative language disorders in association with non-fluent progressive aphasia, at least 20 clinical cases of pure progressive apraxia of speech (PAoS) have been reported in the literature (Didic et al., 1998; Duffy, 2006; Joseph et al. 2006). Impairment in AoS is usually ascribed to the programming of speech gestures (phonetic encoding in the models of speech production). Converging evidence in the literature point to a frequency organization of syllable-sized gestural scores (Cholin, et al., 2006; Laganaro and Alario, 2006) and to an effect of syllable frequency in the programming difficulties accompanying AoS after stroke (Aichert and Ziegler, 2004; Steiger and Ziegler, 2008).

One central issue for both theoretical and diagnostic purposes is whether the same characteristics are observed in AoS after focal lesion and in progressive AoS. Here we present a single case study aimed at analyzing whether syllable frequency affects the progressive disruption of speech production in PAoS.

Method

We present an 18 months follow-up study of a 68-year-old men presenting with PAoS. The patient displayed progressive disruption of speech production, characterized by phonetic errors, intersyllabic pauses, syllable lengthening and dysprosodia, without positive signs of aphasia. Error analyses and word and syllable duration measures were carried out on spontaneous speech and word repetition at first examination and 18 months later. Across the two sessions words and syllables with the same syllabic structure and word position were analyzed.

Results

Error rate increase and speech rate decrease were observed in the comparison of the two assessment periods. More interestingly, syllable duration (1) no longer differentiated according to its position in the word and (2) correlated negatively with syllable frequency in the second assessment only (see Figure 1).

Conclusion

Progressive isolated AoS is affected by syllable frequency during the worsening of speech production. This finding suggests that P AoS progressively displays the same features of AoS after stroke and that its manifestation can be exploited for theoretical and clinical investigations of phonetic encoding.

References

- Aichert, I., & Ziegler, W. (2004). Syllable frequency and syllable structure in apraxia of speech. *Brain and Language*, 88, 148–159.
- Cholin, J., Levelt, W. J. M., & Schiller, N. O. (2006). Effects of syllable frequency in speech production. *Cognition*, 99, 205–235.
- Didic, M., Ceccaldi, M. & Poncet, M. (1998). Progressive Loss of Speech: a Neuropsychological Profile of Premotor Dysfunction. *European Neurology*, 39, 90–96
- Duffy, J.R. (2006). Apraxia of speech in degenerative neurologic disease. *Aphasiology*, 20, 511–527
- Josephs, K.A., Duffy, J.R. et al. (2006). Clinicopathological and imaging correlates of progressive aphasia and apraxia of speech. *Brain*, 129, 1385–1398
- Laganaro, M. & Alario, F.X. (2006). On the locus of syllable frequency effect. *Journal of Memory and Language*, 55, 178–196.
- Staiger, A. & Ziegler, W. (2008). Syllable frequency and syllable structure in the spontaneous speech production of patients with apraxia of speech. *Aphasiology*, 22, 1201–1215.

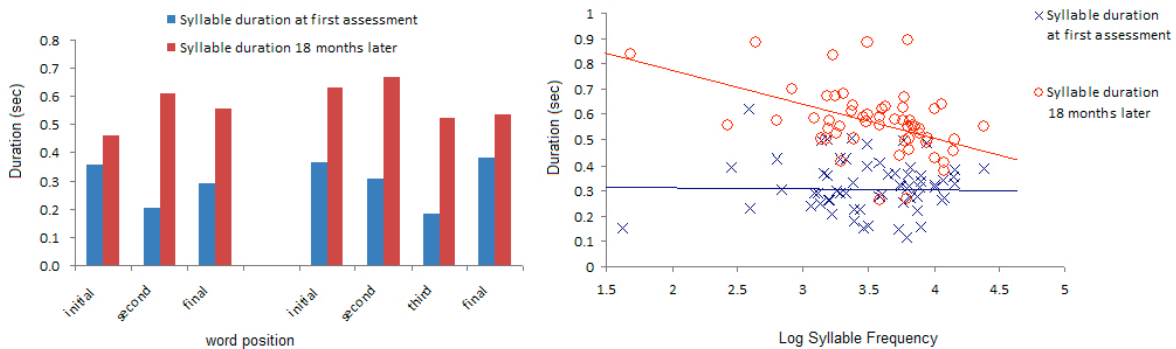


Figure 1. Duration of CV syllables

Presented by: **Laganaro, Marina**