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Neural Systems Underlying the Influence of Sound Shape Properties of the Lexicon on Spoken Word Production: Do fMRI Findings Predict Effects of Lesions in Aphasia?

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Abstract

Previous behavioral work has shown that the phonetic realization of words in spoken word production is influenced by sound shape properties of the lexicon. A recent fMRI study (Peramunage et al., 2011) showed that this influence of lexical structure on phonetic implementation recruited a network of areas that included the supramarginal gyrus (SMG) extending into the posterior superior temporal gyrus (pSTG) and the inferior frontal gyrus (IFG). The current study examined whether lesions in these areas result in a concomitant functional deficit. Ten individuals with aphasia and 8 normal controls read words aloud in which half had a voiced stop consonant minimal pair (e.g. *tame*; *dame*), and the other half did not (e.g. *tooth*; **dooth*). Voice onset time (VOT) analysis of the initial voiceless stop consonant revealed that aphasic participants with lesions including the IFG and/or the SMG behaved as did normals, showing VOT lengthening effects for minimal pair words compared to non-minimal pair words. The failure to show a functional deficit in the production of VOT as a function of the lexical properties of a word with damage in the IFG or SMG suggests that fMRI findings do not always predict effects of lesions on behavioral deficits in aphasia. Nonetheless, the pattern of production errors made by the aphasic participants did reflect properties of the lexicon, supporting the view that the SMG and IFG are part of a lexical network involved in spoken word production.

Introduction

Current models of spoken word production assume there are multiple stages of processing involving the mapping in successive stages from the conceptual/semantic representation of a word on to an abstract sound shape representation and ultimately on to phonetic planning stages needed for articulatory implementation. Discrete models of spoken word production propose that each processing stage generates a single representation on the basis of its input which is then sent to later processing stages (Levelt, Schriefers, Vorberg, Meyer, Pechmann, & Havinga, 1991; Norris, McQueen, & Cutler, 2000). Interactive accounts, on the other

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hand, maintain that there is both bottom-up and top-down interaction between lexical and phonological levels (Dell, Schwartz, Martin, Saffran, & Gangon 1997; Rapp & Goldrick, 2000) such that information at one level of processing can influence activation at other levels of processing. For example, the Dell account holds that a word candidate activates its phonological representation, which then activates a group of phonologically similar sounds through cascading activation. These activated phonological representations feed back to the lexical level, activating a number of lexical competitors. The production of a target word then requires the selection of that word from the set of phonologically competing words.

Consistent with this view, there are a number of studies showing that indeed phonological planning and lexical selection processes influence acoustic-phonetic patterns in the speech output of normal subjects. In particular, competing phonological representations can induce alterations in the acoustic/articulatory output, leaving acoustic traces of the originally intended target (Frisch & Wright, 2002; Goldrick & Blumstein, 2006; Mowrey & MacKay, 1990). Additionally, the number of competing phonological neighbors (lexical density) influences both phonological and articulatory implementation stages of production. Speech errors at the phonological level are more likely to occur in a word from a sparse neighborhood than a dense neighborhood (Vitevitch 1997; 2002), and the production of the vowel space of words is wider in words from dense neighborhoods than in words from sparse neighborhoods (Wright, 2004; Munson & Solomon, 2004).

Further evidence for the influence of lexical/phonological properties of words influencing their acoustic-phonetic output comes from a recent study by Baese-Berk & Goldrick (2009). They showed that voiceless stop-initial words (like ‘teen’) with a voiced minimal pair neighbor (‘dean’) were produced in a reading task with a longer voice onset time (VOT) than words with no voiced stop consonant minimal pair (‘tooth’ *’dooth’). They called this effect lexically conditioned phonetic variation since the properties of the lexicon modulate and hence have a cascading effect on production processes downstream from it. In particular, at the lexical phonological level, a word with a voiced competitor neighbor (‘teen’) requires higher activation to ensure its selection over its competitor (‘dean’) than a word without a voiced competitor (‘tooth’). As a consequence, more ‘extreme’ articulatory realizations are required to override the presence of a phonologically similar lexical competitor. Hereafter, we use the term lexically conditioned phonetic variation to refer to this effect.

Of interest are the neural systems underlying these competitor effects. In a recent fMRI study conducted in our lab, Peramunage, Blumstein, Myers, Goldrick, & Baese-Berk (2011) used the stimuli from the Baese-Berk and Goldrick (2009) study, asking participants to read aloud the stimuli while in an fMRI scanner. Results showed reduced activation for minimal pair (MP) words compared to non-minimal pair (NMP) words in a network of areas that included the supramarginal gyrus (SMG) extending into the posterior superior temporal gyrus (pSTG), the inferior frontal gyrus (IFG), and the precentral gyrus.

That there was reduced activation in the production of words with a minimal pair competitor is consistent with behavioral findings in the literature showing facilitation in the production of words which have many phonologically similar words compared to those that do not. Such facilitation effects have been shown across a number of behavioral paradigms including picture naming (Baus, Costa, & Carreiras, 2008; Vitevitch, 2002; Vitevitch, Armbrüster, & Chu, 2004; but see Jescheniak & Levelt, 1994, for a null result, and Vitevitch & Stamer, 2006, for a reversal of this effect in Spanish speakers), in speech errors (spontaneous: Vitevitch, 1997; experimentally induced: Vitevitch, 2002; Stemberger, 2004; aphasic errors: Nickels & Best, 1996; Gordon, 2002; Kittredge, Dell, Verkuilen, &

Schwartz, 2008; Newman & German, 2005; repetition: Lallini & Miller, 2011) and in analysis of tip-of-the-tongue states (Harley & Bown, 1998; Vitevitch & Sommers, 2003).

fMRI studies have shown similar areas activated in both spoken word production and auditory lexical processing, consistent with the view that these areas are involved in the processes involved in accessing the phonological sound shape of a word and ultimately in its selection from among the set of activated candidate words in the lexicon (Paulesu, Frith, & Frackowiak 1993; Indefrey & Levelt, 2004; Binder & Price, 2001). Indeed studies on lexical processing in auditory recognition have also shown modulation of activation as a function of the phonological similarity of lexical candidates in a similar network to that shown by Peramunage et al. (2011) including the SMG, the pSTG and IFG. These studies have used various paradigms including lexical decision (Prabhakaran, Blumstein, Myers, Hutchinson, Britton, 2006; Okada & Hickok, 2006) and visual world eyetracking (Righi, Blumstein, Mertus, & Worden, 2010). In particular, increased activation has been shown in the posterior superior temporal gyrus and the supramarginal gyrus in a lexical decision task for words that are from a dense neighborhood, and hence have a lot of competitors, compared to words from a sparse neighborhood, and hence have few competitors (Prabhakaran, Blumstein, Myers, Hutchinson, Britton, 2006; Okada & Hickok, 2006). Additionally, using the visual world paradigm coupled with fMRI, Righi et al. (2009) showed increased activation in the SMG and IFG for words that shared phonological onsets (e.g. hammock, hammer) compared to words that did not.

These findings show that the presence of phonological competition not only modulates activation in the pSTG and SMG, areas implicated in phonological processing and lexical access, but also has a modulatory effect on activation in frontal areas and in particular the IFG, an area implicated in selecting among competing semantic alternatives (Thompson-Schill, D'Esposito, Aguirre & Farah 1997; Thompson-Schill, D'Esposito & Kan, 1999). Taken together, they support cascade models of the lexical processing system since they show that neural activation at the lexical level modulates activation in those brain regions involved in phonological processing and lexical access (SMG and pSTG), lexical selection and lexical competition resolution (IFG), and in motor plans for production (IFG and precentral gyrus).

Neuroimaging studies generally assume that neural areas activated in a particular task are associated with a particular cognitive 'function'. However, fMRI show only a correlation between a cognitive function and neural activation patterns. Thus, although functional neuroimaging studies provide a rich source of data about the neural areas activated in speech and lexical processing, they do not provide evidence that these areas play a *necessary and sufficient* role in such processing (cf. Price, Mummery, Moore, Frackowiak, & Friston, 1999; Rorden & Karnath, 2004). Examining the behavioral effects of damage to these areas provides a crucial test of the functional/cognitive role that a neural area may play. In particular, if an area that shows activation in normal individuals using fMRI is in fact necessary and sufficient for a particular cognitive function or task, then damage to that area should result in a behavioral deficit. Indeed, Thompson-Schill and colleagues have shown such a relationship. They used neuropsychological evidence to support their findings from fMRI studies with normals suggesting that the inferior frontal gyrus is recruited in selecting among competing semantic alternatives. They showed that lesions in the inferior frontal gyrus resulted in concomitant behavioral deficits using both naming and verb generation tasks in which subjects were required to select and produce a word from among a set of semantically similar words (Schnur et al., 2009; Thompson-Schill et al., 1998).

Failure to find a deficit despite damage to an area that is implicated in neuroimaging studies with normals is more difficult to interpret. Such findings raise the possibility that the brain

region or regions in question may not be *necessary* for the function/task in question (cf. Price et al., 1999). Alternatively, they could indicate that there is residual functional integrity in peri-lesional structures or functional reorganization involving ipsilateral and/or contralateral homologous neural regions. Nonetheless, if aphasic patients have lesions in neural areas implicated in fMRI findings but no functional impairments, it would challenge the strongest form of the hypothesis that the brain regions in question are functionally *required* for the successful completion of the task.

It is the goal of the current study to investigate whether lesions to those neural areas recruited in spoken word production using fMRI result in behavioral deficits in aphasic patients. In particular, we will examine whether aphasic participants with damage including the SMG and/or IFG show a different pattern of performance from normal participants in the production of voice onset time in initial voiceless stop consonant words that have voiced minimal pair competitors compared to words that do not. We will also examine the potential effects of lesions in the pSTG (Wernicke's area) on lexically conditioned phonetic variation, as this area has been implicated in access of the sound shape of words. Such results not only provide a means of comparing fMRI findings to those from lesion-based behavioral studies, but they also provide a window into spoken word production processes in aphasic patients.

Given that the fMRI findings with normal participants showed modulatory effects in a network involving anterior (IFG) and posterior (SMG extending into the psSTG) regions (Peramunage et al., 2011), we expect an impaired pattern of performance in patients with lesions in these areas. If the SMG is involved in accessing the phonological sound shape of a word from the lexicon, then damage to this area should result in an impairment in the activation of a target word relative to its competitors. Because this information is ultimately cascaded to frontal areas for selection, phonological planning and implementation, there should be a weakening or loss of lexically conditioned phonetic variation, i.e. there should be no difference in VOT between minimal pair and non-minimal pair words beginning with voiceless stop consonants.

A similar pattern of results should emerge for participants with lesions involving the IFG, but for different reasons. The IFG has been implicated in lexical selection processes and the resolution of competition. Thus, although information flow from the SMG should be normal, damage to the IFG should result in a potential loss or weakening of competitor effects. Thus, the encoding of voicing in phonological planning and articulatory implementation stages of processing should be similar for both minimal pair and non-minimal pair words.

Voice onset time analyses examine the pattern of correctly produced word targets. In contrast to normals, aphasic participants typically produce errors in their speech output. An analysis of production errors may provide additional insights into the neural systems underlying spoken word production. It has been hypothesized that the SMG and IFG are part of a lexical network involved in lexical access, selection, and production. Hence, production errors should reflect properties of the lexicon. In particular, there should be a difference in the number of production errors as a function of whether the target word has a voiced minimal pair (a competitor) or not, and errors should more likely result in the production of a word than a nonword.

Methods

Experiment

Participants—Participants included nine older normal controls (five males and four females) and ten subjects with aphasia (seven males and three females). The data from one of the normal female participants were excluded from the study due to technical difficulties

when collecting the stimuli. All participants were native English speakers and all but one patient were right-handed. The normal subjects ranged in age from 57 to 74 years old with a mean age of 66.6 years, and the aphasics ranged from 61 to 82, with a mean age of 71.9. The subjects were paid for their participation in the experiment, which lasted approximately 30 minutes for the older normal controls, and 60 to 90 minutes for the aphasic participants, who took longer to respond and required both longer and more frequent breaks.

Clinical assessment of the aphasic participants was conducted using the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1972) which provided a profile of language abilities and impairments across a range of language functions. Diagnosis was made by review of performance on the BDAE and consensus by a team of researchers after interviews with and evaluation of the patient. Based on this assessment, 5 patients were diagnosed with Broca's aphasia and 5 were diagnosed with Conduction aphasia. Table 1 shows the BDAE percentile scores on a number of the subtests relevant to the current experiment including severity of aphasia, fluency, auditory comprehension, single word reading, repetition, and literal paraphasias (only a few scores were available for B3), and, in addition, the time post onset for each patient.

In addition to clinical diagnosis, extent of lesion analysis was performed on retrospective MRI or CT scans. ROI analyses were conducted to determine the percent of lesion in the IFG and SMG. Additionally, ROI analyses were conducted of other cortical and subcortical structures which have been shown to be implicated in speech production and lexical processing deficits in aphasia (see Table 2) (Goodglass, 1993; Damasio, 1998; Blumstein, 1973; Lecours & Lhermitte 1969; Naeser, Helm-Estabrooks, Haas, Auerbach, & Srinivasan, 1987; Naeser, Palumbo, Helm-Estabrooks, Stiassny-Eder, & Albert 1989). ROIs were defined according to previously published methods by Naeser and colleagues (Naeser, Helm-Estabrooks, Haas, Auerbach, & Srinivasan, 1987; Naeser, Palumbo, Helm-Estabrooks, Stiassny-Eder, & Albert 1989).

OsiriX Imaging Software, an open-source DICOM viewer (<http://www.osirix-viewer.com>) was used to assess percent of lesion within each ROI for subjects for whom digital images were available (B1, B2, C1, C2, C3, C5). ROIs were traced on each slice where they appeared. Lesion within each ROI was then traced on each slice where lesion appeared. Each ROI appeared on two or three slices with the exception of the pars opercularis which was present on only one slice, and the anterior limb of the internal capsule (ALIC) which is rated as the degree to which the lesion crosses the ALIC on the slice where interruption of the ALIC fibers is most extensive. Area of ROI and area of lesion was then totaled across all slices and percent of ROI with lesion calculated. For ROIs that appear on more than one slice, OsiriX calculates the total ROI volume (cm³) and lesion volume (cm³) within the ROI taking into account slice thickness. The ROIs and lesion which were rated on one slice only were calculated as areas (cm²). The percent of lesion within any given ROI refers to the percent of the specific ROI that is lesioned.

Digital scans were not available on all subjects. In this case, extent of lesion was visually assessed, i.e., rated subjectively by eye, for these subjects based on scans on film (B3, B4 and C4). In order to determine accuracy of this method compared to the calculated volumes using OsiriX, visual assessments were also rated on the digital scans blind to the volumes computed using OsiriX. The inter-method reliability was highly significant ($r=0.98$, $p<.0005$), suggesting that subjective visual assessment was an acceptable method for rating extent of lesion within ROIs for subjects without digital scans.

One subject (B5) did not have an available scan on film or in digital format for an analysis of extent of lesion. However, a clinical reading of the original CT scan indicated this subject had a subcortical lesion that did not extend to either the IFG or SMG.

Table 2 shows for each participant the percent area lesioned for all ROIs. Based on this analysis, 3 of the five Broca's aphasic participants had lesions in the IFG and no involvement of the SMG (B1, B2, B3), one of the Broca's aphasic participants had lesions in both the IFG and SMG (B4), and another had a subcortical lesion that did not extend cortically to either the IFG or SMG (B5). Four of the five Conduction aphasic participants had lesions in the SMG and no involvement of the IFG (C1, C2, C3, C4) and one had neither IFG nor SMG damage (C5). Additionally, all of the participants diagnosed with Conduction aphasia had lesions that also included Wernicke's area (pSTG), although to varying degrees, as can be seen in Table 2. It is worth noting that most of the aphasic participants had lesions involving subcortical as well as cortical structures. The presence of subcortical lesions is not surprising since lesions in chronic aphasics are typically large and deep, extending into subcortical areas (Damasio, 1998).

In addition to participants with lesions involving either the SMG or IFG, there was one participant who had a lesion that included both the SMG and IFG. The inclusion of this patient allowed us to determine whether damage to the network including these structures would show a greater decrement in performance than lesions to either area alone. Additionally, we included two aphasic participants who had no lesion in either the IFG or SMG. This would allow us to determine whether a failure to show lexically conditioned phonetic variation was a function of damage specifically to the SMG and/or IFG rather than to a more general left-hemisphere 'lesion effect'.

Materials—The stimuli were a subset of those used by Baese-Berk and Goldrick (2009). They consisted of 30 pairs of monosyllabic words beginning with the voiceless stops /t/ and /k/. Each target pair shared both the same initial voiceless stop consonant and vowel. In each pair, one member had a minimal pair competitor (MP) with an initial voiced consonant (e.g. 'cap' with neighbor 'gap'), while the other did not (NMP) (e.g. 'cad' but no *'gad'). Of the original Baese-Berk and Goldrick stimuli, two /k/-initial pairs and one /t/-initial pair were removed due to concerns that the voiced competitors were uncommon words. Two new /k/-initial pairs were added. In total, 12 /k/-initial pairs and 18 /t/-initial pairs were included as targets in the experiment (see Appendix 1 for the word list).

As described by Baese-Berk and Goldrick (2009), all stimulus pairs were matched for initial consonant and vowel, sum segmental probability, sum biphone probability, number of phonemes, and neighborhood density. Sum segmental probability and sum biphone probability were calculated using the Phonotactic Probability Calculator (Vitevich & Luce, 2004). All words were low frequency, defined as a score of 45 or lower (Ku era & Francis 1967) using the University of Western Australia MRC Psycholinguistic Database. Each target pair was matched for lexical frequency.

The 60 target words were presented in a list along with 128 monosyllabic filler words, which were included to provide a richer phonological set for production than the initial voiceless stop targets. For the MP target words, their voiced minimal pair competitor was not presented. Approximately half of the fillers began with a stop consonant, evenly distributed between /p/, /b/, /d/, and /g/-initial words (e.g. 'pool', 'boss', 'dig', 'geese') and /t/ and /k/-initial words (e.g. 'tend', 'keep'), none of which had minimal pair competitors. The remainder of the fillers began with non-stop consonants and included fricatives, affricates, nasals, and glides (e.g. 'soup', 'jack', 'near', 'wrong'). None of the fillers

included the voiced minimal pair competitor of any of the target words and none of the fillers rhymed with any of the targets.

Stimuli were presented singly on a computer screen, centered on an IBM Thinkpad laptop computer running the BLISS software suite (Mertus, 2002). The data were recorded as 24-bit uncompressed WAV files using a Sony stereo microphone connected to an Edirol R-09 24bit Digital Recorder. The data were then transferred to a computer for acoustic analysis and at a sampling rate of 22050 Hz with a 16 bit quantization for acoustic analysis (Mertus, 2002).

Procedure—Stimuli were presented in a self-paced reading task. Single words appeared one at a time in the center of a laptop computer screen. Participants were instructed to read the word aloud and press the space bar on the keyboard to advance to the next stimulus. The list was presented in three blocks with stimuli in each block presented in pseudorandom order, with a short break in between each list presentation. Thus, the experiment consisted of a total of 564 words, 188 words produced 3 times, once in each block. Before the experiment began, each participant completed a short practice trial that consisted of 10 monosyllabic words, none of which appeared in the experimental set. Participants were instructed to speak clearly and naturally. They were given breaks between blocks, and on occasion, the experiment was paused in the middle of one of the lists to allow the participant to rest. This occurred either when the individual asked to take a break or made more than 5 errors in a row. One subject (B5) was unable to complete the entire experiment due to fatigue. Hence, the third block was omitted for that individual, and the first and second blocks were used for later analysis. In addition, 7 pairs were excluded for one older normal control due to recording error.

Analysis of Results—Two measures were used in the analysis of the target word stimuli: acoustic analysis of voice onset time (VOT) of the initial voiceless stop consonant for correct target productions and analysis of errors for incorrectly produced tokens. VOT was determined with the aid of a software waveform editor Mev (Mertus, 2002) by placing cursors at the release burst of the stop consonant and at the onset of vocal cord vibration. Visual inspection, linear predictive coding (LPC) analysis, and spectrogram analysis were used to verify the onset of glottal excitation. In cases where there was a double burst, VOT was calculated from the first burst. In the cases where it was difficult to discern the resolution of the burst and onset of vocal vibration, the tokens were edited using a highpass filter set at 100 Hz. VOT was then measured in the edited files using the procedure described above. Utterances were not included in the VOT analysis if subjects stuttered or produced a different word for the target token. For each individual, the average VOT of the correctly produced trials for each of the 60 target words was computed and served as the VOT measure for that word.

There were a number of tokens that could not be analyzed. If subjects leaned too close to the microphone, they occasionally produced utterances that had large bursts of air that obscured the normal waveform associated with the production of the initial stop consonant. Such utterances were discarded. Utterances in which the burst was obscured by a cough or by the subject clearing his/her throat were eliminated. Instances in which it was too difficult to identify the stop burst or the transition from initial consonant to vowel were also not included.

Error rate was computed by counting the number of target tokens produced incorrectly. Errors for MP and NMP targets were categorized as phonemic paraphasias, semantic paraphasias, perseverations, stutters, no response, or unclassifiable. In a handful of cases (n

= 4) in which the error production neither a semantic paraphasia nor a clear phonemic paraphasia, the error was marked as unclassifiable.

VOT analyses—The MP and NMP experimental stimuli were created to control for possible variation in VOT caused by the vowel context. Thus, following Baese-Berk and Goldrick (2009), we compared the VOT productions only for those word pairs in which tokens for both members of the particular MP-NMP pair were spoken at least once. Each word's VOT value was derived from averaging the word's correctly produced tokens. Table 3 shows the number of pairs analyzed for each aphasic participant.

Results of the VOT analyses are shown in Table 4. As the Table shows, with the exception of one IFG patient and one normal control, all participants (aphasics and normal controls) showed the minimal pair effect. In particular, the VOT was on average longer in target words with a voiced minimal pair competitor (MP) than in target words having no voiced minimal pair competitor (NMP). Of interest, patient B4, with a lesion that included both the IFG and SMG showed the minimal pair effect, as did patients C5 and B5, with no damage in either area. Although both groups of subjects showed variability in the magnitude of the minimal pair effect, the range of differences in the VOT of MP and NMP words for the aphasics (−.67 to 3.65) fell within that of the older normal controls (−2.62 to 4.55).

Figure 1 shows the VOT analysis of participants with lesions in the IFG or in the SMG. As can be seen, similar to normal controls, both groups show the MP effect. Results of a 2×2 ANOVA with the factors of participant group (older normal controls and aphasics with IFG damage or SMG damage) by word type (MP/NMP) revealed a main effect of condition ($F(1,13) = 18.96, p < .001$), a main effect for group that approached significance ($F(1,13) = .06, p < .881$) and no interaction ($F(1,13) = .779, p < .393$). An additional 2×2 ANOVA comparing the aphasic participants with IFG damage to those with SMG damage revealed only a main effect of condition ($F(1,5) = 11.813, p < .018$).

The first VOT analysis used only the stimuli for which each token of the MP-NMP pair was produced correctly. However, this meant that if a subject failed to correctly produce a word in either the MP or NMP condition, the word pair was not included in the VOT analysis, resulting in a reduction of the data set included in the statistical analysis. To determine whether the VOT patterns described above were due to the reduced statistical power from limiting the analysis to only correctly produced target pairs, a second analysis was conducted using all correctly produced tokens, regardless of whether their matched pairs were produced correctly. Table 3 shows the number of tokens gained for each aphasic participant and Table 5 shows the results of the VOT analysis for each participant using these data. As can be seen, the results are similar to the paired target stimulus analysis with all participants except for 2 aphasic participants (one IFG only and one SMG only) and 1 older normal control showing the VOT minimal pair effect. Again, the patient with a lesion that included both the IFG and SMG showed the minimal pair effect, as did the 2 aphasic participants with no damage in either area.

Analysis as a function of lesion site revealed the same pattern as in the paired target word analysis. Figure 2 shows the results. Results of a 2×2 ANOVA with the factors of participant group (older normal controls and aphasic participants with IFG damage or SMG damage) by word type (MP/NMP) revealed a main effect of condition ($F(1,13) = 11.489, p < .005$), but no main effect for group ($F(1,13) = .066, p = .801$) and no interaction ($F(1,13) = .621, p = .445$). An additional 2×2 ANOVA comparing the aphasics participants with IFG damage to those with SMG damage did not reveal a main effect of condition ($F(1,5) = 2.221, p = .196$), group ($F(1, 5) = .327, p = .592$), or an interaction $F(1, 5) = .009, p = .927$). Despite the failure to show a significant effect of condition, five out of the 7 of those participants with

lesions only in the IFG or SMG still showed the MP effect, and, as can be seen in Figure 2, the patterns of results for the IFG only (N=3) and SMG only (N=4) patients is similar to that of the paired analysis shown in Figure 1.

Error Analyses—As indicated above, aphasic participants produced a large number of production errors that were not analyzed in the VOT analysis. The numbers of errors patients made ranged from 3 to 71. Table 6 shows the total number of errors made for target words in the MP and NP conditions for each aphasic participant. As can be seen, all aphasics made more errors on the NMP words than the MP words. As elaborated in more detail in the discussion section, these findings are consistent with prior research showing a facilitatory effect on the production of words that share phonological attributes compared to words that do not (Vitevitch, 2002).

The types of errors participants made also provide some insight into the locus of the failure to produce the correct target. In particular, it is possible that production errors were driven by phonological, semantic, or articulatory implementation factors. To examine this issue, errors were classified into 6 categories: *phonemic paraphasias*, which reflect errors in accessing the phonological properties of the words, *verbal paraphasias*, which reflect errors in accessing the semantic properties of the words, *perseverations*, which reflect the continued activation of earlier responses in working memory, *stutters*, which reflect articulatory implementation difficulties, and *no responses*, which could occur for a variety of reasons. A sixth category, *unclassifiable*, included error productions that could not be classified as either phonemic or verbal paraphasias.

Results for the errors made by each of the patients as a function of error category are shown in Table 7. They reveal that the largest category of errors produced by the aphasic participants was phonemic paraphasias, followed by perseveration errors. Stutters and no responses were also prevalent, while verbal paraphasias and unclassifiable tokens were relatively rare.

Given that the largest category of errors was phonemic paraphasias, it was of interest to examine the pattern of errors within this error type. One characteristic and common type of phonemic paraphasia found in aphasic patients is phoneme substitution errors (Blumstein, 1973; Lecours & Lhermitte, 1969). Phoneme substitution errors were analyzed in terms of the phonetic feature distance between the substituted phoneme and the target phoneme. Results, shown in Table 8, are consistent with earlier findings indicating more single feature errors than errors of more than one feature in spoken word production (Blumstein, 1973; Lecours & Lhermitte, 1969).

Finally, it is of interest to determine the extent to which phoneme substitution errors resulted in words or in nonwords. Table 9 shows that the erroneous utterances produced tended to be real words phonemically related to the target rather than nonwords.

Lesion Analyses—As Table 2 shows, the extent of lesion in the IFG and SMG, the 2 neural areas implicated in lexically conditioned variation in the Peramunage et al. (2011) fMRI study with normals, varied across the aphasic participants. It is possible that the magnitude of VOT differences between MP and NMP words could have been affected by the extent of lesion in these areas. To examine this question, we conducted correlation analyses examining the percent damage to either the IFG or the SMG with the magnitude of VOT differences between MP and NMP words. A third ROI, Wernicke's area (pSTG), was also included since this area has not only been implicated in fMRI studies with normals in phonological encoding processes (Hickok & Poeppel, 2007) but also in speech output deficits in participants with fluent aphasia (Goodglass, 1993; Damasio, 1998). Results failed

to show any statistical correlations in any of these areas, IFG $r(9) = .22$; SMG $r(9) = .43$; Wernicke's area $r(9) = .018$.

Although we did not find that the percent lesion data correlated with the magnitude of the VOT differences between MP and NMP target words, it is possible that the number of errors that patients made was affected by the extent of lesion and also lesion localization. To examine this question, we conducted correlations between the percent damage to the IFG, SMG, or Wernicke's area and number of errors made. Results once again failed to show any significant correlations IFG $r(9) = .407$, SMG $r(9) = .644$, and Wernicke's $r(9) = .110$, although the correlation between percent area lesioned and number of errors approached significance ($p = .061$) for the SMG.

Nonetheless, earlier lesion studies have implicated a number of subcortical structures in speech output deficits. In particular, damage to the anterior limb of the internal capsule, periventricular white matter and the medial subcallosal fasciculus appears to interrupt connections for initiation and preparation of speech movements and for motor execution and sensory feedback for spontaneous speech (Naeser et al., 1989). Thus, it is possible that the number of errors produced by the patients will be correlated with these areas (see Table 2 which shows the percent of lesion in these areas for the patients). Correlation analyses showed that indeed there was a significant relationship between error performance and percent lesion in subcortical areas: the anterior limb of the internal capsule $r(9) = .803$, the middle periventricular white matter $r(9) = .778$, and the basal ganglia $r(9) = .848$. Other subcortical areas did not reach significance including the anterior periventricular white matter $r(9) = .468$, $p = .072$ and medial subcallosal fasciculus $r(9) = .545$. These findings indicate that the extent of subcortical damage was a predictor of the number of production errors made by the aphasic patients.

Discussion

The current study examined the influence of sound shape properties of the lexicon on the phonetic realization of words in spoken word production in aphasic patients. Such study provides a unique window into spoken word production in aphasic participants for it allows for an examination of the potential integrity of cascading processes and in particular whether higher level deficits, i.e. deficits in lexical access and lexical selection processes, influence acoustic-phonetic output. Previous fMRI research suggests that the neural system underlying this lexically conditioned phonetic variation includes the left supramarginal gyrus (SMG) extending into the pSTG, inferior frontal gyrus (IFG), and precentral gyrus (Peramunage et al., 2011). The goal of the current study was to determine the extent to which damage to the IFG and/or SMG resulted in concomitant impairments. We also examined the potential role of the pSTG (Wernicke's area) as well. Voice onset time analyses revealed surprisingly that aphasic participants with damage in these areas nonetheless showed, as do normals, VOT lengthening effects for words with minimal pair competitors compared to words without minimal pair competitors. Indeed, one aphasic participant with damage that included both the IFG and SMG also showed the minimal pair effect.

That aphasic participants and older normal controls show modulation of voice onset time as a function of the lexical properties of the word, i.e. whether or not there is a voiced phonetic competitor, provides further support for cascading models of spoken word production. Finding that the older normals display lexically conditioned phonetic variation indicates that this effect is robust throughout life, despite other changes in speech production associated with aging such as slower speaking rates (Parnell & Amerman, 1987; Duchin & Mysak, 1987; Smith, Wasowicz, & Preston, 1987), declining quality of speech (Hartman &

Danhauer, 1976), and increased variability in VOT production (Neiman, Klich, & Shuey, 1983; Petrosino, Colcord, Kurcz, & Yonker, 1993; Sweeting & Baken, 1982).

Nonetheless, our results suggest a conflict between the neuroimaging and clinical results concerning the role of the IFG and SMG in processing lexical information. Similar to Price et al.'s (1999) neuropsychological approach, we examined the relation between the behavioral performance of patients on a specified task with lesions in neural areas that were recruited in functional neuroimaging of normal individuals doing the same task. We found that aphasic patients with lesions to the SMG and/or IFG show lexically conditioned variation, suggesting that they are sensitive to the sound structure properties of the lexicon and that competition induced by these properties ultimately influences phonetic planning and articulatory processes.

As described in the introduction, both the IFG and SMG are implicated in studies of spoken word production and auditory word recognition in aphasia (Caplan et al., 1995; Yee et al., 2008). How then can one explain the normal patterns obtained in lexically conditioned phonetic variation? One possibility is that neither the SMG nor the IFG are necessary for processing the lexical information to be used in articulatory planning and production, and the clusters revealed in the Peramunage et al. (2011) fMRI study reflected some other cognitive processes recruited during the spoken word reading task. A second possibility is that the phonological/lexical system is broadly distributed in the perisylvian areas of the left dominant hemisphere and recruits other neural areas in addition to the SMG and IFG. Thus, it is possible that we have not yet identified the combination of ROIs which are the most critical before reaching the threshold of damage within the network that would consistently produce impairments. A third possibility is that there is functional recovery in chronic aphasics, as those tested here. That is, it is possible that there may be residual functional integrity in tissue surrounding the lesion (peri-infarct tissue) or functional reorganization involving ipsilateral and/or contralateral homologous neural regions in our patients. While this is a possibility, there is evidence in the literature showing that deficits still emerge in particular language and/or cognitive functions for chronic patients who have lesions in neural areas identified in neuroimaging studies. For example, using fMRI, Schnur et al. (2009) showed that the IFG was recruited in normal participants in a semantic blocking naming paradigm designed to induce the selection of words from among competing semantic alternatives. Using the same task, they demonstrated that aphasic patients with lesions in the IFG showed a deficit in naming words presented in blocks of semantically related words, consistent with their neuroimaging findings. All participants were chronic aphasics who were tested between 16 and 175 months post-insult. A correlation analysis failed to show any relation between their degree of deficit and time post-onset. Thompson-Schill et al. (1998) also showed deficits in selecting verbs from among competing alternatives in chronic brain-injured participants (time post-onset ranged from 12 to 131 months) who had lesions in the IFG, the area activated in normals performing the same task. Thus, chronic aphasic participants can and do show impairments in similar neural areas as those implicated in language or cognitive functions using fMRI, and performance is not related to time post-onset. A correlation analysis between time post-onset and magnitude of the minimal pair effect in the current study was not significant ($r = .28$). Thus, similar to the findings of Schnur and Thompson-Schill, time post-onset is not a predictor of performance. That is, there failed to be a larger MP effect as a function of potential recovery time.

Nonetheless, it is not always the case that there is a relation between fMRI findings in normals and performance with brain-lesion participants. For example, Price et al. (1999) demonstrated normal performance in a patient 4 years post-stroke in a semantic similarity judgment task. This participant's lesion included areas activated in a neuroimaging study

with normals using the same task. Based on these findings, the authors concluded that these neural areas were not necessary for performing this task.

How then do we interpret the results of the current study? That our chronic aphasic participants showed lexically conditioned phonetic variation challenges the strong form of the claim that damage to areas showing activation in neuroimaging studies necessarily result in concomitant behavioral deficits in brain-lesioned patients. When the aphasic participants succeeded in accessing the correct target word, they implemented the words normally, showing sensitivity to the lexical (minimal pair) properties of the word. These findings were not predicted by the fMRI findings showing modulation of activation as a function of context-conditioned phonetic variation in the SMG, pSTG, and IFG.

Nonetheless, the interpretation of such failures needs to be made with caution. The operational measure used in the current experiment was the same measure as that of Peramunage et al. (2011). Here the VOT measure failed to show deficits. However, further analysis of the data also revealed that the patients made errors in their production of some of the target words. Indeed, examination of the pattern of errors suggests an impairment in the spoken word production system. In particular, all aphasic participants made more errors on non-minimal pair words than minimal pair words. These findings are consistent with the view that the selection of a target word is influenced by the sound structure properties it shares with other words in the lexicon (Dell & Gordon, 2003; Baese-Berk & Goldrick, 2009). As described earlier, the normal literature has shown facilitation in production of words which have many phonologically similar words compared to those that do not (Baus, Costa, & Carreiras, 2008; Vitevitch, 2002; Vitevitch, Armbrüster, & Chu, 2004; Vitevitch, 1997, 2002; Stemberger, 2004; Nickels & Best, 1996; Gordon, 2002; Kittredge, Dell, Verkuilen, & Schwartz, 2008; Newman & German, 2005; Lallini & Miller, 2011; Harley & Bown, 1998; Vitevitch & Sommers, 2003). With respect to the current study, the fewer errors made by the aphasic participants on minimal pair words compared to non-minimal pair words are likely due to the degree of overlap between the phonological representation of the target word and its minimal pair neighbor. Minimal pair words share all phonological properties of the word except for the voicing of the initial consonant. It is this large overlap in the number of sound segments that the competitor shares with the target word that facilitates its production by increasing the activation of the shared segments in relation to the other sound segments in the lexicon.

Further analysis of errors revealed that every aphasic participant made speech output errors that were predominantly phonological in nature resulting in a preponderance of phonemic paraphasias involving single feature substitutions. Single phonetic feature substitutions are among the most common types of phonemic paraphasias occurring in aphasic patients in spoken word production in patients clinically diagnosed with Broca's and Wernicke's aphasia (Blumstein, 1973; Lecours & Lhermitte, 1969).

Finally, these phonological errors made by the patients were more likely to result in a word than a nonword, a finding consistent with the literature examining errors in spoken word production in aphasia (Nickels & Best 1996; Gagnon, Schwartz, Martin, Dell, & Saffran 1997; Blanken 1998; Laine & Martin, 1996). This lexical bias favoring real word responses in such errors supports the interactive functional architecture of the speech/lexical processing system. Thus, if the retrieval of a sound segment is disrupted, the target's phonological neighbors that have been activated by feedback from the correctly retrieved sound segments are more likely to be selected than a phonologically related nonword that lacks lexical activation (Dell, 1986).

Taken together, the pattern of these errors indicate that the aphasic participants have an impairment in the processes involved in lexical access and phonological implementation of words, supporting the original claim that the SMG and IFG are part of a neural network involved in spoken word production. In this view, phonological/lexical information from the SMG cascades to the IFG for selecting the word from among the competing set of potential word candidates (Righi et al., 2010; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997) and for phonological planning and implementation processes (Guenther, 2006; Huang, Carr, & Cao, 2002; Bookheimer, Zeffiro, Blaxton, Gaillard, & Theodore, 1995; cf. also Indefrey & Levelt, 2004).

That a deficit in spoken word production emerged in the aphasic participants only in the error analysis suggests that their deficit was sufficiently mild that it did not show up in voice onset time measures of correctly produced targets. It is possible that testing a larger group of patients would have identified individuals with more severe deficits that affected not only the pattern of errors produced but also the production of lexically conditioned phonetic variation.

Finally, the lesion analyses indicate that the aphasic patients tested have extensive lesions involving more than the inferior frontal gyrus and/or supramarginal gyrus extending into the pSTG. Of interest, the extent of subcortical damage was the only factor that predicted the number of errors made by the patients. These findings support earlier research showing that damage to subcortical areas influences the extent to which nonfluent patients showed improvement in speech output post-stroke (Naeser et al., 1989).

In sum, the current experiment provides further evidence of the importance of the lesion-based approach for understanding the neural systems underlying language and speech processing. While in recent years, there has been increasing focus and reliance on functional neuroimaging in studying the cognitive neuroscience of language, it is clear that no single methodology can inform our understanding of the neural systems underlying language. Indeed, each methodology has both its strengths and its limitations, requiring the convergence of multiple methods in the examination of the cognitive neuroscience of language. The study of aphasia is a critical linchpin in that endeavor.

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APPENDIX 1

Experimental Stimuli

K-initial MP	Stimuli NMP	T-initial MP	stimuli NMP
CAB	CAD	TAB	TAT
CAP	CAT	TAME	TAINT
CAPE	CAKE	TAN	TAG
COAL	CONE	TANK	TAP
COAT	COKE	TART	TAR
COD	COP	TAUNT	TAUT
CODE	COMB	TEAL	TEAT
COO	COOT	TED	TEMPT
CORE	CORN	TEEM	TEETHE
CURL	CURB	TENSE	TENTH
CUSS	CUB	TICK	TIFF
KILT	KILN	TILE	TIGHTS
		TOE	TOAST
		TOMB	TOOTH
		TORE	TORCH
		TORQUE	TORN
		TOTE	TOAD
		TUCK	TUFT

Highlights

Phonetic output of words is influenced by sound shape properties of the lexicon.

FMRI findings show this effect recruits a network of neural areas.

Lesions in this network fail to show deficits in producing this phonetic effect.

Percent area lesioned did not predict the magnitude of the phonetic effect.

FMRI findings do not always predict effects of lesions on deficits in aphasia.

Figure 1.
VOT results for paired MP and NMP words produced by IFG (3), SMG (4), and control (8) participants

Figure 2.
VOT results for all correctly produced MP and NMP words produced by IFG (3), SMG (4), and control (8) participants

Table 1

Percentile scores on BDAE subtests

	Time post onset (mos.)	Severity	Fluency	Auditory Comp	Oral Reading Single word	Repetitn	Literal Paraphasia
B1	6	86.00	61.25	81.25	88.00	93	90.00
B2	95	85.00	52.50	97.50	88.00	76	70.00
B3	151	90.00	---	91.25	---	---	---
B4	160	85.00	57.50	90.00	90.00	83	80.00
B5	280	85.00	56.25	92.50	90.00	75	70.00
C1	71	95.00	68.75	76.25	88.00	40	50.00
C2	24	85.00	67.50	63.50	90.00	43	33.00
C3	36	88.00	71.25	75.00	90.00	80	60.00
C4	58	95.00	72.50	90.00	90.00	67	80.00
C5	9	95.00	66.25	86.25	83.00	75	60.00

Table 2

Percent Lesion Analysis of ROIs

Study ID	Clinical Diagnosis	IFG	SMG	Wernicke's Area	ALIC	Middle 1/3 Periventricular White Matter	Medial Subcallosal Fasciculus	Anterior 1/3 Periventricular White Matter	Basal Ganglia
B1	Broca	20	0	0	0	8	0	25	0
B2	Broca	39	0	0	60	81	38	79	74
B3	Broca	95	0	0	15	20	20	85	10
C1	Conduction	0	99	74	0	70	0	30	35
C2	Conduction	0	7	41	89	51	0	10	58
C3	Conduction	0	84	12	0	5	0	0	6
C4	Conduction	0	5	45	0	0	0	0	0
B4	Broca	100	80	0	100	80	95	85	20
B5*	Broca	--	--	--	--	--	--	--	--
C5	Conduction	0	0	3	0	0	0	0	0

* The original CT scan for this patient was not available for the percent lesion analysis

Table 3

Stimuli Used in VOT Analyses (see text)

Participant	# pairs in analysis (out of 30)	# tokens gained in unpaired analysis
B1	29	1
B2	21	9
B3	29	1
B4	17	11
B5	7	17
C1	23	7
C2	24	5
C3	26	4
C4	29	1
C5	28	2

Table 4

VOT results (in ms) for MP and NMP target words paired by vowel context

Paired VOTs					
Subject#	Lesion	Clinical Type	MP	NMP	Difference
B1	IFG	B	99.84	98.91	0.93
B2	IFG	B	74.59	72.72	1.87
B3	IFG	B	62.90	63.57	-0.67
C1	SMG	C	137.65	136.85	0.80
C2	SMG	C	68.66	65.62	3.05
C3	SMG	C	87.62	83.98	3.65
C4	SMG	C	73.93	71.07	2.86
B4	SMG and IFG	B	119.51	109.64	9.87
B5	no SMG or IFG	B	105.33	96.08	9.25
C5	no SMG or IFG	C	86.63	84.70	1.92
N1	N/A	Normal	99.99	98.33	1.66
N2	N/A	Normal	88.52	84.66	3.86
N3	N/A	Normal	85.55	81.00	4.55
N4	N/A	Normal	99.94	96.44	3.50
N5	N/A	Normal	78.43	74.80	3.63
N6	N/A	Normal	64.71	60.80	3.91
N7	N/A	Normal	90.85	87.84	3.01
N8	N/A	Normal	67.02	69.64	-2.62

Table 5

VOT results (in ms) for all correctly produced MP and NMP target words

Unpaired VOTs					
Subject#	Lesion	Clinical Type	MP	NMP	Difference
B1	IFG	B	99.20	98.91	0.29
B2	IFG	B	76.82	71.80	5.02
B3	IFG	B	63.49	63.57	-0.08
C1	SMG	C	135.55	138.86	-3.32
C2	SMG	C	68.78	65.21	3.56
C3	SMG	C	88.43	84.45	3.98
C4	SMG	C	74.77	71.07	3.70
B4	SMG and IFG	B	121.67	110.79	10.88
B5	no SMG or IFG	B	98.20	94.17	4.03
C5	no SMG or IFG	C	87.53	84.70	2.83
N1	N/A	Normal	99.99	98.33	1.66
N2	N/A	Normal	88.52	84.66	3.86
N3	N/A	Normal	85.55	81.00	4.55
N4	N/A	Normal	101.92	95.75	6.17
N5	N/A	Normal	78.43	74.80	3.63
N6	N/A	Normal	64.71	60.80	3.91
N7	N/A	Normal	90.85	87.84	3.01
N8	N/A	Normal	67.02	69.64	-2.62

Table 6

Total production errors by MP and NMP conditions

Total errors					
Subject#	Lesion	Clinical Type	Total	MP	NMP
B1	IFG only	B	3	0	3
B2	IFG only	B	45	16	29
B3	IFG only	B	21	8	13
C1	SMG only	C	34	11	23
C2	SMG only	C	50	19	31
C3	SMG only	C	26	7	19
C4	SMG only	C	17	4	13
B4	SMG and IFG	B	71	25	46
B5	no SMG or IFG	B	71	31	40
C5	no SMG or IFG	C	4	1	3

Table 7

Percent Error Type for MP and NMP Targets

Subject#	Lesion	Clinical type	Phonemic Paraphasias	Verbal Paraphasias	Perseverations	Stutter	No response	Unclassifiable
			MP/NMP	MP/NMP	MP/NMP	MP/N MP	MP/NMP	MP/NMP
B1	IFG only	B	0/33	0/0	0/67	0/0	0/0	0/0
B2	IFG only	B	24/38	0/0	2/20	2/0	7/7	0/0
B3	IFG only	B	19/33	0/0	10/14	0/0	10/14	0/0
C1	SMG only	C	18/47	0/0	15/21	0/0	0/0	0/0
C2	SMG only	C	24/32	0/0	12/20	2/10	0/0	0/0
C3	SMG only	C	8/31	0/0	0/31	15/8	4/4	0/0
C4	SMG only	C	18/29	0/0	0/35	6/6	0/6	0/0
B4	SMG and IFG	B	4/10	0/0	6/13	7/13	18/28	0/1
B5	no SMG or IFG	B	14/11	3/4	15/24	1/4	7/11	2/1
C5	no SMG or IFG	C	25/25	0/0	0/50	0/0	0/0	0/0

Table 8

Phonetic Feature Analysis of Phoneme Substitution Error

	1 Phonetic Feature	>1 Phonetic Feature
MP	45	6
NMP	73	13

Table 9

Phonemic Paraphasias Resulting in Words or Nonwords

	Word	Nonword
MP	36	15
NMP	54	32