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Notes and discussion

Sensitivity to sub-syllabic constituents in brain-damaged patients: evidence from word games

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Abstract

Two experiments were conducted to examine whether left- (LHD) and right-hemisphere-damaged (RHD) patients exhibit sensitivity to sub-syllabic constituents (i.e., onsets and codas) in the generation of nonwords, using a word games paradigm adapted from Treiman (1983). Four groups of individuals (including LHD fluent and nonfluent aphasic patients, RHD patients and normal controls) were trained to add syllables to monosyllabic CVC nonwords either after the initial consonant (Experiment 1) or prior to the final consonant (Experiment 2) to create bisyllabic nonwords. Experimental stimuli consisting of CCVC or CVCC nonwords tested whether participants would preserve or split the onset and coda constituents in producing the novel bisyllabic nonwords. Results revealed that the majority of subjects demonstrated sensitivity to the sub-syllabic constituents, preserving the onsets and codas. The fluent aphasic patients exhibited a greater than normal tendency to split the onset and coda constituents; however, the small number of individuals in that group whose data met inclusion criteria limits the conclusions that may be drawn from these findings. The results are discussed in relation to theories of phonological deficits in aphasia.

1. Introduction

Phonological processing plays an important role as an entry-point to auditory word recognition and higher-level language comprehension. It follows, therefore, that anything that interferes with that entry-point may have serious consequences for higher-level processing. Thus, impairments in phonological processing commonly associated with aphasia resulting from focal brain damage to the left hemisphere may have significant effects on language understanding in general. Whereas no clear relationship has been demonstrated between speech perception skills and higher-level

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auditory comprehension abilities in aphasic patients (see Blumstein, 1998 for review), there has been increasing interest in the role of phonological form processing (and impairments thereof) in lexical access (e.g., Baum, 1997; Baum & Leonard, 1999; Blumstein et al., 2000; Gordon & Baum, 1994; Leonard & Baum, 1997; Milberg, Blumstein, & Dworetzky, 1988). Relatedly, influences of lexical-level variables on phonetic perception have also been explored (e.g., Blumstein, Burton, Baum, Waldstein, & Katz, 1994; Boyczuk & Baum, 1999).

Investigations that have examined phonological effects in word recognition in brain-damaged patients have yielded mixed findings. For instance, Milberg and colleagues (1988) found that nonfluent Broca's aphasic patients failed to show phonologically mediated priming effects (e.g., "gat" priming "dog" via "cat") in a lexical decision task, whereas fluent Wernicke's aphasics showed greater than normal mediated priming which was as strong as direct associative priming for these individuals. In an effort to ascertain the potential locus of the absence of mediated priming in the nonfluent aphasic patients, Gordon and Baum (1994) investigated direct phonological (rhyme) priming effects (e.g., "gat" priming "cat" or "rat" priming "cat") in both fluent and nonfluent aphasic patients, as well as a group of normal controls. Gordon and Baum (1994) reported relatively normal rhyme priming effects in both fluent and nonfluent aphasic patients, suggesting that these individuals were appropriately activating phonological word forms—in particular, the syllable rimes (see also Baum & Leonard, 1999; Leonard & Baum, 1997). Baum and Leonard (2000) also found relatively normal priming effects of phonology and orthography in left-hemisphere-damaged (LHD) fluent and nonfluent aphasic patients (as well as right-hemisphere-damaged (RHD) patients) for word-initial (i.e., onset) overlap as well.[1] While these studies do not present a fully consistent picture, they do provide evidence that both fluent and nonfluent LHD aphasic patients are sensitive to some aspects of sublexical phonological form (cf. Blumstein et al., 2000). Interestingly, in the literature on normal language processing, the role of the syllable and sub-syllabic constituents as units of processing (and/or units of representation in memory) has been the focus of a good deal of research (e.g., Cutler, Butterfield, & Williams, 1987; Finney, Protopapas, & Eimas, 1996; Mehler, Dommergues, Frauenfelder, & Segui, 1981; Pitt, Smith, & Klein, 1998; Smith & Pitt, 1999; Treiman, 1983, 1986). Evidence from a range of paradigms suggests that syllables and subsyllabic units are important in auditory word recognition, but their precise role as 'units of perception' remains open to question (see e.g., Norris & Cutler, 1985). To date, little direct evidence for sensitivity to sub-syllabic constituents (larger than, or at a level higher than, the phoneme) in brain-damaged patients has been advanced. In fact, in terms of speech perception, direct evidence for sensitivity to syllabic structure in aphasic patients is scant (but see Berndt, Haendiges, Mitchum, & Wayland, 1996 for evidence concerning patients with acquired reading impairments).

Another means of assessing underlying sensitivity to sub-syllabic constituents is by examining production rather than recognition. In particular, studies of speech errors have generally supported the notion that LHD aphasic patients are sensitive to syllable structure, producing substitution errors that typically obey syllabic constraints, substituting onsets for onsets, codas for codas, etc. (see Blumstein, 1990 for

[1] It should be noted that, while both facilitory and inhibitory effects of initial overlap have been found for normal individuals, depending on the specific task requirements, there is clearly an influence of shared onset phonology on word recognition. The locus of that effect remains under debate (see e.g., Hamburger & Slowiaczek, 1996; Slowiaczek, McQueen, Soltano, & Lynch, 2000, among others).

review). Further, syllable nuclei tend to be most resistant to error and consonant cluster errors seem to reflect syllable structure constraints (see Gordon, 2000 for review). A more direct test of brain-damaged patients' sensitivity to syllable-internal constituents was the goal of the present preliminary investigation, using a "word games" paradigm adapted from Treiman (1983).

In two of Treiman's (1983) experiments, normal subjects were trained to separate monosyllabic nonword stimuli into two syllables by inserting a syllable after the consonantal onset (Experiment 1: e.g., [kIg] + "add [æz]" → [kæz Ig]) or prior to the coda (Experiment 2: e.g., [fug] + "add [vi]" → [fu vig]). Following training, experimental test stimuli were presented that included consonant clusters (in onset or coda positions, depending on the experiment) to determine whether subjects would preserve the cluster constituent in creating the new two-syllable target or utilize a more segmental rule, inserting the additional syllable after the initial (or before the final) segment (i.e., phoneme) and thus splitting the onset or coda constituent. Results revealed that normal subjects tend to preserve syllable-internal constituents such as onsets and codas in this task, indicating their sensitivity to these structural components. This basic paradigm, with minor modifications, was adopted in the present investigation to provide a preliminary test of the methodology for use with brain-damaged patients and to evaluate patients' sensitivity to syllable-internal constituents or sublexical phonological structure. The primary question under investigation is whether LHD and RHD patients use similar processing routines or units of representation as do normal individuals in phonological analysis. One might hypothesize that LHD fluent aphasic patients may be the most likely to display aberant results, given their more frequent errors of phonological form in both production and perception. However, recent investigations of phonological perception using functional neuroimaging methods in normal individuals have supported a (somewhat surprising) role for the inferior frontal cortex in speech perception tasks that require overt segmentation (i.e., the separation of individual phonemic or sub-syllabic segments from the whole stimulus) or articulatory recoding (e.g., Burton, Blumstein, & Small, 2000; Zatorre, Meyer, Gjedde, & Evans, 1996). Although these results do not reflect sensitivity to syllable-internal constituents per se (i.e., onsets, rimes, nuclei, and codas), they do reflect the processes involved in breaking down syllables into their segmental components. On the basis of these findings with normal individuals, then, one might expect that patients with focal damage to the inferior frontal lobe (more likely to be nonfluent aphasics) would exhibit deficits in tasks requiring overt segmentation (see Baum, 2002 for related data). It is anticipated that the performance of the RHD patients will be comparable to normal.

2. Methods

2.1. Experiment 1

2.1.1. Subjects

Four groups of individuals participated in the experiment. Five LHD nonfluent aphasic patients, five LHD fluent aphasic patients, five RHD patients, and 12 age-matched normal controls with no history of speech or language disorders. The brain-damaged patients had all suffered a single, unilateral cerebrovascular accident confirmed by CT or MR scan. All participants were right-handed native speakers of English who passed a pure-tone audiometric screening at 35dB HL in the better ear at the speech frequencies (.5, 1, and 2 kHz). The brain-damaged patients were

diagnosed based on clinical reports and a battery of screening tests that varied depending on lesion laterality. All patients were administered the Bells Test for hemifield neglect (Gauthier, Dehaut, & Joanette, 1989), the Spoken Word-Picture Matching subtest of the *Psycholinguistic Assessment of Language* (*PAL*; Caplan, 1992), and the Auditory Sentence Comprehension subtest of the *PAL*. In addition, LHD patients were classified as fluent or nonfluent according to results of the *Boston Diagnostic Aphasia Examination* (*BDAE*; Goodglass & Kaplan, 1983) and ratings of their speech production characteristics. RHD patients were administered a test battery adapted from the *Test of Language Competence-Expanded Edition* (Wiig & Secord, 1987) to assess comprehension of figurative language and inferencing. Normal control subjects were also screened to rule out potential cognitive deficits using a modified version of the *Mini-Mental State Examination* (*MMSE*; Folstein, Folstein, & McHugh, 1975). Background characteristics on the participants appear in Table 1.

2.1.2. Stimuli

Three sets of nonword stimuli were designed to test whether subjects preserve syllable onsets as a unit in creating new nonwords by the addition of a syllable. The three sets varied in the syllable to be added: (a) add [æz]; (b) add [of]; (c) add [ib] (following Treiman, 1983). Four CVC nonwords were created as training stimuli for each set such that, upon addition of the syllable, both syllables remained nonwords (e.g., for the "add [æz]" set, [kIg] → [kæz Ig]). Instructions were provided by example so as not to influence the results; that is, mention of the initial consonant or initial sound was specifically avoided. The experimental stimuli for each set consisted of 10 CCVC nonwords with the same constraints as noted above. In addition, 5 CVC stimuli served as memory controls (e.g., [las] → [læz as]) to ensure that subjects recalled the original pattern on which they were trained; 5 additional CVC stimuli served as letter controls (e.g., [ʃog] → [ʃæz og] vs [sæz hog]). For the latter stimuli, the onset phoneme is realized orthographically as two letters in English. These stimuli were included to determine whether subjects might use a spelling-based strategy to perform the task. The entire list of stimuli is provided in Appendix A.

2.1.3. Procedure

Subjects were trained on a single set of training stimuli by first having them simply repeat the monosyllabic and bisyllabic nonword stimuli after the experimenter. Subsequently, the same CVC stimuli were presented in random order and subjects were asked to apply the rule (e.g., "add [æz]"). If errors were made, they were corrected. This training procedure continued until subjects reached a criterion of three successive trials correct for all four training items. After several attempts at the task with a number of brain-damaged patients, it was determined that the task requirements were too difficult for the patients. Thus, the procedure was modified slightly to include both auditory and written presentation of the monosyllabic stimuli. With this modification, all subjects were able to complete the task.[2]

Upon reaching criterion, subjects were presented with the novel experimental and control stimuli in random order and were again asked to apply the same rule. Monosyllables were presented orally and in orthographic form[3] by the examiner and

[2] It is likely that memory limitations were at the root of the difficulty with the auditory-only presentation. That is, although the rule was available throughout, retention of the nonword stimuli proved difficult for the patients–probably *because* they were nonwords.

[3] The orthographic forms of the nonwords were verified in a pre-test with a separate group of normal adults. Adequate spellings were determined for all stimuli.

Table 1
Background Information on participants

Subject	Sex	Age (years)	Education[a] (years)	MPO[b]	Lesion site	Diagnosis
Nonfluent aphasics						
1	M	79	9	52	Left frontal	Mild nonfluent (anomic)
2	F	67	9	74	Left fronto-temporo-parietal	Moderate-severe nonfluent
3	F	48	14	97	Left fronto-parietal	Nonfluent
4	M	51	14	143	Left parietal	Mild-moderate nonfluent
5	M	73	16	28	Left MCA[c]	Severe nonfluent
	Mean	64	12	79		
	SD	14	3	44		
Fluent aphasics						
1	F	85	9	95	Left paraventricular, deep parietal region	Mild fluent
2	F	76	9	95	Left temporo-parietal	Mild fluent (anomic)
3	F	87	9	34	Left fronto-parietal	Fluent (anomic)
4	M	71	8	53	N/A[d]	Mild-moderate fluent (anomic)
5	M	71	14	21	Left MCA[c]	Moderate fluent (anomic)
	Mean	65	11	68		
	SD	20	4	36		
Right-hemisphere-damaged patients						
1	M	71	12	42	Right parietal	
2	F	58	13	115	Right posterior communicating artery	
3	M	89	11	35	N/A[d]	
4	M	78	11	32	Right temporo-parietal	
5	F	65	13	58	Right internal capsule, right basal ganglia	
	Mean	72	12	56		
	SD	12	1	34		

Table 1 (*continued*)

Subject	Sex	Age (years)	Education[a] (years)	MPO[b]	Lesion site	Diagnosis
Normal controls						
1	F	58	11			
2	M	70	9			
3	F	73	13			
4	M	70	9			
5	F	68	11			
6	F	68	11			
7	M	66	9			
8	F	66	12			
9	F	72	11			
10	M	55	11			
11	F	80	9			
12	F	67	13			
	Mean	67	11			
	SD	13	2			

[a] Best estimated conversion into years, based on information from subject (e.g., 2 years college, high school).
[b] Months post onset.
[c] Middle cerebral artery.
[d] Information not available.

the rule to apply was written in orthographic form and remained visible throughout the experiment. Responses were recorded by the examiner. Those individuals whose performance on the memory controls was below 60% accuracy were eliminated from further analyses. Results for the letter control stimuli were not used as exclusionary criteria because performance on these stimuli merely served to verify whether the experimental results may have been due to application of an orthographic rather than a phonological strategy.

Testing of the other two sets of stimuli was completed in a comparable fashion and the order of rules to be applied was counterbalanced across subjects.

2.2. Experiment 2

2.2.1. Subjects

The participants included the same individuals as in Experiment 1.

2.2.2. Stimuli

As in Experiment 1, three sets of nonword stimuli were designed, this time to test whether subjects preserve coda constituents. The three rules to be applied in creating novel nonwords were: (a) add [vi]; (b) add [za]; (c) add [jo]. Training, experimental, and control stimuli are listed in Appendix B. The experimental stimuli in this experiment contained consonant clusters in coda position (e.g., [bild], [wɔst]), rather than in onset position. All other conditions were comparable to those of Experiment 1, but with the manipulations in coda position.

2.2.3. Procedure

Training and testing procedures were the same as in Experiment 1.

3. Results

The data for the two experiments were analyzed together to compare onset and coda preservation. As noted earlier, individuals whose performance on the memory control stimuli was less than 60% accurate were excluded from the experimental analyses. This criterion resulted in the exclusion of two LHD fluent aphasic patients. For the remaining participants, performance on the letter controls was then examined, but not analyzed statistically.

For the normal control subjects, only a single split of a coda (by a single subject) in the letter control stimuli occurred; for all other letter control stimuli, all subjects preserved the two letters together, yielding a single phoneme. In contrast, for the brain-damaged patient groups, more instances of splitting the letter control stimuli occurred, with a total of 35 (of 150 trials) instances for the nonfluent aphasic patients, 54 for the fluent aphasic patients (including all 5 subjects) and 46 for the RHD patients. The relatively high rates of letter splitting across both onset and coda stimuli for the brain-damaged patient groups may have been partially a result of the testing procedure required. Recall that in order for any of the brain-damaged patients to perform the task, we had to modify the original procedure to include both auditory *and written* presentation of the stimuli. The orthographic representation may have misled the patients, yielding more "errors." (Normal controls were tested in the same manner, but were probably less susceptible to possible orthographic biases.)

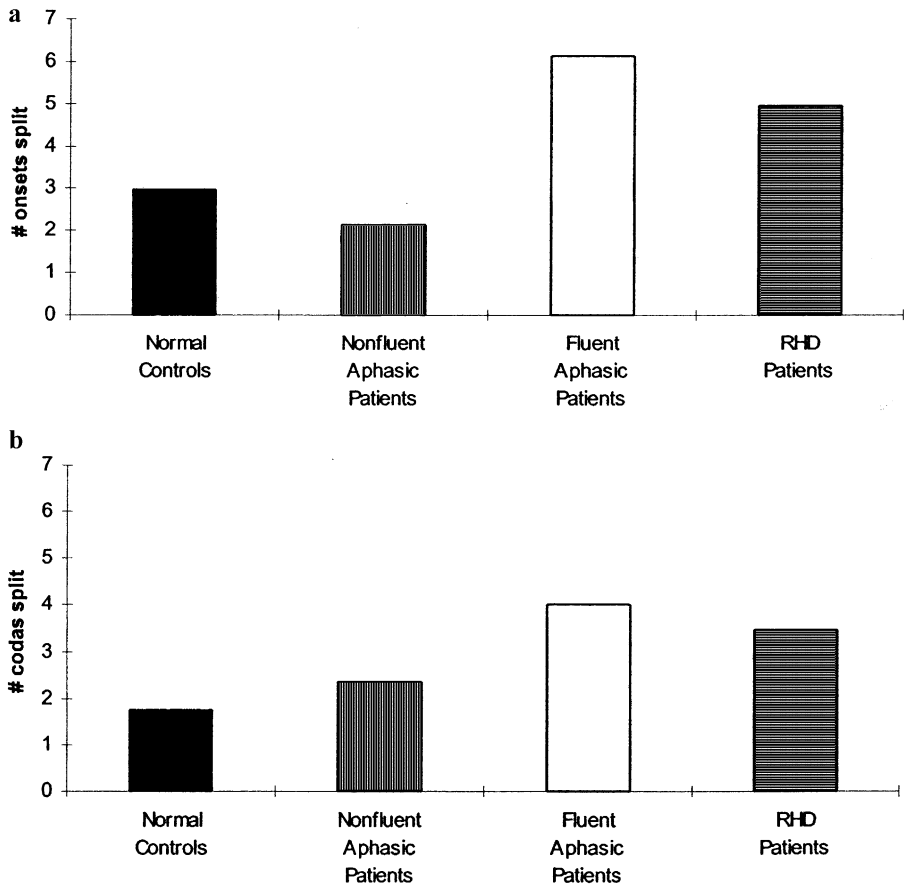

Fig. 1. Mean number of onset (a) and coda (b) stimuli split (maximum possible = 10) by subjects in each group.

The data for the experimental trials are illustrated in Fig. 1, which plots the mean number of onset and coda stimuli that were split by subjects in each group (collapsed across the three specific syllables added in each Experiment). As may be seen, the normal controls produced somewhat more split stimuli than anticipated; however, this pattern is largely the result of two individual subjects who tended to consistently (and surprisingly) split onset (and some coda) stimuli. With these individuals excluded, the mean number of split stimuli drops to 1.57 (out of 10). The nonfluent aphasic patients produced a comparable number of splits of the stimuli (when compared to the entire normal control group). In contrast, the fluent aphasic patients and the RHD patients tended to produce the novel words with more onset and coda splitting (as shown in Fig. 1). Because of the small numbers of participants in each group, statistical power is of potential concern. Nonetheless, a Group × Onset/Coda × Syllable analysis of variance (ANOVA) was conducted on the number of splits produced. The ANOVA yielded main effects of Group ($F(3,20) = 3.295$, $p < .05$) and Onset/Coda ($F(1,20) = 5.413$, $p < .05$). Post hoc analysis of the Group effect using the Newman–Keuls procedure revealed a significant difference only between the normal control group and the fluent aphasic group. No other comparisons reached significance. The main effect of Onset/Coda was due to a larger number of splits in the onset stimuli ($M = 3.67$) relative to the coda stimuli ($M = 2.31$). None of the interactions

reached significance. As noted, however, the statistical results must be interpreted with great caution given the limited numbers of subjects per group. Nevertheless, the patterns within the raw data (largely supported by the statistical analyses) are quite clear, as illustrated in Fig. 1.

4. Discussion

The main objective of the present preliminary investigation was to determine, using a word games paradigm, whether LHD and RHD patients display sensitivity to sublexical, syllable-internal phonological constituents in a manner similar to non-brain-damaged individuals. The findings suggest that, for the most part, both patient groups analyze word forms in keeping with phonological constituent structures. That is, with the exception of a small number of LHD fluent aphasic patients, the brain-damaged patients' performance on the word games task did not differ significantly from that of the normal control subjects. These results are consistent with studies of speech errors in LHD aphasic patients that have demonstrated that paraphasic errors tend to adhere to syllabic constraints and that patients are sensitive to onset and coda constituents as possible units of representation (see Blumstein, 1990 for review). The findings are also in keeping with the demonstrated sensitivity of both LHD and RHD patients to sub-syllabic constituents in rhyme and onset priming tasks (e.g., Baum & Leonard, 2000; Gordon & Baum, 1994).

It is interesting to note that the one group whose performance differed significantly from normal was the fluent aphasic patient group. These participants exhibited a tendency to split onsets as well as codas, suggesting less sensitivity to phonological structure in the fluent aphasic patients, consistent with more frequent phonemic paraphasic errors in speech production, characteristic of this aphasic patient group. However, it must be noted that only three of the five fluent aphasic patients were included in the statistical analysis due to the exclusion of individuals who failed to correctly perform the task with the memory control stimuli. Thus, as mentioned earlier, the findings for the fluent aphasic group must be interpreted with caution. Given the fact that the RHD patients also produced a larger than expected number of splits (see Fig. 1), it is possible that the modification of the experimental procedure required for the patients to complete the task drew on cognitive resources other than phonological processing. Alternatively, and perhaps more likely, the RHD patients may have been influenced by the orthographic representations (as described above) to a greater extent than the individuals with LHD due to their less-impaired language and (presumably) reading skills.

One additional finding is of potential interest—that is, the finding that there were, overall, more splits within the onset stimuli than within the coda stimuli. Close inspection of the experimental stimuli reveals that the clusters within the onsets were mainly composed of stops followed by liquids or glides. In contrast, the coda clusters included a broader range of consonant types including stops followed by fricatives and fricatives followed by stops (see Appendices A and B). It may be that the difference in sonority between the consonants adjacent to the vocalic nucleus (i.e., more sonorous liquids and glides in the onset stimuli relative to the coda stimuli) may have influenced the number of splits produced, yielding more groupings of the liquids and glides with the vowel, split from the initial obstruent in the onset stimuli.[4] However,

[4] Thanks to an anonymous reviewer for pointing this out.

it must be borne in mind that the difference in numbers of splits, while significant, was not of great magnitude. Nonetheless, if this interpretation of the findings is correct, it provides further support for the claim that the subjects were sensitive to phonological form, in this case demonstrating sensitivity to the sonority hierarchy.

In sum, the data collected in the present investigation—while preliminary—suggest that both LHD and RHD patients, like normals, are sensitive to sub-syllabic phonological constituents such as onsets and rimes. These findings add to a growing body of evidence from speech error studies, which indicates that despite sometimes significant impairments in various aspects of phonological processing, the brain-damaged patients remain sensitive to linguistic (in this case syllabic) constraints in speech processing (see also, e.g., Fromkin, 1971; Laubstein, 1987; MacKay, 1970, 1972; Shattuck-Hufnagel, 1983 for data on normal speech errors; and Blumstein, 1973; Gagnon & Schwartz, 1997; Kohn & Smith, 1990 for data on aphasic speech errors).

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Appendix A. Onset

	Add [æz]	Add [of]	Add [ib]
Training stimuli	kigg	zull	zeck
	tep	koot	paff
	nopp	dobb	saol
	boof	mipp	div
Experimental stimuli	kloof	pleece	glun
	kwib	kwudd	drook
	friss	glepp	traff
	skeff	dret	clem
	blesh	klep	brull
	twull	bloog	bligg
	prem	grib	crav
	droog	grith	skoace
	swool	skoom	froosh
	glawsh	broosh	twoff
Letter controls	chep	shull	chig
	chiv	thomm	sheck
	shogue	chev	shapp
	thobb	thook	thoace
	shoon	shibb	chool
Memory controls	tull	bazz	zudd
	mib	gudd	lev
	sim	noog	tav
	foon	pim	voon
	pove	zet	fick

Appendix B. Coda

	Add [vi]	Add [zA]	Add [jo]
Training stimuli	foog	bupp	nepp
	poace	deg	sull
	tawn	voom	rabb
	zight	kib	zim
Experimental stimuli	fict	fids	gemp
	zoalk	lusk	puds
	thoaks	mupps	thailed
	wudds	nense	julp
	bemp	ruld	zant
	villed	thoomp	zaste
	zoaft	kest	voond
	lunz	voned	kevs
	kelm	dift	vits
	fask	shulk	liffs
Letter controls	zoath	besh	futh
	vith	dach	vooth
	koosh	vooch	gich
	gech	luth	zesh
	fash	rish	rooch
Memory controls	kugg	dack	paz
	zawn	koaf	vaught
	ress	tig	regg
	shim	fudd	tuss
	tebb	pev	vape

References

Baum, S. (2002). Consonant and vowel discrimination by brain-damaged individuals: Effects of phonological segmentation. *Journal of Neurolinguistics, 15*, 447–461.

Baum, S. (1997). Phonological, semantic, and mediated priming in aphasia. *Brain and Language, 60*, 347–359.

Baum, S., & Leonard, C. (1999). Automatic versus strategic effects of phonology and orthography on auditory lexical access in brain-damaged patients as a function of interstimulus interval. *Cortex, 35*, 647–660.

Baum, S., & Leonard, C. (2000). The role of sound and spelling in auditory word recognition: Further evidence from brain-damaged patients. *Aphasiology, 14*, 1055–1063.

Berndt, R., Haendiges, A., Mitchum, C., & Wayland, S. (1996). An investigation of nonlexical reading impairments. *Cognitive Neuropsychology, 13*, 763–801.

Blumstein, S. (1973). *A phonological investigation of aphasic speech*. The Hague: Mouton.

Blumstein, S. (1990). Phonological deficits in aphasia: Theoretical perspectives. In A. Caramazza (Ed.), *Cognitive neurospychology and neurolinguistics: Advances in models of cognitive function and impairment*. Hillsdale, NJ: Erlbaum.

Blumstein, S. (1998). Phonological aspects of aphasia. In M. T. Sarno (Ed.), *Acquired Aphasia* (third ed.). New York, NY: Academic Press.

Blumstein, S., Burton, M., Baum, S., Waldstein, R., & Katz, D. (1994). The role of lexical status on the phonetic categorization of speech in aphasia. *Brain and Language, 46*, 181–197.

Blumstein, S., Milberg, W., Brown, T., Hutchinson, A., Kurowski, K., & Burton, M. (2000). The mapping from sound structure to the lexicon in aphasia: Evidence from rhyme and repetition priming. *Brain and Language, 72*, 75–99.

Boyczuk, J., & Baum, S. (1999). The influence of neighborhood density on phonetic categorization in aphasia. *Brain and Language, 67*, 46–70.

Burton, M., Blumstein, S., & Small, S. (2000). The role of segmentation in phonological processing: An fMRI investigation. *Journal of Cognitive Neuroscience, 12*, 1–13.

Caplan, D. (1992). *Language: structure process and disorders*. Cambridge, MA: MIT Press.

Cutler, A., Butterfield, S., & Williams, J. (1987). The perceptual integrity of syllabic onsets. *Journal of Memory and Language, 26*, 406–418.

Finney, S., Protopapas, A., & Eimas, P. (1996). Attentional allocation to syllables in American English. *Journal of Memory and Language, 35*, 893–909.

Folstein, M., Folstein, S., & McHugh, P. (1975). Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Fromkin, V. (1971). The non-anomalous nature of anomalous utterances. *Language, 47*, 27–52.

Gagnon, D., Schwartz, M. (1997). Serial position effects in aphasics' neologisms. Paper Presented at Academy of Aphasia, Philadelphia, PA.

Gauthier, L., Dehaut, F., & Joanette, Y. (1989). The Bells test: A quantitative and qualitative test for visual neglect. *International Journal of Clinical Neuropsychology, 11*, 49–54.

Goodglass, H., & Kaplan, E. (1983). *Assessment of aphasia and related disorders*. Philadelphia, PA: Lea & Febiger.

Gordon, J. (2000). *Aphasic speech errors: Spontaneous and elicited contexts*. Doctoral dissertation, McGill University. Unpublished.

Gordon, J., & Baum, S. (1994). Rhyme priming in aphasia: The role of phonology in lexical access. *Brain and Language, 47*, 661–683.

Hamburger, M. B., & Slowiaczek, L. (1996). Phonological priming reflects lexical competition in auditory word recognition. *Psychonomic Bulletin and Review, 3*, 520–525.

Kohn, S., & Smith, K. (1990). Between-word speech errors in conduction aphasia. *Cognitive Neuropsychology, 7*, 133–156.

Laubstein, A. (1987). Syllable structure: The speech error evidence. *Canadian Journal of Linguistics, 32*, 339–363.

Leonard, C., & Baum, S. (1997). The influence of phonological and orthographic information on auditory lexical access in brain-damaged patients: A preliminary investigation. *Aphasiology, 11*, 1031–1041.

MacKay, D. (1970). Spoonerisms: The structure of errors in the serial order of speech. *Neuropsychologia, 8*, 323–350.

MacKay, D. (1972). The structure of words and syllables: Evidence from errors in speech. *Cognitive Psychology, 3*, 210–227.

Mehler, J., Dommergues, J., Frauenfelder, U., & Segui, J. (1981). The syllable's role in speech segmentation. *Journal of Verbal Learning and Verbal Behavior, 20*, 298–305.

Milberg, W., Blumstein, S., & Dworetzky, B. (1988). Phonological processing and lexical access in aphasia. *Brain and Language, 34*, 279–293.

Norris, D., & Cutler, A. (1985). Juncture detection. *Linguistics, 23*, 689–705.

Pitt, M., Smith, K., & Klein, J. (1998). Syllabic effects in word processing: Evidence from the structural induction paradigm. *Journal of Experimental Psychology: Human Perception and Performance, 24*, 1596–1611.

Shattuck-Hufnagel, S. (1983). Sublexical units and suprasegmental structure in speech production planning. In P. MacNeilage (Ed.), *The production of speech*. New York: Springer.

Slowiaczek, L., McQueen, J., Soltano, E., & Lynch, M. (2000). Phonological representations in prelexical speech processing: Evidence from form-based priming. *Journal of Memory and Language, 43*, 530–560.

Smith, K., & Pitt, M. (1999). Phonological and morphological influences in the syllabification of spoken words. *Journal of Memory and Language, 41*, 199–222.

Treiman, R. (1983). The structure of spoken syllables: Evidence from novel word games. *Cognition, 15*, 49–74.

Treiman, R. (1986). The division between onsets and rimes in English syllables. *Journal of Memory and Language, 25*, 476–491.

Wiig, E., & Secord, W. (1987). *Test of Language Competence—Expanded Edition*. San Antonio, TX: Harcourt Brace Jovanovich.

Zatorre, R., Meyer, E., Gjedde, A., & Evans, A. (1996). PET studies of phonetic processing of speech: Review, replication and reanalysis. *Cerebral Cortex, 6*, 21–30.