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Production of Stress Retraction by Left- and Right-Hemisphere-Damaged Patients

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An acoustic-perceptual investigation of a phonological phenomenon in which stress is retracted in double-stressed words (e.g., *thir**TEEN*** vs ***THIR**teen MEN*) was undertaken to identify the locus of functional impairments in speech prosody. Subjects included left-hemisphere-damaged (LHD) and right-hemisphere-damaged (RHD) patients and nonneurological controls. They were instructed to read sentences containing double-stressed target words in the presence of a clause boundary or its absence. Whereas all three groups of subjects were capable of manipulating the acoustic parameters that signal a shift in stress, there were some differences between the performance of the patient groups and that of the normal controls. Further, stress production deficits were more severe in LHD aphasic patients than in RHD patients. LHD speakers exhibited deficits in the control of both temporal and F_0 cues. Their F_0 disturbance appears to be secondary to a primary deficit in temporal control at the phase or sentence level, as an increased number of continuation rises found for the LHD patients seemed to arise from lengthy pauses within sentences. Findings are highlighted to address the nature of breakdown in speech prosody and the competing views of prosodic lateralization.

Key Words: speech prosody; phonology; English; stress retraction; rhythm rule; speech production; aphasia; left-hemisphere-damaged; right-hemisphere-damaged; lateralization of prosody.

The neural substrates for the processing of speech prosody have been the focus of increasing interest in recent years. A large number of investigations have been conducted with brain-damaged patients in an effort to clarify the nature of breakdowns in prosodic processing subsequent to focal brain damage and, more broadly, to define the neural circuitry underlying the production and perception of prosody (see Baum & Pell, 1999, for review). Prosody is of particular interest because it serves numerous functions in communication, ranging from signaling linguistic stress

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contrasts to providing syntactic cues and expressing emotion. All of these communicative functions are cued by the same set of acoustic parameters, namely duration, fundamental frequency (F_0), and amplitude.

Acoustic investigations of LHD (left-hemisphere-damaged) and RHD (right-hemisphere-damaged) patients' ability to produce lexical and phrasal stress contrasts have generally found a stress deficit in LHD patients only (Emmorey, 1987; Behrens, 1988; Ouellette & Baum, 1994; cf. Bryan, 1989). Instead of characterizing the deficit as a stress deficit, LHD patients appear to have difficulty with the manipulation of durational cues, whereas the other acoustic correlates of stress remain relatively intact (Ouellette & Baum, 1994; cf. Emmorey, 1987). Ouellette and Baum's findings lend support to the hypothesis of differential lateralization for the processing of different acoustic parameters. It is possible that deficits in the processing of linguistic prosody attributed to LHD patients may be a secondary consequence of a more basic impairment in speech timing. This finding is consistent with previous acoustic investigations of other aspects of speech prosody (Danly & Shapiro, 1982; Gandour et al., 1989).

In terms of the production of contrastive or emphatic stress contrasts at the phrase level, most investigations have reported impairments in temporal cue manipulation in LHD patients, but spared production of contrastive stress in RHD patients (Behrens, 1988; Ouellette & Baum, 1994; Pell, 1999a, 1999b). These findings may be interpreted in support of the functional lateralization of prosody, which claims that linguistic prosody is processed via LH mechanisms and emotional prosody via RH mechanisms (Van Lancker, 1980). The results are also partly in agreement with the theory that differential lateralization depends on acoustic cues. Durational parameters are a function of the LH and spectral (specifically F_0) parameters a function of the RH (Van Lancker & Sidtis, 1992; cf. Baum, 1998; Pell & Baum, 1997).

Another stress-related phenomenon that has recently been investigated in brain-damaged patients is that of *stress clash* [also referred to as the *rhythm rule* or *stress retraction* (Grela & Gandour, 1998; Gussenhoven, 1991; Hayes, 1984; Liberman & Prince, 1977)]. In normal speech production in English, there appears to be a definite preference to maintain the alternation of strong and weak syllables to preserve a certain speech rhythm. That is, speakers tend to avoid the production of two successive stressed syllables within a phrase, i.e., a clash context. This is accomplished by shifting the stress in a multisyllabic word away from the second stressed (or strong) syllable or reducing the stress on the initial stressed syllable, thereby averting a stress clash (Grabe & Warren, 1995; Vogel et al., 1995). For example, the word *thirTEEN*, when produced in isolation, bears stress on the final syllable. However, if immediately followed by a word with initial syllable stress (e.g., *WOmen*), the stress on *thirteen* will be shifted to the first syllable (i.e., *THIRteen WOmen*).

Grela and Gandour (1998) explored the ability of LHD fluent and nonfluent aphasic patients and a RHD patient to implement the rhythm rule in contexts in which as many as two steps were required to resolve stress clashes (e.g., *NINEteen BOMbay BOXERS*). Using both acoustic and perceptual analyses, they found that all three patients exhibited impairments relative to a normal speaker in producing the expected stress shift. These findings were interpreted to support the theory that prosodic disorders in patients with brain damage are due to phonetic-motoric deficits rather than an inability to apply a phonological rule. When compared to the normal talker, the LHD nonfluent and RHD talkers produced appropriate rhythmic patterns in short phrases, both experimental and control, but were unsuccessful in longer phrases. If a loss of word-level stress, they would not be expected to produce appropriate rhythmic patterns in the one-word phrases. If a loss of the rhythm rule, then we would have expected appropriate rhythmic patterns across all phrase lengths in the no clash contexts, but abnormal rhythmic patterns in the clash contexts. However, both LHD

nonfluent and RHD talkers produced abnormal rhythmic patterns not only in the longer experimental phrases, but also in the longer control phrases. Similarly, the LHD fluent talker's production of the rhythm rule showed no evidence of a phonological deficit. Although the LHD fluent talker appeared to experience no difficulty in resolving stress clashes in the longer two- and three-word phrases, relative changes in duration between the initial and final syllables were smaller in magnitude than those of the normal talker. Thus, the deficit in all brain-damaged speakers was attributed to an impairment in phonetic implementation rather than a higher level disorder in phonological planning.

In a companion study, Grela and Gandour (1999), the linguistic/prosodic complexity of sentence stimuli was decreased by requiring a one-step stress shift only (e.g., *FOURteen CAKES*). Stress shift items in nonclash contexts occurred as the terminal element of a sentence-internal clause instead, as in Grela and Gandour (1998), at the end of a sentence. Experimental stimuli were composed of sentences where double-stressed target words (*fourteen, nineteen, TV, Chinese*) were presented in the presence and absence of a clause boundary. Results revealed that, despite occasional lack of stress shift on the part of the LHD fluent aphasic and RHD speakers, it was mainly the LHD nonfluent aphasic patient who differed from the normal speaker in terms of both acoustic correlates to stress (notably duration) and perceptibility of stress shifts as judged by three phonetically trained listeners. The authors again concluded that the deficit—this time exhibited only by the LHD nonfluent aphasic patient—was due to an inability to implement the contrast, rather than to impaired underlying knowledge of the rule. Specifically, the LHD nonfluent aphasic patient demonstrated a significant deficit in speech timing, consistent with the earlier literature (Baum, 1992, 1993; Baum & Boyczuk, 1999; Baum & Ryan, 1993; Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980; Blumstein, Cooper, Zurif, & Caramazza, 1977; Cooper, Soares, Nicol, Michelow, & Goloskie, 1984; Gandour, Dechongkit, Ponglorpisit, & Khunadorn, 1994; Gandour, Dechongkit, Ponglorpisit, Khunadorn, & Boongird, 1993; Kent & Rosenbek, 1983; Strand & McNeil, 1996). However, it is noteworthy that the deficit was not *restricted* to temporal parameters (Grela & Gandour, 1999).

More research is required to attempt to tease apart temporal and nontemporal factors that underlie LHD patients' prosodic deficits. Moreover, it is not clear whether the individual cases studied by Grela and Gandour (1998, 1999) are representative of their respective populations. Consequently, the present study was undertaken in an effort to clarify LHD and RHD patients' ability to implement the rhythm rule by including a larger number of patients, stimuli, and listeners.

Based on previous findings and theories of prosodic lateralization, the following predictions may be made. It is anticipated that the LHD patients will exhibit greater difficulty relative to normal speakers in the instantiation of stress shift in clash contexts. At the very least, it is likely that these individuals will display reduced temporal differentiation of the clash and nonclash stimuli (e.g., Baum, 1992, 1993; Baum & Boyczuk, 1999; Baum & Ryan, 1993; Kent & Rosenbek, 1983). If only LHD patients differ from normals, the findings would be in keeping with the functional lateralization of prosody (Van Lancker, 1980). If only temporal parameters are impaired in the LHD patients, the differential lateralization of acoustic cues may be supported, especially if RHD patients exhibit deficits in manipulating F_0 (Van Lancker & Sidtis, 1992). Should both LHD and RHD patients demonstrate similar impairments relative to normal speakers—a less likely outcome—it would suggest that the control of prosody may be diffusely represented in the brain or may be mediated through subcortical mechanisms (e.g., Cancelliere & Kertesz, 1990).

METHODS

Subjects

The subjects included seven LHD aphasic patients, nine RHD patients, and nine age-matched normal controls. All participants were right-handed, native speakers of Canadian English and passed audiometric screening for thresholds <35 dB HL at the speech frequencies (.5, 1, and 2 kHz) in the better ear. All of the brain-damaged patients had suffered a single, unilateral cerebrovascular accident at least 4 months prior to testing, as confirmed by CT or MRI. Patients underwent a battery of screening and diagnostic tests. In addition, the LHD patients were screened for various speech and language skills using subtests of the *Psycholinguistic Assessment of Language* (Caplan, 1992). The RHD patients were screened for communication skills often impaired in this population (e.g., comprehension of inferences and figurative language). A summary of background information on the participants is provided in Table 1.

Materials

Stimuli consisted of locally ambiguous sentence pairs (Table 2). Each pair contained a double-stressed target word (e.g., *fourteen*) which occurred in the presence and absence of a clause boundary. In the

TABLE 1
Background Information on Subjects

Subjects	Sex	Age (years)	Education (years)	Months postonset	Lesion site
Left-hemisphere-damaged patients					
LHD1	M	50	14	126	Left parietal
LHD2	F	81	8	55	N/A
LHD3	F	46	14	81	Left fronto-parietal
LHD4	M	78	9	37	Left frontal
LHD5	F	66	11	37	Left fronto-parietal
LHD6	F	70	12	62	Left parietal
LHD7	F	74	13	6	Left temporo-parietal
	M	66.4	11.6	57.7	
	SD	13.6	2.4	38.2	
Right-hemisphere-damaged patients					
RHD1	M	77	11	11	Right-fronto-temporo-parietal area
RHD2	F	64	13	39	Right internal capsule and basal ganglia
RHD3	M	85	11	17	N/A
RHD4	M	69	12	25	Right parietal
RHD5	F	70	21	30	Right posterior limb of internal capsule and corona radiata
RHD6	F	32	13	36	Right MCA
RHD7	F	57	13	98	Right posterior communicating artery distribution
RHD8	M	68	13	20	Subcortical-right thalamus
RHD9	F	85	5	68	Right MCA
	M	67.4	12.4	38.2	
	SD	16.2	4.1	27.9	
Normal controls					
NC1	M	73	9		
NC2	M	67	9		
NC3	M	65	9		
NC4	M	70	8		
NC5	F	60	10		
NC6	M	68	10		
NC7	F	67	11		
NC8	F	81	8		
NC9	F	29	18		
	M	64.4	10.2		
	SD	14.5	3.1		

TABLE 2
Sentence Stimuli

1.	When eating a **sardine** dinner, I like to drink tea.
	When eating a **sardine,** dinner has a funny smell.
2.	When seeing the **UK** cities, it can be tiring.
	When seeing the **UK,** cities are a popular stop.
3.	When discussing **Taipei** problems, arguments arise.
	When discussing **Taipei,** problems arise.
4.	While watching **TV** soaps, I drink coffee.
	While watching **TV,** soaps are not my favorite.
5.	After eating **fourteen** cakes, he threw up.
	After eating **fourteen,** cakes did not tempt him.
6.	While visiting **nearby** lakes, we often have a picnic.
	While visiting **nearby,** lakes are nice for boating.
7.	After buying **bamboo** furniture, the couple was content.
	After buying **bamboo,** furniture of other kinds had no appeal.
8.	After **nineteen** patients, the dentist smiled.
	After **nineteen,** patients are given the number twenty.
9.	When drinking **champagne** cocktails, I feel tipsy.
	When drinking **champagne,** cocktails make me tipsy.
10.	In **Burmese** words, letters are written with brushes.
	In **Burmese,** words sound funny.

Note. Bold indicates double-stressed target words. Each pair consists of a sentence containing the target word in the absence of a clause boundary, i.e., the clash context (top) and a sentence containing the target word in the absence of a clause boundary, i.e., the nonclash context (bottom).

nonclash context, the double-stressed word appeared immediately before a clause boundary. Stress occurred on the final syllable of the double-stressed word. In the *clash* context, the double-stressed word appeared before a *trigger* word, i.e., a monosysllabic noun which carried primary stress or a polysyllabic noun which carried primary stress on the first syllable. Stress occurred on the initial syllable of the double-stressed word. For purposes of ease of segmentation, all but one of the target words were chosen so that each syllable began with either an obstruent or nasal; all but one of the trigger words began with an obstruent.

Recording Procedure

The stimuli were divided into two sets such that only one version of a particular stimulus (i.e., clash or nonclash context) appeared in each set. The order of presentation of the two sets was counterbalanced across subjects within each group. Five repetitions of each stimulus were elicited in a fixed random order. Stimuli were printed in orthographic form in large font for presentation to the subjects who were instructed to read each aloud. No further specific instructions were provided. If mistakes (unrelated to stress pattern) were made in reading, subjects were permitted to repeat the sentence in its entirety. In all but a few cases, all recording was completed in a single session. Stimuli were recorded onto DAT tape using a Sony TCD-D100 portable recorder and head-mounted directional microphone (AKG Acoustics C420) to maintain a constant microphone-to-mouth distance.

Acoustic Analysis

A total of 2500 (25 talkers & 10 sentences $\times$ 2 stress conditions $\times$ 5 repetitions) tape-recorded utterances were digitized at a sampling rate of 10 kHz by means of a 16-bit A/D converter with a 5-V dynamic range using the Kay CSL (Computerized Speech Lab) 4300B. The low-pass filtering operation was done automatically by Kay CSL during the digitization process to avoid aliasing effects. Duration, voice fundamental frequency (F_0), and rms (root-mean-square) intensity were selected for measurement in the rhyme portion of the target syllables. The rhyme portion has been shown to be a better correlate of stress than either the syllable nucleus or the whole syllable (Sluijter & van Heuven, 1995). Rhyme onset and offset were determined from a simultaneous display of the wideband spectrogram (8-kHz

frequency range and 300-Hz bandwidth) and audio waveform. Those utterances contaminated by voice overlap, background noise, or word omissions were discarded from our corpus (2.7%), resulting in 2433 utterances being retained for acoustic analysis. Acoustic analysis was performed using the Kay Elemetrics CSL (Computerized Speech Lab) 4300 installed on a Gateway 2000 P5/120 computer.

Duration. To measure the duration of the rhyme portion of the target syllable, cursors were positioned on a simultaneous display of an audio waveform and conventional wideband spectrogram. Spectrograms were demarcated in time following conventional rules for segmentation of the speech signal (cf. Klatt, 1976).

Fundamental frequency. The F_0 was computed directly from the waveform using a CSL algorithm that employs a time domain approach to F_0 analysis (modified autocorrelation with center clipping) with nonoverlapping variable frame length. For a given speaker, frame length was determined by his/her F_0 range to ensure that there were at least two complete cycles within a frame. Using the wide-band spectrogram, F_0 onset was defined as the first F_0 value after obstruents that coincided with vertical striations in the second and higher formants; the first F_0 value after a nasal; or in the case of the first syllable of *UK*, the first F_0 value that coincided with syllable onset. The F_0 offset was defined as the last F_0 value preceding the abrupt cessation of the second and higher formants of the vowel or the last F_0 value preceding the sudden offset of a nasal. Mean F_0 was computed from onset to offset for each syllable.

Intensity. Intensity calculation in decibels (dB) was performed on the rhyme portion of the target syllable in a nonoverlapping frame-by-frame, F_0 asynchronous manner using a CSL algorithm which defines intensity as the sum of the absolute amplitude values within a frame. The raw intensity value was converted into decibels by computing 20 times the log (base 10) of the square root of the ratio between the intensity to the number of samples per frame. No smoothing function was applied to the resulting intensity contour. The rms intensity was computed from onset to offset for each syllable.

Perceptual Evaluation

Subjects. Fourteen phonetically trained individuals served as listeners. All were native speakers of English, with no known hearing loss, between the ages of 19 and 40 ($M = 24$).

Stimuli and procedure. Two separate listening tests were prepared for each speaker in each group. A total of 5 repetitions of each sentence in each condition were presented in random order for each test (i.e., a total of 50 stimuli per test). For some speakers (mainly brain-damaged patients), a limited number of utterances was not adequately produced and was thus excluded from the perception tests. Therefore, for some subjects, fewer than 50 stimuli per test were presented.

The stimuli for each speaker were split into two lists to reduce repetition and to avoid presenting both clash and nonclash contexts for the same sentence in the same test. Each listener attended between four and five sessions over a 2- to 3-week period to complete the perception tests for the 25 speakers. Stimuli for approximately 6 speakers were presented in each 1-h session. Order of listening tests (i.e., speakers) was randomized across listeners.

Stimuli were presented over closed headphones at a comfortable level using the Kay Elemetrics CSL system. The listeners' task was to circle on a response sheet the stress pattern transcription (indicated by bold print) corresponding to the stimulus presented. Individual sets of 15 practice stimuli were prepared for each speaker to accustom listeners to the task and the speaker's speech patterns. The actual test sentences were presented multiple times, as controlled by the listener, until (s)he was confident of his/her judgment. Although use of this procedure yields different numbers of stimulus presentations across items and listeners, it was decided upon as the method which would yield the most accurate stress judgments, particularly for production by brain-damaged speakers. If anything, permitting repeated stimulus presentations decreases the likelihood of finding differences across groups—our predicted outcome.

Data Analysis

Acoustic correlates. Because of absolute differences in speaking rate, loudness, and pitch between subjects, ratios were chosen to measure acoustic correlates of stress shift for individual subjects. The syllable duration ratio measured timing of the rhyme portion of the final to the initial syllable of the target word for each utterance. A pause duration was defined as the silent interval between the offset of the target word and the onset of the trigger word. The pause duration ratio measured timing of the silent interval of clash utterances to nonclash utterances for clash and nonclash utterances paired at random.

The intensity ratio measured rms intensity of the rhyme portion of the final to the initial syllable of the target word relative to the peak intensity of the target word for each utterance.

The F_0 ratio measured mean F_0 of the rhyme portion of the final to the initial syllable of the target

word for each utterance. In addition, a dynamic property of F_0 was obtained by calculating the proportion of rises in F_0 during the last 50% of the final syllable of the target word in clash and nonclash utterances separately. A rise in F_0 was defined to be equal to or greater than 10 Hz. Proportions were defined relative to each speaker's total number of productions for each sentence in a clash context and nonclash context.

Perceptual evaluation. To evaluate a speaker's ability to shift stress in clash and nonclash contexts, each of the 2433 target word productions was assigned a Final Syllable Stress score (FSS = % of final stress responses for the target word). FSS values ranged from 0 to 100. If a target word was heard as stressed on its final syllable by all 14 listeners, an FSS score of 100 was assigned to that particular production; if 7 listeners heard the final syllable as stressed, an FSS score of 50 was assigned; if no listeners heard the final syllable as stressed, an FSS score of 0 was assigned (cf. Vogel et al., 1995, p. 120).

The kappa coefficients (Cohen, 1960) computed for each pair of listeners indicated moderately strong agreement between listeners in their stress judgments. About 70% of the pairwise coefficients were larger than 0.69 for the NC group ($M = 0.75$, $SD = 0.9$), larger than 0.48 for the RHD group ($M = 0.54$, $SD = 0.13$); and larger than 0.42 for the LHD group ($M = 0.47$, $SD = 0.9$).

RESULTS

Perceptual Evaluation

Pooled across listeners and sentence pairs, a two-way (group × stress context) analysis of variance (ANOVA) on arcsine-transformed FSS scores yielded a significant two-way interaction [$F(2, 22) = 9.80$, $p < .0009$]. Tukey–Kramer multiple comparisons ($\propto = .01$) revealed that FSS scores were significantly different between the clash and nonclash contexts for all three groups. This means that the NC, RHD, and LHD groups were able to signal successfully the stress contrast between clash and nonclash contexts (Fig. 1). In the nonclash context, however, FSS scores of both the LHD and RHD groups were significantly smaller than that of the NC group, indicating that brain-damaged speakers had difficulty in signaling stress in the presence of a clause boundary. In the clash context, the FSS score of the LHD group was significantly larger than that of the NC group, meaning that LHD aphasics also had difficulty in signaling stress in the absence of a clause boundary. No other differences in FSS scores between groups reached significance in the clash context. Espe-

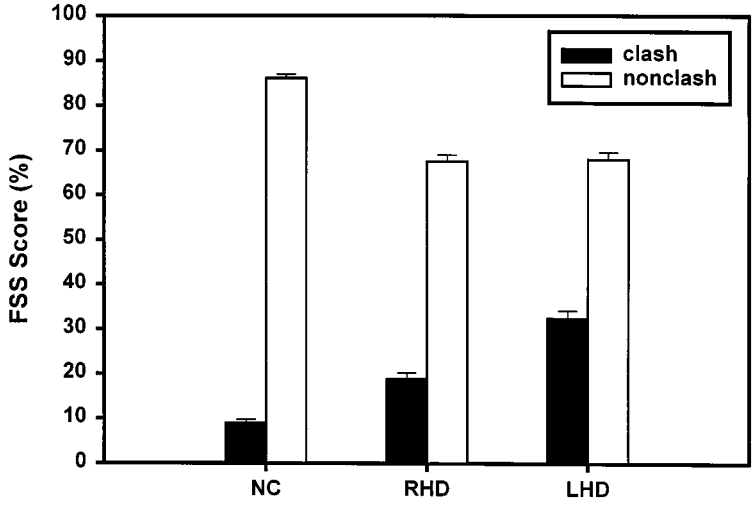

FIG. 1. Average FSS (final syllable stress) scores for target words produced in clash and nonclash contexts by normal control (NC), right-hemisphere-damaged (RHD), and left-hemisphere-damaged (LHD) subjects. Error bars = ±1 *SE*.

cially noteworthy is the absence of any significant differences in FSS scores between the LHD and RHD groups in either stress context.

Acoustic Analysis

Syllable duration. Pooling across sentence pairs, a two-way (group × stress context) ANOVA on log-transformed syllable duration ratios yielded a significant two-way interaction [$F(2, 22) = 8.12$, $p < .0023$]. Tukey–Kramer multiple comparisons ($\propto = .01$) revealed that syllable duration ratios were significantly different between the clash and nonclash contexts for the NC and RHD groups, but not for the LHD group (Fig. 2). This means that intraword timing cues were vulnerable to disruption in the LHD group.

A two-way (group × sentence pair) ANOVA on log-transformed pause duration ratios indicated no significant main group effect [$F(2, 22) = 2.18$, $p < .1373$] or

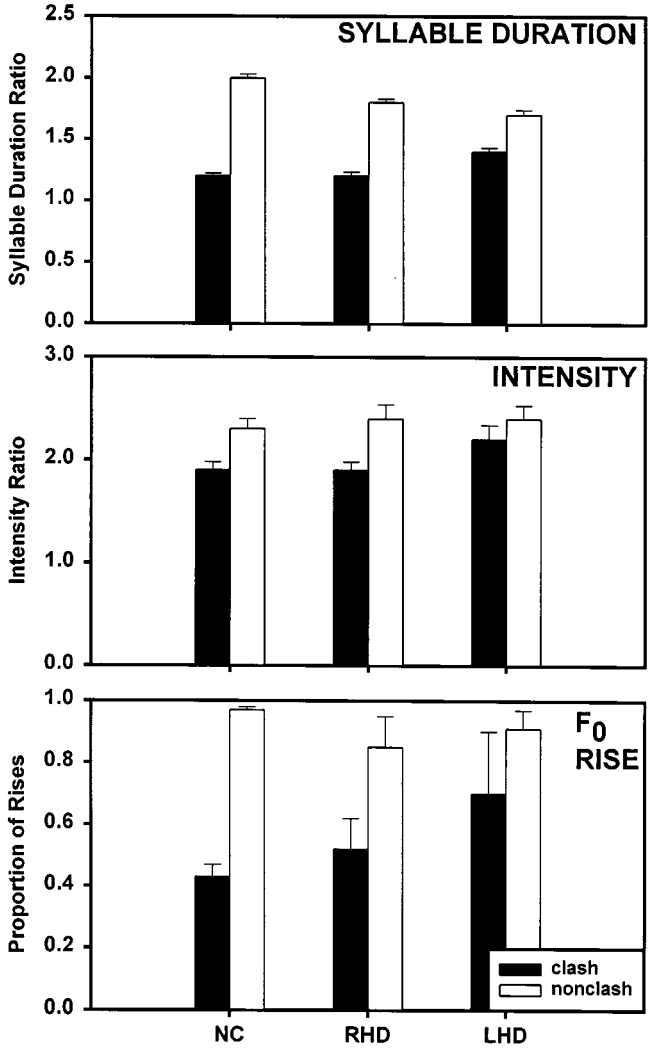

FIG. 2. Comparison of average syllable duration (top panel), intensity (middle panel), and F_0 (bottom panel) ratios for target words produced in clash and nonclash contexts by normal control (NC), right-hemisphere-damaged (RHD), and left-hemisphere-damaged (LHD) subjects. Error bars = ±1 *SE.*

two-way interaction [$F(2, 198) = 1.15$, $p < .3076$]. Pooling across sentence pairs, average (*SD*) pause duration ratios for the NC, RHD, and LHD groups, in order, were 0.64 (0.14), 0.72 (0.12), and 0.78 (0.09). Regardless of group, pauses were longer between the target word and trigger word in the nonclash context. Despite intraword timing disturbances, the LHD group managed to control interword timing, i.e., the duration of the silent interval between the target word and the trigger word.

Fundamental frequency. A two-way (group × stress context) ANOVA on mean F_0 ratios of the rhyme portion of the final to the initial syllable of the target word revealed no significant main group [$F(2, 22) = 0.48$, $p < .6274$] or stress context effects [$F(1, 22) = 0.31$, $p < .5851$] or two-way interaction [$F(2, 22) = 1.04$, $p < .3689$]. Average mean F_0 ratios for the NC, RHD, and LHD groups, in order, were 0.95, 0.96, and 0.95 in the clash context and 1.01, 0.95, and 0.94 in the nonclash context. That is, changes in mean F_0 between the initial and final syllable of the target word did not vary systematically as a function of group or stress context.

However, a two-way ANOVA on arcsine-transformed proportions of F_0 rises in the final syllable of the target word showed a significant two-way interaction between group and stress context [$F(2, 22) = 4.72$, $p < .0198$]. Tukey–Kramer multiple comparisons ($\propto = .01$) indicated that the proportion of rises in the final syllable was significantly greater in the nonclash than in the clash contexts for the NC and RHD groups only (Fig. 2). Whereas no group differences emerged in the nonclash context, the LHD group showed a significantly greater proportion of rises in the clash context when compared to the NC group. No other group differences reached significance in the clash context.

Intensity. A two-way (group × stress context) ANOVA on intensity ratios yielded a significant main effect of stress context only [$F(1, 22) = 9.74$, $p < .0050$]. Neither the group main effect [$F(2, 22) = 0.33$, $p < .7207$] nor the interaction between group and stress context [$F(2, 22) = 3.93$, $p < .6947$] reached significance. For all three groups (NC, RHD, and LHD), changes in intensity between the initial and final syllable of the target word varied systematically depending on stress context (Fig. 2). Intensity was greater on the final syllable in the nonclash context than in the clash context.

Total sentence duration. A three-way (group × sentence pair × stress context) ANOVA on total sentence duration revealed a main group effect [$F(2, 22) = 11.17$, $p < .0004$] and a significant two-way interaction between group and sentence pair [$F(18, 199) = 5.08$, $p < .0001$]. For clash and nonclash contexts separately, Student–Newman–Keuls multiple comparisons ($\propto = .01$) showed that LHD speakers were significantly slower than RHD and NC speakers for all sentence pairs. RHD speakers, on the other hand, were significantly slower than NC speakers in their production of *nearby* in the clash context only. Pooling across stress context and sentence pairs, average sentence durations for the NC, RHD, and LHD groups, in order, were 3.08, 3.23, and 6.12 s. Thus, the LHD group's sentence productions were about twice as long as those produced by the NC and RHD groups.

DISCUSSION

In agreement with results reported by Grela and Gandour (1998, 1999), our findings demonstrated that the LHD aphasic patients exhibited impairments in the phonetic implementation of the rhythm rule. The LHD patients produced fewer and less consistent acoustic cues, signaling a shift in stress relative to the normal controls. It is noteworthy that deficits emerged for both temporal and F_0 parameters—a somewhat

unusual finding (see Baum & Pell, 1999 for review). The reduced acoustic differentiation resulted in less perceptible cues to stress for the productions of the LHD patients. In contrast, results for the RHD speakers, albeit not exactly equivalent to normal, reflected fewer significant impairments in signaling stress shifts (Grela & Gandour, 1999; cf. Grela & Gandour, 1998).

Results of the acoustic analyses indicated that, as expected, normal speakers consistently shift stress in a clash context (i.e., in the absence of a clause boundary), as reflected in syllable duration, F_0, and intensity (Grabe & Warren, 1995; Vogel et al., 1995). In addition, the clause boundary, when present, was signaled by an increase in pause duration between the target and trigger words (Cooper, Paccia, & Lapointe, 1978; Price, Ostendorf, Shattuck-Hufnagel, & Fong, 1991; Scott, 1982; Streeter, 1978). Results for the RHD speakers were similar to those of the non-brain-damaged individuals. This pattern contrasts with that reported for a single RHD speaker in Grela and Gandour (1998), but is consistent with that found for the same RHD speaker in Grela and Gandour (1999). We can only speculate that linguistic/prosodic complexity underlies the disparity in results between the two studies for the RHD speaker. In Grela and Gandour (1998), stress shift required as many as two steps to resolve stress clashes, whereas only one step was required in Grela and Gandour (1999), the same as in the present study.

Whereas the LHD speakers in the present investigation manipulated all of the acoustic parameters measured to signal stress shifts in the appropriate contexts, their productions differed from both the RHD patients and normal controls in a number of important ways. In agreement with numerous previous studies (e.g., Baum, 1993; Gandour et al., 1994; Kent & Rosenbek, 1983; McNeil et al., 1987; Strand & McNeil, 1996), the speaking rate of the LHD patients was dramatically slower than that of both other groups. When controlled for speaking rate differences, the LHD speakers produced relatively normal pauses signaling clause boundaries (see also Baum et al., 1997; Baum et al., 2001), supporting claims that the timing impairment is not pervasive (e.g., Blumstein, 1998). However, syllable duration cues to stress were less clear in the productions of the LHD speakers (see also Ouellette & Baum, 1994), consistent with their oft-cited impairment in temporal control, particularly within words (e.g., Baum, 1992; Baum & Boyczuk, 1999; Gandour et al., 1993).

Of particular interest was the finding that the LHD speakers exhibited deficits not only in the production of certain temporal cues, but also in the control of F_0. The LHD speakers unexpectedly produced a greater number of F_0 rises in the clash context. One might argue that the LHD speakers do not successfully control F_0 to signal word-level stress; however, the majority of previous data suggest otherwise (e.g., Emmorey, 1987; Ouellette & Baum, 1994). An alternative, more plausible, explanation is that the unexpected F_0 rises occur secondary to a primary deficit in temporal control at the phrase or sentence level. That is, due to their extremely slow speaking rate and lengthy pauses (e.g., Kent & Rosenbek, 1983), the LHD speakers may produce an increased number of continuation rises as a signal that their utterance is incomplete (cf. Cooper et al., 1984; Danly & Shapiro, 1982; Danly, Cooper, & Shapiro, 1983; Kent & Rosenbek, 1982). The continuation rise serves to maintain the coherence of the sentence despite lengthy and sometimes inopportune pauses between words. The rising F_0 contour on the second syllable, albeit an effective means of preserving sentence coherence, severely compromises stress cues for double-stressed words. That is, an increase in F_0 as a signal of stress placement on the first syllable of a double-stressed word in a clash context may be overridden by an F_0 increase on the second syllable, utilized inappropriately as a continuation rise. These aberrant F_0 patterns in LHD speakers were revealed only through a time-varying measure of F_0, thus high-

lighting the importance of such measures of prosody when interpreting underlying causes of prosodic disturbances in brain-damaged patients (cf. Gandour, Petty, & Dardarananda, 1989).

With respect to the perceptual evaluations, our findings largely support the acoustic data, with the most consistent and accurate perception for the normal speakers' utterances and the worst, albeit better than chance, performance for the utterances produced by the LHD speakers. It is important to note that the perceptual data, as is true of the acoustic data as well, indicate that all three groups of speakers did produce perceptible cues reflecting appropriate stress shifts in clash contexts. The LHD speakers were simply less successful in cueing the stress shifts due to deficits in phonetic implementation (Grela & Gandour, 1998, 1999).

The current findings are not entirely consistent with any of the current theories of the neural bases of prosody. Contrary to the theory of differential lateralization of acoustic parameters (Van Lancker & Sidtis, 1992), the LHD patients displayed impairments in the control of *both* temporal and spectral properties, whereas the RHD patients exhibited relatively few deficits in either property. Neither are the present results entirely congruent with the theory of functional lateralization of prosody (Van Lancker, 1980). Although stress clash is clearly a linguistic, as opposed to emotional, prosodic distinction, LHD patients, albeit impaired in the implementation of certain acoustic cues, were still capable of signaling stress shift. These experimental outcomes are consistent with a large number of previous studies that have demonstrated prosodic impairments in some but not all linguistic functions of prosody in LHD speakers (see Baum & Pell, 1999, for review). The present results, however, unequivocally point to a specific deficit in the control of temporal parameters by LHD speakers—a deficit that, as noted above, probably underlies the unexpected rising F_0 patterns as well.

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