

# THE TEMPORAL ORGANIZATION OF AFFECTIVE AND NON-AFFECTIVE SPEECH IN PATIENTS WITH RIGHT-HEMISPHERE INFARCTS

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## ABSTRACT

To evaluate the right hemisphere's role in encoding speech prosody, an acoustic investigation of timing characteristics was undertaken in speakers with and without focal right-hemisphere damage (RHD) following cerebrovascular accident. Utterances varying along different prosodic dimensions (emphasis, emotion) were elicited from each speaker using a story completion paradigm, and measures of utterance rate and vowel duration were computed. Results demonstrated parallelism in how RHD and healthy individuals encoded the temporal correlates of emphasis in most experimental conditions. Differences in how RHD speakers employed temporal cues to specify some aspects of prosodic meaning (especially emotional content) were observed and corresponded to a reduction in the *perceptibility* of prosodic meanings when conveyed by the RHD speakers. Findings indicate that RHD individuals are most disturbed when expressing prosodic representations that vary in a graded (rather than categorical) manner in the speech signal (Blonder, Pickering, Heath et al., 1995; Pell, 1999a).

Key words: brain damage, right hemisphere, prosody, speech timing, emotion

## BACKGROUND

In his seminal report on a woman with left fronto-temporo-parietal insult resulting in altered "melody of language", Monrad-Krohn (1947) was the first to focus specifically on abnormalities in the *prosodic* quality of his patient's speech, a native speaker of East Norwegian. By the term 'prosodic quality of speech', the author was referring to those alterations in pitch, stress, and rhythm (including pauses and prolongations) that specify meaning at levels of linguistic structure above the segment, such as word stress or pitch contours. Following his patient's brain damage, the inability to modulate pitch appropriately to express a number of linguistic (but not musical) forms was interpreted as evidence of a selective disruption of the prosodic faculty, or 'dysprosody' (Monrad-Krohn, 1947). Since that time, empirical data have confirmed the idea that prosodic attributes of speech are susceptible to selective disruption following acquired brain injury, and therefore, distinct in some way from other aspects of the language code (Baum and Pell, 1997; Baum, Pell, Leonard et al., 1997; Behrens, 1988, 1989; Blonder et al., 1995; Breitenstein, Daum and Ackermann, 1998; Cancelliere and Kertesz, 1990; Cooper, Soares, Nicol et al.,

1984; Danly, Cooper and Shapiro, 1983; Danly and Shapiro, 1982; Emmorey, 1987; Gandour, Larsen, Dechongkit et al., 1995; Gandour et al., 1992c; Kent and Rosenbek, 1982; Klouda, Robin, Graff-Radford et al., 1988; Kurowski, Blumstein and Alexander, 1996; Ouellette and Baum, 1993; Pell, 1998; Pell and Baum, 1997a, 1997b; Ross, Thompson and Yenkosky, 1997; Ryalls, 1982; Shapiro and Danly, 1985; Weintraub, Mesulam and Kramer, 1981).

Despite consensus that cerebral (especially cortical) dysfunction can adversely affect the formulation and evaluation of speech prosody, the specific impact of lesions confined to left or right hemisphere regions on prosodic functions remains opaque. This lack of clarity likely reflects the multifaceted nature of prosody which relies on a complex of (currently under-specified) sensory-motor and cognitive mechanisms, coupled with the possibility that distinct patterns of hemispheric sensitivity characterize at least some of these different facets of prosodic behaviour. For instance, there is now considerable support for the notion that the *functional* significance of prosodic cues is in some way an important determinant of hemispheric involvement in speech production and perception (Borod et al., 1990; Emmorey, 1987; Gandour, Larsen, Dechongkit et al., 1995; Gandour, Ponglorpisit, Khumadorn et al., 1992c; Heilman, Bowers, Speedie et al., 1984; Pell, 1998; Pell and Baum, 1997a; Perkins, Baran and Gandour, 1996; Ross, 1981; Tompkins and Flowers, 1985; Tucker, Watson and Heilman, 1977). Most commonly, mechanisms of the right hemisphere have been viewed as critical in regulating affective components of the speech stream, whereas left hemisphere mechanisms are most active when a linguistic analysis of prosodic features is necessary (Van Lancker, 1980). Alternatively, explanations that emphasize the influence of specific perceptual-acoustic parameters in biasing hemispheric activation on prosody tasks have been formulated (Robin, Tranel and Damasio, 1990; Van Lancker and Sidtis, 1992). These hypothetical descriptions, which converge with a sizable literature on nonlinguistic auditory capabilities (e.g., Carmon and Nachshon, 1971; Sidtis, 1980; Sidtis and Volpe, 1988; Zatorre, Evans, Meyer et al., 1992), ascribe a superior right hemisphere processing substrate for pitch attributes of intonation contours and a left hemisphere processing advantage for temporal parameters of the same stimuli (Cooper, 1981; Robin et al., 1990; Van Lancker and Sidtis, 1992; cf. Gandour, Wong and Hutchins, 1998; Pell and Baum, 1997b). Despite their value in illuminating some of the component processes of prosodic behaviour, neither the “functional” nor “cue-based” approach has led to a unitary description of how the brain subserves prosody production or comprehension.

Of the two primary acoustic parameters that encode prosodic meaning (e.g. Bolinger, 1986), duration (or speech timing) has received somewhat less attention than fundamental frequency ( $F_0$ ) where the domain of interest is larger than the individual segment. Nonetheless, it is well established that changes in duration contribute important cues to phonemic and emphatic stress placement (Eady and Cooper, 1986; Ferreira, 1993; Klatt, 1976; Morton and Jassem, 1965), to the location of syntactic phrase boundaries (e.g., Beach, 1991; Lehiste, Olive and Streeter, 1976), and to a speaker’s emotional disposition while producing the utterance (Huttar, 1968; Lieberman and Michaels, 1962; Williams and Stevens, 1972). Problems implementing the temporal correlates of linguistic and emotional prosody following

unilateral brain lesion have been observed on several occasions. For example, Cooper and colleagues (1984) found that both RHD and LHD patients displayed timing irregularities when required to read emotionally-neutral sentences of varying complexity (utterance duration and individual clause/word durations were computed), but that this effect was far greater in the LHD sample. Impairments in signalling phonemic and emphatic stress contrasts via duration have also been uncovered, again primarily in the LHD population (Emmorey, 1987; Ouellette and Baum, 1993; cf. Vijayan and Gandour, 1997).

The opinion that right hemisphere dysfunction minimally affects the timing of sentence prosody – at least when elicited in a non-affective context – is further complemented by data derived from both case and group analyses of RHD speakers (Baum et al., 1997; Behrens, 1988; Blonder et al., 1995; Emmorey, 1987; Klouda et al., 1988; Ouellette and Baum, 1993; Ryalls, Joannette and Feldman, 1987), although not all (Dykstra, Gandour and Stark, 1995; Grela and Gandour, 1998; Kent and Rosenbek, 1982; Vijayan and Gandour, 1997). Notably, Gandour and colleagues (Gandour et al., 1994; Gandour, Dechongkit, Ponglorpisit et al., 1993; Gandour et al., 1995; Gandour et al., 1992a; Gandour et al., 1992b; Gandour et al., 1992c) systematically explored speech timing characteristics of brain-damaged speakers of Thai, probing a number of different levels of linguistic structure (e.g., individual segments, words, sentences) and eliciting both linguistic- and emotional-prosodic stimuli. In general, RHD speakers of Thai displayed little difficulty encoding duration as a linguistic device (subtle deficits did emerge for some larger-sized linguistic units – see Gandour et al., 1993), whereas comparable LHD speakers exhibited temporal abnormalities at all levels of linguistic structure (segment, word, and sentence levels). The temporal correlates of *emotional* distinctions were also relatively preserved in the RHD speakers based on a single report (LHD patients were excluded from the study of emotional prosody – see Gandour et al., 1995). Collectively, the authors' findings imply that timing deficits, when they appear in RHD speakers, tend to be of a more subtle nature than those witnessed in LHD aphasic speakers under most experimental conditions (Gandour et al., 1993; Kent and Rosenbek, 1982).

The subtlety of many speech timing deficits, and the risk that prevailing deficits in timing may not have been highlighted in past reports (interestingly, pitch irregularities originally described by Monrad-Krohn in his patient may have been secondary to a more basic disturbance of timing – see Moen, 1991), warrant renewed investigation of the temporal structure of speech following unilateral brain damage. In particular, the lack of clarity concerning the right hemisphere's involvement in timing operations is addressed in the present study. In addition, novel attempts are made to illuminate the demands of regulating duration to signal linguistic- and emotional-prosodic distinctions *in tandem* (certainly, a natural feature of speech production) in an invariable sample of right-brain-damaged speakers. This approach could prove useful in informing previous hypotheses about the neural mechanisms that govern vocal signals (Ross, Harney, deLacoste-Utamsing et al., 1981; Van Lancker, 1980; Van Lancker and Sidtis, 1992). Current findings may also speak to recent proposals that ascribe a right-hemisphere advantage for prosodic cues that operate in a graded rather than categorical manner in the speech signal (Blonder et al., 1995; Pell, 1999a).

## MATERIALS AND METHODS

*Subjects*

Experimental subjects consisted of 10 individuals with unilateral right-hemisphere damage and 10 healthy control (NC) subjects without neurological dysfunction. Recruitment was restricted to dextral adults who were native speakers of Canadian English without a premorbid history of neurologic or psychiatric illness, or substance abuse<sup>1</sup>. All subjects displayed acceptable hearing acuity following a pure-tone air-conduction screening of both ears (inclusion criteria: 30 dB HL at .5, 1 and 2 kHz, for the better ear). The RHD and NC groups were balanced for mean age in years (RHD = 64.3; NC = 66.1) and were closely balanced for gender.

Clinical subjects were tested following a period of rehabilitation after suffering an occlusive event in the distribution of the right middle cerebral artery. Lesion information was documented by CT scan and derived from radiologic reports in patients' medical records. Bilateral or brainstem lesions, or evidence of a condition associated with cognitive decline (e.g., Parkinson's disease) served as grounds for exclusion from the study. Patients with comparable left-hemisphere lesions were excluded from initial analyses owing to the complexity of the linguistic materials utilized in the current study (Gandour et al., 1995; Pell, 1999a). Aphasic or dysarthric deficits were not present in any of the RHD patients as determined by an aphasia screening and through clinical observation. A persistent hemispatial visual neglect was identified in four RHD patients following administration of the Bells Test (Gauthier, Dehaut and Joannette, 1989). A brief evaluation of discourse inferencing abilities, figurative language comprehension, and emotional prosody discrimination and recognition was also conducted; deficits on these subtests (where observed) are reported in Table I, along with other defining features of the RHD participants. Testing was always conducted at least 3 months post-onset of stroke (mean = 36 months, range = 7-83) to ensure stability in the performance measures.

TABLE I  
*Demographic and Clinical Attributes of the RHD Patients*

Subject	Sex/ Age	Post- onset	Lesion	Major clinical signs	VN
R1	F/54	5.5	(R) posterior	(L) hemiparesis, "flat affect"	-
R2	M/59	2.6	(R) temporo-parietal	(L) hemiparesis, "inappropriate mood", impaired inferencing, comp. of figurative language	+
R3	M/74	4.0	(R) parietal	(L) hemiparesis, impaired comp. of figurative language	-
R4	F/61	0.7	(R) basal ganglia	(L) hemiparesis, "flat affect"	-
R5	F/29	0.6	(R) MCA	(L) hemiplegia	-
R6	F/82	3.3	(R) temporal	(L) hemiplegia, impaired recognition of emotional prosody	+
R7	M/69	4.9	(R) temporo-parietal	(L) hemiparesis, impaired recognition of emotional prosody	-
R8	F/87	6.9	(R) MCA	(L) hemiparesis, impaired inferencing/comp. of figurative language	-
R9	F/62	0.8	(R) external capsule	(L) hemiparesis, "flat affect", impaired inferencing/comp. of figurative language/emotional prosody recognition	+
R10	F/66	1.0	(R) fronto-parietal	(L) hemiparesis, impaired comp. of figurative language	+

Note: Age, post-onset = years, VN = hemispatial visual neglect.

<sup>1</sup>Although functionally right-handed for most activities, subject R8 reported a preference to use the left hand as a child. However, the absence of aphasic signs following acute right MCA infarction, coupled with positive evidence of what many consider to be "right hemisphere signs" (review Table 1), suggested that it was acceptable to enter this patient into the study.

### Stimuli

Following research on normal prosodic characteristics (Cooper, Eady and Mueller, 1985; Eady and Cooper, 1986), two short sentences served as base stimuli (*Mary sold the teapot/Barry took the sailboat*). Each target sentence was composed of high frequency content words matched for syllable length and stress location between the stimuli. Furthermore, when elicited from experimental subjects, each stimulus was constructed to facilitate multiple variations in the prosodic form of the utterance as a function of three prosodic variables: sentential location of emphatic stress (initial, final, none); linguistic “modality” (statement, question); and emotional tone (sad, happy, angry, neutral). To explore the potential interaction of these forms on temporal encoding, an exhaustive combination of the three prosodic variables was elicited from each subject, culminating in 24 distinct readings per stimulus (or 48 per speaker). It is important that each target utterance represented a unique combination of the three prosodic attributes *without* altering the influence of segmental/semantic content across utterances. “Neutral” semantic content was adopted that did not suggest a particular linguistic or affective interpretation, forcing subjects to signal these distinctions solely through the prosodic contour of the utterance.

To systematically bias subjects’ reading of the stimuli (thereby ensuring that specific prosodic “targets” were met), a story completion paradigm was utilized (e.g., Behrens, 1989; Eady and Cooper, 1986). Short passages were prepared to complement each trial, influencing the manner in which subjects read the target sentence. For example, the scenario preceding a neutral, declarative reading of *Mary sold the teapot* that lacked sentence focus (i.e., [no focus, declarative, neutral]) was the following:

*You are holding a garage sale at your house with the help of some friends. After the sale, someone tells you that Mary sold the teapot. When another friend asks you what happened, you say:* [MARY SOLD THE TEAPOT]

Trials differing in emphasis placement, sentence modality, and emotional tone were biased by modifying the corresponding priming scenario (see Pell, 1999a, for details of this procedure). Cue cards containing the target utterance and information reinforcing how the sentence was to be read (e.g., emphasized words were written in bold italics) further prompted the desired reading of each trial. All scenarios were pre-recorded by a male speaker in a sound-attenuated chamber using a high-quality Sony tape recorder and a Sony ECM-909 directional microphone. The speaker was encouraged to produce the passages in a “reporting” tone that did not lend undue prominence to specific lexical items within the passage. Scenarios were subsequently randomized for order of presentation separated by a five second inter-stimulus pause to allow subjects adequate time to initiate a response during the experiment.

### Procedure

Subjects were tested individually in their home (RHD patients) or in a laboratory setting (control subjects). Two separate 30 minute sessions were conducted to ensure that subjects remained alert and attentive to the content of priming scenarios. Each subject was seated comfortably at a table with a directional microphone (Sony ECM-909) placed 20 centimeters in front of his or her mouth. A small binder containing the stimulus cards corresponding to each trial was placed on the table in front of them. The experimenter encouraged subjects to listen closely to the priming “stories” (presented free-field) and then complete the story by reading the card placed in front of them. The examiner was permitted to direct the subject’s attention to the written display, although this was seldom necessary owing to the amount of redundancy in the auditory priming scenarios and the target utterances. Reading errors or dysfluencies were followed by a request to repeat the sentence. At any time, subjects were free to repeat a response when not totally satisfied with their performance, although such “corrections” occurred highly infrequently (the final token produced for a given trial was always considered in subsequent analyses). Five practice trials acquainted subjects with the experimental procedure, and despite occasional comments that priming scenarios were repetitious, neither the NC nor the RHD subjects exhibited any difficulty in

performing the task. All responses were recorded by a Sony (TCD-D3) digital audio tape recorder for later analysis.

### *Acoustic Analyses*

Target productions were digitized using the BLISS speech analysis system (Mertus, 1989) at a sampling rate of 20 kHz, with a 9 kHz low-pass filter setting and 12-bit quantization. The 48 utterances generated for each speaker were then acoustically analyzed using the BLISS waveform editor. The modulation of  $F_0$  parameters of the stimuli reported herein, elicited from the same patient sample, was considered in a companion report (Pell, 1999a.) In the present study, the following duration measures were derived to capture each subject's ability to encode the various distinctions of interest in the stimuli:

#### (1) *Keyword Duration*

The duration of "keywords" within each utterance was computed to chart point-by-point transitions in syllable duration for the NC and RHD speakers (Cooper et al., 1985; Eady and Cooper, 1986; Eady, Cooper, Klouda et al., 1986). The duration of the full vowel (transition and steady state) was measured on the stressed syllable of each content word (i.e., *Ma-ry*, *sold*, *tea-pot*). (Consonants have generally been assigned minimal importance in prosodic highlighting, e.g., Brown and McGlone, 1974; Klatt, 1976.) Through both visual and auditory analysis of the oscillographic display, boundaries were demarcated by placing cursors at zero crossings at the onset and offset of periodicity corresponding to the vowel. The duration (in milliseconds) between the cursors was then recorded.

#### (2) *Speech Rate*

The speaking rate of each utterance (a cue considered especially pertinent to emotional communication, Scherer, 1986) was calculated as the number of syllables present divided by the total sentence duration (in milliseconds).

### *Statistical Analyses*

Keyword duration measures were normalized prior to statistical inspection to adjust for inter-speaker differences in speaking rate; this was accomplished by dividing each keyword value by the corresponding utterance duration, for each speaker. Data derived for the two test items were collapsed within each condition prior to statistical analysis via analysis of variance (ANOVA). Group membership (NC, RHD) served as the between-subjects factor for each analysis and repeated measures comprised a subset of the following: EMPHASIS (none, initial, final), MODALITY (declarative, interrogative), EMOTION (neutral, sad, happy, angry), and KEYWORD position (K1-K3). Where appropriate, posthoc analyses were conducted using Tukey's HSD procedure ( $p < .05$ ).

### *Perceptual Analyses*

To appraise the *perceptibility* of emphasis and emotion in brain-damaged speakers, ten young normal listeners rated a subset of the stimuli elicited from each NC and RHD subject (i.e., declarative renditions of the utterance *Mary sold the teapot*). During one session (*emotion perception*), listeners determined the emotional tone which "most resembled" that used by the speaker for each exemplar (stimuli were those elicited from experimental subjects in a sad, happy, or angry tone, or 30 items per emotion, per diagnostic group). A second testing session (*emphasis perception*) required listeners to detect the position in which emphasis was perceived within each utterance (stimuli were those elicited with either sentence-initial or sentence-final targets, or 40 items per emphasis position, per diagnostic group). Perceptual raters were always offered an additional interpretation in judging the prosody which corresponded to a "neutral" tone (emotion perception) or a "no emphasis" designation (emphasis perception). Productions elicited from the RHD and NC subjects

were fully randomized within each task, and the emotion and emphasis perception tasks were counterbalanced for order of presentation among the ten listeners. Pearson product-moment correlation coefficients (computed between each of the ten rater's correct judgements for each speaker) indicated, on average, a moderate yet stable level of consistency across the ten listeners when judging utterances elicited from the normal (emotion = .54; emphasis = .56) and RHD (emotion = .60; emphasis = .70) speakers.

## RESULTS

Statistically significant findings are reported independently by acoustic measure, focussing on those effects of greatest theoretical interest. All significant main and interactive effects emerging from each analysis are listed in the Appendix.

### (1) *Keyword Duration*

Mean (normalized) duration values for the three "keywords" of interest (e.g., *Mary, sold, teapot*) are furnished for the RHD and NC speakers in Table II, by emphasis position, sentence modality, and emotional context. To accentuate potential group differences in the ability to encode temporal cues at specific keyword locations (parameters most characteristic of linguistic-prosodic forms such as emphatic stress), four GROUP  $\times$  MODALITY  $\times$  FOCUS  $\times$  KEYWORD ANOVAs were performed on the keyword duration data, independently for each emotional context. Generally, analysis of keyword measures pointed to similarities in how RHD and normal speakers regulated duration to mark sentence focus, but also revealed important group differences in some of the emotional contexts.

Before examining group differences in the production of keyword duration,

TABLE II  
*Mean Vowel Durations (ms) Produced by the NC and RHD Speakers at Three Keyword Positions, as a Function of Sentence Modality, Focus Position, and Emotional Context*

Group	Mod.	Focus	Neutral			Sad			Happy			Angry		
			K1 Mary	K2 sold	K3 teapot									
NC	(.)	K1	151	76	94	152	76	89	151	81	97	155	69	90
		K3	113	76	116	115	79	111	116	72	119	112	73	117
		none	133	80	106	125	82	97	121	80	113	119	73	108
	(?)	K1	157	74	101	154	73	96	142	68	98	148	70	102
		K3	120	74	109	117	68	101	113	69	115	117	68	113
		none	114	79	104	123	79	102	124	80	109	113	79	107
RHD	(.)	K1	155	83	100	149	77	99	142	90	107	141	79	98
		K3	120	87	128	114	88	115	118	84	117	112	83	125
		none	138	86	107	130	91	109	122	87	117	129	89	114
	(?)	K1	130	85	112	133	81	112	134	83	108	128	80	113
		K3	119	81	120	118	82	118	116	85	115	109	77	114
		none	127	88	111	129	92	108	122	89	114	117	85	111

Note: (.) = declarative utterances, (?) = interrogative utterances, focus = position of emphasis within the utterance

common interactions emerged from each of the keyword analyses which describe general tendencies in how emphatic and modality specifications are encoded across emotional contexts. Most notably, each of the four ANOVAs yielded a significant three-way interaction of MODALITY  $\times$  FOCUS  $\times$  KEYWORD ( $F_{\text{NEUTRAL}} = 6.61$ ; d.f. = 4, 72;  $p < .001$ ;  $F_{\text{SAD}} = 2.82$ ; d.f. = 4, 72;  $p < .05$ ;  $F_{\text{HAPPY}} = 2.62$ ; d.f. = 4, 72;  $p < .05$ ;  $F_{\text{ANGRY}} = 9.42$ ; d.f. = 4, 72;  $p < .001$ ), depicted graphically in Figure 1. Posthoc inspection of the interaction assessed how sentence modality influenced keyword duration across the three focus conditions. In the declarative speaking mode (Figure 1a), sentence-initial emphasized words were always significantly longer than matched words without emphasis (i.e., sentence-final or no focus conditions). Moreover, sentence-initial words spoken in utterances with final focus tended to be *further* reduced in duration when compared to matched words in utterances without emphasis (this was true of three of the four emotional contexts: neutral, sad, angry). This latter finding suggests that emphasis produced toward the end of an utterance is *anticipated* through reduction of the initial vowel (cf. Cooper et al., 1985). Sentence-final keywords tended to be less distinctive with respect to duration across the three emphasis conditions; words emphasized in sentence-final position were only longer than the same word spoken in utterances with alternate (i.e., initial) focus but *not* those in utterances lacking sentence focus altogether.

Continued examination of the interaction revealed that interrogative utterances (presented in Figure 1b) had a somewhat different impact on keyword duration as a function of emphasis placement. Sentence-initial focussed words were again significantly longer than matched words without focus (sentence-final or no focus). However, in sentence-final position differences in vowel length did not serve to differentiate words with and without emphasis in the interrogative speaking mode. Vowel durations of “intervening” keywords (i.e., *sold*) were invariably unaffected by differences in sentence modality or focus placement.

General patterns in how keyword duration was regulated, although mostly true of the RHD speakers, were not uniformly noted in each emotional context. The analysis of utterances elicited in a “neutral” (i.e., non-affective) tone yielded a significant four-way interaction of GROUP  $\times$  MODALITY  $\times$  FOCUS  $\times$  KEYWORD ( $F = 3.02$ ; d.f. = 4, 72;  $p < .05$ ). Posthoc analysis of the interaction indicated that in the declarative mode (Figure 2a), RHD and normal speakers each prolonged emphasized words relative to matched words spoken without emphasis on both initial and final keywords (this pattern reflects normal expectancies reported earlier). However, in the interrogative speaking mode (Figure 2b), normal speakers used temporal cues to demarcate focussed and unfocussed words in sentence-initial position (as expected), whereas RHD speakers failed to produce temporal differences across the three focus conditions in either sentence-initial or sentence-final position.

Eliciting emphatic stress tokens from subjects in various affective modes extends previous studies of keyword duration (Cooper et al., 1985; Eady and Cooper, 1986; Ryalls, Le Dorze, Lever et al., 1994); as such, the demands of implementing the temporal correlates of linguistic-prosodic forms on brain-damaged speakers in concert with emotional messages is not yet understood. Results of the ANOVAs

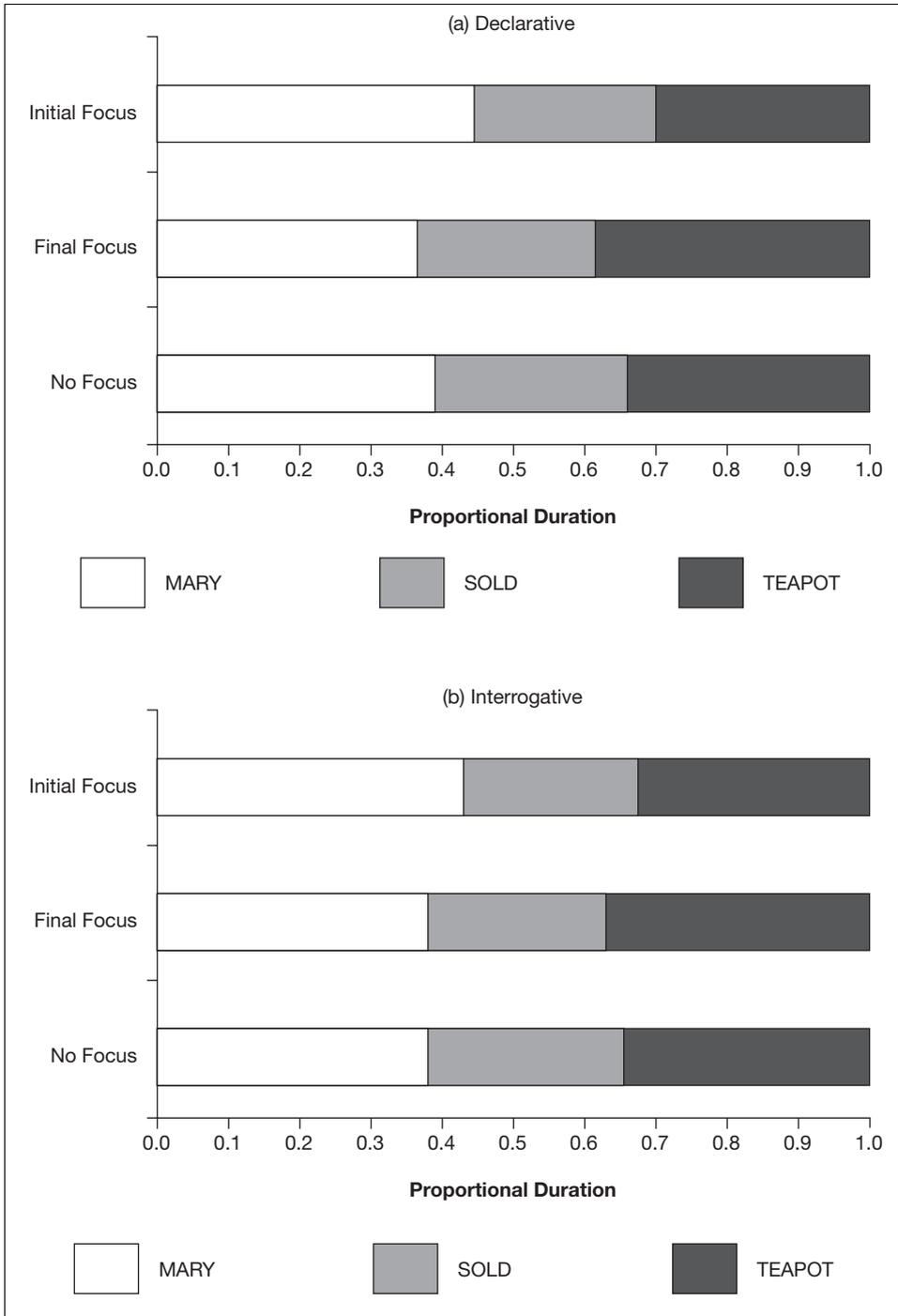


Fig. 1 – Interaction of sentence modality, focus location and keyword position on vowel duration (across speaker groups).

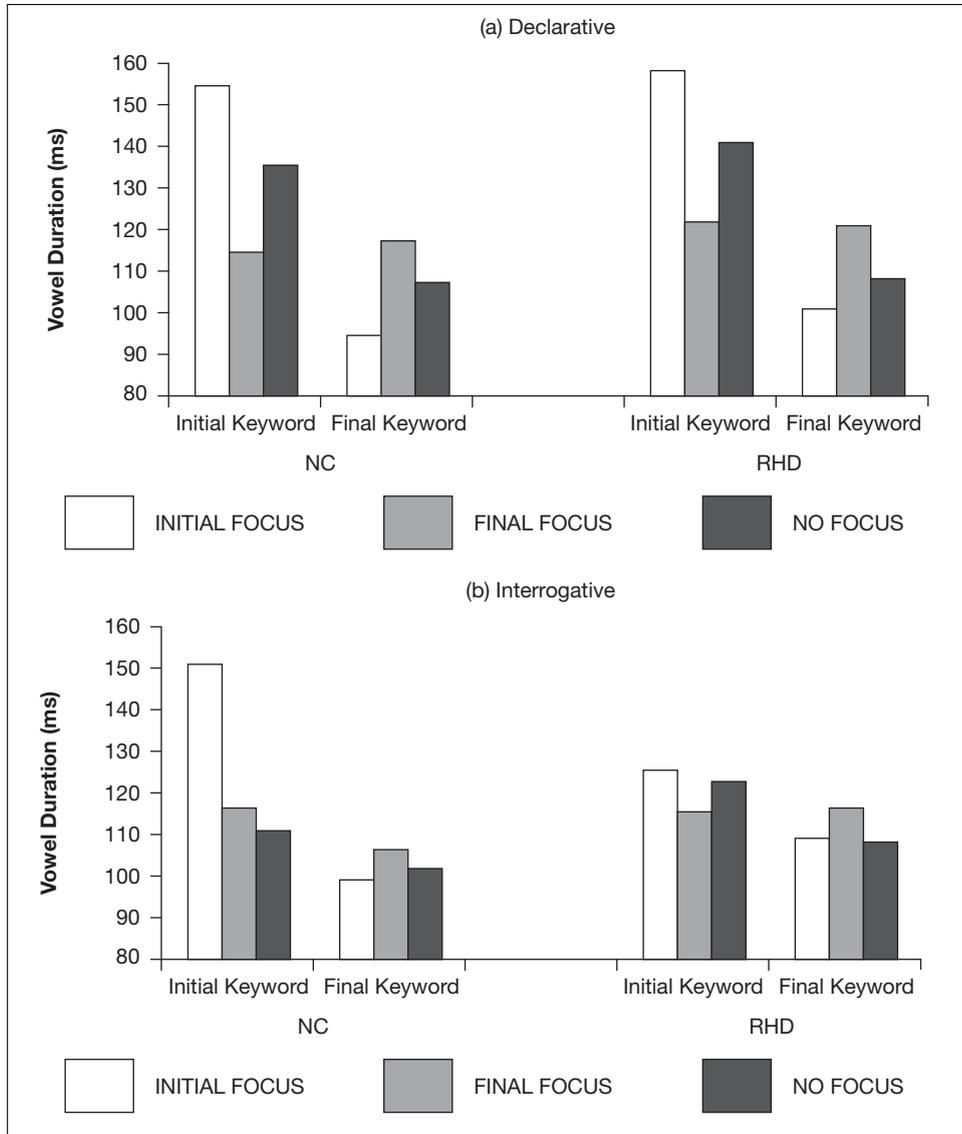


Fig. 2 – Effects of focus location and keyword position on vowel durations produced by the NC and RHD subjects in the (a) declarative and (b) interrogative mode (neutral context).

performed on stimuli elicited in a sad and happy tone did not yield significant main or interactive effects with the group factor ( $p > .05$ ). This outcome implies a high degree of correspondence in how the RHD and normal speakers employed duration as a cue to emphasis for a large subset of the data (however, cf. Pell, 1999a, for evidence that the two groups differed in the ability to modulate certain  $F_0$  features of the happy and sad stimuli.) Analysis of stimuli elicited in an angry tone produced a

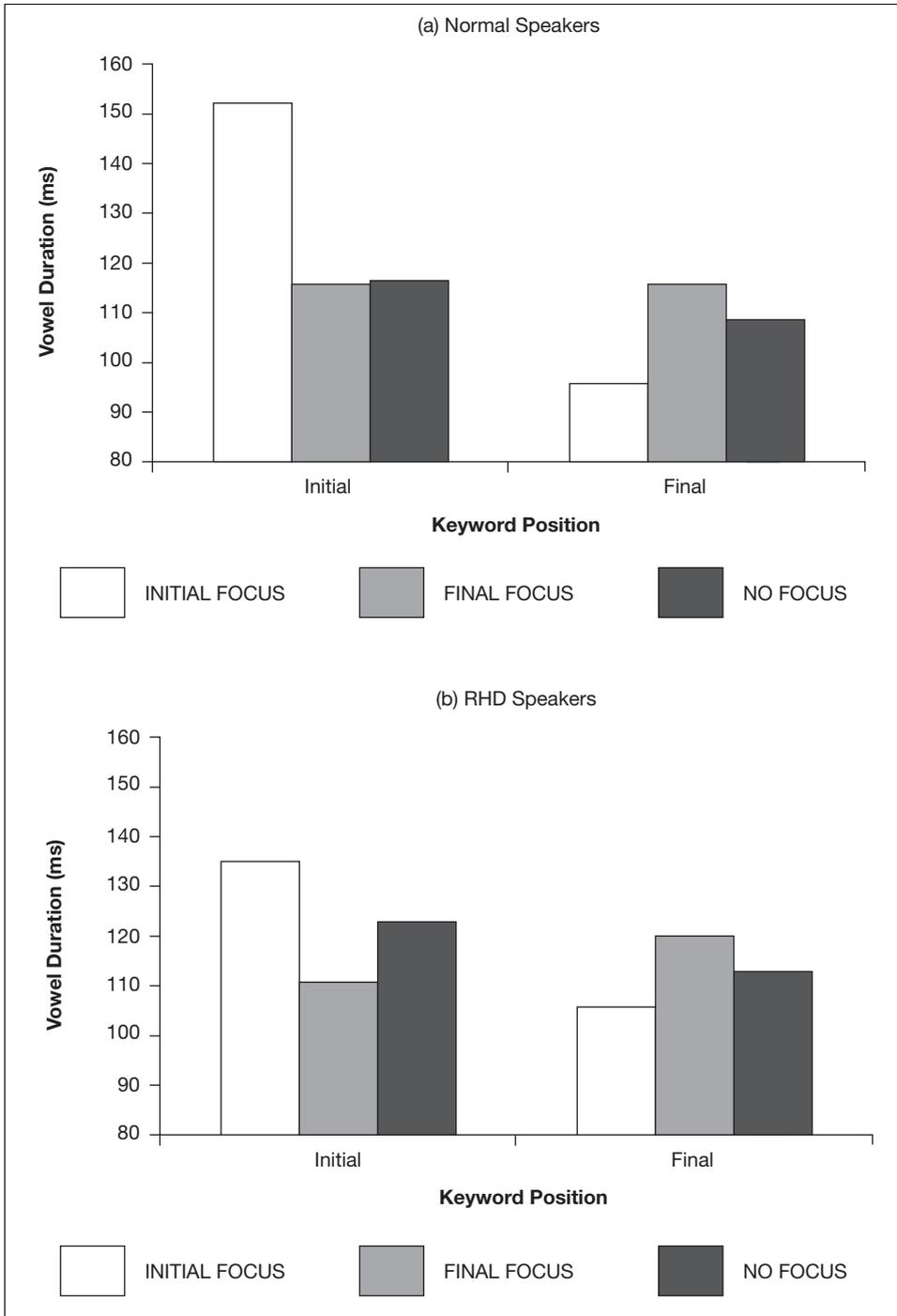


Fig. 3 – Effects of focus location and keyword position on vowel durations produced by the (a) NC and (b) RHD subjects in the angry context.

significant interaction of GROUP  $\times$  FOCUS  $\times$  KEYWORD ( $F = 2.98$ ;  $d.f. = 4, 72$ ;  $p < .05$ ], presented graphically in Figure 3. This interaction was explained by the RHD speakers' tendency to produce significantly shorter vowel durations than normal speakers to mark focus on sentence-initial keywords. (Remember that both groups reliably distinguished focused from unfocused words in this position through significant vowel lengthening.) Moreover, RHD speech tended to exhibit fewer temporal distinctions across the three focus conditions than normal speech in the angry context; namely, for the RHD individuals, only words produced in sentence-initial position displayed duration differences indicative of emphasis placement, and unlike normal speakers, these cues were only present between focused words and corresponding words in utterances spoken with alternate (sentence-final) focus<sup>2</sup>.

## (2) Speech Rate

To assess whether gross differences in speaking rate are utilized to signal prosodic meaning by normal and RHD speakers (particularly emotional meanings, e.g., Scherer, 1986), the number of syllables produced per second was computed for specific utterance types. The resulting data are reported for the NC and RHD speakers in Table III, as a function of sentence modality, focus position, and emotion type.

TABLE III  
*Mean Speech Rate (Syllables/S) of the NC and RHD Speakers as a Function of Sentence Modality, Focus Position, and Emotional Context*

Group	Mod.	Focus	Speech rate			
			Neutral	Sad	Happy	Angry
NC	(.)	K1	4.80	3.89	4.54	4.33
		K3	4.85	3.57	4.40	4.06
		none	4.97	3.93	4.68	4.01
	(?)	K1	4.50	3.93	4.27	4.36
		K3	4.43	3.62	4.35	4.19
		none	4.43	3.94	4.55	4.20
RHD	(.)	K1	4.64	4.06	4.33	4.27
		K3	4.68	3.99	4.28	4.15
		none	4.45	4.07	4.38	4.24
	(?)	K1	4.10	4.13	4.23	4.10
		K3	4.34	4.09	4.41	4.25
		none	4.36	4.16	4.46	4.24

Note: (.) = declarative utterances, (?) = interrogative utterances, K1 = emphasis on sentence-initial word, K3 = emphasis on sentence-final word.

<sup>2</sup>More extensive temporal abnormalities were noted in a study that elicited longer (10 syllable) utterances from the current RHD speakers (Pell, 1997). These irregularities were again characterized by the tendency of RHD speakers to provide fewer (or smaller) duration cues to emphasis contrasts than NC subjects, a pattern observed in all four affective contexts for the longer stimuli. These findings imply that utterance length may influence the success of RHD patients in implementing the temporal parameters of linguistic-prosodic forms to some degree (Behrens, 1989; Gandour et al., 1994). Alternatively, as suggested by an anonymous reviewer, increased attentional demands imposed by the elicitation paradigm for longer stimuli may be linked (in part) to the RHD patients' performance in this condition, possibly in a non-specific manner.

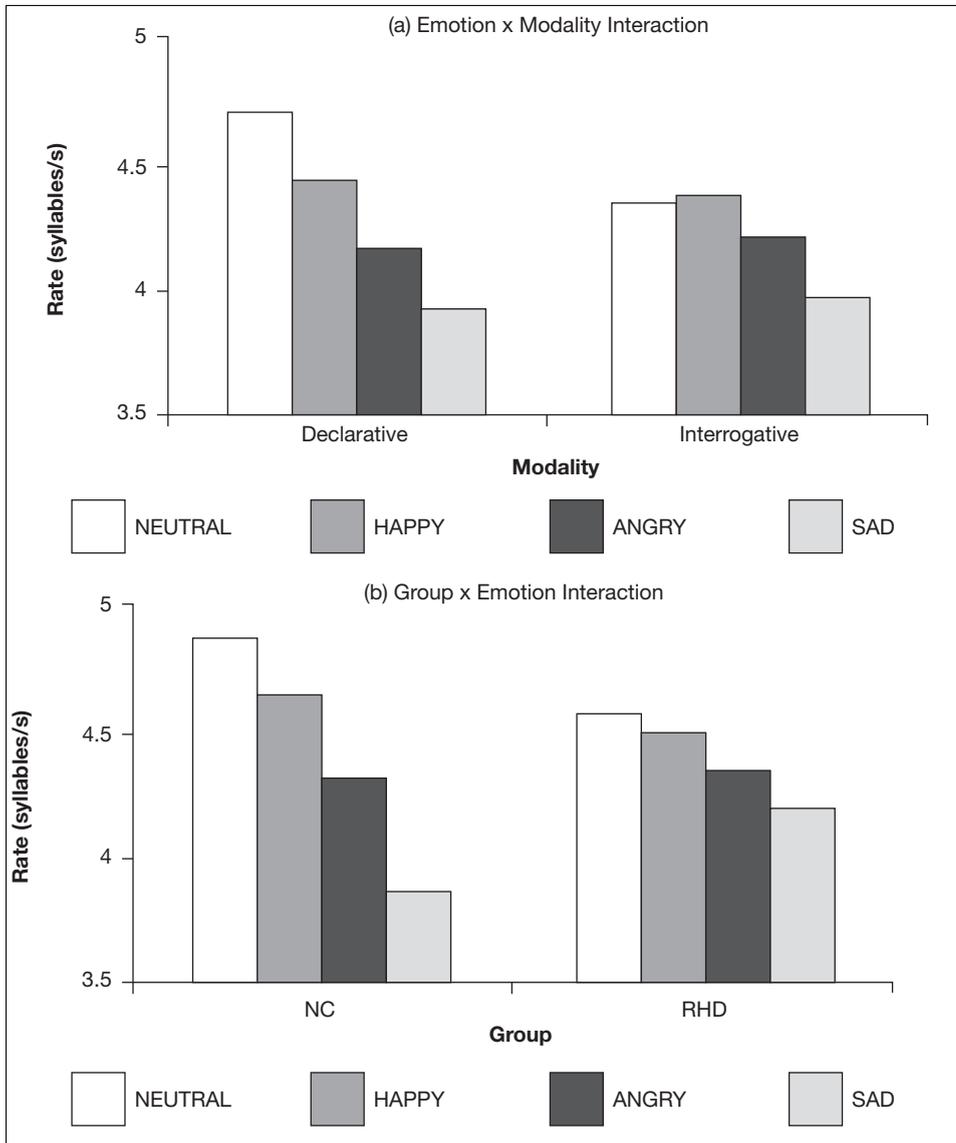


Fig. 4 – Interaction of (a) emotion and sentence modality and (b) emotion and group membership on speaking rate.

Analysis of the speech rate data yielded a significant main effect of EMOTION ( $F = 44.92$ ;  $d.f. = 3, 54$ ;  $p < .001$ ) and interactive effect of EMOTION  $\times$  FOCUS ( $F = 3.05$ ;  $d.f. = 6, 108$ ;  $p < .01$ ). Posthoc tests on the interaction indicated that neutral and happy prosody (which did not significantly differ) were conveyed at a significantly faster rate than sad prosody, and that neutral prosody also exceeded angry prosody, irrespective of emphasis

placement. Happy sentences were also faster than angry sentences in certain conditions (i.e., when stimuli lacked focus), and angry sentences tended to be faster than sad sentences (i.e., when sentence-initial or sentence-final focus was present). In a given affective tone, speaking rate never varied significantly as a function of emphasis position within the utterance.

A significant EMOTION  $\times$  MODALITY interaction (presented in Figure 4a) was additionally observed ( $F = 12.42$ ; d.f. 3, 54;  $p < .001$ ). In the declarative mode, all four emotions could be distinguished by significant differences in speech rate; neutral exceeded happy, happy surpassed angry, and angry exceeded sad in rate. In the interrogative mode, rate differences were far less evident and only emerged between sad prosody (slowest) and neutral, happy, and angry prosody (which did not significantly differ).

Only neutral prosody differed in rate as a function of linguistic modality, neutral declaratives being delivered at a significantly increased rate than neutral interrogatives overall.

General tendencies in how speakers regulated speaking rate as a function of the prosodic variables were tempered by group differences in the data. Specifically, a significant GROUP  $\times$  EMOTION interaction ( $F = 7.80$ ; d.f. = 3, 54;  $p < .001$ ), illustrated in Figure 4b, established that the RHD patients expressed sad prosody at an accelerated rate when compared to normal speakers. This difference attenuated the number of rate distinctions present in the RHD patients' speech when viewed across the four emotions: although normal speakers employed speech rate to demarcate three of the four emotions (neutral and happy prosody were both significantly faster than angry prosody, which was faster than sad prosody), RHD displayed similar tendencies but fewer differences overall (neutral and happy prosody surpassed sad prosody, but angry prosody did not differ in rate from either neutral, happy, or sad prosody).

### *Perceptual Analyses*

The success of the RHD and normal speakers at projecting emphatic and emotional distinctions to a group of unbiased *listeners* was assessed quantitatively in an earlier report (Pell, 1999a). Briefly, these analyses indicated that RHD speakers were significantly less able to convey both emphasis and emotional prosody to listeners than normal speakers, but that the RHD patients' success in communicating emphasis and emotion was also contingent on specific category variables (i.e., RHD patients were least able to convey sentence-final emphasis and "happy" prosody to listeners, respectively). A more qualitative evaluation of the perceptual data may complement acoustic findings reported here and in Pell (1999a). These supplementary analyses, although not specifically related to temporal parameters of the stimuli, may help clarify some of the factors underlying the RHD patients' performance on expressive prosody tasks which could not be captured quantitatively (Pell, 1999a) or through the present acoustic measures.

Table IV supplies matrices that reflect the total responses obtained in judging emphatic targets (focus perception) and emotional targets (emotion perception) from the RHD and normal speakers (expressed as a percentage of the total for each

TABLE IV  
 Mean Percentage of Responses Obtained for Emotional Targets (emotion perception) and Emphatic Targets (focus perception) Following Perceptual Presentation of the NC and RHD Speakers' Utterances to Ten Listeners

Speaker	Actual response (%)								
	Target	Emotion perception				Focus perception			
		Sad	Happy	Angry	Neutral	Target	Initial	Final	None
NC	Sad	61	6	19	14	Initial	80	6	14
	Happy	4	78	41	14	Final	3	77	20
	Angry	6	19	64	11	–	–	–	–
RHD	Sad	56	2	21	21	Initial	80	4	16
	Happy	16	37	21	26	Final	12	56	32
	Angry	13	11	58	18	–	–	–	–

Note: initial = sentence-initial emphasis, final = sentence-final emphasis.

target). In the focus perception condition, inspection of the data revealed that when judging sentence-final targets, a substantially larger portion of the total responses fell within the “no focus” category when elicited from RHD speakers (32%) than from normal speakers (20%). This pattern conflicts with that obtained for sentence-initial targets where a similar incidence of “no focus” responses was observed for the RHD (16%) and NC (14%) speakers. The resilience of temporal distinctions provided for sentence-initial, but not always sentence-final, keywords by the RHD speakers may have been a contributing factor to the perceptual confusability of these forms (see also Pell, 1999a, for related data on fundamental frequency).

Finally, analysis of the emotional ratings indicated that for all three emotions, a substantially larger proportion of utterances produced by the RHD patients were misidentified as *lacking* emotional inflection (i.e., a “neutral” response was recorded) than utterances produced by the normal speakers (for sad, happy, and angry targets, respectively: RHD = 21%, 26%, 18%; NC = 14%, 14%, 11%). This qualitative pattern suggests a blurring or “flattening” of affective-prosodic distinctions in the RHD patients’ speech towards a “neutral” baseline (Pell, 1999a, 1999b). A tendency for emotional messages to converge acoustically in the RHD speakers was recently attributed to difficulties modulating certain fundamental frequency parameters of the emotional stimuli (Pell, 1999a). Speech rate data reported herein provide further evidence of such a pattern, as substantially fewer distinctions were observed among the four emotional categories for the RHD speakers. Taken together, the perceptual data re-affirm that acoustic irregularities noted between normal and RHD speakers – inclusive of temporal abnormalities in the data – have a discernable impact on the patients’ ability to communicate prosodic meanings in an effective manner. There was no apparent relationship between the acoustic or perceptual findings and prominent clinical variables in the RHD group such as lesion site, time post-CVA, or hemispatial neglect based on cursory inspection of individual performance characteristics.

## DISCUSSION

Together with a companion report (Pell, 1999a), the current study represents an initial attempt to understand the *interactive* influence of linguistic and emotional prosody on speech production following focal right hemisphere lesion, and estimate its perceptual consequences. The present investigation also afforded a rare look at whether the right cerebral hemisphere regulates speech timing at both the syllabic and sentence levels. These data were motivated by the need to inform previously articulated (but conflicting) views about the right hemisphere's role in encoding prosody (e.g., Behrens, 1989; Pell, 1999a; Ross, 1981; Shapiro and Danly, 1985; Van Lancker and Sidtis, 1992). Current findings also highlight important aspects of normal speech production previously undocumented in a structured manner.

The success of RHD patients in modulating temporal parameters of emphatic stress tokens in the present study was determined by computing the vowel duration of designated "keywords" in the utterances elicited. In general, the production of duration as a cue to sentence focus patterned in a similar manner for the RHD and normal speakers in many important respects. For example, emphasized vowels in both sentence-initial and sentence-final words tended to be longer in duration (i.e., more prominent) than vowels in matched words spoken without emphasis for both speaker groups (Behrens, 1988; Cooper et al., 1985; Eady and Cooper, 1986; Ouellette and Baum, 1993). Further, emphasis of one sentence element through vowel lengthening (e.g. *Mary* in *Mary sold the teapot*) often resulted in "de-emphasis" of potentially contrastive sentence elements (e.g., *teapot*) beyond that observed in utterances that lacked focus altogether. Finally, "intervening" words (i.e., *sold*) were always less prominent in terms of duration relative to content words in sentence-initial and sentence-final position for both RHD and NC speakers. These patterns in the temporal organization of the utterances emerged in a highly regularized manner for both speaker groups, despite (new) evidence that keyword duration was contingent on differences in focus position and sentence modality during speech planning.

However, it is inappropriate to claim that the temporal correlates of emphatic stress were consistently implemented in a uniform manner by the RHD and normal speakers. For instance, RHD patients failed at times to lengthen initial focussed vowels to the extent displayed by normal subjects (see Ouellette and Baum, 1993, for similar trends). Moreover, at designated keyword positions, duration did not always signal distinctions among the three focus conditions as distinctively for the RHD speakers (particularly when interrogatives were elicited, in which case differences in focus distribution were sometimes completely unmarked by duration in the RHD patients' speech). Perhaps of greatest significance, *perceptual* judgements of each group's speech indicated that RHD patients were less proficient at conveying emphasis to listeners overall, although this effect was associated primarily with sentence-final tokens (Pell, 1999a). Thus, current findings suggest a trend whereby RHD patients tended to exploit somewhat *fewer* of the normal expected cues to emphasis in their utterances (Behrens, 1988; Hird and Kirsner, 1993; Pell, 1999a). Such difficulties appeared to be exacerbated by the (assumed) increased processing

demands of encoding interrogative intonation and linguistic focus in tandem, a pattern commented on earlier for  $F_0$  aspects of the same stimuli (Pell, 1999a).

Signs that the amount of acoustic detail supplied by RHD speakers in particular speaking conditions is somewhat attenuated – although potentially revealing of their diminished success in communicating sentence focus to listeners – does not override evidence that RHD patients modulated duration in a qualitatively normal manner in combination with a broad range of concurrent features. Accordingly, the present data converge with previous findings that argue that the capacity to produce emphatic stress following focal right hemisphere compromise is largely spared (Behrens, 1988; Ouellette and Baum, 1993). Similar conclusions were arrived at by Pell (1999a) in his examination of  $F_0$  parameters of the same stimuli, suggesting that the right hemisphere's role in encoding linguistic stress features is likely negligible (Behrens, 1988; Behrens, 1989; Emmorey, 1987; Gandour et al., 1992b; Grela and Gandour, 1998; Pell, 1999a).

The contribution of temporal parameters to emotional communication is represented most clearly through rate of speech (Fairbanks and Hoaglin, 1941; Pell and Baum, 1997b; Scherer, 1986; Williams and Stevens, 1981). The four affect types elicited from subjects in the present study (angry, sad, happy, neutral) separated well as a function of speaking rate; happy and neutral prosody tended to be produced at a relatively high rate when compared to angry prosody, which in turn tended to be produced more quickly than sad prosody. The RHD subjects' ability to regulate speech timing to transmit emotional messages was of great interest in the present study, owing to multiple claims that emotional prosody is specifically disrupted by right hemisphere lesions (Borod, 1993; Edmondson, Chan, Seibert et al., 1987; Gandour et al., 1995; Ross et al., 1981; Shapiro and Danly, 1985). Examination of the speech rate measures demonstrated parallelism between the RHD and normal speakers when *general* dimensions of their emotional utterances were considered (Baum and Pell, 1997; Gandour et al., 1995). The RHD patients' use of speech rate diverged from normalcy for only one emotion type, "sad" prosody. For normal speakers, the slow rate at which sad utterances were produced rendered these exemplars highly distinctive from the other three emotions. In contrast, RHD patients produced sad utterances significantly faster than normal subjects, a pattern which led to poor separation of the four emotions in their speech as a function of speaking rate (review Figure 4b). Curiously, similar imprecision in how RHD patients modulated the  $F_0$  underpinnings of emotional contrasts – a tendency which again culminated in fewer normal contrasts within the emotional dataset under study – was noted on a previous occasion for this clinical sample (Pell, 1999a).

In fact, it is worthwhile to compare patterns described in the present investigation of temporal parameters and those reported by Pell (1999a) for fundamental frequency parameters of the same stimuli, spoken by the same diagnostic groups. Based on these earlier analyses, RHD speakers were characterized by "subtle difficulties" in the ability to modulate  $F_0$  properties of emotional stimuli (mean  $F_0$ ,  $F_0$  variability) to signal discrete emotional qualities in their speech (see also Colsher, Cooper, and Graff-Radford, 1987; Dykstra et al., 1995; Edmondson et al., 1987; Shapiro and Danly, 1985). More specifically,

analysis of the  $F_0$  data suggested that RHD speakers lack precision in modulating  $F_0$  to achieve (known) emotional targets which diverge substantially from a neutral “baseline” or resting state for that vocal parameter (Pell, 1999a). Thus, for  $F_0$ , emotional representations specified at an elevated  $F_0$  level with considerable long-term  $F_0$  variation (i.e., happy prosody) posed particular difficulties for the RHD speakers when compared to their production of neutral (i.e., non-affective) utterances. As reported earlier and elaborated herein, irregularities in the acoustic parameters were reflected in less reliable transmission of emotional messages to an unbiased group of listeners in the case of the RHD speakers (also Gandour et al., 1995).

Interestingly, patterns in the temporal data are conducive to a similar interpretation to that furnished by Pell (1999a); the ability of RHD patients to differentiate emotional messages through speech rate was most deficient for precisely those contrasts which were most ‘marked’ from neutral utterances on the basis of that cue (i.e., sad prosody). Positing a continuum of emotional features within which discrete alternatives are represented or interpreted is not novel (Levenson, 1992; Ohala, 1984; Scherer, 1986). Although not uncontroversial, the notion that “neutral” signals represent an independent point along an emotional continuum which are then “coloured” through emotional inflection also finds precedence in the literature (e.g., Bolinger, 1986; Gandour et al., 1995; Ladd, 1980; Young et al., 1997). Following suggestions by Peretz (1990) and Blonder, Pickering, Heath et al. (1995), Pell’s (1999a) data on  $F_0$  were viewed as evidence of phonetic difficulties in achieving normal acoustic landmarks following RHD when required to convey prosodic meaning that varies in a graded (rather than categorical) manner. Such a deficit would seem to explain normal qualitative tendencies in the group data as well as a reduction in the number of inter-stimulus differences produced by RHD speakers across prosodic conditions, or a tendency for utterances varying in emotional or emphatic content to “converge” acoustically for the RHD patients (Pell, 1999b).

A related, if somewhat more subtle, pattern in the current data provides further support for the idea that RHD speakers are limited in their ability to control continuous or graded aspects of intonation contours during speech production (Blonder et al., 1995; Pell, 1999a). If this hypothesis is validated, it is of little surprise that deficits in *emotional* expression (which are associated with a speaker’s level of activation and therefore inherently “continuous” in nature) have been cited most frequently in the RHD population relative to deficits in the production of categorical distinctions such as linguistic stress (Borod, 1993; Edmondson et al., 1987; Gandour et al., 1995; Ross et al., 1981; Shapiro and Danly, 1985). The failure of RHD patients to “fine-tune” representative acoustic properties of emotional utterances in a manner which adequately distinguishes them from neutral exemplars is also predictive of subjective impressions of reduced emotional inflection or “flat affect” in this population (Baum and Pell, 1997; Behrens, 1989; Blonder et al., 1995; Borod et al., 1990; Pell, 1999a, 1999b; Ross et al., 1981; Ross and Mesulam, 1979; Ryalls et al., 1987).

The hypothesis that duration and  $F_0$  parameters of prosodic stimuli rely on processing mechanisms residing in the left and right hemispheres of the brain,

respectively (Robin et al., 1990; Van Lancker and Sidtis, 1992), merits brief commentary. A uniform group of RHD speakers examined here and by Pell (1999a) presented with (selective) deficits in modulating both temporal and  $F_0$  aspects of the stimuli, indicating that more than  $F_0$  properties of intonation tend to be disrupted following right hemisphere insult (Blonder et al., 1995; Dykstra et al., 1995; Grela and Gandour, 1998; Pell and Baum, 1997b). Given the particular importance of  $F_0$  cues in signalling prosodic meaning (e.g., Bolinger, 1986; Lieberman and Michaels, 1962), it remains nonetheless possible that the type of quantitative impairment attributed to RHD patients in the present research tends to emerge more frequently for  $F_0$  parameters, as intimated by the "cue hypothesis" (Robin et al., 1990; Van Lancker and Sidtis, 1992; Zatorre et al., 1992). Certainly, the concept that unilateral right-brain-damage yields a sensitivity to selective elements of the physical form of prosodic contours is corroborated by this and other findings (Pell, 1998, 1999a), highlighting the need for further consideration of how the acoustic determinants of prosodic meaning influence hemispheric performance on such tasks.

The current study is not without certain limitations. For example, left-hemisphere-damaged aphasic patients were not included in the study due to the complexity of the verbal stimuli presented; future attempts to elicit linguistic and emotional prosody from such patients will be necessary before right-hemisphere-based neural networks can be invested with *exclusive* jurisdiction over continuous attributes of prosody in expressive language. Similarly, the current study was not designed to examine inter-speaker variability in prosodic abilities in detail, in spite of evidence that not all speakers conformed to the group pattern in each condition (e.g., R2 was particularly effective in transmitting emotion to listeners (82% correct) when compared to the group average (49.5%); see Pell, 1999a, 1999b, for details). Finally, the manner in which emotional utterances are elicited on structured tasks is always somewhat problematic; despite considerable efforts to evoke a genuine emotional response from RHD listeners by placing them into a hypothetical priming context, findings are nonetheless reflective, to some extent, of subjects' ability to *pose* certain emotions. Certainly, it is probable that structured elicitation paradigms impose demands that are atypical of natural speech production, and possibly, that exceed resources available to some brain-damaged (including RHD) individuals (e.g., Tompkins, Bloise, Timko et al., 1994; Tompkins and Flowers, 1985). This latter shortcoming – a pervasive difficulty in studies of emotional expression and behaviour – may be mitigated by comparing individual speech characteristics on both structured and spontaneous speech tasks, a project currently underway in our laboratory.

Finally, as alluded to in the discussion of Monrad-Krohn's dysprosodic patient, the phenomenon whereby acquired brain lesion produces a primary disturbance of speech patterns is characteristic of patients with so-called 'foreign accent syndrome' (Blumstein, Alexander, Ryalls et al., 1987; Graff-Radford, Cooper, Colsher et al., 1986; Kurowski et al., 1996; Monrad-Krohn, 1947). A recent analysis of this condition suggests that perceived changes in the prosodic quality of these patients' speech resemble those of a non-native speaker, but importantly, *not* those of a speaker whose speech has been disrupted by acquired

(left) hemispheric lesion (i.e., their speech does not deviate from potentially normal combinations of parameters, Kurowski et al., 1996). In a similar vein, pathological limitations in how RHD patients implement graded aspects of prosodic contours may often be treated by listeners as utterances which are qualitatively normal, but which lack the extent of affective detail typical of the majority of speakers (or which fail to complement other cues in the communicative interaction). Importantly, in the case of both disturbances, it is the impact on the *listener* that is most instrumental in defining the nature of the deficit. Thus, RHD patients do not lose knowledge of how emotional representations are encoded vocally, but rather, become less efficient at regulating aspects of the speech signal that evoke an emotional response in listeners (or which, for example, convey the degree of emphasis intended). The neurological substrates of this regulatory device within the right hemisphere, if indeed validated, awaits future research.

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#### APPENDIX Significant ANOVA Results

	Main effects	Interactions with Group	Interactions without Group
(1) Keyword duration			
Sad	F: F = 6.03; d.f. = 2, 36; p < .01 K: F = 73.51; d.f. = 2, 36; p < .001		F × K: F = 25.97; d.f. = 4, 72; p < .001 M × F × K: F = 2.82; d.f. = 4, 72; p < .05
Happy	F: F = 5.04; d.f. = 2, 36; p < .05 K: F = 86.37; d.f. = 2, 36; p < .001		F × K: F = 32.88; d.f. = 4, 72; p < .001 M × F × K: F = 2.62; d.f. = 4, 72; p < .05
Angry	F: F = 3.14; d.f. = 2, 36; p = .055 K: F = 72.56; d.f. = 2, 36; p < .001	G × F × K: F = 2.98; d.f. = 4, 72; p < .05	F × K: F = 30.85; d.f. = 4, 72; p < .001 M × K: F = 7.14; d.f. = 2, 36; p < .01 M × F × K: F = 9.42; d.f. = 4, 72; p < .001
Neutral	F: F = 12.87; d.f. = 2, 36; p < .001 K: F = 129.68; d.f. = 2, 36; p < .001	G × F: F = 5.20; d.f. = 2, 36; p = .01 G × M × K: F = 4.22; d.f. = 2, 36; p < .05 G × F × K: F = 2.80; d.f. = 4, 72; p < .05 G × M × F × K: F = 3.02; d.f. = 4, 72; p < .05	F × K: F = 34.63; d.f. = 4, 72; p < .001 M × K: F = 6.84; d.f. = 2, 36; p < .01 M × F × K: F = 6.61; d.f. = 4, 72; p < .001
(2) Speech rate			
	E: F = 44.92; d.f. = 3, 54; p < .001	G × E: F = 7.80; d.f. = 3, 54; p < .001	E × F: F = 3.05; d.f. = 6, 108; p < .01 E × M: F = 12.42; d.f. = 3, 54; p < .001

Note: G = Group, F = Focus, M = Modality, E = Emotion, K = Keyword.