

CHAPTER

**28**

**Sentential Stress  
Production by  
Normal and Left  
Anterior Lesion  
Subjects**

Karen A. Colson  
Donald A. Robin  
Erich S. Luschei

In recent years, much research has been devoted to examining the neuroanatomic correlates of disturbances in speech prosody (Ross and Mesulam, 1979; Kent and Rosenbek, 1982; Shapiro and Danly, 1985; Emmorey, 1987; Ryalls, Joannette, and Feldman, 1987; Behrens, 1988). The majority of findings indicate that unilateral cortical insult may result in differential deficits of prosodic processing. Further, the findings suggest that the encoding of prosodic features may be impaired selectively according to the lesion site within a hemisphere. Thus it is possible that focal unilateral cortical damage may be associated with primary disorders of prosodic output, in which case patients with similar lesion sites may manifest common patterns of prosodic impairment.

Current evidence indicates that dysprosody is a primary symptom in apraxia of speech (Rosenbek and Wertz, 1976; Rosenbek, 1985; Kent and Rosenbek, 1982; Wertz, LaPointe, and Rosenbek, 1984) and an important clinical feature for distinguishing Broca's aphasia from other aphasic disorders (Goodglass, Quadfasel, and Timberlake, 1964; Benson, 1967, Kerschensteiner, Poeck, and Brunner, 1972; Blumstein, 1973a, 1973b). It has been well documented that patients with left frontal opercular damage frequently manifest apraxia of speech and/or Broca's aphasia (Mohr, 1973; Mazzochi and Vignolo, 1979; Knopman et al., 1983; Kertesz, Ferro, and Shewan, 1984, Marquardt and Sussman, 1984). Thus, in general, the majority of present findings suggest that disturbances in prosodic output may be similar among left anterior lesion patients. However, systematic study of prosodic deficits in relation to the locus of left-hemisphere subjects' lesions has, as yet, provided only limited descriptive data (Kent and Rosenbek, 1982; Emmorey, 1987). Therefore, it is unclear if there are commonalities in the prosodic disturbances of patients with focal left anterior lesions that may be relevant to an understanding of the expressive impairments of this clinical population. More extensive study of the prosodic output of patients with well-defined left anterior insult should contribute to an understanding of the relationship between dysprosody and left cerebral damage and the most productive methods for management.

Descriptions of the prosodic disturbances of patients with apraxia of speech and/or Broca's aphasia, and presumably left anterior damage, typically have identified abnormal stress patterning as a salient characteristic (Darley, Aronson, and Brown, 1975; Rosenbek, 1985; Kent and Rosenbek, 1982, 1983). Findings from several studies indicate that abnormalities in the patients' stress patterns may influence the grammatical structure and/or fluency of their productions (Goodglass, Fodor, and Schulhoff, 1967; Lebrun, Buysens, and Henneaux, 1973; Danly and Shapiro, 1982). Nevertheless, data pertaining to how adequately the patients are able to convey varied stress patterns at a perceptual level are scarce. Moreover, it is unclear if there are differences in the patients'

spontaneous and imitative stress-patterning skills. Given the impact that disturbed stress production may have on these patients' communication and their clinical management, the perceptual adequacy of their spontaneous and imitative stress patterning should be examined more thoroughly.

The present study was designed to examine the stress-production ability of patients with well-defined lesions of the left frontal opercular region. Specifically, this study sought to determine if these patients differ from normal adults in their ability to produce varied patterns of sentential stress spontaneously and/or imitatively. This study also examined the brain-damaged patients' sentential stress production to determine if the group differed in their ability to vary stress placement spontaneously and imitatively.

## METHOD

Subject information is presented in Table 28-1. All subjects were native English speakers and demonstrated hearing sensitivity thresholds of at least 30 dB HL in the better ear. The normal and the brain-injured groups were matched for age and sex. Selection of the patients was based primarily on neuroanatomic data obtained from standard CT and/or MRI scan localization protocol as described by Damasio (1983, 1987). Each patient had a medically diagnosed history of a single focal left-hemisphere lesion in any or part of the areas defined as precentral cor-

**TABLE 28-1. SUBJECT INFORMATION: AGE, SEX, ETIOLOGY, YEARS AFTER ONSET**

<i>Subject</i>	<i>Age</i>	<i>Sex</i>	<i>Etiology</i>	<i>Years after onset</i>
Left:				
LAL1	44	M	Thrombotic	3.9
LAL2	50	M	Embolic	2.3
LAL3	68	F	Thrombotic	1.7
LAL4	67	M	Thrombotic	4.2
LAL5	64	M	Thrombotic	1.0
Normal:				
N1	44	M		
N2	50	M		
N3	69	F		
N4	68	M		
N5	63	M		

tex, premotor cortex, Broca's area and surrounding structures (e.g., Brodmann's areas 44 and 45), and the insular cortex. Localization data for each left-anterior-lesion (LAL) subject are shown in Table 28-2. Sample size was limited due to the availability of patients who met the rigid LAL selection criteria. Other selection criteria excluded patients whose medical records indicated a history of seizures, signs of dementia or psychosis, and/or major health complications. Speech-language testing (Schuell and Sefer, 1973; Goodglass and Kaplan, 1983) indicated that all patients were able to respond appropriately and reliably to verbal input (e.g., commands and questions), recall three-part verbal sequences, and match printed to spoken words. The patients also were able to repeat and spontaneously produce intelligible three-word (monosyllabic) utterances. There were no signs of structural speech anomalies, oral paralysis, or a marked oral apraxia during an oral peripheral examination and performance of oral, nonverbal movement tasks (DeRenzi, Pieczuro, and Vignolo, 1966; Darley, Aronson, and Brown, 1975; LaPointe and Wertz, 1974).

Stimuli for the imitation task were recorded repetitions of 10 declarative sentences composed of three monosyllabic content words. Stress placement was varied in four randomized repetitions of each sentence

**TABLE 28-2. LESION LOCALIZATION DATA FOR EACH BRAIN-INJURED SUBJECT**

<i>Subject</i>	<i>Lesion locus</i>
LAL1	Left frontal lobe: most superior tip of frontal operculum and premotor region immediately above (cortices and underlying white matter)
LAL2	Left frontal lobe: posterior part of frontal operculum and premotor and motor regions immediately behind, anterior portion of insula (cortices and underlying white matter)
LAL3	Left frontal lobe: the frontal operculum and premotor and motor regions immediately behind and superiorly, anterior part of insula (cortices and underlying white matter)
LAL4	Left frontal lobe: frontal operculum and premotor region immediately above, extends deep into white matter almost to frontal horn of lateral ventricle, involves insular cortex and white matter
LAL5	Left frontal lobe: premotor cortex and white matter just behind frontal operculum, extends deep in white matter to the frontal horn of lateral ventricle and part of anterior limb of internal capsule, involves insular cortex and white matter and most of lenticular nucleus

so that one of the three words was stressed or all three words were given equal stress in a "neutral" version. Each of the 40 stimulus sentences had been judged perceptually by a panel of four naive listeners to convey a particular target stress pattern unambiguously.

Stimuli for the spontaneous stress task were recorded productions of the neutral version of each three-word sentence, followed by a question designed to prompt a response with a particular stress pattern. Four different questions, structured to elicit stress placement on each of the three words or equally on all three words, were asked for each stimulus sentence (randomly).

Stimuli were presented by means of headphones, with all subjects performing the spontaneous production task first. In the spontaneous task, subjects were instructed to listen to each stimulus item and respond by producing the entire sentence in a manner that appropriately answered the question. The subjects' responses were tape-recorded. For the imitative stress task, recordings were made as each subject repeated the stimuli. The subjects were instructed to repeat the sentences exactly as they were produced on the recording.

Responses for both production tasks were analyzed perceptually by three certified speech-language pathologists and three naive listeners to

**TABLE 28-3. INTRAJUDGE RELIABILITY, EXPRESSED AS A PERCENTAGE OF AGREEMENT, FOR PERCEPTUAL JUDGMENTS OF STRESS PLACEMENT IN SPONTANEOUS AND IMITATIVE UTTERANCES PRODUCED BY ALL SUBJECTS, NORMAL SUBJECTS, AND LAL SUBJECTS**

<i>Judge*</i>	<i>All subjects</i>	<i>Normal only</i>	<i>LAL only</i>
<b>Spontaneous utterances:</b>			
1	90%	86%	86%
2	80%	80%	89%
3	97%	100%	95%
4	84%	87%	86%
5	92%	92%	92%
6	90%	89%	89%
<b>Imitative utterances:</b>			
1	83%	92%	85%
2	86%	89%	80%
3	82%	82%	83%
4	80%	89%	83%
5	80%	89%	78%
6	88%	86%	85%

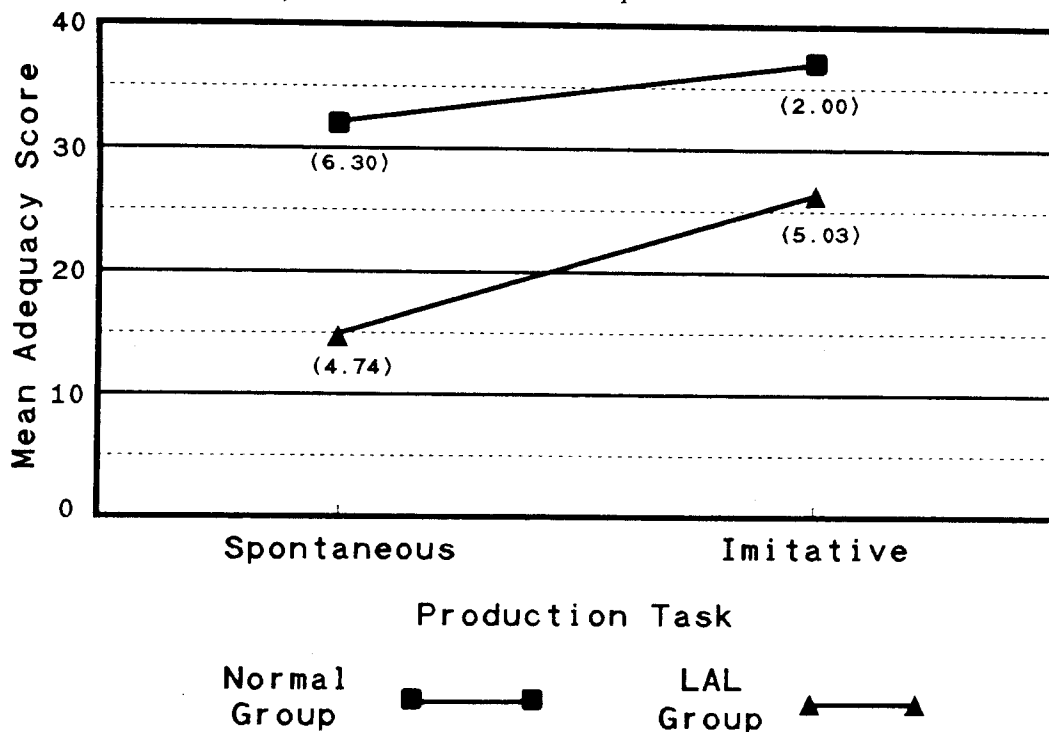
\*Judges 1 to 3 were certified speech-language pathologists, and judges 4 to 6 were naive listeners.

determine how adequately each subject conveyed the stress patterns. Each production perceived by four or more judges to have a stress pattern different from the target pattern was classified as inadequate. Each inadequate production was considered to have a substituted stress pattern when at least four of the judges agreed that a particular stress pattern was used instead of the target pattern. Intrajudge reliability measures for each of the six listeners' perceptual judgments are shown in Table 28-3. Interjudge reliability, expressed as a mean percentage of agreement, was determined to be 92 percent for the judgments of both spontaneous and imitative stress placement.

## RESULTS

Group means and standard deviations for the LAL and normal subjects' scores on the stress-production tasks are presented in Figure 28-1. Evaluation of the measures with a split-plot, two-factor analysis of variance (Kirk, 1982) revealed a significant task by group interaction [ $F(1,8) = 5.67, p < .05$ ], suggesting that the subjects' performance on the stress-production tasks differed according to group membership.

Fig. 28-1. Group means and standard deviations (shown in parentheses) for the LAL and normal subjects' scores on the stress-production tasks.

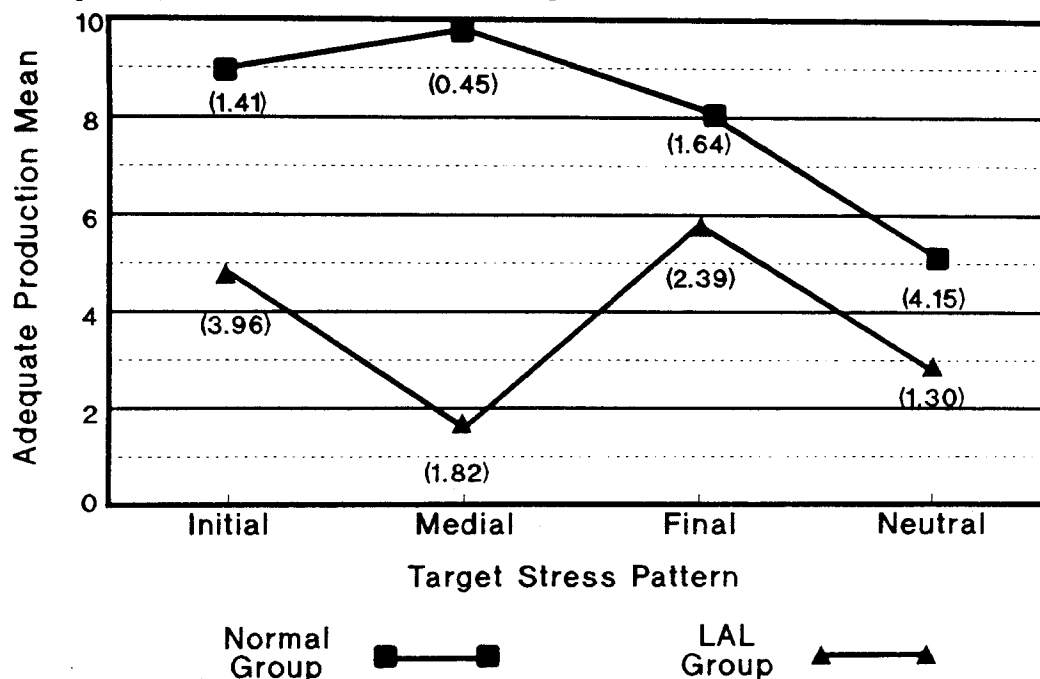


Based on the six listeners' perceptual judgments, the LAL group was significantly inferior [ $F(1,8) = 23.78, p < .005$ ] to the controls in spontaneously producing the target stress patterns. As well, the LAL group performed significantly poorer [ $F(1,8) = 19.17, p < .005$ ] than the normal controls in imitatively producing the stress patterns. Differences in the groups' ability to vary their stress placements are most apparent from inspection of Figures 28-2 and 28-3, which shows the groups' means for adequate production of each target pattern in the spontaneous and the imitative tasks, respectively. In both tasks, the LAL group averaged fewer adequate productions of each of the target stress patterns than did the control subjects.

Analysis of the groups' errors in the production tasks indicated that the LAL subjects had greater difficulty overall than the control subjects in producing sentences that were differentiated appropriately as to the neutral pattern with equal stress and the target patterns involving specific stress placement. This trend is illustrated clearly by the summary of the groups' error data for the imitation task in Table 28-4.

Comparison of the LAL groups' mean adequacy score for the two experiments indicated that the subjects' spontaneous stress-production ability was significantly inferior [ $F(1,8) = 33.76, p < .001$ ] to their imitation skills. As reflected by their performance data in Figures 28-2 and 28-3 the LAL group averaged fewer adequate productions of each target

Fig. 28-2. LAL and normal groups' average number of adequate spontaneous productions of each target stress pattern (according to the perceptual analysis). Group standard deviations are shown in parentheses.



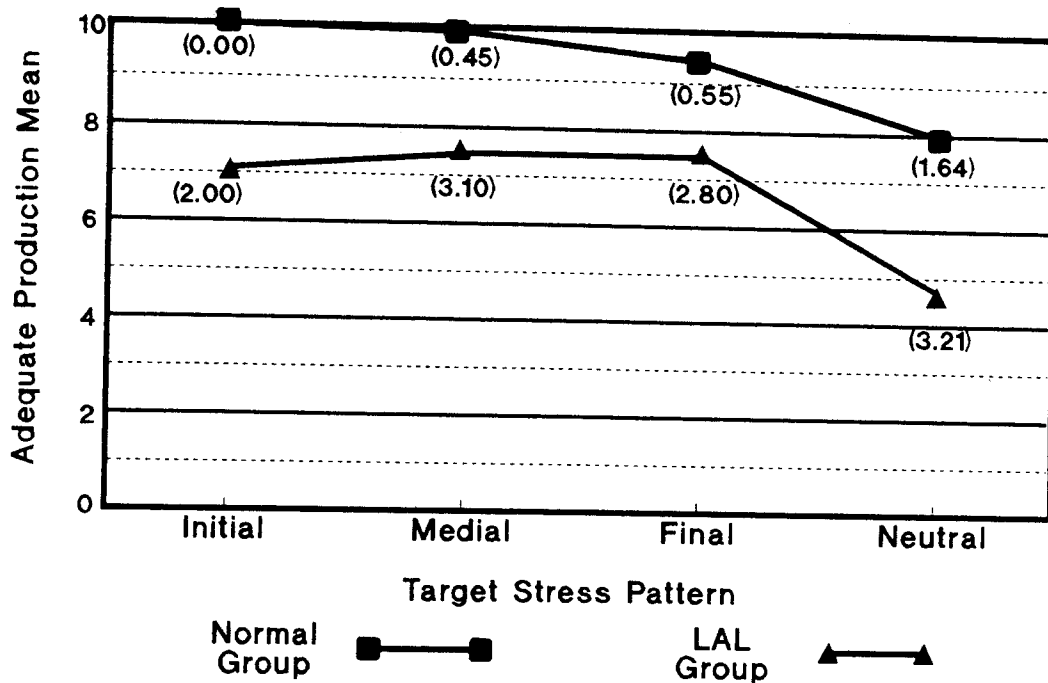


Fig. 28-3. LAL and normal groups' average number of adequate repetitions of each target stress pattern (according to the perceptual analysis). Group standard deviations are shown in parentheses.

stress pattern in the spontaneous task than in the imitative task. It is notable that in both tasks, the LAL subjects tended to show the same substitution pattern in response to each target pattern (e.g., neutral pattern used instead of initial stress placement).

## DISCUSSION

From the results of this study, it can be inferred that there may be commonalities in the propositional stress-production skills of patients with focal damage to the left frontal opercular region. The findings suggest that these patients may exhibit marked disturbances in encoding stress placement in short utterances, regardless of the target stress pattern. The results also indicate that the patients may show significantly reduced ability to produce variations in propositional stress placement in both spontaneous and imitative speech. These findings extend the results of previous studies that have documented disturbances in LAL patients' propositional stress-production skills (Kent and Rosenbek, 1983; Emmorey, 1987) and provide support for the view that abnormal stress patterning is a prominent symptom of LAL patients' dysprosodic output



**TABLE 28-4. LAL AND NORMAL GROUPS' AVERAGE NUMBER OF INADEQUATE IMITATIVE PRODUCTIONS AND SUBSTITUTION PATTERN FOR EACH TARGET STRESS PATTERN**

<i>Group</i>	<i>Target pattern</i>	<i>Average number of inadequate productions</i>	<i>Substituted pattern*</i>
LAL	Initial	3.00	Neutral
	Medial	2.60	Neutral
	Final	2.60	Neutral
	Neutral	5.40	Final
Normal	Initial	0.00	
	Medial	0.02	Final
	Final	0.06	Medial
	Neutral	2.20	Final

\*The substituted pattern occurred in 80 to 100 percent of a group's inadequate productions of the target pattern.

(Benson, 1967). In addition, the present findings are consonant with the view that disturbances in linguistic prosody may be a primary communication disorder associated with left anterior cerebral damage (Kent and Rosenbek, 1982, 1983).

The current findings are among the first to provide evidence that LAL patients may demonstrate significant differences in their spontaneous and imitative stress-production skills. The results indicate that perceptual inadequacies in the patients' spontaneous and imitative stress placement, such as the use of equalized stress, may be similar in type but occur most often in spontaneous speech. It is possible, therefore, that consistent use of imitative tasks in the study and clinical management of LAL patients' stress-patterning deficits may influence the resulting measures and description. In particular, reliance on imitative tasks may result in inflated measures of LAL patients' stress-production skills and a false impression of the efficiency of their verbal communication. Thus it may be useful to reevaluate and modify many of the current techniques for assessing and treating LAL patients' abnormal stress patterning, which are based solely on imitation of verbal stimuli.

## ACKNOWLEDGMENTS

The authors wish to thank Dr. Neil Graff-Radford for his assistance in locating the subjects for this study. A special note of thanks is due to

Dr. Hanna Damasio for providing the lesion localization data. This research was supported in part by NIH Grant No. PONS19632, on which the second author is an investigator.

## REFERENCES

- Behrens, S. (1988). The role of the right hemisphere in the production of linguistic stress. *Brain and Language*, 33, 104–127.
- Benson, D. (1967). Fluency in aphasia: Correlation with radioactive scan localization. *Cortex*, 3, 373–394.
- Blumstein, S. (1973a). Some phonological implications of aphasic speech. In H. Goodglass and S. Blumstein (Eds.), *Psycholinguistics and aphasia*. Baltimore: John Hopkins University Press.
- Blumstein, S. (1973b). *A phonological investigation of aphasic speech*. The Hague: Mouton.
- Damasio, H. (1983). A computer tomographic guide to the identification of cerebral vascular territories. *Archives of Neurology*, 40, 138–142.
- Damasio, H. (1987). Vascular territories defined by computed tomography. In J. Wood (Ed.), *Cerebral blood flow: Physiologic and clinical aspects*. New York: McGraw-Hill.
- Danly, M., and Shapiro, B. (1982). Speech prosody in Broca's aphasia. *Brain and Language*, 16, 171–190.
- Darley, F., Aronson, A., and Brown, J. (1975). *Motor speech disorders*. Philadelphia: Saunders.
- DeRenzi, E., Pieczuro, A., and Vignolo, L. (1966). Oral apraxia and aphasia. *Cortex*, 2, 50–73.
- Emmorey, K. (1987). The neurological substrates for prosodic aspects of speech. *Brain and Language*, 30, 305–320.
- Goodglass, H., Quadfasel, F., and Timberlake, W. (1964). Phrase length and the type and severity of aphasia. *Cortex*, 1, 133–153.
- Goodglass, H., Fodor, C., and Schulhoff, C. (1967). Prosodic factors in grammar: Evidence from aphasia. *Journal of Speech and Hearing Research*, 10, 5–20.
- Goodglass, H., and Kaplan, E. (1983). *The assessment of aphasia and related disorders*, 2d Ed. Philadelphia: Lea & Febiger.
- Kent, R., and Rosenbek, J. (1982). Prosodic disturbance and neurologic lesion. *Brain and Language*, 15, 259–291.
- Kent, R., and Rosenbek, J. (1983). Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, 26, 231–249.
- Kerschensteiner, M., Poeck, K., and Brunner, E. (1972). The fluency-nonfluency dimensions in the classification of aphasic speech. *Cortex*, 8, 233–240.
- Kertesz, A., Ferro, J., and Shewan, C. (1984). Apraxia and aphasia: The functional-anatomical basis for their dissociation. *Neurology*, 34, 40–47.
- Kirk, R. (1982). *Experimental design: Procedures for the behavioral sciences*, 2d Ed. Monterey, CA: Brooks Cole Publishing.
- Knopman, D., Selnes, O., Niccum, N., Rubens, A., Yock, D., and Larson, D. (1983). A longitudinal study of speech fluency in aphasia: CT correlates of recovery and persistent nonfluency. *Neurology*, 33, 1170–1178.

- LaPointe, L., and Wertz, R. (1974). Oral-movement abilities and articulatory characteristics of brain-injured adults. *Journal of Perception and Motor Skills*, 39, 39-46.
- Lebrun, Y., Buysens, E., and Henneaux, J. (1973). Phonetic aspects of anarthria. *Cortex*, 9, 126-135.
- Marquardt, T., and Sussman, H. (1984). The elusive lesion: Apraxia of speech link in Broca's aphasia. In J. Rosenbek, M. McNeil, and A. Aronson (Eds.), *Apraxia of speech: Physiology, acoustics, linguistics, management*. Austin, TX: PRO-ED.
- Mazzochi, F., and Vignolo, L. (1979). Localization of lesions in aphasia: Clinical CT scan correlations in stroke patients. *Cortex*, 15, 627-654.
- Mohr, J. (1973). Rapid amelioration of motor aphasia. *Archives of Neurology*, 28, 77-82.
- Rosenbek, J., and Wertz, R. (1976). Veterans administration workshop on motor speech disorders. Unpublished manuscript, Madison, Wisconsin.
- Rosenbek, J. (1985). Treating apraxia of speech. In D. Johns (Ed.), *Clinical management of neurogenic communicative disorders*. Austin, TX: PRO-ED.
- Ross, E., and Mesulam, M. (1979). Dominant language functions of the right hemisphere? Prosody and emotional gesturing. *Archives of Neurology*, 35, 144-148.
- Ryalls, J., Joannette, Y., and Feldman, L. (1987). An acoustic comparison of normal and right-hemisphere-damaged speech prosody. *Cortex*, 23, 685-694.
- Shapiro, B., and Danly, M. (1985). The role of the right hemisphere in the control of speech prosody in propositional and affective contexts. *Brain and Language*, 25, 19-36.
- Schuell, H., and Sefer, H. (1973). *Differential diagnosis of aphasia: Revised*. Minneapolis, MN: University of Minnesota Press.
- Wertz, R., LaPointe, L., and Rosenbek, J. (1984). *Apraxia of speech in adults*. Orlando, FL: Grune & Stratton.