Aphemia
With Dysarthria or Apraxia of Speech?

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INTRODUCTION

The term aphemia emphasizes the "isolated loss of the ability to articulate words without loss of the ability to write and to comprehend spoken language" (Albert, Goodglass, Helm, Rubens and Alexander, 1981; p. 86). A recent paper by Shiff, Alexander, Naeser, and Galaburda (1983) depicts aphemia as a "syndrome of dysarthria following the appearance of small left frontal-lobe lesions" (p. 720). According to these authors, aphemia with dysarthria is a recognizable clinical syndrome with a predictable course and highly uniform symptoms. Initial mutism in these patients evolves into persistent dysarthria in the absence of any language impairment. Patients typically present some lower facial paresis that improves over time, but is not sufficient to explain the observed speech deviations. Key features of speech output include effortful articulatory struggle, speech articulation errors, and dysprosody. Cerebral lesions are typically small and confined to the cortical frontal operculum and/or frontal subcortical fiber systems. The description of aphemia with dysarthria offerred by Schiff et al. is almost a duplicate of that offered by Wertz, LaPointe, and Rosenbek (1984) for apraxia of speech. However, Schiff et al. reject the term apraxia of speech in referring to aphemic patients. The issues raised by the apparent terminological conflict include a better understanding of the speech deficits seen in aphemic patients. In this paper, we take the perspective that the expressive deficits in aphemia are poorly classified as a dysarthria, but do represent a distinct apraxia of speech. The specific purpose of this paper then, is to evaluate neurologic, linguistic, sensorimotor and acoustic aspects of a single aphemic patient with consideration for the concepts of dysarthria versus apraxia of speech.

CASE DESCRIPTION

JR was a 45-year-old black male who was admitted to the hospital on 10/30/84, following the sudden and complete loss of speech earlier that morning. Upon admission, he presented right perioral weakness and reduced gag reflex, but other cranial nerves were grossly intact. Neurological tests were unremarkable except for the presence of mild buccofacial apraxia. He was able to write but not to speak. Comprehension appeared adequate. The patient was right-handed, monolingual (English), and had completed education through the fifth grade. He remained as an inpatient for eight days. During that time neuroradiologic and language testing revealed a picture of a specific motor speech impairment accompanied by a small frontal left-hemisphere lesion (Figure 1).

Neuroradiologic Findings. CT scan taken on the day of admission was unremarkable. A repeat scan taken eight days later identified a luxury perfusion lesion in the areas of pars opercularis in the inferior frontal gyrus and the inferior prerolandic gyrus in the left hemisphere. The affected area could be identified only in one 10mm slice. Figure 1 presents a reproduction of the transection of the lesion area and a lateral perspective drawn according to procedures used by Mazzocchi and Vignolo (1978). Though it is not possible

to define the specific functional extent of this insult, the focal deficits appear to be located in the superior aspect of Broca's area and the inferior precentral gyrus.

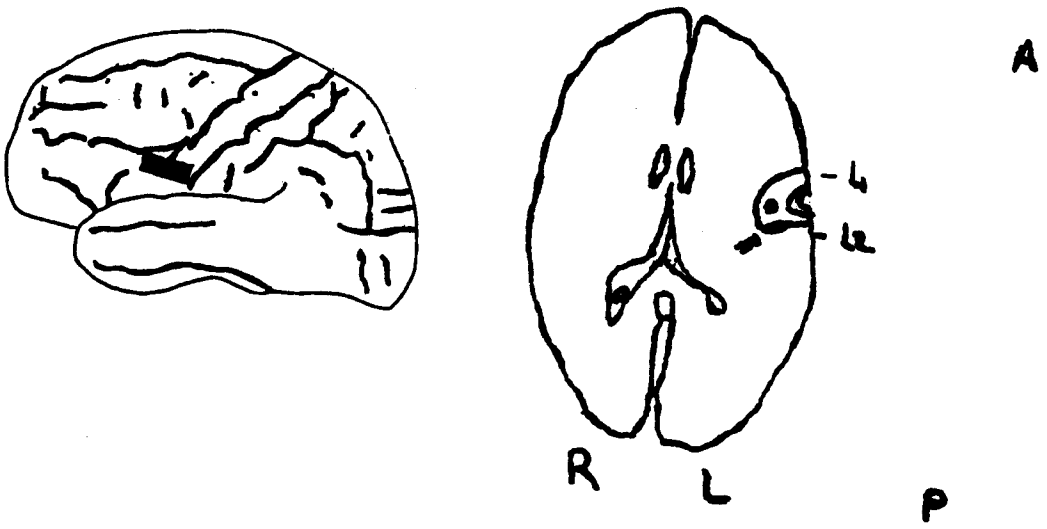

Figure 1. Graphic representation of JR's lesions drawn to scale from CT scan. Both transection (10 mm cut) and lateral mapping are shown.

Language Findings. Detailed language testing was initiated two days after onset on 11/1/84. Table 1 presents results of the Western Aphasia Battery (WAB: Kertesz, 1982). The patient obtained an AQ of 91.2, with lowest scores in writing and repetition. Fluency was scored high relative to grammaticality, but low relative to effort. The patient's verbal output was continually interrupted by pauses, postural fixes, articulatory groping and attempts at self-correction. Verbal output was limited at this time, but was grammatically appropriate.

Table 1. JR's Western Aphasia Battery profile on 11/2/84, three days after sudden loss of speech.

Information Content	9/10
Fluency	
Effort	2/10
Grammar	9/10
Comprehension	10/10
Repetition	8.6/10
Naming	9/10
Aphasia Quotient	91.2

Supplemental language testing was completed to identify any subtle linguistic deficits. The overall obtained score on the Revised Token Test (RTT: McNeil and Prescott, 1978) was 14.7, with a range of 14.2 to 15.0. Testing for specific syntactic comprehension revealed no difficulties with

passives, possessives, with/to agency, prepositions of direction, prepositions of time, plurals, or direct/indirect objects. Elaborate grammatic expression was not evaluated beyond the basic aphasia battery. Phonologic/articulatory analysis identified only limited segmental errors incorporating fricatives, liquids and clusters. In brief, this patient had no detectable language limitations. His sole problems were speech articulation and prosody deficits, apparently secondary to motoric impairments.

Oral Sensorimotor Findings. In an attempt to delineate sensorimotor limitations that might have contributed to the observed speech difficulties, a battery of objective measures was completed six days after onset (11/6/84). At this time, the only motor deviation noted was the presence of lingual-mandibular dependency. This symptom was most pronounced when the mandible was stabilized through use of a bite-block. Under this condition lingual movement for speech or nonspeech movements demonstrated significant postural groping.

The oral motor battery provided for an assessment of maximum strength of bite force (interdental), lip force and lingual protrusive force. Sensory measures included an assessment of labial and bite force discrimination, replication of jaw position, interdental thickness discrimination, and oral stereognosis.

Strength Measures. Measures of biting force and bilabial compression were accomplished through use of a straingauge scale (Williams, LaPointe and Blanton, 1984). The mechanism consists of two metal bars protruding from a small hand-held box. The end of each bar is covered with a thin plastic cap designed for contact with the teeth or lips. The scale can measure compression force exerted on the two bars in one-gram increments up to 10 kgms. To assess maximum strength, the patient was required to compress the bars with as much force as possible. In sampling bilabial strength of compression, two conditions were employed. One measure was obtained with the jaw free and one with the jaw stabilized by having the patient clench his teeth. This condition was added to evaluate potential assistance of the mandibular musculature in bilabial strength tasks. To evaluate potential asymmetry of strength in the labial musculature, maximum force measures were obtained in the midline and the right and left corners of the mouth opening.

The assessment of protrusive lingual force was accomplished by use of a modified tension-compression gauge. A plastic disk, one inch in diameter, was attached to the end of the gauge, which was then mounted to a fixed stand containing stabilization bars for the patient's forehead and chin. The plastic disk was positioned directly in front of the lips. The task required the patient to protrude his tongue against the disk with as much force as possible.

In order to provide a normal comparison to our patient's performance, five age-matched males with no present or past speech difficulty completed the same tasks. The average performance of these five control subjects was used for comparison.

Table 2 presents data for maximum bite force (interdental), maximum lip force and maximum lingual protrusive force. The obtained value for bite force was equivalent to that of the group of normal speakers, indicating no weakness in the musculature utilized in mandibular elevation. JR's performance on the labial compression tasks was superior to the average performance of the control group. However, an obvious right-left asymmetry was noted, with the midline being closer to the weakened right side. In addition, the lower values obtained in the jaw-stable condition suggested that he was using mandibular activity to assist in labial compression in the jaw-free condition. Finally, lingual protrusive force was reduced relative to the group of controls. JR's performance was less than one-half of that of the normal speakers.

Table 2. Results of maximum force assessment for mandibular, labial, and lingual mechanisms. JR's performance was compared to the mean value of five age-matched normal-speaking males. Values represent force of compression in grams.

Mechanism	JR (gms)	$\bar{X}$(S.D.)Norm(gms)
Mandibular (midline)	10 Kgm+	10 Kgm+
Labial (jaw free)		
Midline	1050	660(358)
Right	1033	780(503)
Left	1366	730(399)
Labial (jaw stable)		
Midline	766	639(406)
Right	750	740(408)
Left	983	760(365)
Lingual (midline)	208	562(95)

Force Discrimination Measures. Measures of bite and bilabial force discrimination were assessed using the same strain-gauge mechanism that was employed in the maximum strength tasks. This device has the capability to permit the investigator to change the amount of resistance between the two compression bars in one-gram increments. Also, compression force may be monitored on a modified voltage meter. In the force discrimination tasks, the patient was required to compress the bars with a preset level of resistance build in until the VU meter reached a zero setting. The amount of resistance between the bars was then increased or decreased by a fixed amount. The patient then completed a second compression of the bars again reaching a zero setting on the meter. The task required the patient to make a judgment as to whether the second compression required more, less, or the same amount of force to reach the zero setting. The amount of change in resistance between the first and second compressions was manipulated until a threshold of force discrimination or difference limen (DL) was identified.

Comparison of JR's performance with that of normal speakers was accomplished in two ways. For bite force discimination, JR's performance was compared to that of data obtained from six normal adult males published by Williams, LaPointe, Mahan, and Cornell (1984). For the labial task, unpublished data obtained from twenty young adults were used as a normal comparison. Only midline testing was completed on these normal speakers.

Table 3 presents data from the force discrimination tasks. In all cases, JR's performance was equivalent or superior to averaged data from the normal controls. Right-left asymmetries were noted in JR's data especially in the bilabial tasks. However, though performance asymmetries were noted, the overall performance on force discrimination tasks was superior to the performance of control normal speakers.

Supplemental Sensory Testing. Supplemental sensory testing was undertaken which evaluated the patient's abilities on tasks of oral stereognosis, the replication of jaw position, and interdental thickness discrimination. No deficits were identified in any of these sensory tasks.

Summary of Sensorimotor Findings. Collectively, the results of sensorimotor testing depicted an intact mandibular sensorimotor system, intact left and midline labial functions, and intact intraoral sensation. A right-left

Table 3. Results of force discrimination tasks for mandibular and labial mechanisms. JR's performance was compared to averaged data from six normal-speaking adults published by Williams, et al. (1984). Only midline comparisons were available. Values represent difference limen (DL) in grams.

Mechanism	JR(DL)	$\bar{X}$(S.D.)Norm(DL)
Mandibular		
Midline	100	339(206
Right	75	--
Left	100	--
Labial (jaw free)		
Midline	10	42(18)
Right	50	--
Left	30	--
Labial (jaw stable)		
Midline	15	44(38)
Right	50	--
Left	30	--

perioral asymmetry was noted in strength and force discrimination, and lingual protrusive force was reduced. Though these deviations cast doubt upon the integrity of labial and lingual sensorimotor systems, they do not deviate sufficiently from normal speakers to explain the pronounced speech abnormalities which remained at this time.

Speech Acoustic Findings. In an attempt to examine JR's speech production deficits more objectively, acoustic analyses were completed. JR read ten words within the carrier phrase "say _____ again." The target stimuli were chosen to represent pre- and post-vocalic distinctions for a variety of places of articulation. Three trials of each phrase were recorded and subjected to spectrographic analysis. Each spectrograph was evaluated relative to: (1) Initial segment duration (ISD). This measure was defined as the duration between the termination of the vowel in the word "say" and the spike-release of the initiating consonant in the target word. In this respect, ISD incorporated intersyllabic duration and initial consonant closure duration. (2) Voice onset time (VOT). This measure was defined as the duration between the spike-release and the onset of the vowel formant structure in the target word. (3) vowel duration (VD). This measure was defined as the duration of the total vowel formant structure including on-glide, steady-state, and off-glide. (4) Final segment duration (FSD). This measure was defined as the duration between the end of the vowel formant structure and the spike-release of the final consonant in the target word. In this respect, FSD represented the closure duration of the final consonant. All measures were made in millimeters and converted to milliseconds. Three samples were collected. The first was recorded six days after onset (11/5/84). The second eight days after onset (11/7/84). The final sample was recorded six weeks after onset (12/12/84).

Initial Segment Duration. Figure 2 presents data for change in initial segment duration. A systematic reduction of ISD is observed from the first to the third sample. This decrease in duration is evident for both voiced and voiceless consonants at all three places of articulation. Since ISD incorporates intersyllabic pause duration in addition to initial consonant closure

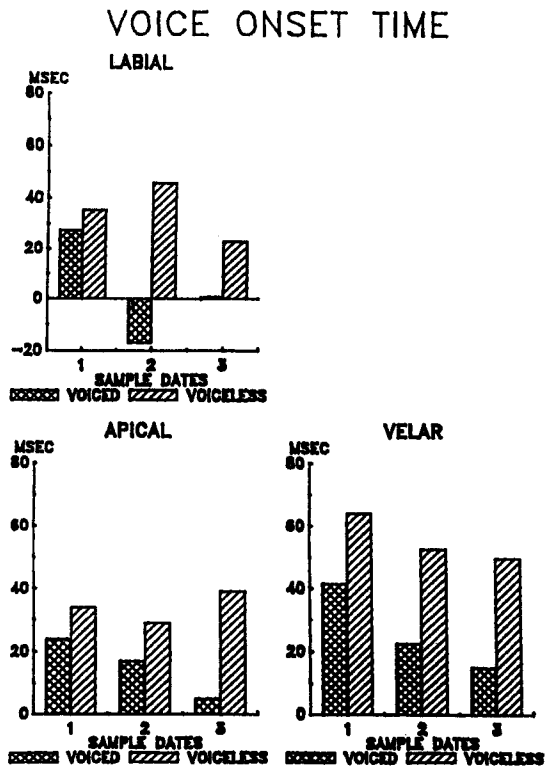

Figure 2. Mean values (msec) for initial segment duration of voiced and voiceless stimuli at labial, apical, and velar points of articulation. Samples were recorded five days (1), seven days (2), and six weeks (3) after onset.

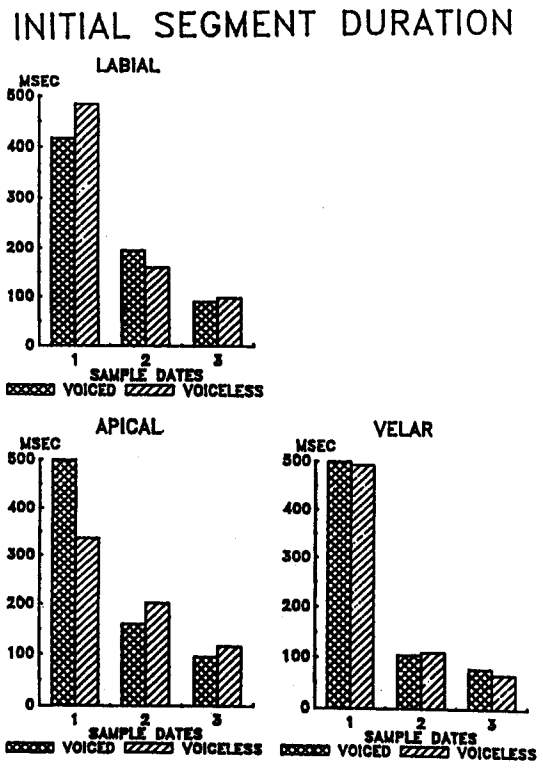

Figure 3. Mean values (msec) for voice onset time (VOT) of voiced and voiceless stimuli at labial, apical, and velar points of articulation. Samples were recorded five days (1), seven days (2), and six weeks (3) after onset.

duration, these data represent a change in prosodic as well as segmental production abilities. Kent and Rosenbek (1983) described a pattern of "syllable segregation" and prolonged consonant closure duration in their patients presenting apraxia of speech. The prolonged ISD values noted in the present case in the first sample identify similar production patterns in our patient. However, as a result of the speech timing changes noted over time, these production patterns are absent by the final sample.

Voice Onset Time. Figure 3 presents data for change in VOT. Initially, VOT facilitates only minimal differentiation of the linguistic voicing feature in prevocalic consonants. This result is due primarily to an increase of VOT duration in each voiced case and a decrease in VOT duration in each voiceless case (normal comparative data available from Shewan, Leeper, and Booth, 1984). A similar observation was made from the speech-apraxic patient of Freeman, Sands, and Harris (1978). Across samples, however, VOT values for voiced consonants were reduced in duration, establishing discrete voicing categories. VOT values for voiceless consonants were more variable over time. However, with the exception of apical consonants, these values also demonstrated decreased duration from sample one to sample three

Vowel Duration. Figure 4 presents data for change in vowel duration (VD). Only vowels from stimulus pairs containing a final voicing contrast were included in this analysis. Two observations may be made from these data. First, vowel duration was reduced over time in all contexts. Second, in the initial sample, vowel duration did not facilitate a contrast of the voicing feature in post-vacalic consonants. Typically, vowels preceding voiced consonants have a greater duration than vowels preceeding voiceless consonants (Clatt, 1973; 1976). This expected pattern began to emerge in the second sample and was well established by the third sample. In comparing JR's performance to comparable data published by Duffy and Gawle (1984) we noted that the emerging pattern was not that of a progression toward normal vowel durations. Rather, the pattern was that of a reduction of vowel durations in both voicing contexts, but with the establishment and maintenance of the post-vocalic voicing distinction over time. This pattern may reflect the patient's attempts to execute a known linguistic rule in the presence of initial and residual motor speech timing limitations.

Final Segment Duration. Figure 5 presents data for change in final segment duration. These data are very similar to those for initial segment duration, presenting a gradual reduction of values in all contexts over time. As with the ISD data, we interpret change in FSD to reflect an increase in the control of segmental-articulatory timing.

DISCUSSION

Two primary issues may be raised relative to our patient's performance. (1) Was his expressive deficit a dysarthria or an apraxia of speech? (2) What possible mechanisms or limitations might explain the observed deficits?

Darley, Aronson, and Brown (1975) remind us that the speech deviations in dysarthria "are directly attributable to [these] alterations in muscle function" (p. 251). No evidence of significant sensorimotor dysfunction could be identified in the present case. He did present a reduction in right perioral strength and lingual protrusive force. However, it is unlikely that these selective limitations could have been the sole factor responsible for JR's initial mutism or the observed severe speech difficulties that followed. We would like to offer the argument that our patient presented an apraxia of speech manifest in a deficit in the ability to control the temporal sequencing

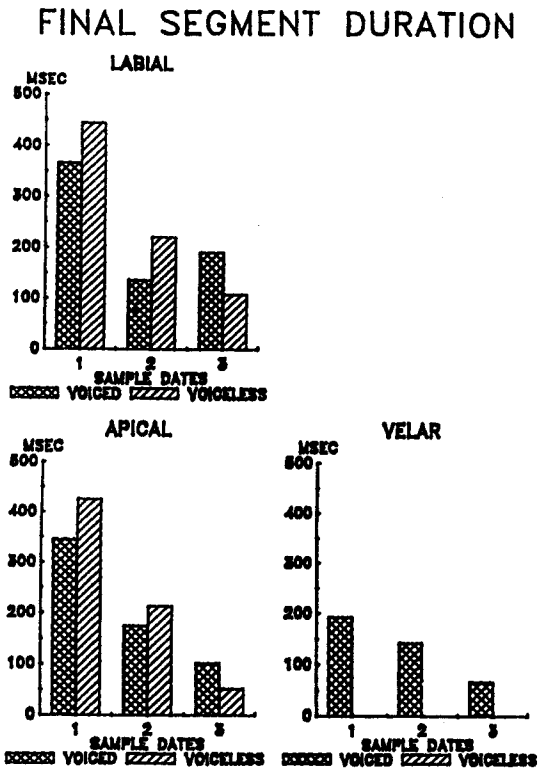

Figure 4. Mean values (msec) for vowel durations in stimulus pairs containing postvocalic voicing contrasts. No postvocalic velar contrasts were included. Samples were recorded five days (1), seven days (2), and six weeks (3) after onset.

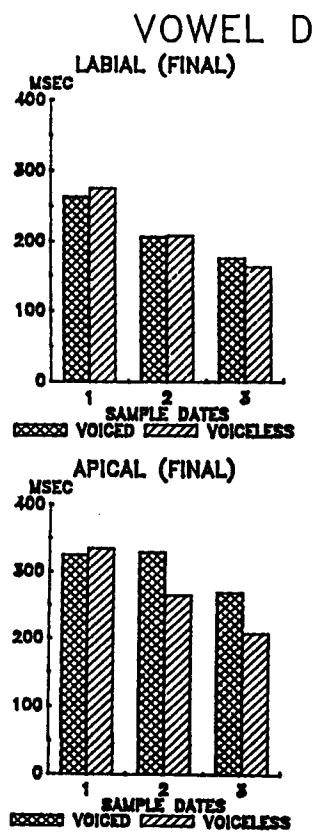

Figure 5. Mean values (in msec) for final segment durations of voiced and voiceless stimuli at labial, apical, and velar points of articulation. Samples were recorded five days (1), seven days (2), and six weeks (3) after onset.

of motor activity required for speech, rather than dysarthria secondary to sensorimotor dysfunction.

The position we offer with reference to apraxia of speech is, for the most part, formed by the acoustic analyses of motor speech timing changes in our patient. Following initial mutism he demonstrated increased intersyllabic and segment durations that contributed to the elimination of certain linguistic/phonologic properties of speech. These timing errors may represent an underlying deficit in the execution or coordination of discrete motor activities required for speech. The area of brain involvement in this case would support such a contention. Stimulation of inferior frontal cortex is known to produce an executive block to all motor activity, including speech (Ojemann and Mateer, 1979). A sudden-onset lesion in the speech control centers in this area could have similar effects; i.e. the initial mutism might have been secondary to an executive motor block of all speech activity. Also, in studying limb apraxia secondary to small left premotor lesions, many investigators have reported executive deficits. Historically, limb-kinetic apraxia has been considered to lie intermediate between a paresis and a true apraxia (Hecaen and Albert, 1978). The symptoms include "difficulty...in isolated movements, with loss of fine movements, or partial movements of a sequence" (Brown, 1972; p. 180). Kleist (in Hecaen and Albert, 1978) considered limb-kinetic apraxia (melokinetic) to be an executive deficit "resulting from an impaired ability to link or to separate closely related but independent muscle groups having separate innveration " (p. 107). Leipmann (in Brown, 1972) described a limb-kinetic apraxia of the vocal tract that was manifest in dyssynergistic functions among the components involved in speech. Motor timing characteristics previously reported in apraxia of speech include reduction in independent articulator movement (Shankweiler, Harris and Taylor, 1968) and temporo-spatial dyscoordination among multiple speech activities (Fromm, Abbs, McNeil and Rosenbek, 1982). JR's symptoms might represent a reduction of independent articulator movement. In the bilabial compression tasks there was evidence that he was using mandibular activity to assist in bilabial compression. This pattern was not observed in control subjects. Also, we noted a strong lingual-mandibular dependency in which pronounced lingual groping was evident when the mandible was stabilized with a bite-block. The speech timing deviations noted in JR's speech also may have been representative of underlying motor timing impairments. For example, a reduction in coordination between laryngeal and supralaryngeal articulations would create the observed VOT deviations (Kewley-Port and Preston, 1974). Though we are far from proving the existence of the underlying motor impairments, our data suggest a reduction in independent articulator movement and a dyscoordination of timing among multiple articulators. In this respect, we feel that the speech deficits demonstrated by JR represent an executive deficit of speech that would be akin to a limb-kinetic apraxia of the vocal mechanism.

Schiff et al. (1983) reject the term apraxia of speech as a descriptor for the executive deficit of speech noted in their aphemic patients because they "favor reserving it for learned motor actions that are impaired in some settings but not in others" (p. 726). In our opinion, this distinction is appropriate for an ideomotor variety of apraxia (e.g. Geschwind, 1975) but does not apply to the more executive variety traditionally referred to as limb-kinetic.

In summary, we would agree with many of the observations made by Schiff et al. (1983) regarding aphemic patients. We cannot, however, accept their position that the speech difficulties observed represent a dysarthric syndrome. We agree with Johns and LaPointe (1976) that an explanatory approach and label

to such problems must be undertaken. It is our position that the speech deficits noted, at least in our patient, represent an executive form of apraxia of speech.

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REFERENCES

Albert, M., Goodglass, H., Helm, N., Rubens, A., and Alexander, M. Clinical Aspects of Dysphasia. New York: Springer-Verlag, 1981.

Brown, J. Aphasia, Apraxia, and Agnosia. Springfield, IL: Charles C. Thomas, 1972.

Darley, F., Aronson, A., and Brown, J. Motor Speech Disorders. Philadelphia: W.B. Saunders, 1975.

Duffy, J. and Gawle, C. Apraxic speakers' vowel duration in consonant-vowel-consonant syllables. In J. Rosenbek, M. McNeil, and A. Aronson (Eds.), Apraxia of Speech: Physiology-acoustics-linguistics-management. San Diego: College Hill Press, 1984.

Freeman, F., Sands, E., and Harris, K. Temporal coordination of phonation and articulation in a case of verbal apraxia: A voice onset time study. Brain and Language, 6, 106-111, 1974.

Fromm, D., Abbs, J., McNeil, M., and Rosenbek, J. Simultaneous perceptual-physiological method of studying apraxia of speech. In R.H. Brookshire (Ed.), Clinical Aphasiology: Conference Proceedings, 1982. Minneapolis, MN: BRK Publishers, 1982.

Geschwind, N. The apraxias: Neural mechanisms of disorders of learned movement. American Scientist, 63, 188-195, 1975.

Hecaen, H. and Albert, M. Human Neuropsychology. New York: John Wiley and Sons, 1978.

Johns, D. and LaPointe, L. Neurogenic disorders of output processing: Apraxia of speech. In H. Whitaker and H. Whitaker (Eds.), Studies in Neurolinguistics (Vol. 1). New York: Academic Press, 1976.

Kent, R. and Rosenbek, J. Acoustic patterns of apraxia of speech. Journal of Speech and Hearing Research, 26, 231-249, 1983.

Kertesz, A. The Western Aphasia Battery. New York: Grune and Stratton, 1982.

Kewley-Port, D. and Preston, M. Early apical stop production: A voice onset time analysis. Journal of Phonetics, 2, 195-210, 1974.

Klatt, D. Interaction between two factors that influence vowel duration. Journal of the Acoustical Society of America, 54, 1102-1104, 1973.

Klatt, D. Linguistic uses of segmental duration in English: Acoustical and perceptual evidence. Journal of Acoustical Society of America, 59, 1208-1221, 1976.

Mazzochi, F. and Vignolo, L. Computer assisted tomography in neuropsychological research: A simple procedure for lesion mapping. Cortex, 14, 136-144, 1978.

McNeil, M. and Prescott, T. Revised Token Test. Baltimore: University Park Press, 1978.

Ojemann, G. and Mateer, C. Human language cortex: Localization of memory, syntax and sequential motor-phoneme identification systems. Science, 205, 1401-1403, 1979.

Schiff, H., Alexander, M., Naeser, M., and Galaurda, A. Aphemia: Clinical-anatomic correlations. Archives of Neurology, 40, 720-727,

Shankweiler, D., Harris, K., and Taylor, M. Electromyographic studies of articulation in aphasia. Archives of Physical Medicine and Rehabilitation, 49, 1-8, 1968.

Shewan, C., Leeper, H., and Booth, J. An analysis of voice onset time (VOT) in aphasic and normal subjects. In J. Rosenbek, M. McNeil, and A. Aronson (Eds.), Apraxia of Speech: Physiology-acoustics-linguistics-management. San Diego: College-Hill Press, 1984.

Wertz, R., LaPointe, L., and Rosenbek, J. Apraxia of Speech in Adults: The Disorder and Its Management. New York: Grune and Stratton, 1984.

Williams, W.N., LaPointe, L.L., and Blanton, R.S. Human discrimination of different bite forces. Journal of Oral Rehabilitation, 11, 407-413, 1984.

Williams, W.N., LaPointe, L.L., Mahan, P.E., and Cornell, C.E. The influence of T.M.J. and central incisor sensory impairment on bite force discrimination. The Journal of Craniomandibular Practice, 2, 119-124, 1984.

DISCUSSION

Q: What is the difference between aphemia with dysarthria and aphemia without dysarthria? For example, the neurology literature refers to dysarthria as any abnormality in articulation that is part of aphemia.

A: Your question reflects one of the focal points of our paper. The Schiff paper identified the articulatory disturbance associated with aphemia as a dysarthria. Yet, as we understand dysarthrias, they are typically associated with motor disturbance in the vocal tract. In the present case, there was no evidence of motor impairment in the vocal tract sufficiently severe to explain the speech deviations. We do not argue with the term aphemia as a descriptor for speechless patients without language impairment -- only with the interpretation of the subsequent articulatory deficits. We do not agree that the presence of an articulatory deficit automatically implicates dysarthria.

Q: I was interested in your observation that the strength and force differences you found were not sufficient to explain the speech difficulties. Would you explain to me the data on which that distinction was made?

A: Basically, the only deviation demonstrated by our patient from the normal-speaking controls was in the lingual protrusive force task. In this case his performance was more than two standard deviations below our small group of controls. Though I can't prove it, it is difficult for me to conceive that a weak tongue alone could create the degree of dysprosodia and articulatory groping that was noted in JR's speech. JR did demonstrate a right-left asymmetry for bilabial tasks. However, his performance was within the realm of "normalcy" established by our control group.

Q: Do you have any data that would indicate how much deviation on these tasks would be required before the results could be attributed to observed symptoms?

A: No. We recently have begun to test patients with confirmed neuromotor disorders involving the vocal mechanism but our data are insufficient to draw any conclusions.

Q: Could you give us an idea of what the patient sounded like?

A: The primary perceptual feature would most likely be dysprosodia. He spoke with an equal and even stress and loudly. The rhythm of his speech was continually interrupted by postural fixations, groping of the articulators and attempts at self-corrections. His articulatory errors were limited and mostly confined to fricatives, clusters and liquids. Also, voicing errors were frequent as indicated by the acoustic data.

Q: Were his errors more phonetic or phonemic and was automatic speech better than spontaneous speech?

A: His errors were more of the phonetic type. We did not complete a specific comparison of automatic speech to spontaneous speech, but my impression is that there would have been no difference.

Q: Some people who have reviewed the literature in this area have suggested that all aphasic adults would have some sort of phonological selection problem and that the more anterior patients may, in addition to that, show an articulatory programming aspect. It would appear that you were trying to separate the motor speech aspects. Was there any consideration of a phonological selection disorder in this patient?

A: No. His articulatory errors were so few and confined to specific sound classes that this consideration was never undertaken. Also, there was no evidence of language impairment, which I think would speak against the possibility of a phonological selection impairment in this case.

Q: Did you administer any standardized or published test for apraxia of speech?

A: No. First of all, I am not aware of any standardized test for apraxia of speech. Second, I'm not sure if some of the published batteries would address the range of possibilities that different types of apraxias would present. I think that the best we can do at present would be to document a pattern of response behaviors across several tasks that we believe to be associated with apraxia of speech. My problem is that I am not confident that we know all there is to know about the clinical profiles of different apraxic speakers.

Q: Geschwind didn't believe in apraxia of speech but he has defined apraxia, and I think speech pathologists have talked about apraxia as being the inability to produce a voluntary movement in response to a stimulus that would normally elicit it. If he made the substitution of /t/ for /s/ in the word "say," would he be able to produce the word correctly in another setting?

A: I'm going to answer your question indirectly because it gets at an issue that is now being raised relative to apraxia of speech in both adults and children. The context-sensitive response attributed to apraxia of speech is an outgrowth of the work in ideomotor limb apraxia. We know, for example, that if we ask an adult with ideomotor apraxia to show us how he uses a screwdriver that he may not be able to demonstrate its use. However, if given an actual screwdriver as a prop, this same patient may perform flawlessly. This is somewhat analogous to the issue of the effects of different types of speech elicitation techniques. In the type of apraxia that we have suggested to explain JR's speech deficits, no such context-sensitive changes in performance would be expected. This was one of Geschwind's objections and it has been handed down to others who object to the concept of apraxia of speech on similar grounds. Our point is that we

must look at other behaviors and begin to accept that more than one type of apraxia probably exists and that this differentiation may apply to deficits in speech production. Historically, context-sensitive performances are not characteristic of the executive variety of apraxia commonly termed limb-kinetic.