The Syndrome of Subcortical Apraxia of Speech: An Acoustic Analysis (Abstract)

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Reports of pure motor aphasia resulting from subcortical lesions deep to Broca's area have intermittently appeared in the literature during this century. Marie (1906) reported that lesions in the region surrounding the lenticular nucleus or in the anterior limb and knee of the internal capsule resulted in anarthria. Dejerine (1926) reported that pure motor aphasia resulted not from damage to Broca's area itself but from damage to association fibers connecting Broca's area to the rolandic operculum or to bulbar nuclei.

Several contemporary reports of anarthria or pure apraxia of speech have supported the views of Marie (1906) and Dejerine (1926). That is, in patients with apraxia of speech, Broca's area may be spared with damage occurring subcortically (Goodglass and Kaplan, 1972; Lecours and Lhermitte, 1976; Mazzocchi and Vignolo, 1978; Square, 1981; Square, Darley and Sommers, 1982). Others have reported that in cases of pure apraxia of speech which ameliorate quickly, Broca's area is only minimally involved (Mohr, et al., 1978).

We identified a 49-year-old female patient who had sustained an infarct to the left basal ganglia, especially the caudate nucleus. Site of lesion was confirmed by CT scan. Neurobehavioral correlates included only mild right hemiplegia and mild right facial weakness. No aphasic impairment was measurable. Overall PICA score was 14.41 with modality means being 14.47, 14.79 and 14.41 on verbal, gestural, and graphic subtests, respectively. No errors were made on the Token Test nor the Keenan and Brassell sentences. Forward digit span was seven and backward span was six.

The patient was judged by speech pathology consultants at the Mayo Clinic to demonstrate pure apraxia of speech, because the following behaviors characterized her speech output:

1. Inconsistent articulatory errors with more occurring on phonetically complex and longer stimuli.
2. Prosodic aberrations including slowed rate, even stress, inaudible and audible gropes, phoneme reapproaches, syllable segregation, prolonged phonemes (especially vowels), and struggle behaviors.
3. Islands of error free production and acute awareness of errors accompanied by attempts to self-correct which were inconsistently successful.

A number of experienced speech clinicians were asked to listen to the speech of this patient. A great proportion of these listeners asked if this patient was foreign. She wasn't. It was presumed that she had a mid-western dialect premorbidly. A small proportion questioned if this patient's aberrant speech was due to dysarthria. However, aside from a mild right facial weakness, no significant weakness, slowness, or incoordination could be demonstrated.

Recently, several groups of researchers have alluded to the existence of clinical subvarieties or subtypes of verbal apractic patients (Square, Darley, and Sommers, 1982; Rosenbek, et al., 1981). It has been hypothesized that subcortical lesions may interrupt cortico-cerebellar-cortico tracts and possibly even the coalitional influences of the gamma motoneuron regulatory influences on speech production (Abbs, 1973). For these reasons, we felt it necessary to fully specify the characteristics of this patient's unique, apractic-like speech. This was accomplished retrospectively by acoustically analyzing samples of this patient's speech.

The purposes of the present investigation were:

1. To acoustically analyze monosyllabic, polysyllabic, and continuous speech samples of this subject, who had a lesion confined to the caudate nucleus and who was judged to demonstrate apraxia of speech and no measurable aphasia.
2. Compare the results of the acoustic analysis of this patient's speech to those obtained for a normal adult female and those reported previously in the literature for apractic, pseudo-foreign dialects, and dysarthric (particularly ataxic) patients.
3. Determine if the characteristics of the acoustic analysis of the speech of this patient more clearly represented apraxia of speech or dysarthria.

The results of this descriptive investigation were in general agreement with those of Kent and Rosenbek (1982) for apractic speakers. Our patient's production of isolated words as well as continuous speech was generally prolonged. However, this durational increase was not found to result from prolongation of all segments but varied from word to word. For instance, in some words the lengthening was primarily due to consonant prolongation (i.e., prolongation of VOT or frication) while in others it was due to prolongation of the steady state portion of the word (i.e., vowel lengthening). In still others the placement of inappropriate pauses before voiceless fricatives and affricates in the final position was responsible for abnormal word lengthening.

Our patient often imposed additional features on segments not normally found. She would consistently produce frication and/or aspiration at the end of voiced stop plosives in word final position. She produced intermittent voicing during the production of voiceless fricatives and affricates and the use of the additive schwa. Articulatory variability was also noted when the spectrograms of the same words were compared across different modes of elicitation (i.e., reading, naming, and repeating) and across self-repeated trials (i.e. successive approximations or conduite d'approache to the target word).

When our patient's continuous speech was compared to that of speakers with apraxia of speech and ataxic dysarthria, certain similarities and differences were noted. Like the apractic speaker, our patient's speech was characterized by syllable segregation (i.e., temporal separation of syllables in a syllabic series) especially when approaching and/or producing multisyllabic words. This pattern was not apparent when the patient produced strings of monosyllabic words in a sentence. However, it was found that acoustically her speech was characterized by a relatively flat fundamental frequency contour, a pattern similar to that of ataxic dysarthric speaker-(Kent, Netsell and Abbs, 1979; Kent and Rosenbek, 1982).

Overall, it is our opinion that the acoustic description of our patient's speech offers support to the notion that apraxia of speech can be caused by subcortical lesions. Whether our patient is qualitatively different from

those patients whose apraxia is due to cortical lesions is uncertain. The presence of a pseudo-foreign dialect may be a key to this differentiation. Further case studies describing individuals with subcortical lesions resulting in apraxia of speech are needed.

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Q: I wonder if you would tell us when your scan was done, who read the scan, with what reliability, what kind of scanner was used, and how much information the readers or reader of that scan had about the other clinical sumptoms of that patient?

A: First, the readers had no knowledge of the patient's symptomatology, language or speech-wise. Second, the scan was done at about six months post-onset. Third, I have no idea of the reliability of the reading. We've been trying to get the scan, but we can't locate the patient to obtain her permission to release this information. This was a patient from the Sister Kinney Institute in Minneapolis who is apparently now living in Wyoming. There may be no problems with the readings, but we would like to establish the reliability of them before publishing these data.

Q: What did your patient sound like? I wish you'd brought a tape . . .

A: I did bring a tape, and anybody who would like to view it is more than welcome to. This patient sounded pseudo-foreign and apractic. It's interesting, because Richard Peach, in his presentation the other day of subcortical patients, said that their patient had reduced articulatory agility, but sounded dysarthric. Several speech clinicians who listened to our patient said she sounded dysarthric. We assumed this was because of her vowel distortions which, according to Alajouanine et al. (1939), in their report of a similar patient, were dystonic in nature. A lot of other speech clinicians who listened to our patient said, "No, that's apraxia we hear." I think this is an issue we have to grapple with. What is this disorder? We do see these kinds of patients on occasion. They are the types of patients who have minimal aphasia and strange apractic-like speech with trial and error groping, extreme struggling, reapproaches, and yet some of the phonetic aspects of the speech are reminiscent of dysarthria. Nevertheless, our patient, when asked to program longer utterances, longer phrases and sentences, really struggled. I rarely observed islands of error-free speech, although there were, on occasion, single words which sounded fairly normal. In other words, the longer the utterance that had to be programmed, the worse this patient's speech became.

Q: If I recall your introduction of that topic in the paper, you said that the patient had a "mild facial weakness," but that no other weakness nor abnormal tone nor discoordination could be demonstrated for the oral musculature.

A: That's right. Clinically, we could not demonstrate neuromuscular dysfunction.

Q: Could not be demonstrated by whom?

A: Several of us on different occasions, independently, did a full motor speech examination a la Mayo Clinic, and could not demonstrate dysfunction.

Q: Mild facial weakness, then, does not necessarily mean dysarthria?

A: This is another issue we have all grappled with. We all know that a lot of these patients who have resolved all of their speech disabilities other than fricative distortions, and who have mild right facial weakness

and a unilateral tongue weakness, of course, have a dysarthria. But we couldn't demonstrate any significant problems on either our nonspeech motor or motor speech exam, of muscular incoordination, weakness, or slowness.

Q: The more fine-grained our analysis, the more careful our subject selection is going to have to be. We can make instrumental and other kinds of analyses and we're going to find all kinds of aberrations in that system, and I think it's important that we make very careful patient selection in light of this technology.

A: I agree with that. However, when one is dealing with neurogenic patients, how clean is clean? We can only make our judgments based upon our best clinical tools at the time. And the best clinical tools at this time are thorough motor speech examinations for both nonspeech and speech activities.

Q: I'm curious about the PICA score. That was unbelievably high for someone you would think to be an apractic. Can you explain that?

A: Yes. Her one-word responses were generally quite good and unless transcribed and listened to carefully appeared to be quite normal. She performed much worse on Subtest 1, but I didn't bring these data. Another PICA, administered one year later, demonstrated a somewhat lower verbal PICA mean, around 13, instead of 14. Two different administrators gave these tests. I didn't give either test. But, the one thing I would like to stress is that one-word responses emitted by this patient often sounded quite normal. When, however, transcriptions were made, and we really listened to her speech, we could hear all kinds of distortions, especially those of VOT. These aberrations were not necessarily obvious on single word responses which were not closely analyzed, and may not have been scored as abnormal by a PICA administrator.

Q: I'm interested in your normal subject. How old was she?

A: Forty-nine, the same age as our pathologic subject.

Q: I was interested because she showed a lack of variability in VOT productions, and many of the normal subjects whom we have tested demonstrated a somewhat greater variability of VOT productions at that age.