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Phonemic characteristics of apraxia of speech resulting from subcortical hemorrhage

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Abstract

Reports describing subcortical apraxia of speech (AOS) have received little consideration in the development of recent speech processing models because the speech characteristics of patients with this diagnosis have not been described precisely. We describe a case of AOS with aphasia secondary to basal ganglia hemorrhage. Speech-language symptoms consistent with subcortical AOS and aphasia are reported as well as description of the patient's speech characteristics using broad phonemic transcription. Significantly more phoneme substitution errors were observed than other types of errors and these occurred significantly more often in the initial rather than the medial or final positions of words. No differences were observed in the number of errors produced for the various types of phonemes included in this analysis. These findings challenge the hypothesis that speech motor planning disorders are uniquely the result of cortical damage. They also argue against the notion that subcortical AOS is indistinguishable from the cortical syndrome.

Learning outcomes: The reader will learn (1) the phonemic characteristics of AOS due to subcortical brain damage, (2) the similarities and differences between AOS due to cortical versus subcortical lesions, and (3) the implications of these patterns for current models of speech motor processing.

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Keywords: Apraxia of speech; Subcortical hemorrhage; Phonemic transcription

1. Introduction

Apraxia of speech (AOS) is a phonemic-motoric disorder of speech production that frequently coexists with aphasia but may, on occasion, appear in pure form (Ballard,

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Granier, & Robin, 2000; McNeil, Doyle, & Wambaugh, 2000; Square, Martin, & Bose, 2001). It is an impairment of volitional speech production characterized by effortful groping for articulatory gestures and difficulty with the initiation of utterances. It is further characterized by perceived substitutions, distortions, omissions, additions, and repetitions of phonemes and abnormalities in speech rate, prosody and fluency (Duffy, 1995).

Early studies of the phonological characteristics of AOS using broad phonemic transcription found (a) more consonant errors than vowel errors; (b) more substitution than distortion, omission, or addition errors; (c) more errors in word initial than word final position; (d) more errors of simplification than complication; (e) more single-feature than multiple-feature sound substitutions; and (f) more place than manner or voicing errors (McNeil, Robin, & Schmidt, 1997). With regard to phoneme type, consonant clusters are more frequently in error than singletons and affricates and fricatives are more frequently in error than other manners of production (Duffy, 1995). Recent accounts (McNeil et al., 1997, 2000) have suggested that AOS is characterized primarily by distortions of segments and intersegment transitionalizations that are perceived as sound substitutions and by inappropriate assignment of stress and other prosodic abnormalities. The symptoms of AOS are not attributable to deficits of muscle tone or reflexes or to deficits in the processing of auditory, tactile, kinesthetic, proprioceptive, or language information (McNeil et al., 2000).

To distinguish AOS from its related speech and language disorders, (Darley, Aronson, & Brown, 1975) described a three-level model of motor speech programming consisting of a central language processor (CLP), a motor speech programmer (MSP), and the downstream motor cortex (see Table 1). For language, the CLP selects the words and word sequences that transform meaningful content into language. For speech, the CLP identifies the phonemes and meaningful sequences of phonemes for language transmission. The CLP then converts these phoneme sequences into a neural code that drives the MSP. The MSP activates the appropriate musculature to produce the desired speech sounds in their proper order. The MSP then projects directly to the motor cortex.

Darley et al. attributed AOS to a disturbance in the programming of movements for speech. That is, the linguistic plans (syntactic, phonologic) for an intended message are

Table 1
Comparison of formal stages in Darley et al. (1975) and Van der Merwe (1997) models of speech motor processing

Darley et al. (1975)	Van der Merwe (1997)
Central language processor	Linguistic-symbolic planning
Phoneme identification	Selection of phonemes
Phoneme sequences	Combination of phonemes
Chains of neural codes computed to direct MSP	Motor planning
	Recall of core motor plans
Motor speech programmer	Motor programming
Activation of preprogrammed chains for specific muscles to produce desired speech	Selection of articulatory motor programs
Motor cortex	Execution
Speech execution	Muscle movements

assembled accurately but the speaker is unable to program the musculature for its motor expression. The difficulty then is not related to deficits in the neuromotor execution of speech (i.e., disturbances to the basic motor processes involved in muscular control of the speech mechanism), but rather to an inability to transform phonologic plans into a code that is recognized by the speech motor system.

More recently, Van der Merwe (1997) has described a four-phase model of speech sensorimotor control that makes explicit the planning and programming stages described by Darley et al. (see Table 1). In Van der Merwe's first phase, phonologic representations (i.e., phonemes) are activated and transformed to motor code during the motor planning or second phase of speech production. Motor planning involves activating and organizing the temporal and spatial specifications for the production of sequences of phonemes. Core motor plans for particular phonemes are adapted to the specific speech contexts in which they will appear and entered into subroutines that enable movement of the articulatory structures. In the third phase, the motor plan subroutines are fed-forward to the motor programming system where muscle-specific motor programs for articulatory movements are selected and sequenced. Motor programs specify spatio-temporal and force dimensions such as muscle tone, movement direction, force, range of movement, and rate. In the last phase, the plans and programs of the preceding phases are transformed to muscle movements. Following from this model, Van der Merwe conceived AOS as a motor planning disorder since (a) AOS is not attributable to deficits of muscle tone or reflexes and (b) motor programming deficits allow for alterations in muscle tone, force, direction, speed, and timing. The latter characteristics would instead be encompassed within the group of speech disorders known as the dysarthrias.

Close inspection of these models reveals an important difference between the two with regard to AOS. For Darley et al., the linguistic and phonemic planning functions associated with the CLP show little or no impairment while selective activation of the programming routines for the speech musculature is deficient. In the Van der Merwe approach, the fundamental deficit of AOS is found in the planning rather than the programming stage of speech motor production, i.e., at the point in Darley et al.'s CLP where neural codes are computed for specifying the spatial and temporal characteristics of the output (see Table 1). Accordingly, AOS is a motor programming disorder for Darley et al. resulting from damage to the hierarchically later MSP but a motor planning disorder for Van der Merwe that follows from damage to the earlier motor planning phase of speech motor production.

These theoretical accounts have important implications for identifying the neurological foundations of AOS. Despite the above differences, both Darley et al. and Van der Merwe assume that the specification and programming of motor speech output are mediated by the cortical association areas, i.e., the premotor cortex, the supplementary motor area (SMA), the prefrontal areas and the posterior parietal areas. Both accounts also suggest then that AOS should result from lesions to the cortical association areas while dysarthria should result from lesions to the basal ganglia and other lower segmental structures.

Mohr et al. (1978) determined that circumscribed damage to inferior premotor cortex (Brodmann's area 44, i.e., portions of Broca's area) produces AOS. Dronkers (1996) offered a strong hypothesis stating that the insula must be damaged in order for AOS to occur. Recent work, however, has failed to find support for this view (Jones, Peach, & Schneck, 2003). In their recent reviews, Square et al. (2001) and Duffy (1995) have

supported the traditional notion that AOS occurs following lesions to the frontal as well as the parietal association areas. But they also note that AOS may be the result of subcortical lesions as well. For example, Kertesz (1984) found AOS with aphasia in 10 participants with lesions restricted to the basal ganglia and the internal capsule. Marquardt and Sussman (1984) also described several cases of AOS with aphasia resulting from damage confined to subcortical structures. These findings create problems for the site of lesion account offered in the models of Darley et al. (1975) and Van der Merwe (1997).

However, drawing conclusions from these studies about the contributions of subcortical structures to motor speech production has been problematic. In both studies, as well as others like them, the characteristics of the participants' speech production were not described precisely using phonemic transcription to distinguish their speech errors from those that might be due to related disorders of speech production, e.g., phonemic paraphasia. As McNeil et al. (2000) have stated, under these circumstances, "it is difficult to have confidence in any relationships drawn between lesion locus and signs of AOS in these patients" (p. 231). Precise description of AOS due to subcortical damage is needed to address the debate concerning AOS and its neuroanatomical substrates. In this paper, we offer specific data regarding one such case.

2. Case report

2.1. Participant

A 71-year-old, right-handed man presented with acute loss of speech and right hemiplegia following a cerebral vascular accident. His previous medical history was unremarkable. CT scan performed 3 days post-onset demonstrated a massive left frontal hemorrhage probably originating in the region of the basal ganglia and extending into the frontal white matter (Fig. 1). There was no evidence for involvement of the lower half of the primary motor cortex, the posterior portion of the inferior frontal gyrus, the insula, or the postcentral opercular structures.

The patient was admitted to rehabilitation 10 days after his stroke. At the time of the initial speech-language evaluation, he was alert and inconsistently responsive. His communication status was characterized by muteness with only occasional simple gestures in apparent reference to his inability to speak or to understand questions. Comprehension performance consisted of undifferentiated pointing responses to auditory word discrimination tasks or visual word–picture matching tasks. No appropriate responses were observed for questions in either formal or informal conversational contexts. He occasionally followed functional commands when presented in an appropriate context. Writing was characterized by unintelligible scribble using the non-preferred hand.

The patient was enrolled in a program of speech-language treatment. Minimal vocalization was observed during the following 2 weeks; a few grammatically well-formed sentences were produced spontaneously during this period. Imitation of oral movements was impeded by bucco-facial apraxia. By the end of the fourth week post-stroke, he began to respond successfully to tasks eliciting automatic verbal responses although speech remained aphonetic. He was frequently frustrated by his inability to imitate speech

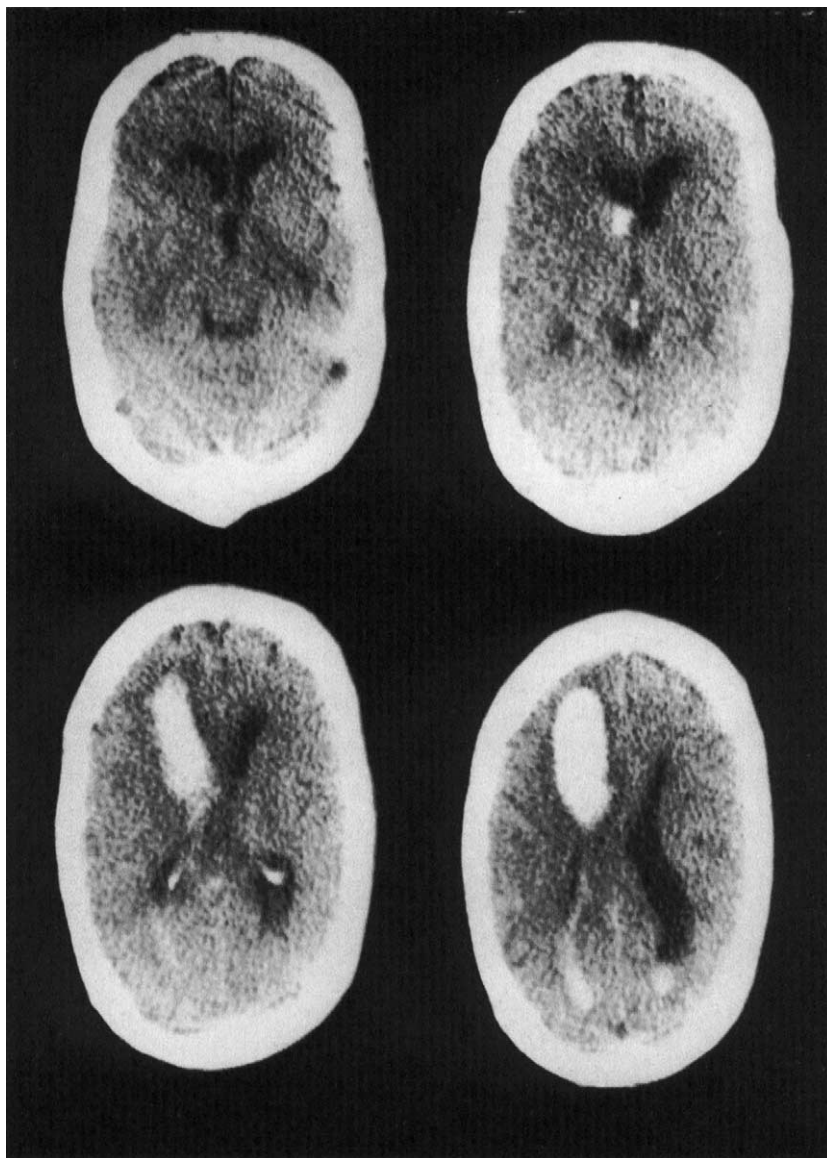


Fig. 1. CT scans demonstrating origin of basal ganglia infarct and anterior-superior progression.

consistently. Auditory comprehension for yes–no questions and word recognition was moderately impaired. He could spell one simple word out of three by pointing to an alphabet board. At approximately 5 weeks after his stroke, his speech output was much improved from his initial appearance but he continued to be dysfluent (reduced phrase length with poor grammatical complexity) and to have substantial difficulty initiating speech. He was echolalic and perseverative. However, occasional but more frequent

grammatically well-formed sentences of normal lengths were observed in conversation. He was able to read sentences aloud but with substantial difficulty. He used self-cuing to increase the consistency of vocalization during speech production. Auditory comprehension for word recognition was only mildly impaired but his ability to follow commands using parts of his body was complicated because of limb apraxia. Naming was poor and influenced by a number of linguistic variables (i.e., frequency of occurrence, open versus closed class, concrete versus abstract). Verbal paraphasias were observed.

At approximately 6 weeks after the participant's stroke, the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1983) was administered to obtain a formal measure of speech and language functioning. Significant speech apraxia persisted characterized by aphonics, effortful, trial and error, groping articulatory movements, attempts at self-correction, dysprosody unrelieved by extended periods of normal rhythm, stress, and intonation, difficulty initiating utterances, and perceived phoneme substitutions. The following speech sample was obtained in response to the Cookie Theft picture:

(Examiner: What's happening with her?) With her? She's overflowing the sink . . . and . . . he's /s/ . . . (Examiner: How 'bout over there?) "Over there? He /kæraher/ it in /ɛ/ /ki/ it's a . . . (expletive) . . . /fo fo folling/ it's cookie jar . . . he's about to fall off . . . and hurt himself.

The patient's severity rating and scale profile of speech characteristics appear in Fig. 2.

The patient also demonstrated mild to moderate aphasic deficits (see Table 2). Oral expression had improved substantially; written confrontation naming was disproportionately impaired relative to his other language skills. Inspection of these ratings as well as the subtest results reveal that the patient's aphasia did not correspond to any of the classical aphasia categories as might be expected following a subcortical lesion. The ratings are also consistent with previous research reporting concurrent AOS and literal paraphasia in patients with lesions of the basal ganglia (Robin & Schienberg, 1990).

2.2. Phonemic analysis and results

To allow a better description of the characteristics of the patient's speech, the patient orally read a series of word lists. The lists sampled 45 voiced and unvoiced stop, fricative, affricate, nasal, semi-vowel, and consonant cluster phonemes. The patient read 10 different words or phrases in different vowel contexts that sampled each phoneme or blend in the initial, medial, and final positions of words (as appropriate). Words sampling single phonemes were one to two syllables in length while words and phrases sampling blends were one to three syllables in length. All responses were audiotaped. The procedure yielded a total of 1057 responses. To facilitate transcription, two experienced judges (the authors) jointly listened to the patient's responses and converted them to broad phonemic transcriptions. Repeated listening and consensus were used to resolve instances of disagreement. An analysis of the patient's phonemic errors was performed by inspecting each target phoneme only in the words that were intended to elicit that particular phoneme in either the initial, medial, or final positions of the words. Error productions of a given phoneme in words intended to produce a different target phoneme were ignored. Also, errors due to substitutions of voiceless for voiced cognate phonemes were also omitted from this analysis by necessity because of the patient's aphonics

Patient's Name _____ Date of rating _____
 Rated by _____

APHASIA SEVERITY RATING SCALE

0. No usable speech or auditory comprehension.
1. All communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. The range of information that can be exchanged is limited, and the listener carries the burden of communication.
2. Conversation about familiar subjects is possible with help from the listener. There are frequent failures to convey the idea, but patient shares the burden of communication with the examiner.
3. The patient can discuss almost all everyday problems with little or no assistance. Reduction of speech and/or comprehension, however, makes conversation about certain material difficult or impossible.
4. Some obvious loss of fluency in speech or facility of comprehension, without significant limitation on ideas expressed or form of expression.
5. Minimal discernible speech handicaps; patient may have subjective difficulties that are not apparent to listener.

RATING SCALE PROFILE OF SPEECH CHARACTERISTICS

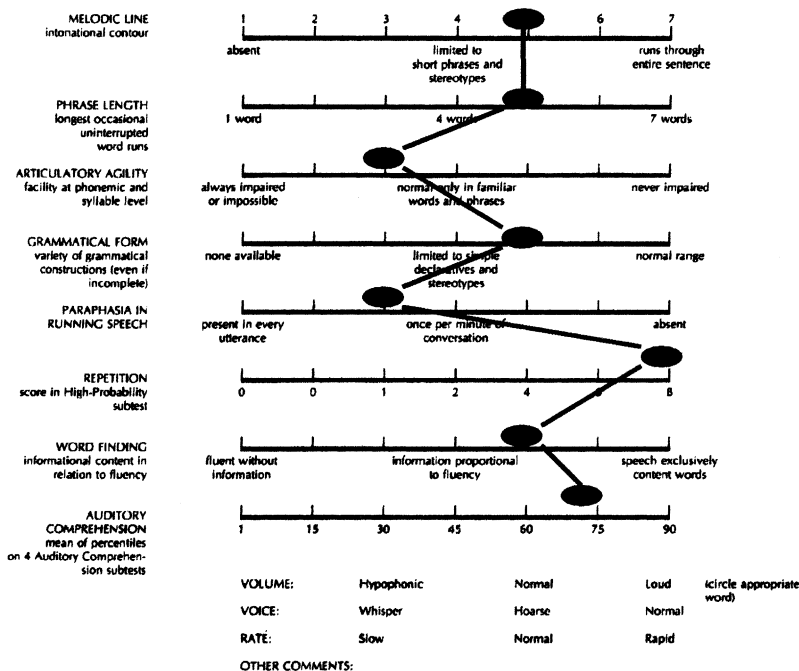


Fig. 2. BDAE aphasia severity rating scale and rating scale of profile of speech characteristics for participant.

Table 2

Results obtained from administration of the Boston Diagnostic Aphasia Examination at approximately 1-month post-onset of the participant's stroke

Subtest	Score
Auditory comprehension	
Word discrimination	69/72
Body part identification	17/20
Commands	12/15
Complex ideational material	8/12
Oral expression	
Oral agility	10/12
Verbal agility	14/14
Automatized sequences	5/8
Repetition of words	8/10
Repeating phrases	
High probability	8/8
Low probability	7/8
Word reading	30/30
Responsive naming	29/30
Visual confrontation naming	102/105
Body part naming	29/30
Oral sentence reading	8/10
Reading comprehension	
Symbol and word discrimination	10/10
Word recognition	8/8
Comprehension of oral spelling	7/8
Word–picture matching	9/10
Reading sentences and paragraphs	9/10
Writing	
Mechanics of writing	1/3
Primer-level dictation	14/15
Written confrontation naming	3/10

speech production. Approximately 25% (77) of the total errors produced by the patient were included in this analysis as a result of this approach.

Using the phoneme error types defined by Johns and Darley (1970), significantly more substitution errors were observed ($\chi^2 = 70.9$, $P = 0.000$) than other error types followed next by errors of addition (Fig. 3). With regard to error position, significantly more errors ($\chi^2 = 28.4$, $P = 0.000$) occurred in the initial position than in the medial and final position of words (Fig. 4). No differences emerged with regard to the relative difficulty of the different types of phonemes ($\chi^2 = 3.04$, $P = 0.69$), i.e., similar percentages of error occurred across the phoneme groupings (Fig. 5).

3. Discussion

We have described a patient with brain damage limited to subcortical regions of the left hemisphere who presented AOS with aphasia. His speech production was characterized

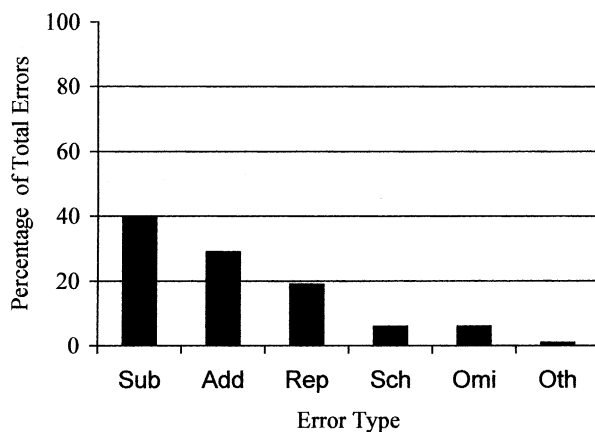


Fig. 3. Percentages of different error types produced by patient based on 10 productions of each target phoneme. Sub: substitution, Add: addition, Rep: repetition, Sch: intrusive schwa, Omi: omission, Oth: other.

by labored output that improved in automatic contexts and subsequently, by dysfluency and difficulty initiating speech. More importantly, we have demonstrated for the first time using broad phonemic transcription that the patient's profile of speech errors was similar to but not identical with that observed in patients with AOS due to cortical lesions. That is, the patient produced significantly more phoneme substitution errors than other types of errors and these occurred significantly more often in the initial rather than medial or final positions of words. No differences were observed in the number of errors produced for the various types of phonemes included in this analysis (e.g., consonant clusters, fricatives, affricates). Since consonant clusters are more frequently in error than singletons and affricates and fricatives are more frequently

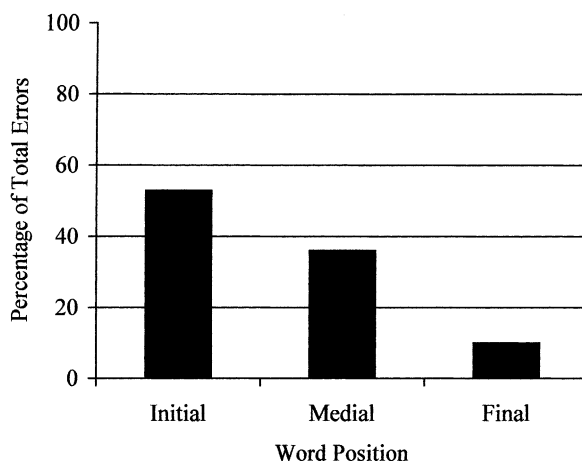


Fig. 4. Distribution of errors by word position.

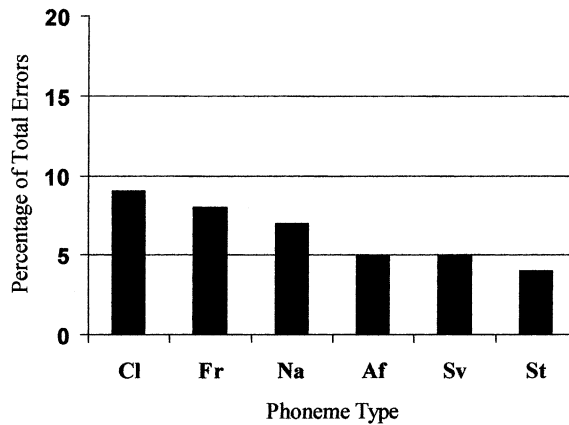


Fig. 5. Percentages of error according to type of phoneme. Cl: consonant cluster, Fr: fricative, Na: nasal, Af: affricate, Sv: semivowel, St: stop.

in error than other manners of production in patients with AOS due to cortical lesions (Duffy, 1995), the latter finding argues against the notion that the subcortical syndrome is indistinguishable from the cortical syndrome (Kertesz, 1984).

Previous studies investigating subcortical AOS and aphasia have not provided similar specificity of speech patterns, leading to the “lack of confidence” to which McNeil et al. (2000) refer. The earliest reports describing pronounced articulatory difficulties and accompanying aphasia with CT scan documentation of basal ganglia damage were those of Naeser et al. (1982) and Damasio, Damasio, Rizzo, Varney, and Gersh (1982). Naeser et al. (1982) provided examples of speech output littered with articulatory substitutions, simplifications, and omissions in three cases with lesions to the putamen and the anterior limb of the internal capsule. All three cases had accompanying buccofacial apraxia. Damasio et al. (1982) described at least two cases (Cases 1 and 4) in their series that presented nonfluent speech and defective articulation following damage to portions of the putamen and/or the caudate nucleus and the white matter surrounding these structures. Neither transcriptions nor samples of the patients’ speech were provided.

Kertesz (1984) used a scale rating volume, phonemic paraphasias, semantic paraphasias, phonemic perseverations, neologistic jargon, rate of speech, and dysarthria to describe speech production in his cases with subcortical lesions. Impressions regarding the presence of AOS in this group were based on the frequency of phonemic paraphasias and alterations in speech rate. Phonemic paraphasias “were defined as clearly articulated, recognizable alterations of a target phoneme” (p. 76). Nine of the 10 ratings suggested mild (3–4) or occasional (1–2) phonemic substitutions while the remaining case exhibited severe phonemic paraphasia (more than 2 per sentence or per 4 words). All of the cases showed slowing and mild hesitation or occasional hesitation but were still considered to be fluent in most cases. Unfortunately, the methodology in this study was inadequate for distinguishing between errors due to AOS versus aphasia, i.e., phonemic paraphasia. In addition, the overall impression of fluent speech in these cases is inconsistent with that observed in AOS.

It is difficult to determine, therefore, whether these patients did indeed demonstrate an AOS secondary to subcortical involvement.

Marquardt and Sussman (1984) examined the CT scans of 15 patients with Broca aphasia in an attempt to correlate lesion centroids and volume with clinical measurements of verbal, oral, and limb apraxia. Aphasia diagnosis was based on the patients' performance on the BDAE (Goodglass & Kaplan, 1972). A battery of tasks comprised of word and sentence imitation and diadochokinetic rate was used to measure the presence and severity of verbal apraxia. Correlation and multiple regression analyses did not reveal any meaningful relationships between lesion- and clinically-derived measures. Case analyses revealed subjects who had either extensive cortical lesion or lesion confined primarily to subcortical structures but similar lesion volumes and apraxia scores.

Alexander, Naeser, and Palumbo (1987) described 19 cases with aphasia due to subcortical infarction or hemorrhage. Speech and language assessment was limited to administration of the BDAE (Goodglass & Kaplan, 1972). All articulatory and prosodic disturbances were designated as dysarthric in nature despite the association of at least several of these symptoms with AOS (e.g., slowed speech rate, phoneme cluster simplification, reduced phoneme transitionalization, and dysprosody). Comparison of these cases to the present case is difficult, therefore, because of these terminological differences and the lack of specific procedures or examples to illustrate their patients' speech production. Nonetheless, one case (Case 5), described as having abnormal articulation, prosody and volume due to extensive damage to the putamen and anterior limb of the internal capsule accompanied by lesser damage to the superior periventricular white matter and genu of the internal capsule, may be similar to the case reported here.

Robin and Schienberg (1990) found AOS and aphasia in 5 of their 10 patients with basal ganglia lesions and in one patient with a hemorrhagic stroke of the thalamus. The findings were based on administration of the BDAE (Goodglass & Kaplan, 1972) and motor speech examination. The putamen and the anterior limb of the internal capsule was involved in five of the six patients with basal ganglia lesions; the anterior limb of the internal capsule was also involved in the patient with thalamic hemorrhage. No description of the patients' AOS was provided.

The current results provide support for the hypothesized influences of the basal ganglia and frontal white matter in the functional systems responsible for motor speech processing and production (Square et al., 2001; Square, Roy, & Martin, 1997). Motor speech appears to be governed by several functionally distinct cortical motor areas including primary motor (Brodmann's area 4) and nonprimary motor areas (premotor areas 44 and lateral 6 and mesial SMA 6) and the parietal lobe. Square et al. (1997, 2001) have suggested that the nonprimary motor cortex is involved in movement preparation and sensory guidance while the primary motor cortex is involved in the execution of movements. The parietal lobe appears to mediate the integration of sensory input for the preparation of motor behavior. These cortical areas relate to the basal ganglia through their projections to specific subcortical nuclei that send reciprocal projections to the supplementary and primary motor areas. The primary and non-primary motor areas project to the putamen, which then projects to the globus pallidus and substantia nigra. Globus pallidus and substantia nigra project to the SMAs by way of their thalamic connections. Projections from prefrontal,

parietal and temporal association areas project to the caudate and then the globus pallidus and substantia nigra, which project via the anterior thalamus to the lateral premotor area (Square et al., 2001). The basal ganglia, therefore, contain structures that are central to the integration of afferent and efferent impulses associated with perception and skilled motor movement.

AOS may be a result of lesions to the cortical association areas and, as in the present case, to the basal ganglia. Importantly, this finding presents problems for both of the speech programming accounts reviewed previously. In both the Darley et al. (1975) and Van der Merwe (1997) models, the core deficits that result in AOS are related to the cortical association areas. Furthermore, basal ganglia damage is only associated with specific dysarthrias in each of these approaches.

AOS following damage to the basal ganglia, however, is likely not a direct consequence of damage to these structures as in the case of cortical damage (Square et al., 1997). Brooks (1986) suggests that the basal ganglia allow high-level plans to be translated into motor action. Damage to the basal ganglia, therefore, might produce disruption in the implementation of speech motor planning routines (Alexander et al., 1987). This may explain the lack of differences in phoneme error types that were observed in this patient. Van der Merwe (1997) contends that AOS, when due to damage to the basal ganglia, might actually represent a dysarthric rather than apraxic disturbance. But this argument is simply based on anatomic notions that highlight a further weakness in the model.

Results of positron emission tomography have demonstrated that basal ganglia lesions have substantial effects on frontal lobe metabolism (Metter et al., 1988a; Metter, Riege, Hanson, Phelps, & Kuhl, 1988b) and may, therefore, impair motor speech processing associated with premotor and prefrontal areas. So, while it appears reasonable to identify different processes for speech motor planning, programming, and execution, these findings suggest that specification of separate segmental levels for the implementation of these processes is at best, premature. From an anatomic perspective, the data seem to argue for a more distributed functional unit than has been proposed in the aforementioned models.

In conclusion, the description of this patient's speech patterns provides important evidence for a theory of the neuroanatomical substrates of AOS. As in the most frequent presentation of the cortical syndrome, this case demonstrated an accompanying aphasia, which complicates the specification of motor versus linguistic mechanisms underlying speech processing. Identification and description of a case of pure AOS secondary to subcortical damage is needed to extend this line of inquiry. However, given the extremely rare occurrence of cases of pure AOS due to cortical origin (McNeil et al., 1997) and the paucity of cases that have been reported describing AOS due to subcortical damage, it is recognized that this will be a difficult goal to achieve.

Finally, the patient's speech samples were transcribed using broad phonetic transcription because of the exploratory nature of this investigation. The clear association of dysarthria with subcortical damage, and the tendency to use this term to describe all neuromotor speech errors (Alexander et al., 1987) suggests that the additional precision in the description of acquired articulation disorders gained through the use of narrow phonemic

transcription would be worthwhile (McNeil et al., 1997). This would be a reasonable direction to extend the current work.

Appendix A. Continuing education

A.1. Phonemic characteristics of apraxia of speech resulting from subcortical hemorrhage

A.1.1. Questions

1. The phonemic characteristics of AOS include:
 - a. More distortion, omission, or addition than substitution errors
 - b. More errors in word final than word initial position
 - c. More errors in affricates and fricatives than other manners of production
 - d. More errors of complication than simplification
 - e. More multiple-feature than single-feature sound substitutions
2. Both Darley et al.'s and Van der Merwe's accounts of speech motor processing suggest that AOS is the result of damage to:
 - a. Primary cortical areas
 - b. Cortical association areas
 - c. Heteromodal association areas
 - d. Subcortical nuclei
 - e. Both cortical and subcortical areas
3. The participant's AOS was accompanied by aphasia and:
 - a. Fluent, grammatically well-formed speech
 - b. Easy initiation of speech
 - c. Mild naming deficits
 - d. Phonemic paraphasias
 - e. Aphonia
4. In this study, AOS following subcortical hemorrhage was characterized by:
 - a. Fewer substitution errors than other phoneme error types
 - b. More errors in the medial and final than initial position of words
 - c. No differences with regard to the relative difficulty of the different types of phonemes
 - d. Greater numbers of errors in voiced than voiceless phonemes
 - e. More errors in affricates and fricatives than other types of phonemes
5. The authors concluded in this study that:
 - a. AOS only results from lesions to the basal ganglia
 - b. AOS following damage to the basal ganglia is a direct consequence of damage to these structures
 - c. AOS due to subcortical damage cannot be considered a motor planning disorder
 - d. The lack of differences in phoneme error types may be evidence for disruptions to cortical motor planning routines
 - e. Subcortical brain damage produces few effects on cortical speech processes

References

- Alexander, M. P., Naeser, M. A., & Palumbo, C. L. (1987). Correlations of subcortical CT lesion sites and aphasia profiles. *Brain*, *110*, 961–991.
- Ballard, K. J., Granier, J. P., & Robin, D. A. (2000). Understanding the nature of apraxia of speech: Theory, analysis, and treatment. *Aphasiology*, *14*, 969–995.
- Brooks, V. B. (1986). *The neural basis of motor control*. New York: Oxford University Press.
- Damasio, A. R., Damasio, H., Rizzo, M., Varney, N., & Gersh, F. (1982). Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. *Archives of Neurology*, *40*, 129–134.
- Darley, F. L., Aronson, A., & Brown, J.R. (1975). *Motor speech disorders*. Philadelphia: Saunders.
- Dronkers, N. F. (1996). A new region for coordinating speech articulation. *Nature*, *384*, 159–161.
- Duffy, J. R. (1995). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis: Mosby.
- Goodglass, H., & Kaplan, E. (1972). *The assessment of aphasia and related disorders*. Philadelphia: Lea & Febiger.
- Goodglass, H., & Kaplan, E. (1983). *The assessment of aphasia and related disorders* (2nd ed.). Philadelphia: Lea & Febiger.
- Johns, D., & Darley, F. L. (1970). Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research*, *13*, 556–583.
- Jones, K. C., Peach, R. K., & Schneck, M. J. (2003, May). *Must the insula be damaged in apraxia of speech?* Paper presented to the Clinical Aphasiology Conference, Orcas Island, Washington.
- Kertesz, A. (1984). Subcortical lesions and verbal apraxia. In J. C. Rosenbek, M. R. McNeil, & A. Aronson (Eds.), *Apraxia of speech: Physiology, acoustics, linguistics, management* (pp. 73–90). San Diego: College-Hill Press.
- Marquardt, T. P., & Sussman, H. (1984). The elusive lesion—Apraxia of speech in Broca’s aphasia. In J. C. Rosenbek, M. R. McNeil, & A. Aronson (Eds.), *Apraxia of speech: Physiology, acoustics, linguistics, management* (pp. 91–112). San Diego: College-Hill Press.
- McNeil, M. R., Doyle, P. J., & Wambaugh, J. (2000). Apraxia of speech: A treatable disorder of motor planning and programming. In S. E. Nadeau, L. J. Gonzales Rothi, & B. Crosson (Eds.), *Aphasia and language: Theory to practice* (pp. 221–266). New York: Guilford Press.
- McNeil, M. R., Robin, D. A., & Schmidt, R. A. (1997). Apraxia of speech: Definition, differentiation, and treatment. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (pp. 311–344). New York: Thieme.
- Metter, E. J., Riege, W. R., Hanson, W. R., Jackson, C., Kempler, D., & Van Lanker, D. (1988a). Subcortical structures in aphasia: Analysis based on FBG, PET, and CT. *Archives of Neurology*, *45*, 1229–1234.
- Metter, E. J., Riege, W. R., Hanson, W. R., Phelps, M. E., & Kuhl, D. E. (1988b). Evidence for a caudate role in aphasia from FBG positron computed tomography. *Aphasiology*, *2*, 33–43.
- Mohr, J. P., Pessing, M. S., Finkelstein, S., Funkenstein, H. H., Duncan, G. W., & Davis, K. R. (1978). Broca aphasia: Pathologic and clinical. *Neurology*, *28*, 311–324.
- Naeser, M. A., Alexander, M. P., Helm-Estabrooks, N., Levine, H. L., Laughlin, S. A., & Geschwind, N. (1982). Aphasia with predominately subcortical lesion sites: Description of three capsular/putaminal aphasia syndromes. *Archives of Neurology*, *39*, 2–14.
- Robin, D. A., & Schienberg, S. (1990). Subcortical lesions and aphasia. *Journal of Speech and Hearing Disorders*, *55*(1), 90–100.
- Square, P. A., Martin, R. E., & Bose, A. (2001). Nature and treatment of neuromotor speech disorders in aphasia. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (4th ed., pp. 847–882). Philadelphia: Lippincott Williams & Wilkins.
- Square, P. A., Roy, E. A., & Martin, R. E. (1997). Apraxia of speech: Another form of praxis disruption. In L. J. Gonzales Rothi & K. M. Heilman (Eds.), *Apraxia: The neuropsychology of action* (pp. 173–206). Hove, East Sussex, UK: Psychology Press.
- Van der Merwe, A. (1997). A theoretical framework for the characterization of pathological speech sensorimotor control. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (pp. 1–25). New York: Thieme.