Subcortical Aphasia: A Report of Three Cases

Richard K. Peach
West Paces Ferry Hospital, Atlanta, Georgia

John D. Tonkovich
Veterans Administration Medical Center, Decatur, Georgia

The correlation between damage to specific regions of the cerebral cortex and well-defined aphasia syndromes is firmly established (Benson and Geschwind, 1981; Damasio, 1981; Goodglass and Kaplan, 1972). The relationship between subcortical brain damage and aphasic symptomatology, however, has been both a topic of historical controversy and recent interest. Following the localizing doctines of Broca in 1861 and Wernicke in 1874, Pierre Marie argued in 1906 that the clinical characteristics of Broca's aphasia were not the result of involvement of only anterior cortical areas, which was the accepted belief at that time. Citing evidence in which cases of Broca's aphasia were found that did not involve lesions of Broca's area (Benton, 1981), Marie hypothesized that these clinical characteristics did not truly represent aphasia, but rather were the result of a severe disturbance of verbal output which he called anarthria. The pathological basis for this condition involved a lesion in the subcortical area which he referred to as the "quadrilateral of anarthria" (Hecaen and Albert, 1978). Marie's quadrilateral space, as it has come to be known, included the deep structures such as the caudate nucleus, the putamen, the internal capsule, and the thalamus. True aphasia was conceived only when this subcortical lesion extended posteriorly to involve the temporal isthmus, thereby resulting in Wernicke's aphasia. Later reports consistently demonstrated specific language disturbances associated with lesions of Broca's area, so that little more appeared concerning this approach to aphasic phenomena until approximately the past decade. It is clear now that Marie's work was the harbinger of the recent increase in emphasis on the contributions of subcortical structures to the integration and production of language.

With the advent of CT scan localization, atypical syndromes of language disturbances which do not completely conform to classical aphasic syndromes are being identified and associated with lesions of subcortical structures. The language deficits have been described in patients who consistently present destruction of the thalamus, the basal ganglia, and the internal capusle. The syndromes however, as Benson (1979) has suggested, must be considered tentative while clinical observations are accumulated and reported. Naeser, Alexander, Helm-Estabrooks, et al., (1982) estimated that 10% of their aphasic patients during the two-year period previous to their report presented speech and language disturbances secondary to subcortical lesions. It would seem that this is a relatively substantial proportion of the population of aphasic patients in general. These informal prevalance figures suggest a need for continued investigation and discussion of observations related to subcortical brain damage. To our knowledge, such discussion at this forum has been only briefly alluded to in the work of Rosenbek, McNeil, Teetson, Odell, and Collins (1981). Reliable identification of these aphasic syndromes has obvious effects upon prognostic statements concerning recovery and development of treatment procedures. In our own clinical practice, we have observed three cases of subcortical aphasia resulting from damage involving basal ganglia structures and the internal capsule in the past ten months. The purpose of

this presentation is fourfold. First, to review the literature concerning aphasic disturbances resulting from subcortical damage. Second, to describe three additional cases of subcortical aphasia as they relate to previous reports of this type of aphasia. Third, to discuss the proposed neuropsychological mechanisms associated with the speech and language characteristics of subcortical aphasia. Fourth, to establish a dialogue on aspects of treatment for aphasia resulting from subcortical lesions.

THE BASAL GANGLIA AND APHASIA

Identification of aphasic deficits arising from subcortical lesions anterior to the thalamus and involving the basal ganglia and the internal capsule has been the focus of several recent reports. Specifically, a "striatal aphasia" (Benson, 1979) has been described following both hemorrhagic (Hier, Davis, Richardson, and Mohr, 1977; Alexander and LoVerme, 1980) and nonhemorrhagic (Brunner, Kornhuber, Seemuller, Suger, and Wallesch, 1982; Naeser, et al., 1982; Damasio, Damasio, Rizzo, Varney, and Gersh, 1982) lesions of the dominant left cerebral hemisphere. In addition, a rare case of crossed aphasia in a right handed patient has been observed following right cerebral hemisphere infarction of the basal ganglia and the internal capsule (Assal, Parentes, and Deruaz, 1981). The reports of language deficits resulting from thalamic and putaminal hemorrhages were previously unable to distinguish specific aphasic syndromes associated with damage to each of these structures (Alexander and LoVerme, 1980). The failure to identify such differences may have been partially attributable to the imprecise CT scan localization which resulted from hemorrhagic displacement of these structures. In the past year however, observations of language impairment in twenty-one cases with nonhemorrhagic lesions limited to the basal ganglia have been reported with CT scan verification. The outcome of the observations has been a better description of the role of the basal ganglia in language performance as well as a more definite characterization of aphasic deficits arising from striatal versus thalamic involvement.

As has been the case in descriptions of thalamic aphasia, the symptoms of language impairment resulting from basal ganglia and internal capsule lesions have not lent themselves readily to definition by comparison with classic aphasic syndromes. Differential patterns of speech and language production have been identified in these cases based upon the degree of fluency, the quantity of verbal output, the presence or absence of dysarthria or dysprosody, comprehension functioning, and presence or absence of paraphasia. Based upon nine cases, Naeser et al. (1982) defined three aphasia syndromes resulting from lesions involving the putamen and internal capsule. The syndromes included 1) good comprehension with grammatical, dysarthric speech following capsular/putaminal (C/P) lesions with anterior-superior white matter extension, 2) poor comprehension with fluent Wernicke-type speech resulting from C/P lesions with posterior white matter lesion extension across the auditory radiations in the temporal isthmus, and 3) global aphasia from C/P lesions with both anterior-superior and posterior extension. Lesions with anterior-superior white matter extension included the periventricular white matter and corona radiata lateral to the body of the lateral ventricle and deep to Broca's area. Lesions with posterior extension included the area inferior to the Sylvian fissure and superior to the temporal horn. In all instances, the patients demonstrated lasting right hemiplegia. Additional support for an aphasic syndrome similar to that characterized by capsular-putaminal lesions

with anterior-superior extension has also been provided by the report of Damasio, et al., (1982).

The cases with C/P lesion sites and anterior-superior extension resembled Broca's aphasia in their impaired articulatory agility, good comprehension, and right hemiplegia. However, their near normal phrase length was inconsistent with the agrammatic output of classic Broca's aphasia. The cases with C/P lesions and posterior extension were like Wernicke's aphasia in their comprehension deficits, fluent paraphasic speech and relatively better naming of letters than objects. However, they differed from Wernicke's patients in that they presented lasting right hemiplegia and a very large discrepancy between oral and verbal agility.

Naeser, et al. (1982) and Damasio, et al. (1982) have provided tentative neuroanatomical explanations for the speech and language deficits resulting from basal ganglia and capsular lesions. The preservation of grammatical form in the speech of these patients has been attributed to the deep locus of these lesions, leaving Broca's area intact. Dysarthric impairment results from white matter lesions which involve fibers in or descending to the anterior limb of the internal capsule. Several interpretations are offered for the disturbed comprehension and paraphasic output in these patients. Naeser, et al. (1982) suggest that damage to the ascending auditory radiations in the temporal isthmus might be responsible for the comprehension defect. This proposal is based upon their observations of comprehension impairment only in cases where there is posterior extension of the lesions. Damasio, et al. (1982) have proposed a similar disconnection hypothesis, but attribute comprehension deficits to damage to the fiber system of the auditory cortex which projects to the head of the caudate nucleus. Alternatively, they also suggest that direct damage to the striatum itself may be responsible for the aphasia, because of the striatum's role as a multimodal processor of perception. Their approach to this question is motivated by their findings of auditory comprehension impairment in patients with lesions strictly limited to the anterior limb of the internal capsule, the caudate nucleus, and the putamen. Lesion size was a critical factor in both studies, one in which small differences contributed to large discrepancies in behaviors. It is also a factor to consider in the presentation of our own cases.

REPORT OF CASES

Case 1. A 66-year-old right-handed man with a history of arteriosclerotic vascular disease and hypertension suffered a cerebrovascular accident involving the left middle cerebral artery. Impressions derived from neurological consultation suggested multiple lacunar infarcts, probably of the deep internal capsule with relative sparing of cortical areas. Results of CT scan demonstrated deep low density abnormalities. Right hemiplegia was present.

Testing with the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass and Kaplan, 1972) at three days after the stroke revealed a mild to moderate comprehension deficit. Performance scores were as follows: word discrimination, 52/72; body-part identification, 18/20; following commands, 12/15; complex ideational material, 8/12. Speech output was characterized by normal phrase length and grammatical form with mildly reduced melodic line. Marked dysarthria was present with consistently impaired articulatory agility and occasional paraphasic errors. Naming appeared generally intact (95/105 for visual confrontation naming, 30/30 for responsive naming, 29/30 for body-part naming), while word fluency was mildly reduced (9/60). Repetition performance revealed marked difficulty for polysyllabic words and low probability phrases (3/8),

frequently resulting in paraphasic or omitted items. For example, in response to "The spy fled to Greece" he said "The fly skek the Greek" and for "The phantom soared across the foggy heath" he produced "The phantom soared across the fomly heath." Oral word (7/30) and sentence (1/10) reading were characterized by marked paraphasic and paralexic substitutions. Examples of these errors were: cridle/circle, tentree/fifteen, sengy twenty once/seven twenty one, To get home form work/I got home from work, and Limes are sugar/Limes are sour. Word recognition was only mildly impaired (6/8). Reading for sentence comprehension (1/10) was severely impaired. Graphic expression could not be tested due to the patient's unilateral (right) hemiplegia and a stated inability to perform with his left hand.

The aphasia pattern in this case is consistent with C/P lesions with anterior-superior extension that show relatively good auditory comprehension but dysarthric speech with reduced articulatory agility. Repetition, oral reading, and silent reading for sentences were markedly impaired.

Case 2. A 72-year-old right-handed woman suffered an intracerebral hemorrhage that was confirmed by CT scan in September, 1982. The scan showed a lesion generally located in the area of the internal capsule resulting in dense right hemiplegia.

Informal evaluation at two weeks after the stroke revealed fluent, grammatical verbal output characterized by frequent perseveration and marked confusion. Utterances were also distorted by paraphasic errors. The patient's confusion had cleared sufficiently by two months post stroke to allow formal evaluation.

Expressive output was fluent and grammatical with multiple paraphasic responses and occasional neologisms, as illustrated by this excerpt from the patient's description of the cookie theft picture: "I never...I didn't hever have...a pisher...a train running over the sink...and nothing like that...I had a rainch...I had a river...let it run over one and run over...a fiftent... we just had one sink stopped up at times..." On tasks of auditory comprehension, the patient's scores were 1/30 (prorated) for word discrimination, 12/20 for body-part identification, and 0/6 (prorated) for following commands. Supplementary testing using real objects demonstrated 30% accuracy and 10% accuracy in object identification by name and function respectively. These scores suggested moderate to severe auditory comprehension impairment. The patient's word repetition was characterized by a score of 4/10 and literal and verbal paraphasic responses. Examples included lababu for W, emaside for emphasize, and 1777 for 1776. The sentence repetition score was 1/8, high probability, and 1/8, low probability; responses were composed of primarily verbal paraphasic errors. For example, for "Go ahead and do it if possible" she said, "Go and get down when it's possible," and for "Near the table in the dining room" she said, "Bring the table in the country room." Repetition performance was markedly to severely impaired. Naming was characterized by scores of 7/30 for responsive naming and 79/105 for confrontation naming. This large discrepancy was likely a result of the patient's auditory comprehension deficits. Her score for oral word reading was 25/30; supplementary word list reading was also only mildly to moderately impaired but contained paralexic errors (chain for chin, charge for cart, water for week). Oral sentence reading was markedly impaired (2/10) with paralexic substitutions; e.g. for "They heard him speak on the radio last night" she produced "They made him fish from the road lost night." Word and sentence reading comprehension were both severely impaired. Spontaneous written production of her name was not possible; writing was limited to copying, with occasional perseverative errors.

This patient's aphasic symptoms are representative of those associated with C/P lesions with posterior extension. Poor comprehension, fluent speech with paraphasic and neologistic errors, poor repetition, and impaired reading and writing with a dense right hemiplegia comprise this symptom complex.

Case 3. A 50-year-old male suffered a CVA in June, 1982, with right hemiparesis and aphasia. Significant medical history included previous CVA with a completely resolved left hemiplegia and hypertension. CT scan showed a small area of diminished attenuation in the right thalamic region, a fairly well-defined area of diminished attenuation in the posterior limb of the internal capsule, and a slight impression on the posterior portion of the left lateral ventricle. Remaining sections showed no abnormalities.

Portions of the BDAE were administered to the patient on 7/9/82. Aphasic deficits were noted primarily in the areas of speech and writing. The patient's auditory comprehension appeared to be only mildly involved, although reading comprehension was moderately impaired. The patient recognized single words and short sentences, but showed decreased performance on longer sentences and paragraphs. The patient was mute and unable to imitate oral-motor movements. Functional writing and copying was absent with the non-preferred hand. He was discharged to his home on 8/3/82. Subsequent evaluation on 9/10/82 showed mildly increased speech production which was "labored" and "slurred" and generally unintelligible out of context. This pattern is similar to aphasia due to C/P lesion with anterior-superior and posterior extension with deficits in all language modalities.

DISCUSSION

The neuropsychological mechanisms for aphasia resulting from subcortical lesions have been briefly alluded to in this paper. The reader is referred to the reports of Naeser, et al. (1982) and Damasio, et al. for a more extensive discussion of the role of subcortical structures in language function. Additional discussion in these works has also focused on the etiology of such lesions and their size and site as determiners of aphasic syndromes. Brunner, et al. (1982) have discussed the profile of speech and language disturbances resulting from lesions limited to the basal ganglia versus lesions which include both cortical and basal ganglia regions. Our focus in discussing subcortical aphasias includes three areas: prognosis, treatment, and some generally unresolved issues.

The prognosis for recovery from aphasia resulting from subcortical lesions appears to be good. Most speech and language deficits resolve quickly, usually within a few weeks or months after onset. This recovery is probably the result of sparing of cortical areas after subcortical infarcts or hemorrhages. Recovery occurs less rapidly when the lesion extends into cortical areas (Brunner, et al., 1982) or involves the thalamus (Glosser, Kaplan, and LoVerme, 1982). Of those patients with strictly capsular-putaminal lesions, some continue to demonstrate chronic deficits and are subsequently enrolled in programs of speech and language treatment. It is unknown at this time whether these patients follow a course of recovery similar to that demonstrated by patients with cortical insults. For example, in evaluating the landmarks of recovery, additional data will be required to more specifically describe the nature and extent of these patients' spontaneous recovery, the optimal period of recovery and responsiveness to treatment, and the long-term severity of these deficits. Some insight into what the pattern of this recovery might be is provided by Glosser, et al. (1982) in their longitudinal investigation of a patient with subcortical hemorrhage. However, their patient had thalamic as

well as basal ganglia involvement so that generalization of their findings requires caution when discussing patients with lesions limited to the internal capsule and basal ganglia.

With regard to treatment, traditional techniques which are used to stimulate recovery of speech and language functioning from cortical lesions seem appropriate for treatment of subcortical aphasias. Such approaches, of course, include those which are based upon compensatory mechanisms, unmasking of collateral areas, and transfer of function to contiguous areas. Our patients were treated using traditional techniques. Case 1 received therapy designed to increase the precision of motor speech production and improve his articulatory agility. Case 2 received intensive auditory-verbal stimulation, while Case 3 was treated with both auditory comprehension tasks (initially) and nonverbal communication training (later). Helm-Estabrooks (1983) has recently advocated Visual Action Therapy and Voluntary Control of Involuntary Utterances for treatment of globally-impaired subcortically damaged patients.

Finally, there remain certain unresolved issues. Damasio, et al. have suggested that isolated nonhemorrhagic lesions of the internal capsule, caudate nucleus, and putamen will need to be identified in order to better understand the specific roles of these structures in language processing. Lesions of the basal ganglia and capsular regions, however, are commonly lacunar, consisting of multiple small infarcts (Fisher, 1965 as cited by Miller, 1983). This phenomenon could confound investigations of subcortical language mechanisms. The nature and location of lesions involving subcortical structures will have to be consistently reported.

The factors which contribute to rapid resolution of aphasic deficits in some individuals with subcortical lesions versus those which underlie chronic impairment are also as yet incompletely understood. Identification of such factors will play an important role in formulating prognoses for these aphasias. A patient at the rehabilitation unit at one of our facilities presented severe speech and writing reductions secondary to putaminal hemorrhage. Yet he almost completely recovered within a few weeks after his admission. Given the significant role of third-party reimbursement in the delivery of speech and language treatment services, the need for identification of these factors takes on increasing importance.

It appears that we have only begun to consider the relationships which exist between subcortical structures such as the basal ganglia and the internal capsule and the process of language functioning. This area should prove to be an exciting one in terms of future research and clinical practice in aphasia.

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DISCUSSION

Comment: Since I'm going to be arguing for the use of adjectives in aphasia on Thursday, I feel a little silly saying what I'm about to say now but, it would seem to me that, because the relationship between subcortical lesion and the presence or absence of aphasia is a phenomenon that is only recently been opened to investigation, what we're going to see over time is basically the variety of symptoms that we see in patients who have aphasia. I'm suggesting that one of the likely outcomes of our interest in patients with subcortical lesions is that we're going to see aphasias resulting from subcortical lesions rather than an entity called subcortical aphasia. I think it's an important kind of distinction to keep in mind. I'm very busily studying the spontaneous recovery period in patients who have strokes regardless of hemisphere or whether they are brainstem, subcortical, cortical, whatever and a couple of interesting things are emerging from those data that I think address some of your points. The first is that the incidence of people with subcortical involvement is much higher, I think, than the literature would predict. Second, the number of patients who are symptomatic who have subcortical involvement, not necessarily purely isolated subcortical lesions, is higher than the present data would suggest. Third, I think the issue is in large measure compromised by the frequency and the tendency of damage to those areas to result from hemorrhage rather than infarction. I think

the problem is not going to turn out to be a syndrome called subcortical aphasia but rather it's going to be aphasias in their many variants resulting from subcortical lesions.

Response: I think those are all very good points. I think that some of the observations made by Jeff Metter yesterday and alluded to by Don Robin are issues that have not really been approached in this whole area. We're looking again at the putamen as a structure that has some function in generating language behavior. We know that there are some strong cortico-striatal-thalamic-cortical loops and that a disconnection along any one of those areas certainly could have distance effects. So while we may be seeing syndromes that appear to be occurring concurrent with certain areas of involvement, we may be unsure of what we're looking at when we consider this whole other area of disconnection phenomena.

Q: What can you say about the dysarthria you mentioned one of your patients had? Would either of you like to predict the form of dysarthria to be seen with subcortical lesions?

A: There was nothing that distinguished this particular type of dysarthria from that which might be seen with any other case with capsular involvement. Damasio, et al. made no real attempts to differentiate their dysarthrias. They do discuss dysprosodic deficits. It's a point that will have to be thought about in the future.