Isolated Thalamic Lesion and Aphasia: A Case Study

Donald A. Robin
Case Western Reserve University, Cleveland, Ohio and
Great Lakes Regional Rehabilitation Center
of Lorain Community Hospital, Lorain, Ohio

Steven Schienberg
University of Pittsburgh, Pittsburgh, Pennsylvania and
Great Lakes Regional Rehabilitation Center
of Lorain Community Hospital, Lorain, Ohio

INTRODUCTION

The belief that the cortex operates as a closed neuronal circuit responsible for language functioning has recently been challenged (Brunner, et al., 1982; Glosser, et al., 1982; Kornhuber, 1977; Ojemann, 1976). Kornhuber (1977) emphasized that a complex act such as speech or language requires information processing at several levels of the central nervous system. The concept of subcortical language processes has been supported by evolutionary/phylogenetic studies (Brown, 1982; Kuypers, 1973), electrical stimulation studies (Ojemann, 1976), and numerous case reports of patients with subcortical lesions and resultant aphasia (Alexander and LoVerme, 1981; Brunner et al., 1982; Cappa and Vignolo, 1979; Glosser et al., 1982; McFarling et al., 1982; Reynolds et al., 1979).

Two subcortical areas thought to be involved in linguistic processing are the thalamus and the basal ganglia (Brunner et al., 1982; Glosser et al., 1982; Kornhuber, 1974, 1977). Glosser and co-workers (1982) reported data on a patient who suffered a putaminal-thalamic hemorrhage with a resulting auditory comprehension deficit, word-finding difficulties, and paraphasic speech which resolved after 2-1/2 years. The majority of recovery occurred four months post episode. Brunner and colleagues (1982) studied 40 patients, eight of whom had isolated basal ganglia lesions. These eight patients exhibited either Broca's aphasia with "transcortical features" or an anomia. Resolution of deficits in these eight patients occurred within eight months post stroke.

Studies examining isolated thalamic lesions have documented intact repetition, fluent paraphasic speech, variable comprehension deficits, mild dyslexia, and dysgraphia (Alexander and LoVerme, 1981; Cappa and Vignolo, 1979; Metter et al., 1983; Mohr et al., 1975; McFarling et al., 1982; Reynolds et al., 1979). A key finding of all of these studies has been the transient nature of the aphasia Typically, resolution of impaired behaviors occurred within six months after insult.

The purpose of this study was to describe a patient with a lesion of the thalamus who suffered an unusual syndrome of aphasic behaviors. Unlike previously reported cases, our patient demonstrated a severe and persistent language deficit. Diagnostic, therapeutic, and neuroanatomical correlates are considered.

METHOD

The Subject. The patient was a 66 year-old, right-handed female who suffered a cerebrovascular accident (CVA). CT scans were performed on the day of the CVA and four months later. Scans were independently read by three radiologists and two neurologists with similar interpretations by each. The

first scan revealed low density in the left thalamic and left parietal regions (Figure 1). The second scan documented only a left thalamic lesion with resolution of the low density in the parietal lobe (Figure 1).

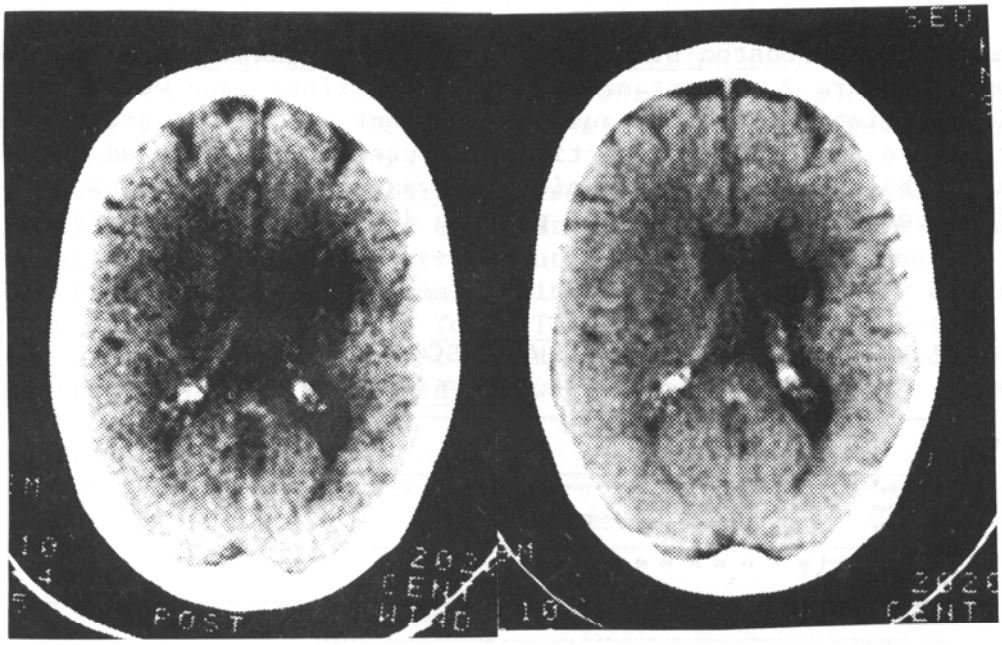

Figure 1. CT Scans. (Left) = at onset. (Right) = 4 months post-onset.

Assessment. Assessment of language and related areas was performed two to four months post-stroke by the authors. The patient's behavioral profile did not change over her period of treatment. At the time of assessment the patient had a right-sided hemiplegia. Auditory and visual acuity were unaffected by the CVA. Visual fields were intact. The Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972), the Token Test (DeRenzi and Vignolo, 1962), the Reading Comprehension Battery for Aphasia (LaPointe and Horner, 1979), and informal procedures were used to assess the areas outlined in Table 1.

Table 1. Speech-Language parameters assessed.

1. Spontaneous Speech
   a. Conversation
   b. Picture description
2. Naming
   a. Confrontational
   b. Responsive
3. Repetition of Speech
   a. Single word
   b. Phrase/sentence
4. Auditory comprehension
   a. Word discrimination
   b. Commands
   c. Complex ideational material
5. Oral-reading
   a. Single word
   b. Phrase/sentence
6. Reading comprehension
   a. Single word
   b. Sentence
   c. Paragraph
7. Writing
   a. Spontaneous
   b. Dictation
   c. Copying
8. Prosody
   a. Rate
   b. Pitch
   c. Stress
   d. Rhythm
9. Gestural
10. Pragmatics (Searle, 1969)
    a. Request
    b. Assert
    c. Question
    d. Order
    e. Argue
    f. Advise
    g. Warn

RESULTS

The patient's Boston Diagnostic Aphasia Examination (BDAE) z-score profile is shown in Figure 2. Spontaneous speech was fluent and well articulated, but essentially unintelligible because of frequent neologisms and paraphasias. The patient used a variety of syntactic structures which appeared intact despite aberrant verbal output. Self-monitoring was not evident in spontaneous speech. No differences were noted between picture description and conversational speech.

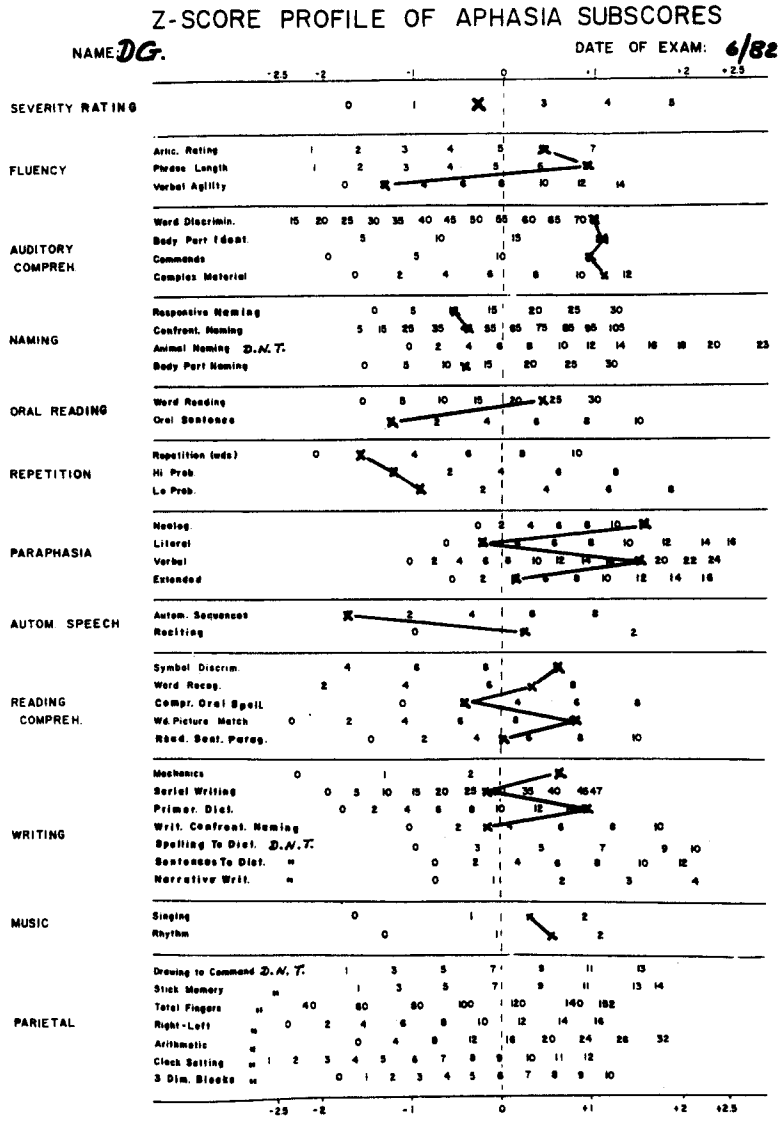

Figure 2. Boston Diagnost Aphasia Examination Z-scor profile.

Naming was moderately to severely impaired. Visual confrontation naming elicited 15/35 correct responses on the BDAE. On a responsive naming task 4/10 correct responses were recorded. Errors consisted of verbal paraphasias, neologisms, and perseverations. For instance, the patient named the color "green" as

"gray" and when attempting to say "falling" produced /dɪpəlɪn/. Unlike in spontaneous speech, the patient attempted to self-correct speech errors on these tasks. However, most attempts at self-correction were unsuccessful. Repetition of speech was severely impaired for words and phrases. Eight of ten error words were neologistic in nature with a tendency toward perseveration. She was unable to repeat phrases.

Auditory comprehension was mildly impaired. On the BDAE, word discrimination, right-left discrimination, and body-part identification were essentially normal. Sequential commands were carried out correctly. Responses to complex ideational materials were within normal limits. The mild auditory comprehension disturbance was most apparent on the Token Test (Table 2). No specific pattern of dissolution was noted. The patient did show a minimal improvement on a second administration of the Token Test two weeks later (Table 2).

Table 2. Results of Token Test.

Subtest	Date of Testing 7/12/82	7/23/82
I	10/10*	10/10
II	10/10	9/10
III	5/10	9/10
IV	6/10	7/10
V	17/22	17/22

*number correct

Oral reading of words was mildly impaired. However, oral sentence reading was severely disordered even at the simple sentence level. Errors were paraphasic, neologistic, and perseverative in nature. Reading comprehension was moderately impaired. The patient had difficulty on the sentence-paragraph subtest of the BDAE when stimulus length reached paragraph level (Figure 2). Results of the Reading Comprehension Battery for Aphasia (RCBA) indicated inability to comprehend written material of increased length, increased linguistic complexity, and of a functional nature (Figure 3).

A moderate to severe dysgraphia was noted. Although the non-dominant hand was used for all writing tasks, letters were well formed and legible. Spelling errors were made during dictation of words and confrontational writing. The patient typically wrote the correct initial letter of words. The patient was obviously critical of her performance on all writing tasks since she frequently attempted to correct her spelling errors.

Prosody was informally assessed during conversational speech samples. Three of four prosodic elements--pitch, stress, and rhythm--were normal. However, rate of spontaneous speech was judged as excessive. The patient used a crude but effective gestural system to augment her communication. Pointing was a typical gestural response, but occasionally the patient pantomimed an activity to convey a message.

Pragmatic skills appeared intact. Observation of the patient in various communicative interactions revealed appropriate use of the following speech acts: request, assert, question, order, argue, advise, and warn. However, the most

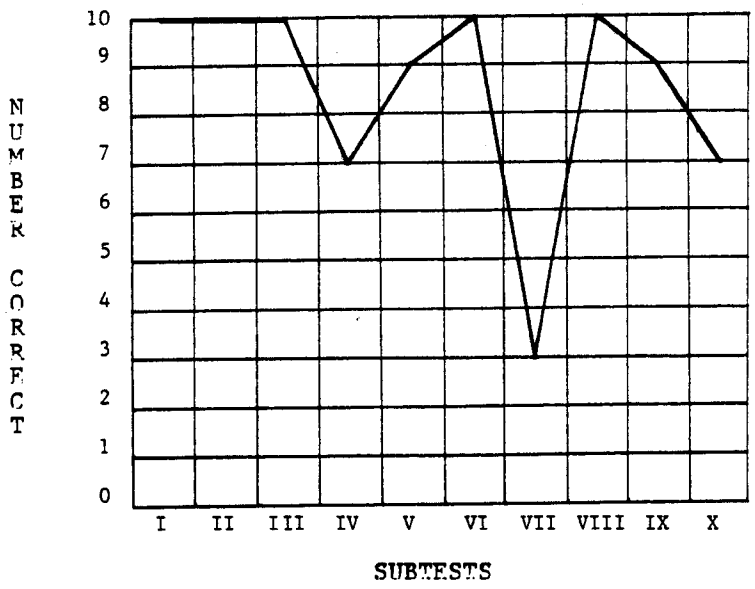

Figure 3. Reading Comprehension in Aphasia subtest profile. (I = word, visual. II = word, auditory. III = word, semantic. IV = functional reading. V = synonyms. VI = sentence, picture. VII = paragraph, picture. VIII = paragraph, factual. IX = paragraph, inferential. X = morpho-syntax.

frequent speech acts observed were request and question, which was in part due to limited opportunities to use a larger repertoire. In most instances the patient conveyed her message through a combination of speech, gestures, and/or writing.

DISCUSSION

Clinical Implications. The language performance of this patient revealed moderate to severe deficits in spontaneous speech, naming, repetition, reading, and writing, with relatively preserved auditory comprehension. These behaviors were the result of a lesion of the thalamus. This syndrome of aphasia was unique in that it did not correspond with traditional categories of aphasia. Thus, our findings supported Glosser and colleagues (1982, P. 111) who stated that subcortical aphasias "stand apart from instances of cortical aphasia."

Thalamic aphasia, as summarized by Mateer and Ojemann (1983), can be characterized by the following: (1) fluent paraphasic speech, (2) anomia, (3) intact repetition, (4) variable auditory comprehension, (5) mild dyslexia, (6) dysgraphia, and (7) transient symptoms. Our patient differed from these cases in two ways. First, our patient demonstrated a persistent and severe disorder. Secondly, the disordered repetition demonstrated in this study clearly distinguished this patient from others who reportedly have shown "transcortical" features on the basis of intact repetition with thalamic aphasia (Cappa and Vignolo, 1979; McFarling et al., 1982; Mohr et al., 1975; Reynolds et al., 1979) Additionally, while many symptom complexes result from thalamic lesions, the occurrence of fluent, paraphasic speech in the presence of intact auditory comprehension made this case of thalamic aphasia unusual.

The unusual symptom complex and persistent nature of our patient's aphasia dictated the therapeutic regime. Given this patient's near normal level of

auditory comprehension she was trained to monitor her spontaneous speech and utilize phonemic and placement cues so that paraphasic errors could be decreased. While the number of paraphasic errors were decreased, the overall severity of the patient's aphasia did not change.

The persistent, severe language disorder our patient demonstrated clearly indicates the role of the speech-language pathologist in cases of thalamic aphasia. The clinician's responsibility in these cases includes careful examination of patients' speech-language abilities so that appropriate treatment plans can be formulated. The variety of symptom complexes in cases of subcortical aphasia now reported in the literature illustrates the confusion encountered when attempting to diagnostically categorize these behaviors. Until stronger correlations are discovered between site of lesion and disrupted behavior we caution against the grouping of patients.

Theoretic Implications. The role of the thalamus in linguistic processing is as yet unclear. It has been suggested that the thalamus acts as an "alerting system" for language areas (Mateer and Ojemann, 1983; Ojemann, 1976). Thus, the disorder associated with thalamic lesions may be related to decreased attention for language because of a disorder of the arousal mechanism. Anomia, found in all cases of thalamic aphasia, can be explained by this hypothesis. Further, Ojemann (1976) has found decreased verbal memory associated with thalamic lesions. Metter and colleagues (1983) have shown that loss of verbal memory, in both cortical and subcortical aphasias, are correlated with decreased metabolic rates in the thalamus. They concluded that abnormal metabolic functioning of the thalamus interferes with specific arousal mechanisms for memory.

The alerting mechanism hypothesis of thalamic function in language does not account for the entire symptom complex observed in thalamic lesions. We hypothesize that complex neuronal circuitry between the cortex, thalamus, and basal ganglia accounts for a variety of symptoms found in subcortical aphasia (Figure 4). Each area in the circuit has a role in language function (primary or secondary), as well as an influence over the two adjoining areas through neuronal pathways. The primary language function of the cortex has been well documented (Goodglass and Kaplan, 1972; Kornhuber, 1974; 1977). Part of the function of the thalamus is that of an alerting mechanism. The thalamus may also play a role in the processing of complex information. For example, the lateral geniculate body processes complex visual stimuli via interactions between on and off cell transients (Phillips and Singer, 1974; Singer and Phillips, 1974). Rolls and co-workers (1982) have recorded potentials from neurons in the thalamus during visual recognition and memory tasks. Neurons in the thalamus responded to familiar and novel stimuli. Further, different response latencies were recorded when visual discrimination was hard or easy. Thus the role of the thalamus as part of the language circuit probably involves numerous functions.

The basal ganglia have been implicated as motor programmer (Marsden, 1982). Researchers believe that complex motor movements are the result of basal ganglia motor programs in conjunction with cortical activation. The basal ganglia receive increased blood flow during complex motor acts prior to the cortex (Roland et al., 1982). In order for these movements to be precise, sensory input from the thalamus may be necessary. Thus, speech production may be the result of initiation of motor programs at the level of the basal ganglia. The thalamus may also be responsible for accurate transmission of information from the basal ganglia to the cortex (Kornhuber, 1974). Apparently, for language to be processed efficiently and correctly, the specific neurons in the cortex, thalamus, basal ganglia, and their connections must all be intact or error patterns will result.

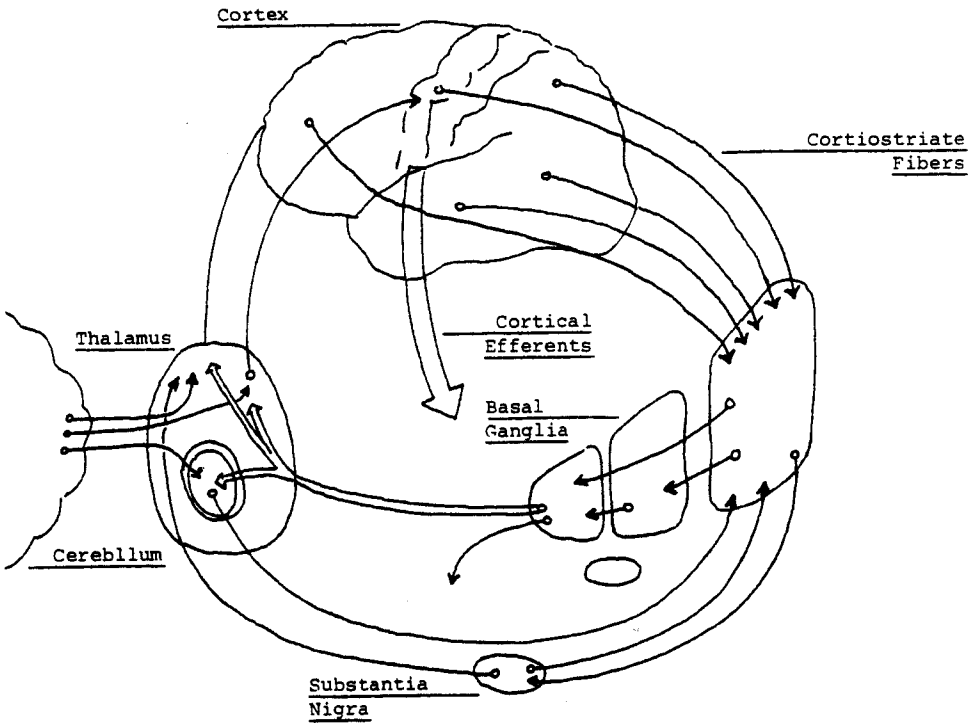

Figure 4. Functional neuronal circuit. (Adapted from Côté, 1982)

The fluent, paraphasic speech of patients with thalamic lesions may be the result of an inability to select and sequence the appropriate motor programs for phonemic movements due to disruption of the cortical-thalamic-basal ganglia circuit. As a result, these patients activate speech at a normal or excessive rate but their production is faulty. Naming disturbances are explained by decreased arousal mechanisms as postulated by Ojemann (1976). That repetition was impaired in our patient and not in other studies is probably related to the size and place of the lesion. Kornhuber (1974) wrote that patients with intact repetition cannot extract information internally from the basal ganglia, due to fiber disruption between the basal ganglia and thalamus, and must rely on external stimuli to generate this function. However, if the thalamic lesion disrupts flow from the basal ganglia to the cortex, repetition would be impaired. Intact auditory comprehension would indicate that the auditory pathway, including the medial geniculate body, is unaffected by the lesion. Thus, a functional circuit, with each part dependent upon the next, can explain a variety of symptoms seen in subcortical aphasia.

A second hypothesis as to the role of the subcortical area in language was provided at this conference by Metter (Metter et al., 1983). He suggested that the aphasia seen with subcortical lesions might be the result of distance effects. In other words, his studies have shown that metabolic depression of the cortex occurs with structural lesions of the thalamus and basal ganglia. We believe that the best explanation may be a combination of many factors including abnormal language processing at a subcortical level, decreased arousal mechanisms, and distance effects.

We have discussed a case in which a thalamic lesion created a persistent fluent aphasia with relatively intact comprehension and disordered repetition. The presence of aphasia following subcortical lesions seems related to disruption of complex neuronal circuitry as well as disruption of subcortical processing of language.

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DISCUSSION

Q: Did you mention utterance length on this individual?

A: It was essentially 7-8 words. There was no nonfluency or hesitancy of speech.

Q: Why did you label the verbal symptoms as neologistic and paraphasic when your treatment goals were articulatory in nature?

A: She made a number of different error types. Some of the errors were clearly verbal paraphasias (e.g., "gray" for "green"). Other errors were neologistic. None of these errors were accounted for by muscular weakness or apraxia found in patients with anterior lesions. Our explanation involves the functional neuronal circuit discussed in the paper. The interaction between motor-speech mechanisms and language may be at a subcortical level.

Q: Why did you consider her aphasic?

A: She exhibited language deficits in all modalities.

Q: Can you discuss your treatment approach and outcome?

A: The first goal was to slow her rate of spontaneous speech. Secondly, using a picture naming task or oral reading, we paired initial sounds of words with phoneme placement. This strategy was extended to phrases which were functional in nature. While she eventually produced many phrases without

errors there was no generalization to materials found on the BDAE. Thus, her behavioral profile did not change, but functionally she showed improvement.

Q: Was she aware of her errors?
A: When we first saw her she was not aware of her errors in spontaneous speech. Her verbal output reminded us of a patient with Wernicke's aphasia. On more structured tasks (e.g., naming, writing) she was very aware of her errors. She was most aware of her errors in writing. Interestingly her writing did not parallel her fluent speech.

Q: Was she hypophonic?
A: She was not.

Q: Was her auditory comprehension absolutely intact?
A: She showed a mild deficit.

Q: I want to challenge the literature that states that recovery is rapid or immediate. We presented a patient here last year who had severe deficits after a year. Do you agree that short-term aphasia from subcortical lesions may be a myth?
A: We feel that for the most part, thalamic language symptoms are transient (see Mateer and Ojemann, 1983). However, some cases (ours included) do show lasting effects. These are the cases that are most important for speech-language pathologists to be aware of.

Q: In terms of her general level of arousal, her general ability to focus and maintain attention and vigilance, would you say they were all intact?
A: Essentially, yes.