An Assessment of Internal Speech in Phonologically Impaired Brain-damaged Adults with Minimal Aphasia

Cynthia Shuluk-Rome
Perkins School for the Blind, Watertown, Massachusetts

Jon G. Lyon
Veterans Administration Medical Center, Reno, Nevada

A long controversy continues over the nature of articulatory errors in brain-damaged adults who otherwise exhibit minimal receptive language deficit (Martin, 1974; Kearns, 1982; Kent and Rosenbek, 1983). A nonlinguistic, motor speech disorder, apraxia of speech, has been identified and labelled by one constituency (Johns and Darley, 1970; Rosenbek, Wertz and Darley, 1973; Mohr et al., 1978) while another group has promoted a linguistic, phonological dysfunction as the basis of this disorder (Martin, 1974; Dunlop and Marquardt, 1977; Shewan, 1980). Resolution of this issue has not proven easy since an unhindered examination of phonological form prior to its realization is unobtainable. Inferentially then, proponents of apraxia of speech cite recent physiologic and acoustic findings (Itoh et al., 1979, 1980, 1982; Kent and Rosenbek, 1983) that suggest faulty timing of articulatory and laryngeal movements within phonemic boundaries while advocates of the other view cite induced articulatory errors caused by manipulation of linguistic variables (Martin and Rigodsky, 1974; Dunlop and Marquardt, 1977). However, to our knowledge there are few investigations that have attempted to assess the intactness of internal speech in such patients (Nebes, 1975; Shewan, 1980; Square, Darley and Sommers, 1980). The present investigation was proposed with this objective in mind.

METHOD

Subjects. Fourteen adult male subjects (seven brain-damaged and seven non-brain-damaged), participated in this investigation. The brain-damaged adults, ages 49-75, had all suffered from a single left cerebrovascular accident at least 2 months prior to their inclusion. All had been diagnosed independently by two speech and language pathologists as manifesting a speech disorder consistent with Darley's (1969) definition of apraxia of speech. These subjects exhibited articulatory errors at the monosyllabic word level while receptive language skills remained relatively intact. Criteria for subject selection included: (a) Performance of 90% or higher on the Auditory Comprehension Section of the Boston Diagnostic Aphasia Examination. (b) Performance at or above the 50th percentile on the Porch Index of Communicative Abilities. Subjects demonstrated a mean of 73% Overall, 85% Gestural, 54% Verbal and 75% Graphic on the various modalities. Verbal modality response scores were markedly lower than other modalities. (c) A score of 80% or higher on the retention of a series of three maximally varied nouns from the Denver Phoneme Sequencing Test. Individual test scores are summarized in Table 1. The non-brain-damaged adults, ages 49-69 served as a control group.
Table 1. Results of speech and language tests for Experimental Group.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>MPO</th>
<th>PICA OA</th>
<th>PICA Gest</th>
<th>PICA Verb</th>
<th>PICA Graph</th>
<th>Boston Aud.</th>
<th>P.S.T. #4 Max. Varied</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>85%</td>
<td>87%</td>
<td>52%</td>
<td>93%</td>
<td>100%</td>
<td>90%</td>
</tr>
<tr>
<td>2</td>
<td>132</td>
<td>52%</td>
<td>81%</td>
<td>45%</td>
<td>47%</td>
<td>95.8%</td>
<td>90%</td>
</tr>
<tr>
<td>3</td>
<td>72</td>
<td>68%</td>
<td>94%</td>
<td>54%</td>
<td>63%</td>
<td>97.0%</td>
<td>90%</td>
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<tr>
<td>4</td>
<td>6</td>
<td>66%</td>
<td>67%</td>
<td>49%</td>
<td>76%</td>
<td>98.0%</td>
<td>90%</td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>72%</td>
<td>87%</td>
<td>54%</td>
<td>74%</td>
<td>94.0%</td>
<td>80%</td>
</tr>
<tr>
<td>6</td>
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<td>93%</td>
<td>64%</td>
<td>95%</td>
<td>98.5%</td>
<td>100%</td>
</tr>
<tr>
<td>7</td>
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<td>76%</td>
<td>85%</td>
<td>58%</td>
<td>78%</td>
<td>100%</td>
<td>80%</td>
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<tr>
<td>Mean</td>
<td></td>
<td>73%</td>
<td>85%</td>
<td>54%</td>
<td>75%</td>
<td>98%</td>
<td>89%</td>
</tr>
</tbody>
</table>

\(^{1}\)Months Post Onset

Materials. Four test batteries were constructed to assess internal speech. Two test batteries contained pictured stimuli and the other two contained printed stimuli. One picture battery and one print battery contained rhyming word pairs, i.e., "bird/burn," a response choice that differed phonemically from the stimulus in only the final portion of the word.

All stimuli depicted monosyllabic words with a high frequency of occurrence. Each test item contained a stimulus picture or printed word, with six response pictures or printed words below on the same page. None of the printed word pairs could be identified from similarity of letters (date/straight, rule/tool). For both printed batteries, response choices were selected so that there were both visual and acoustic confusions. For all four batteries, the degree of phonemic similarity of inaccurate response choices was systematically controlled.

Procedure. Two test sessions were required to administer the four test batteries to each subject. Order of presentation of test batteries was counterbalanced. To minimize any confusion regarding the task, both rhyming batteries and both acoustically similar batteries were presented in the same session.

Two screening measures were constructed to assure that the subjects could 1) discriminate rhymed versus unrhymed word pairs and acoustically similar versus dissimilar word pairs, and 2) recognize all pictured and printed words used in the test. The first screening test consisted of thirty word pairs, presented on tape, half rhymed or acoustically similar. An accuracy of 85% was necessary before the test batteries were presented.

The second screening test was incorporated into the actual test battery. Fourteen stimulus words (from every two test items) were randomized and inserted into the test booklet prior to administration of those items. The examiner presented each word verbally, and the subject demonstrated recognition by pointing to the word named.

For the internal speech measure, the subject was instructed to point to the word on the lower portion of the page which corresponded to the stimulus word in either the rhyming or acoustically similar mode. This was to be completed without any verbalization by the subject. Finally, subjects were asked
to name all test stimuli within the test sets after rhymed or acoustically similar selections had been made. These output data were useful in determining whether any discrepancy may have existed (particularly in the pictured batteries) between the intended word and the subject's representation for that word. In addition, these data provided some general information regarding articulatory errors. Following the test, each subject's verbal responses were examined for accuracy. For the purposes of this study, judgment was made as to whether each word was produced accurately or in error. A specific error analysis was not performed. Tapes were randomly selected and interjudge reliabilities were 90% or better in judging articulatory accuracy.

RESULTS

Figure 1 illustrates percent correct scores for normal and aphasic subjects on the four test batteries. The mean correct responses for the experimental group was 49.2, compared to 91.7 for the control group. A three-factor analysis of variance with repeated measures was performed on the data obtained from the internal speech measures. The effects of type of stimuli (rhymed versus acoustically similar) and mode of presentation (pictured versus printed) were not statistically significant. However, group differences (Experimental versus Control) were found to be highly significant (p < .001). This finding suggests that internal speech was disordered during the rhyming and acoustically similar pair selection for the brain-damaged group. However, when internal speech proficiency was compared to articulatory proficiency on rhymed and acoustically similar pairs, distinct differences emerged from within the brain-damaged group.

Figure 2 illustrates the percent of correct and incorrect responses on the internal speech measure and the subsequent articulation of stimulus words. One distinct profile was shown by Subject 1. He displayed good ability to perform the internal speech measures, scoring within one standard deviation of the control group mean, although his own articulation was highly disordered. Subjects 2, 4 and 5 likewise exhibited a high percentage of misarticulations, but they grossly failed the internal speech measures. A still different profile was seen with Subjects 3, 6 and 7 who scored within a moderate range (accuracy between 50-65%) on the internal speech measure, and exhibited fewer articulatory errors.

DISCUSSION

The findings of this investigation indicate that among subjects with apraxia of speech, the underlying causes can and do vary. Table 2 provides a model for the interactions between internal speech measures and articulation. Intact internal speech, in conjunction with disordered articulation, suggests that the underlying speech form was present during the task. This type of performance is consistent with the explanation of apraxia of speech as a motor programming disorder. Nebes (1975) found similar results in his work with a single patient, whose ability to perform similar internal speech tasks was comparable to that of normal subjects, in spite of her severe articulation disorder following a CVA.

Other subjects in this investigation (2, 4 and 5) presented a disordered phonological profile. For this group, both internal speech and corresponding articulation were greatly disordered, suggesting that the underlying speech form was not present. Shewan (1980) also found evidence of a
Figure 1. Percent correct responses for control and experimental subjects on each internal speech measure.
phonological disorder in her investigation of subjects with aphasia and verbal apraxia. In comparing speech production abilities to performance on a receptive phonological task, she found a correlation between receptive and productive phonological impairments in six of nine subjects. She discussed involvement of a central processing mechanism and suggested that these subjects were unable to maintain a phonological representation on the receptive task.
The performance of the remaining subjects (3, 6 and 7) is more difficult to categorize. Compared to normal controls, these subjects perform poorly on the internal speech task. However, examination of their individual scores indicates that they were performing the task correctly 50-65% of the time. As illustrated in Figure 2, the articulation of these subjects was less severely involved compared with the other subjects in the study. Examination of the total percent of incorrect responses on the internal speech task, as well as the overall frequency of articulatory errors, indicates a close correspondence for each subject. Given the variability of articulatory errors in apraxia of speech, it is impossible to know the specific articulation corresponding to each internal speech item. One possibility, given consistencies in internal speech and articulatory error scores for these three subjects, is that when these subjects erred on the internal speech measure, they did not have the underlying speech form in mind. This would be indicative of a phonological disorder which presents with less severity than that exhibited by Subjects 2, 4 and 5. A second possibility is that the performance of these subjects was inconsistent, at times characteristic of an apraxia of speech disorder, in other instances characteristic of a phonological disorder.

The task presented in this investigation may not have been the most sensitive test for Subjects 3, 6 and 7. More challenging internal speech measures, involving polysyllabic words, such as syllable count, or cancelling silent "e's" (Neves, 1975) might yield more information regarding the internal speech of subjects with few articulatory errors. The present tasks may be best suited for subjects who exhibit a preponderance of articulatory errors at the time the test is administered (as did Subjects 1, 2, 4 and 5).

The existence of distinct subgroups within the population exhibiting apraxia of speech has been suggested by other authors. Shewan's (1980) results indicated that while some subjects were comparable to normals on the receptive task, others exhibited a disorder involving both receptive and productive elements. Buckingham (1981) has also proposed more than one form of apraxia of speech.

The heterogeneity within the present subject group suggests that our standard clinical diagnostic tools are not complete enough to enable us to predict the variability observed in subjects' performances. Phonological aspects of language are rarely assessed beyond phonemic discrimination abilities. Internal speech measures may provide a tool for the clinician to understand the nature of articulatory errors exhibited by this population. More importantly, for those brain-damaged adults who exhibit a predominance toward either a motor programming or phonological disorder, specification of treatment may be improved by such an assessment.

Logue and Dixon (1979) and Fedor (1981) have suggested therapeutic use of rhyming words for anamia. Logue and Dixon found that word retrieval skills improved when subjects were trained to use a number of retrieval strategies, including phonological association using rhyming words. Perhaps articulatory accuracy may also be improved by incorporating rhyming words and other internal speech tasks into treatment sessions. One question raised is whether the phonologically impaired patient stands to benefit more than the patient exhibiting a motor programming disorder from such treatment strategies. Whether or not different treatment strategies are indicated for the various subgroups designated in this study has yet to be demonstrated.

In conclusion, internal speech measures may be useful in differentiating apraxia of speech from phonological disorders in subjects with minimal aphasia.
While physiologic and acoustic differentiation entails instrumentation not readily accessible or familiar to many, internal speech measures are easily utilized by the clinician. Additional research is needed to further examine the relationship between internal speech abilities and specific articulatory performance.

ACKNOWLEDGMENT

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REFERENCES

DISCUSSION

Q: How would aphasic patients without apraxia of speech perform on your internal speech task?
A: I don't think they'd be able to do it. First of all, I think the naming would be too difficult for many of the aphasic patients. We needed to demonstrate (and did so) that subjects included in this study were not anomic for test stimuli.

Q: But they don't produce any phonological errors, then. If the aphasic patient without apraxia of speech does not produce any "phonological-phonetic, phonemic-phonetic" types of errors, then how can you say that the inability to perform internal speech tasks results in phonological errors?
A: I'm not clear on what you're saying.

Q: I'm saying that four years ago here, Square, Darley and Sommers presented a study where they looked at patients with pure apraxia of speech, an aphasia and apraxia of speech group, and a pure aphasic group. What they found on tasks very similar to yours is that there was no overlap between the pure apractic group and the apraxia and aphasia group, but there was overlap between the apractic-aphasic group and the aphasic group. The point is, without having a pure aphasic group and comparing that against it, that it is very difficult to come out with a conclusion that their phonological errors were due to an internal speech problem.
A: What was done in this study was to select patients who had minimal aphasia and try to control for other linguistic variables such as word retrieval that would have otherwise affected their performance on our internal speech task.

C: Yeah, I realize that pure apractic patients are rare and very difficult to get. However, it's very difficult to make those types of statements unless you're looking at pure syndromes. I would suggest that if you're not aware of that study, it was reported in the CAC Proceedings of 1980.

Q: Do you have a feeling that a classification system that groups together people who have intact internal speech and impaired internal speech may not be the best classification? If you had your druthers would you call these folks something else, that perhaps highlights something more basic about what's going wrong with them?
A: I think I might. The most important consideration in attaching a label to a specific group is understanding what that label implies. I think what we're seeing in this study and a couple of other studies I've cited, is that within this group, there seems to be more variability than is recognized by the label apraxia of speech.
Q: Did you look at the relationship between apraxia of speech in these patients and some of your measures of aphasia that were not influenced by verbal performance, such as the Boston Auditory Comprehension score, or the PICA gestures, or the PICA graphics?
A: We looked at them informally, and couldn't discern any apparent relationship.

Q: You didn't run correlations between performance on aphasia tests and your internal speech tests or your experimental test.
A: No.

Q: I'm a little intrigued by the dichotomy between the phonologically impaired and the non-phonologically-impaired. I was wondering whether you might comment upon what you think is creating the errors in the phonologically impaired group? At what level of the phonological system? Is it maybe an acoustic-perception level or is it more representative of the encoding process? Perhaps it's a motor or speech perception deficit?
A: I think it's difficult to know with any certainty. I don't think we know enough about internal speech. The most I can really say is that normals could perform this task and seemingly had an intact internal representation for the word while our disordered group did not.

Q: Is there nothing in their speech that differentiates this group, other than their internal representation? Is there anything we can hear or feel or see?
A: We didn't look at that in this study, but I think that would be a logical next step. One thing I did observe clinically in administering the test, was that for the experimental group, Subjects 3, 6 and 7, who were hard to categorize, exhibited primarily initiation problems. They had more groping, struggling behavior than any of the other subjects. There were very few substitutions. I wondered about that at the time...a more careful examination of those features might prove valuable.

Q: What degree of confidence do you have in your perceptual judgments? In other words, you had to classify the group as phonologically impaired or not phonologically impaired on some basis. What kind of confidence do you have in the way that you made the perceptual judgments?
A: I have a fair degree of confidence. Given the high frequency of articulatory errors for Subjects 1, 2, 4 and 5, where they were making errors on 80-90% of the words, I was very confident that they did not have intact internal speech. Subject 1 was still making errors with roughly the same frequency and was able to do the task, indicating intact internal speech. I have a lot of confidence in that distinction. It's much more difficult for Subjects 3, 6 and 7, and these we did not feel we could accurately categorize in these two groups.