The Frequency and Amplitude of Fluctuating Auditory Processing in Aphasic and Nonaphasic Brain-Damaged Persons

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INTRODUCTION

Traditionally, auditory comprehension disorders associated with aphasia have been viewed in terms of the linguistic properties of the stimulus and the linguistic deficits of the aphasic (Gainotti, Caligirone and Ibba, 1975), or in terms of a more basic processing disorder such as a limitation in some aspect of memory (span, sequencing, rehearsal. etc.; Adamovich, 1978) or as an overall slow processing system (Weidner and Lasky, 1976). Brookshire's (1974) assembly of several patterns of auditory processing disorders redirected attention to the notion that failure to comprehend by an aphasic listener may occur in more than one way, and potentially because of differing underlying disturbed mechanisms. During the past four years there has been a renewed interest in these auditory processing patterns and in exploring the mechanisms underlying these auditory deficits exhibited by virtually all aphasic patients. Given that the test instrument used to derive these patterns is reliable, has enough items of equal difficulty, and the behaviors are evaluated with a sensitive scoring system (one that captures more than the right-wrong qualities of the response). much can be learned about the nature of the patterns. Using the Revised Token Test (RTT) because it meets those psychometric requirements, McNeil and Hageman (1979) found that the pattern-types were not predicted by the type of task being performed, age, sex, severity of aphasia, etiology or lesion site of the aphasic patient. McNeil and Hageman (1980) further found that nonaphasic right-hemisphere-damaged persons exhibited nearly identical patterns to those of a group of left-hemisphere-damaged aphasic patients, suggesting that aphasia per se was not the mechanism causing the pattern-type. Hageman (1980) found that the frequency with which each pattern occurred was nearly identical between a left-hemisphere-damaged aphasic group and a group of normal non-brain-damaged subjects who were given the test under difficult (competing) listening conditions. Thus, it was again demonstrated that whatever the mechanisms for the patterns, they are not aphasia specific. Finally. Norris (1980) demonstrated that the frequency with which particular patterns occurred was not because of the length of the verbal stimulus (single noun vs. sentences) nor because of the verbal versus nonverbal (words vs. environmental sounds) nature of the stimuli. All of these findings are supportive of the notion that patterns are due to factors which are independent of the task being performed and are caused by internal states fluctuating within the subject.

When fluctuations are measured within subtests across homogeneous items, the four patterns which have been documented (McNeil and Hageman, 1979) are flat, tuning-in, tuning-out. and intermittent. The intermittent pattern occurs substantially more often than all others for aphasic,

right-hemisphere-damaged and stressed normal subjects and may, in fact, be the only reliable pattern that occurs (Hageman et al., 1982; this volume). McNeil (1982) speculated that the internal states responsible for the intermittent pattern might be reduced and/or inefficient allocation of effort or attention.

With the goals of describing and ultimately defining the mechanism(s) for the patterns, two additional, more finite analyses of the patterns were conducted in the present study. The frequency of changes across items within subtests was calculated, as was a measure of the amount of fluctuation or change from item to item (amplitude).

Answers to the following experimental questions were sought:
1. Do frequency and amplitude measures correlate highly and positively across all aphasic subjects and across all nonaphasic subjects for each subtest?
2. Is there a significant difference among subtests in the frequency or amplitude of changes which occur across items within subtests, for either the aphasic or non-aphasic brain-damaged subjects?
3. Is there a significant difference in the frequency and amplitude for each subtest between the aphasic and nonaphasic groups?

METHODS AND PROCEDURES

Thirty left hemisphere damaged aphasic and thirty right hemisphere damaged nonaphasic subjects were administered the Revised Token Test (McNeil and Prescott, 1978) under standard administration and scoring conditions. The aphasic subjects were heterogeneous with respect to etiology, lesion location, age, severity and any classification to which they may have belonged. Each of the 10 items in each of the 10 RTT subtests was scored according to convention so that a single mean was derived for each item. The number of frequency changes across all items in each subtest was calculated as the number of times a subsequent item changed by .20 or more from the preceding one. The .20 criterion was derived from previous research on the internal consistency of the RTT (McNeil and Prescott, 1978). A change in direction was not required for a frequency shift to be recorded. Amplitude of the changes was calculated by deriving the standard deviation for the amount of fluctuation for each subtest. Figure 1 illustrates frequency and amplitude as measured for one subtest. Note that a change of .20 was not reached between items 4 and 5 and between items 8 and 9, thus giving a frequency of 7 for this example.

Data analysis involved the calculation of Spearman rank order correlations, analysis of variance, Tukey Honestly Significant Different Procedures, and t tests.

RESULTS AND DISCUSSION

The overall goal of this investigation was to gain insight into the mechanisms causing fluctuations in performance on successive equally difficult items. As another and continuing step in the development of a model or theory which could initially describe, and finally explain, the
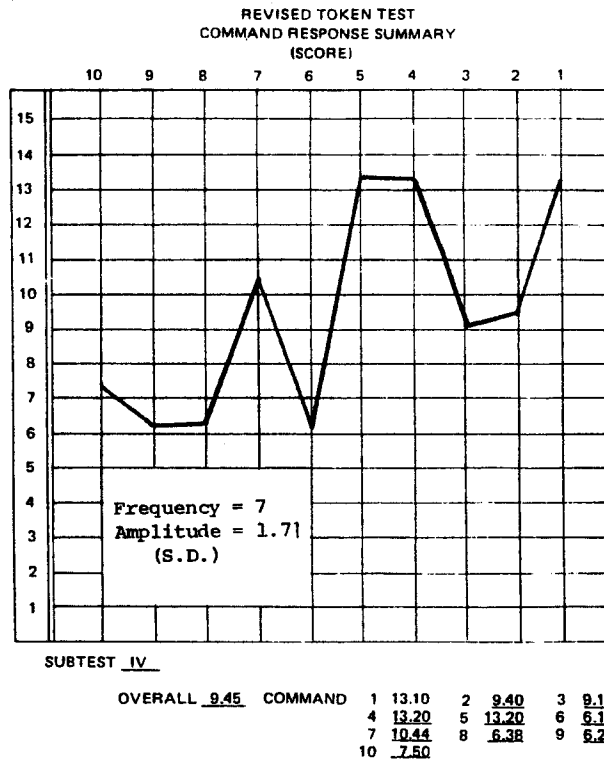
Figure 1. Hypothetical example of across item-within subtest performance illustrating the frequency and amplitude measures.

mechanisms for fluctuating behavior in aphasia, we asked if there was a predictable relationship between the frequency with which fluctuations occur and the amplitude of those fluctuations. If little or no relationship existed between the two measures, then separate mechanisms might be hypothesized. If on the other hand, the two measures varied together in highly predictable ways, then it might be reasonable (although cautiously) to pursue a single model/mechansim course to explain both qualitative characteristics of the patterns.

Relationship Between Amplitude and Frequency

To answer this initial question, rank order correlations were computed between the amplitude and frequency measures for each individual subtest of the RTT for the aphasic and nonaphasic groups. These correlations are summarized in Table 1. Only subtest 1 for the aphasic group and subtest 2 for the nonaphasic group showed a strong relationship between the amplitude and frequency of the pattern. Since there is little reason to believe that subtest I and II are fundamentally different from the other subtests, an alternative explanation for these two correlations must be sought. A closer examination of the data suggested that a ceiling effect may have occurred for these subtests, with little frequency and amplitude change occurring, resulting in a large correlation coefficient. This suggestion

Table 1. Spearman Rank Order Correlations for frequency with amplitude across RTT subtests for 30 left-hemisphere-damaged aphasic and 30 right-hemisphere-damaged nonaphasic subjects.

	SUBTEST									
	I	II	III	IV	V	VI	VII	VIII	IX	X
Left-Aphasic	.85	.57	.04	.28	.21	.23	.45	.14	.61	.47
Right-Nonaphasic	.58	.83	.61	.50	.51	.43	.61	.62	.55	.39

is supported by strong negative correlations between subtest scores and frequency and amplitude measures (see Table 2). These data suggest that these two measures of auditory processing vary independently, and there is little reason to believe that a single mechanism serves both aspects of the fluctuating performance.

Table 2. Spearman Rank Order Correlations for frequency and amplitude measures with subtest score, across RTT subtests for 30 left hemisphere damaged aphasic and 30 right hemisphere damaged nonaphasic subjects.

	SUBTEST									
	I	II	III	IV	V	VI	VII	VIII	IX	X
Left-Aphasic										
Frequency	-.86	-.67	-.52	-.34	-.31	-.08	-.17	+.20	-.45	-.40
Amplitude	-.88	-.83	-.36	-.45	-.02	-.02	+.11	+.20	-.59	-.61
Right-Nonaphasic										
Frequency	-.76	-.86	-.66	-.57	-.81	-.52	-.70	-.41	-.78	-.70
Amplitude	-.94	-.86	-.86	-.64	-.64	-.46	-.81	-.25	-.84	-.79

Consistent with the notion that the cause of changed performance is within the subject, and is task independent, it was hypothesized that frequency would be task independent and thus no significant differences would be found across subtests on the RTT. Conversely, we hypothesized that the amplitude of fluctuations would be task dependent, and thus significant differences would be found as task demands varied across subtests of the RTT. To measure the independence of frequency and amplitude from the task being performed differences were tested across subtests for each of the measures, for each group.

Frequency

Left-hemisphere subjects. A one way analysis of variance (Nie et al., 1975) for the frequency measure across subtests was significant ($F = 7.351$; $df = 9,290$; $P < 0.001$). The Tukey Honestly Significant Difference Procedure revealed that subtest 1 was significantly different from all other subtests in the number of changes or fluctuations (frequency) that occurred. Table 3 summarizes the result of these post hoc comparisons.

Table 3. Significant (P < .05) planned comparisons (Tukey Honestly Significant Difference Procedure) for the <u>frequency</u> measure of the within subtest pattern for 30 left hemisphere damaged aphasic and 30 right hemisphere damaged nonaphasic subjects.

		SUBTEST COMPARISONS	
Left- Aphasic Subjects	1 < 2 1 < 3 1 < 4 1 < 5	1 < 6 1 < 7 1 < 8	1 < 9 1 < 10 2 < 4
Right Nonaphasic Subjects	1 < 3 1 < 4 1 < 5 1 < 6 1 < 7	1 < 8 1 < 9 1 < 10 2 < 4 2 < 5	2 < 6 2 < 7 2 < 8 2 < 9 3 < 6 6 < 10

Figure 2 illustrates the mean frequency scores across all 10 subtests for the aphasic and nonaphasic groups. In order to account for the frequency of fluctuations differences among subtests, it was hypothesized that a ceiling might have occurred on that subtest which concealed the actual fluctuations which were occurring. In order to address this hypotheses, although indirectly, the frequency scores were again correlated with subjects' overall severity scores for that subtest. It was reasoned that if a high negative correlation was found for the subtests which were significantly different from the others (only subtest 1, or possibly subtest 2 in this instance), then it could be argued that a ceiling effect had occurred. The results of these rank order correlations for both subject groups are summarized in Table 2. These correlations are interpreted to support the hypothesis that a ceiling effect may account for this finding.

<u>Right-hemisphere subjects.</u> Another one way analysis of variance for the frequency measure across subtests for the nonaphasic group was also significant (F = 10.288; df = 9,290; P <0.000). The T.H.S.D.P. revealed that, like the aphasic group, the frequency of fluctuations for subtests 1 and 2 occurred significantly fewer times than for other subtests (Table 3) This is also illustrated in Figure 2. As with the data for the aphasic group, the frequency scores were correlated with subjects' overall correlations (Table 2), and again support the hypothesis that the significance for these two subtests may also be explained by a ceiling effect.
Amplitude

<u>Left-hemisphere subjects.</u> A one way analysis of variance for the amplitude measure across subtests was significant (F = 4.575; df = 9,290; P <0.00). The T.H.S.D.P. revealed that subtests 9 and 10 were different from several other subtests on this measure. Table 4 summarizes those comparisons which were significant (P <.05). All other comparisons were nonsignificant. Figure 3 illustrates the relationship of the amplitude scores among subtests for the aphasic and nonaphasic groups. It is apparent that subtest 9 and, to a lesser extent, subtest 10 are different
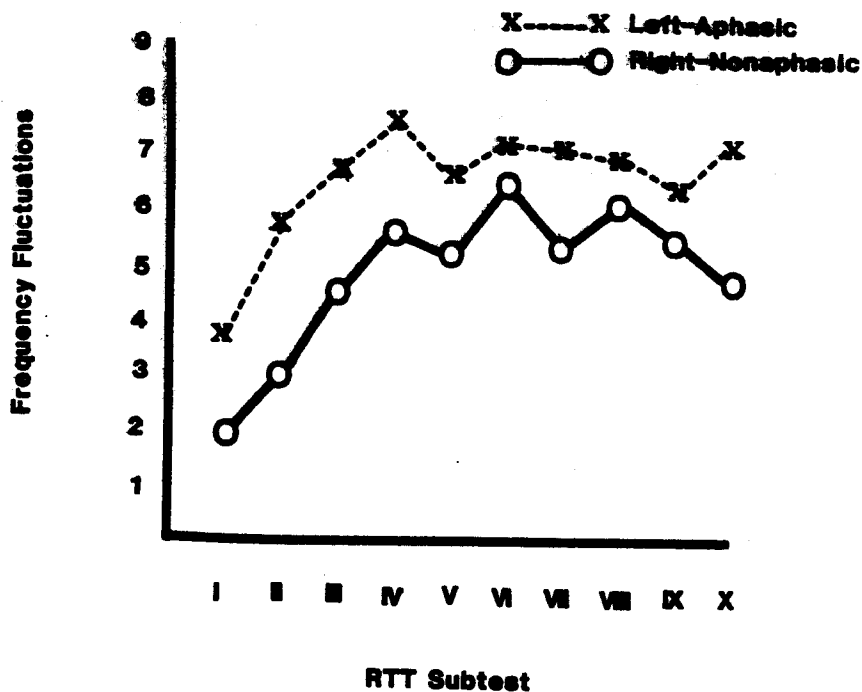
Figure 2. Mean frequency across RTT subtests for 30 left-hemisphere-damaged aphasic subjects and 30 right-hemisphere-damaged nonaphasic subjects.

Table 4. Significant (P < .05) planned comparisons (Tukey Honestly Significant Difference Procedure) for the <u>amplitude</u> measure of the within subtest pattern for 30 left hemisphere damaged aphasic and 30 right hemisphere damaged nonaphasic subjects.

	SUBTEST COMPARISONS		
Left	3 > 1	9 > 5	9 > 8
Aphasic	9 > 1	9 > 6	10 > 1
Subjects	9 > 2	9 > 7	10 > 8

from other subtests. A ceiling effect may explain this finding as it did for the frequency measure, since subtests 9 and 10 correlate strongly with subjects' level of performance (Table 2).

<u>Right-hemisphere subjects.</u> A one way analysis of variance for the amplitude measure across subtests was also significant for the right hemisphere group (F = 5.811; df = 9,290; P < 0.000). The T.H.S.D.P. revealed that, unlike aphasic subjects' performance, subtests 1 and 2 were significantly different (P < .05) from other subtests on this measure (Table 4). All other comparisons were nonsignificant (P > .05). Figure 3 illustrates the relationship of amplitude scores among subtests for this nonaphasic group. It is apparent from this figure that the nonaphasic group differed across subtests, primarily on subtests 1 and 2.
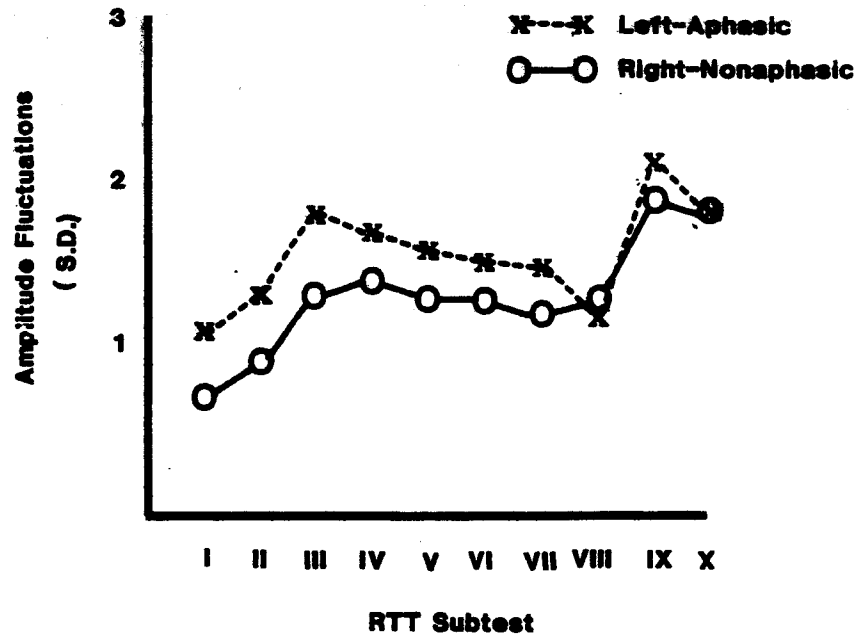
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Figure 3. Mean amplitude across RTT subtests for 30 left-hemisphere-damaged aphasic subjects and 30 right-hemisphere-damaged nonaphasic subjects.

As stated earlier, few differences between left-hemisphere-damaged aphasic and right-hemisphere-damaged nonaphasic subjects have been found on auditory processing pattern types. This has led to the suggestion that a single underlying mechanism may explain their existance. In order to assess this single mechanism theory further with the current refined frequency and amplitude measures, a two-tailed t test was computed within subtest, for amplitude and again for frequency for each group. It was reasoned that if the frequency measure was caused by an internal state factor common to all people and processes, then the groups should not differ significantly on the amount of frequency changes recorded unless there was a ceiling effect whereby a threshold for detection was not reached. On the other hand, severity of involvement might be expected to manifest itself in the amplitude measure, with aphasic subjects exhibiting significantly larger amplitude fluctuations than nonaphasic subjects. Therefore, no significant differences for frequency would be predicted, while significant differences would be predicted for the amplitude.
Group Differences

Frequency. Table 5 summarizes the two-tailed t test results between subject groups for frequency for each subtest. All but two subtest comparisons (6 and 9) were significantly different between the aphasic and nonaphasic groups. It is clear from these data that this method of pattern analysis yields fewer fluctuations for the nonaphasic than for the aphasic subjects and that these differences are not likely to have occurred by

chance. Therefore, either the nature of the deficit or the laterality of the lesion appears to be related to the frequency with which performance varied on these measures. Our prediction of no differences between subject groups was not supported.

Table 5. t values by subtest for frequency and amplitude for 30 left hemisphere damaged aphasic subjects compared to 30 right hemisphere damaged nonaphasic subjects.

| | | | | SUBTESTS | | | | | |
I	II	III	IV	V	VI	VII	VIII	IX	X
Frequency 3.00*	4.71*	3.97*	7.03*	2.65*	1.74	2.98*	2.09*	1.67	4.07*
Amplitude 2.30*	2.64*	3.46*	1.44	1.53	1.85	1.00	0.29	1.00	0.00

* = significant $P < .05$

Amplitude. Table 5 also summarizes the two-tailed t test results between subject groups for amplitude for each subtest. Again, contrary to our predictions, few amplitude differences were found between groups (with subtests 1, 2 and 3 as exceptions). These data are interpreted as evidence that the amount of fluctuation (measured by the standard deviation) differs little between groups. While the nonaphasic group performed significantly better than the aphasic group overall, and on all subtests, the depth of their fluctuations does not differ significantly from the depth of fluctuations for the aphasic group. Therefore, there does not appear to be a simple one-to-one relationship between the severity of involvement of auditory processing (as measured by the RTT) and the amplitude (amount) of moment to moment changes which occur in both groups.

SUMMARY

The following conclusions seem warranted:
1. The frequency and amplitude of auditory processing patterns can and do vary independently, suggesting separate mechanisms for their generation.
2. When the task is difficult enough to reach a threshold (determined by the limited evaluative system) for observing deficits, the frequency of fluctuations is task independent for both aphasic and nonaphasic groups. This suggests that whatever is causing the fluctuations is within the organism and the fluctuations may not be influenced in any reliable way by sensory information.
3. The amplitude of fluctuations appears to be task independent, for aphasic and nonaphasic groups. A ceiling effect appears to account for all differences between groups on this measure.
4. Aphasic subjects demonstrated significantly more fluctuations but not of greater amplitude (amount) from moment to moment than nonaphasic subjects. This suggests a process (e.g.

aphasia) or hemisphere lesion-dependent (even though task independent) mechanism for both parameters of the auditory processing variability. At this time it is not possible to speculate further about underlying mechanisms without either building straw men or over-turning stones unnecessarily.

It has been the purpose of this investigation to explore the utility of and possible mechanisms underlying two refined measures of subject variability. The frequency measure differentiated groups relative to measures of variability and auditory processing patterns. This measure may prove to be an important descriptive tool in our search for internal factors which account for the great majority of deficits in processing auditory information by aphasic and other brain damaged individuals.

The amplitude measure poses some important problems. First, it did not differ between tasks or between subject groups, and thus became un-interpretable relative to our preconceptions of what it actually represente. It may be that using the standard deviation as the metric for measuring the amount of fluctuations is inappropriate. The coefficient of variability ma provide a measure which will capture the depth of fluctuations which might vary across conditions and across subject groups. Secondly, the test-retes reliability (unreliability) of the amplitude measure (Hageman et al., this volume) makes it clear that it is a measure to be used with great caution, if at all.

Finally, the clinical relevance of the frequency and amplitude measure and the data presented about them deserve comment. At this time, neither tool nor the studies about them reveal insights that we believe have immediate clinical applicability. We believe that with further research, the frequency measure may have relevance as a refined tool for describing subject variability.

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