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ABSTRACT

This paper describes the results of two studies of auditory processing in child aphasia, and their implication for understanding deviant language development. The term "aphasia" is discussed as it is used to describe adult and child language disorders. A first experiment on the auditory functioning in aphasic and nonaphasic children suggests that the auditory processing defect in child aphasia lies in short-term auditory storage or memory. A subsequent study, which sought to measure the relationship between stimulus duration and signal detection, had equivocal results which lead to speculation about signal threshold and internal filter. It is suggested that low frequencies present special problems for temporary storage and detection. A final conclusion states that either a phonological base is a requisite foundation of subsequent linguistic development, or that auditory dysfunction does not adequately account for developmental aphasia. (AM)

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AUDITORY AND LINGUISTIC INTERACTION IN DEVELOPMENTAL APHASIA: EVIDENCE FROM TWO STUDIES OF AUDITORY PROCESSING

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INTRODUCTION

The following paper describes the results of two studies of auditory processing and the implications of those studies for understanding deviant language development. The studies were conducted on children with the delayed language syndrome known as congenital, childhood, or developmental aphasia. Characteristic of this syndrome is the failure to develop adequate linguistic skills, even though other developmental milestones are generally normal and peripheral sensory systems are intact.

Historically, aphasic children have been viewed as suffering from a kind of high-level symbolic or cognitive disorder, which is ultimately expressed in a delayed and deviant linguistic system. Close inspections of such children frequently turn up both auditory and visual perceptual disorders. But a direct causal relationship between these modality specific perceptual disorders and language delay has never been explicitly demonstrated.

In recent years usage of the term aphasia, as applied to children with certain kinds of developmental language disorders, has been severely criticized. Lenneberg (1960), for example, has argued that adult aphasia, which is a consequence of traumatic injury or a disease process directly affecting the cerebral cortex, bears only a superficial relationship to the developmental syndrome which bears the same name. Frequently, if not usually, brain damage is not documented in children who have the developmental syndrome. In contrast to the adult aphasic, the developmentally aphasic child does not lose the ability to communicate verbally; he does not learn language. But when linguistic skills are acquired, the child will continue to use them.

From a linguistic standpoint, the spoken language of aphasic adults is frequently bizarre, characterized by telegraphic speech, categorical confusions, jargon, etc. Developmentally aphasic children, on the other hand, do not exhibit the deviant linguistic forms of the adult. As a matter of fact, recent developmental language data collected by Morehead and Ingram (1972) indicate that the language of aphasic children does not differ basically from that of much younger, normal children. They found that as a group, deviant children tend to fixate for long periods at stages of linguistic development characteristic of earlier, normal stages. Slight differences were found in types of base sentence forms and kinds of transformations used. But these differences were apparently due to plateau effects, in which deviant children expanded and elaborated on available linguistic forms from a restricted repertoire.

TEMPORAL ORDER PERCEPTION FOR SPEECH AND NON-SPEECH SIGNALS

If, as these recent studies suggest, developmental aphasia is most accurately described as severely delayed and prematurely arrested linguistic development, then the problem remains to further identify the nature of that delay. We have turned to an evaluation of auditory functioning in children with this disorder for two reasons. First, from a developmental standpoint, the principal normal channel of linguistic activity is auditory-vocal. Second, at the time we began our investigations in this area, rather surprising auditory processing disorders associated with aphasia had already been established experimentally, for both adult subjects and children (Efron, 1963; Lowe and Campbell, 1965). Those results are now well known. Basically, it was discovered that aphasic subjects could not accurately report the order of occurrence of two different relatively brief auditory signals unless those signals were separated by excessively large, silent intervals. Frequently, those intervals had to be increased by a factor of 10 before aphasic subjects could approach the accuracy of normal controls.

The initial interpretation of those results was generally naive. It was suggested that if aphasic subjects could not correctly perceive the order of experimental sounds, then they should also be unable to understand spoken language because of the need to keep the acoustic and linguistic segments of the speech signal in proper sequence. This was a gross oversimplification. Closer examination revealed that even subjects with relatively little receptive language difficulty still had great difficulty in ordering experimental stimuli.

As a matter of fact, in our own experience, all of the older children who have served as subjects have had quite usable language, in spite of the fact that in most cases we have found abnormal auditory processing in those same individuals.

In our own study of temporal order effects (Rosenthal and Eisenson, 1970), we decided to try to isolate some of the factors associated with poor order perception. We used short speech sounds as stimuli and measured subjects' ability to order those sounds compared to similarly contrasting non-speech sounds. For example, one stimulus pair consisted of two vowels. Performance on that pair was compared with two pure-tones which differed in frequency. Similarly, a vowel-affricate pair was compared to a tone-noise pair. Figure 1 shows sound spectograph recordings of the latter two stimulus pairs. They are illustrative of the type of signals and time domains used in this experiment. The time between members of each pair was systematically varied and the correct response rate was determined for subjects at each change.

The results of this study may be summarized in the following way:

1) When presented singly, individual stimulus items were learned and subsequently identified with high accuracy by both aphasic and normal children. Therefore, the inability to report correctly the order of pairs of stimuli was apparently not a simple function of identification or discrimination difficulty.

Figure 2 is illustrative of other major findings. That figure shows the rate of correct order responses as a function of interstimulus intervals for tone-noise and vowel-affricate pairs.

2) Aphasic children were consistently poorer at ordering stimuli regardless of the interval duration between those stimuli.

3) Both aphasic and normal children ordered speech pairs more accurately than similarly contrasting non-speech stimulus pairs.

In Figure 3 the contrast measured was between two sets of speech stimuli. The first set consisted of two fricative sounds, /s/ and /š/, which had exactly identical duration and rise times. The second set consisted of /š/ and /tš/. These sounds had identical spectral characteristics, but differed in duration and rise time.

4) Stimuli differing in features defined by spectrum were relatively easier for aphasic children to order in contrast to sounds differentiated on the basis of temporally coded distinctions, such as signal duration and signal rise time. The case was reversed for normal subjects.

The identification of singly presented stimuli was not a particularly difficult task for aphasic subjects. However, when those same stimuli were paired in close temporal proximity and subjects were required to report the order of their occurrence, aphasic performance disintegrated. These results, taken together, suggest that the nature of the auditory processing defect in aphasic children is primarily in the domain of short-term auditory storage or memory. Apparently, the auditory trace is either not retained or cannot be recalled for a time period sufficiently long to allow accurate perceptual analysis. Furthermore, within the limits of this study the degree to which that auditory storage function is affected depends on both the degree and type of contrast between paired auditory stimuli.

The results of the temporal order study still leave unanswered basic questions about auditory processing in aphasic children. The effects we observed tended to implicate some kind of fundamental difficulty in doing temporal analysis of auditory signals. But, at the same time, the effects of cognitive analysis were also apparent in the differences between speech and non-speech stimuli. The possibility of interaction between cognitive functions on the one hand and rather base, perceptual analysis on the other, could not be ruled out.

THRESHOLD-DURATION FUNCTIONS: BASE AUDITORY PERCEPTUAL FUNCTIONING

In a subsequent study (Rosenthal, 1971) we have attempted to refine our understanding of the nature of the auditory processing disorder in aphasic children by measuring the degree to which stimulus duration is related to signal detection. In normal subjects, and at near threshold levels, the auditory system appears to add up or integrate the moment to moment energy in continuous signals. Consequently, within certain limits, progressively less signal intensity is required for detection as signal durations are increased. This relationship between intensity and duration is approximately linear for pure-tone signals over a range of durations from 3 msec to about 250 msec. Within that range, a detection threshold advantage of about 10 dB is realized for each ten-fold increase in signal duration. Since aphasic children appear to be at a severe disadvantage when temporal constraints are put on auditory processing, we reasoned that signal intensities would have to be increased abnormally in order to maintain normal detectibility.

The results of this study were more equivocal than those obtained on temporal-order tasks. Only about half of the subjects tested showed significantly abnormal relationships between signal duration and detection thresholds. Figure 4 illustrates the difference between threshold-duration functions of five normal and five aphasic subjects for one ear and frequency combination. As a group, aphasic subjects required substantially greater increases in signal intensity than did normal subjects. But this difference was present only at the shortest signal durations. Furthermore, the vertical lines, which indicate the range of individual thresholds, show extreme variability among aphasic subjects in contrast to normals. Similar results were obtained for the right ear at this frequency (Figure 5). However, much to our puzzlement, no reliable differences between aphasic and normal subjects were found for a higher frequency, 4000 Hz signal (Figures 6 and 7).

AN AUDITORY MODEL OF DEVIANT PERCEPTION

Our interpretation of these results is admittedly speculative. However, we believe they are best explained by adopting two, somewhat different models. The first is useful in understanding the implications of base, auditory findings.

With some latitude of interpretation the critical-band concept provides an attractive basis for explaining the mechanism underlying abnormally increased thresholds at short durations. Briefly, the critical-band concept states that the auditory system can be viewed as a narrow-band-pass filter when the signal is a sinusoid presented in wide-band noise. The present experimental conditions met those requirements. Acceptance of the concept also implies that the ear is not responsive to sounds which are outside the frequency range of the critical-band. In his review of critical-bands, Scharf (1970) has pointed out that most research indicates that the critical-band for a particular frequency widens at short signal durations. In the

present study the data at 1000 Hz are consistent with the hypothesis that in the language deviant subjects such a widening of the critical-band does not occur to the same degree as in normal listeners. Consequently, signal energy "spills" outside the region of the critical-band filter, raising signal threshold.

The principal obstacle to this interpretation is the inability of auditory theory to resolve the issue of whether critical-bands are a fixed property of cochlear mechanics and anatomy, or whether they are a function of internal filtering which is subject to cognitive control. In view of the fact that critical-band widths appear to vary according to stimulus and experimental conditions, the latter view seems most supportable. Furthermore, the notion of an adjustable internal filter under cognitive control helps explain one possible basis for the frequency specific results in the present study. The problem posed to the subjects in the present experiment was to detect at near-threshold levels the presence of a periodic signal in a background of non-periodic continuous noise. The details of the decision process under these conditions are not fully understood.

However, auditory theory generally views the detection task as made easier when progressively more signal parameters (e.g., frequency, phase, duration, intensity) are known to the listener beforehand (for detailed review see Green and Swets, 1966). It is presumed that the listener is able to "pre-set" his internal filter in anticipation of expected signals. The events leading to a detection decision may thus go through the following steps: 1) the onset of the test signal or some non-signal event requires the listener to test the hypothesis that a signal is present; 2) the pre-set internal filter examines the input for the proper parameters; 3) the decision for or against the presence of the signal is made. If we assume, however, that the internal filter is not fully pre-set in anticipation of all signal parameters, then when Step 1 occurs, time must be spent in adjusting the internal filter. If that adjustment is sufficiently long and the signal duration is short, little may remain of the actual signal when adjustment is complete. At very short durations of a 1000 Hz tone, for instance, only one or a few periods may be available at peak amplitude for testing the hypothesis that a signal is present. At higher frequencies and longer signal durations this delay in adjustment would be mitigated.

A second related possibility is that low frequencies, which are assumed to be processed by a "counting" mechanism involving direct neural tracking of signal amplitude peaks, may present unique problems for temporary storage and detection. On the other hand, higher frequencies, coded primarily by "place" information in the cochlea, may require less rigorous adjustment of the properties of the internal filter.

LINGUISTIC MODEL OF LANGUAGE DELAY

The nature of the interaction between abnormal auditory processing and language dysfunction is understood best by assuming an analysis-by-synthesis model of linguistic processes. That model holds that generalized rules for perception and production govern phonological, morphological, syntactic and semantic relations. Such rules are the basis for testing hypotheses about the content of messages received. One of the major features of such a system is its ability to function adequately under adverse, noisy listening conditions.

To a great extent, mature linguistic systems function independently of the constraints of primary perceptual processes. That is, adult speakers of a language are not entirely dependent on the physical parameters of the acoustic speech signal. For adults, both language experience, redundancy in the signal, and contextual cues assist effective communication even when listening conditions are poor.

On the other hand, children who are actively acquiring linguistic rules are more directly dependent on the acoustic speech signal, especially while learning phonemic feature distinctions. If accurate tracking of the speech signal is impossible, then normal development of the child's linguistic system is likely to suffer.

The implications of the foregoing analysis may represent a strain on linguistic theory. It is suggested that, initially, the disorder of auditory functioning in aphasic children interferes with the establishment of a firm phonological base. However, analysis of the established language systems of aphasic children show morphological, syntactic and semantic deviations as well. Consequently, one must assume either that a phonological base is a requisite foundation of subsequent linguistic development, or that auditory dysfunction does not adequately account for the developmental aphasic syndrome.

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LEGENDS FOR FIGURES

Figure 1 - Sound spectrograph pictures of stimulus pairs T_1N and V_1A.

Figure 2 - Mean percentage of correct order responses by aphasic and normal groups plotted as a function of interstimulus interval (ISI) for the tone-noise (T_1N) and vowel-affricate (V_1A) stimulus sets.

Figure 3 - Mean percentage of correct order responses by aphasic and normal groups plotted as a function of interstimulus interval (ISI) for the fricative (F_1F_2) and fricative-affricate (F_2A) stimulus sets.

Figures 4-7 - Thresholds relative to a 1000 msec signal are shown plotted as a function of 5 signal durations. Each data point, based on not less than 800 observations, is the group mean threshold for the .70 detection level measured under constant response criteria. Vertical lines indicate the range of individual thresholds of normal and aphasic subjects at each duration. There are 5 subjects at 1000 Hz, and 4 subjects at 4000 Hz in each group.

FIGURE 1

SOUND SPECTROGRAPH PICTURES OF STIMULUS PAIRS T_1-N AND V_1-A

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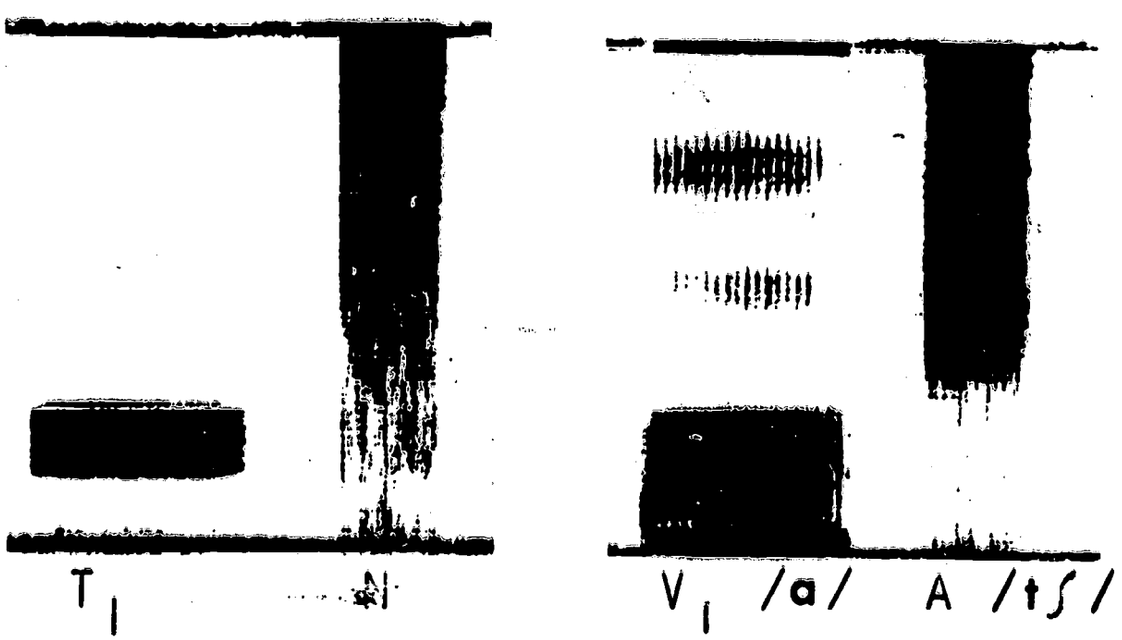

200 MSEC.

FREQ. RANGE SHOWN 0-5KHz

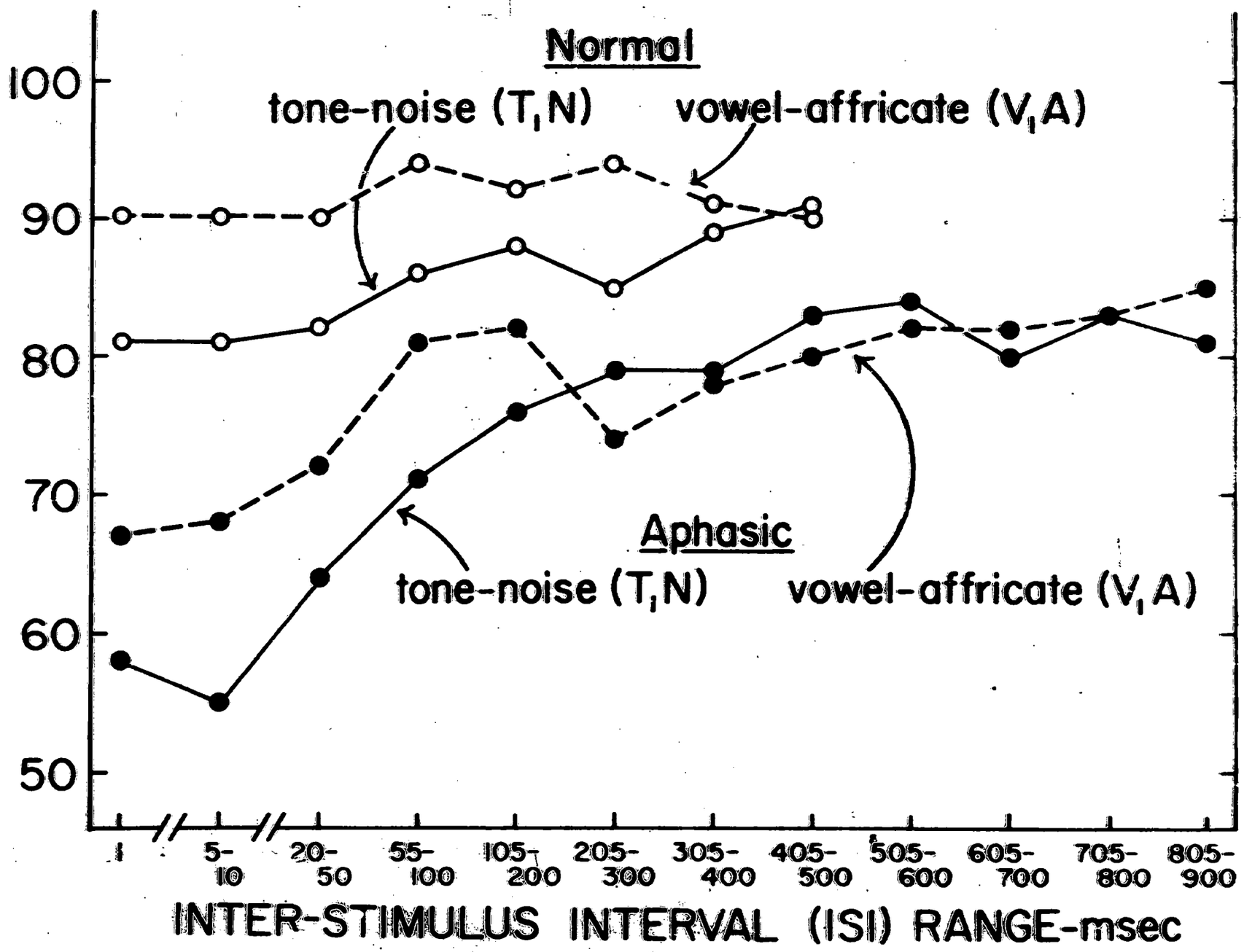
Normal
tone-noise (T,N)
vowel-affricate (V,A)
Aphasic
tone-noise (T,N)
vowel-affricate (V,A)
AVERAGE PER CENT CORRECT ORDER RESPONSE
100
90
80
70
60
50
1
5-10
20-50
55-100
105-200
205-300
305-400
405-500
505-600
605-700
705-800
805-900
INTER-STIMULUS INTERVAL (ISI) RANGE-msec

Figure 2

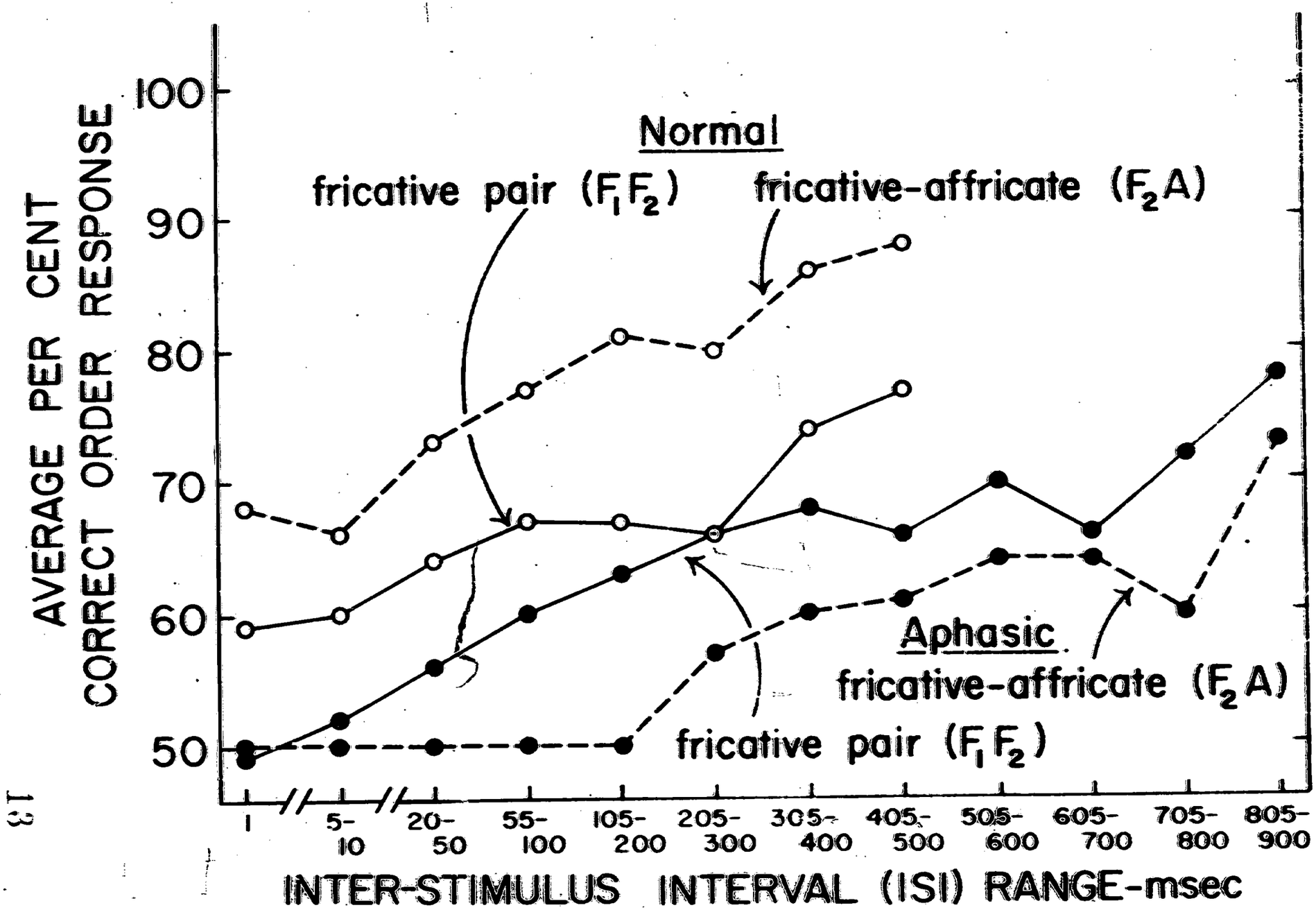

Figure 3

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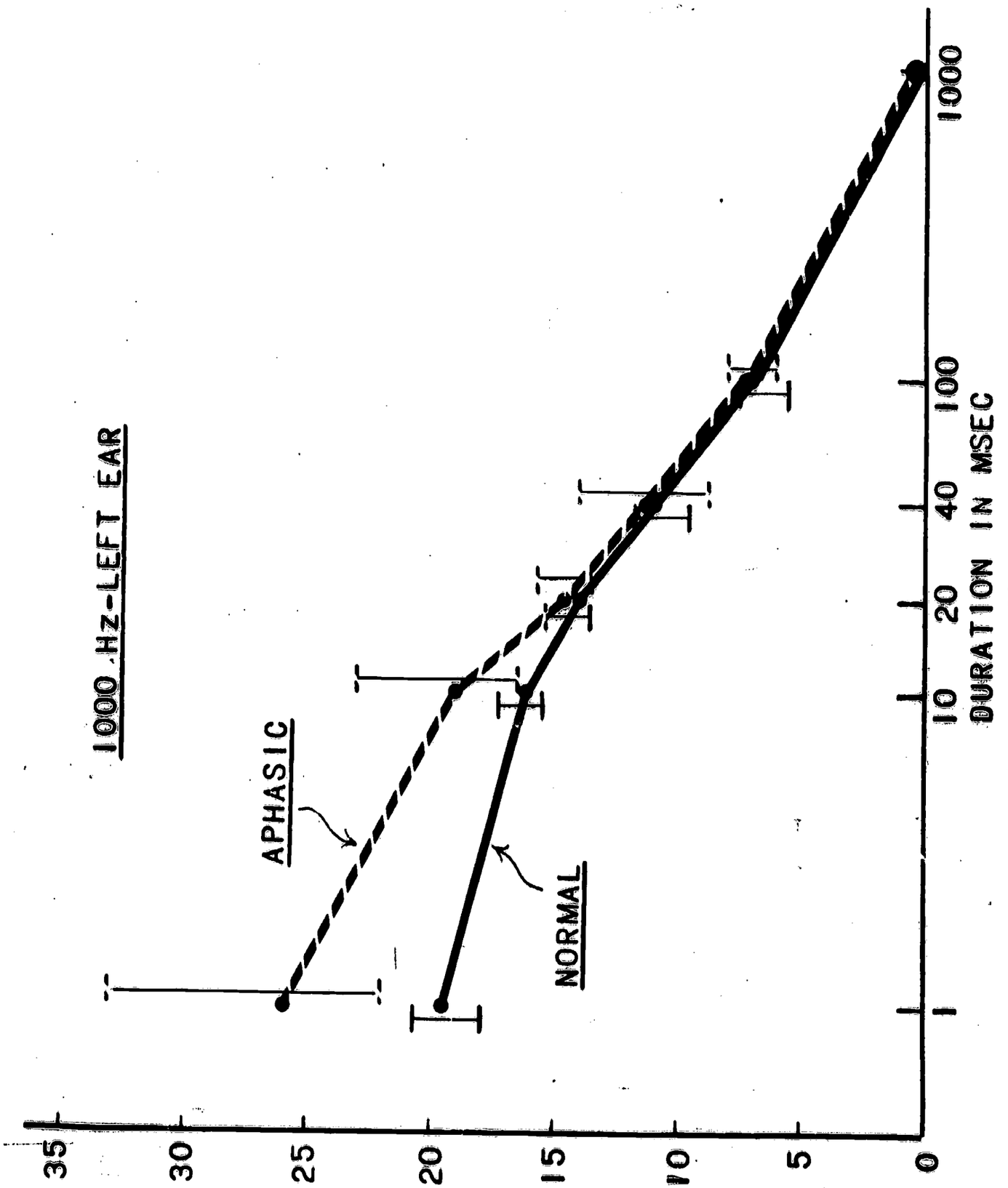

1000 Hz-LEFT EAR
APHASIC
NORMAL
RELATIVE THRESHOLD IN dB
35
30
25
20
15
10
5
0
1
10
20
40
100
1000
DURATION IN MSEC

Figure 4

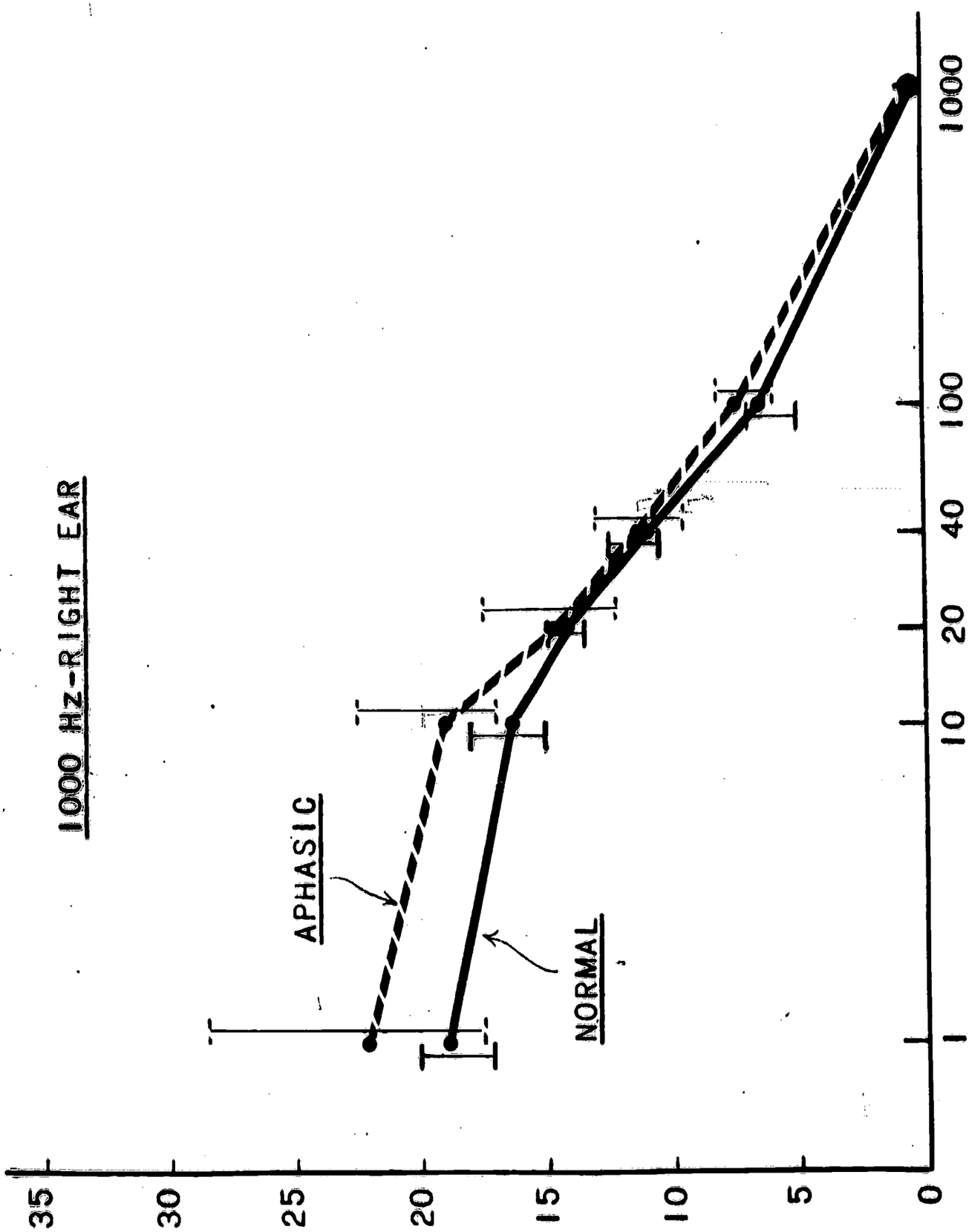
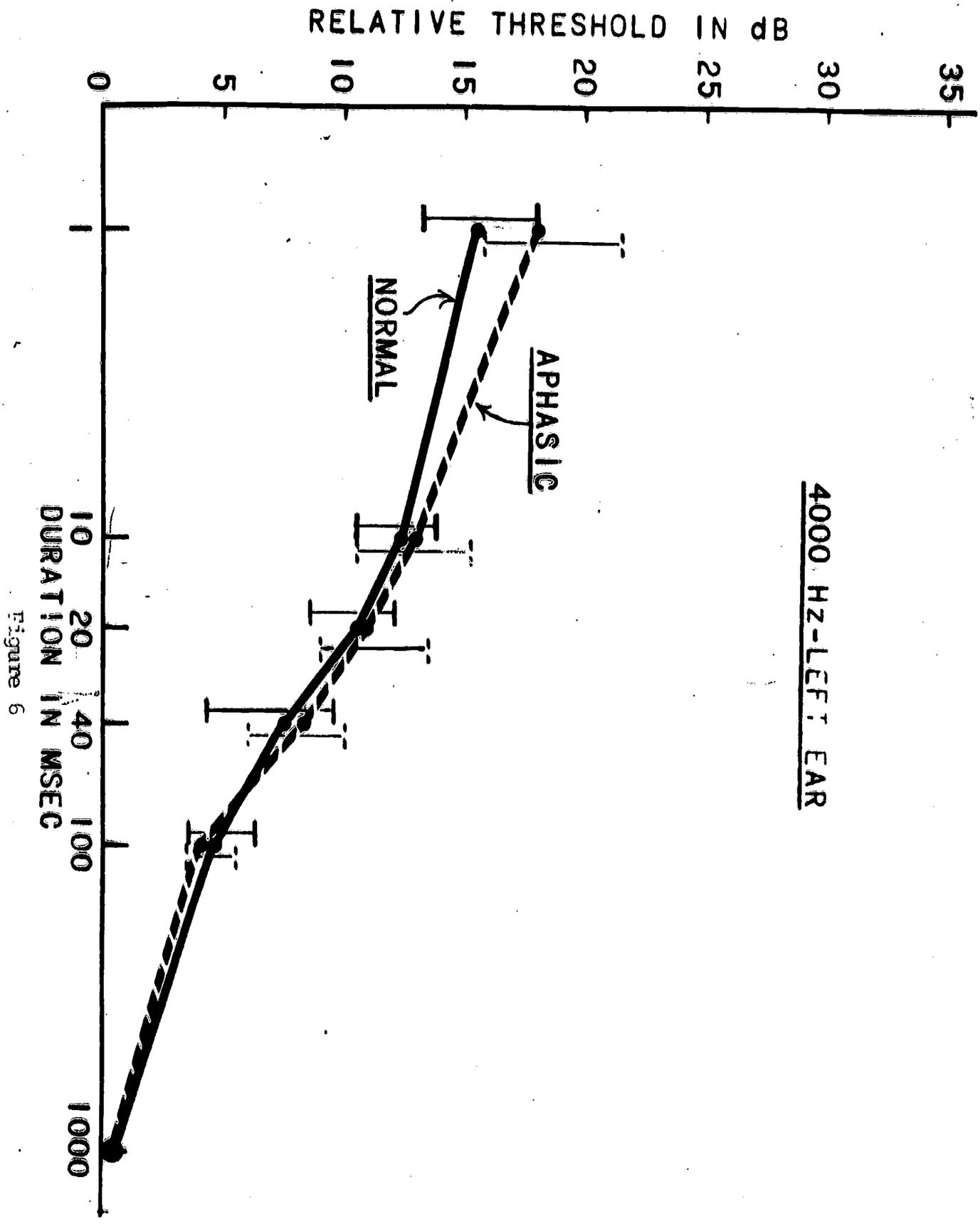

1000 Hz-RIGHT EAR
APHASIC
NORMAL
RELATIVE THRESHOLD IN dB
35
30
25
20
15
10
5
0
1
10
20
40
100
1000
DURATION IN MSEC

Figure 5


RELATIVE THRESHOLD IN dB
0
5
10
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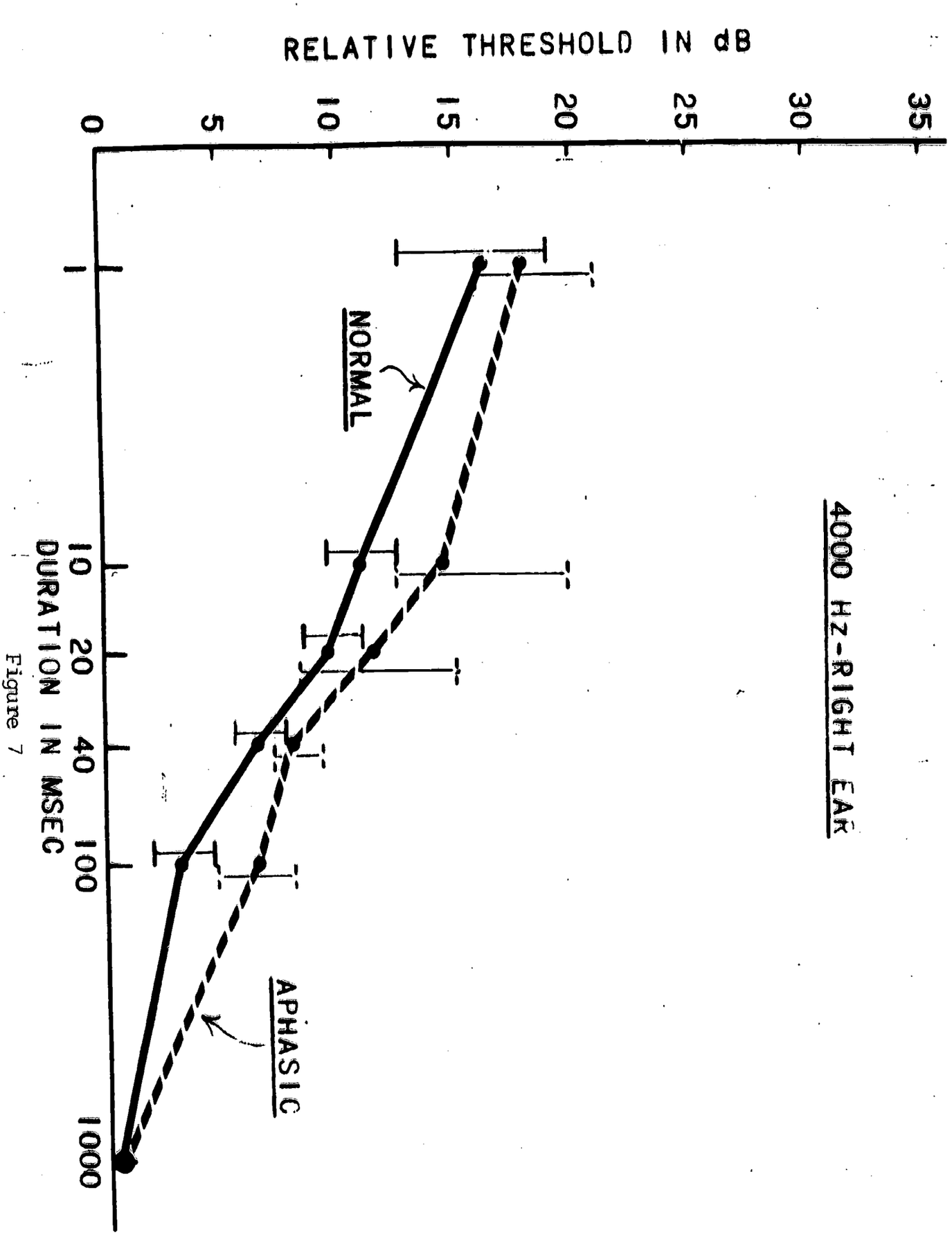
1000
DURATION IN MSEC
NORMAL
APHASIC
4000 Hz-LEFT EAR

Figure 6


RELATIVE THRESHOLD IN dB
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1000
DURATION IN MSEC
NORMAL
APHASIC
4000 Hz-RIGHT EAR

Figure 7