DEFICITS IN TEMPORAL PROCESSING ABILITY WITH APHASIA

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1.0 INTRODUCTION

Aphasia is a communication disorder, typically due to a stroke affecting the left hemisphere of the cortex. In a schema proposed by McNeil and Kimelman,[1] it was suggested that observed language deficits in aphasia are secondary to primary deficits in the processing of the intensity, duration and frequency of sound. Research studies support this view. Individuals with temporal lobe pathology and speech perception deficits also have reduced acuity for a change in frequency and duration.[2,3] Comprehension deficits specific to aphasia are related to difficulty with the processing of temporal order.[4] The present study was conducted to determine the relevance of both site of pathology and the severity of language processing difficulties to the processing of nonlinguistic auditory temporal stimuli in aphasic individuals.

2.0 METHODS AND MATERIALS

2.1 Subjects

One group of normal subjects (N=20) and two groups with confirmed pathology of the left posterior (N=16) and left anterior (N=6) hemisphere from stroke, participated. Months post onset of symptoms ranged from 2-37 months. Subjects in all three groups had screened normal hearing and were right-handed, native English speakers, and 30-70 yrs of age.

2.2 Procedure

Standardized tests of speech-language performance were administered to each subject in a quiet room. The tests included the Porch Index of Communicative Abilities (PICA),[5] the Boston Diagnostic Aphasia Examination (BDAE),[6] and the Revised Token Test (RTT).[7] Each test included subscales which required subjects to listen, speak, read, write, gesture and manipulate objects.

The psychoacoustic tests were carried out in a double-walled sound proof booth. The apparatus has been previously described.[8] Duration difference limens (DLs) were measured in each ear for standard durations of 50 ms (R/D=10 ms) and 300 ms (R/D=50 ms), using a two-interval forced-choice procedure.[9] The short standard represented the shortest duration encountered in speech (e.g. fricative) and the long standard was the average duration of a syllable. The stimulus was a one-third octave noise band centred at 2 kHz, presented at a comfortable listening level. Subjects responded by means of a response box placed on the side contralateral to the lesion.

3.0 RESULTS AND DISCUSSION

The mean DLs observed for the short standard were 14 ms, 35 ms and 45 ms for the normal, left posterior and left anterior groups respectively. The DLs for the long standard were 45 ms, 79 ms and 82 ms. There was no difference between ears within group. Outcomes for the normal group were comparable to published values.[8] The differences in the DLs for the normal and left posterior groups were statistically significantly ($p<0.001$). Individual results for the six subjects with left anterior lesions were within the range observed for the left posterior group.

The left posterior group also showed significantly lower scores on each of the three speech tests, relative to normal. Again, the range of values for the left anterior subjects overlapped those of the posterior group. For individuals with left posterior lesions, the DLs for the short standard measured in either ear and the DL for the the long standard measured in the right ear were negatively correlated with the results of the PICA ($p<0.01$).

The results confirm that individuals with left hemisphere lesions have reduced duration discrimination ability. Site of lesion, posterior versus anterior, does not appear to be a signicant factor. Severity of language processing deficit as measured by the PICA is a significant correlate of outcome.

Acknowledgments

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