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Language-Stuttering Relationship

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Introduction

Developmental and persistent speech disfluency has been observed in many cultures and languages, in general affecting the male more than the female, nonetheless the cause of stuttering has remained elusive (Rosenfield, 1997). Growing evidence from cortical imaging points to a neurological cause for the developmental persistent stuttering. Implicated are: cortical structural asymmetries, increased cortical tissue, enhanced metabolic activity, greater EEG activity in the right brain, and compensating right hemispheric participation. The confluence of these factors falls in line with earlier observations of acquired disfluency in post-stroke aphasics. Tantalizingly much neurological/neuroradiological evidence is focused upon cortical centers dedicated to language in the left brain (Foundas et al., 2001).

A stuttering dysfluency intertwined with language has not been collectively examined but it suggests that subjects who stutter perform slowly or differently on language tasks, even when responding non-verbally. There are language components and computations that are either associated with stuttering or trigger dysfluent speech movements. Marked with “slow-lexicalization,” stutterers take more time in silent searching, making decisions and monitoring phonological and lexical manipulations. Further, children with developmental stuttering have a restricted variety and number of verbs. Needless to say, specific speech-language deficiencies can contribute to stuttering.

Further support for our suspicion of a neurolinguistic bases for stuttering comes from the early work by Penfield and Roberts (1959), who 50 years ago evoked disfluencies from the stimulation of language and somatosensory cortices.

We present our own observations of evoked disfluency from stimulation mapping of language cortex by focusing on the linguistic components that were simultaneously associated with stimulation-evoked disfluencies.

Methods and Procedure

Our data comes from cortical stimulation of exposed brains on three male subjects with a history of medically intractable seizures involving the temporal lobes. Assessed were: handedness, preoperative IQ, auditory-verbal language, and cerebral dominance by Wada testing. All subjects were right-handed and had IQ scores ranging from 85 to 95, and left-hemispheric dominance for language.

Various linguistic (sentence formation, grammatical completion, and object naming) tasks were used during language mapping. Experimental trials were mixed with controlled trials. Multiple samples of language behaviors were obtained from multiple cortical sites in each patient to determine the statistical significance of the evoked errors. (Bhatnagar, Mandybur, Buckingham, Andy, 2000).

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Results

We evoked multiple errors of speech disfluency (hesitancy, syllable (CV)/part word repetitions, vowel distortion and prolongation) similar to those seen in developmental and persistent stuttering. All the cortical sites where we found speech disfluencies were also the sites associated with evoked errors of language functions.

Discussion

Penfield and Roberts (1959) noted evoked stuttering during mapping of the language cortex. Similarly, we pinpointed brain sites that were associated with both stuttering and language errors. With growing evidence of altered functional anatomy in the language cortex of stutterers, who at the same time reveal altered performance on language tasks, we hope to further outline the language dynamics and cortical substrates identified with speech disfluency.

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