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Dirk B. den Ouden
University of South Carolina - Columbia, denouden@sc.edu

Charles F. Adams
University of South Carolina - Columbia, cfadams@mailbox.sc.edu

Allen A. Montgomery
University of South Carolina - Columbia, amontgom@mailbox.sc.edu

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SIMULATING THE NEURAL CORRELATES OF STUTTERING

Dirk den Ouden, Charles Adams, Allen Montgomery
University of South Carolina, Columbia, United States of America

Introduction
Neural activation associated with dysfluent speech in developmental stuttering is characterized by hyperactivity of right hemisphere motor and premotor cortex, combined with overall reduced left-hemisphere perisylvian activation, particularly in auditory cortex. However, two challenges exist for neuroimaging studies of stuttering, namely (1) the elicitation of naturally stuttered versus fluent speech and (2) the separation of activation associated with abnormal motor execution from activation that reflects the cognitive substrates of stuttering (see De Nil et al. 2008). We made use of a speaker’s insight into his own stuttering behavior, to create a list of single-word trials on which he is likely to stutter, versus a matched list of ‘fluent’ words. In addition, a speech pathologist was trained to imitate the articulatory and facial motor pattern associated with this speaker’s stuttering. Both performed an fMRI experiment of single word reading, with the same lexical items.

Methods
A sparse scanning design was used, with a fixed SOA of 10 seconds. Participants read aloud words presented on a screen, with a control condition showing a nonsense letter string, not requiring a response. One brain volume was acquired 3 seconds after each word presentation, for a total of 48 trials per condition. The dysfluent speaker’s (DS) recorded responses were analyzed for whether his stuttering pattern matched the anticipated pattern. For the fluent speaker (FS), word trials were color coded to achieve complete matching between his output and the real stutters of DS. Data were analyzed separately, using one-way ANOVAs by trial with condition as a three-level factor.

Results & Discussion
Both DS and FS show bilateral temporal and auditory cortex activation in fluent as well as dysfluent speech. In contrast to De Nil et al.’s (2008) group results, we do find differences between dysfluent and fluent speech in FS, which are overall very similar to those observed in DS. However, for the contrast of dysfluent speech versus the control condition, DS does show a greater right-hemisphere activation bias than FS, visible in motor cortex, supramarginal gyrus and anterior middle temporal gyrus. In addition, DS shows bilateral prefrontal activation that is not observed in FS’ simulated stuttering. These results suggest that some of the classically reported neural activation patterns associated with stuttering are driven more by nonspecific motor patterns than by the cognitive substrates underlying stuttering. Nevertheless, the generally observed right-hemisphere lateralization in speakers who stutter appears to reflect a true characteristic neural correlate of developmental stuttering.

Reference