Simulating the neural correlates of stuttering
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Introduction
Neural activation associated with developmental stuttering has been characterized by hyperactivity of right hemisphere motor and premotor cortex and overall reduced left-hemisphere persylvian activation, particularly in auditory cortex (Fox et al., 1996). Other areas that have been found to play a role underlying stuttering are left anterior cingulate cortex (De Nil et al., 2000), the thalamus (Bhatnagar & Andy, 1989), cerebellum (De Nil et al., 2001) and basal ganglia (Giraud et al., 2008).

Two challenges for neuroimaging studies of stuttering:
(1) the elicitation of naturally stuttered versus fluent speech
(2) the separation of activation associated with abnormal motor execution from activation that reflects the cognitive substrates of stuttering.

A study by De Nil et al. (2008) did not find differences between simulated stuttering and fluent speech in a nonstuttering group, but this may be due to the fact that the simulated stuttering was too ‘weak’. De Nil et al. suggested that a ‘true stuttering’ condition be included, but pointed to practical problems, which we have tried to address in the present feasibility study.

We made use of a speaker’s insight into his own stuttering behavior, to create a list of words on which he is likely to stutter, versus a list of ‘fluent’ words. 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.

Table 1: matching data for the lists of fluently spoken vs nonfluently spoken words

<table>
<thead>
<tr>
<th>Fluent</th>
<th>Nonfluently spoken</th>
<th>Statistics</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kucera-Francis written frequency</td>
<td>122.6</td>
<td>163.2</td>
<td>F(1,6) = .755</td>
</tr>
<tr>
<td>Imagability</td>
<td>446.7</td>
<td>392.8</td>
<td>F(1,6) = 3.01</td>
</tr>
<tr>
<td>Length in graphemes</td>
<td>8.6</td>
<td>6.8</td>
<td>F(1,6) = 1.86</td>
</tr>
<tr>
<td>Length in phonemes</td>
<td>7.8</td>
<td>6.8</td>
<td>F(1,6) = 2.36</td>
</tr>
<tr>
<td>Length in syllables</td>
<td>3.1</td>
<td>3.2</td>
<td>F(1,6) = 0.83</td>
</tr>
<tr>
<td>Inset type (vowel, consonant, cluster)</td>
<td>(k&gt;3)</td>
<td>(k&gt;3)</td>
<td>.000</td>
</tr>
</tbody>
</table>

Methods
Participants
Both left-handed monolingual speakers of American English. The disfluency speaker (DS) was 65 yrs old, while the fluent speaker (FS; a speech-language pathologist familiar with DS’ stuttering pattern) was 47 yrs old.

Materials
Based on DS’ insight into his own stuttering pattern, two lists of 24 ‘fluent’ and ‘nonfluential’ words were matched for frequency, length (in phonemes, graphemes and syllables), imageability and onset type (see table 1)

Training
FS was trained to imitate DS’ stuttering pattern during two 2-hour training sessions, in addition to home study of video recordings of DS’ stutterers.

fMRI experiment
Participants openly read words presented on a screen, with a control condition in which nonsense letter strings were presented, not requiring a response (see figure 3). Participants were allowed to speak (with or without stuttering) only during word presentation.

fMRI scanning was performed at 3 Tesla, with a sparse scanning design (TR and fixed SOA: 10 seconds). One whole brain volume was acquired 3 seconds after each word presentation, with an acquisition time of 2 seconds, for a total of 144 acquisitions over 2 runs (48 trials per condition).

DS’ recorded responses were coded offline to register whether his stuttering pattern did indeed match the anticipated pattern. DS stuttered on 5 ‘fluent’ trials (10.4%) and was fluent on 1 ‘dysfluential’ trial (2.1%). For participant FS, word trials were colored on the presentation screen (green for fluent, red for nonfluential), to achieve complete matching between his output and the real stutterers of DS. Data were analyzed separately for each participant in SPMS, using one-way ANOVAs by trial, with condition as a three-level factor. The significance threshold was set at \(p<.05\) (FWE correction; \(k>3\)).

Results & Discussion
Both DS and FS show temporal and auditory cortex activation in fluent as well as dysfluent speech. In addition to other activation foci, FS shows activation in left anterior cingulate (area of dysfluent speech, which is not present during the natural stutters of DS. In contrast to De Nil et al.’s (2008) group results, we find differences between dysfluent and fluent speech in FS, which are overall similar to those observed in DS (Fig 1). However, for the contrast of dysfluent speech versus the control condition, DS shows a greater right-hemisphere activation bias than FS, visible in motor cortex, supramarginal gyrus and anterior middle temporal gyrus (Fig 2), as well as prefrontally.

Conclusions
This study shows that it is feasible to match stuttering speakers’ utterances more closely to simulated stutters, for the investigation of neural correlates of real stuttering, by (1) eliciting words that are known to be ‘stutter prone’ in individual speakers, and (2) training nonstuttering speakers to imitate closely the individual motor behavior of real stutters.

Results suggest that:
1. Some of the classically reported activation associated with stuttering is driven more by nonspecific motor patterns than by cognitive substrates of stuttering.
2. Anterior cingulate activation may reflect awareness of (upcoming) dysfluencies, rather than a process that is specific to natural stuttering.
3. The generally observed right-hemisphere lateralization appears to reflect a true neural correlate of stuttering, whether due to neurogenetic, early developmental or later adaptive behavioral factors.

References