Simulated stuttering in non-stuttering healthy subjects: differentiating cause from consequence in developmental stuttering

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Introduction

Over the past decade several functional neuroimaging studies in developmental stuttering have been published\(^1,2,4,5,6\). In comparison to non-stuttering volunteers, these studies revealed that adults who stutter show overactivation in areas such as the cerebellum, supplementary motor area (SMA), right frontal operculum (RFO), and the right insula\(^1,2,4,5,6\). This overactivation mostly occurs bilaterally but a right-hemisphere bias can be detected\(^5\). In addition, stuttering speakers show reduced activation in areas involved in auditory processing\(^6\) and severity of stuttering is correlated with basal ganglia (BG) activation\(^2\). However, it is still unknown whether these differences point to potentially causal factors in stuttering or, alternatively, may be the result of increased effort while producing speech. Comparison of activation patterns during simulated stuttering in healthy volunteers with activation patterns in stuttering speakers might help in differentiating the cause from the consequence of the stuttering. In a previous study, using an auditory paradigm, simulated stuttering did not reveal differences in activation patterns in comparison to non-stuttered speech in healthy controls\(^5\). In the present study we aimed at investigating the effects of simulated stuttering with a different, more language-based, paradigm.

Materials and Methods

At present five healthy right-handed adult volunteers (3 ♀, 2 ♂) without history of speech and language problems participated in the study. The fMRI scanning was performed on a 3T MR-system (Philips INTERA) with a 8-channel head coil. Stimuli were presented visually in a block design paradigm (90 volumes, TR/TE = 3000/33 ms, FOV = 230x230 mm, matrix = 80x80, slice thickness = 4 mm, 35 contiguous transversal slices, flip angle 90°, SENSE factor 2). Stimuli consisted of 30 words (W), 30 pseudowords (PW) and 30 strings of characters (C) (see Fig. 1). A new word was presented every 3 seconds, with a duration of 30 seconds per block of stimuli. The experiment consisted of 5 different conditions:

1. silent reading of words and pseudowords (read)
2. overt reading of words and pseudowords (speak)
3. silent simulated stuttering: read words and pseudowords silently and repeat the first letter of the word multiple times (e.g. b-b-baard) (stutter silently)
4. overt simulated stuttering: read words and pseudowords overtly and repeat the first letter of the word multiple times (stutter overtly)
5. passive looking at non-sense characters (baseline)

Each condition was presented twice to each subject and the order of presentation was randomized. Analysis of the functional images was done with SPM2 using standard procedures for head motion correction, non-linear spatial normalization, spatial smoothing and general linear model statistics. Additionally, to reduce confounding signal changes from bulk head motion, especially during overt reading, motion parameters were added in the general linear model as covariates of non-interest. For quantitative analysis, spherical regions of interest (ROI’s) of 7 voxels each were drawn around the local maxima extracted from the different contrasts. Percent signal change was extracted in each ROI using a custom-designed procedure in SPM2. The obtained mean values for the percentage MR signal change were compared using an unpaired two-tailed student t-test. Significance threshold was set at FWE p < 0.05 corrected for multiple comparisons.

Results

Silent reading (W and PW) compared to the baseline condition (C) resulted in a typical network of brain activity in the left ventral occipitotemporal cortex, supramarginal gyrus (SMG), and receptive language cortex. Overt versus silent reading resulted in activation in the motor, auditory cortex and in the cerebellum bilaterally during both the reading and the stuttering task. In addition, activation in the thalamus and hippocampus is shown during both the reading and the stuttering task. The main condition of interest in this study, overt simulated stuttering compared to overt fluent reading, resulted in higher activation in the right primary and secondary auditory cortex, right basal ganglia, right insula, SMA, left cerebellum, and left supramarginal gyrus (Fig. 2, Table 1).

Conclusion

In this study we used a language-based paradigm to compare simulated stuttering to fluent speaking, in order to differentiate cause from consequence in developmental stuttering. We found that normal subjects performing simulated stuttering engage areas, notably the SMA, premotor cortex, basal ganglia, insula and cerebellum, that were formerly implicated in developmental stuttering. This observation supports the hypothesis that activation in these regions may result from higher demands on speech and language processing during dysfluent speech, even in normal subjects. This suggests that these differences reflect the increased effort in producing speech rather than the cause of the stuttering.

In contrast, while in our study on normally fluent speakers simulated stuttering resulted in increased activation in the primary auditory cortex, stuttering speakers have been reported to show decreased activation in this brain region during dysfluent speech. This may point to the auditory cortex as a region causally implicated in developmental stuttering.

References


Fig. 1: Stimuli consisted of strings of characters (baseline condition - C), words (W) and pseudowords (PW), presented in a block-design paradigm. Tasks were “look” in the baseline condition, and either read, speak or stutter in the PW and W conditions.

Fig. 2, Table 1: Group SPM showing regions with significant increased activation during the overt simulated stuttering task in comparison to the overt reading task.