Spatial and Subject Variability of Long-term Respiration Effects in fMRI

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INTRODUCTION

Physiological fluctuations due to respiration are the dominant source of confounding variability in BOLD fMRI at high fields [1-4]. Short-term respiration effects result from B0 modulation due to bulk susceptibility changes from thoracic organ movement and gas volume as well as head motion [3-5]. Recently low-frequency fluctuations in the depth and the rate of respiration have been identified as a source of long-term respiration effects, mediated by the carbon dioxide content of arterial blood [1,2]. Long-term effects are particularly problematic in resting-state functional connectivity analysis since their frequency range overlaps with the frequencies of fluctuations believed to reflect resting brain activity [1,6]. Therefore, it is vital to precisely characterize the impact of breathing on the BOLD signal. Respiration volume per time (RVT) and respiration response function (RRF) have been proposed as global models of respiration effects [1,7]. The global models implicitly assume that the respiration effects are space-invariant and subject-invariant. This assumption is probably not strictly met. A technique was previously introduced to estimate voxel-specific physiological impulse response function (PIRF) for characterizing the impact of respiration on the BOLD signal [8]. The goal of this study is to examine space and subject variability of long-term respiration effects by clustering the voxel-specific PIRFs.

METHODS

Eight subjects were scanned on a Siemens 3T Trio (Siemens Medical Solutions, Malvern, PA) MR scanner. Resting-state BOLD signal were acquired (Z-SAGA GE-EPI [9], TR =2.02s, TE1/TE2 =30ms/66ms, FA=90°, FOV =22cm, 19 axial slices with no gap, 3.44 x 3.44 x 5 mm^3, 210 volumes each run). Heart beat and respiration were recorded with a pulse-oximeter and a respiratory bellow, respectively. The datasets underwent slice-timing correction, motion correction, normalization with MNI152 template, and spatial smoothing with a 6mm using SPM5. The retrospective technique in [3,4] was applied to reduce short-term physiological effects. Then, voxel-specific respiration PIRFs were estimated as described in [8] with Fourier basis.

K-means clustering, based on using sample correlation as distance measure, was performed on all brain PIRFs from eight subjects using 5 clusters. The average and standard deviation of PIRFs were calculated for each cluster. To compensate for inter-subject variability in BOLD signal change, the signal was normalized before taking the average.

RESULTS and DISCUSSION

Results of the clustering are shown in Fig. 1. Cluster A was the most dominant component (34%) and was located in mostly grey matter. This cluster resembles the average PIRF across brains from all subjects (not presented here). We believed that the global model is related to this cluster. However, other clusters (B, C, and D) were highly dominant in some subjects. Cluster E was found in CSF (subject 1-3). It also overlapped with the default mode network (subject 6).

The patterns of the clusters varied substantially across subjects. The substantial variation in the average cluster PIRFs (Fig. 1B) indicates the spatial variability of respiration effect. The small standard deviations of the average PIRFs show that clusters are relatively tight.

CONCLUSIONS

The significant variability of respiration effect is observed across subject and space based on the shape of respiration PIRFs. The dominant cluster exists which can be related to global respiration effects. But, the spatial pattern is not consistent across subjects.

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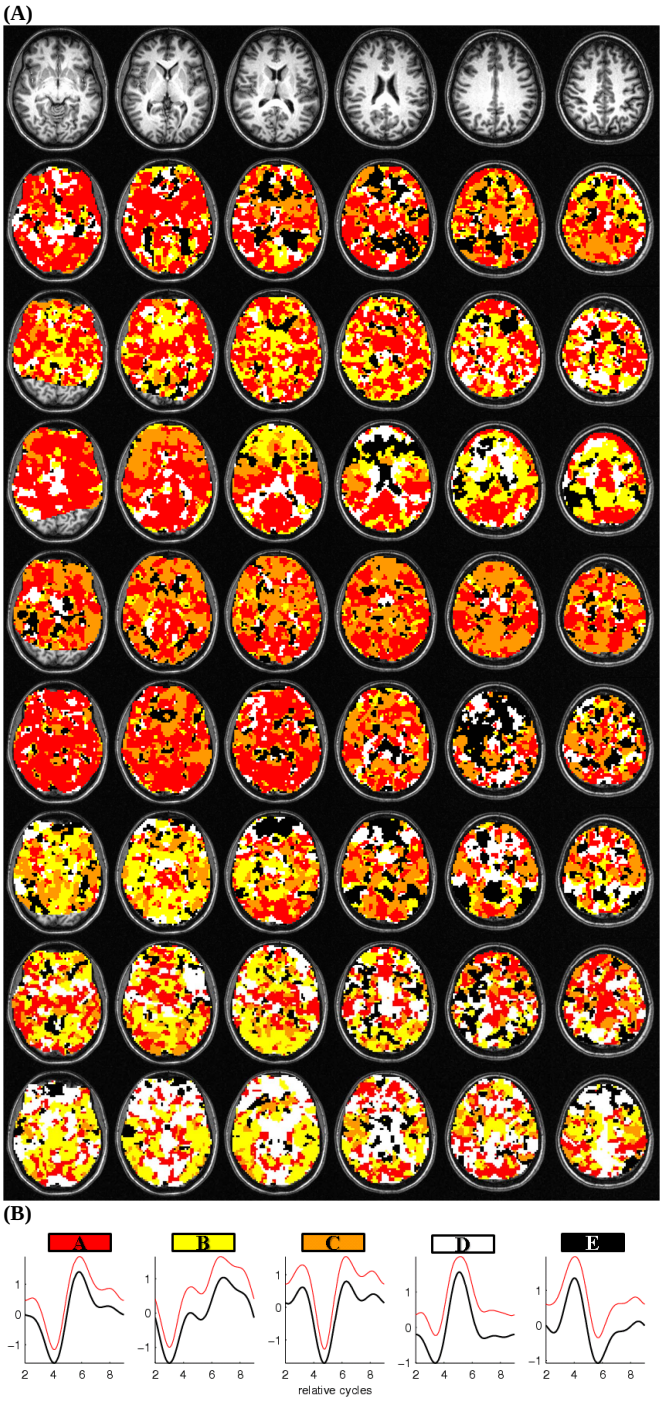

Figure 1. (A) Clustering of respiration PIRFs in 6 slices in all 8 subjects. First row represent anatomical location. Each subsequent row represents a subject. **(B)** Average PIRFs of each cluster (black line) and one standard deviation (red line).