Event related negative BOLD response observed in an attention switching task

Z. Li[1], S. Peltier[1], X. Hu[1]

[1]Emory University/Georgia Tech, Atlanta, Georgia, United States

Introduction The impulse response function (IRF) of the BOLD signal in an event-related fMRI study is commonly a positive function representing a stimulus induced signal increase. However, in a task requiring subjects to continuously shift attention in both the visual spatial and the verbal working memory domains, we observed an event-related, widespread, negative BOLD response. We investigated the physiological basis of this response. In addition, the possibility of using it to study the cognitive brain function is also discussed.

Fig.1. The task

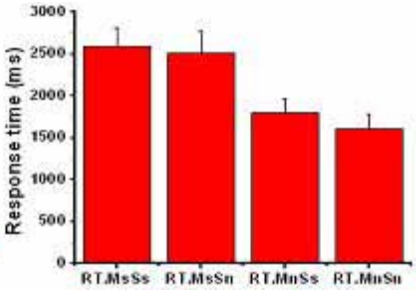

Fig. 2. The RTs of the 4 experiments

Methods Subjects (5M, 5F, age 22±1.6) were required to count a sequence of central circles/arrows (mental attention shift [1]) and at the same time to judge the color of the peripheral circles (spatial attention shift [2]) (Fig.1). According to the presence/absence of the mental and spatial attention switch, four trial types were defined (MsSs: mental shift, spatial shift; MsSn: mental shift, no spatial shift; MnSs: no mental shift, spatial shift; MnSn: no mental shift, no spatial shift). Subjects proceeded through each task block at their own pace but as quickly and accurately as possible. They could take a short break (1~5sec) between task blocks. EPI-BOLD fMRI data were collected (3T Trio Siemens scanner, matrix=64×64, TR/TE/FA/FOV=900ms/30ms/60°/22cm, 15 axial thick slices, thickness/gap=5mm/1mm) throughout the task and resting period. The impulse response function for each of the 4 stimulus type were generated by a deconvolution algorithm [3] using the AFNI software package (http://afni.nimh.nih.gov).

Results The response time (RT) data showed both the mental-shift ($F(1,9)<0.001$) and the spatial-shift effects ($F(1,9)=0.05$) with the presence of either attention shift requiring a longer RT. The presence of the mental shift led to the largest RT increase (Fig.2). Negative IRFs were observed in virtually all activated brain areas for all the four experimental conditions. Representative IRFs of the medial frontal area are shown in Fig.3. Except in 2 subjects whose IRFs were too noisy (fitting Chi-sqr>1), the difference of the width of the mental-shift (Ms) vs. no-mental-shift (Mn) IRFs was significantly correlated with the corresponding RT difference (Fig.3).

To investigate the underlying mechanism of the negative IRFs, we scanned an additional subject with a constant interstimuli interval (ISI, 3.5s) and a prolonged resting period (55s). An additional continuous arterial spin labeling (CASL) scan (8-ch array head coil, 14 axial slices, thickness/gap=5mm/1mm, TR/TE/FA/FOV=3s/17ms/90°/20cm, matrix=64×64, labeling duration=2s, sequence adapted from Dr. Wang, UPENN) was also conducted to ascertain the cerebral blood flow change.

An increased task vs. resting response was observed in both the BOLD and CBF data. Given the fixed ISI, the BOLD signal for the task blocks (30 blocks) was averaged together and a stimulus related signal dip could clearly be seen (Fig.4) while the CBF data exhibits a task related increase.

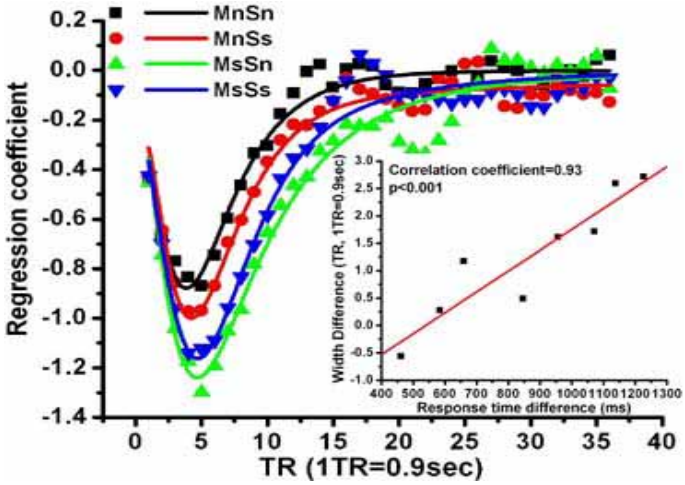

Fig.3. The IRF of medial frontal area (data points and the Gamma fitting curve). The linear correlation of the IRF peak width difference (Ms vs. Mn) and the RT difference is shown in the lower right part of the figure.

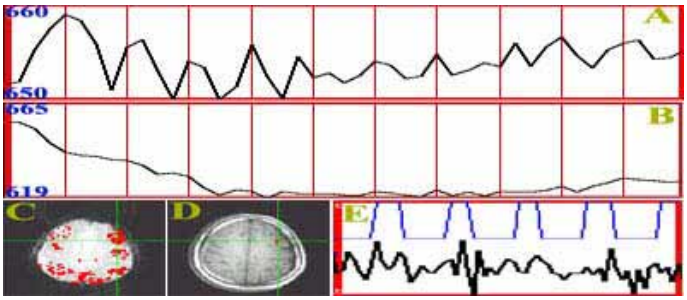

Fig.4. The mean BOLD signal of the task blocks (A) and the resting blocks (B). The vertical red grid marks the onset of each stimulus in the task block and the blue numbers indicated the signal intensity level. The CBF perfusion is higher in the task block than the rest (E). The 3 time courses are all from the same voxel activated both in the BOLD (C) and the CBF data (D).

Conclusion Because of the relatively constant CBF during the task blocks, the negative response might be generated by a similar mechanism as the initial dip [4]. The correlation between the peak width and the behavioral RT suggests the feasibility of using it to investigate brain function [5].

Reference [1]. Garavan H. 1998. Mem. Cong. 26:263. [2].Yantis S., et al. 2002. Nat. Neurosci. 5:995. [3]. Ward BD. 2002. AFNI document. [4]. Yacoub E., et al. 2001. NMR Biomed. 14:408. [5]. Richter W., et al. 1997. Neuroreport. 8:3697.

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