Detection of Brain Activation Induced by NREM Sleep Using Independent Component Analysis

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Introduction

Functional neuroimaging techniques such as PET (1) or fMRI (2) have been widely used to study brain activation associated with different sleep stages. FMRI is superior in localizing brain activation because of its excellent spatial and temporal resolution. However, the challenge of fMRI lies in the fact that temporal information is unavailable for fMRI sleep studies. For this reason, a simultaneous fMRI/EEG technique (3) has been used by many investigators to localize brain activities. This technique requires the use of specially designed MRI-compatible EEG equipment. Several issues must be considered to maintain the quality of fMRI data. For example, the components near the subject's head must not induce eddy currents. And the susceptibility-sensitive sequences, such as echo-planar imaging (EPI), are more sensitive to the signal degradation caused by EEG than are spin-echo sequences. Independent component analysis (ICA) is a widely used paradigm-independent data analysis method that does not require prior knowledge of temporal information (4, 5). Here, we report a novel method to localize brain activation associated with non rapid-eye-movement (NREM) sleep by application of the ICA data processing method, which does not require EEG monitoring.

Methods

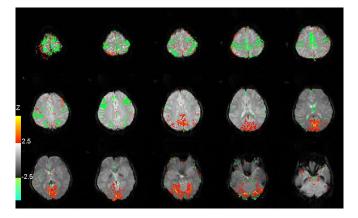
Eight healthy subjects were examined in the afternoon at least 2 hours after lunch to avoid the influence of food. When the one-hour functional scan began, the light in the MRI room was turned down and subjects were told to close their eyes and sleep. During the scan, we monitored the subjects' eye movement through an infrared camera (a dim light and a mirror were set before the eyes of subjects), and the pictures from the camera were recorded on video tapes. Five subjects claimed to have fallen asleep during the scan. 21 slices covering the whole brain were scanned by EPI pulse sequence. Imaging parameters were 5 mm slice thickness with 1 mm gap; TR/TE/ θ = 2000ms/30ms/90°; field of view = 256 x 256 mm²; and matrix = 128 x 128 pixels. 1800 functional images were obtained during the one-hour scan. The Fast ICA algorithm (4) was used for our ICA data analysis. Before applying the ICA, the data dimension was reduced to 30 through principal component analysis (PCA). The resulting 30 independent components were threshold at z = 2.5 and a spatial cluster size of 5, then overlaid on the first echo-planar image of the time series. The brain activation patterns located at the same cortical areas for the five fall-sleep subjects were taken as the true brain activation patterns.

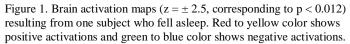
Results

We observed no jerky eye movement during the scan, indicating that all subjects who claimed to fall asleep were in the stage of NREM sleep. Figure 1 shows the brain maps resulting from one subject who fell asleep during the scan. The other four fall-asleep subjects showed similar brain activation patterns located at the same brain areas. For all fall-asleep subjects, we saw a decreased activation in frontal and parietal cortices and an increased activation in the temporal lobes, occipital lobes and bilateral cerebellum. No such brain activation patterns were seen in the subjects who did not enter sleep state.

Conclusion

We successfully detected the brain activations induced by NREM sleep through fMRI and ICA techniques. For the subjects who fell asleep during the scan, decreased brain activation was found in frontal and parietal cortices and increased brain activation was found in the temporal lobes, occipital lobes and bilateral cerebellum, which is consistent with the observations of other researchers (6). This method does not require using EEG equipment and avoids the possible degradation to the quality of fMRI data by the electrophysiological apparatus.





References

- 1. Maquet P, Dive D, Salmon E, et al. Brain Res 1990; 513: 136–143.
- 2. Lovblad KO, Thomas R, Jakob PM, et al. Neurology 1999; 53: 2193–2195.
- 3. Huang-Hellinger F, Hans C, McCormack G, et al. Hum Brain Mapp 1995; 3: 13–23.
- 4. Hyvarinen A, Oja E. Neural Netw 2000; 13: 411–430.
- 5. McKeown MJ, Makeig S, Brown GG, et al. Hum Brain Mapp 1998; 6: 160–188.
- 6. Andersson JLR, Onoe H, Hetta J, et al. J. Cereb. Blood Flow Metab., 1998; 18: 701-715.