Simple Method for Estimation of Cerebral Perfusion Reserve by BOLD Images

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Introduction

Measurement of cerebrovascular reserve capacity is important in assessing patients with cerebrovascular disease. Blood oxygenation level-dependent (BOLD) imaging can noninvasively estimate reserve capacity of the cerebral circulation based on changes in signal that reflect differences in magnetic susceptibility of intravascular oxy- and deoxyhemoglobin. We showed a simple method to detect cerebrovascular perfusion reserve using BOLD imaging.

Methods

We selected 10 male volunteers and 2 illustrative patients with cerebrovascular disease for investigation of this method. Subjects were instructed via a headphone to hold their breath following exhaling three times for 24 s each. Between the periods of breath holding, subjects were instructed to breathe normally. A 1.5 T MR instrument was used for this study. The gradient-echo mode of echo planar imaging (EPI) was carried out as follows: TR/TE, 3000/50 ms; FA, 60 Å; receiver bandwidth, 100 kHz; FOV, 24 cm. Results were sent to a workstation, and statistical software was used for analyses. A paradigm was established as follows. "Off" was breathing at rest, while "On" was breath holding; sine waves were used as the template function. The correlation coefficient of each matrix was calculated. Compared with signal changes ordinarily seen in functional MRI, the onset of signal changes following breath holding was delayed to some extent, and this delay varied among individuals. Therefore, the entire template was shifted along the time axis within one paradigm cycle to obtain maximum correlation coefficients concerning the entire brain. A color scale was used to indicate pixel-by-pixel correlation coefficients.

Results

Changes in BOLD signal in healthy volunteers;

- After 24 s of breath holding, PaCO2 rose by 4.2 to 8 mm Hg (mean, 5.5 mm Hg), and pH decreased by 0.013 to 0.064 (mean, 0.036). Breath holding-induced BOLD signal changes were seen in all healthy individuals. Immediately upon initiation of breath holding, BOLD signal transiently decreased, but then increased while the breath was being held. Immediately after breathing resumed, BOLD signal further increased (Fig. 1). Changes in BOLD signal caused by breath holding in cerebral cortex were greater than in white matter; 6.2Å/2.4% in cortex within the territory of the middle cerebral artery (MCA), or 4.5Å/0.7% in the thalamus; and 1.4Å/0.5% in white matter. The correlation coefficient distribution was higher in cortex than that in white matter (Fig. 1).

Illustrative cases of cerebrovascular disease examined by BOLD signal change;

Case 1. A 50-year-old woman was hospitalized for subarachnoid hemorrhage caused by a ruptured internal carotid artery (ICA) aneurysm. On performing coil embolization, the left ICA had to be occluded because of inadvertent migration of the coil into the ICA. During the procedure, the patient developed right homonymous hemianopsia due to embolism to the calcarine artery (presumably the operculoinsular area, and the interhemispheric fissure. The degree of breath holding change in BOLD signal in the territory of the MCA (temporal lobe) was 6.2Å/2.4%, while in the thalamus it was 4.5Å/0.7% and in the white matter (centrum semiovale) it was 1.4Å/0.5%. Thus, changes in the cerebral cortex and thalamus were greater than those in the white matter. These findings agreed with previously reported results of acetazolamide-activation CBF and BOLD studies.2

- Interesting results were observed in patients with cerebrovascular disease. In patient 1, who had occlusion of the left internal carotid artery and embolism to the left calcarine artery, SPECT CBF study before and after acetazolamide administration disclosed reduced reserve capacity in the left hemisphere where PET showed a slight increase in OEF. BOLD signal changes during breath holding were absent in this area. In patient 2, diagnosed with moyamoya disease, an abnormality was found in the right temporoparietal area on a BOLD correlation map. The time-intensity curve of BOLD signal changes in this area showed delay compared to those in the other areas, resulting in a negative correlation. Compared to the other side, the degree of the BOLD signal change in this area was slightly decreased. In this patient, SPECT CBF study showed a mildly reduced response to acetazolamide in the right temporal lobe, but was more favorable in the parietal region. Detecting a response delay in such a patient is facilitated by taking an enough intervals between breath holding periods. Since BOLD signal is dependent on magnetic susceptibility changes reflecting hemoglobin saturation, this examination is easily affected by intravoxel phase dispersion. In particular, air in the nasal cavity and sinuses obscures changes in BOLD signal in the basal frontal and temporal lobes. Also, since breath holding requires the cooperation of subjects, this method cannot be performed in any patient. Nonetheless, this assessment usually can be performed easily following conventional MRI, and it can be used to estimate reserve capacity in a larger area than transcranial Doppler examination. We believe that further examination will confirm the clinical usefulness of BOLD signal change in assessing cerebrovascular reserve.

References


Figures omitted at submission.