Quantitative NIRS, BOLD and FAIR in a Human Breath Hold Challenge

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Flow-sensitive Alternating Inversion Recovery (FAIR) and Blood Oxygenation Level Dependent (BOLD) images with simultaneous Near Infrared Spectroscopy (NIRS) were acquired during repeated breath holds (BH). Time series for the BOLD signal, cerebral blood flow (CBF) and quantitative concentration changes for deoxyhemoglobin ([Hb]) and total hemoglobin ([HbT]) were generated. The data show that the positive BOLD signal originates from a decrease in the susceptibility difference between the vessels and the tissue since tissue [Hb] does not change, but vascular [Hb] does. The BOLD undershoot after the BH arises from a combination of increased [Hb] and [HbT] and restored baseline CBF, consistent with the Balloon model.

INTRODUCTION

The BOLD signal is dependent on the concentration of Hb, be it through a change in blood oxygenation (Y) or a change in cerebral blood volume (CBV). Experiments in piglets have demonstrated that R_2^* is linearly dependent on the absolute [Hb] over large changes in physiology due to hypoxia (1). However, in order to understand the transient dynamics that relate [Hb] with the BOLD signal one must also have knowledge of CBV and cerebral blood flow (CBF) responses. NIRS provides a measure of CBV from [HbT], when assuming a constant hematocrit. Also, fractional changes in CBF can be obtained using a variety of MR spin-labeling techniques such as Flow-sensitive Alternating Inversion Recovery.

A breath hold is a transient hypercapnic challenge that has been well studied. It causes a global change in hemodynamics and therefore alleviates the issue of partial voluming (2) with the NIRS sampling. Transient features of the BOLD signal such as the initial dip and the post-task undershoot have been the subject of much work with a concerted effort to understand the underlying physiology behind the BOLD signal. With increased temporal resolution compared to earlier experiments of simultaneous BOLD and NIRS measurements and knowledge of CBF, HbT and Hb, we look to better understand how transient features in hemodynamics affect the BOLD signal.

METHODS

Experiments were conducted on a Varian Unity INOVA 4 Tesla whole-body system equipped with a Siemens Sonata gradient system (SR120 / 40mT/m) in two parts. In the first session, BOLD and NIRS measurements were made simultaneously on six subjects as they performed six 30 s breath holds after expiration with 60 s of rest. Four transverse 128 x 128 echo planar images were collected with four shots (TR/TE/FA = 500 ms / 10 ms / 45°) achieving identical temporal resolution as the NIRS acquisition of 2 s. In the second session, a single 64 x 64 FAIR image (TI/TR/TE = 1400 ms / 4000 ms / 3 ms) and NIRS measurements were made simultaneously on five subjects as they performed ten 32 s breath holds after expiration with 64 sec of rest. Our method for computing concentration values of near infrared absorbing hemoglobin (oxy and deoxyhemoglobin) is described in full elsewhere (3).

RESULTS

Figure 1 shows the percent signal change maps for an individual subject from part I, BOLD (left), and part II, FAIR (right), of the study. In order to draw a meaningful conclusion about the transient dynamics of BOLD (black figure 1), Hb (gray), HbT (gray dashed), and CBF (black figure 2), an average response for each of these time series data from the two part study are shown in figure 2 and 3, respectively.

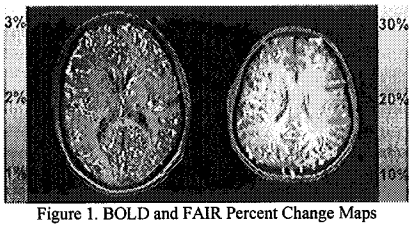

Figure 1. BOLD and FAIR Percent Change Maps

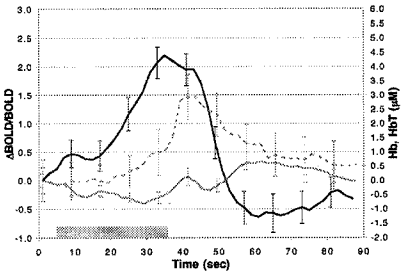

Figure 2. BOLD and NIRS

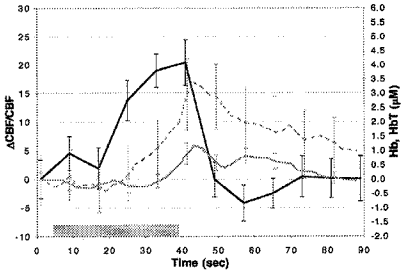

Figure 3. FAIR and NIRS

DISCUSSION

This study involved monitoring the transient global changes in hemodynamics brought on by hypercapnia during a BH task. Although the measured changes in hemodynamic parameters are not mediated by an altered state of cellular metabolism, as is the case in a functional activation study, there are parallels that we can draw about hemodynamics in general. FAIR measures CBF changes at the arterioles and capillaries, [Hb] changes originate in the capillaries and veins, while [HbT] derives from the entire blood volume. BOLD signal increases in the task despite the fact that the *tissue* concentration of [Hb] remains relatively constant. This is because [Hb] in the *vessel* decreases as the CBF and CBV increase, reducing the susceptibility difference between the vessels and the extracellular space. Towards the end of the BH, the tissue [Hb] begins to climb, causing the BOLD signal to level out. Upon cessation of the task, CBF returns to normal and the passive distension of the veins with this small increase in vessel [Hb], causes a post-task undershoot in BOLD until the venous volume normalizes (4).

REFERENCES AND ACKNOWLEDGEMENTS

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