Frequency cutoff in neurovascular coupling of resting-state fMRI

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Introduction In resting state MRI studies in humans, BOLD fluctuations typically exhibit high power only in the low frequencies (0-0.1 Hz). This is in contrast to the power distribution observed in neural recording of field potentials, which is typically inversely proportional to frequency in the mammalian cortex [1]. Non-linear neurovascular coupling is a plausible explanation, as the vasculature's property of limited frequency response may limit BOLD fluctuations to below a certain frequency. To test this hypothesis, we altered the properties of the vasculature in opposite directions by using two different anesthetics with opposite effects [2] to estimate the possible vasculature-dependence on the frequency cutoff in neurovascular coupling. The studies were conducted by simultaneous intracortical slow potential recording and fMRI in rat [3].

Materials and methods: Seven SD rats, 200-300g (male), were used in the studies. Simultaneous intracortical direct current (DC) recording at somatosensory (FLS1) and BOLD fMRI imaging were conducted under isoflurane (ISO, 1.5%) or medetomidine (MED) anesthesia [3]. The intracortical potentials (0-100 Hz) were recorded with micro glass electrodes (Ag/AgCl). The GE-EPI imaging of single coronal slice, which covered recording pipette tips, was obtained on a 9.4 T Bruker scanner with the following parameters: TR/TE, 500ms/15ms; thickness, 2mm; in-plane resolution, 300×300mm^2. Each resting-state study session lasted 8 min. MED data were obtained 2 hr after switching from ISO. The date preprocessing is described elsewhere [3, 4]. Coherences between BOLD and DC across 0-1 Hz were computed. DC/BOLD correlations were conducted within a high power frequency range or neighboring range as a control.

Results: Significant BOLD/DC correlations were found in recording sites within frequencies of 0.01-0.25 Hz under MED, and 0.01-0.1 Hz under ISO (Fig. 1.). When comparing frequency contributions to DC/BOLD coherence, the tight coupling in recording site (S1) was within the frequencies of high BOLD powers, demonstrated in Figure 1. In addition, we monitored BOLD dynamics in power spectra every 30 min over several hours when switching ISO to MED, and then MED to ISO, and observed significant frequency shift in BOLD from <0.1 Hz to 0.2 Hz during prolonged MED, and back to 0.1 Hz gradually in ISO again (not shown). These power shifts were not observed in the concurrent DC signals (not shown).

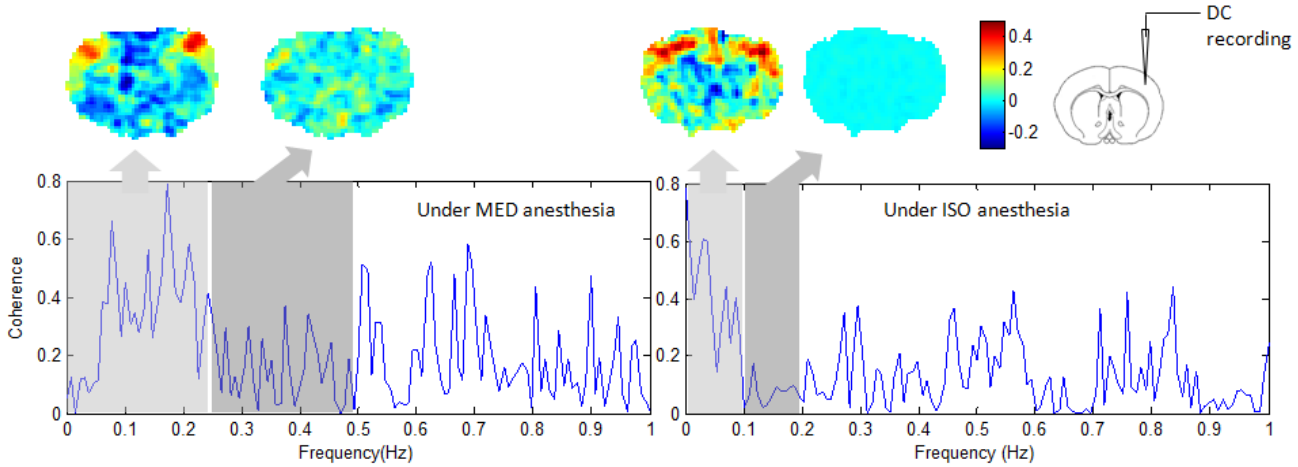

Fig.1. Frequency range of BOLD/DC coupling. The recording position is illustrated in the top right corner. The maps show DC/BOLD correlation in space with the two frequency ranges (light or dark gray, BOLD lags: 2.5s in MED, 4s in ISO).Tight DC/BOLD coherence were observed below 0.25 Hz in MED data, below 0.1 Hz in ISO data (light gray), but not the corresponding neighboring frequencies (dark gray).

Discussions: The present studies demonstrate that the relatively high amplitudes of low frequency BOLD fluctuations compared to the corresponding power distribution of neural activity is likely the result of lowpass filtering via neurovascular coupling., The range of the filtered signal can be modulated by altering the frequency response of the vasculature. ISO and MED have inverse effects on the vasculature, with ISO causing vasodilation and MED causing vasoconstriction [2]. The vessel smooth muscle under ISO may become relaxed and reduce the vessel's ability to pulsate quickly; on the contrary, the vessel wall under MED could become tighter, facilitating rapid movements. By using the nature of vascular modulations of the anesthetics, we demonstrated neurovascular coupling can be modulated by vascular frequency properties. Therefore, the BOLD signals of resting state should be sensitive to slow neural fluctuations due to limited vascular movement capability. The frequency limitation in neurovascular coupling mechanism would not reflect full-frequency information in neural activities and should be considered when interpreting resting-state fMRI data.

References: [1] Buzsaki G. et al., Science, 2004, 304:1926-1929 [2] Ohata H. et al., Anesth Anolg 89:370-377 [3] Pan W-J., et al., Brain Connectivity, 2011, 1:119-131 [4] Pan W-J., et al., ISMRM, 2010 (949), Montreal, 13: 3266-3283