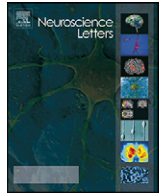
Neuroscience Letters 436 (2008) 185–188

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet



Changes in BOLD transients with visual stimuli across 1–44 Hz

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ARTICLE INFO

Article history:
Received 10 July 2007
Received in revised form 6 February 2008
Accepted 7 March 2008

Keywords:
Positive BOLD
Post-stimulus undershoot
Neurovascular coupling
Flicker
SSVEP

ABSTRACT

The dependency of positive BOLD (PBOLD) and post-stimulus undershoot (PSU) on the temporal frequency of visual stimulation was investigated using stimulation frequencies between 1 and 44 Hz. The PBOLD peak at 8 Hz in primary visual cortex was in line with previous neuroimaging studies. In addition to the 8 Hz peak, secondary peaks were observed for stimulation frequencies at 16 and 24 Hz. These additional local peaks were contrary to earlier fMRI studies which reported either a decrease or a plateau for frequencies above 8 Hz but in line with electrophysiological results obtained in animal local field potential (LFP) measurements and human steady-state visual evoked potential (SSVEP) recordings. Our results also indicate that the dependency of PSU amplitude on stimulus frequency deviates from that of PBOLD. Although their amplitudes were correlated within the 1–13 Hz range, they changed independently at stimulation frequencies between 13 and 44 Hz. The different dependency profiles of PBOLD and PSU to stimulation frequency points to different underlying neurovascular mechanisms responsible for the generation of these BOLD transients with regard to their relation to inhibitory and excitatory neuronal activity.
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The blood oxygenation level-dependent (BOLD)-based fMRI has been used to measure neuronal metabolic activity [12]. BOLD fMRI signal reflects combined effects of several physiological parameters like cerebral blood flow (CBF), cerebral blood volume (CBV) and cerebral metabolic rate of oxygen ($CMRO_2$) during the neuronal activity. Combined effects of all three physiological parameters could be observed as three transients in BOLD response as the initial dip, the positive BOLD (PBOLD) and the post-stimulus undershoot (PSU) [2,3]. Differences in the amplitude of these transients could reflect differences in the neurovascular coupling and/or differences in neuronal activity [9].

The dependency of PBOLD- and CBF-based fMRI on the temporal frequency of the stimulation has been studied before [13,17,19]. An increase in PBOLD has been reported up to 8 Hz, which was followed by either a decrease or a plateau for higher stimulation frequencies. These results were also in agreement with previous PET findings [5].

On the other hand, electrophysiological experiments have shown that flickering light leads to synchronized EEG responses, which mainly consist of oscillations with the same frequency as the flickering stimulus and its harmonics, namely steady-state visual evoked potentials (SSVEPs) [15]. The systematical analysis of these driven electrical responses to visual stimuli between 1 and 100 Hz with 1 Hz steps demonstrated that amplitude of the SSVEP selectively increased around 10 Hz, which is close to the peak observed in BOLD and PET responses [7]. However, distinct from the fMRI and PET results additional peaks were observed around 20 and 40 Hz.

So far, most of the researches on BOLD transients were studied with stimulation frequencies up to 30 Hz [13,17,19]. Furthermore, the higher ranges were sampled with fewer frequency inputs. An interesting question that still begs for an answer is how BOLD transient components including the PSU change when stimulated up to 40 Hz with a finer frequency resolution. Unlike those studies that only investigated PBOLD around 8 Hz and its adjacent frequencies, we focused on both PBOLD and PSU responses to the stimulation frequencies beyond 8 Hz. We explored additional local peaks emerging from a stimulation scheme with a finer frequency resolution. Additionally, we analyzed the correlation between the PBOLD and PSU across different stimulation frequencies to investigate the underlying physiological mechanisms.

Nine healthy subjects (mean age 28 ± 5) were recruited for this study. Written consents were obtained from all subjects. Photic stimulation was applied by using two white light-emitting diodes coupled to a fiber bundle. Fiber bundles housed on both glasses of a specially designed light-proof goggles transferred the light into the magnet room. Stimulation frequency was controlled by a digital I/O card (NI DAQCard-6062E). To keep the energy of the visual stimuli constant across frequencies, the duty cycle was 50%. Each measurement consisted of two blocks of 30 s stimulation preceded and followed by 30 s darkness. The total duration of each experiment was therefore 180 s. Experiments were performed for 24 different

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doi:10.1016/j.neulet.2008.03.021

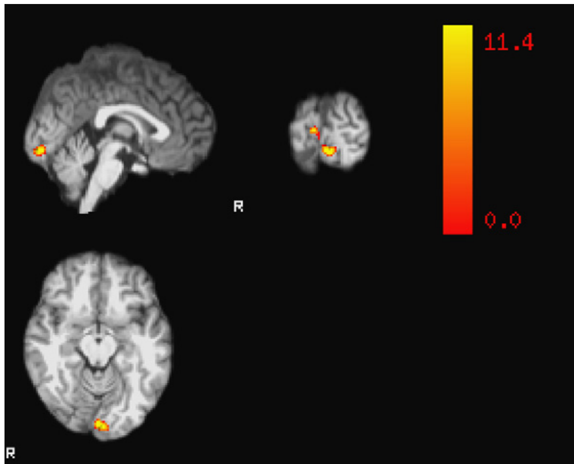

Fig. 1. Functional mask in primary visual cortex created due to visual stimulation of the subject OZ, overlaid on the structural image. A Z threshold of 3 was chosen to reflect a significance level of $p < 0.001$ of detected activation. Mask for this subject consists of 250 voxels.

frequencies from 1 to 44 Hz in randomized order. Stimulation frequencies were sampled with 1 Hz steps between 1 and 14 Hz, with 2 Hz steps between 14 and 24 Hz and with 4 Hz steps between 24 and 44 Hz. It could be considered to sample the whole stimulation frequency range with steps of 1 Hz for a finer frequency resolution [7]. However, the electrophysiology literature shows that the amplitudes of both local field potential (LFP) [14] and EEG responses [7] to steady-state stimuli at higher frequencies do not show sharp but rather broader peaks.

The experiments were approved by the Local Ethics Committee of Istanbul University, Istanbul Faculty of Medicine.

All experiments with fMRI were conducted on Siemens Symphony 1.5 T MR System at Department of Radiology, Istanbul Faculty of Medicine. A single shot T_2^* weighted gradient echo (GE) echo planar imaging (EPI) sequence was used for BOLD measurements. 20 transverse slices over a field of view of 192 mm × 192 mm with 128 × 128 resolutions, were acquired with a slice thickness of 3.5 mm. Other imaging parameters were chosen as TR 2000 ms, TE 60 ms and flip angle 90°. The high-resolution structural scans were obtained with a standard 3D MPRAGE sequence.

For each measurement of each subject, all the volumes were registered to the first volume in order to remove motion artifacts by using MCFLIRT from the FSL (FMRIB's Software Library, http://www.fmrib.ox.ac.uk/fsl) program package [8]. First level analysis was carried out using FMRI expert analysis tool (FEAT) Version 5.63, part of FSL. The following pre-statistical processes were applied: spatial smoothing using a Gaussian kernel of FWHM 5 mm; mean-based intensity normalization of all volumes by the same factor; highpass temporal filtering (Gaussian-weighted least-squares straight line fitting, with sigma = 67.5 s). Time-series statistical analysis was carried out using FILM with local autocorrelation correction [24]. This produced a fairly large region of activation within the visual cortex. A fixed-effect group analysis was performed to combine statistical parametric maps of the first level analysis for individual subjects across all stimulation frequencies. This resulted in a Z-score map of statistically significant stimulus related activity across all stimulation frequencies for each subject. Z (Gaussianised T/F) statistic map was thresholded using clusters determined by $Z > 3$ and a (corrected) cluster significance threshold of $p = 0.05$. Then, a mask that consists of a most active cluster including 200–250 voxels in the visual cortex was created for each subject, Fig. 1. For each stimulation frequency of each subject, average BOLD signal time course was computed from all activated voxels within the mask.

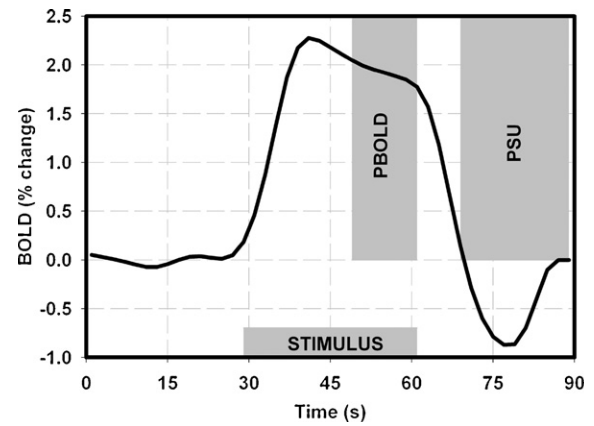

Fig. 2. The average time course of the BOLD percent change at 8 Hz frequency. The shaded areas represent stimulation, PBOLD and PSU intervals.

The percent change of the BOLD signal is defined as the absolute difference between the BOLD and the baseline (Fig. 2). The baseline signal level was defined as the average signal during the time period of 10 s before the stimulus was turned on [25]. PBOLD was quantified as the average value of the BOLD response between 20 and 30 s after the stimulus onset, and PSU was quantified as the average BOLD value between 10 and 30 s after the stimulus offset. In order to eliminate intersubject variability, an additional normalization was performed by dividing the percent change responses due to different frequencies by the maximum percent change response observed for each subject [13,17]. The grand mean values over nine subjects were obtained by averaging normalized PBOLD and PSU for each stimulation frequency and plotted against these stimulation frequencies (Fig. 3). The significance of the major peaks observed in the PBOLD response was tested using a repeated-measures ANOVA design with two within-subject factors: amplitude (two levels: peak PBOLD amplitude vs. mean PBOLD amplitude at adjacent frequencies) and frequency (peak frequencies). These adjacent frequency ranges were determined by observing the minimum values around each peak. The post hoc analyses performed using a paired t-test between the PBOLD value at each peak frequency and the mean value at its adjacent stimulation frequencies provided a regional estimation of significance for observed local peaks.

To investigate the underlying physiological mechanisms of PBOLD and PSU, the change of them across stimulation frequencies

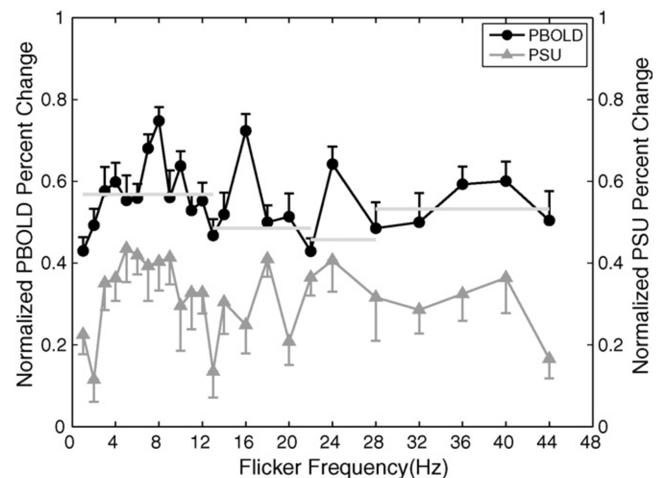

Fig. 3. Absolute mean normalized PSU (right y-axis) and PBOLD (left y-axis) change. The error bars represent the standard errors. Significance of the local peaks of PBOLD was tested against the mean of the adjacent frequencies (straight gray lines).

were compared by computing the Pearson correlation coefficients for the whole range of stimulation frequencies and for different frequency bands that show distinct patterns of correlations.

The functional map due to visual stimulation from one of the subjects, overlaid onto the anatomical image is shown in Fig. 1. This map shows statistically significant activation ($p < 0.001$) across all frequencies in the primary visual cortex, which was replicated for all subjects.

The plot of mean normalized percent change for all nine subjects as a function of stimulation frequency was illustrated in Fig. 3. The global peak of PBOLD response was at 8 Hz, which was significantly higher compared with the mean PBOLD amplitude between 1 and 44 Hz ($p < 0.03$). Secondary peaks were observed for stimulation frequencies at 16 and 24 Hz. Additionally, a broader peak was observed around 36–40 Hz. This latter one will be referred as the 40 Hz peak and is quantified as the mean of PBOLD values at 36 and 40 Hz stimulation frequencies. To test the significance of these local peaks, the amplitudes of 8, 16, 24 and 40 Hz peaks were compared with the mean PBOLD amplitudes of their adjacent values within regions defined as 1–13, 13–22, 22–28 and 28–44 Hz, respectively. The overall result of the ANOVA test showed that the peak amplitudes were significantly higher than the amplitudes at the adjacent frequencies ($F(1, 8) = 6.53$, $p < 0.05$). The post hoc pair-wise tests showed that the 8 Hz ($p < 0.02$), 16 Hz ($p < 0.05$) and the 24 Hz peaks ($p < 0.05$) were significantly higher than the mean amplitude of responses at their adjacent frequencies. The comparison of the local maximum at 36–40 Hz with the mean amplitude within the 28–44 Hz range turned out to be non-significant ($p = 0.37$).

The correlation between the PBOLD and PSU profiles throughout the whole range of the stimulation frequencies was non-significant ($r^2 = 0.391$, $p = 0.059$). Later, three specific bands of the whole spectrum were analyzed separately. These bands were determined by observing the regions in Fig. 3 that show distinct patterns of correlations: the PSU amplitudes changed in-line with the PBOLD within the 1–13 and 22–44 Hz frequency ranges, whereas they had the opposite trend between 13 and 22 Hz. The absolute amplitudes of PBOLD and PSU showed a significant positive correlation between 1 and 13 Hz range ($r^2 = 0.615$, $p < 0.03$), whereas they were correlated neither within the 13–22 Hz range ($r^2 = -0.164$, $p = 0.756$) nor within the 22–44 Hz frequency range ($r^2 = 0.387$, $p = 0.391$).

Our findings are in agreement with previous studies for the stimulation frequency range up to 16 Hz [13,17]. These studies consistently reported a peak at 8 Hz for PBOLD and that PBOLD reached a plateau or decreased monotonically at stimulation frequencies above 8 Hz. These studies usually employed stimulation frequencies up to 30 Hz by sampling the frequency range sparsely. In this study, we explored BOLD responses to 24 different stimulation frequencies ranging from 1 to 44 Hz. In addition to the peak observed at 8 Hz, our results indicate that there are statistically significant local peaks for 16 and 24 Hz, which, to the best of our knowledge, is the first observation of the local peaks beyond 8 Hz in BOLD studies. A less prominent fourth local peak around 40 Hz turned out to be non-significant.

It has been shown that PBOLD changes correlated with LFP better than with multiple unit activity (MUA) [10]. This suggests that the BOLD signal reflects the synaptic activity, which is directly related to stimulus processing. In an animal study [14], LFP and MUA recordings from neurons in Brodmann's areas 17 and 18 of the cat were analyzed for stimulation frequencies between 2 and 50 Hz. The authors reported two main peaks in LFP amplitudes at around 6–8, and 16–30 Hz with decaying amplitudes towards 50 Hz. A closer inspection of the figure presenting the frequency-dependent changes in LFP amplitudes (see Fig. 7c in ref. [14]) indicated that local peaks were also present at 16, 24, and 40 Hz. This study used 20 μs light flashes in contrast to our 50% duty cycle light stimuli, which we preferred to obtain a constant energy of visual input independent of the stimulation frequency. This allowed us to perform a comparison of responses to different stimulation frequencies that purely depend on the respective frequency but not on a change of the mean stimulus intensity. Despite such difference in the design of visual stimuli, we assume that the peak powers in the study of Rager and Singer [14] may reflect effects of stimulation frequency superimposed on the effect of increasing stimulus strength with increasing stimulation frequency. Therefore, the frequency effects on PBOLD in our study are in-line with those obtained for LFPs in Rager and Singer [14]. Also, the literature on SSVEPs in human subjects shows that the visual evoked potentials have amplitude maxima around 10 Hz and weaker peaks in the 20–30 and 35–45 Hz range [7]. Taking these findings together, the stimulation frequencies leading to local peaks in PBOLD seem to correspond to the maxima of LFPs measured in the cat visual cortex [14]. Furthermore, scalp recorded SSVEPs in humans with light stimulation for different flickering rates yielded results in the same direction [7]. This suggests that the frequency dependency of the PBOLD is related to changes in neuronal activity that shows frequency selectivity probably due to the resonance of oscillatory mechanisms tuned to certain frequencies [1,14].

From the early fMRI studies, it has been observed that the BOLD signal often has a PSU after prolonged stimulation [6]. However, there are still inconsistencies about the physiological origins of PSU, which represents an increased level of deoxy-hemoglobin compared with the baseline condition. Three hypotheses have been proposed: The first one based on the balloon model is the delayed return of CBV to baseline [3]. The second claims a slower return of the $CMRO_2$ to the baseline than the CBF [6]. The last one is based on the assumption that CBF may temporarily fall below the baseline [21].

One possibility to test the above hypotheses is to investigate the dependency of PSU and PBOLD amplitudes under various experimental conditions. The wide SSVEP literature, which revealed that rhythmic responses are driven in the EEG using stimulus trains at various frequencies, and the extensive literature on the mechanisms of the oscillatory electrical activities of neuronal circuits, suggests that stimulation at different temporal frequencies as in SSVEPs studies might modulate the PSU–PBOLD relationship in a manner, that could give hints on the PSU generation mechanisms. Indeed, our results demonstrate that the amplitude of the PSU has a dependency on stimulation frequency, which, in fact, deviates from that of PBOLD. Their amplitudes are globally correlated within the 1–13 Hz range, although the sharp peak at 8 Hz in PBOLD does not have an exact counterpart in PSU profile. Furthermore, PBOLD and PSU seem to change independently with stimulation frequencies between 13 and 44 Hz. These discrepancies between the PBOLD and PSU amplitudes across stimulation frequencies might give hints about the physiological origins of the PSU. The hypothesis claiming that the PSU is the result of the delayed return of the CBV to baseline level dictates that the PSU amplitude should be correlated with PBOLD throughout the whole range of stimulation frequencies. However, this is not the case in our data. The CBV delay mechanism alone could only explain the positive global correlation between PBOLD and PSU in the stimulation frequency range between 1 and 13 Hz. The discrepancy at 8 Hz showed that additional contribution of neurally mediated effects (hypotheses) cannot be excluded even in this frequency range. However, PBOLD and PSU were fully uncorrelated in the 13–44 Hz band, which indicates that neurally mediated mechanisms such as CBF and $CMRO_2$ considerably contribute to the PSU amplitude within this range.

The discussion of what type of neural activities might explain the different levels of CBF and $CMRO_2$ contribution to the PSU at different stimulation frequencies needs the consideration of electrophysiological results obtained with similar steady-state visual stimulation experiments. According to Rager and Singer [14], who

observed selectively increased LFP amplitudes at certain flicker frequencies, there are strongly damped oscillators along the retino-cortical path, which are tuned to different frequency bands and get recruited to variable extents at different stimulation frequencies. This view is also shared by many authors, who analyzed scalp recorded transient and SSVEPs in humans by using frequency domain analyses and time–frequency transforms [1,4,7,15]. This conjecture is supported by evidence on diverse oscillatory mechanisms that are tuned to different frequencies at the retinal (60–90 Hz), thalamic (0.1–40 Hz) and cortical levels (beta and gamma ranges).

It has been proposed that the gamma rhythm serves to build local patches of synchrony, whereas lower frequency oscillations are more robust for the establishment of long-range interactions [22]. Long-range inputs to a cortical area are exclusively excitatory [11], whereas local inputs may have a significant inhibitory component due to the inhibitory inter-neurons that act mainly within short distances. In line with these considerations, a number of studies have clearly shown that the interplay between excitatory pyramidal cells and inhibitory inter-neurons is essential for the generation of oscillatory activities within gamma and beta frequency ranges [20,23]. A conclusive result on the contribution of excitatory and inhibitory neuronal activities on PSU and PBOLD generation would need the concurrent measurement of cellular activity and the BOLD, which was not possible within the limits of this study. However, following the string of ideas in the above studies, the difference we observed for PBOLD and PSU relationship between 1–13 Hz and 13–44 Hz bands might be tentatively attributed to the different weights of excitation and inhibition in these two frequency bands. In addition to these results, a biophysical model of the coupling between neuronal activity and the balloon model [3] was proposed that allowed for evaluating the role of both excitatory and inhibitory activity [18]. Their results showed that increases in excitatory activity amplified both the PBOLD and the PSU, whereas increasing the inhibitory activity evoked decrease in the PBOLD signal without altering the PSU. These simulation results are in good agreement with our findings in the sense that a correlated change of PBOLD and PSU across the frequency range 1–13 Hz might be attributed to distant excitatory input, whereas an uncorrelated pattern across the frequency range 13–44 Hz might originate from the interplay of excitatory and inhibitory activities within that local area.

Additionally, there have been reports on a post-stimulus undershoot in both the LFP and MUA of V1 neurons below the baseline [16] resembling the PSU in BOLD responses discussed in our study, which supports our point that neuronal inhibition could contribute to PSU along with other factors.

In conclusion, the analysis of BOLD changes for a series of stimulation frequencies, which produced electrical oscillations of various strength in both intracranial LFP and superficial SSVEP recordings in previous studies, revealed that several other local maxima are present in the BOLD dynamics beyond the 8 Hz peak. Their correspondence with electrophysiological results should be further investigated by measuring the SSVEPs with the same stimuli on the same subjects. Additionally, the investigation of PBOLD–PSU relationship across a series of stimulation frequencies revealed that the delayed CBV response alone could not explain the PSU. Furthermore, we obtained hints on the underlying neurovascular mechanisms with regard to their relation to inhibitory and excitatory neuronal activity. Present results may be improved by changing the stimulus design: one can, for instance, reduce the effect of the CBV on the PSU amplitude by equalizing the PBOLD responses to the same level for different stimulation frequencies and measure the CBF in addition to the BOLD.

Acknowledgments

The authors would like to thank Dr. Kubilay Aydin for his expert assistance in MRI scanning. This study was supported by Brain Research Society (Turkey)—Abdi Ibrahim and Gen Ilac grants.

References

[1] E. Basar, T.H. Bullock, Induced Rhythms in the Brain, Springer Verlag, 1992.
[2] R.B. Buxton, K. Uludag, D.J. Dubowitz, T.T. Liu, Modeling the hemodynamic response to brain activation, NeuroImage 23 (2004) S220–S233.
[3] R.B. Buxton, E.C. Wong, L.R. Frank, Dynamics of blood flow and oxygenation changes during brain activation: the balloon model, Magn. Reson. Med. 39 (6) (1998) 855–864.
[4] T. Demiralp, J. Yordanova, V. Kolev, A. Ademoglu, M. Devrim, V.J. Samar, Time–frequency analysis of single-sweep event-related potentials by means of fast wavelet transform, Brain Lang. 66 (1) (1999) 129–145.
[5] P. Fox, M. Raichle, Stimulus rate dependence of regional cerebral blood flow in human striate cortex, demonstrated by positron emission tomography, J. Neurophysiol. 51 (1984) 1109–1120.
[6] J. Frahm, G. Kruger, K.D. Merboldt, A. Kleinschmidt, Dynamic uncoupling and recoupling of perfusion and oxidative metabolism during focal brain activation in man, Magn. Reson. Med. 35 (2) (1996) 143–148.
[7] C.S. Herrmann, Human EEG responses to 1–100 Hz flicker: resonance phenomena in visual cortex and their potential correlation to cognitive phenomena, Exp. Brain Res. 137 (3–4) (2001) 346–353.
[8] M. Jenkinson, S. Smith, A global optimisation method for robust affine registration of brain images, Med. Image Anal. 5 (2) (2001) 143–156.
[9] B. Krekelberg, G.M. Boynton, R.J.A. van Wezel, Adaptation: from single cells to BOLD signals, Trends Neurosci. 29 (5) (2006) 250–256.
[10] N.K. Logothetis, J. Pauls, M. Augath, T. Trinath, A. Oeltermann, Neurophysiological investigation of the basis of the fMRI signal, Nature 412 (6843) (2001) 150–157.
[11] P.L. Nunez, Neocortical dynamics of macroscopic-scale EEG measurements, IEEE Eng. Med. Biol. Mag. 17 (5) (1998) 110–117.
[12] S. Ogawa, D.W. Tank, R.S. Menon, J.M. Ellerman, S.-G. Kim, K. Ugurbil, Intrinsic signal changes accompanying sensory stmulation: functional brain mapping with magnetic resonance imaging, Proc. Natl. Acad. Sci. 89 (1992) 5951–5955.
[13] L.M. Parkes, P. Fries, C.M. Kerskens, D.G. Norris, Reduced BOLD response to periodic visual stimulation, Neuroimage 21 (1) (2004) 236–243.
[14] G. Rager, W. Singer, The response of cat visual cortex to flicker stimuli of variable frequency, Eur. J. Neurosci. 10 (5) (1998) 1856–1877.
[15] D. Regan, Human Brain Electrophysiology: Evoked Potentials and Evoked Magnetic Fields in Science and Medicine, Elsevier, 1989.
[16] A. Shmuel, M. Augath, A. Oeltermann, N.K. Logothetis, Negative functional MRI response correlates with decreases in neuronal activity in monkey visual area v1, Nat. Neurosci. 9 (4) (2006) 569–577.
[17] M. Singh, S. Kim, T.-S. Kim, Correlation between BOLD-fMRI and EEG signal changes in response to visual stimulus frequency in humans, Magn. Reson. Med. 49 (1) (2003) 108–114.
[18] R.C. Sotero, N.J. Trujillo-Barreto, Modelling the role of excitatory and inhibitory neuronal activity in the generation of the BOLD signal, Neuroimage 35 (1) (2007) 149–165.
[19] R. Srinivasan, E. Fornari, M.G. Knyazeva, R. Meuli, P. Maeder, fMRI responses in medial frontal cortex that depend on the temporal frequency of visual input, Exp. Brain Res. 180 (4) (2007) 677–691.
[20] R.D. Traub, M.A. Whittington, I.M. Stanford, J.G. Jefferys, A mechanism for generation of long-range synchronous fast oscillations in the cortex, Nature 383 (6601) (1996) 621–624.
[21] K. Uludag, D.J. Dubowitz, E.J. Yoder, K. Restom, T.T. Liu, R.B. Buxton, Coupling of cerebral blood flow and oxygen consumption during physiological activation and deactivation measured with fMRI, Neuroimage 23 (1) (2004) 148–155.
[22] A. von Stein, J. Sarnthein, Different frequencies for different scales of cortical integration: from local gamma to long range alpha/theta synchronization, Int. J. Psychophysiol. 38 (3) (2000) 301–313.
[23] M.A. Whittington, R.D. Traub, N. Kopell, B. Ermentrout, E.H. Buhl, Inhibition-based rhythms: experimental and mathematical observations on network dynamics, Int. J. Psychophysiol. 38 (3) (2000) 315–336.
[24] M.W. Woolrich, B.D. Ripley, M. Brady, S.M. Smith, Temporal autocorrelation in univariate linear modeling of fMRI data, Neuroimage 14 (6) (2001) 1370–1386.
[25] E. Yacoub, K. Ugurbil, N. Harel, The spatial dependence of the poststimulus undershoot as revealed by high-resolution BOLD- and CBV-weighted fMRI, J. Cereb. Blood Flow Metab. 26 (5) (2006) 634–644.