B0 changes around the head induced by the cardiac cycle at 7T

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Purpose: Our aim is to assess the magnitude of B_0 changes during the cardiac cycle without the confounding influence of tissue motion. The two major sources for physiological noise in resting state fMRI and other sensitive sequences, are respiration and the cardiac cycle. The influence of respiration is well understood, as the varying lung volume during breathing causes a varying B_0 field distortion, which can largely be corrected by real time B_0 measurement and shimming based on phase images.[1)] The influence of the cardiac cycle on brain images is much more complex as it consists of three different components: 1) field fluctuations induced by the pulsating heart and blood volume in the lungs, 2) brain tissue motion induced by pulsation of the major cerebral arteries and 3) potential BOLD effects due to fluctuation of blood flow in small arteries and veins. These effects can cause signal instabilities in various sequences. As a first step towards disentangling the different components related to the cardiac pulsation, we assessed B_0 changes during the cardiac cycle using field probes[2)], so pulsatile tissue motion could not influence the measured B_0.

Methods: For six volunteers sixteen field probes were set up around the headrest of the MRI bed in a 7T bore. For each volunteer a total of 2400 measurements at an interval of 100ms were acquired and for each time point the field strength at the location of the probes was determined. The probe positions were determined by use of the linear field gradients of the scanner. The volunteer's cardiac pulse was measured by use of a peripheral pulse unit (PPU). The cardiac peak width was estimated from the PPU signal, as $F_{width} = 2*\sigma_{Fcard}$. The mean cardiac frequency (F_{card}) was taken from the PPU directly. The amplitude of the cardiac frequency was defined as the maximum value of the spectrum in the range of F_{card} +/- $2*\sigma_{Fcard}$. (Figure 1) The same analysis was performed on the respiratory frequencies, for which the frequency range was manually selected. Per volunteer the minimum, maximum and mean amplitudes over all probes was determined. To assess signal quality, the power in the cardiac frequency was compared to that of the thermal noise and averaged over all probes and volunteers.

Results: The mean (min-max) amplitude of field strength changes found for the cardiac frequency was 1.4 (0.8-3.8) nT, and 5.7 (1.9-13.6) nT for the respiratory frequency. This resulted in a mean (min-max) ratio of 3.7 (2.5-3.6) between the respiratory and the cardiac amplitude, see Table 1. The probes with the highest amplitudes for the cardiac frequency were the most caudal and anterior probes, in front of the chin, which were closest to the heart. The amplitude dropped as probes were positioned more cranially (Figure 2). The mean ratio of power in the cardiac frequency to thermal noise over all probes and volunteers was 16.9 (range 2.7-448.9).

Discussion: The maximum amplitude of field fluctuations at the cardiac frequency measured was 7.0 nT. On average the cardiac field fluctuation amplitude was only 27% of the amplitude of field fluctuations at the respiratory frequency. These are pure B_0 changes; there was no pulsatile motion in the probes which could have confounded scanner acquired B_0 maps. For MRI sequences sensitive to phase changes or B_0 changes over time, these B_0 changes could be an important contributor to signal instability. The maximum intracranial respiratory amplitude measured by P. van Gelderen et al[1)] was approx. 75 nT (calculated from figure 4 of the referenced paper), which is the same order of magnitude as our maximal extracranial respiratory amplitude of 23 nT. This shows that our measured values are realistic. We conclude that the field fluctuations induced by the cardiac cycle can be a measurable contributor to B0 instability. This effect should be taken into account in imaging methods focusing on the information that may be present in the 'cardiac noise' in resting state fMRI data, and methods aiming at measuring brain tissue motion with phase sensitive methods, such as velocity phase contrast MRI at high field.

References: 1. *P. van Gelderen et al*, MRM 2007, Real-Time Shimming to Compensate for Respiration-Induced B_0 fluctuations. 2. *De Zanche et al*, MRM 2008, NMR Probes for Measuring Magnetic Fields and Field Dynamics in MR Systems.

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	Amplitude: mean (min-max) in nT		
V	cardiac	respiratory	resp/card
1	1.4 (0.9-3.1)	3.0 (1.4-7.7)	2.1 (1.6-2.5)
2	1.2 (0.8-2.4)	2.5 (1.0-6.0)	2.1 (1.2-2.5)
3	1.4 (0.3-5.6)	5.6 (1.0-23.3)	4.1 (3.9-4.2)
4	1.1 (0.8-1.8)	2.3 (1.0-4.6)	2.1 (1.2-2.5)
5	1.4 (1.0-3.0)	10.3 (3.9-20.6)	7.4 (4.0-6.9)
6	2.2 (1.1-7.0)	10.4 (3.4-19.6)	4.7 (3.0-2.8)
mean	1.4 (0.8-3.8)	5.7 (1.9-13.6)	3.7 (2.5-3.6)

Table 1: Max, min and mean field fluctuation amplitudes at the cardiac and respiratory frequencies and their ratio, for all volunteers (V).

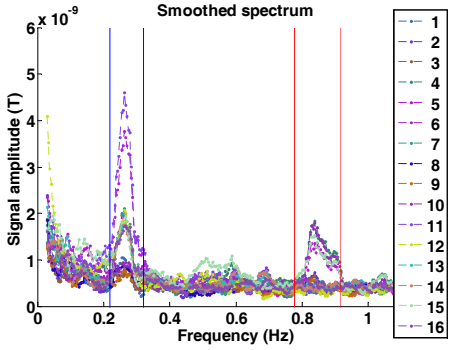

Figure 1: Spectrum of all probes for volunteer 1. The red lines show F_{card} +/- $2*\sigma_{Fcard}$ and the blue lines show the manually selected respiratory frequencies, for which the amplitudes were calculated. The spectrum was smoothed for visualisation by a moving average filter.

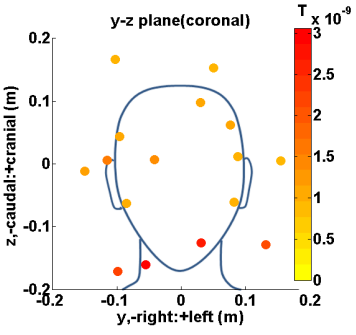

Figure 2: Coronal view of probe positions for volunteer 1, with a sketch of the approx. head location. Color coding shows amplitude of cardiac frequency in Tesla for each probe.