# Small-scale Phase and Magnitude fluctuations in fMRI time series

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## Introduction

The information conveyed by phase fMRI is limited by dominating physiological noise and scanner instabilities (1). Magneto-mechanical effects caused by slow variations in respiration volume and cardiac rhythm over time that in general have an extended spatial scale can be removed. More localized time variations in the gray matter and in the vasculature have been reported and are likely caused by vascular dephasing mediated by a BOLD susceptibility change (2). In the present study we investigated resting state dynamics of 7T phase and magnitude fMRI signals at a local scale (voxels of 98 and 64nl) at two echo times (13 and 36ms) in rats. We could confirm the presence of TE-dependent signal dephasing mediated by BOLD susceptibility in the vasculature, and found evidence of variations in dephasing over time in white matter structures in both magnitude and phase images.

## Materials and Methods

fMRI was performed at 7T (Bruker Biospec 70/30, Bruker Medical, Ettlingen Germany 5) using a BGA-9S gradient insert, a 86 mm volume transmission coil and a circularly polarized surface coil for reception. Rats were anesthesized with alfa chloralose and ventilated at 70 min-1, physiological signs (heart rate, body temperature, pCO2, pO2) were monitored during the experiment (duration ca.4 h), according to the study protocol, approved by the local ethics review board. Double echo gradient EPI was performed while the animals were at rest, TE:13 and 36 ms, TR=2sec, 1 mm axial slices covering the somatosensory cortex, 170 volumes, FOV: 3x2cm. Two acquisition protocols were used: BW=300kHz, 96x64, 98 nl voxel volume; and BW=441kHz, 128x73, 64 nl voxel volume. Venography data were acquired with a flow compensated 3D gradient echo sequence in corresponding locations with a 0.117x0.117x1mm3 voxel size, TE=15 TR=37, FA=10, 2NEX. The temporal signal-to-noise-ratio in magnitude images, tSNR was evaluated as the signal average divided by the standard deviation of the detrended time series signal. The phase stability, SDph was evaluated in terms of the standard deviation of the phase for each voxel after regression with the dork trace (3), temporal unwrapping and detrending. The phase stability relative to the 1/tSNR was evaluated. Voxels that showed local signal reduction in EPI magnitude and venography data, assumed to be dominated by the venous signal in the sagittal sinus, and an intra-cortical vein were selected for further analysis (Fig.1). Likewise time series from voxels next- to and outside the intra-cortical vein and voxels in the corpus callosum were analysed.

## Results

The tSNR value decreased as expected based on T2* decay and voxel size. The phase stability reached levels expected from the magnitude data for small voxels and short TE only, while for the remaining conditions 2-3 fold higher levels were observed. In areas with low SNR, due to the sensitivity profile of the receiving surface coil, and close to air-tissue regions even greater differences were obtained. For the high-resolution data, the TE dependent increase in 1/tSNR was greatest in the corpus callosum, followed by voxels with contributions from large veins. The phase signal showed a similar tendency although the increase in SDph outsized 1/tSNR by 15-70%, except in the corpus callosum where the SDph increase was as high as expected from the magnitude 1/tSNR (Fig 3).

## Discussion

Our data confirm that phase instabilities tend to increase with TE and voxel size faster in phase than in the magnitude data (2). For maximizing phase stability, a high spatial resolution is advantageous, and TE should be kept short to increase the available tSNR and phase stability. In areas were receiver noise dominates over physiologic noise, phase stability will inevitably decrease. Signal dephasing are greatest in voxels with strong vascular components and decrease with distance from such areas. Interestingly, both high phase and magnitude instabilities were observed in the callosal fibers, containing oriented white matter structures. In view of differences in susceptibility between these structures and the surrounding grey matter, instabilities may result from minute displacements caused for instance by heart pulsations.

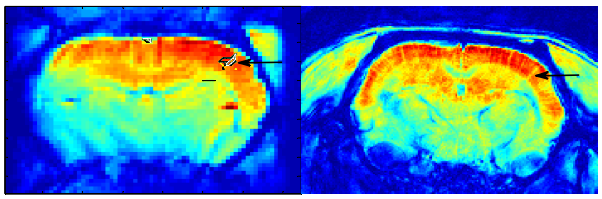

Fig 1 Average magnitude EPI and venogram. Black lines indicate voxels with venous contributions, and part of corpus callosum, the arrow an intra-cortical vein

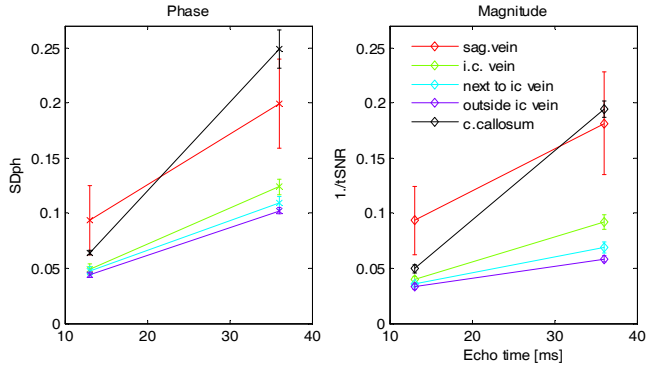


Fig 2:Phase stability and inverse magnitude stability in selected voxel sets, classified according to their contribution from large veins or callosal location

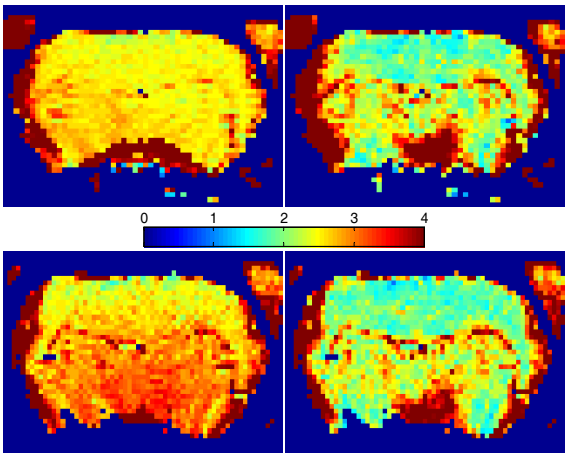


Fig 3: Illustration of the increase in instabilities at longer TE, in phase data by the ratio of SDph(36ms)/SDph(17ms) [left] and tSNR(17)/tSNR(36) [right] in 64nl (upper row) and 98nl (lower row) voxels.

**References:**

(1) Hagberg et al., MRI 26: 1026 (2008)
(2)Petridou N etal., MRI 27:1046(2009);
(3) Pfeuffer J et al., MRI 25:869 (2007)