Real-Time Observation of Spatiotemporal Dynamics of Arterial Pulsatility with MR-Encephalography

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Introduction: The concept of one-voxel-one-coil(OVOC) acquisition has been introduced as an extreme realization of parallel imaging where spatial information is predominantly encoded through the sensitive volumes of multiple small coils. In implementations as MR-encephalography(MREG) or Inverse Imaging (InI) this allows very rapid examination of functional activation (1,2). The signal time course of MREG-acquisitions shows strong pulsatility due to breathing and ECG. For fMRI studies these are treated as confounds and removed by suitable physiological noise correction algorithms (3). The purpose of this study was to explore the feasibility of MREG/acquisition to measure the spatiotemporal dynamics of ECG-pulsatility.

Methods: Experiments on 8 volunteers were performed on a 3T Tim Trio (Siemens) using a custom made 8 channel head coil array (MGH, L.L.Wald). Data acquisition was performed using a using COBRA-acquisition (TE=10 ms, FA 15 deg) with four projections at TR= 20 ms per projection or 80 ms per image for a total of 140s (4). A single transverse slice was located through the area of the calcerine fissure as in fMRI-studies. Images were reconstructed by constrained reconstruction using Tikhonov regularization as previously described (4). ECG-signals were coregistered with data acquisition in a time-locked manner. For retrospective synchronization trigger signals were identified as the maxima of the registered signals. One dataset had to be discarded because of strong gradient induced spikes in the ECG-signal. After removal of the first 20s to ensure steady state the resulting signal timecourse was high-pass filtered with a cut-off frequency of 0.5 Hz to remove low frequency signal fluctuations. Data was then resampled to the ECG-cycle using constant time- as well as constant phase resampling. In constant time resampling data are reordered starting with each ECG-trigger keeping the data acquisition constant. The duration of the resampling cycles is set to the length of the shortest ECG-cycle. This is based on the asumption that variations in the pulse period are mainly caused by variations in the endsystole whereas the systolic phase remains constant. In constant phase resampling each ECG-interval is stretched to a constant length by nonlinear interpolation.

Results: Fig.1 shows images demonstrating the propagation of the arterial pulse wave at 20 ms time intervals.Fig.2 shows signal timecourses in pixels along a line from the cortex towards the ventricles It is shown, that the signal maximum is reached first at the cortex with a subsequent inward propagation to white matter with a clear delay of the peak maximum between grey and white matter. No significant asymmetry could be discerned in the observed pulsatility in corresponding areas between the hemispheres. No significant difference was observed in the variance of the resampled signals between constant time and constant phase resampling.

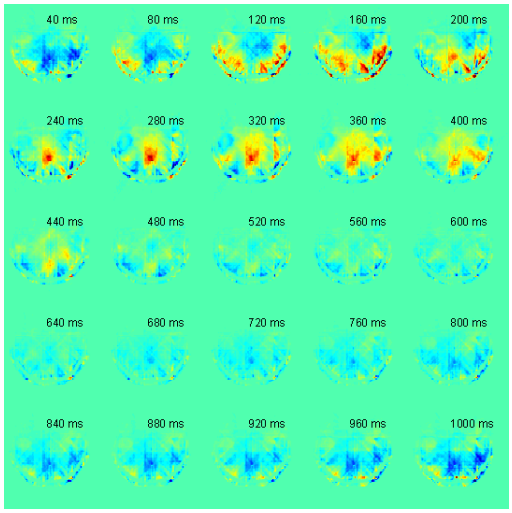

Fig. 1: Pulsatility maps along the ECG-cycle at 20 ms intervals

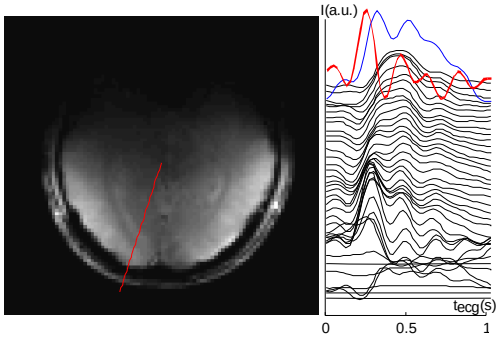

Fig. 2: Single pixel signal time courses along positions indicated by the red line. Signals from a superficial artery(red) and the sagittal sinus (blue) are shown for comparison.

Discussion: The observed apparent spread of arterial pulsatility from the cortex inwards is a consequence of a superposition of three hemodynamic events: First arterial pulsatility is observed in the cortex which is supplied through the leptomeningeal arteries. The central structures are supplied somewhat delayed. A broad hump at the center can be attributed to CSF-pulsatility and/or brain pulsation.
The lack of distinction between constant time- and constant phase average is most likely due to the small variation in pulse rate in the cohort of healthy volunteers examined in this study. It should be noted that the measured absolute time within the ECG-cycle is rather meaningless due to the somewhat artificial generation of the trigger signal at the maximum of the measured ECG-signal.
The mechanism underlying the observed signal changes is a superposition of various mechanisms as shown by the complex signal-time courses of vascular signals in Fig.2: A signal increase may be attributed to inflow effects in the current single slice experiment. Variations in vessels size especially of downstream venules and veins will increase CBV and thus spin density. Negative contributions arise from flow dependant dephasing and (potentially) from a dynamic BOLD-effect due to a shift between arterial and venous contributions over the ECG-cycle. An exact analysis of the physiological basis of the observed signal changes therefore is highly complex and requires further studies.

Conclusions: Our results indicate, that MREG can be used as a robust and reliable tool to monitor arterial pulsatility. The results shown are generated from periodic averaging over 2 min to allow for quantitative comparison of pulsatility effects. The high sensitivity of the methods allows a qualitative assessment directly on the acquired data in real-time. The method thus can be used as a vey fast and sensitive method to add information about the spatiotemporal dynamics of arterial pulsatility.
Potential areas of clinical applications are the diagnosis and assessment of territories with pathological flow dynamics in stroke patients, territorial/ hemispheric pulsatility asymmetries in patients with atherosclerotic disease and other pathologies leading to alterations in arterial pulsatility.

References:

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