

# Complex Constant Phase Activation Model Removes Venous BOLD Contribution in fMRI

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**Introduction** The measured MRI signal is the complex valued Fourier Transform (FT) of the proton spin density (PSD) of the object being imaged. In image reconstruction, generally a discrete inverse Fourier Transform (IFT) is performed to return to a representation of the PSD. Ideally, this reconstruction would result in a real valued representation of the PSD, as the true PSD is real valued. However, the IFT returns a complex valued representation of the PSD as a result of imperfections in the magnetic field. The real and imaginary data can be converted to a magnitude and phase combination through a unique mapping. However, fMRI brain activation is most often determined through the statistical analysis of time courses of magnitude images assuming Gaussian noise contributions (1). The magnitude operation of taking the modulus of the complex data is a non-linear, non-unique transformation, allowing multiple complex points to be mapped to the same magnitude values and changing the statistical distribution of noise contributions from Gaussian to Ricean. Thus, the phase portion of the data is omitted from analysis and the assumption of Gaussian noise is only valid at high SNR (2). However, studies have suggested that important information, such as the size of the vessel contributing to the BOLD signal, may be contained in the phase data (3,4). Furthermore, proper modeling of the noise is essential to optimal activation computations (5). To preserve the complete data and model the noise contribution without the approximation to the Ricean distribution, Rowe and Logan developed a method for computing activation based upon the complex data, assuming constant phase (5). The phase behavior that is observed depends upon the pulse sequence being used. It affects the measured BOLD signal, with gradient echo (GE) sequences based upon dynamic averaging of signal dephasing, much of which is caused by large veins which may not be as closely related to activation as capillaries, while spin echo (SE) pulse sequences correct for the large scale dephasing caused by larger veins, thus omitting them (6).

**Methods** A block designed bilateral finger tapping experiment of 20 seconds off followed by 8 epochs of 16 seconds on and 16 seconds off provided the data for this experiment. An axial slice through the motor cortex is examined here, with the first three time points omitted to account for magnetic field stabilization. Scanning used a Bruker Medspec 3T/60cm scanner, where 10 axial slices of 96x96 were acquired. Voxels were isotropic and 2 mm<sup>3</sup>. Both SE EPI and GE EPI pulse sequences with TE=80ms, TR=2000ms, and 138 time points were used to collect comparable sequential data sets. A navigator echo of the center line was used to correct the phase of the data to an accuracy of one pixel. The activations were computed for each slice with the traditional magnitude method and with the constant phase complex (CPC) method, both as described by Rowe and Logan (4). The chi-squared statistical maps created through the activation calculations were then thresholded using a Bonferroni correction to determine the regions of activation (7).

**Results** The activations from the SE and GE experiments using the two models are in the activation maps in Figures 1 and 2. The overlay activation maps of Figures 3 and 4 illustrate the voxels declared active in the listed cases. The CPC model and the magnitude model yield strikingly different activation maps in the case of GE images (Figure 4 [left]). However, the computed activations through both models in the SE images are found to be quite similar (Figure 4 [right]). Additionally, both SE activation maps closely relate to the CPC model of activation in the GE image (CPC SE and Magnitude GE overlay map in Figure 3 [right]).

**Discussion** The two activation models yield differing results for data acquired through a GE pulse sequence, in support of other reports (4). The CPC model finds less activation, and confines it more to the gray matter than the magnitude activation model. This suggests that the CPC activation model does omit a portion of the large vessel contribution to the bold signal in GE. With the SE pulse sequence, both the CPC and magnitude models find nearly identical activation, suggesting no major difference between the two methods with SE. This suggests that the pulse sequence's suppression of BOLD contributions from large veins results in signal from the capillary bed, already at constant phase. Thus, the CPC method which assumes constant phase and the magnitude method both result in similar activation. Additionally, the CPC method implemented on a GE image yields results which are similar to both magnitude and CPC activations in SE images, further suggesting that the CPC method removes the varying phase contributions from large veins and returns activation from the capillary bed.

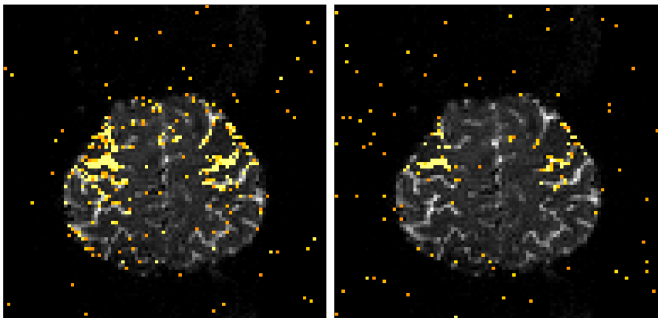


Figure 1 GE Magnitude Activation [left], GE Constant Phase (CPC) Activation [right]

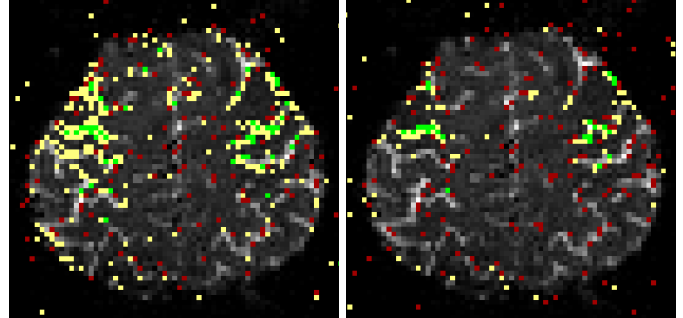


Figure 3 Magnitude Activation: SE (red), GE (yellow), Both (green) [left]  
Constant Phase Activation: SE (red), GE (yellow), Both (green) [right]

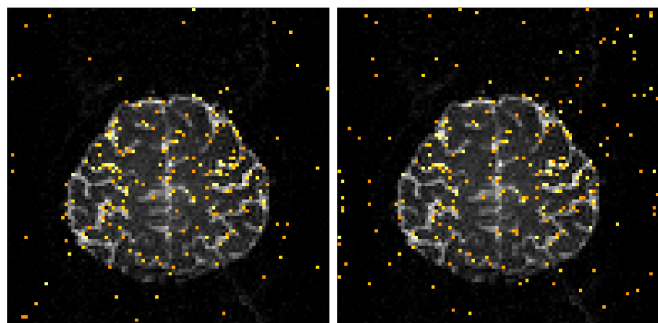


Figure 2 SE Magnitude Activation [left], SE Constant Phase Activation [right]

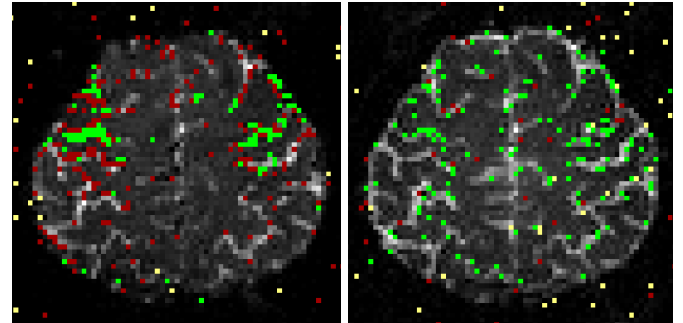


Figure 4 GE Activation: Magnitude (red), Constant phase (yellow), Both (green) [left]  
SE Activation: Magnitude (red), Constant Phase (yellow), Both (green) [right]

**References:** 1. Bandettini, et al., Magn. Reson. Med. 1993. 2. Gudbjartsson, Patz, Magn. Reson. Med., 1995. 3. Bodurka, et al., J. Magn. Reson. 1999. 4. Menon, Magn. Reson. Med. 2002. 5. Rowe, Logan, NeuroImage, 2004. 6. Kennan in *Functional MRI* 1999. 7. Logan, Rowe, NeuroImage, 2004.