S1: Validation of cardiac phase mapping code and multiband phase correction

The phase calculation outlined in the Methods of the main manuscript was tested with random and real time series data. All simulations described below were implemented in MATLAB.

Random data test

Random time series with zero mean were simulated, an example is given in Figure SF1 together with its frequency-independent power spectrum. As desired, a 2D slice of random time series (pure noise scan) results in a random phase map and a uniform phase distribution when run through the phase mapping algorithm as displayed in Figure SF2.

Multiband phase correction test

To test the multiband phase correction approach we used a pulse-oximetry cardiac signal from an example non-random time series. To simulate the case of instantaneous transmission (no phase differences) we set up an imaging volume of $90 \times 90 \times 24$ voxels with this time series. We further added a random noise fluctuation on top of each voxel’s signal for a typical SNR of 50. A TR of 320 ms was chosen to down-sample the time series to a realistic temporal resolution. To introduce a multiband acquisition slice timing we shifted the time series along the slice direction by $[0; 160; 52.5; 265; 107.5; 212.5]$ ms, which would be a realistic timing for a multiband factor of 6. Figure SF3 shows the time series at slices 1 and 4. We then ran the phase mapping code with and without phase correction on this artificial 4D dataset. Figure SF4 shows the phase map along an in-plane and slice dimension (equivalent to a sagittal or coronal view). The strikes in the phase map correspond to
Figure SF1: An example random time series (left) and the corresponding random frequency power spectrum.

Figure SF2: A phase map of randomly fluctuating voxels (left) exhibits a uniform phase histogram distribution (right).
Figure SF3: The cardiac time series from a pulse-oximetry measurement that was used for code testing. The black solid line is the original data (sampled at 1kHz) and the red line is shifted by 265 ms, to represent the signal at a different slice in the multiband acquisition order. The dotted lines are the same signals down-sampled to a representative TR of 320 ms.

the uncorrected shifts in the time domain. The phase distribution has 6 discrete phase bands. Figure SF5 shows the same dataset being phase mapped with phase correction. The phase differences are negligible and revert to the added noise (SNR) and sub-sampling noise.

As a final verification of the phase mapping code we tested if a temporal delay in the signals at different voxel locations was correctly reflected in the calculated phase difference. We added a time delay of 100 ms to every even slice and ran the phase mapping code, including the multiband phase correction. The dominant cardiac frequency at which the phase maps were calculated was \( f_c \approx 1.1 \) Hz for this particular time series. The expected phase shift between slices is then \( \Delta \phi = 2\pi f_c \Delta t \approx 0.7 \) radians as is indeed shown in the phase distribution in Figure SF6.

**S2: General linear model fitting for cardiac mask creation**

Figure SF7 shows an average whole brain spectrum with cardiac and respiratory spectra plotted on top. The physiological spectra were obtained with an external measurement (pulse-ox and respiratory belts) during the scan. Above the low frequency range the EPI spectrum almost assembles
Figure SF4: Simulated time series that is instantaneously transmitted over 24 slices and sampled with a multiband factor of 6. The phase map (left) and phase distribution (right) show the 6 phase stripes introduced by the acquisition timing. Note, that the splitting in the histogram at the second band is due to the histogram binning.

Figure SF5: The same simulated data as in Figure SF4, but with phase correction along the slice dimension. The phase distribution is only affected by signal differences that are introduced by noise and the TR sub-sampling.

Figure SF6: A time delay of 100 ms between even and odd slices was added. The pattern can be seen in the phase map and the phase distribution. The expected phase difference between signals is 0.7 radians.
Figure SF7: A representative single subject rs-fmri spectrum (red) and the simultaneously acquired respiratory (green) and cardiac (blue) spectra.

A scaled replica of the physiological spectra and suggest that the EPI spectrum could be modelled by a general linear model (GLM) with the external spectra as explanatory variables. Let $Y(f)$ be the power spectrum of a single voxel’s rs-fmri time course. The slow nature of the haemodynamic response is assumed to sub-sample any neuronally driven signal below 0.2 Hz. The power distribution above can then be modelled as a solely combination of respiratory $X_r(f)$, cardiac $X_c(f)$ and thermal noise spectra. Thermal noise is frequency independent and is modelled as a constant. A voxel-by-voxel linear regression

$$Y(f) = \alpha + \beta_r X_r(f) + \beta_c X_c(f), \quad \text{with } f > 0.2 \text{ Hz}$$

provides spatial regressor maps $\beta_r$ and $\beta_c$ that quantify how much respiration and cardiac fluctuations contribute to the signal power in each voxel. In order to compare within and across subjects all spectra are normalised, i.e.

$$\sum_{f_i=0,...,f_{\text{max}}} Y(f_i) = 1.$$  \hspace{1cm} (2)

The same holds for the cardio-respiratory spectra. Figure SF8 illustrates the model with example regressor maps. The p-values of the GLM fit can be used to create masks of significantly cardiac modulated voxels. Cardiac regressor maps $\beta_c$ simply need to be binarised if their p-value is below a desired limit.
Figure SF8: The GLM with example maps from a single subject. The EPI spectrum above 0.2 Hz is modelled by a constant noise base line, a respiratory and a cardiac regressor.