Realistic simulations on the dark rim artifact for myocardial perfusion protocols

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Background and Aims
Motion and Gibbs artifacts have been previously shown separately as probable sources of Dark Rim Artifacts (DRA) in myocardial perfusion imaging [1,2]. Their relative importance through the cardiac cycle for a range of typical perfusion protocols has not been fully examined. Therefore, the appearance of Gibbs, motion, and T1 and T2* k-space modulation were studied by numerically simulating typical perfusion protocols.

Methods
The raw-data of a short axis image of the heart was simulated in MATLAB; some data was simulated with a true perfusion defect all around the subendocardium with a third of the myocardial wall thickness. Due to the fact that the raw-data is finite and discretely sampled, all magnitude images are corrupted with Gibbs artifacts. Myocardial motion was also simulated by considering a radial motion of the endocardium during the raw-data acquisition, according to measured radial myocardial velocities by Jung et al. [3].

Three different sequences were simulated: GRE (gradient recalled echo), bSSFP (balanced steady state free precession), and hEPI (hybrid echo planar imaging) with an EPI factor of 4. The sequence protocols were (GRE/bSSFP/hEPI): TR 2.4/2.2/5.8 ms; TE 1.2/1.0/1.22 ms; Time from saturation pulse to centre of k-space 80/91/108 ms; flip angle 12/50/30 (degrees); FOV 370x370 mm, TSENSE R=2. The GRE and bSSFP used linear coverage, while the hEPI sequence k-space trajectory used is described by Ding et al [4]. The signal intensity of both the left ventricle and myocardium were taken from the first pass of contrast agent in real perfusion studies done with each sequence. Matrix sizes of 128, 160 and 192 at a range of phase-encode resolutions were simulated. All the magnitude images were zero filled to a matrix size of 2048x2048, before FFT, in order to reduce the dependency of the artifacts’ edge sub-pixel position.

Motion simulations were done for central rawdata at three different times in the heart cycle: 100/300/400 ms after R-wave. This time corresponds to central rawdata coverage for the three sequences coinciding with peak-systole/end-systole/peak-diastole respectively. T1 (30/730 ms for blood/myocardium) and T2* (15/15 ms for blood/myocardium) relaxation in the raw-data were also simulated for each sequence. Some of the simulated raw-data was filtered with a Hamming filter in order to study its effects in Gibbs artifacts and real perfusion defects.

Results and Discussion

Figure 1 shows on the left a simulated short axis image with no motion, showing a thin DRA all around the subendocardium, Figure a. After being Hamming filtered the Gibbs artifacts are nullled, Figure b. Figure c and d are identical to a and b but with an added true defect around the subendocardium. The Hamming filter blurs slightly the true defect, although, because a high resolution of 192x192 was used, the defect is still clearly visible.

On the centre it is shown a comparison for the motion artifacts simulation between a bSSFP sequence and an hEPI sequence. The results obtained for the GRE sequence (not shown) are similar to the ones obtained with the bSSFP sequence. It is shown the magnitude images for the bSSFP sequence at two different resolutions, 128x128 and 192x192, and for three different trigger times, 100/300/400 ms Figures c-j; the same results are shown, for the hEPI sequence, Figure k-p. The raw-data was not filtered; therefore Gibbs artifacts are visible in all images with the lower resolution of 128x128. As resolution increases, raw-data acquisition time increases, thus motion artifacts become more prominent. Motion blurs the endocardial border, reducing its sharpness which reduces the visibility of Gibbs artifacts, this happens mainly for trigger times 100 ms and 400 ms. For the 300 ms trigger time, the radial velocities of the endocardium are much smaller when compared to the other two trigger times making motion artifacts less prominent and Gibbs artifacts more visible. Motion artifacts also create DRAs all around the subendocardial border except along the frequency encode direction for the bSSFP sequence at 100 and 400 ms, while the hEPI sequence shows ghosting of the bright blood pool in the phase encode direction instead of a dark rim in the endocardial region for the same trigger times.

Figure q and r show hEPI magnitude images simulated with myocardial motion during diastole with the true defect at 128x128 and 192x192 matrix sizes respectively. The motion artifacts of the hEPI sequence do not render the added true defect invisible.

T1 and T2* k-space modulation introduces some blurring with a spread of the point spread function of up to 25% FWHM, but no DRA is created.

Conclusion
The faster acquisition of hEPI sequences reduces the strength of motion artifacts, while its centre-out k-space sampling trajectory distributes them rather than concentrating them near the endocardial border. Even at a matrix of 192x192, hEPI perfusion images can potentially gain from an increase of the resolution to a bigger matrix. An increase in resolution, makes possible to window the raw-data with a Hamming filter nulling Gibbs artifacts while maintaining the visibility of thin true perfusion defects.

References