

Compensation of signal loss due to cardiac motion in point-resolved spectroscopy of the heart

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Introduction: In cardiac magnetic resonance spectroscopy (MRS) signals are acquired using a combination of ECG triggering and navigator gating to compensate for cardiac and respiratory motion^{1,2}. While the effects of respiratory motion have been studied in detail³, only little work has been carried out to study the impact of cardiac bulk motion on signal-to-noise ratio (SNR) performance so far⁴.

In the current work the sensitivity of point-resolved cardiac MRS to cardiac motion is examined using a numerical motion model and in-vivo measurements. It is demonstrated that SNR is strongly dependent on sequence timing relative to cardiac motion. An optimized sequence design employing reduced FID spoiling is proposed to limit sensitivity to motion. Finally the optimized sequence is tested in-vivo to probe creatine and triglyceride concentrations in the heart.

Methods: A 3D numerical motion model (Figure 1a) based on 3D tagging data⁵ was implemented using Matlab (Mathworks, Natick, USA). A PRESS single-voxel (SV) sequence as implemented on a 1.5T clinical scanner (Philips Healthcare, Best, The Netherlands) was simulated with FID spoilers of $40\frac{mT\cdot ms}{m}$ and $60\frac{mT\cdot ms}{m}$ for the first and the second refocusing pulse (Figure 2) corresponding to a minimum dephasing of 64.2 and 80.3 radians across the voxel. In the optimized sequence, FID spoilers were reduced to $20\frac{mT\cdot ms}{m}$ and $25\frac{mT\cdot ms}{m}$ corresponding to a minimum of 26.8 and 33.4 radians dephasing across the voxel. Thereby the required dephasing of the excitation sidebands and FIDs was still maintained, which was confirmed by Bloch simulations of the employed refocusing pulse shapes.

In vivo experiments were performed using cardiac-triggered and respiratory gated PRESS in a total of 6 volunteers upon informed consent was obtained. A single voxel (SV) of $5 \times 15 \times 30 \text{mm}^3$ was selected in the septum. A series of scans w/o water suppression was obtained at different trigger delays covering the entire cardiac cycle in 2 out of 6 volunteers. Water-suppressed (WS) data were acquired at 4-9 systolic trigger delays. For WS scans, voxel size was $10 \times 20 \times 40 \text{mm}^3$ and 32 signal averages were acquired. With a repetition time of 2sec scan duration was 3.3minutes assuming 40% navigator gating efficiency. Echo times were 32 and 22ms for standard and optimized PRESS. Signal-to-noise ratios (SNR) of water (W) and triglycerides (TG) were calculated using JMRUI⁶. TG to water ratios (TG/W) are reported upon correction for T_1 and T_2 relaxation⁷.

Results: In Figure 1b simulation results of relative signal as a function of trigger delay for standard and optimized PRESS are shown. In-vivo SNR values of the water signal are presented in Figure 1c. On average, SNR of water increased by a factor of 2.7 with optimized PRESS. Figure 3a shows in-vivo SNR of TG depending on cardiac trigger delay in one subject. Using optimized PRESS SNR of TG increased from 32 ± 11 to 53 ± 18 corresponding to a gain of 1.7 ± 0.1 relative to standard PRESS. Spectra of TG acquired at 280ms and 340ms with standard and optimized PRESS are given in Figures 3b-c. Significant loss of signal is seen with standard PRESS at a trigger delay of 340ms. TG/W ratios are presented in Figure 4. The inter-scan variance was significantly lower for optimized relative to standard PRESS.

Discussion: This study has identified contractile motion of the heart as a source of signal loss in cardiac MRS. Using an optimized implementation of PRESS, significant gains in SNR of a mean factor of 1.7 and improved inter-scan reproducibility have been achieved. As multi averages are acquired in cardiac spectroscopy, the SNR gain can be translated into a 2.9 fold data acquisition acceleration with optimized PRESS compared to standard PRESS, keeping SNR levels constant. Hence, this work facilitates the integration of cardiac spectroscopy into clinical scan protocols by maximizing the achieved SNR per time unit and increasing reproducibility and reliability of myocardial triglyceride quantification.

References:

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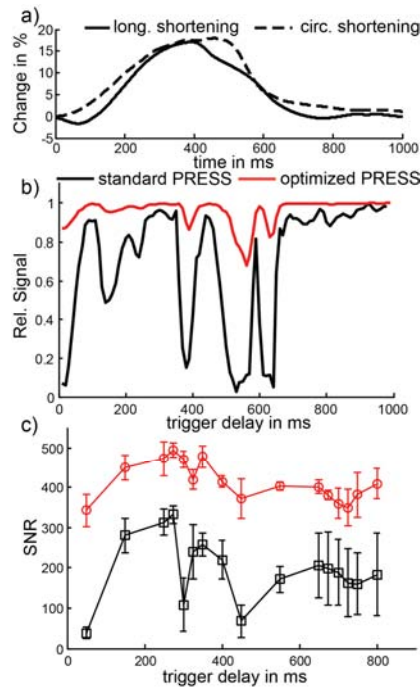


Figure 1: a) Longitudinal and mean circumferential shortening of the heart as used for the numerical simulations. b) Relative signal dependent on the cardiac trigger delay as estimated from numerical simulations. c) SNR of the water signal in a healthy volunteer. Every point represents 8 measurements. The bars indicate one standard deviation.

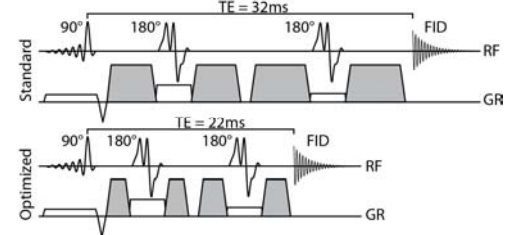


Figure 2: RF pulses and gradient design (GR) of the standard and the optimized PRESS sequence. The spoiler gradients are indicated by the gray areas.

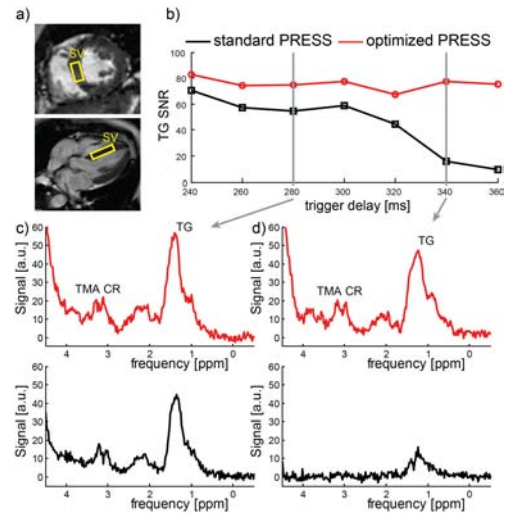


Figure 3: a) SV position inside the septal wall as used for all measurements shown in a short axis and 4 chamber view. b) TG SNR dependence on the cardiac trigger delay measured in a healthy volunteer. c) Spectra at 280ms trigger delay and d) 340ms trigger delay. The resonances of TG, creatine (CR) and trimethylammonium (TMA) are labeled in c) and d).

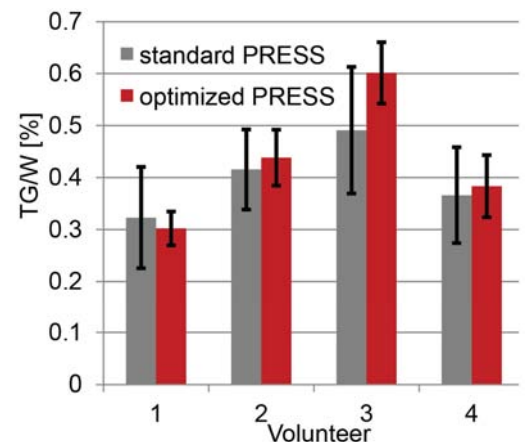


Figure 4: Estimated mean values and standard deviations of myocardial TG content for the cardiac trigger delays (N= 4 to 9). For all volunteers the standard deviation is smaller for optimized PRESS.