Simultaneous Nulling of Fat and Viable Myocardium in Delayed Enhancement Imaging - A New Approach to Fat Suppression at 1.5 and 3 Tesla Employing Multiple SPAIR Pulses

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Introduction: In contrast enhanced delayed enhancement (DE) imaging, the classic approach to suppress fat signal consists of a fat-frequency selective saturation followed by immediate readout of one segment of data. This approach is not optimal in clinical protocols which typically have a large number of lines per segment (21-29) and the common linear reordering scheme; by the time the k-space center is acquired, the fat magnetization has significantly recovered due to its short T1 (T1fat = 230 ms at 1.5T, 290 ms at 3T) and is thus poorly suppressed. Centric reordering works better, but is prone to artifacts and hence not the method of choice. Dixon-type techniques suppress fat very well, but no combination with DE has yet been described. In the turbo-spin echo sequence, fat suppression works extremely well using the SPAIR (Spectral Selection Attenuated Inversion Recovery) pulse and thus, combining SPAIR with DE imaging might appear desirable. However, due to the non-selective inversion recovery (NSIR) pulse played about 300 to 400 ms before the center of k-space to null normal myocardium in the presence of contrast agent, simply playing a SPAIR pulse about 160 ms (T1w at 1.5T) or 200 ms (T1w at 3T) before the k-space center does not work. Fat has experienced the NSIR and has not fully recovered when the SPAIR pulse is played, resulting in a shorter effective T1 to null fat. In fact, the required T1 may be so short that using a standard readout length, fat nulling is impossible. Therefore, we developed a new approach that combines NSIR for DE imaging with two SPAIR pulses to accurately null fat, without any of the above limitations.

Methods: The combined delayed enhancement and SPAIR sequence is illustrated in Figure 1. The NSIR is immediately followed by a first SPAIR pulse to selectively re-invert fat so that its magnetization is virtually untouched. A second SPAIR pulse is played at time T1w before readout (RO) of the center of k-space. Magnetization of normal myocardium and infarct are not affected by the second pulse. Fat is properly nulled at the same time that normal myocardium is approximately nulled. Dark grey myocardium and black fat are obtained simultaneously. To evaluate the quality of the new technique, we acquired three images per patient in 12 patients (6 at 1.5T and 6 at 3T) using a) no fat suppression (NONE), b) classic fat saturation (FS), and c) the new combined DE SPAIR method (SPAIR). Typical parameters for the segmented IR TurboFlash (gradient echo) sequence were at 1.5T (MAGNETOM Avanto, Siemens) and 3T (MAGNETOM Verio), respectively: T1 adjusted to null normal myocardium (300-400 ms), trigger pulse 2, fov 360 x 270 mm, matrix 256 x 125, segments 21, flip angle 25° (15°), receiver bandwidth 130 (399) Hz/pixel, TE 3.85 (1.66) ms, TR 8.9 (4.4) ms, RF spoiling on, slice thickness 7 (6) mm. We measured the signal-to-noise ratio (SNR) in 3-4 regions of fat (e.g. pericardial, abdominal), see red dashed regions of interest (ROIs) in figure 2, in each set of three images. To quantify the efficiency of FS and SPAIR in reducing fat signal, SNR of FS and SPAIR in each ROI was normalized to the SNR of NONE in the same ROI and expressed as percent. An ANOVA with Bonferroni correction was applied to test for statistical differences between the three groups NONE, FS, and SPAIR. Cavity and normal myocardium SNR were measured to evaluate if FS or SPAIR would affect SNR.

Results: Figure 2 shows a representative example of delayed enhancement images using no fat suppression (NONE), FS, and SPAIR at 1.5 Tesla. Visual inspection shows the excellent fat signal nulling of SPAIR and no significant fat suppression effect of FS. At 1.5T, statistical analysis revealed a significantly reduced relative fat SNR for SPAIR (20.5% ± 2.3%, mean ± std error of the mean, p< 0.001), whereas FS (104.1% ± 4.1%) provided no fat suppression and was similar to NONE (p>0.05). Similarly at 3T, relative fat SNR was significantly reduced for SPAIR (27.1% ± 3.3%, p<0.001) compared to NONE. At 3T FS performed much better than at 1.5T (relative SNR 45.7% ± 5.1%) and was different from NONE (p<0.001), but did not suppress fat as strongly as SPAIR (p<0.01 for FS at 3T vs SPAIR at 3T). At 1.5T, the cavity SNR was 24.2 ± 3.7 (NONE), 29.9 ± 4.8 (FS), and 20 ± 3.9 (SPAIR). Myocardial SNR was 4.6 ± 1.0 (NONE), 6.1 ± 1.4 (FS), and 4.5 ± 0.8 (SPAIR). The 3T cavity SNR was 69.3 ± 18.9 (NONE), 65.5 ± 16.2 (FS), and 61.5 ± 16.5 (SPAIR). The 3T myocardial SNR was 10.7 ± 2.2 (NONE), 9.8 ± 1.7 (FS), and 9.1 ± 2.6 (SPAIR). Neither cavity SNR nor myocardial SNR were statistically different (p>0.05) in any of the three techniques, at both field strengths.

Conclusion: The presented combined SPAIR DE approach reliably nulled the fat signal at both field strengths and for a clinically relevant number of lines per segment. SPAIR worked much better than FS at both field strengths, but the difference was particularly strong at 1.5T. This could possibly be attributed to the longer T1 at 3T and the bigger frequency difference between water and fat making fat-selective excitation more efficient. Neither FS nor SPAIR had a significant effect on SNR in cavity and myocardium. The described SPAIR DE method did not require any manual adjustment of parameters.