Contrast Optimization of Black-Blood Viability Imaging

M. Salerno[1], W. G. Rehwald[1,2], E. L. Chen[1], R. Judd[1], and R. J. Kim[1]

[1]DCMRC, Duke University Medical Center, Durham, NC, United States, [2]Siemens Medical Solutions, NC

Introduction: Delayed enhancement imaging using inversion recovery (IR) FLASH has become the clinical gold standard for myocardial viability imaging (1). This technique has high contrast between infarct and normal myocardium, but often has poor contrast between the blood-pool and sub-endocardial infarcts. We previously described a technique to simultaneously null the blood pool and normal myocardium (2). This technique (SSSR-NSIR) consists of a slice-selective saturation pulse (SSSR), followed by a time delay to allow blood to flow out of the imaging slice (TD_1), and then by a non-selective inversion (NSIR) and a second time delay (TD_2) set to the null time for the blood signal (TI_{blood}) (Fig. 1). TD_1 is chosen so that the combination of both pulses will also null myocardium. To provide increased time for blood exchange, we developed a second sequence (SSIR-NSIR) where the SSSR above is replaced by a slice-selective inversion (SSIR) which increases the TD_1 needed to null myocardium, but lowers the available magnetization of the infarct. For our implementation, the user provides the inversion times to null normal myocardium (TI_{normal}) and blood (TI_{blood}) for a standard IR sequence. From these parameters, the appropriate time delays to null blood and myocardium are calculated by the sequence. The magnetization response of these pulse sequences as a function of these inversion times has not been evaluated.

Purpose: (1) To simulate the magnetization response of these pulse sequences and determine the sensitivity of blood-infarct and normal-infarct contrast to the user defined inversion times, and to evaluate for combinations of parameters which may inadvertently null infarcted myocardium. (2) To determine the CNR between normal myocardium, infarct, and blood-pool in a chronic dog infarct model.

Methods: The magnetization evolution for normal myocardium, infarct, and blood pool, for the SSSR-NSIR and SSIR-NSIR were simulated using MATLAB for typical T_1 values which occur about 15 minutes after IV injection of 0.125 mmol/Kg Gd-DTPA (T_{1blood} = 330 ms, $T_{1normal}$ = 490, $T_{1infarct}$ = 280 ms) [4, 5]. The time delays were calculated from the following equations:

$$(1)\quad TD_2 = TI_{Blood} = T_{1blood}\ln(2), \qquad (2)\quad TD_1 = \frac{TI_{Normal}}{\ln(2)}\ln\left(\frac{M_1 - M_0}{M_2 - M_0}\right) \text{ where } M_2 = M_0\left(\exp\left(\frac{TD_2\ln(2)}{TI_{Normal}}\right) - 1\right)$$

M_1 is the magnetization after the first pulse (0 for SSSR and $-M_0$ for SSIR) and M_2 is the desired magnetization before the second inversion pulse. For the given T_1 values, the TD_1s are 443ms and 782 ms for the SSSR-NSIR and SSIR-NSIR sequences respectively and TD_2 is 229 ms for both sequences. TI_{blood} and TI_{normal} for the IR sequence are 229ms and 340ms respectively. The TI_{blood} and TI_{normal} were simulated over a range of +/- 50 ms from these "true" values. This was repeated for a variety of $T_{1\ infarct}$ values. Contrast C_{AB} was defined as the difference in the absolute values of the longitudinal magnetization $|M_{zA}|$-$|M_{zB}|$ available at the center of data collection. Segmented viability images were obtained in a dog with chronic infarct using IR-FLASH, SSSR-NSIR and SSIR-NSIR pulse sequences on a 1.5T Siemens MAGNETOM Sonata. Parameters included: FOV 300x180 mm, TH 6mm, Matrix 256x114, TE 3.85ms, FA 25°, TI_{blood} 280ms, TI_{normal} 340ms, single breath-hold.

Results: Figure 2 shows the contrast for infarct-to-blood and infarct-to-normal myocardium for the SSSR-NSIR (2.a-b) and SSIR-NSIR (2.c-d) as a function of the user-specified TI_{blood} and TI_{normal}. When the blood and normal myocardium are nulled, the infarct magnetization is 21% and 17% of M_0 for the SSSR-NSIR and SSIR-NSIR sequences respectively. For the infarct-blood plot, the contrast drops quickly as TI_{blood} is chosen shorter than the null time for both sequences (2.a,c), but falls off more shallowly for TI_{blood} chosen longer than the null time. The infarct-blood contrast is not strongly dependent on the user input of TI_{normal}. The infarct-normal contrast has a relatively flat response as a function of TI_{blood} and TI_{normal} for both sequences (2b,d) with a trend towards increased infarct-normal myocardial contrast at higher values for TI_{blood}. If the T_1 of the infarct is set to the same value as that of the blood pool, the infarct longitudinal magnetization is still 13% and 9% of M_0 for the SSSR-NSIR and SSIR-NSIR while blood and myocardium are still nulled. Figure 3 shows representative images from a dog with an infarct for the IR-FLASH (3a), SSSR-NSIR (3b) and the SSIR-NSIR (3c) pulse sequences. For the IR-FLASH pulse sequence the CNR between blood-infarct and normal-infarct are 6 and 23 respectively, for the SSSR-NSIR sequence the CNRs are 12 and 12 respectively, and for the SSIR-NSIR they are 11 and 10 respectively.

Discussion: For both sequences the optimal blood-infarct contrast occurs at the null point for blood, however, the contrast decreases at a slower rate for longer values of TI_{blood} as compared to shorter values. Thus the user should err on the side of setting TI_{blood} slightly longer than its null value. The infarct-normal contrast is also improved when the TI_{blood} is set longer. For the SSSR sequence the contrast between infarct and normal myocardium is improved for a TI_{normal} greater than its null time, but for the SSIR sequence, infarct-normal contrast is optimal when TI_{normal} is equal to the null time for normal myocardium. As the infarcted myocardium experiences both rf pulses, while the blood pool only experiences the second rf pulse, infarct will not be nulled even when the T_1 of infarct and the blood pool are similar. For the SSSR-NSIR pulse sequence there is a 23% higher theoretical magnetization for the infarct as compared to the SSIR-NSIR pulse sequence, however the CNR in the dog model is similar for both sequences. In practice, either pulse sequence is a reasonable choice depending on timing parameters related to the patient's heart rate, contrast dose, and timing after contrast without losing significant CNR.

Conclusion: A viability sequence using a slice-selective preparation pulse followed by a non-selective inversion provides a means for improving blood-infarct contrast to enhance visualization of the sub-endocardial extent of infarct. The signal of the myocardium or blood pool can be increased to enhance endocardial definition while preserving overall contrast. The magnetization response curves indicate that it is unlikely that infarcted myocardium will be inadvertently nulled by these pulse sequences.

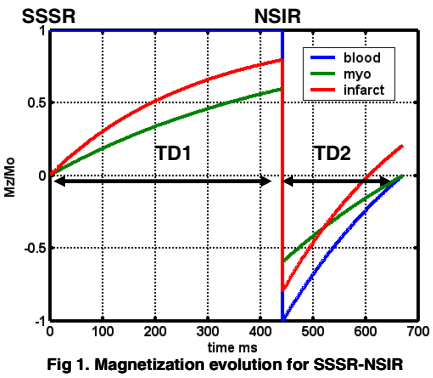

Fig 1. Magnetization evolution for SSSR-NSIR

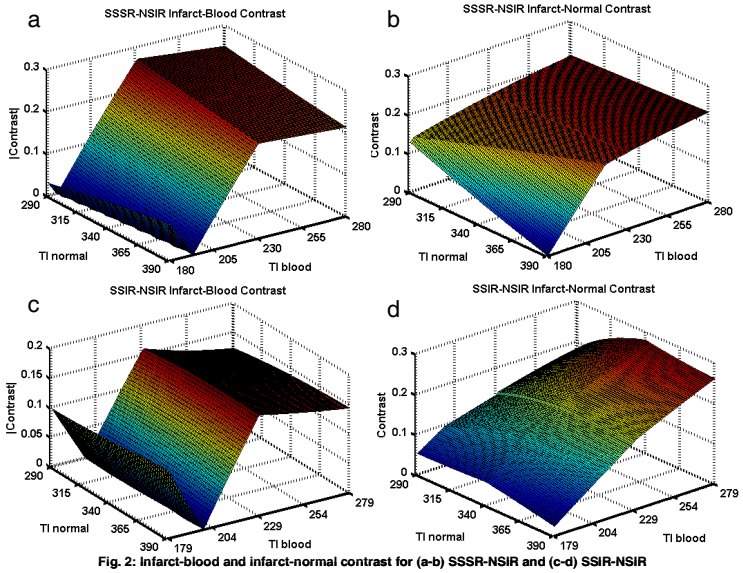

Fig. 2: Infarct-blood and infarct-normal contrast for (a-b) SSSR-NSIR and (c-d) SSIR-NSIR

References

[1] Nat Clin Pract Cardiovasc Med. 2005 Mar; 2(3):150-8.

[2] Rehwald et al. SCMR 2007. (accepted oral presentation)

[3] Radiology. 2006 Mar; 238(3):1004-12.

[4] Radiology. 2005 Sep; 236(3):1041-6.

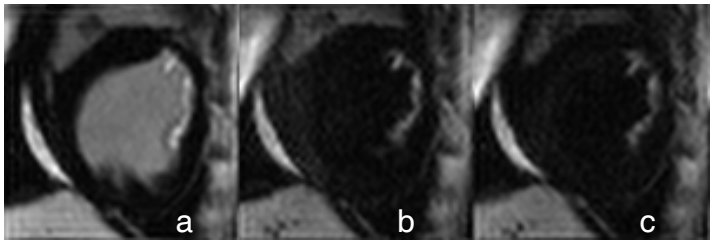

Fig.3 Viability images using (a) IR-FLASH, (b) SSSR-NSIR, and (c) SSIR-NSIR