Wideband Arrhythmia-Insensitive-Rapid (AIR) Cardiac T₁ mapping Pulse Sequence for suppressing Image Artifacts induced by ICD

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Introduction: Cardiac T₁ mapping is emerging as promising method for assessment of diffuse cardiac fibrosis in heart failure (HF) patients with non-ischemic cardiomyopathy. Despite the fact that MRI can be performed safely in most patients with cardiac devices at 1.5T [1], many HF patients who would derive benefit from MRI do not undergo MRI largely due to image artifacts arising from implantable cardioverter-defibrillator (ICD). Specifically, an ICD located 5-10 cm away from the heart can induce a center frequency shift as large as 2-6 kHz [2], which renders standard radio-frequency (RF) pulse modules for T₁ weighting to be ineffective. Recently, the feasibility of wideband late gadolinium enhanced (LGE) was demonstrated for assessment of myocardial scarring in patients with ICD [2], where "wideband" refers to the inversion pulse. The purpose of this study was to develop and evaluate a wideband arrhythmia-insensitive-rapid (AIR) cardiac T₁ mapping pulse sequence [3] for assessment of myocardial T₁ in human subjects with ICD.

Methods: (Pulse Sequence) We developed a wideband AIR T₁ mapping pulse sequence by incorporating a saturation RF pulse with wide frequency bandwidth (8.9 kHz), in order to achieve uniform T₁ weighting in the heart with the presence of ICD. Specifically, we used B₁-insensitive train to obliterate signal (BISTRO)[4] as a train of 15 hyperbolic secant adiabatic inversion RF pulses (β=750 radians/s; µ=10; individual RF duration=3.07 ms; total duration = 106 ms) with crusher gradients in between RF pulses to minimize stimulated echoes. We note that in the context of magnetization saturation, T2 relaxation during the RF time (46 ms out of 106 ms) of BISTRO is a benefit since the objective is to set the magnetization to zero. (Human Experiment) We tested the performance of original (saturation pulse frequency bandwidth=2.5 kHz) and "wideband" AIR cardiac T₁ mapping pulse sequences in 11 human volunteers at 1.5T (Siemens Espree) with and without ICD. To mimic a realistic situation [2], we taped an ICD on each subject's left shoulder approximately 5-10 cm superior to the left nipple, and performed original and wideband AIR cardiac T₁ mapping in short- and long-axis planes without administration of contrast agent. We used original AIR without ICD as the control to compare the results produced by wideband AIR. Original and wideband AIR T1 mapping acquisitions used ultra-fast gradient echo read-out and the following relevant imaging parameters: FOV = 360 x 270 mm², slice thickness = 8 mm, acquisition matrix = 128 x 96 (PE), TE = 1.1 ms, TR = 2.2 ms, receiver bandwidth = 1000 Hz/pixel, center-out k-space ordering, readout duration = 132 ms, saturation-recovery time delay (TD) = 600 ms, flip angle = 10°, breath-hold duration = 2-3 heart beats, acceleration factor (GRAPPA) = 1.6. (Image Analysis) AIR cardiac T₁ maps were generated using the Bloch equation describing T₁ relaxation in saturation recovery [3]. Left ventricular cavity and wall contours were drawn carefully to avoid partial volume averaging effects. For statistical analysis, for each measurement type (native blood and myocardial T₁) per cardiac plane, a single-factor analysis of variance was used to compare the mean T₁ values between four groups (original AIR without ICD (control), original AIR with ICD, wideband AIR without ICD, and wideband AIR with ICD), and Bonferroni

correction was used to compare the mean values between the control and other three groups.

Results: Figure 1 shows representative native T_1 maps in short-axis and long-axis planes of the heart. Compared with original AIR without ICD as the control, original AIR with ICD produced less accurate T_1 results, whereas wideband AIR with and without ICD produced more accurate T_1 results. Averaging the results over 11 human subjects, the mean myocardial and blood T_1 measurements were significantly different between the four groups (Table 1; p < 0.001). Compared with original AIR without ICD as the control, only original AIR with ICD was significantly different in both imaging planes (p < 0.05). We note that native T_1 measurements (myocardial $T_1 \sim 1100$ ms; blood $T_1 \sim 1500$ ms)

made with original AIR without ICD and wideband AIR with and without ICD are comparable to ex-vivo [5] and in vivo [6] myocardial and blood T_1 measurements reported in literature. We note that intra-

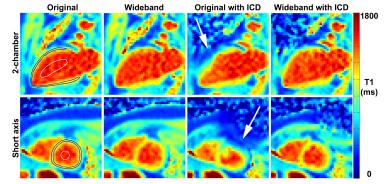


Figure 1. Representative original and wideband T_1 maps with and without ICD as shown. White arrows point to image artifacts caused by ICD.

Table 1. Mean myocardial and blood T_1 measurements over 11 human subjects. Percent error (relative to control) is reported in parenthesis. *p < 0.05 with respect to original AIR without ICD as the control.

Original (ms)	Wideband (ms)	Original with ICD (ms)	Wideband with ICD (ms)
1093.6 ± 42.1	1158.8 ± 44.5 (6.0%)	884.8 ± 84.8 (-19.1%)*	1114.9 ± 68.8 (2.0%)
1069.8 ± 29.3	1137.7 ± 28.6 (6.4%)	837.9 ± 115.8 (-21.7%)*	1091.4 ± 82.7 (2.0%)
1470.0 ± 75.3	1498.3 ± 57.6 (1.9%)	1321.6 ± 124.7 (-10.1%)*	1489.7 ± 60.8 (1.3%)
1473.9 ± 89.2	1522.5 ± 52.3 (3.3%)	1241.0 ± 268.9 (-15.8%)*	1496.6 ± 58.3 (1.5%)
	1093.6 ± 42.1 1069.8 ± 29.3 1470.0 ± 75.3	1093.6 ± 42.1 1158.8 ± 44.5 (6.0%) 1069.8 ± 29.3 1137.7 ± 28.6 (6.4%) 1470.0 ± 75.3 1498.3 ± 57.6 (1.9%)	1093.6 ± 42.1 1158.8 ± 44.5 (6.0%) 884.8 ± 84.8 (-19.1%)* 1069.8 ± 29.3 1137.7 ± 28.6 (6.4%) 837.9 ± 115.8 (-21.7%)* 1470.0 ± 75.3 1498.3 ± 57.6 (1.9%) 1321.6 ± 124.7 (-10.1%)*

cardiac leads do not generate significant artifacts (phantom data not shown due to space constraint).

Conclusion: This study demonstrates the feasibility of wideband cardiac AIR T₁ mapping for imaging human subjects without significant image artifacts induced by ICD.

References: [1] Nazarian S, et al. Heart Rhythm. 2009;6(1):138-43. [2] Rashid S, et al. Radiology. 2014;270(1):269-74. [3] Fitts M, et al. Magn Reson Med. 2013;70(5):1274-82. [4] Luo Y, et al. Magn Reson Med. 2001;45(6):1095-102. [5] Stanisz GJ et al. Magn Reson Med. 2005;54(3):507-12. [6] Chow K et al., Magn Reson Med. 2014;71(6):2082-95. **Funding:** NIH (HL116895-01A1), American Heart Association (14GRNT18350028).