SSFP in the Steady-State can Detect Myocardial Edema in Canines

J. D. Green[1,2], A. Kumar[2], J. Sykes[3], A. Mitchell[3], P. Ephrat[3], J. L. Carson[3], G. Wisenberg[3], and M. G. Friedrich[2]

[1]Siemens Medical Solutions, Calgary, AB, Canada, [2]Stephenson CMR Centre, University of Calgary, Calgary, AB, Canada, [3]Lawson Health Research Institute, University of Western Ontario, London, ON, Canada

Introduction: Determining whether myocardial edema is present or absent following a cardiac event can have a profound impact on clinical decision making, because it allows the physician to distinguish acute from chronic myocardial infarction. MRI can play an important role in detecting edema, but current techniques (such as the triple-inversion recovery prepared (STIR) T_2-weighted turbo spin echo sequence) suffer from long acquisition times and occasional imaging artifacts in patients with poor ejection fraction.

SSFP (steady-state free precession) sequences are T_2/T_1-weighted in the steady-state, and therefore should see elevated signal intensity in edematous segments of myocardium because of the long T_2/T_1 ratio of water relative to other myocardial components. However, achieving steady-state-level magnetization with SSFP in cardiac imaging can be challenging due to cardiac motion: outside of mid-diastole, spins will be moving in and out of the imaging slice. However, with a specially designed magnetization prepared sequence, it is possible to achieve equivalent T_2/T_1-weighted contrast while compensating for cardiac motion.

Purpose: To demonstrate the feasibility of using T_2/T_1-weighted SSFP imaging sequence to detect myocardial edema in dogs.

Methods: T_2/T_1-contrast was achieved by applying a train of approximately 51 radiofrequency (rf) pulses during the trigger delay period. The rf pulses were unspoiled and had a repetition time of 8 ms and a flip angle of 70°, which has been shown previously to achieve T_2-weighted contrast at 1.5 T. The rf train was preceeded by an $\alpha/2$ pulse of o reduce off-resonance effects and succeeded by a $\alpha/2$ pulse and a gradient spoiler to "store" the magnetization on the longitudinal plane. All pulses in the magnetization preparation had a excitation thickness of 7 cm, so that effectively the entire heart was affected by the magnetization preparation. Following preparation, a train of 8 rf pulses with a linearly increasing flip angle were played out prior to data acquisition to minimize signal oscillations. This was followed by a conventional segmented SSFP acquisition scheme in mid-diastole when cardiac motion is minimal. The linearly-increasing flip angle dummy pulses and acquisition all excited a thin slice (10 mm). Additional sequence parameters were: TR/TE/flip angle = 3.2 ms/1.6 ms/70°; Field-of-view = 178 x 300 mm^2 (phase x readout); matrix = 152 x 256; Bandwidth = 975.0 Hz/Pixel; 21 lines acquired per cardiac cycle.

This study was carried out in five healthy mongrel dogs and was approved by the local animal care and use committee. We chose an acute infarct animal model which is well known to produce myocardial edema (acute ischemia followed by reperfusion). All scanning was done on a 1.5 T Siemens Avanto scanner (Siemens Medical Solutions, Erlangen, Germany). Each dog (all female, weight 19-22kg) underwent a thoracotomy and transient occlusion of the left anterior descending (LAD) coronary artery distal to the first diagonal branch. After 3-4 hours of ischemia time, the occlusion was released and the dogs were allowed to recover. After 3 days, a MRI study was performed. T2/T1-weigthed SSFP images were acquired in a short-axis orientation across the entire heart. Contrast agent (0.1 mmol/kg Gd-DTPA) was then injected, and after approximately ten minutes, a late enhancement (LE) sequence was applied using the same slices and slice thickness.

Data was analyzed using a clinically validated software package (cmr[42], Circle International Ltd., Calgary, Canada) using a modified 17-segment model. In each slice, a region of interest was drawn in myocardial territory (adjacent to the septum) which should not be affected by the occlusion in the LAD. All pixels which had a signal intensity two standard deviations greater than the signal intensity of this remote area were considered positive for edema. If a group of 7 or more contiguous pixels were present in a myocardial segment, that segment was considered to be positive under the SSFP sequence. A similar method was used to determine if a segment was positive for late enhancement.

Results: A typical results is shown in the Figure. A total of 162 segments were analyzed over the ten dogs. Out of the 98 segments that were negative for LE, 53 were also negative for T_2-enhancement. Of the 64 segments that were rated positive LE, 44 were also rated positive for T_2-enhancement.

Discussion: Because the T_1 of scar only increases as the infarct ages, any signal increase can only be due to a larger increase in T_2 values. Many of the myocardial segments which were positive under SSFP but negative under LE were in segments adjacent to those that were positive for LE, so these could represent an edematous area at risk which had not undergone infarction yet. Areas which were rated negative using SSFP and positive under LE are possibly part of the zone of no-reflow, which is commonly encountered in this type of animal model (3-4 hours of ischemic injury followed by reperfusion).

Conclusion: These results show thatit is feasible to use T_2/T_1-weighted SSFP for detecting T_2 changes consistent with myocardial edema in acute infarcts. Further studies need to be performed to compare the efficacy of the SSFP sequence to the conventional T_2-weighted STIR sequence.

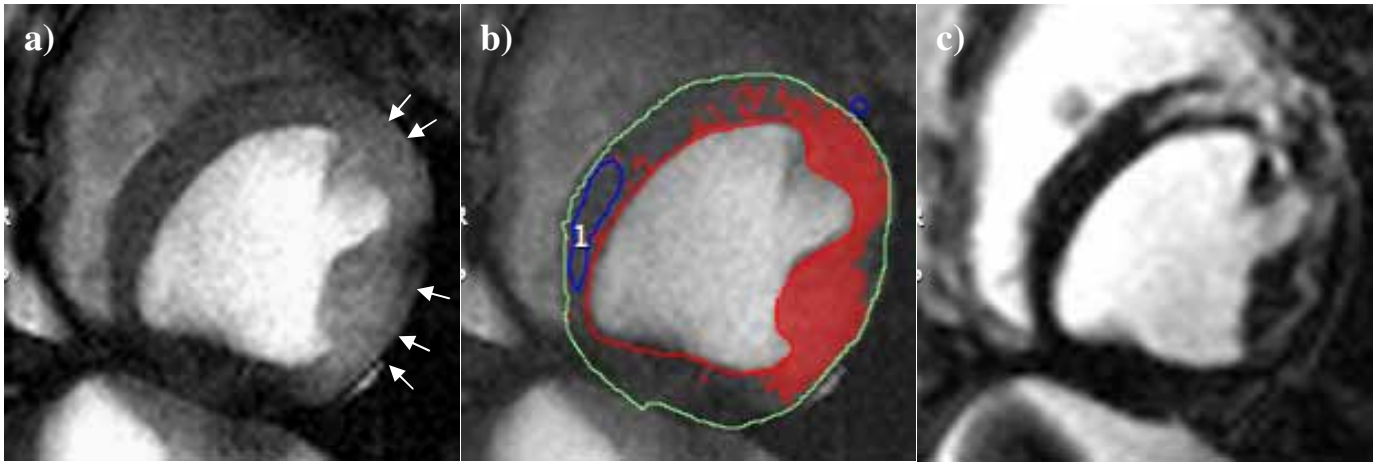

Figure: a) Short axis T2/T1 weighted SSFP image. Note bright areas in the lateral wall (arrows), corresponding to areas of increases in T2. **b)** The same image as a), but with pixels with signal intensity two SD greater than the remote area (curve "1") highlighted. **c)** Late enhancement image in the same slice position. Large areas of contrast uptake are present in the lateral wall.