Detection of Recent Myocardial Infarction using CINE SSFP Imaging

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Introduction: T_2 weighted imaging has been used to detect acute myocardial infarction (MI)[1] and is reported to accurately define the myocardial area at risk[2]. Image quality with this technique often suffers from long breathhold times, image artifacts and poor contrast between myocardium and the left ventricular bloodpool. Edema lengthens both the T_1 and T_2 myocardial relaxation times. We investigated the use of a CINE SSFP (steady-state free precession) technique with T2/T1 contrast for the detection of myocardial edema resulting from recent MI.

Materials and Methods: Sixteen subjects participated in this study. Eight subjects had experienced an MI within two months of CMR imaging (MI age, 0.05 years±0.03; range, 0.03-0.13) and eight subjects had no history of cardiovascular disease. Imaging was performed using a 1.5T clinical scanner (Siemens Magnetom Sonata, Erlangen, Germany). Subjects underwent volumetric short and long axis CINE imaging using a steady-state free precession (SSFP) CINE technique (TR/TE=3.1/1.6 ms; FA= 65; BW= 1085 Hz/pixel; Matrix= 127x256; Resolution = 2.0x1.3 mm^2; Slice thickness = 8 mm). This was followed by contrast injection (0.2 mmol/kg gadodiamide) and delayed hyperenhancement (DHE) infarct imaging in patients with recent MIs. Left ventricular (LV) epicardial and endocardial contours were drawn on DHE and CINE short axis images. Myocardial segments with MI were identified on DHE images using the full width half maximum (FWHM) criterion. The signal intensities from SSFP-CINE images were compared between infarcted and viable segments. Contours were drawn for all cardiac phases allowing analysis of signal intensities changes as a function of cardiac phase.

Results: In patients with MIs within the last two months, areas of myocardial edema in regions with MI shown by DHE imaging were well depicted using precontrast SSFP CINE imaging. An example is given in Figure 1. In normal subjects, we detected a significant variation in image signal intensity over the cardiac cycle of the myocardium and LV bloodpool (Figure 2). The myocardial peak percent signal difference was 42.0%±9.9% (Range: 20.5 - 50.4). There were two distinct peaks, one occurring at peak systole and the other at the end of rapid ejection. The LV bloodpool peak percent signal difference was 18.5% ± 2.9% (Range: 13.0 - 21.1). The LV bloodpool signal decreased slowly through systole and rose sharply during rapid filling. These characteristic curves for the LV bloodpool and myocardium were consistently seen in the normal subjects.

In patients with MIs, segments with DHE had greater signal intensity on SFFP CINE images (Figure 1). Signal intensity percent difference between infarcted and viable segments was 17.5%±19 (Range 2.8-54%) The phase with the greatest percent difference having the best contrast was at the end-of systole 252ms±76 (Range= 122-359).

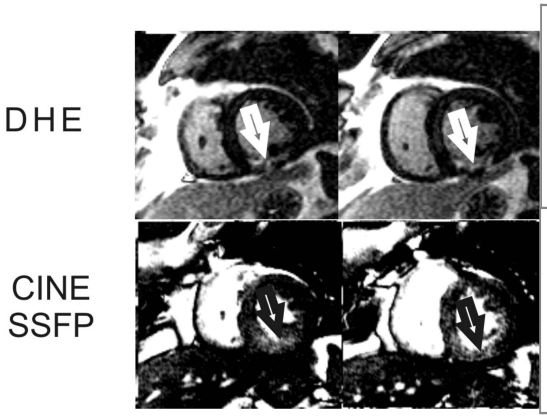

Figure1
Precontrast CINE SSFP images show a hyperintense region in the same inferior segment as delayed hyperrenhanced images show myocardial infarction.

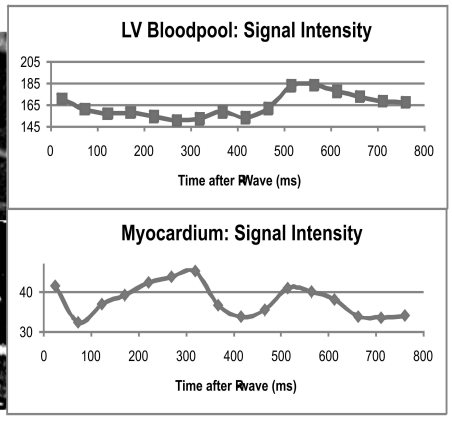

Figure 2

Graph shows the significant changes in LV bloodpool and myocardial signal intensity over the cardiac cycle in a normal volunteer.

Conclusions: As a result of recent MI, myocardial edema causes a signal increase on CINE SSFP images. In CINE SSFP images, the LV bloodpool and myocardial signal intensities vary normally with cardiac phase and myocardial edema can be best detected at the end of systole when the contrast is the greatest with adjacent myocardium.

[1]Abdel-Aty H, Zagrosek A, Schulz-Menger J, Taylor et al. Circulation. 2004;109:2411-6.
[2]Aletras AH, Tilak GS, Natanzon A, et al. Circulation. 2006;113:1865-70.