

Comparison of Cardiac Stress MR Perfusion Imaging With and Without Non-Rigid Motion Correction

Pamela K. Woodard¹, Matthew R. Lyons², Sven Zuehlsdorff³, Gary McNeal⁴, Agus Priatna⁴, Cylen Javidan-Nejad⁵, Ibrahim M. Saeed⁶, Hui Xue⁷, Christopher Glielmi⁴, and Robert J. Gropler⁵

¹Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri, United States, ²Cardiology, Case Western School of Medicine, Cleveland, Ohio, United States, ³Siemens Medical Solutions, Erlangen, Germany, ⁴Siemens Medical Systems, Malvern, PA, United States, ⁵Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, MO, United States, ⁶Cardiology, Saint Lukes Hospital, Kansas City, MO, United States, ⁷Siemens Corporate Research, Princeton, New Jersey, United States

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Objective: To compare the visibility of perfusion defects on stress myocardial perfusion imaging performed with and without non-rigid motion correction (MOCO).

Background: A framework for non-rigid multimodal registration has been created and applied to compensate for distortions between EPI images and anatomical MRI volumes [1]. Initially applied in brain MR imaging, this technique has now been applied to a cardiac perfusion sequence to be used with pharmacologic stress imaging. The implemented algorithm uses a key frame of the dynamic data set during the first pass of contrast agent as the reference frame. Subsequently, all other frames are registered using a nonrigid registration algorithm [2].

Method: 22 patients (15 M, average age 60 yrs, SD 9.6) with a reversible myocardial perfusion defect on SPECT-MPI underwent a cardiac perfusion MRI within 7 days of the SPECT-MPI. The first-pass stress cardiac MR perfusion examination was acquired on a 1.5T MAGNETOM Avanto (Siemens Healthcare, Erlangen, Germany) using a six element body phased array coil and a six to nine element spine matrix coil. MR exams consisted of short and long axis cine steady state free precession (SSFP) imaging, SR prepared tfl cardiac first pass perfusion (TI=100ms, TE=1.05ms, TR = 2.2ms, 650 Hz/px, TPAT 2, 160 base resolution) with matched slice positions and delayed contrast-enhanced (DCE) T1 GRE imaging. First-pass perfusion images were obtained 30 seconds after regadenoson 400 micrograms administered in a single IV bolus and during power injection of 0.075 mmol/Kg of gadobenate dimeglumine at 5 mL/sec IV followed by normal saline flush. Perfusion images were reconstructed without and with MOCO. DCE imaging was obtained 10 minutes after injection of an additional 0.025 mmol/Kg of contrast agent. In separate sessions a physician with cardiac MRI expertise (16 years) graded the images for the presence or absence of a perfusion defect in each myocardial segment using the American Heart Association 17-segment model[3]. Correlation for hypoperfusion at the base, mid and apex of the left ventricle was assessed using a one-sided McNemar's test.

Results: In total, there were 39 defects identified during stress perfusion imaging with MOCO and 28 on the non-MOCO stress perfusion imaging, with more hypoperfusion identified on MOCO imaging in comparison to non-MOCO. Differences between MOCO and non-MOCO imaging were significant only for defects in the mid left ventricle (LV) (P = .03), with base (P = .06) approaching significance. Differences at the apex were less significant (P=.25) (table).

Discussion: More defects were visible on MOCO cardiac MR perfusion imaging than non-MOCO. Differences were significant for defects present in the mid LV, and approached significance at the LV base. Of note, although SPECT-MPI imaging was performed, this is not a true reference standard, in that as many as 5-10% of standard SPECT-MPI imaging may have attenuation correction artifacts attributed to perfusion defect. Anecdotally, perfusion defects identified on non-MOCO imaging appeared smaller in comparison to their MOCO counterparts and often covered fewer portions of adjacent myocardial segments.

Conclusion: More myocardial MR perfusion defects are identified on MOCO MR perfusion imaging in comparison to non-MOCO imaging.

TABLE: Correlation of myocardial perfusion defects on MOCO and non-MOCO imaging.

	MOCO	Non-MOCO	P value
LV Base	11	7	.06
LV Mid	15	10	.03
LV Apex	13	11	.25
TOTAL	39	28	

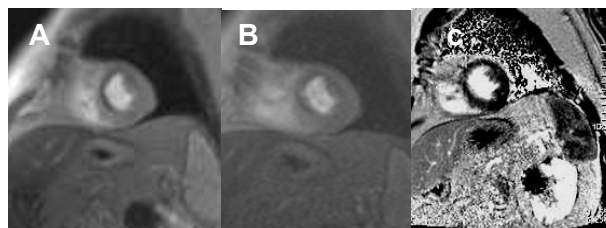


FIGURE: Anteroseptal hypoperfusion, apical LV in (A) motion-corrected (MOCO) and (B) non-MOCO stress perfusion imaging. Corresponding delayed enhanced image (PSIR) shows no infarction, indicating that this region is ischemia (C).

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