

Fusion of T₁-Mapping and Semi Quantitative Perfusion (T₁Per-Fusion) Imaging Provides Additional Insight into Myocardial Tissue Viability

Matthias Alexander Dieringer^{1,2}, Marcel Prothmann², Florian von Knobelsdorff-Brenkenhoff^{1,2}, Andreas Greiser³, Christina Eichhorn⁴, Thoralf Niendorf^{1,5}, and Jeanette Schulz-Menger^{1,2}

¹Berlin Ultrahigh Field Facility (B.U.F.F.), Max-Delbrueck Center for Molecular Medicine, Berlin, Germany, ²Working Group on Cardiovascular Magnetic Resonance, Experimental and Clinical Research Center (ECRC), Berlin, Germany, ³Siemens Healthcare, Erlangen, Germany, ⁴Max-Delbrueck Center for Molecular Medicine, Berlin, Germany, ⁵Experimental and Clinical Research Center (ECRC), a cooperation of the Charité Medical Faculty and the Max-Delbrueck Center for Molecular Medicine, Berlin, Germany

Target audience: This work targets basic researcher, clinical scientists, and clinicians interested in myocardial tissue characterization using T₁-mapping and perfusion imaging.

Purpose: Cardiovascular MR (CMR) offers the unique capability for non-invasive characterization and differentiation of myocardial tissue. Native tissue contrasts as well as contrast media enhanced imaging allow the discrimination of healthy from diseased tissue. Visual - but subjective - evaluation of T₁-weighted and contrast enhanced myocardial perfusion imaging has arrived in the daily clinical routine of CMR. Viability imaging including detection of myocardial infarction is usually based on late Gadolinium enhanced (LGE) imaging. The current gold standard of LGE quantification is eye-ball assessment; different (semi-)quantitative post processing methods are in discussion. To deliver more precise information about pathogenesis and classification of cardiac disorders, quantitative MR imaging is a promising means to characterize stage and progress of complex pathophysiological processes. Notwithstanding its utility, myocardial T₁ mapping and semi-quantitative perfusion imaging have seen some inertia when moved into the clinical scenario. One reason for this hesitation is that inter-subject variance of single quantitative parameters renders delineation of healthy and diseased tissue challenging if not elusive. A combination of complementary quantitative tissue information together with subject-specific data evaluation might refine tissue characterization and improve diagnostic quality. Therefore, we propose a method that combines quantitative T₁ and semi quantitative perfusion imaging to support tissue characterization and to enable intra-individual assessment of impact and progress of cardiac diseases on myocardial tissue viability.

Materials and Methods: A myocardial perfusion sequence and a myocardial T₁-mapping sequence were adjusted, so that spatial resolution, slice position, field of view, and data acquisition window were identical for both sequences. Semi-quantitative rest perfusion (signal upslope) was assessed using a fast gradient echo based saturation recovery perfusion sequence (TE/TR=0.93/2.0ms, Flip angle=17°, GRAPPA R=2, voxel size= voxel size=(2.8x2.1x8)mm³, scan time=49s) together with the application of contrast agent (Gadovist, Bayer, Leverkusen, Germany) dose of 0.2 mmol/kg body weight, given in one bolus. An inversion recovery prepared SSFP sequence [1] (MOLLI, TE/TR=1.02ms/2.4ms, Flip angle=35°, 5-3-3 acquisition pattern, GRAPPA R=2, voxel size=(2.8x2.1x8)mm³, scan time=12s) was applied post contrast to allow pixelwise T₁-mapping. T₁ map and perfusion images were co-registered using an in-house implementation in Matlab (TheMathworks, Natick, MA). A region of interest (ROI) was placed on the ventricle in the perfusion image contouring endo- and epicardial borders. The ROI was transferred to the T₁-map and perfusion signal upslope and T₁ values for each pixel were extracted. Correct registration was confirmed in seven volunteer datasets acquired on a 1.5T whole body MRI (MAGNETOM Avanto, Siemens Healthcare, Erlangen, Germany) equipped with a 12 channel receiver cardiac coil array. Mid-ventricular short axis views were acquired from two patients with chronic myocardial infarction on a 3T whole body MRI (MAGNETOM Verio, Siemens Healthcare, Erlangen, Germany) equipped with a 32ch receiver cardiac coil array (Invivo, Gainesville, FL, USA). K-means cluster analysis was conducted for normalized T₁ values versus normalized perfusion signal upslope in Matlab. Cluster plots consisting of one, two, three and four clusters were generated, where the four clusters represented four categories of myocardial tissue: healthy myocardium ("healthy"), scarred myocardium ("scar"), rest-perfusion defect ("PD"), and scarred myocardium exhibiting rest-perfusion defect ("Scar+PD"). The respective cluster plot was selected visually by plausibility.

Results: Figure 1 shows T₁-map (a), corresponding LGE (b), rest-perfusion upslope (c), and k-means cluster analysis (d) of a patient with chronic myocardial infarction (CMI) in the lateral wall after 14 weeks of acute event. Best data separation was found for three regions by the clustering algorithm, represented by tissue suffering from rest-perfusion deficit only (16.5 %), scar tissue with impaired rest-perfusion (37.5 %), and healthy tissue (46.0 %).

Figure 2 shows a patient with CMI in the inferior wall 13 weeks after acute event. Cluster analysis yielded three tissue types featuring healthy tissue (29.5 %), tissue without scar but with perfusion defect (36.3 %), and tissue with scar accompanied by perfusion defect (34.2 %).

Discussion: The presented results offer the possibility to quantify different pathophysiological processes. T₁ mapping represents conventional LGE. Beyond localization of myocardial injury, combination of quantitative discrimination provides additional tissue viability information and potentially enables follow-up during progress of disease. While this study tested only the feasibility of the proposed method in one mid-ventricular slice, imaging has to be extended to a 3D whole heart coverage approach.

Conclusions: In this study we demonstrated a method to combine information of T₁ mapping and perfusion imaging. The k-means clustering algorithm provided stable results also for iterations with different starting points. We anticipate extending this approach to stress perfusion (including mean perfusion reserve) and fully quantitative perfusion measurements.

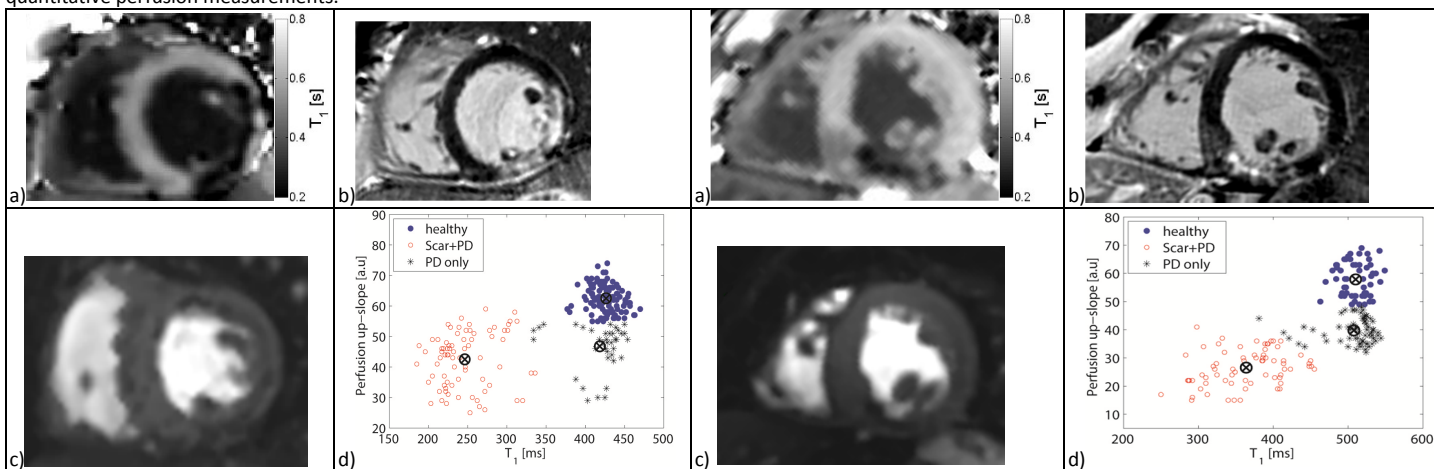


Figure 1: T₁-map (a), corresponding LGE image (b), perfusion (up-slope) measurement (c), and pixelwise cluster evaluation of perfusion vs. T₁ using k-means clustering algorithm (d) of a CMI patient. This patient was classified with areas of rest-perfusion deficit only (16.5 %), scar tissue with impaired rest-perfusion (37.5 %), and healthy tissue (46.0 %).

Figure 2: T₁-map (a), corresponding LGE image (b), perfusion (up-slope) measurement (c), and pixelwise cluster evaluation of perfusion vs. T₁ using k-means clustering algorithm (d) of another CMI patient. This patient was classified with areas of healthy tissue (29.5 %), tissue without scar but with perfusion defect (36.3 %), and tissue with scar accompanied by perfusion defect (34.2 %).

References: [1] Messroghli et al., Magn Reson Med, 2004