Quantitative 3D Myocardial Perfusion Imaging at High Dose with Accurate Arterial Input Function Assessment

Lukas Wissmann¹, Markus Niemann^{1,2}, Robert Manka^{1,2}, and Sebastian Kozerke^{1,3}

¹Institute for Biomedical Engineering, University and ETH Zurich, Zurich, Switzerland, ²Department of Cardiology, University Hospital Zurich, Zurich, Switzerland, ³Division of Imaging Sciences & Biomedical Engineering, King's College London, London, United Kingdom

Purpose: Concurrent qualitative diagnosis and myocardial blood flow (MBF) quantification from first-pass myocardial perfusion images suffers from an inherent compromise. While high contrast agent dose increases myocardial contrast-to-noise ratio (CNR) and thus improves the detection of ischemic regions (1), a low dose image is preferred for MBF quantification to maintain the approximate linearity between signal intensity and concentration in the arterial input function (AIF) (2). In general, however, high CNR and MBF quantification is desirable. To this end, integrated dual-image approaches have been proposed (1,3,4), yielding perfusion information in up to four 2D slices.

In this study, interlacing of a 2D AIF navigator with highly accelerated 3D whole-heart myocardial perfusion imaging is presented. It is demonstrated that this method allows accurate AIF assessment and MBF calculation, while maintaining the CNR benefits of high dose imaging.

Methods: <u>In-vivo acquisitions</u>: An interleaved saturation recovery gradient echo sequence was set up on a Philips Achieva 1.5T system (Philips Healthcare, Best, The Netherlands). The sequence consists of a WET saturation pulse (5), followed by a 2D and a high-resolution 3D scan triggered to end-systole (Fig. 1). Acquisitions were performed using a 5-channel cardiac receive array. 2D AIF scan parameters were: spatial resolution = 3.21x3.21 mm², slice thickness = 10 mm, flip angle=15°, TR=2.3 ms, TE=1.06 ms, 2.5x SENSE acceleration, time from saturation to k-space centre (saturation delay) = 26 ms, acquisition window = 84 ms. 3D perfusion image parameters



Figure 1. Interleaved 2D-3D sequence and schematic of ECG signal used to trigger the 3D perfusion scan to end systole.

were: spatial resolution= 2.05x2.05x10 mm³, field-of-view: 360x360x80 mm³, flip angle=15°, TR=2.0 ms, TE=0.8 ms, 10-fold *k-t* PCA undersampling (6), saturation delay = 180 ms, acquisition window = 255 ms. 30 time frames were acquired during breath-hold with a temporal resolution of 1 heartbeat. 6 healthy volunteers were measured according to local ethics regulations. Contrast-enhanced imaging was performed after administering a bolus of Gadovist (Bayer Schering Pharma, Germany) at 0.025 mmol/kg, followed by a high dose scan at 0.1 mmol/kg 20 minutes later.

<u>Phantom experiments</u>: Phantoms containing saline and variable amounts of contrast agent were produced to optimize the sequence timing w.r.t. linearity between signal intensity and concentration. Concentrations were varied from 0 to 5 mmol/l, which corresponds to peak left-ventricular concentration with a T_1 of 30 ms (1).

<u>Post-processing</u>: After SENSE reconstruction of the 2D images and *k-t* PCA reconstruction (7) of the 3D images, segmentation was performed to extract the signal intensity curves from the left ventricle (LV) in the 2D and 3D images (AIFs), and the myocardium in the 3D scans. The 2D AIFs were rescaled to the 3D AIFs by means of the first and last five time frames with low contrast agent concentration in the LV. After conversion of the signal intensity to contrast agent concentration by baseline subtraction, MBF quantification was performed in 6 angular sectors over all segmented slices, using Fermi deconvolution (8). CNR was compared in myocardial regions for low and high dose.

Results: Fig. 2 illustrates the phantom signal intensities vs. concentration using the sequence shown in Fig. 1. While the 2D signal increases linearly with concentration, the 3D signal strongly deviates from linear behaviour. Fig. 3 shows example 2D images and apical, mid-ventricular and basal slices of the 3D high dose scan from one volunteer at three different time points. The 2D AIF image was only used for AIF extraction, while the high-resolution 3D scan provided myocardial residue curves. Fig. 4 shows mean AIFs from low and high dose scans of 6 volunteers. While the 3D AIF at low dose is only mildly saturated, strong underestimation of the 3D AIF due to nonlinearity is observed at high dose. Fig. 5 shows MBF bull's eye plots using the 3D scan for saline phantoms with various



low and high dose 2D AIFs and 3D myccardial residue curves from one volunteer. MBF mean and standard deviation over all sectors and slices were 0.90±0.48 ml/g/min and 0.92±0.26 ml/g/min for low and high dose, respectively. Myocardial high concentrations. Non-linear behaviour is observed for the 3D image at high concentrations.

dose and 6.0 ± 4.7 for low dose acquisitions.

Discussion: The feasibility of quantitative high-dose 3D myocardial perfusion imaging with accurate AIF assessment has been demonstrated in this study. A similar approach to whole-heart high dose imaging using a pencilbeam probe has been presented previously (9). The sequence at hand, however, is less sensitive to inter-scan and intra-scan motion, because it acquires a whole 2D image for AIF assessment. Further evaluation in patients using stress and rest perfusion acquisition is necessary to confirm the presented results.



DCE-MRI time frames Figure 3. 2D and 3 slices of the 3D image at peak enhancement in the right ventricle, left ventricle and myo-cardium.





Figure 4. 2D and 3D signal intensities in the left ventricular blood pool for low dose (left) and high dose (right). 20 minutes were allowed for contrast washout in-between the low-dose and high-dose scans.

Figure 5. Myocardial blood flow [ml/g/min] over 8 slices and 6 angular sectors in 1 volunteer using 2D AIFs at low and high contrast agent dose.

References: 1. Gatehouse, JMRI 2004;20:39–45. 2. Jerosch-Herold, JCMR 2010;12:57. 3. Kim, JMRI 2006;23:81–86. 4. Breton, JMRI 2011;34:676–684. 5. Ogg, JMR B 1994;104:1–10. 6. Vitanis, MRM 2011;65:575–587. 7. Pedersen, MRM 2009;62:706–716. 8. Jerosch-Herold, Med Phys 1998;25:73–84. 9. Wissmann, Proc. ISMRM 2012;20:89.