

POSTER PRESENTATION

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A protocol for the measurement of myocardial blood volume and water exchange

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Objective

Absolute myocardial perfusion MR imaging (ml/min/100g tissue) has the potential to timely diagnose and reduce patient mortality from coronary artery disease.

Background

Organ perfusion can be quantified by direct calibration of relative perfusion images using absolute blood volume (ml/100g) [1]. It is well known that for an intravascular gadolinium-based T1 shortening contrast agent, the parenchymal T1 change reflects tissue blood volume [2]. However, to accurately quantify blood flow from blood volume, we must describe the compartmentalization effects of intra- to extra-vascular water exchange [2,3].

Materials and methods

Protocol

In an instrumented dog we measured T1 using a cardiac gated Modified Look Locker Inversion Recovery (MOLLI)(4) pulse sequence (slice thickness 8 mm, FOV 171 x 343 mm2, matrix 96 x192, TR 173 ms, effective TI 100 ms). Images were acquired on a 1.5 T Espree scanner (Siemens Medical Systems, Erlangen, Germany), during a short breath-hold, 5 minutes after injections of 0.003 mmol/kg of MS-325 (Ablavar, Lantheus Medical Imaging, Billerica, MA).

Image processing

We estimated the myocardium and left ventricle blood pool T1 through fitting of MOLLI signal to the regrowth curves of the Look-Locker equation by an automatic image processing program developed in MATLAB R2009a (Mathworks, Natick, MA, USA). Myocardial blood volume (MBV) was calculated from the baseline to post-contrast change in T1 in the blood pool and myocardium.

Results

Low dose injections (1/10th of single dose for humans) of MS-325 effected significant changes in myocardial T1's (Table 1). The measured MBV was 40% of total myocardial volume, or 28 ml/100g, a value that overestimates those quoted in the literature [5]. Water exchange in the myocardium was shown to approach the slow or no-exchange limit (Figure 1).

Conclusions

We have established an imaging protocol to measure MBV and water exchange. Over-estimation of MBV may be caused by extravasation of MS-325, and to a lesser extent by T2 bias on the T1 measurements with the steady-state free precession MOLLI sequence. Future steps include measuring MBV with a strictly intravascular USPIO contrast agent, application of a more sophisticated fit that includes T2 effects, and determination of the water exchange constant by Monte-Carlo simulations.

Table 1 Blood pool and myocardium T1

	Baseline	0.003 mmol/ kg	0.009 mmol/ kg	0.015 mmol/ kg	0.021 mmol/ kg	0.027 mmol/ kg
T1 in left ventricle blood pool (ms)	1259±63	1018 ±33	761 ±15	645 ±19	580 ±16	548 ±33
T1 in myocardium (ms)	871±88	818 ±76	727 ±52	676 ±49	634 ±69	624 ±42

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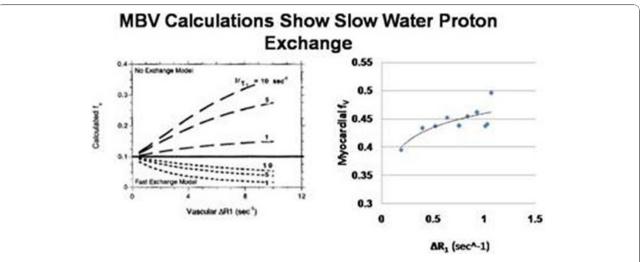


Figure 1 Vascular fractions for predictions based on "No exchange" and fast exchange limits for a range of exchange values (left). Preliminary results from our experiments suggest the "No exchange" limit is appropriate for the quantification of myocardial blood volume (right).

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