Quantification of Myocardial Oxygen Consumption Rate: Initial Experience in Humans

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Purpose
Myocardial oxygen supply and demand has to match to maintain normal myocardial contractility. Myocardial oxygen consumption (MVO₂), which determines the total myocardial oxygen demand, may provide accurate assessments of this balance in the heart. Recent studies in animals have shown the promise for the non-invasive quantification of MVO₂ by cardiac MR (CMR) techniques [1]. The purpose of this study is to assess the ability of our newly developed CMR methods to quantify regional myocardial MVO₂ at rest and during pharmacologically-induced hyperemia in normal volunteers.

Methods
Five volunteers without documented coronary artery disease were recruited (4M, age = 35 ± 4 old). All participants underwent CMR study at rest and then during adenosine vasodilation. This study was performed with a 1.5T Siemens Sonata system. Adenosine was infused intravenously for 6 minutes at a constant rate of 0.14 mg/kg/min by using a MR compatible infusion system (Medrad Continuum, Indianola, PA). CMR methods include a dark-blood (DB) turbo-spin-echo (TSE) sequence and a bright-blood (BB) T₂-prep-gradient-echo (T₂-prep-GE) sequence for acquiring T₂-weighted images. Myocardial perfusion was measured using a turboFLASH sequence to collect 80-100 dynamic images. A bolus injection of 0.02 mmol/kg Multihance (Bracco Diagnostic, Princeton, NJ) was started 5 sec after the start of the perfusion measurement. T₂-weighted imaging and dynamic perfusion imaging were performed at rest and during the adenosine injection. The volunteers were instructed to hold their breath during each imaging session. Table 1 lists the imaging parameters for each sequence.

Global myocardial oxygen extraction fraction (OEF) at rest was determined in the coronary sinus using the T₂-prep-GE sequence. The quantification of OEF was different from a previously reported method [2]. A predefined relationship between OEF-blood T₂ was used to calculate OEF in the coronary sinus. Regional myocardial OEF during adenosine vasodilation was calculated by a two-compartment model with hyperemic myocardial T₂ (DB-TSE) or with hyperemic T₂-weighted signals (BB-T₂-prep-GE) [3]. Myocardial blood flow (MBF) and blood volume (MBV), both at rest and during pharmaceutical stress, were determined using a newly developed model-independent algorithm [4]. Global MVO₂ was calculated by drawing an ROI on the MBF and OEF maps and using Fick’s law: MVO₂ ≈ OEF x MBF. Figure 1 shows one set of images from one volunteer.

Results
MBF, MBV, and MVO₂ results can be seen in Table 2. As expected, injection of adenosine increased MBF 165% and MBV 41%. Myocardial OEF was reduced 102% and MVO₂ increased 29% with a concomitant increase in the rate-pressure product. MBF is linearly correlated with MVO₂ at rest (R² = 0.98) (Figure 2), but adenosine infusion disrupts this correlation (R² = 0.33), indicating a mismatch of myocardial perfusion and oxygen demand. No significant correlation was observed between MBV and MVO₂, at rest or during adenosine vasodilation. In comparison, the BB-T₂-prep-GE method yielded a hyperemic OEF of 0.42 ± 0.09 and a hyperemic MVO₂ of 8.6 ± 2.8 µmol/g/min (no significant differences vs. the DB-TSE method). The sensitivity for 100% MBF increase with adenosine vasodilation is approximately 3.2% using the BOLD TSE sequence and 16% using the T₂-prep sequence.

Conclusions
Our CMR methods may non-invasively quantify myocardial perfusion and MVO₂. The BB-T₂-prep-GE method shows much higher sensitivity to the changes in MBF.

References