Measurements of Myocardial Perfusion and Metabolism with MR: Validation Study with PET in a Canine Model

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Purpose

Historically, assessments of myocardial perfusion have relied heavily on measurements of myocardial blood flow (MBF). However, because only ~50% of capillaries are functional at rest [1], the recruitment of capillaries, or changes in myocardial blood volume (MBV), may also play a significant role in supplying O₂ to the myocardium [2]. Our MRI methods can quantify MBF, MBV, oxygen extraction fraction (OEF), and estimate oxygen consumption (MVO₂) with Fick’s law: 

\[ \text{MVO}_2 \propto \text{OEF} \times \text{MBF} \]

In this ongoing study, we are imaging beagles with MRI and PET to determine the accuracy of MRI methods.

Methods

So far, eight beagle dogs were used in this ongoing study, divided into 4 groups (Table). Stenosis was created by using an MR-compatible, adjustable clamp in the proximal left anterior descending coronary artery (LAD). MR imaging was performed on a 1.5T Sonata scanner (Siemens Medical Solutions, Erlanger, Germany). MR first-pass perfusion scans using a turboFLASH sequence were performed at rest and during hyperemia. 0.015 mmol/kg Gadomer (Bayer Schering Pharma AG, Berlin, Germany), an intravascular contrast agent, was injected as a bolus. A validated perfusion quantification method designed in our lab [3], was applied to obtain MBF and MBV maps (Figure 1). OEF during hyperemia was determined by a two compartment model with myocardial T₂ that was measured with a 2-D segmented black blood turbo spin-echo (TSE) sequence [4]. Rest OEF was assumed to be 0.6, which is based on values measured in normal dogs using AV blood sampling at rest [5]. PET imaging was performed on a Focus 220 MicroPET scanner (Concorde Medical Systems, Knoxville, TN). MBF was determined with \(^{15}\)O-water (avg. 7.59 mCi), and MVO₂ was measured with \(^{11}\)C-acetate (avg. 8.39 mCi). OEF was then determined with Fick’s law. Data from both LAD-perfused anterior and left circumflex (LCx)-perfused inferior myocardial beds was determined.

Results

As expected, dipyridamole and dobutamine both produced large increases in MBF (47.4 ± 16.4% and 55.6 ± 16.7%, respectively) in normal myocardial regions. Both agents also produced significant increases in MBV (17.9 ± 11.4% for dipyridamole, 32.8 ± 12.8% for dobutamine), as well as in MVO₂ (15.7 ± 9.9% for dipyridamole and 43.6 ± 18.4% for dobutamine) in normal myocardium. The considerably larger increases with dobutamine are due to the combination of inotropic and chronotropic stimulation.

Thus far, our PET results show good agreement with our MR measurements (MBF \(R^2 = 0.796\), OEF \(R^2 = 0.629\), MVO₂ \(R^2 = 0.664\)) (Figure 2). Once more subjects are completed; these correlations are expected to become more significant.

Conclusions

The “gold standard” PET measurements appear to validate our MRI results for the measurements of myocardial perfusion and oxygenation.

References


![Figure 1. MR MBF (A), MBV (B), and OEF (C) maps of a dog with a 95% LAD stenosis during dipyridamole. (D) is the PET \(^{11}\)C-acetate (MVO₂) image for this dog.]

![Figure 2. Correlation plots between MRI and PET MBF (A), OEF (B), and MVO₂ (C). OEF data is stress only due to the assumed MR rest data.]

<table>
<thead>
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<th>Group</th>
<th>Stenosis</th>
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<th>n</th>
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<tr>
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<tr>
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<td>Dipyridamole</td>
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Table. Dog Groups