MR Quantification of Regional Myocardial Oxygen Consumption Rate

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Purpose

Myocardial oxygen supply and demand has to match to maintain normal myocardial contractility. Myocardial oxygen consumption (MVO_2), which determines the total myocardial oxygen demand, may provide accurate assessments of this balance in the heart. The purpose of this study is to test the ability of a cardiac MR BOLD method to determine changes in myocardial MVO_2 during pharmacologically-induced hyperemia in both normal and stenotic dogs.

Methods

31 dogs were divided into six groups as seen in the Table. Stenosis was created by an MR-compatible occluder on the proximal left-anterior descending coronary artery (LAD) and stenosis severity was confirmed via Doppler flow reduction. MVO_2 was calculated by the Fick principle: $MVO_2 \propto OEF \times MBF$.

Table. Dog groups.

Group (*n*)	Stenosis (Area)	Hyperemia
1 (14)	normal	Dipyridamole
2 (4)	70%	Dipyridamole
3 (3)	90%	Dipyridamole
4 (4)	normal	Dobutamine
5 (3)	50%	Dobutamine
6 (3)	70-90%	Dobutamine

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 [1]. The study was performed in a 1.5 T Siemens Sonata system. The imaging sequence was repeated several times at rest and during either Dipyridamole-induced vasodilation or Dobutamine-induced hyperemia. Rest OEF was assumed to be 0.6, which is based on values measured in normal dogs using an arterial and coronary sinus blood sampling approach at rest [2]. MBF values, both at rest and during pharmaceutical stress, were determined with a quantitative first-pass perfusion MR method. First-pass images were denoised and MBF maps were created with an algorithm that was developed and validated in our laboratory [3]. MVO_2 values were determined in the stenotic LAD perfused anterior region and the remote left-circumflex coronary artery (LCx) perfused inferior region.

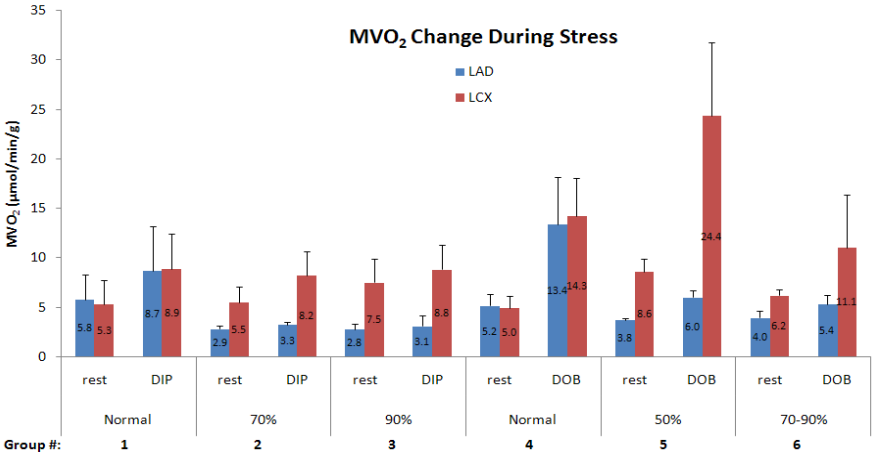

Figure 1. Changes in MVO_2 during Dipyridamole or Dobutamine with various LAD stenosis. (DIP: Dipyridamole, DOB: Dobutamine).

Results

MVO_2 results can be seen in Figure 1. As expected, in normal dogs, Dobutamine induced a dramatic increase (186%) in MVO_2 (group 4), while injection of Dipyridamole shows only a mild to moderate effect (62%) (group 1). In the anterior region with LAD stenosis, after the injection of Dipyridamole, a small increase in MVO_2 was observed at 13.8% and 10.7% for the 70% and 90% stenosis groups, respectively, whereas the remote normal LCx region shows 49.1% and 17.3% increases in MVO_2. This is different from conventional wisdom that Dipyridamole would induce no changes in MVO_2, but is consistent with a report using adenosine injection in dogs [4]. With Dobutamine, MVO_2 in the anterior regions increased significantly at 57.9% and 35% for the 50% and 70-90% stenosis groups, respectively, whereas there were 183.7% and 79% increases in the remote inferior LCx regions accordingly. It is interesting to note that severe single-vessel stenosis not only attenuated the increase in MVO_2 in the stenotic perfused region with Dobutamine, but also attenuated MVO_2 in the remote normal myocardial region, a similar finding in patients with a single LAD stenosis [5]. Figure 2 shows the relationship of the ratio of MBF/MVO_2 in the LAD and remote LCx regions, within Dobutamine groups. There is a marked difference in the normal dogs and stenotic dogs, which again agrees well with a report in patients [5].

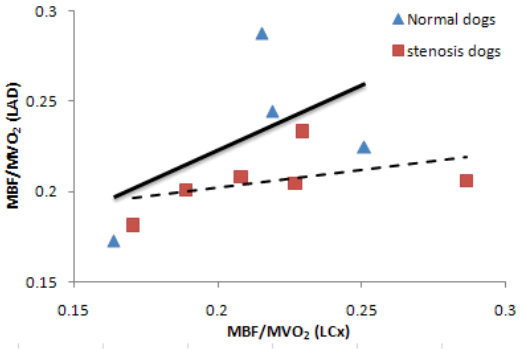

Figure 2. Correlation between MBF/MVO_2 ratio for the LAD region and LCX region in normal and stenotic dogs.

Conclusions

Our cardiac MR methods may non-invasively quantify regional differences in myocardial MVO_2. Evaluation of MVO_2 changes in our coronary artery stenotic dogs reveals similar findings as in patients.

References

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