Regional OEF Determination with the BOLD Effect in Normal and Stenotic Dogs

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Purpose

Recently, we have shown that a cardiac MRI method can non-invasively determine the global and regional myocardial oxygen extraction fraction (OEF) in canine models during hyperemia [1,2,3], with assumed myocardial blood volume (MBV) values. Recently, we have developed fast mapping techniques for MBV mapping with a first-pass perfusion approach [4]. The purpose of this study is to determine the changes in regional myocardial OEF with the complementary MBV data during pharmacologically induced hyperemia, in both normal and coronary artery stenotic dogs.

Methods

There were 31 dogs used in 6 groups (Table). Coronary artery stenosis was introduced by using an MRI-compatible occluder in the proximal left anterior descending coronary artery (LAD) with an open-chest model. Single-slice T2-weighted images were gathered by use of a 2-D multi-contrast segmented turbo spin-echo (TSE) sequence with double inversion recovery preparation pulses. T2 maps can be generated with these images with three different TEs (8ms, 36ms, 58 ms). This T2-imaging was performed several times at rest and during either Dipyridamole or Dobutamine-induced hyperemia in all dogs. Using a two-compartment model validated in normal dogs [1], the OEF during hyperemia can be determined assuming a resting OEF of 0.6, which is based on OEF values measured in normal dogs using an arterial and coronary sinus blood sampling approach at rest [1]. It is assumed that this value changes little with moderate stenosis [3,5], but for very severe stenoses (> 95%), this may be a potential source of error. The two-compartment model also requires the input of rest and hyperemic MBV values. Instead of assuming the MBV, as in previous studies [1-3], these values were determined with a validated first-pass perfusion method developed in our laboratory [4,5]. This technique utilized a blood pool agent, Gadomer (Bayer Schering Pharma AG, Berlin) as a tracer, injected at rest and during hyperemia. Myocardial OEF values were determined in the stenotic LAD perfused region and a remote normal left-circumflex coronary artery (LCX) subtended region.

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

Results

The changes in OEF during hyperemia are shown in the Figure. In normal dogs, Dipyridamole-induced vasodilation increased myocardial blood flow (MBF) with minimal changes in myocardial oxygen consumption (MVO_2), resulting in a dramatic decrease in myocardial OEF in both LAD and LCX territories (from 0.6 to 0.35). In stenotic dogs during vasodilation, the normal LCX regions decreased a similar amount, while the stenotic LAD regions remained near resting OEF values (11% decrease and 0% change in dogs with 70% and 90% stenosis, respectively); due to restricted oxygen supply (limited MBF). During Dobutamine-induced hyperemia, myocardial OEF was preserved in normal dogs, because increased MBF (supply) met the demand of the increased MVO_2. Minor elevations were seen in the 50% LAD stenotic regions due to restricted MBF and increased oxygen consumption (demand). However, in 70-90% stenotic dogs, OEF values on both LAD and LCX regions were slightly reduced. Reviewing source T2-weighed images found moderate flow artifacts in the anterior (LAD) myocardial region, leading to false OEF reduction.

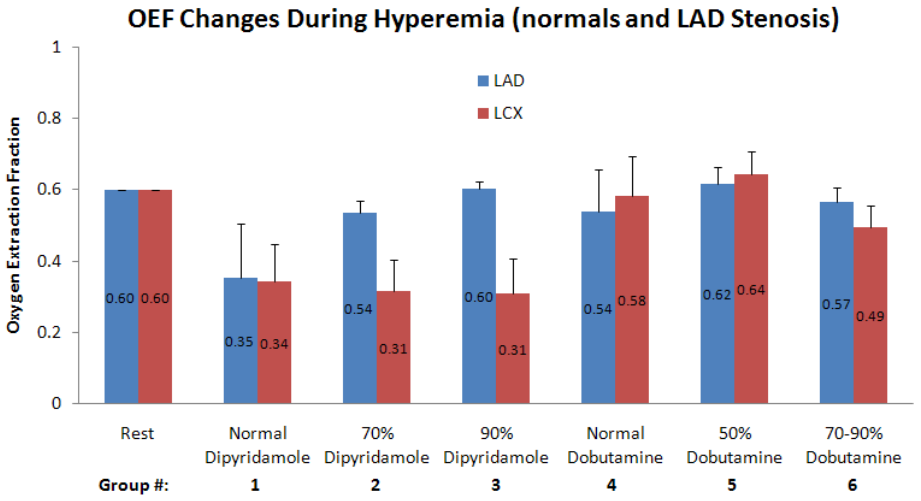

Figure. OEF Changes after vasodilation or hyperemia with varying stenosis severity.

Conclusions

Using our cardiac MR methods, regional differences in myocardial OEF induced by a single-vessel stenosis during hyperemia can be readily detected. However, these methods need further improvement to prevent flow artifacts for more accurate quantification, particularly during the Dobutamine-induced hyperemia when the heart rate increases dramatically.

References

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