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Meeting abstract

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231 Alternation of myocardial oxygen consumption during hyperemia: detection with a CMR method

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Introduction

Myocardial oxygen consumption (MVO_2) directly reflects myocardial oxygen supply and demand. The purpose of this study is to test the ability of a cardiovascular magnetic resonance (CMR) method to determine changes in myocardial MVO_2 during pharmacologically-induced hyperemia in a canine stenosis model.

Methods

13 dogs were divided into four groups, which can be seen in Table 1. Stenosis was created by an occluder around the proximal left-anterior descending (LAD) and stenosis severity was confirmed via Doppler flow reduction. MVO_2 was calculated by the Fick principle: $MVO_2 \propto OEF \times MBF$, in which OEF is the oxygen extraction fraction and MBF represents myocardial blood flow.

OEF during hyperemia was determined by a two compartment model with measured myocardial T2 that is measured with a 2-D segmented turbo spin-echo (TSE) sequence [1]. This sequence was performed 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 the quantitative first-pass perfusion CMR 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 (LCX) perfused inferior region.

Results

MVO_2 results can be seen in Figure 1. As expected, Dobutamine causes a dramatic increase in MVO_2, while injection of Dipyridamole shows only a moderate effect.

In the anterior area 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. With Dobutamine, MVO_2 increased significantly at 57.9% and 35% for the 50% and 70–90% stenosis groups, respectively.

In the remote normal LCX perfused region, Dipyridamole induced moderate increases in MVO_2 at 49.9% and 17.3% in the 70% and 90% stenosis groups, respectively. 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]. As expected, Dobutamine induced much higher changes in

Table 1: Dog groups

Group (*n*)	Stenosis	Stress
1 (4)	70%	Dipyridamole
2 (3)	90%	Dipyridamole
3 (3)	50%	Dobutamine
4 (3)	70–90%	Dobutamine

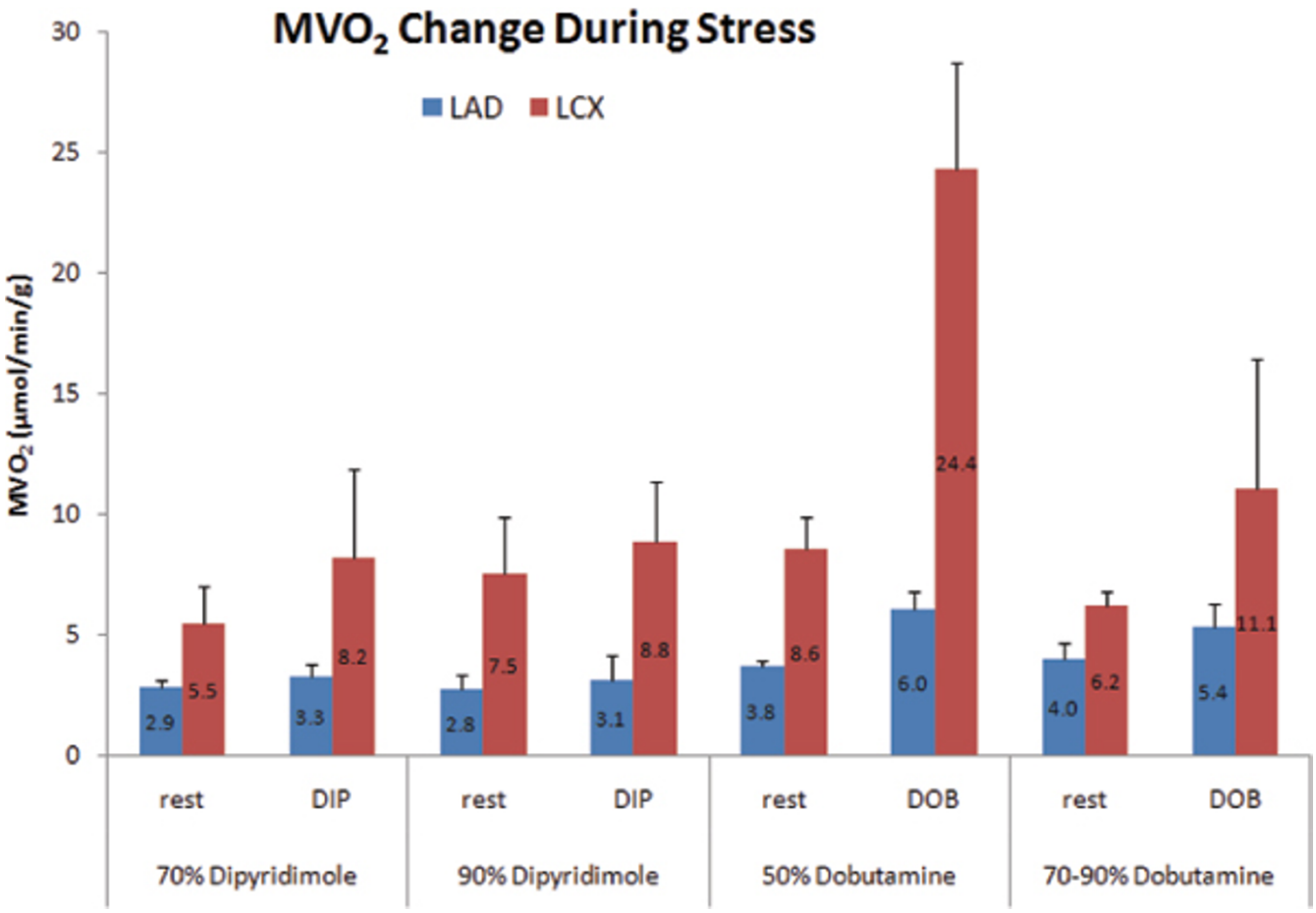

Figure 1
Changes in MVO_2 during dipyridamole or dobutamine with various LAD stenosis. A quantitative CMR method is introduced to detect changes in myocardial MVO2 during hyperemia in a canine stenosis model. While severe stenosis attenuated MVO2 increase in the stenosis subtended region, the remote region also showed reduced increase in MVO2.

MVO_2, 183.7% and 79% increases in the 50% and 70–90% stenosis groups, respectively. It is interesting to note that severe single-vessel stenosis not only attenuated the increase in MVO_2 in stenotic perfused region with both Dipyridamole and Dobutamine, but also attenuated MVO_2 in the remote normal myocardial region.

Conclusion

Our CMR method can non-invasively quantify regional myocardial MVO_2. Determination of the changes in MVO_2 is important in the diagnosis and management of patients with coronary artery disease.

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