

WORKSHOP PRESENTATION



Fast, heart-rate independent, whole-heart, free-breathing, three-dimensional myocardial BOLD MRI at 3T with simultaneous ¹³N-ammonia PET validation in canines

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Background

Myocardial BOLD MRI is a non-contrast approach for examining myocardial perfusion. Although recent developments have shown promising technical advancements, current myocardial BOLD MR methods are still limited by: (a) poor spatial coverage; (b) imaging confounders; and (c) imaging artifacts, particularly at 3T. To address these limitations, we developed a heart-rate independent, free-breathing 3D T₂ mapping technique at 3T that utilizes near 100% imaging efficiency, which can be completed within 3 minutes. We tested our method in a canine model and validated our findings with simultaneously acquired ¹³N-ammonia PET perfusion data in a whole-body PET/MR system.

Methods

Canines with and without LAD stenosis (n = 11) were studied in a PET/MR system. The proposed sequence was prescribed at rest and under adenosine stress (140 mg/min/kg). Dynamic ¹³N-ammonia PET scans were acquired for validation purpose. PET images were analyzed using qPET software. In healthy dogs, mean myocardial T₂ (T2avg) were measured at rest and stress and mean myocardial blood flow (Qavg) were derived from PET images in the corresponding slices. Myocardial BOLD Response (MBOLDR = T2avg (stress):T2avg (rest)) and perfusion reserve (MPR = Qavg (stress):Qavg (rest)) were computed and compared. In the stenosis study, the affected regions were identified from the PET

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images and matched to the corresponding slices in BOLD data. Mean myocardial T_2 and myocardial perfusion were measured at rest and stress in the affected and remote territories. MBOLDR and MPR from affected and remote regions were computed and compared against to each other.

Results

A representative set of BOLD and PET images acquired from a healthy dog under rest and stress are shown in Figure 1. T2avg measured under stress were significantly higher than at rest (T2avg: 38.5 ± 1.0 ms (rest) vs. $44.4 \pm$ 3.1 ms (stress), p < 0.05). As expected, Qavg were significantly higher during adenosine stress relative to rest (Qavg: 0.8 ± 0.1 ml/mg/min (rest) vs 2.0 ± 0.9 ml/mg/min (stress); p < 0.05). Linear regression of MBOLDR and MPR showed high correlation ($R^2 = 0.67$, p < 0.05). In Figure 2, a set of PET and BOLD images from a dog with LAD stenosis acquired during stress are presented (A and C). Perfusion defect was consistently observed in the LAD territory from both PET and BOLD images. Panel B shows MPR was significantly higher in the remote regions (2.8 \pm 1.7) compare to the affected regions (1.4 ± 1.0) , p < 0.05. Significant higher MBOLDR was also observed in panel D (Remote: 1.09 ± 0.04 , Affected: 1.00 ± 0.03 , p < 0.05).

Conclusions

The proposed BOLD CMR approach permits rapid whole LV assessment of BOLD changes between rest and adenosine stress. The BOLD responses were very closely correlated with PET perfusion, suggesting that the proposed BOLD CMR method is a viable approach



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for imaging myocardial perfusion. The method remains to be validated in patients.

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