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Feasibility Study of Myocardial Perfusion and Oxygenation by Non-Contrast MRI: Comparison with PET Study in a Canine Model

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Abstract

The purpose of this study was to examine the feasibility of quantifying myocardial blood flow (MBF) and rate of myocardial oxygen consumption (MVO_2) during pharmacologically induced stress without using a contrast agent. The former was measured by the arterial spin labeling (ASL) method and the later was obtained by measuring the oxygen extraction fraction (OEF) with the magnetic resonance imaging (MRI) blood oxygenation level-dependent (BOLD) effect and Fick's law. The MRI results were compared with the established positron emission tomography (PET) methods. Six mongrel dogs with induced acute moderate left coronary artery stenosis were scanned using a clinical PET and a 1.5T MRI system, in the same day. Regional MBF, myocardial OEF, and MVO_2 were measured with both imaging modalities. Correlation coefficients (R^2) of the three myocardial indexes (MBF, OEF, and MVO_2) between MRI and PET methods ranged from 0.70 to 0.93. Bland-Altman statistics demonstrated that the estimated precision of the limits of agreement between MRI and PET measurements varied from 18% (OEF), to 37% (MBF), and 45% (MVO_2). The detected changes in these indexes, at rest and during dobutamine stress, were similar between two image modalities. The proposed non-contrast MRI technique is a promising method to quantitatively assess myocardial perfusion and oxygenation.

Keywords

MRI; PET; myocardial blood flow; oxygen consumption; non-contrast; comparative studies

1. Introduction

Myocardial ischemia results from an imbalance between oxygen supply (MBF) and demand (MVO_2). Therefore rapid, accurate, noninvasive assessments of regional differences of the OEF (defined as $([O_2]_{\text{artery}} - [O_2]_{\text{vein}}) / [O_2]_{\text{artery}}$) are critical for the accurate diagnosis of CAD since OEF reflects the combined effects of MBF and MVO_2 . Positron emission tomography (PET) has been used at our institution and elsewhere to non-invasively quantify regional MVO_2 [1,2,3,4] and MBF [5,6,7]. However, low spatial resolution, limited availability, relatively high costs, and the need for ionizing radiation limit the widespread use of PET for this purpose.

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MRI techniques using the blood oxygenation level-dependent (BOLD) effect along with T₂ contrast have yielded promising results for myocardial oxygenation assessments in humans and animals [8,9,10,11,12,13]. Recently, the T₂ method has been used to quantify coronary venous oxygen content in the heart [14] and OEF in the brain [15]. Consequently, if MBF can be measured directly in the heart, then the MVO₂ can be quantified using Fick's Law:

$$MVO_2 = [O_2]_a \times OEF \times MBF. \quad (1)$$

The constant $[O_2]_a$ is defined as the total oxygen content of arterial blood, (which is equal to $(1.39 \times [Hb] \times \%Sat)/100$). The value 1.39 represents the maximum binding capacity of oxygen per unit mass of hemoglobin (ml O₂/g hemoglobin), [Hb] refers to the hemoglobin concentration of the blood (hemoglobin/ml blood), and term %Sat refers to the percentage saturation of oxygen in arterial blood. Recently, we have proposed to measure regional myocardial OEF [16] and MBF [17] without the use of any contrast agents. In this paper, the aim was to compare these MRI techniques in the measurements of these cardiac indexes (MBF, OEF, and MVO₂) with the corresponding PET outcomes.

2. Materials and Methods

2.1 Animal preparation

All animal procedures were approved by the Animal Studies Committee of our institute. Six mongrel dogs (23.0 ± 1.3 kg) were fasted for 24 hours prior to the study and sedated with 1-2 mg/kg morphine sulfate subcutaneously. Thirty to sixty minutes after the morphine was given, the dogs were anesthetized with 12.5 mg/kg sodium thiopental and 72 mg/kg α -chloralose, administered intravenously. The dogs were intubated with a 9.0 French endotracheal tube, and placed on a Harvard ventilator with 100% oxygen. Anesthesia was maintained throughout the entire procedure with intravenous administration of 5-10 ml α -chloralose titrated as needed. Each dog was monitored by an electrocardiographic system throughout the study.

Coronary stenosis was created in the proximal to middle portion of the left anterior descending (LAD) artery by an intracoronary Teflon ring that was made in our laboratory. The diameter reduction of the LAD was equal to or larger than 50%, to sufficiently impair the myocardial perfusion reserve. To generate a Teflon ring stenosis in a coronary artery, the neck skin was shaved, cleaned, and incised over a carotid artery. An 8.0 French catheter sheath advanced proximally into the vessel through an arteriotomy. A guide catheter was advanced into the left coronary artery and a baseline left coronary angiogram was obtained to ensure correct positioning. A 0.014-inch x 300cm exchange guide wire was then advanced through the guide catheter into the LAD branch. A Teflon ring was advanced over the wire into the coronary artery with use of formacath, a radio-opaque polyethylene catheter material. The guidewire and formacath were withdrawn leaving the stenosis in place in the artery. Left coronary angiography was repeated to confirm the site and severity of the diameter-narrowing stenosis by the visualization. A venous line was used for the administration of fluids as well as drug intervention. After the dogs became stable, they were first moved to the PET imaging suite, followed by the MRI study. Both MRI and PET imaging procedures were performed on the same day for optimal comparison. The time for the entire study lasted 6-7 hours including the surgical procedures.

2.2 PET Imaging Protocol

All PET studies were performed on a conventional tomograph (Siemens ECAT 962 HR+, Siemens Medical Systems, Iselin, New Jersey). The electrocardiogram, arterial blood pressure, and blood gas values were monitored throughout the procedure (Propaq, Protocol Systems, Welch Allyn, Beaverton, OR). The PET imaging protocol is outlined in Figure 1. A

transmission scan was first performed to correct for photon attenuation. ^{15}O -water (up to 1.0 mCi/kg) was administered as an intravenous bolus and dynamic PET data was acquired for 5 minutes to measure the resting myocardial perfusion. Approximately 10 min after the decay of H_2^{15}O radioactivity was allotted for (half-life of ^{15}O is 2.5 min), ^{11}C -acetate (0.40 mCi/kg) was administered as a bolus and 30 minutes of dynamic data collection occurred to determine the resting MVO_2 measurements. Venous blood samples were obtained at predetermined intervals to measure the $^{11}\text{CO}_2$ concentrations. These time intervals varied: 4-5 min at rest and 1-3 min during the dobutamine stress. Approximately 100 min after the ^{11}C radioactivity decay (half life of ^{11}C is 20 min), dobutamine was administered, and the same procedure with both ^{15}O -water and ^{11}C -acetate scans was followed to determine MBF and MVO_2 during hyperemia. Stress was induced by intravenous infusion of an initial dose of 10 $\mu\text{g/kg/min}$ dobutamine and increased at 5 min intervals to 20, 30, and a maximum of 40 $\mu\text{g/kg/min}$. The peak dose of dobutamine was determined when the heart rate increased to greater than 130 beats/min. As soon as PET imaging was finalized, the dogs were transported to the MR scanning room under the same anesthesia.

2.3 MRI Imaging Protocol

All MR imaging was performed on a 1.5-T whole-body Sonata system (Siemens Medical Solutions, Erlangen, Germany), equipped with a fast gradient system (maximal gradient strength = 40 mT m^{-1} ; maximal slew rate = 200 mT m^{-1}/ms). A four-element phased array coil was used for signal reception and a body coil was used as a transmitter. The dogs were positioned supine, and ventilated with 100% oxygen. Heart rate and blood pressure were monitored continuously with an MR-compatible vital-signs monitoring system (Millennia; Invivo Research, Orlando, FL). Scout imaging was performed to obtain a short-axis image of the left ventricle (LV) at the middle level of the papillary muscle. Cine MR imaging was performed at rest to determine the motionless period during the cardiac cycle. As reported previously for myocardial OEF measurement, multiple myocardial T_2 -weighted MR imaging was performed in the same section location at rest and during pharmacological stress using a multi-contrast segmented turbo spin echo (TSE) sequence [16]. T_1 -weighted images were acquired as well for MBF measurement using a recently developed arterial spin labeling (ASL) pulse sequence [17]. Stress was induced by the same procedure as in the PET study, i.e., through the infusion of dobutamine, and T_1 -weighted and T_2 -weighted images were taken for determining the MBF and OEF, respectively, at the hyperemic state.

2.4 MR Sequences

For OEF measurements, the basic sequence structure proposed for in vivo T_2 mapping was a multi-contrast 2-D segmented turbo spin-echo (TSE) sequence or segmented Carr-Purcell-Meiboom-Gill (CPMG) multi-echo sequence. The sequence was triggered by an electrocardiographic signal. Double inversion recovery pulses were placed in each cardiac cycle to minimize the effect of fast pulsatile blood flow within the LV. The turbo spin-echo train was placed in mid-diastole to minimize cardiac motion. Other image parameters included field-of-view = 220 x 131 mm^2 ; data acquisition matrix size = 256 x 61; section thickness = 8 mm; inversion time = 350 to 500 ms, depending on the duration of the RR interval; and data acquisition time = 24 x RR interval, or 14.4 s for a typical RR interval of 600 ms. To reduce respiratory motion, ventilation was turned off to simulate breath-holding in dogs during each MRI scan.

The ASL method applied a pair of slice-selective and volume-selective inversion prepared (IR) pulses to estimate MBF by measuring myocardial T_1 values. The sequence was described in detail in a previous publication [17]. The ASL sequence parameters included: TR/TE = 2.15/1.2 ms; flip angle = 5°; field of view = 220 x 131 mm^2 ; slice thickness = 8 mm; and interpolated matrix size = 256 x 128.

3. Data Analysis

3.1 PET Data

Data reconstruction was performed in a 2-D manner with filtered backprojection and transferred to a Sun workstation (Sun Microsystems, Menlo Park, CA) for image analysis with software developed in our laboratory. Myocardial images were reformatted to the true short-axis views on which measurements of perfusion and metabolism were performed. Since the reformatted PET images had an isotropic voxel size of 3.43 mm, three slices that approximately matched to the location of MR slice (8 mm slice thickness) were selected, based on the distance from the central slice to the base of LV. The distance was measured on MRI scout images. Regions of interest (ROIs) were then drawn on this PET image to generate myocardial and blood time activity curves for each set of tracer data. Three ROIs were located on the anterior (ANT), lateral (LAT), and inferior (INF) areas (Fig. 2). All ROI contours were made as similarly as possible among PET and MR images. Regional MBF (ml/g/min) was quantified with a previously validated compartmental modeling method [5,18]. Blood and myocardial time-activity curves were used in conjunction with a one-compartment kinetic model to estimate the rate (k_2) at which ^{11}C -acetate is converted to $^{11}\text{CO}_2$ (k_2 , min^{-1}) [19,20]. Regional MVO_2 ($\mu\text{mol/g/min}$) was determined using a previously published relationship between k_1 and MVO_2 [1]. Once the MVO_2 and MBF were determined by PET, the regional OEF could then be calculated by the means of Fick's law [see Eq. 1].

3.2 MRI Data

The calculation of OEF by this MR method has previously been reported [16]. In brief, two T_2 maps were acquired at rest with different echo spacing to obtain two parameters for modeling the OEF calculation. ROI measurements were performed in the same regions, indicated in the PET analysis, on all T_2 maps. Myocardial OEF during the dobutamine stress was then calculated. To perform these calculations, myocardial OEF and myocardial blood volume (MBV) at rest were assumed at 0.6 and 6.4 ml/100g, respectively [16]. The rest OEF value was based on OEF values measured in normal dogs using an arterial and coronary sinus blood sampling approach at rest. It was assumed that this value changes little with moderate stenosis [18] although this value did vary slightly with different dogs (see PET results below). For this reason, we also performed a second calculation using the OEF value measured by PET at rest for each dog to calculate its OEF value during the stress in our MR data analysis. Regional MBV at rest may also vary, depending on the stenosis status (see discussion section).

Since MVO_2 is approximately proportional to the rate-pressure product (RPP), [1,3,19], the ratio of MVO_2 during stress to MVO_2 at rest was assumed to be the ratio of the RPP during stress and RPP at rest. Using the relationship between myocardial T_2 and OEF [16], myocardial OEF during the dobutamine stress could then be estimated.

MBF was calculated pixel-by-pixel based on a two-compartment model [21]:

$$\text{MBF} = \lambda \frac{T_{1,GS}}{T_{1,Blood}} \left(\frac{1}{T_{1,SS}} - \frac{1}{T_{1,GS}} \right) \quad (2)$$

where λ is a constant for the blood-tissue partition coefficient of water ($\lambda = 0.92$ ml/g for canine myocardial tissue); $T_{1,GS}$ is the T_1 of myocardium obtained with the volume-selective IR pulse; $T_{1,Blood}$ is the average T_1 of the LV blood pool and can be taken as a constant; and $T_{1,SS}$ is the T_1 of myocardium obtained with the slice-selective IR pulse. Similar ROIs as on myocardial T_2 maps were drawn on these maps to obtain regional MBF values. Knowing OEF and MBF, MVO_2 can then be calculated by means of Fick's law (Eq. 1).

3.3 Statistical Analysis

Data was expressed as mean $\pm$ standard deviation (SD). Each data set (MRI or PET) consisted of 3 ROI values. All ROI data were pooled together for statistical analysis. Differences between MRI and PET measurements in OEF, MBF, and MVO₂ values were analyzed by linear regression and Bland-Altman plots. Paired comparison was also made by use of a Student's *t* test with a *P* < 0.05 significance level.

4. Results

All dogs were hemodynamically stable throughout the PET and MRI studies. Table 1 lists the blood pressure and heart rate data for the dogs scanned. The hemodynamic data during dobutamine in the PET study was averaged over the entire period of data acquisition since the PET imaging time was relatively long (over 40 min) comparing to corresponding MRI time (5 min). As expected, both heart rate and blood pressure increased during dobutamine stress, resulting in a more than 2-fold higher RPP (*P* < 0.01). Any difference of these parameters between PET and MRI was not significant (*P* = NS).

Because of some severe motion artifacts during the dobutamine stress MRI sessions, some MR data (MBF or OEF) in the six dogs was excluded for further analysis, resulting in comparable (MRI vs. PET) data sets for MBF (*n* = 5), OEF (*n* = 4), and MVO₂ (*n* = 3). Figure 2 shows image examples from one dog with 50-60% diameter narrowing in the middle portion of the LAD.

4.1 MBF in Dogs

Figure 3 shows the correlation of MBF values measured by the MRI ASL method and PET perfusion, with a good agreement (*R*² of 0.83). The Bland-Altman plot demonstrates a small overestimation by MRI with a bias of 8.1% (lower and upper limits of agreement of 28.6% and -44.9%, respectively). Comparing the regional differences (Fig. 4a), MBF in the INF bed increased from 0.99 ± 0.20 ml/g/min at rest to 2.06 ± 0.69 ml/g/min (*P* < 0.02) during dobutamine stress with MRI ASL measurements, whereas MBF increased from 0.76 ± 0.09 to 1.93 ± 0.50 ml/g/min (*P* < 0.001) with PET measurements. In the perfusion deficit ANT bed, MBF increased less, from 0.80 ± 0.14 ml/g/min at rest to 1.28 ± 0.57 ml/g/min (*P* = NS) during stress (MRI), or from 0.74 ± 0.14 to 1.50 ± 0.45 ml/g/min (PET) (*P* < 0.01). The increase in MBF in the LAT region was similar to that in the INF bed with both MR and PET methods.

4.2 OEF in Dogs

Correlations between MRI and PET in the OEF values showed the most agreement. When the rest OEF of 0.6 was used for the calculation of OEF during stress, the correlation coefficient (*R*²) was 0.87 (Figure 5b) and MRI slightly underestimated the OEF during the stress (Figure 5a). If we selected the rest OEF from the PET OEF data at rest, the *R*² was slightly improved to 0.93 (Fig. 5d). However, the MRI underestimation increased using this method (Figure 5c). Nevertheless, the regional OEF values were in a good agreement between MRI and PET measurements (Fig. 4b). MRI measured the OEF during dobutamine infusion to be 0.65 ± 0.19 (ANT), 0.69 ± 0.24 (LAT), and 0.55 ± 0.11 (INF). No significant differences were found between PET and MR derived OEF nor between the three anatomical regions.

4.3 MVO₂ in Dogs

With a rest OEF of 0.6, the MRI MVO₂ overestimated the PET MVO₂ by a difference of 16.4% (Fig. 6a). After the selection of the rest OEF from PET data, the MRI overestimation was decreased to 4.7% (Fig. 6c). The correlation between MRI and PET was slightly improved with *R*² increasing from 0.70 (Fig. 6b) to 0.75 (Fig. 6d). As shown in Figure 4c, MVO₂ in all vascular

beds increased significantly from the rest to stress conditions, as consistently observed by both PET and MR images ($P < 0.05$). However, the ANT MVO₂ during dobutamine tended to increase less compared to the LAT and INF regions ($P = 0.05$ and $P = 0.06$, respectively); No significant differences were found between corresponding MR and PET measurements.

5. Discussion

The goal of this study is to evaluate the capability of MRI to measure regional myocardial MBF, OEF, and MVO₂ during the stress imaging session without using any contrast agent. Because PET is considered the “gold standard” for estimations of OEF and MVO₂, we compared our MRI results with the PET method. In order to evaluate regional differences, an acute stenosis model was created in dogs that were scanned in the same day using both PET and MRI modalities. This is a relatively difficult study because it requires the hemodynamics of the dogs to be maintained at a comparable level during both imaging studies. For this reason, only moderate coronary artery stenoses (diameter narrowing stenosis of 50-60%) were created.

In this MRI study, MBF was determined by a fast ASL method that has previously been validated in dogs [17]. Myocardial OEF was measured through another two-compartment model using the apparent myocardial T₂ values [16]. By integrating both OEF and MBF measurements, MVO₂ was calculated at rest and during dobutamine-induced stress by Fick's Law [see Eq. 1]. In comparison with PET measurements performed on the same day and in the same subjects, MRI results show an excellent agreement in the three myocardial parameters separately. The correlation coefficients (R^2) were 0.83, 0.93, and 0.75 for MBF, OEF, and MVO₂ respectively.

In general, dobutamine infusion increases MVO₂ by increasing the cardiac workload, which in turn increases MBF. In normal tissue, oxygen demand and supply are matched, which results in no change in OEF. With progressively worsening coronary artery stenosis, MBF will decrease (Fig. 4a), and eventually MBF will not be able to meet oxygen demand by myocardial tissue, resulting in increased OEF to prevent myocardial ischemia. Thus, one should see a slightly higher OEF in tissue subtended by a coronary artery with stenosis compared with tissue from a normal artery (Figure 4b). However, for a coronary artery stenosis of 50-60% in diameter narrowing, increases in OEF may not be easily observed due to auto-regulation of the heart. On the other hand, MVO₂ should always increase with dobutamine in normal beds. With a stenosis of 50-60%, MVO₂ will still increase in a stenotic territory, but only a fraction of the increase of a normal perfusion bed, as we have observed with both MRI and PET methods.

The errors associated with our proposed MRI methods are relatively moderate. With PET results as standard references, the errors for the measurement of MBF and OEF can be estimated from the standard deviations of the differences between MRI and PET data. They are approximately 37% and 18% for MBF and OEF, respectively. These errors are in accordance with previously reported theoretical values of 36% [17] and 15% [16]. With a simple calculation of error propagation for Fick's equation, the error for measuring MVO₂ would be 40%, which corresponded well with our measured MRI results (45%). For normal myocardial tissue, the change in MVO₂ from rest to stress was approximately over 150%. For stenotic tissue, this change was over 85%. Furthermore, during dobutamine stress, the difference in MVO₂ between normal and stenotic tissues was approximately 76%, also confirming that our MRI methods are sensitive enough to differentiate MVO₂ on a regional basis. This moderate accuracy should allow for the assessment of changes in MVO₂ from rest to inotropic stress in normal tissues as well as tissues with moderate MBF deficits.

The MR measured values of MBF and myocardial OEF, mainly reflect conditions at the diastolic phase of the cardiac cycle. However, the gold standard measurements using PET

represent average values over the entire cardiac cycle. In normal coronary arteries, most blood flow occurs during diastole as myocardial compression during systole increases distal vascular resistance. This diastolic predominant blood velocity pattern was demonstrated in human subjects by utilizing Doppler transesophageal echocardiography [22,23], and transthoracic echocardiography [24]. This pattern remains the same during reactive hyperemia. Furthermore, it has been reported that the diastolic fraction of coronary blood flow significantly correlated with the transmural distribution of MBF in animal studies [25]. For these reasons, we believe the measured MBF and OEF by MRI at diastole can be compared with the respective gold standards.

5.1 Limitations

There are several limitations in this study. First, myocardial OEF at rest should be directly measured with MRI, rather than a simple assumption. The two different methods we used either assumed that the rest OEF in all myocardial regions was equal to 0.6 [16], or that the resting OEF during MRI was equal to the resting OEF during PET in the corresponding myocardial regions. In PET study, it was found that resting OEF in the anterior region was indeed less than the resting OEF of other regions (0.47 ± 0.09 vs 0.54 ± 0.1). However, there were no statistical differences between these regions in OEF values at rest. Although the later method (using PET OEF at rest) did slightly improve the correlation between MRI and PET measurements in MVO_2 , there is no significant difference in MVO_2 data between two methods.

Secondly, MBV should be measured directly with MRI. This is also true for regional MBV since MBV at rest may increase in the stenotic territory. A 10% increase in MBV may lead to a 5.7% error in the calculated OEF during hyperemia [26]. The measurement of MBV could be accomplished with contrast enhanced first-pass perfusion imaging [27]. This study is currently undergoing in our laboratory. Thirdly, MRI and PET studies were not performed simultaneously. Although the dogs show similar hemodynamic signs in our same day study (Table 1), it was not clear whether the dogs maintained similar physiological conditions during both studies. Lastly, exact matching of the ROIs between MR and PET images was likely not achieved. These restrictions may explain some deviations in the measurements between MRI and PET studies.

6. Conclusions

In summary, myocardial perfusion and oxygenation can be estimated with the proposed non-contrast MRI approach, allowing temporal monitoring of these myocardial indexes. Although accuracy and precision of these MRI measurements need further improvement, this approach has permitted differentiation in the changes of myocardial perfusion and oxygenation from rest to stress conditions. Therefore, it has the potential to facilitate a non-invasive evaluation of cardiac interventions and estimate myocardial oxygenation status underlying cardiac disease of diverse etiologies. Further investigation for the assessment of ischemic myocardium with this approach is warranted prior to its clinical applications.

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Figure 1. The PET protocol for all dog studies. Dashed line represents time at rest and solid line represents dobutamine-induced stress.

Figure 2.

Examples of ROI locations of MR and PET image. Top row are images obtained at rest and bottom row are images acquired during dobutamine stress. (a) and (e) are T_2 maps for measuring the OEF; (b) and (f) are MBF maps created by the ASL method; (c) and (g) are PET source images to calculate MBF; (d) and (h) are PET images to calculate MVO_2 . The scalar bar indicates absolute MRI MBF magnitude in ml/g/min. The anterior (ANT) ROI is in the stenotic vessel-perfused bed and the inferior (INF) ROI is in the remote normal vessel-perfusion bed. Lateral (LAT) regions were also indicated. In all PET images, the signal intensities in each source image were normalized to its own maximal intensity of the image. Therefore, they are not comparable to each other.

Figure 3.

(a) Correlation of MBF measured by MR ASL and PET methods; (b) Bland-Altman plot of the percent-differences in these MBF values versus the averaged MBF values. No significant difference was found between MRI and PET methods.

Figure 4.

The averaged (a) myocardial MBF, (b) OEF, and (c) MVO₂ measured with MRI and PET methods, at rest and during the dobutamine stress (OEF only during dobutamine). Both methods show similar changes in these myocardial indexes. There were no significant differences between MRI and PET on any of the three parameters (MBF, OEF, or MVO₂).

Figure 5.

Bland-Altman plots of the percent-differences of myocardial OEF measured by MRI and PET methods versus their averaged values during the dobutamine stress, (a) when the myocardial OEF was assumed to be 0.6 at rest for MRI stress OEF calculation; and (c) when the myocardial rest OEF was taken from PET measurement values. MRI values during stress were slightly underestimated in (a) (bias = 1.1%) and more drastically underestimated in (b) (bias = 6.5%). There are no significant differences between PET and either MRI methods. The second MRI method demonstrates slightly increased correlations from respective regression lines in (d) than (b). There is no significant difference between the two MRI OEF calculations.

Figure 6.

Bland-Altman plots of the percent-differences of MVO₂ measured by MRI and PET verses their averaged values, at rest and during the dobutamine stress, (a) when the myocardial OEF was assumed to be 0.6 at rest for MRI calculation; and (c) when the myocardial OEF at rest was taken from PET measurement values. MRI values were underestimated in (a) (bias = -16.4%) and less underestimated in (b) (bias = -4.7%). The correlation was slightly improved when myocardial OEF at rest was estimated from PET data in (d) than in (b) when myocardial OEF at rest was fixed at 0.6. There is no significant difference between two sets of MRI MVO₂ data.

Table 1
Hemodynamic data in dogs (n=6) during PET and MRI scans.

	Rest [†]			Dobutamine [‡]		
	Heart Rate	Systolic Pressure	RPP(x10 ³)	Heart Rate	Systolic Pressure	RPP(x10 ³)
MRI [*]	93.0 ± 9.4	127.7 ± 27.1	11.7 ± 1.8	124.8 ± 21.4	168.2 ± 24.0	20.9 ± 4.5
PET [*]	89.2 ± 10.1	115.5 ± 24.8 ^{†‡}	10.2 ± 1.8	141.3 ± 21.8	167.2 ± 22.9	23.5 ± 4.1

^{*} No significant difference in corresponding data between PET and MRI (*P* > 0.05)

[†] Significant difference in all corresponding data between rest and dobutamine in both MRI and PET (*P* < 0.01)

^{†‡} except (*P* < 0.03).