

POSTER PRESENTATION

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Metabolic imaging of *in vivo* myocardium

Charles S Springer^{1,2*}, Craig S Broberg^{3,2}, William D Rooney^{1,2}

From 18th Annual SCMR Scientific Sessions
 Nice, France. 4-7 February 2015

Background

The *equilibrium* cellular water efflux rate constant [k_{i0} ; mean water lifetime inverse] from contrast agent [CA]-enhanced MRI measures on-going cellular Na^+ , K^+ -ATPase activity [turnover]. Good literature [4 different labs] agreement shows substantial k_{i0} decreases in myocardial ischemia, hypertension, or infarct regions (Table). The 3 methods used differ in extracellular ("outside") CA_o level manipulation to change the MR shutter-speed relative to k_{i0} and the MR exchange condition reached: A) CA_o steady-state, slow-exchange-regime; B) CA_o titration, fast-exchange-regime [FXR]; and C) CA_o wash-out, FXR. The independent intracellular volume fraction [ICV] - cell density•volume product and ≈ 1 - ECV [extracellular volume fraction] - also decreases in pathology. We hypothesize that k_{i0} mapping shows metabolic compromise most effectively. We report initial experience with tissue near a repaired ventricular septal defect [VSD].

Methods

We acquired serial 1.5T $^1\text{H}_2\text{O}$ T_1 -weighted data from a 27 yo male before and 3 times after a bolus IV 0.15 mmol/kg CA [Omniscan] injection. Quantitative Look-Locker T_1 measurements [non-selective inversion, 21 recovery times] imaged an 8 mm slice with a mid-ventricular short axis location inferior to the VSD patch. Method C (CA_o wash-out, FXR) determined k_{i0} and ICV values in six LV wall segments.

Results

The Figure shows a post-CA T_1 -w image: the endo- and epicardial LV wall edges as bright orange and green, respectively [light orange circle, an LV ROI]. Segmental ICV and k_{i0} values are given (yellow). Segments S5 and S6 comprise the septum. The ICV values for segments S1 - S4 are reasonable for normal myocardium (Table). Thus, we

have indicated (*) a control myocardial k_{i0} value [5 s^{-1} , Table], since the CA wash-out data quantity [3 points] and quality from these normal myocardium segments yielded insufficient precision. Interestingly, the k_{i0} value is reduced [4.5 s^{-1}] in segment S6, and dramatically so [1.7 s^{-1} ; 66%↓] in segment S5, immediately inferior to the VSD patch.

Conclusions

The k_{i0} biomarker is a sensitive measure of on-going myocardial metabolic activity. Our result suggests that tissue nearby a VSD patch can be, or become, metabolically compromised.

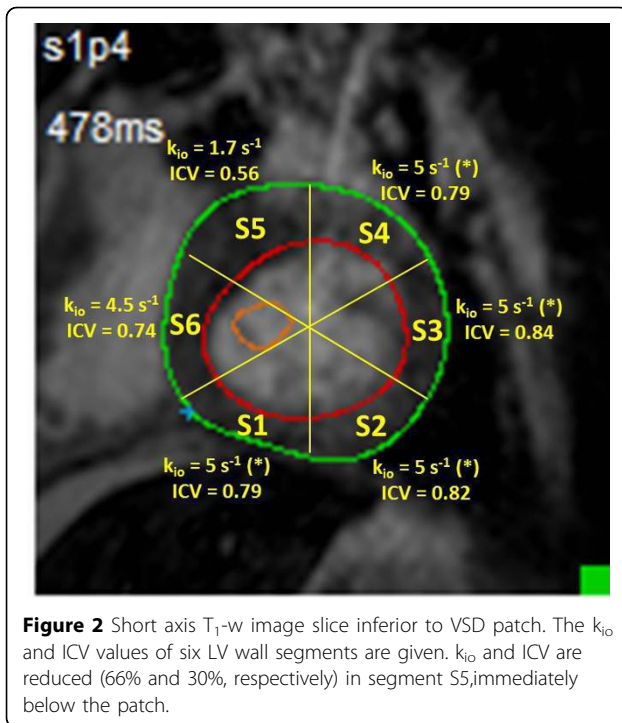
The ultimate goal is pixel-wise k_{i0} and ICV maps. [Here, nominal voxels are $2 \times 2 \times 8 \text{ mm}^3 = 32 \text{ }\mu\text{L}$.] For this, one needs data with good S/N and more than 3 wash-out points. Also, method C has systematic error absent in methods A and B, which cannot be used for humans. It assumes the CA_o concentration equals that of CA_p [in plasma] during wash-out. This is invalid for finite CA intravasation kinetics, which may be particularly slow in myocardial lesions due to common reduced

report date	method	myocardium	ICV	k_{i0} (s^{-1})
2006	A	ex vivo rat (perfused/beating) control		5.6
		(no flow) ischemia		3.7
		change		36%↓
2013	B	in vivo mouse control [n = 13]	0.75	5.3
		chronic hypertension [n = 17]	0.58	2.3
		change	23%↓	57%↓
2013	C	in vivo human control [n = 12]	0.69	
		chronic hypertension [n = 8]	0.55	
		change	20%↓	
2014	C	in vivo human control [n = 4]	0.69	3.2
2014	C	in vivo human control [n = 20]	0.69	10.0
		chronic infarct [n = 20]	0.39	2.5
		change	43%↓	75%↓

Figure 1 Literature reports of active trans-membrane water cycling [k_{i0}] and intracellular volume fraction [ICV] values in normal and pathological myocardia.

¹Advanced Imaging Research Center, Oregon Health & Science University, Portland, OR, USA

Full list of author information is available at the end of the article



vascularization. Possible k_{10} and ICV underestimations can be corrected using K^{trans} [the CA extravasation transfer constant] from the bolus tissue wash-in time-course to calculate the CA intravasation rate constant.

Funding

NIH: RO1-NS040801.

Authors' details

¹Advanced Imaging Research Center, Oregon Health & Science University, Portland, OR, USA. ²Knight Cardiovascular Institute, Oregon Health & Science University, Portland, OR, USA. ³Division of Cardiovascular Medicine, Oregon Health & Science University, Portland, OR, USA.

Published: 3 February 2015

doi:10.1186/1532-429X-17-S1-P251

Cite this article as: Springer et al.: Metabolic imaging of *in vivo* myocardium. *Journal of Cardiovascular Magnetic Resonance* 2015 17(Suppl 1):P251.

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