

Transcytolemmal Water-Exchange Slows in Infarcted Myocardium

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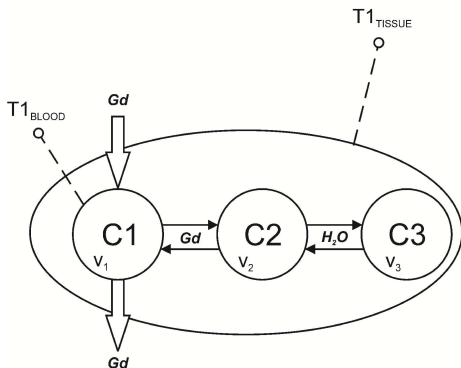
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Purpose: To measure and compare the transcytolemmal cellular water exchange rates in fibrotic and viable myocardium in patients with chronic myocardial infarcts.

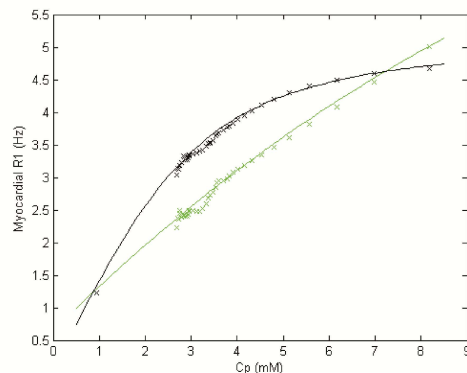
Introduction: Quantitative fibrosis measures using partition coefficient and extracellular volume (ECV) have been recently studied using pre- and post- Gd-contrast T1 measurements. These measurements typically assume fast water exchange conditions. The BOLus Enhanced Relaxation Overview (BOLERO) uses additional model parameters to allow the calculation of not only ECV but the intracellular lifetime of water (τ). The BOLERO method uses a three compartment model. Model parameters are fit using multiple blood and myocardial tissue T1 measurements. A recent study showed that water-exchange could result in a significant underestimate of ECV in hypertensive patients(1). We hypothesized that not only ECV differs between infarcted and viable myocardium, but water exchange also differs and could serve as a biomarker.

Materials and Methods: Twenty patients with chronic myocardial infarction underwent MR imaging at 1.5T with blood and myocardial T1 measurements before and after contrast administration for forty minutes. Plots of myocardial R1 vs blood plasma concentration were constructed for viable and infarcted myocardium for each patient. The BOLERO (2) method was used to fit these curves with model parameters: ECV, τ and longitudinal relaxation times of intracellular and extracellular water. Statistical tests were used to compare model parameters between infarcted and viable myocardium.

Results: A representative patient plot and BOLERO model curve fit are shown below. Viable tissue showed a linear fit and infarcted tissue a non linear shape with greater myocardial R1. On average, ECV was twice as large in infarcted tissue, 0.61 vs 0.31. Intracellular lifetime of water was on average, four times shorter in viable myocardium when compared to infarcted myocardium, 100 vs 408 ms. R1 inside of the cellular compartment was also significantly different, most likely due to the replacement of myocytes.



Catenary Model Used with vascular, extracellular and cellular compartments.



Data samples and model fits from a patient with a chronic myocardial infarct. Note the nonlinear curve shape with fibrosis. Black = infarct; Green = viable myocardium.

| | Viable | Infarct | p |
|-----------|--------|---------|--------|
| R1i (Hz) | 1.72 | 3.15 | <0.001 |
| tau (ms) | 99.5 | 408 | 0.008 |
| ECV | 0.31 | 0.61 | 0.001 |
| R1o0 (Hz) | 0.76 | 0.57 | 0.063 |

R1i = R1 inside cellular compartment, τ = H2O intracellular lifetime, ECV= extracellular volume fraction, R1o0= precontrast extracellular R1

Conclusions: Transcytolemmal water exchange rates differ between infarcted and viable myocardium. Viable myocardium can be assumed to be in the fast exchange limit. Infarcted myocardium has significantly slower exchange which alters the linearity of contrast curve shapes.

References: [1] Coelho-Filho OR et al. "Role of transcytolemmal water-exchange in magnetic resonance measurements of diffuse myocardial fibrosis in hypertensive heart disease." *Circ Cardiovasc Imaging* 2013; 6(1):134-41. [2] Yankeelov, TE et al. "Variation of the relaxographic "shutter-speed" for transcytolemmal water exchange affects the CR bolus-tracking curve shape." *Magn Res Med* 2003; 50:1151-1169.