# Effects of blood fraction and noise on a model-independent deconvolution method for estimating myocardial blood flow

**N. A. Pack[1,2], and E. V. DiBella[1,2]**

[1]Dept. of Bioengineering, University of Utah, Salt Lake City, Utah, United States, [2]Dept. of Radiology, University of Utah, Salt Lake City, Utah, United States

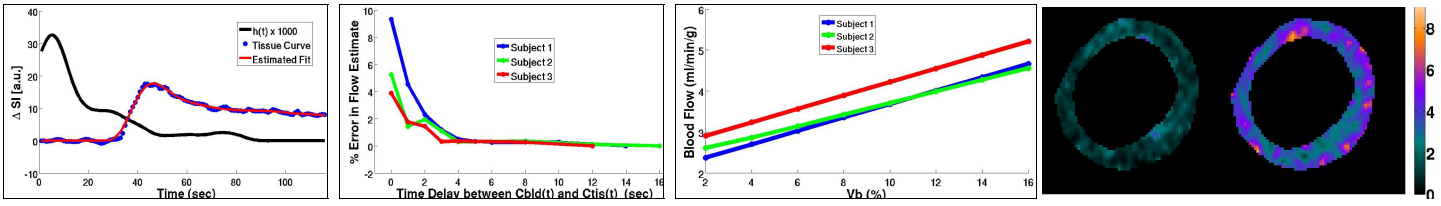
**Introduction:** Myocardial blood flow estimates from MRI perfusion studies have been reported using a number of different analysis methods [1,2,3]. Some analysis methods use tracer kinetic models that assume an exponential model for the system impulse response function, h(t). Other methods use model-independent deconvolution with B-splines [4] or other polynomials to parameterize h(t), which is directly related to blood flow [5]. While some models explicitly account for the delay time ($\Delta t$) between left ventricular (LV) blood pool enhancement and myocardial tissue enhancement and the inclusion of a vascular blood signal (Vb), model-independent analysis does not. The objective of this work was to investigate how variations in $\Delta t$ and Vb affect estimates of blood flow using a model-independent deconvolution method. Also, to evaluate the suitability of model-independent analysis with low SNR data, this method was tested using noisy, pixelwise dynamic MRI perfusion data.

**Methods:** Dynamic cardiac perfusion images were acquired with a Siemens Trio 3T MRI scanner using a TurboFLASH pulse sequence. Three short axis (SA) slices of the LV were imaged in five normal subjects at rest and adenosine (140 µg/kg/min) stress using a low-dose (~0.017 mmol/kg) bolus injection of Gd-BOPTA at 5cc/s followed by a saline flush. Typical imaging parameters were: TR=2.2ms, TE=1ms, flip angle=10-12˚, 8mm slice thickness, FOV=360x270 with GRAPPA (R=1.7). Offline, the MR images were manually registered for respiratory motion and the LV myocardium was segmented. Tissue enhancement curves, $C_{tis}(t)$, were obtained from six equi-angular regions in each SA slice of the LV and a blood enhancement curve, $C_{bld}(t)$, was obtained from a manually selected region in the LV blood pool. A model-independent deconvolution method, which uses iterative minimization and regularization [6], was used to estimate h(t) from the dynamic blood and tissue enhancement data. The cost function used in this process is shown in Eq (1). h(t) represents the system impulse response function and $\nabla$ represents a first-order gradient operator to penalize spurious oscillations in the estimate of h(t). $\lambda$ represents the regularization parameter and $\| \|$ denotes the Euclidean norm. Blood flow estimates were calculated from the maximum amplitude of h(t) in each region, scaled by the inverse of the sampling rate of the perfusion images.

$$\min_{h(t)} \left\{ \left\| C_{bld}(t) \otimes h(t) - C_{tis}(t) \right\|^2 + \lambda^2 \left\| \nabla h(t) \right\|^2 \right\} \quad (1)$$

The delay time between $C_{bld}(t)$ and $C_{tis}(t)$ was incrementally altered to evaluate how estimates of blood flow varied with changes in $\Delta t$ using model-independent analysis. Next, a simulated Vb signal was added to $C_{tis}(t)$ to investigate how changes in myocardial vasculature would affect estimates of blood flow. Finally, estimates of blood flow from pixelwise dynamic MRI perfusion data were compared to the regional estimates to determine how flow estimates vary as SNR decreases.

**Results:** Figure 1 shows a typical h(t) curve overlaid on the corresponding tissue enhancement curve (with the estimated fit). Figure 2 shows the percent error in blood flow estimates versus $\Delta t$ in one slice from three representative subjects in the study. For $\Delta t < 0$, the system is non-causal and the blood flow estimates do not have a sensible physiologic interpretation. For most values of $\Delta t \geq 0$, the maximum amplitude of h(t), and the estimates of blood flow, remain nearly constant. However, for small values of $\Delta t > 0$ or when blood and tissue enhancement occur nearly simultaneously, the blood flow was higher than the steady-state estimate by as much as 10%. Figure 3 shows a plot of blood flow estimates versus Vb in one slice from the same three subjects in the study. As the fraction of Vb within the myocardium increased from 2% to 16%, blood flow estimates increased linearly at a mean rate of 0.16 ml/min/g per 1% increase in Vb. Mean resting pixelwise blood flow estimates were 12 ± 4% higher than the mean 6-region flow estimates in four subjects. In the fifth subject, the mean flow estimate was 42% higher than the 6-region flow estimate. Figure 4 shows a pixelwise flow map at rest (left) and stress (right) in one SA slice from one subject in the study.




**(1) (2) (3) (4)**

**Figures from left to right: (1) A typical h(t) curve overlaid on the corresponding tissue enhancement curve and the estimated fit curve. (2) The percent error in blood flow estimates versus $\Delta t$ in one slice from three representative subjects in the study. (3) Blood flow estimates versus Vb in one slice from three representative subjects in the study. (4) Pixelwise flow maps (ml/min/g) at rest (left) and stress (right) in one slice of the LV from one subject in the study.**

**Discussion and Conclusion:** A model-independent deconvolution method, which uses iterative minimization and regularization, was evaluated to determine how changes in the delay time between LV blood pool enhancement and myocardial tissue enhancement, and variations in the vascular blood signal would affect estimates of myocardial blood flow from dynamic contrast-enhanced MR perfusion images. Blood flow estimates using this method correlated well with results from dynamic PET imaging ($r=0.83, n=5$) [6]. For a physiologic range of $\Delta t$, blood flow estimates using this model-independent method were nearly constant ($p<0.05$), provided the system was causal (ie. $\Delta t \geq 0$). However, for small values of $\Delta t$ or when blood and tissue enhancement occurred nearly simultaneously, the steady-state flow estimates were overestimated by as much as 10%.

Because model-independent analysis does not distinguish between extra-vascular and extra-cellular tissue space, and because it doesn't require knowledge about the local topology and permeability of the myocardial vasculature, it also doesn't discriminate between extra-vascular and intra-vascular perfusion. Subsequently, blood flow estimates are dependent on the fraction of Vb within the myocardium. Thus, regions of tissue with more vasculature will have greater tissue enhancement (and higher blood flow estimates) due to the higher [Gd] in the blood than in the extra-vascular, extra-cellular tissue. This also implies that with model-independent analysis, if Vb is higher at stress than at rest, the ratio of hyperemic to resting blood flow (ie. MPR) will be elevated in proportion to the increase in Vb. Perhaps estimates of "total" blood flow (extra-vascular and intra-vascular) may provide a more sensitive measure for differentiating ischemic tissue from healthy tissue in perfusion analyses. While pixelwise blood flow estimates were significantly different than those from 6-region analysis, which assumes regional uniformity, they may still be useful for localizing small regions of low blood flow near the endo- and epi-cardial borders of the LV.

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**References:** [1] Jerosch-Herold M et al. Int J Card Imaging 1997;13:205-218. [2] Larsson HB et al. Magn Reson Med 1996;35:716-726. [3] Jerosch-Herold M et al. Med Phys 2002;29:886-897. [4] Jerosch-Herold M et al. Med Phys 2002;29:886-897. [5] Zierler K Circ Res1962;10:393-407. [6] Pack NP submitted to SCMR 2008.