Improved Quantitative Cardiac Perfusion in High CA Dose MR First Pass: Patient Study

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Introduction: First pass perfusion imaging was shown to be a robust method for qualitative perfusion analysis and for quantitative perfusion reserve evaluation. Commonly CA dose is limited to low dose, to assure that acquired signal shows a linear dependency on CA concentration. Dependency of signal on tissue and blood native T_1 is neglected, despite the fact, that it differs in myocardium itself and blood pool, and between different first pass experiments in a single protocol due to remaining CA (which shortens T_1 significantly). Purpose of this work was to develop a model for quantitative CA concentration evaluation from first pass data, since common perfusion models are based on tracer kinetics and require tracer concentration as input rather than acquired signal. Model output can be used for quantitative perfusion analysis independent of tissue and blood T_1, and it even corrects for local varying coil sensitivity. Range of CA concentrations described by this model extends those of linear approximation. Feasibility of this model was shown in simulations and in measurements on patient. It offers the possibility to inject a 3-fold higher CA dose for quantitative perfusion analysis. This method was compared to evaluation based on signal intensity.

Methods: CA concentration $c_{calc}(s)$ is derived from signal intensity $s(c_{bolus})$ from T_1-weighted imaging experiment is described in our approach phenomenologically by

$$c_{calc}(s) = ([\exp\{s(c_{bolus})*p/s(c_{bolus}=0)\} * \exp\{-p\}] - 1) * h$$

with $p = 1 / (b*T_1 - a)$ and

(a) Parameters a, b, h depend on measurement parameters derived from simulation or on an additional calibration measurement.
(b) Quantitative T_1 of tissue and blood pool has to be measured before each CA experiment.
(c) Baseline intensity $s(c_{bolus}=0)$ is the apparent signal intensity of precontrast images.
First pass signal was acquired three times with a saturation recovery Snapshot FLASH sequence (T_I=10ms, T_R=2.4ms, T_E=1.1ms, FA=18°, 10 ml Gd-DTPA bolus) in patient with global reduced perfusion (ischemic cardiomyopathy) (*fig.*2). Three datasets were acquired and evaluated with the new model. Two of these were obtained in the resting condition, but greatly different T1 due to remaining CA from previous experiment, and one during Adenosine induced stress. Quantitative T_1 was measured before each first pass in all slices using a fast inversion recovery Snapshot Flash sequence. Stroke volume was also determined. Regional perfusion from six segments in three slices was derived from deconvolution with the Fermi model as a representative deconvolution model. Same data were evaluated additionally with a similar signal intensity based evaluation.

Results: Quantitative perfusion derived from both first pass datasets under same resting vasodynamic state with the proposed model was determined {with signal based evaluation} to be 0.56(±0.12) {0.73(±0.18)} [ml/g/min] with no remaining CA from previous first pass and 0.57(±0.09) {0.92(±0.28)} [ml/g/min] with remaining CA. Perfusion under adenosine induced stress based on the shown model was determined to be 1.63(±0.40) {2.25(±0.62)} [ml/g/min] (*fig.*1). Perfusion reserve from rest with no remaining CA / stress firstpass was calculated with new model to be a factor of 2.82(±0.41) {3.17(±0.52)}. Perfusion reserve from both rest and stress with remaining CA with new model was a factor of 2.86(±0.64) {2.57(±0.30)}. Evaluation from single segments does show similar behaviour to the averaged perfusion. This general behaviour of perfusion overestimation in signal based evaluation was also observed in other patients than the example shown here. Total amount of CA passing the left ventricle (LV) was determined from the quantitative CA concentration time curve. Scaled by stroke volume (67 ml) this was 7.8(±0.2) ml and maximum CA concentration in LV was determined for rest / stress / rest first pass to be 3.7 / 4.9 / 4.2 [mmol/l], respectively.

Conclusion: The proposed exponential model yields a correction for non-linear dependency of signal and CA concentration and for both native T_1 and coil sensitivity. It offers quantitative perfusion evaluation in the high CA dose regime, resulting in increased CNR.

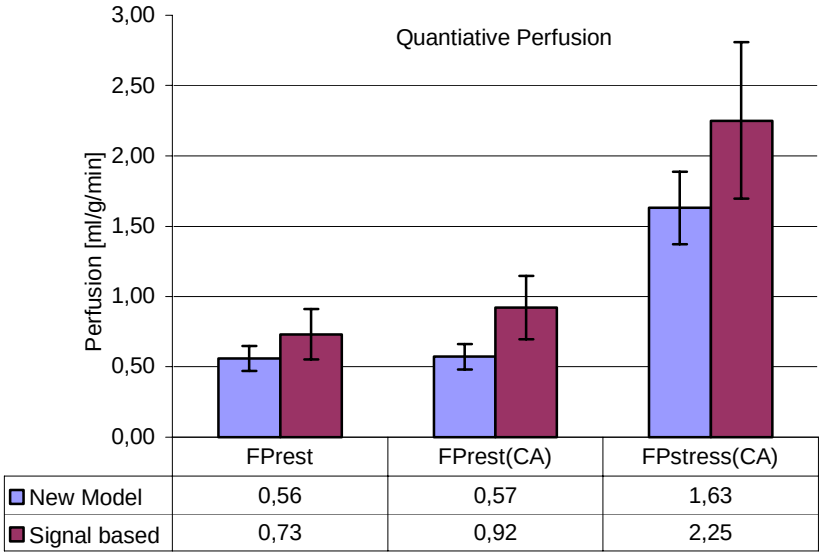

*Figure 1:*Quantitative perfusion from three first pass datasets. In second rest and stress experiment T_1 was reduced due to remaining CA.(exemplarily in one patient).