

WALKING POSTER PRESENTATION

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An instantaneous ECV with no blood sampling: using native blood T1 for hematocrit is as good as standard ECV

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Background

The extracellular volume fraction (ECV) by T1 mapping measures the size of the myocardial interstitium. T1 changes in blood and myocardium are used to measure the contrast partition coefficient (λ), and substituting in the blood volume of distribution (directly measured on a peripheral blood sample as one minus the hematocrit [Hct]) provides the ECV. This methodology is however cumbersome, has significant variability, introduces a delay and is a barrier to wider use of ECV quantification in clinical practice. We have previously observed a strong relationship between ShMOLLI T1_{blood} and Hct [Piechnik, JCMR 2013, 15:13] and hypothesise that this could be used to infer the Hct at the time of scan and permit immediate ECV calculation without blood sampling (ECV_{No Hct}).

Methods

350 subjects (age 61±15 years; 47% male; 36 healthy volunteers, 95 severe aortic stenosis, 95 with a history of anthracycline chemotherapy, 46 hypertrophic cardiomyopathy, and 78 cardiac amyloidosis) underwent T1 mapping with ShMOLLI at 1.5T (Siemens Avanto) prior to and at 15 minutes after administration of 0.1mmol/kg of Dotarem. Venous blood for Hct was obtained prior to scanning. The partition coefficient $\lambda = (\Delta[1/T1_{myo}] / \Delta[1/T1_{blood}])$ and ECV_{Hct} = λ * [1-haematocrit]) were calculated. Hct was approximated from the linear relationship with native T1_{blood} and used to calculate ECV_{No Hct}. This

was then compared to the conventional ECV $_{\text{Hct}}$, partition coefficient and post-contrast $T1_{myocardium}$.

Results

There was strong correlation between ShMOLLI T1_{blood} and Hct across health and disease with a coefficient of explained variation R²=0.50 (p<0.001; Figure 1), i.e. 50% variability of native T1_{blood} apportioned to the Hct. The broad array of cardiac pathologies provided a wide range of Hct (40.0±3.6%; range 28-51%) and native T1_{blood} (1557±81ms; range 1368-1834ms), with similar correlations of Hct versus T1_{blood} in each group. The regression equation was: Hct = 0.9 - (T1_{blood} / 3333).

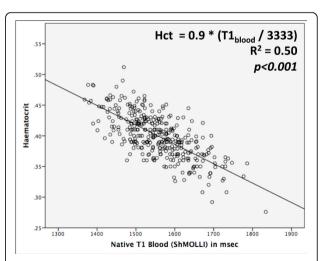


Figure 1 Correlation between hematocrit and native T1_{blood}.

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Table 1 Correlations between ECV with/without hematocrit, partition coefficient, post contrast T1 myocardium and clinical parameters.

	ECV with Hct	ECV no Hct	Partition Coefficient	Post contrast T1 myocardium
Indexed LV mass	0.48*	0.48*	0.48*	-0.33*
Indexed LA area	0.33*	0.34*	0.33*	-0.30*
LVEF, %	-0.53*	-0.55*	-0.54*	0.34*
Indexed Stroke Volume	-0.46*	-0.47*	-0.48*	0.48*
NT-pro-BNP	0.51*	0.52*	0.48*	-0.34*

^{*}p<0.01

Derived ECV $_{\mathrm{No\ Hct}}$ exhibited excellent correlation with conventional ECV $_{\mathrm{Hct}}$ (R 2 =0.99; p<0.001) with small ~2% bias and ~3% SD of differences on Bland-Altman analysis (95% confidence interval -0.7 to +3.9% excluding Amyloid, and -2.6 to +8.0% for Amyloid) close to previously reported 1.4% [Schelbert EB JCMR 2011, 13:16].

 $ECV_{No\ Hct}$ correlated equally well with clinical markers of disease severity (LV mass index, LVEF, stroke volume index, left atrial area index and NT-pro-BNP) as ECV_{Hct} and partition coefficient, and better than post-contrast $T1_{mvocardium}$ (Table 1).

Conclusions

Native $T1_{blood}$ correlates well with the laboratory-measured values of hematocrit. Our data demonstrates that straight-forward derivation of hematocrit from $T1_{blood}$ can be used as an immediate measure of ECV that may pave its application for nearly instantaneous clinical diagnosis. It remains to be confirmed if the high correlation of $ECV_{No\ Hct}$ with the conventional calculations may cause blood sampling to become an obsolete complication in clinical practice.

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