WALKING POSTER PRESENTATION

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 (\( \lambda \)), 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 T1blood 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 (ECVNo 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 \( \lambda = (\Delta[1/T1_{\text{myo}}] / \Delta[1/ T1_{\text{blood}}]) \) and ECV_{Hct} = \( \lambda \times [1-\text{haematocrit}] \)) were calculated. Hct was approximated from the linear relationship with native T1blood and used to calculate ECVNo Hct. This was then compared to the conventional ECV_{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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Derived ECV_noHct exhibited excellent correlation with conventional ECV_Hct ($R^2=0.99$; $p<0.001$) with small $\sim2\%$ bias and $\sim3\%$ 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_noHct 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 myocardium (Table 1).

**Conclusions**

Native T1blood correlates well with the laboratory-measured values of hematocrit. Our data demonstrates that straight-forward derivation of hematocrit from T1blood 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_noHct with the conventional calculations may cause blood sampling to become an obsolete complication in clinical practice.

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