HCM 572±176, normal 745±138 p=0.01; exercise HCM 648±191, normal 845±160 p=0.02). There was a significant correlation between PFR and PCr/ATP at both rest (rs=0.78, p=0.001) and exercise (rs=0.54, p=0.039). There was significantly reduced BOLD SIΔ response in HCM (10±11% vs normal, 18±14% and athletes 17±10%, p<0.0001) as well as MPRI (normal: 1.8±0.6; athletes: 2.0±0.9, HCM 1.3±0.6, p=0.001). There was a weak but significant correlation between BOLD SIΔ and MPRI (R=0.27, p<0.0001) and between BOLD SIΔ and end diastolic wall thickness (R=0.24, p<0.001). MPRI (β 0.2, p<0.001) and wall thickness (β −0.2, <0.001) are independent predictors of BOLD SIΔ. For β myosin heavy chain mutation cohort (n=12), there was a significant relationship between change in PCr/ATP and either BOLD SIΔ (R=0.48, p=0.05).

**Conclusion** During exercise, the pre-existing energetic deficit in HCM is further exacerbated, independent of hypertrophy. Additionally, oxygenation is blunted during stress. This may lead to acute derangement of energy dependent ion homeostasis during acute stress, resulting in ventricular arrhythmias. We offer a possible explanation for the high incidence of exercise related death in HCM and suggest that treatments that optimise energetics may be protective.

## 082 RIGHT VENTRICULAR HYPERTROPHY AND THE ATHLETE'S HEART: UTILITY OF THE ECG AS A SCREENING TOOL

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**Introduction** Right ventricular hypertrophy (RVH) is a manifestation of various congenital and acquired cardiopulmonary disorders which may lead to premature morbidity and mortality. Physiological RVH is also reported among healthy athletes. European (ESC) guidelines define ECG markers of RVH in young athletes as "uncommon and training-unrelated," warranting further investigation to exclude "pathological RV dilatation or hypertrophy." Conversely, recent American guidelines state that evidence is lacking to support such a strategy. There have been no studies to correlate ECG markers of RVH with imaging data in young athletes.

**Methods** 214 asymptomatic, elite athletes underwent ECG and transthoracic echocardiography. Sensitivity and specificity, as well as positive and negative predictive values (PPV and NPV) of published ECG criteria for RVH were assessed against echo findings (see Abstract 082 table 1). RV free wall thickness (RVWT) was measured in the subcostal plane as per ESC recommendations. RV end-diastolic area (RVEDA) was also calculated in each case.

Abstract 082 Table 1

| | Prevalence (%) | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) |
|---|---|---|---|---|---|
| R:S(V1) >1 | 3.7 | 0.0 | 95.1 | 0.0 | 96.6 |
| R:S(V5/V6) <1 | 1.4 | 0.0 | 98.6 | 0.0 | 96.7 |
| R(V1) >7 mm | 8.9 | 14.3 | 91.9 | 5.3 | 97.1 |
| R(V1) + S(V5/V6) >10.5 mm | 14.5 | 28.6 | 86.0 | 6.5 | 97.3 |
| R′(V1) >10 mm | 0.5 | 0.0 | 99.5 | 0.0 | 96.7 |
| qR(V1) | 0.0 | 0.0 | 100.0 | 0.0 | 96.7 |
| Right axis deviation (>110°) | 1.9 | 0.0 | 98.1 | 0.0 | 96.7 |
| Right atrial enlargement (P-wave >2.5 mm) | 0.9 | 0.0 | 99.0 | 0.0 | 96.7 |

**Results** Mean age was 21.4 years, 76.7% male. Mean RVWT was 3.8 mm (range 2–6 mm). Only 7/214 (3.3%) of athletes, all male, demonstrated RVH on echo (RVWT ≥6 mm). Inter- and intra-observer variability for RVWT measurements were 10% and 14% respectively. All ECG criteria for RVH had low sensitivity and PPV for echocardiographic RVH, although specificity and NPV were high. The Sokolow-Lyon voltage criterion for RVH (R(V1) + S(V5/6) >10.5 mm), which is specifically mentioned in the ESC guidelines, was seen in 14% of athletes. Mean RVEDA did not differ between athletes with RVH on ECG and those without (both groups 27.3 $cm^2$, p=1.0).

**Conclusions** Published ECG criteria have an unacceptably low correlation with echo evidence of RVH, which is rare in athletes. Adherence to current ESC guidelines would result in a large number of additional investigations, with the potential for undue distress, disruption to training, and inappropriate resource utilisation. Our data support American guidance that RVH voltage criteria violations should not prompt further investigation, which may have significant implications for the burden of testing required after ECG screening of British athletes.

## 083 ANNULO-APICAL ANGLES AND TAPSE TO RAPIDLY ASSESS RIGHT VENTRICULAR SYSTOLIC FUNCTION: A CARDIAC MAGNETIC RESONANCE STUDY

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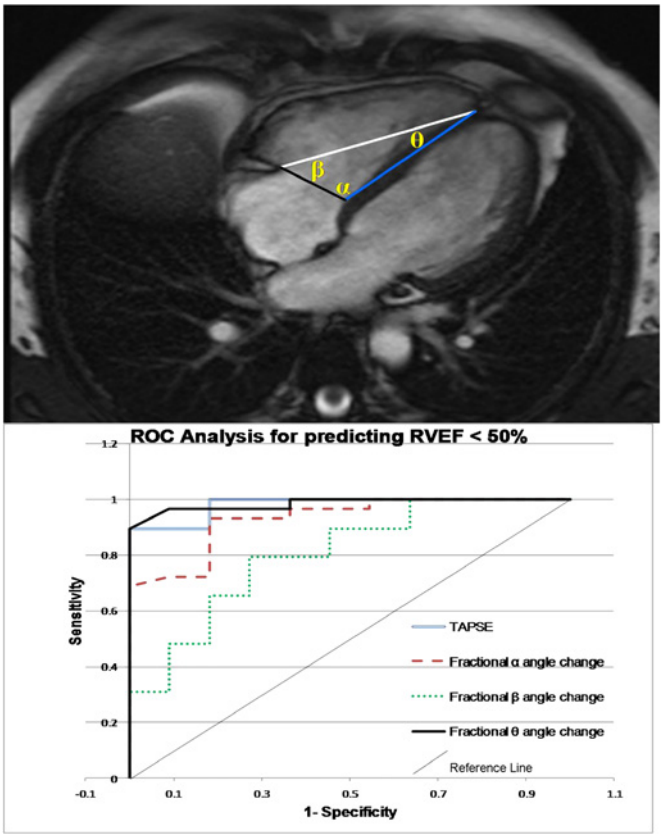
**Background** Volumetric assessment of the right ventricle (RV) by Cardiac Magnetic Resonance (CMR), albeit time-consuming, provides accurate and reproducible measurement of RV ejection fraction (RVEF). Tricuspid annulus peak systolic excursion (TAPSE)




Abstract 083 Figure 1 Top: AAAs in ED on a 4 chamber view. Bottom: ROC curve analysis.

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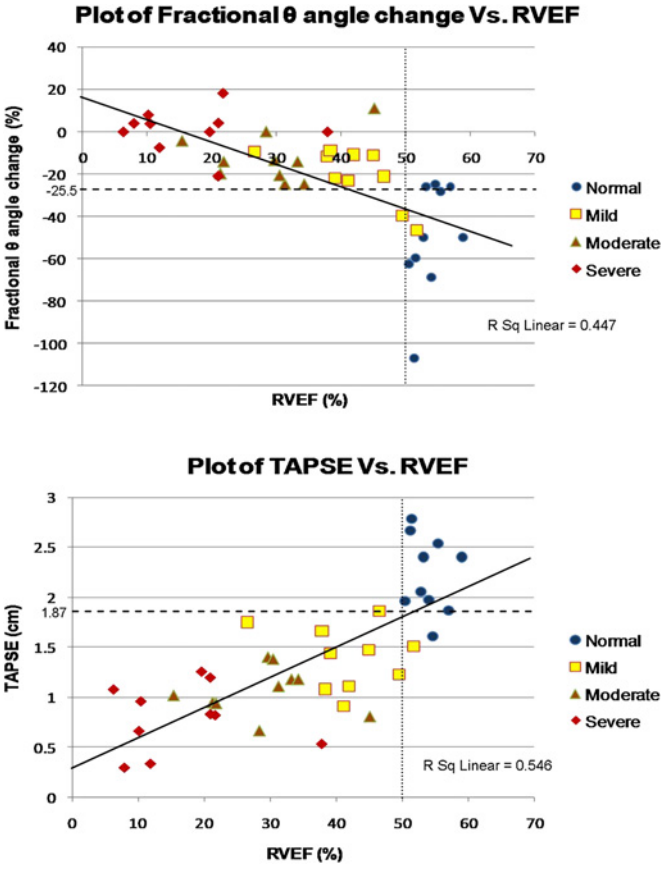
is a predominantly Echo-validated rapidly—derived surrogate of RV function. Correlations between RVEF and systolic changes in annulo-apical angles (AAAs) have not previously been evaluated.

**Objective** To assess the use of changes in AAAs and TAPSE as rapidly-derived surrogate markers of RV systolic function using CMR.

**Methods** We measured RV volumes from short-axis bSSFP stacks in patients undergoing clinically indicated CMR scans. RVEF was calculated from volumes derived by semi-automated endocardial contouring (QMass®MR 7.2). AAAs (α, β, θ angles—see Abstract 083 figure 1), subtended by a triangle connecting the medial and lateral extent of the tricuspid valve annulus and RV apex, and fractional changes in AAAs (ΔAAA/EDAAA×100, whereby ΔAAA-=EDAAA−ESAAA) were measured from end-diastolic (ED) and end-systolic (ES) 4chamber SSFP cine still frames. TAPSE was measured as the change in length of a line connecting the lateral tricuspid valve annulus with the RV apex from ED to ES. Parameters were compared with RVEF using Spearman rank correlations; ROC curves constructed to assess accuracy of the parameters in predicting an RVEF<50%.

**Results** 40 subjects were included: 10 normals, 10 mildly-impaired, 10 moderately-impaired, and 10 with severely-impaired RV systolic function. Median (25th−75th percentile) RVEF for each subgroup was 53.5% (51.4%−55.7%), 41.5% (38.1%−47.2%), 30.0% (21.7%−33.5%), and 15.8% (9.6%−21.2%), respectively. Correlations with RVEF: TAPSE (0.74, p<0.001), fractional changes of α angle (0.64, p<0.001), β angle (−0.39, p<0.05), and θ angle, which had the highest correlation (−0.77, p<0.001). Smaller increases or a decrease in magnitude of the θ angle from ED to ES are associated with lower RVEFs, whereby a fractional θ angle change of ≥ −25.5% predicts RVEF<50% [97% sensitivity, 91% specificity, AUC=0.98]. The cut-off for TAPSE is ≤1.87 cm [100% sensitivity, 82% specificity, AUC=0.98]. Intra- and inter-observer reproducibility is excellent as shown by intra-class correlation coefficients for TAPSE (0.98 and 0.87, respectively) and fractional θ angle change (0.96 and 0.94, respectively).

**Conclusion** Both fractional θ angle change and TAPSE strongly correlate with RVEF, and are accurate predictors of RVEF<50%. These measurements provide an excellent alternative to the more time-consuming derivation of RVEF obtained volumetrically by endocardial chamber tracing.




Abstract 083 Figure 2 Scatter graphs for fractional θ angle change and TAPSE, both plotted against RVEF. Dotted vertical lines represent the ROC cut-offs of RVEF<50%. Dashed horizontal lines represent cut-offs of ≥ −25.5% and ≤1.87 cm for fractional θ angle change and TAPSE, respectively.

## 084 IN VIVO ASSESSMENT OF CELLULAR INFLAMMATION FOLLOWING ACUTE MYOCARDIAL INFARCTION

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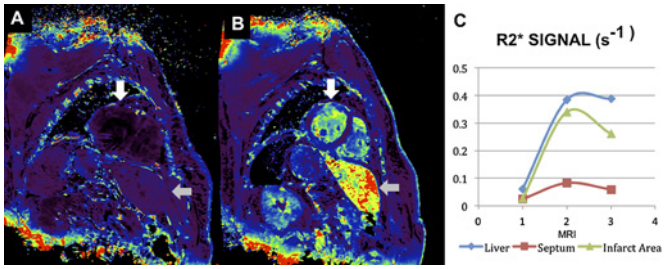
**Background** Inflammation following myocardial infarction has detrimental effects on reperfusion, myocardial remodelling and left ventricular function. MRI using ultrasmall superparamagnetic particles of iron oxide (USPIO) can be used to detect cellular inflammation in tissues.

**Methods** 15 patients were recruited up to 5 days after ST-segment elevation myocardial infarction. Nine patients underwent cardiac MRI (3 Tesla) at baseline, and at 24 and 48 h following infusion of USPIO (4 mg/kg; Ferumoxytol, AMAG). Six control patients underwent the same scanning protocol without infusion of USPIO. T2*-weighted multi-gradient-echo sequences were acquired and R2* maps (inverse of T2*) were generated to assess USPIO accumulation. Baseline scans were registered to subsequent 24 and 48 h scans and the infarct zone was defined on Gadolinium-enhanced T2-weighted images. An "object map" was created that defined corresponding regions of interest (ROI) on all scans for each subject. The ROIs included infarct zone, peri-infarct zone, remote myocardium, liver, blood pool and skeletal muscle. The R2* values for each ROI was calculated.

**Results** In the control group, the R2* value in the infarct zone remained constant: baseline, 0.047 $s^{-1}$ (95% CI 0.034 to 0.059); 24 h, 0.043 $s^{-1}$ (95% CI 0.035 to 0.052) and 48 h, 0.040 $s^{-1}$ (95% CI 0.024




Abstract 084 Figure 1 In this subject, late gadolinium enhancement had revealed an infarct of the anterior left ventricular wall. Panels A and B are R2 acquisition images of the same subject taken on day 1 (A, pre-USPIO), and day 2 (B post-USPIO) in a patient given ferumoxytol. The white arrow indicates the area of infarction corresponding to the late gadolinium enhancement. In this area there is sequential higher uptake of USPIO as indicated by the red/green colour in this area. This is consistent with neutrophil and macrophage influx. Ferumoxytol is also taken up by the liver reticulo-endothelial system (grey arrow). These findings are confirmed by the quantitative analysis of the R2* signal (Panel C).

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