would not be recommended and may be considered in the other three.

Conclusion The current ESC scoring system potentially leaves many high-risk patients unprotected or with ambiguous ICD implant guidance. Lowering the current threshold may improve accuracy.

Imaging

146 CONTRIBUTION OF CONDUCTION AND REPOLARISATION ABNORMALITIES TO THE TYPE I BRUGADA PATTERN: A STUDY USING NON-INVASIVE ELECTROCARDIOGRAPHIC IMAGING

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Introduction In Brugada Syndrome (BrS), the substrate location and underlying electrophysiological mechanisms that contribute to the characteristic ECG pattern are still debated. Using non-invasive electrocardiographical imaging (ECGi), we study whole heart conduction and repolarisation patterns following an ajmaline challenge in individuals with concealed Type I BrS.

Methods 13 participants (mean age 44 ± 12 yrs; 8 males), 11 concealed Type I BrS and 2 controls, underwent an Ajmaline infusion with ECGI and ECG recordings for a research study. ECGi technology reconstructs >1000 electrograms (EGMs) from 252 surface electrode vest and projects this mathematically onto a 3D cardiac image created using a CT scan. Activation time points were determined as the QRS (dP/dtmin) and repolarisation time as (dP/dtmax) for positive T waves and (dp/dtmin) for negative or biphasic T waves, annotated using a custom built semi-automated software off-line. From these data, the local activation recovery interval (ARI), a surrogate of action potential duration, and activation timings across the right ventricle (RV) body, out flow tract (RVOT), and left ventricle (LV) were computed for all participants (Figure 1a). Changes in AT timings and ARI across the RVOT, RV and LV with ajmaline were calculated, and correlated with peak ST elevation (STE) derived from the ECG at the same time point.

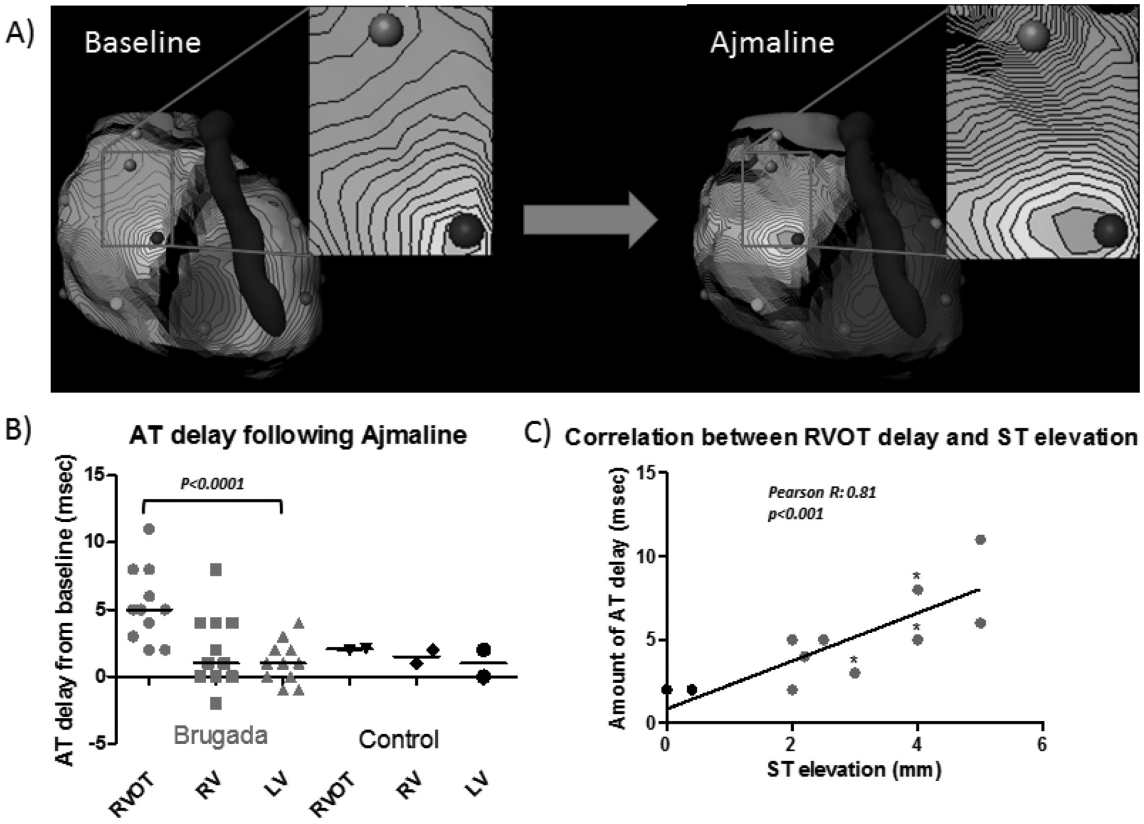
Results Following ajmaline administration, the greatest median increase in conduction delay was noted in the RVOT than in the RV or LV (5[3–8] ms vs 1[0–4]ms vs 1[0–2] ms; $p < 0.0001$) (FigURE 1b). Prolongation of ARI was also observed to have increased the most in the RVOT (68[53–99] ms vs 35 [23–46] ms vs 25[9–30] ms; $p < 0.01$). In the two control patients, no STE was noted with minimal rise in conduction delay or ARI prolongation noted in the RVOT, RV and LV. Only conduction delay in RVOT with ajmaline correlated to amount of STE (Pearson R 0.8, $p < 0.001$) (Figure 1c), but not in the RV or LV (Pearson 0.3 and 0.2 respectively; p=ns). No significant correlation was also seen between STE and ARI prolongation in the RVOT, RV or LV (Pearson 0.5, 0.4, 0.1 respectively; p=ns).

Conclusion Magnitude of STE in the Type I BrS pattern is attributed to degree of conduction delay in the RVOT and not prolongation in repolarisation time.

Abstract 145 Table 1 Summary of risk factors in (primary prevention) patients with and without ICD therapy. Mean ± SD and proportion shown

	Appropriate therapy (n=8)	No therapy (n=44)	
ESC 5 year risk score (%)	4.79 ±1.5	5.37 ±3.3	p=ns
Average no. of risk markers (ACC/AHA)	2	1.6	
Age at implant (yrs)	51 ±13	47 ±13	
LVH (mm)	23±7	24±6	
LVOT gradient (mmHg)	8 ±3	28 ±32	
LA diameter (mm)	44 ±4	43 ±8	
Non-sustained VT	6/8 (75%)	30/44 (68%)	
Unexplained syncope	0/8 (0%)	8/44 (18%)	
Family history of SD	3/8 (38%)	13/44 (30%)	
Family history of multiple (≥2) SD in 1st and 2nd degree relative	2/8 (25%)	3/44 (7%)	
Abnormal BP to exercise	3/5 (60%)	9/32 (28%)	
Massive LVH (≥35mm)	1/8 (12.5%)	3/44 (6.8%)	
AF	2/8 (25%)	9/44(20%)	
Follow up period (years)	6.5 ± 0.5	6.2 ± 5.3	

ESC –– European Society of cardiology; ACC – American college of cardiology; AHA – American Heart Association; LVH – Left Ventricular Hypertrophy; LVOT – Left Ventricular Outflow Obstruction; LA – left atrium; VT – ventricular tachycardia; SD – sudden death; BP – blood pressure; AF – atrial fibrillation)



Abstract 146 Figure 1 a) Isochronal crowding seen RVOT following ajmailne, and measurement of activation times across region b) Activation time (AT) delay across the different regions. c) Correlation of RVOT conduction delay with ST elevation on ECG. Black denotes control and red denotes Brugada participants

Valve Disease/Pericardial Disease/ Cardiomyopathy

147 ELEVATED SERUM TROPONIN I IS ASSOCIATED WITH INCREASED RISK IN HCM

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The European Society of Cardiology recently recommended a new tool[1] to estimate 5 year risk of sudden cardiac death (SCD) in patients with hypertrophic cardiomyopathy (HCM). We investigated the relationship between serum cardiac troponin I (cTnI) levels and HCM-risk score in 100 consecutive patients referred to the West of Scotland Inherited Cardiac Conditions Clinic. The 20 most recent patients had a high sensitivity cTnI assay performed (limit of detection (LOD) 1.2 ng/L) and the remaining 80 had the traditional assay (LOD 10 ng/L). Demographic, clinical, genetic and imaging parameters were collected at first assessment. HCM-risk was calculated retrospectively.

Cardiac TnI was elevated in 27% of the population (n = 100, 60% male, mean age 56 ± 14, left ventricular outflow tract (LVOT) obstruction (i.e. resting gradient ≥30 mmHg) in 20%) and they had significantly higher overall HCM-risk score (3.7% v 2.2%, $p < 0.01$). Of the risk tool's component variables, an elevated cTnI was associated with increased left atrial diameter (50 ± 8 v 42 ± 8 mm, $p < 0.01$) and raised maximum LVOT gradient (33 ± 38 v 19 ± 24 mmHg, $p < 0.03$), but not with maximal wall thickness, family history of SCD, the presence of non-sustained ventricular tachycardia, history of syncope, or age at clinical evaluation. Of non-tool variables, an elevated cTnI was associated with history of atrial fibrillation (37% v 14%, $p < 0.01$) and heart failure (22% v 3%, $p < 0.01$). Finally, in a sub-group (n = 49) of patients who underwent cardiac magnetic resonance imaging, patients with an elevated cTnI were more likely to have late gadolinium enhancement (92% v 38%, $p < 0.01$).

In conclusion, serum cTnI is elevated in a significant proportion of patients with HCM and is associated with clinical markers of disease severity. Biomarkers may be useful as an adjunct to current risk models in identifying patients with adverse cardiac remodelling and underlying atrial fibrillation.

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148 THE COST EFFECTIVENESS OF SCREENING YOUNG ATHLETES WITH ECG IN THE UK

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Introduction High false positive rates and subsequent costs of additional investigations provide major obstacles to state-sponsored screening of young athletes for cardiac disease with