Abstract 10 Figure 2 Correlation scatterplot of LVAs $\leq 0.2\text{mV}$

Figure 2: Correlation coefficients of anatomical segment at voltages consistent with dense scar $\leq 0.2\text{mV}$ (in mm^2 of endocardium fitting that voltage category) between rhythms of SR and AF.

significance in the Posterior (AF = $234 \text{ mm}^2 \pm 150.45$; SR = $138.27 \text{ mm}^2 \pm 112.3$, $p = <0.001$), Roof (AF = $150.61 \text{ mm}^2 \pm 93.17$; SR = $115 \text{ mm}^2 \pm 77.14$, $p = 0.01$), Anterior (AF = $220.87 \text{ mm}^2 \pm 173.07$; SR = $158.53 \text{ mm}^2 \pm 99.22$, $p = 0.002$) and Lateral AS (AF = $137.05 \text{ mm}^2 \pm 104.99$; SR = $87.52 \text{ mm}^2 \pm 66.82$, $p = <0.001$) as seen in table 1. The voltage range of $0.21\text{-}0.5\text{mV}$ demonstrated weak correlation coefficients of $R=0.09\text{-}0.31$, only reaching significance on the Posterior AS, once again shown in table 1.

Conclusions LVA burden $0.21\text{-}0.5\text{mV}$ in AF is significantly higher in SR with poor correlation, suggesting a greater influence of rhythm in this range and so care must be taken in interpretation. Comparatively, at dense scar voltages ($\leq 0.2 \text{ mV}$), we found comparable burden with good correlation between AF and SR, reflecting true readings in both rhythms. These findings should be carefully considered by operators performing additional scar modification and ablation.

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CLINICAL ASSOCIATIONS AND PATHOLOGICAL MECHANISMS OF REDUCED ATRIAL CONDUCTION VELOCITY

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Background The complex pathogenesis of atrial fibrillation (AF) remains unclear. A key concept is that for AF to be sustained, both a trigger and an atrial substrate capable of perpetuating the arrhythmia are required. Pathological remodelling in the atria can be observed after only a few minutes of high atrial rate, with the chronicity of these changes being directly related to the duration of the episodes of high atrial rate. This underpins the often-quoted axiom 'AF begets AF'. Increased atrial fibrosis in patients with AF has been observed in both histological and non-invasive studies using LGE-CMR atrial imaging. Reduced atrial conduction velocity (CV) has been observed in fibrotic areas, which may propagate AF as more wavefronts can be sustained in the atria. It is unclear if reduced atrial CV is a prerequisite for AF, or if it develops after the initial periods of arrhythmia. The use of 3D electro-anatomical mapping (EAM) has led to advancement in the accuracy of intra-cardiac CV measurement and investigations into the mechanism for reduced CV.

Hypothesis We hypothesised that left atrial CV plays a key role in the AF disease process and investigation into atrial CV may improve understanding into the pathogenesis and clinical course of AF. **METHODS** A systematic review was performed to assess current published evidence. This informed the design of a retrospective study to calculate CV from the left atrial

local activation time (LAT) electro-anatomical maps of 90 paroxysmal AF (PAF) and 90 persistent AF (PsAF) patients presenting for first time PVI. Following this, pathological mechanisms were assessed by looking at the associations between left atrial CV determined from EAMs and 3D atrial LGE-CMR in 96 patients. Finally, a pilot study was designed and undertaken to investigate if atrial CV at rest, and at varying heart rates with exercise can be measured non-invasively with the use of electrocardiographic imaging (ECGI) and CMR.

Results There was no statistical difference in the left atrial CV of patients with PAF compared to PsAF (0.68 m/s vs 0.69 m/s respectively, p -value = 0.70). Left atrial CV did not predict recurrence of AF in the first year after PVI. (Figure 1) Regional analysis of 3D atrial LGE-CMR in 96 patients, found significant variation in the distribution of fibrosis, however fibrosis burden was not significantly correlated with either overall or regional CV. (Figure 2). A pilot study using ECGI with CMR imaging was able to non-invasively measure bi-atrial CV during rest and three exercise stages. CV restitution properties

	Fibrosis burden (% of total area)	Conduction Velocity (m/s)	Correlation P-Value
Anterior	28.6 ± 23.5	0.731 ± 0.18	0.57
Lateral	27.6 ± 22.0	0.786 ± 0.18	0.34
Posterior	35.6 ± 24.6	0.735 ± 0.16	0.42
Septal	27.6 ± 21.8	0.793 ± 0.18	0.87
Roof	20.0 ± 21.2	0.832 ± 0.25	0.35

Abstract 11 Figure 1 AF recurrence survival analysis

Abstract 11 Figure 2 Regional variation in left atrial scar and correlation with CV
(A = Anterior LA view, B = Posterior LA view)

were not demonstrated in this healthy cohort in heart rates up to 150bpm.

Conclusions Reduced atrial CV is a common finding in AF, however CV is similar in patients with PAF and PsAF in sinus rhythm during PVI. Alterations in CV did not correspond to fibrosis detected on atrial LGE-CMR, suggesting that pathological changes other than fibrotic remodelling account for the reduction in CV seen in these patients.

12 COMPARISON OF SLOW VS. RAPID AJMALINE INFUSION PROTOCOL FOR THE DIAGNOSIS OF BRUGADA SYNDROME (BrS): TEN YEAR EXPERIENCE OF THE NORTHERN IRELAND INHERITED CARDIAC CONDITIONS SERVICE (NI ICC)

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Introduction BrS is diagnosed in patients with ST-segment elevation with type 1 morphology ≥ 2 mm in one or more leads among the right precordial leads V1 and/or V2 positioned in the second, third or fourth intercostal space. This may be spontaneous or after provocation challenge with intravenous administration of a sodium channel blocker (i.e. ajmaline, flecainide, procainamide or pilsicainide). The specific protocol for ajmaline provocation challenge for diagnosis of BrS has been debated between ICC services worldwide. Concerns regarding safety and false positive rates are perceived to be associated with a more rapid infusion protocol. This retrospective observational cohort study describes the safety and positivity rates for patients undergoing ajmaline provocation challenge by both protocols over ten years.

Methods Data on consecutive adults undergoing ajmaline challenge test from March 2011 to March 2021 were retrospectively collected. Patient demographics, indication for testing, genetic information, adverse events and positivity rates were compared by test protocol used. Slow protocol was defined as total dose of 1mg/kg ajmaline capped at 100mg, given at rate of 10mg/min. Rapid protocol was defined as 1mg/kg ajmaline capped at 100mg given in 10 divided doses over 5 minutes.

Results A total of 414 ajmaline challenges were included (251 (61%) slow vs. 163 (39%) rapid protocol) [Mean age 41 ± 16 yrs; 50% male]. Indications for conducting the test were a) family history of BrS 182 (44%), b) family history of SADS/SUD 138 (33%), c) OOHCA 26 (6%), d) abnormal ECG 47 (11%) and e) syncope 21 (5%). There was no difference in positivity rate between the two protocols (slow (23%) vs. rapid protocol (17%), $p=0.13$) [table 1]. Comparison of patients by provocation result regardless of the protocol used

showed a predominance of (males, 51% in both groups, $p=0.96$) and (similar mean age of patients, $p=0.91$). Positive patients were more likely to have (a family history of BrS, $p=0.006$) or (an SCN5A genetic variant, $p<0.05$). Patients whose indication was (a family history of SADS/SUD, $p<0.05$) or (abnormal ECG, $p<0.05$) were more likely to yield a negative ajmaline test [table 2]. A single patient experienced ventricular ectopy with the slow protocol. While there were no dysrhythmias with the rapid protocol, two patients experienced QRS broadening necessitating early termination of the protocol but this was not statistically significant. Two patients developed jaundice post provocation with slow protocol, both of which resolved spontaneously.

Conclusion The NI ICC service have performed 414 ajmaline test over the last ten years. Patients with a family history of BrS are more likely to have a positive provocation challenge,

Abstract 12 Table 1 Demographics of patients by ajmaline challenge protocol

	Group A: Slow protocol (1mg/kg over 10 minutes)	Group B: Rapid protocol (1mg/kg over 5 minutes)	p-value
No of tests, n (%)	251 (61%)	163 (39%)	$p=0.61$ (ns)
Age (mean $\pm$ sd) years	40 ± 16	43 ± 15	$p=0.09$ (ns)
Gender (male: female)	136 : 115	75 : 88	$p=0.10$ (ns)
Indications:			
a) Family History of BrS	105 78	77 60	$p=0.28$ (ns)
b) Family history of SADS/SUD	17 34	9 13	$p=0.23$ (ns)
c) History of OOHCA	17	4	$p=0.61$ (ns)
d) ECG abnormality			$p=0.08$ (ns)
e) Unexplained syncope			$p=0.05$ (trend)
Positive result, n (%)	57 (23%)	27 (17%)	$p=0.13$ (ns)
Ventricular Arrhythmia	1 (ventricular ectopy)	0	
QRS broadening resulting in shortening of protocol	0	2	
Jaundice	2	0	

*BrS: Brugada syndrome, SADS/SUD: Sudden arrhythmic death syndrome/Sudden unexplained death, OOHCA: Out of hospital cardiac arrest, ns: non-significant, sig: significant.

Abstract 12 Table 2 Comparison of patients by ajmaline challenge result

Ajmaline challenge result	Age (mean)	Gender Male (%)	a) Family Hx BrS	b) Family Hx of SADS/ SUD	c) Hx of OOHCA	d) ECG abnormality	e) Unexplained syncope	SCN5A variant positive
Positive results	41.2	43/84 (51%)	48/84 (57%)	14/84 (17%)	3/84 (4%)	16/84 (19%)	3/84 (4%)	15/84 (18%)
Negative results	41.4	168/330 (51%)	134/330 (41%)	124/330 (38%)	23/330 (7%)	31/330 (9%)	18/330 (5%)	5/330 (2%)
p-value	$p=0.91$ (ns)	$p=0.96$ (ns)	$p=0.006$ (trend)	$p<0.05$ (sig)	$p=0.25$ (ns)	$p<0.05$ (sig)	$p=0.48$ (ns)	$p<0.05$ (sig)

*BrS: Brugada syndrome, SADS/SUD: Sudden arrhythmic death syndrome/Sudden unexplained death, OOHCA: Out of hospital cardiac arrest, Hx: history, ns: non-significant, sig: significant.