

since Fontan completion may largely explain this finding. This review has helped shape the planning of follow up for patients with Fontan circulation in Yorkshire and the Humber. Multicentre analysis would be useful in comparing cohorts and the standard of follow up for this growing population of patients.

**Conflict of Interest Nil**

**19 SIGNAL AVERAGED ECG CHANGES IN HYPERTROPHIC CARDIOMYOPATHY WITH OBSTRUCTIVE SLEEP APNOEA**

<sup>1</sup>Shahid Karim, <sup>2</sup>Shreyas Venkataraman, <sup>2</sup>Anwar Chahal, <sup>2</sup>Virend Somers. <sup>1</sup>Mayo Clinic, 200 1st Street, Rochester, MN 55902, USA; <sup>2</sup>MAYO CLINIC

10.1136/heartjnl-2022-BCS.19

**Background** Hypertrophic cardiomyopathy (HCM) and obstructive sleep apnoea (OSA) are independently associated with increased risk of arrhythmic events. Oximetry based studies suggest a high prevalence of OSA in HCM. Signal Average ECG (SAECG) is a non-invasive method of detecting late ventricular potentials which are low-amplitude, high-frequency signals occurring in the terminal portion of the QRS complex, analysis of which may aid in predicting the risk of reentry ventricular arrhythmias and sudden cardiac death. SAECG

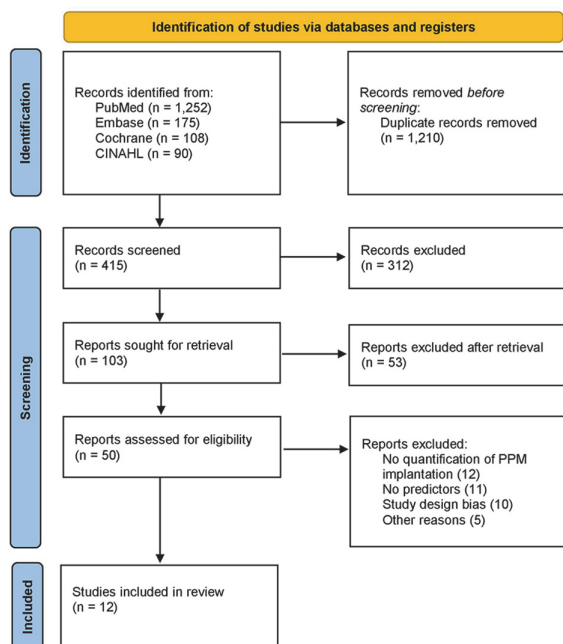
parameter profiles in HCM with concurrent OSA have not been previously studied.

**Methods** In this prospective study, 84 patients with HCM (24 of whom had previously diagnosed OSA and were treated for this) underwent SAECG analysis. The 60 HCM patients with no prior history of OSA underwent polysomnography assessment to determine their OSA status and severity. The final sample included 58 males (69%). An Apnea Hypopnea Index (AHI) >5/Hr was considered diagnostic for OSA (5–15 mild, 15–30 moderate, >30 severe).

**Results** 62 (74%) subjects were found to have OSA (15 mild, 9 moderate, 14 severe, 24 treated). Subjects with OSA were older with higher BMI. For analysis, subjects were split into those without sleep apnoea, those with sleep apnoea and those with sleep apnoea on treatment. No significant differences were found between groups in filtered QRS duration nor terminal QRS duration however the root mean square voltage of the terminal 40 ms of the QRS segment was significantly lower in the sleep apnoea group (mean 36 msec) when compared to the non-OSA group (mean 56 msec, p=0.028) and lower still in the treated OSA group (mean 30 msec, p=0.007). Similarly, the mean voltage of the terminal 40 msec of the filtered QRS segment was significantly lower in the sleep apnoea group (mean 26µv) when compared to the non-OSA group (mean 39µv, p=0.034) and again was lower still in the treated OSA group (mean 22µv, p=0.007). Those

**Abstract 19 Table 1 Study characteristics**

Study	Year	Design	No. of patients; n	Follow-up (years)	New pacemaker; %	Age (years); mean ± SD	Females; %	AF;%	LBBB; %	RBBB; %	Predictors of pacemaker insertion	Criteria for new pacemaker
Keefe et al. [8]	1985	Prospective cohort	100	4.74	3	54.4 ±13.56	NR	63	NR	NR	NR	Transient or permanent AVB
Kim et al. [9]	2001	Retrospective	155	1.08	10.96	54 ± 23	NR	44	NR	NR	AVR + MVR	Second and Third degree AVB
Meimoun et al. [10]	2002	Prospective cohort	115	3	2.6	56 ± 16	43.47	12.17	5	5	Systemic hypothermia	Third degree AVB
Berdajs et al. [11]	2008	Retrospective	391	3	4.34	57.6 ± 13	43.73	1.7	2.6	4.6	Cross-clamp time, anti-arrhythmic drugs (digoxin, sotalol, amiodarone)	Third degree AVB
McClure et al. [12]	2009	Retrospective	707	5.66	1.7	57 ± 13	39	20	NR	NR	NR	AVB
Alsoufi et al. [13]	2010	Retrospective	79	10	11	NR	NR	NR	NR	NR	NR	AVB
Levy et al. [14]	2016	Retrospective	18,402	14	10.8	63.5 ± 15	51.2	NR	NR	NR	Age, male sex, emergency admission, preexisting diabetes, renal impairment, heart failure, MVR, CABG	AVB
Tomsic et al. [15]	2018	Retrospective	83	7	8.43	56.6 ± 12.6	25	15.9	NR	NR	NR	AVB
Moskowitz et al. [16]	2019	Retrospective	14,686	1	4.5	64.92 ± 13.88	60.23	46.24	NR	NR	Age, Hx of arrhythmias, conduction disturbances, MVR + AVR	SA node dysfunction, High-degree AV block, Third degree AV block
DeRose et al. [17]	2019	RCT	243	1	14.4	70 ± 9.2	51.4	54.3	NR	NR	Age, AF ablation, multivalve surgery, NYHA class III/IV	SA node dysfunction, AVB
Herrmann et al. [18]	2021	Retrospective	797	8.77	10	Median (IQR) 70 (60 – 76)	41.4	NR	NR	NR	MVR, tricuspid ring annuloplasty, DM	AVB
Helmert et al. [19]	2021	Retrospective	1366	NR	7.75	Median (IQR) 66 (56 – 74)	43	NR	NR	NR	Age, prior MI, concomitant AVR/TVR, MVR, AF	AVB



Abstract 19 Figure 1

subjects with sleep apnoea and with sleep apnoea on treatment were also found to have a higher prevalence of atrial fibrillation and a higher prevalence of family history of sudden death.

**Conclusion** Abnormal SAECG patterns with significantly reduced root mean square voltages and terminal 40 msec voltages are seen in those patients with HCM and concurrent OSA. These changes do not appear to reverse with treatment of OSA and in fact appear to shorten further still. The SAECG may prove useful as a marker of underlying sleep apnoea and also may provide utility in predicting an individual with HCM's risk of developing arrhythmia particularly atrial fibrillation and potentially ventricular arrhythmia.

**Conflict of Interest** Nil

## 20 PREDICTORS OF PERMANENT PACEMAKER INSERTION AFTER MITRAL VALVE REPLACEMENT

<sup>1</sup>Jahanzeb Malik, <sup>2</sup>Hamza Ghauri, <sup>3</sup>Raafae Iqbal. <sup>1</sup>Rawalpindi Institute of Cardiology, Rawal Road, Rawalpindi, 46000, Pakistan; <sup>2</sup>Rawalpindi Institute of Cardiology; <sup>3</sup>Wah Medical College

10.1136/heartjnl-2022-BCS.20

**Objective** As the established surgical mitral valve replacement (MVR) expands towards various contemporary techniques and access routes, the predictors and burden of procedure-related complications including the need for permanent pacemaker (PPM) implantation need to be identified.

**Methods** Digital databases were searched systematically to identify studies reporting the incidence of PPM implantation after MVR. Detailed study and patient-level baseline characteristics including the type of study, sample size, follow-up, number of post-MVR PPM implantations, age, gender, and baseline ECG abnormalities were abstracted.

**Results** A total of 12 studies, recruiting 37,124 patients were included in the final analysis. Overall, 2,820 (7.6%) patients required a PPM with the net rate ranging from 1.7% to

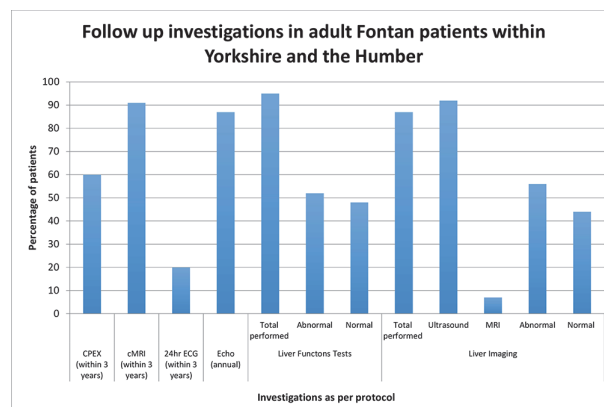


Chart 1. Follow up investigations in adult Fontan patients within Yorkshire and the Humber. CPEX: Cardio-pulmonary Exercise Test, ELF: Enhanced Liver Fibrosis Score.

**Abstract 20 Figure 1** Follow up investigations in adult Fontan patients within Yorkshire and the Humber. CPEX: Cardio-pulmonary Exercise Test, ELF: Enhanced Liver Fibrosis Score.

## Abstract 20 Table 1 Demographics of Adult Fontan Patients in Yorkshire

	Total (n=116)	TCPC (n=83)	LT (n=16)	AP (n=17)
<b>Gender</b>				
Males	70 (60%)	49 (59%)	8(50%)	13 (75%)
Females	46 (40%)	34 (41%)	8(50%)	4 (25%)
<b>Average Age (Range)</b>	27 (15 - 45)	24 (15 - 39)	30 (18 - 35)	36 (28 - 45)
<b>Average number of years since fontan completion (Range)</b>	17 (5 - 34)	14 (5 - 29)	20 (8 - 30)	28 (22 - 34)

10.96%. Post-MVR atrioventricular (AV) block was the most commonly observed indication for PPM, followed by sinoatrial (SA) node dysfunction, and bradycardia. Age, male gender, pre-existing comorbid conditions, prior CABG, history of arrhythmias or using anti-arrhythmic drugs, AF ablation, and double valve replacement were predictors of PPM implantation post-MVR.

**Conclusion** Age, male gender, comorbid conditions like diabetes and renal impairment, prior CABG, double valve replacement, and anti-arrhythmic drugs served as positive predictors of PPM implantation in patients undergoing MVR.

**Conflict of Interest** None

## 21 ATHEROSCLEROSIS IN FABRY DISEASE

<sup>1</sup>Ashwin Roy, <sup>2</sup>Hamza Umar, <sup>2</sup>Antonio Ochoa-Ferraro, <sup>2</sup>Adrian Warfield, <sup>3</sup>Nigel Lewis, <sup>2</sup>Tarekgn Geberhiwot, <sup>4</sup>Richard P Steeds. <sup>1</sup>Queen Elizabeth Hospital, Birmingham, Department of Cardiology, Mindelsohn Way, Birmingham, WMD B15 2GW, United Kingdom; <sup>2</sup>Queen Elizabeth Hospital, Birmingham; <sup>3</sup>Sheffield Teaching Hospital; <sup>4</sup>Queen Elizabeth, University Hospitals Birmingham

10.1136/heartjnl-2022-BCS.21

**Introduction** Fabry disease (FD) is a lysosomal storage disorder characterised by a deficiency in the enzyme  $\alpha$ -galactosidase A resulting in sphingolipid deposition which causes progressive cardiovascular manifestations. Angina is common in FD due to multiple mechanisms, including thickening of fibrocellular