defined as prior myocardial infarction, coronary revascularisation, stroke, transient ischaemic attack, peripheral vascular disease or carotid stenosis. SCORE2 (non-diabetics <70yrs) and ADVANCE (diabetics <70yrs) CVD risk calculators were deployed in the primary prevention cohort. Data was anonymised using a unique reference number for each patient, with the coding sequence stored strictly at the heart failure clinic.

**Results** In total, we assessed 62 patients who were prescribed aspirin. This was an elderly population (mean age 72, SD 9.8), with a high prevalence of diabetes (48%). 21 (34%) were female. 19 (31%) of patients did not have a secondary prevention indication for aspirin therapy. Of these 19 with a primary prevention strategy, 11 (58%) were <70 years of age, automatically contraindicating aspirin for use in this context. CVD risk calculation was applied to the remaining 8 patients <70 years of age. 2 were deemed high-risk, 2 moderate-risk and 4 low-risk for a CV event. Of those patients prescribed aspirin for primary prevention only 42% were co-prescribed a statin. 35/62 (56%) of the total cohort were co-prescribed proton pump inhibitor.

**Conclusions** Contravening contemporary evidence, aspirin use in primary prevention of CVD remains prevalent. While knowledge gaps and nuances to treatment in certain cases exist, clinicians should strive to avoid prescription of aspirin where it has potential to harm. Along with research to address aspirin’s merit in high-risk individuals <70yrs, education is required to empower educated, safe and effective decision making.

**General posters**

**PHENOTYPIC PREDICTORS OF GENOTYPE POSITIVITY IN PROBANDS WITH HYPERTROPHIC CARDIOMYOPATHY (HCM)**

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**Introduction** HCM is the most common inherited cardiomyopathy and a leading cause of sudden cardiac death (SCD). With growing access to genetic testing, and incorporation of genetics in diagnosis and personalised management, it is critical to better understand the phenotypic predictors of pathogenic or likely pathogenic (P/LP) variants. This has implications for family screening, as well as resource planning.

**Aims** To the yield of genetic testing in unexplained left ventricular hypertrophy (LVH) and to further identify the phenotypic predictors of genotype positivity in HCM patients.

**Method** A retrospective single centre study of 213 patients, who had undergone comprehensive HCM testing was carried out. Thirteen patients were excluded (6 - SCD, 7 - significant, non-HCM gene). Demographic information was obtained from clinic data and each patient’s LVH pattern was then classified as sigmoid, concentric, reverse or apical based on trans-thoracic echocardiogram (TTE). Pathogenicity of variants was classified according to the American College of Medical Genetics (ACMG) criteria.

**Results** A total of 200 patients were included in the analysis, of which 167 had TTE undertaken in the study centre, allowing for further detailed phenotype analysis. In the 200 patients, the mean age was 53.85 (SD 14.04) with 151 (75.5%) being male, 66 patients (17.5%) had an underlying diagnosis of hypertension (HTN) with an average of 1 anti-hypertensive agent (58.7%), 30 patients had a family history of SCD (17.5%), 41 patients (24.6%) had underlying atrial fibrillation, 53 patients had a history of ventricular arrhythemia (26.5%), 61 patients had an implantable defibrillator (31.8%), 3 patients had an aborted cardiac arrest (1.5%), 7(3.5%) patients had sepal myomectomy and 4(1%) patients required a cardiac transplant. Echocardiographic analysis showed a mean interventricular septal thickness in diastole (IVSd) of 19.8mm (SD 4.3) and the left ventricular internal diameter in diastole (LVIDd) was 44.2mm (SD 8.3). Genetic testing was appropriate in 195 patients (97.5%), with the remainder 4 patients had IVSd <15mm and 1 patient had moderate aortic stenosis. A core HCM panel (17 genes) was performed in 192 patients, 6 patients had extended HCM (69 genes) and 2 patients had global CM panel (109 genes), with a yield, of
LP/P in 58 (30.2%), 3 (50%) and 1 (50%) within each respective panel. No gene, including likely benign (LB) and benign (B), was identified in 98 patients (49%). MYBPC3 and MYH7 were the most frequently identified variants (figure 1). Sub-analysis of 167 patients with TTE showed the concentric pattern of LVH was most frequent at 31.7% (53 patients) followed by reverse, apical and sigmoid patterns (table 1). Younger patients, females, family history of SCD, normotensive with reverse pattern LVH (p value <0.001) predicted LP/P variant identification (table 2).

Conclusion Genetic testing was appropriately offered and comparable to international guidelines (33.3% ESC, 32% AHA). In patients with unexplained LVH being younger, female, having a family history of SCD, normotensive and a reverse pattern LVH on TTE predicted a higher yield of LP/P variant identification. This study has implications for supporting better phenotype-based genetic counselling and resource usage for HCM patients.

### CHARACTERIZATION, SHORT TERM OUTCOME AND ANATOMICAL DISTRIBUTION OF ROTATIONAL AND FOCAL ACTIVITIES IN PERSISTENT ATRIAL FIBRILLATION

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**Introduction** Pulmonary vein isolation has become the cornerstone treatment of Atrial Fibrillation (AF), although its efficacy varies for up to 72% in cases of paroxysmal AF and around 50% in persistent AF after single procedure. Improving outcomes post ablation in persistent AF (Pe AF) has been challenging, despite of the applications of various strategies. Automated detection and ablation of repetitive rotational and focal activities RAs/FAs in persistent AF has been valued of possible viable therapeutic targets.

**Purpose** We sought to study frequency and anatomical distribution of FAs/RAs across left atrium (LA) post PVI. Also, to compare standard approach of Pulmonary vein Isolation (PVI) with additional ablation lines vs PVI and FA/RA ablation to compare follow-up AF free burden.

**Methods** We retrospectively studied 60 cases with Pe AF who had first time RF ablation treatment (20 cases had FAs/RAs mapped and ablated in plus; 40 cases had standard ablation approach. Following PVI, sequential high-density mapping performed with multielectrode catheter applied for 30 seconds at each anatomical location in LA only. Follow up information were gathered at 3rd, 9th, and at 12th months post index procedure using electronic hospital records. All data were analysed using SPSS software program (IBM Inc).

**Results** Cohort demographics were comparable in both groups. In total 279 area of interest mapped in 20 pts (average 13.95 applications per patient), with a total of 84 FAs and 3 RAs detected. LAA hosted majority of FAs/RAs among all 9 segments of LA (anterior, posterior, septal, lateral, LAA, roof, floor, RPVA and LPVA) (figure 1). There was no significance