bicycle ergometer exercise stress using a WHO25 protocol. An abnormal ESE was defined by the occurrence of: increase in AV mean gradient >20 mmHg, increase in LVOt gradient to >50 mmHg, absence of left ventricular contractile reserve, new dynamic severe MR, PASP >60 mmHg or new wall motion abnormality. An abnormal exercise ECG test was defined by the occurrence of chest pain, ST depression ≥2 mm, a fall in systolic BP or rise <20 mmHg, ventricular arrhythmia or METs <4. The occurrence of pre-conception interventions, and post-conception adverse maternal CV and obstetric events were recorded. Follow up was terminated at 6 months post-partum.

Results At baseline, out of the total of 44 patients, 24 had at least mild congenital aortic stenosis, 8 had undergone previous aortic valve replacement, and 12 had hypertrophic cardiomyopathy. Twenty-three patients (52%) had an abnormal ESE and 21 patients (48%) had an abnormal exercise ECG test. ESE helped guide 3 pre-conception aortic valve replacements and 21 patients (48%) had an abnormal exercise ECG test. Of the available risk scores, ESE had a stronger association with the occurrence of adverse maternal CV events (16%) and 10 adverse obstetric events (23%). Prior cardiac medication (p=0.031) and multiple cardiac lesions (p=0.037) were associated with adverse maternal CV events. Of those with abnormal ESE, 5 (22%) patients suffered an adverse maternal CV event, one of which had a normal exercise ECG test. Patients with abnormal ESE accounted for 71% of all adverse maternal CV events (RR= 2.1, 95% CI: 0.5–9.6, p=0.4) and 50% of obstetric events (RR= 0.8, 95% CI: 0.3–2.5, p=1.0). The abnormal exercise ECG group accounted for 57% of adverse maternal CV events (RR=1.2, 95% CI: 0.3, 4.8, p=0.6) and 50% of obstetric events (RR= 0.91, 95% CI: 0.3–2.7, p= 1.0). Of the available risk scores, on ROC analysis, a mWHO class of III-IV was the strongest predictor of CV events (AUC= 0.77, RR: 9.7, 95% CI: 2.2–42.2, P= 0.01).

Conclusion Patients with abnormal ESE results were twice as likely to suffer an adverse maternal CV event. Abnormal ESE had a stronger association with the occurrence of adverse maternal CV events than exercise ECG testing. These results suggest that ESE provides additive prognostic information among high-risk patients with left heart obstruction.

Conflict of Interest N/A
study cohort. The endpoints were mortality at 30 days and 5 years, and in-hospital length of stay. 1684 patients (median age 83 [79 – 87] years; 32% female) were screened, of whom 121 (7.2%) had RBBB. 41 (33.9%) patients received a prophylactic PPM by clinical preference. Of the remaining 80, 45 (56%) patients received a PPM after TAVI. Baseline demographic and procedural characteristics were similar. Among those patients in sinus rhythm who had a prophylactic PPM, the PR interval was longer than in those who did not receive a prophylactic pacemaker (200 ms [170 – 228] vs. 175 ms [158 – 196]; p=0.05). Similarly, the QRS duration was longer (140 ms [126 – 156] vs. 131 ms [120 – 142]; p=0.01). There was numerically but not statistically more left anterior hemi-block (24 [62%] vs. 29 [45%]; p=0.10). All-cause mortality was similar at 5 years with death in 17 patients (41.4%) in the prophylactic PPM group vs. 27 (33.8%) in the non prophylactic PPM group; hazard ratio 1.27 (95% CI 0.69 to 2.33; p=0.44; Figure 1). In the no prophylactic PPM group 16 (35.6%) deaths occurred in those receiving PPM after TAVI and 11 (31.4%) in those not receiving PPM; hazard ratio 0.95 (95% CI 0.43 to 2.09; p=0.90). 30-day all-cause mortality was also similar with 2 deaths (5.3%) occurring in the prophylactic group vs. 5 deaths (6%) in those without a prophylactic PPM, 3 (8.6%) of which were in those not receiving PPM and 2 (4.4%) receiving PPM after TAVI. There was a trend to a reduced hospital length of stay in those receiving prophylactic PPM compared to those who either received a post TAVI PPM or who were discharged without PPM (2.5 [1.6] days vs. 4.0 [4.8] days, respectively; p = 0.08). When comparing the prophylactic PPM group to those who received PPM after TAVI, there was a statistically significant reduction in length of stay (2.5 [1.6] vs. 4.3 [4.5] days, respectively; p=0.02; Figure 2). Two patients in the prophylactic PPM group developed a pocket haematoma managed non-operatively.

Conclusions Over half of patients with RBBB undergoing TAVI require a pacemaker after their valve implant for high-grade AV block. A prophylactic pacing strategy in this high-risk cohort is safe and reduces length of hospital stay.

Conflict of Interest None

10 DISEASE PENETRANCE IN ASYMPTOMATIC CARRIERS OF FAMILIAL CARDIOMYOPATHY VARIANTS

Adil Mahmood, 2Deborah Morris-Rosendahl, 1Matthew Edwards, 2Andrew Fleming, 2Tessa Homfray, 2Samantha Mason, 2Ellie Quinn, 3James Ware, 2John Baks, 2Sanjay Prasad, 2Antonis Pantazis, 3Brian Halliday, 2Royal Brompton Hospital, Royal Brompton Hospital, Sydney Street, London, LND SW3 6NP, United Kingdom; 2Royal Brompton Hospital, 2Harefield Hospital

Introduction Predictive genetic testing allows the identification of at-risk first-degree relatives of patients with genetic cardiomyopathies. Data on the penetrance of genetic variants associated with cardiomyopathies is limited. The aim of this study was to investigate disease penetrance in asymptomatic carriers of familial cardiomyopathy variants associated with hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC).

Methods We included asymptomatic individuals referred to the Royal Brompton Hospital for predictive testing after the finding of a pathogenic or likely pathogenic genetic variant in a first-degree relative with cardiomyopathy between January 2017 and December 2019. Cardiomyopathy diagnosis was defined by international guidelines. Those with a prior diagnosis or signs or symptoms of heart disease at the time of testing were excluded. Results: A total of 105 genotype-positive individuals from 80 families were evaluated (median age 24.9 years [interquartile range: 16.1 to 45.0 years], 51 [48.6%] males). Variants in genes associated with DCM included: TTN n = 16 (51.6%), MYH7 n = 5 (16.1%), LMNA n = 3 (9.7%), TNNT3 n = 3 (9.7%), RBM20 n = 2 (6.5%), BAG3 n = 1 (3.2%), DMD n = 1 (3.2%); variants in genes associated with HCM included: MYBPC3 n = 31 (47.7%), MYH7 n = 25 (38.5%), TPM1 n = 5 (7.7%), PLN n = 1 (1.5%), TNNC1 n = 1 (1.5%), TNNT3 n = 1 (1.5%), TNNT2 n = 1 (1.5%); and variants in genes associated with ARVC included: PKP2 n = 4 (44.4%), DSP n = 4 (44.4%), FLNC n = 1 (11.1%). On first clinical evaluation 2 of 31 carriers of DCM variants were diagnosed with DCM, 5 with hypokinetic non-dilated cardiomyopathy (HNDC) and 4 with isolated left ventricular (LV) dilatation; 10 of 65 carriers of HCM variants were diagnosed with HCM; and 0 of 9 carriers of ARVC variants were diagnosed with ARVC. Over a median follow-up of 2.4 years (interquartile range: 1.0 to 4.3 years) an additional 13 of 84 carriers developed cardiomyopathy phenotypes (0 of 20 DCM, 3 of 20 HNDC, 3 of 20 isolated LV dilatation, 7 of 35 HCM, 0 of 9 ARVC). Furthermore, 2 individuals with DCM/HCM received implantable cardioverter defibrillators and 2 individuals with DCM/HCM received implantable loop recorders.

Conclusions Approximately one-third of asymptomatic carriers of familial cardiomyopathy variants were diagnosed with a cardiomyopathy phenotype at initial screen or during a short follow-up period. This confirms the importance of predictive testing and the need for follow-up of genotype-positive, phenotype-negative individuals.

Conflict of Interest None

11 IS PULMONARY ARTERY DILATATION PART OF THE ATHLETIC ADAPTATION TO EXERCISE?

Nikhil Chattrath, 2Kashif Quazi, 2Saad Fayaz, 2Raghav Bhatia, 2Harish MacLachlan, 2Christopher Rowntree, 2Sanadep Marwaha, 2Mania Teresa Tome Esteban, 2Sanjay Sharma, 3Michael Papadaklis. 1St. George’s University of London, St George’s University, Cranmer Terrace, London, LND SW17 0RE, United Kingdom; 2St. George’s, University of London and St. George’s University Hospitals NHS Foundation Trust; 3St. George’s University of London

Introduction The ‘Athlete’s Heart’ is a well-established term used to describe the spectrum of structural and electrical cardiac adaptations to intensive exercise. Several studies have demonstrated the development of ventricular dilatation and hypertrophy in athletic individuals and cardiac magnetic resonance imaging (CMR) is well suited to assess these changes. However, there is a paucity of studies looking at pulmonary artery (PA) dimensions in athletic individuals. One could hypothesize that increased stroke volumes and pulmonary flow may cause dilatation of the PA. The aim of this study is to ascertain whether PA dilatation is part of the cardio-pulmonary adaptation to intensive exercise and establish normal parameters of PAs in athletes.

Methods The CMRs of male athletes performed at a single-centre between January 2017 and October 2021 were reviewed. Those with any evidence of a structural abnormality...