

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 no 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

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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%), TNNT1 $n=1$ (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 55 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?

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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