Abstract 183 Table 3 Logistic regression P values, odd ratios and confidence intervals for a 5mmHg increase in BP parameters

	Maximum exercise systolic BP (5mmHg)		Resting systolic BP (5mmHg)		24 Hours ambulatory BP (5mmHg)		Nocturnal Ambulatory BP (5mmHg)	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
CAC Score >100 AU	N/A	0.498	1.21 (1.07-1.37)	0.003	1.23 (0.99-1.54)	0.06	1.28 (1.05-1.57)	0.016
Stenosis >50%	1.26 (1.09-1.46)	0.002	1.31 (1.09-1.58)	0.005	1.74 (1.24-2.46)	0.002	1.50 (1.10-2.03)	0.01
Plaque Vulnerability Markers	1.14 (1.04-1.25)	0.004	1.16 (1.02-1.32)	0.022	1.36 (1.07-1.71)	0.01	1.43 (1.15-1.78)	0.001

1.07-1.33), 18.49% (p=0.024, CI 1.02-1.37), 46.45% (p=0.001, CI 1.12-1.91) and 50.54% (p=0.001, CI 1.18-1.93) respectively.

Conclusions Hypertension is prevalent, under diagnosed, and contributes to the development of potentially unstable coronary artery disease in healthy male endurance athletes. Recommendations for cardiovascular evaluation of master athletes should consider a low threshold for non-invasive exercise testing and ambulatory BP monitoring for for athletes with hypertension and even high-normal BP. Table 1. ESC definitions of hypertension. Table 2. Prevalence of hypertension according to ESC guideline classifications. Table 3. Logistic regression P values, odds ratios and confidence intervals for a 5mmHg increase in BP parameters.

Conflict of Interest Nil

184 ASSOCIATION BETWEEN HIGH-SENSITIVITY TROPONIN AND ONE YEAR MORTALITY IN 20,000 CONSECUTIVE HOSPITAL PATIENTS UNDERGOING A BLOOD TEST FOR ANY REASON

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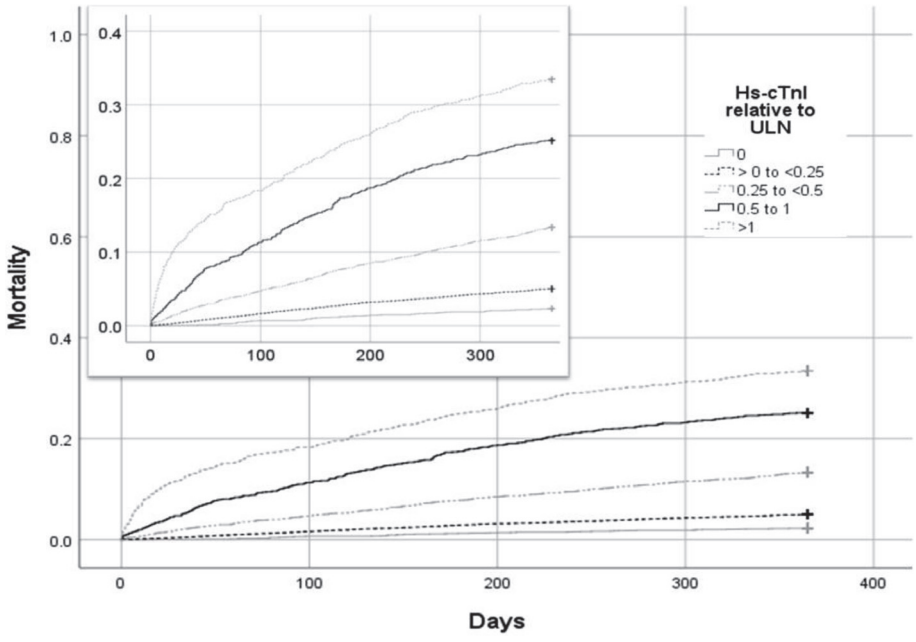
Introduction High sensitivity troponin (hs-cTn) concentrations above the manufacturer recommended upper limit of normal (ULN) are seen frequently in patients without a clinical presentation consistent with an acute coronary syndrome. There is increasing evidence that these concentrations may act as a marker of prognosis in a range of conditions. However, previous studies have been limited because they have only included patients in whom the clinician has requested the test. The aim of this study was to assess the relationship between one year mortality and hs-cTn concentration in a consecutive hospital population, regardless of whether there was a clinical indication for performing the test.

Method This study included 20,000 consecutive patients that had hs-cTnI added onto their blood tests at a large teaching hospital, regardless of the clinical indication (CHARIOT population). One year mortality data was obtained by linkage with NHS Digital. The association between hs-cTnI concentration and one year mortality was evaluated using Kaplan-Meier plots and Cox proportional hazards analyses. After the cohort was considered as a whole, each of the clinical areas (inpatient (IPD), outpatient (OPD), emergency department (ED)) were considered separately.

Results Overall, 1782 (8.9%) patients had died at one year. Multivariable Cox regression analysis showed that a hs-cTnI concentration above the ULN was independently associated



Abstract 184 Figure 1 Kaplan-Meier curve of one year mortality based on the ratio of the hs-cTnI concentration to the ULN (log rank test between each stratum p<0.001)

with the hazard of mortality (HR 2.23; 95% CI 1.97 – 2.52). There was a progressive increase in mortality across the strata of hs-cTnI concentration (figure 1). Furthermore the log (10) hs-cTnI concentration was an independent predictor of the hazard of one year mortality (HR 1.77; 95% CI 1.64 – 1.91). The discriminative ability of hs-cTnI for one year mortality was good with an AUC of 0.75 (95%CI 0.73 – 0.76). Further, the log(10) hs-cTnI concentration was independently associated with mortality across all three locations and most strongly in the OPD cohort (IPD HR 1.49; 95% CI 1.33 – 1.67, OPD HR 2.44; 95% CI 1.95 – 3.04, ED HR 1.99; 95% CI 1.76 – 2.25).

Conclusion In a large, unselected hospital population of both in- and out-patients, 18,282 (91.4%) of whom there was no clinical indication for testing, hs-cTnI concentration was independently associated with one year mortality. These data suggest that hs-cTnI may have a role as a biomarker of future risk.

Conflict of Interest Beckman Coulter paid for all of the assays used in our studies but had no other role in the studies

185 FEMALE SPEAKER REPRESENTATION AT NATIONAL CARDIOLOGY CONFERENCES

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Introduction Women are under-represented in cardiology in the UK (28% of cardiology trainees and 13% of cardiology consultants are female despite women accounting for >50% of medical students). Lack of female role models is one of the commonly cited reasons for the lack of women within the field. Fair representation of female speakers at national cardiology conferences is therefore important to increase the visibility of women within cardiology. We assessed the extent of female representation at UK national cardiology conferences over the last 4 years.

Methods We requested the past programmes for annual conferences from 8 national cardiology societies in the UK. The programmes were reviewed and for each chair, panellist, operator and speaker, a binary definition of gender was assigned (female or male). In cases where gender was unclear from the name, the General Medical Council (GMC) register was referred to. For those not on the GMC register, we searched for the individual online. In any remaining cases of ambiguity, we selected the gender most commonly associated with that name. Speakers presenting abstracts were excluded.

Results Programmes were obtained from 7 societies (British Cardiovascular Society (BCS), British Cardiovascular Intervention Society (BCIS), British Heart Rhythm Society (BHRS), British and Irish Hypertension Society (BIHS), British Society of Cardiovascular Magnetic Resonance (BSCMR), British Society of Echocardiography (BSE), and British Society for Heart Failure (BSH). A total of 25 annual conferences were analysed. There were a total of 3959 slots; 982 of these were female (25%). Of the 951 chair slots, 224 (24%) were female. There were 3007 speaker/panellist slots, of which 760 (25%) were female. See table 1 below for details.

Conclusion There is significant variation in the proportion of female speakers between societies (11-45%). Specialties with the lowest numbers of female consultants had the lowest female representation, however BCS which represents all subspecialties also had poor female speaker representation. No clear improvement in female numbers occurred from 2017-2020. As part of a drive to facilitate gender equality within cardiology, conference committees need to ensure that speaker panels are gender balanced to enhance visibility of women within the specialty.

Conflict of Interest None

Abstract 185 Table 1

Society Total speakers (% female) Year	BCIS	BCS	BHRS	BIHS	BSCMR	BSE	BSH
2020	169 (15%)	No conference	340 (28%)	15 (33%)	No data	98 (41%)	65 (45%)
2019	193 (17%)	367 (23%)	325 (31%)	57 (25%)	30 (30%)	92 (40%)	61 (29%)
2018	200 (14%)	245 (22%)	385 (23%)	65 (15%)	No data	90 (22%)	64 (34%)
2017	128 (13%)	372 (19%)	336 (32%)	73 (26%)	35 (11%)	93 (38%)	61 (21%)

186 EFFECTS OF AN ESTABLISHED DRUG ON SMAD SIGNALLING PATHWAY IN THE RAT MODEL OF MONOCROTALINE PULMONARY HYPERTENSION

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Introduction Pulmonary arterial hypertension (PAH) is a complex multifactorial disease with both genetic and environmental dynamics contributing to disease progression that characterized by unbalanced proliferation and apoptosis of pulmonary arterial smooth muscle cells (PASMCs) (Nasim et al., 2012). Mutations in the bone morphogenetic protein receptor type II (BMPRII) gene have been associated with development of familial pulmonary artery hypertension (PAH). The function of SMAD signal transduction during the pulmonary vasculature and the role BMPRII mutations in the development of PAH are not fully understood. However, drug or toxin-induced PAH has been characterized by raised pulmonary arterial resistance leading to right heart failure. Aim and

Methods in this study, the monocrotaline (MCT) model of PAH was used to examine alterations in SMADs (SMAD3 and SMAD1/5) signal transduction pathways in vivo. The SMAD signalling pathways were investigated in lungs harvested from rats treated with a single 50-mg/kg of MCT and an established drug at concentrations of 0.5 and 1 mg/Kg. The level of phosphorylation of SMAD3 and SMAD1/5 were detected by Western blot and the expression BMPR2, Id1, Pai1 transcripts was measured using quantitative real time PCR (qPCR).

Results MCT-treated rats decreased the level of SMAD 3 phosphorylation, which was restored following the treatment with the established drug. The drug also modulated the expression of SMAD target genes.