Abstract 2 Table 2 Exercise echo data

EXERCISE ECHO DATA	Control (n=20)	TTS (n=20)	P
Exercise time (min)	7.5±2.6	6.8±2.3	NS
Peak HR (bpm)	147±8	152±12	NS
Peak SBP (mmHg)	148±16	170±23	0.005
Peak LVEDV (ml)	81±16	93±21	0.05
Peak LVESV (ml)	17±7	±11	0.001
Peak LVEF (%)	80±7	69±11	0.001
ΔLVEF (%)	19±9	8±11	0.001
Peak WMSI	1±0	1.4±0.4	<0.001

mmHg, respectively). A comparative analysis of the resting and stress echo data is given in table 1. The stress echo data showed similar exercise time and peak HR, but higher peak SBP in TTS patients. TTS patients had blunted contractile response to exercise with lower peak LVEF, ΔLVEF (exercise minus resting EF) and peak wall motion score index (WMSI) compared to controls. Among the 20 TTS patients, 12 had exercise-induced wall motion abnormalities: 6 involved the apical segments and 6 developed global dysfunction. In these 12 patients, the mean ΔLVEF was reduced (4%). In the other 8 patients, the ΔLVEF was 16% and pooled the TTS cohort had blunted contractile response with exercise compared to controls (ΔLVEF 8% vs. 19% in the control group, $p=0.001$).

Conclusion Our study shows that symptomatic patients with previous TTS have a blunted contractile response to exercise manifest as reversible apical or global LV dysfunction. These findings might indicate the need for treatment. Further systematic investigation of the therapeutic and prognostic implications of this reversible exercise-induced myocardial dysfunction is needed.

Conflict of Interest no

3

3D ECHOCARDIOGRAPHY-DERIVED INDICES OF LEFT VENTRICULAR FUNCTION AND STRUCTURE PREDICT LONG-TERM MORTALITY DIFFERENTLY IN MEN AND WOMEN: THE SOUTHALL AND BRENT REVISITED (SABRE) STUDY

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Background Three-dimensional echocardiography (3DE)-derived indices of left ventricular (LV) structure and function predict mortality in various patient groups, although studies are limited. However, whether these indices equally predict mortality in the general population is unknown. Further, whether known sex-differences in cardiac structure and function modify the relationship between these indices and subsequent death is uncertain.

Purpose We therefore sought to examine the associations between 3DE-derived LV ejection fraction (LVEF), volumes and remodelling, and all-cause mortality in a general population and to investigate whether this relationship is modified by sex.

Methods A total of 910 individuals (age, 69.7±6.2 years; 77.7% male) from the SABRE study, the UK-largest tri-ethnic community cohort, underwent a health examination including comprehensive transthoracic echocardiography. Full volume 3D LV datasets acquired over 4 sub-volumes were obtained using a matrix-array transducer, and were analysed offline using Qlab advanced, v7.0. The association between 3D-derived LVEF, body surface area indexed end-diastolic volume (iEDV) and end-systolic volume (iESV), and LV remodelling index (LVRI, the ratio of LV mass and EDV) and risk of all-cause mortality were determined using Cox proportional hazards models.

Abstract 3 Table 1 a) Associations between 3DE-derived indices of LV function and structure and all-cause mortality

	Unadjusted HR (95% CI), <i>p</i>	Model 1 HR (95% CI), <i>p</i>	Model 2 HR (95% CI), <i>p</i>	Model 3 HR (95% CI), <i>p</i>
EF All (N=910; 120 deaths, SD=6.8%)	0.79 (0.67, 0.93), 0.006	0.81 (0.68, 0.97), 0.020	0.81 (0.68, 0.96), 0.015	0.84 (0.71, 1.0), 0.060
EF Interaction term for sex*	1.7 (0.95, 3.2), 0.074	1.6 (0.89, 2.9), 0.114	1.6 (0.88, 3.0), 0.117	1.59 (0.86, 2.9), 0.142
EF Men (N=707; 103 deaths, SD=6.6%)	0.78 (0.66, 0.93), 0.005	0.78 (0.65, 0.93), 0.006	0.78 (0.65, 0.93), 0.005	0.81 (0.67, 0.97), 0.021
EF Women (N=203; 17 deaths, SD=5.6%)	1.35 (0.75, 2.4), 0.313	1.3 (0.74, 2.2), 0.368	1.3 (0.71, 2.4), 0.387	1.31 (0.69, 2.5), 0.399
LVRI All† (N=879, 115 deaths, SD=0.37g/ml)	0.91 (0.75, 1.1), 0.336	0.85 (0.70, 1.0), 0.089	0.84 (0.70, 1.0), 0.065	0.86 (0.71, 1.0), 0.120
LVRI Interaction term for sex*	1.9 (1.2, 3.2), 0.011	1.8 (1.1, 3.1), 0.023	1.7 (1.0, 2.9), 0.034	1.5 (0.94, 2.6), 0.085
LVRI Men (N=689; 99 deaths, SD=0.37 g/ml)	0.83 (0.67, 1.0), 0.072	0.78 (0.64, 0.96), 0.020	0.77 (0.63, 0.94), 0.012	0.79 (0.65, 0.98), 0.030
LVRI Women (N=190; 16 deaths, SD=0.34 g/ml)	1.6 (0.99, 2.5), 0.052	1.4 (0.86, 2.2), 0.184	1.5 (0.95, 2.5), 0.082	1.4 (0.88, 2.2), 0.159

Model 1: adjusted for age, sex and ethnicity

Model 2: model 1 + SBP, antihypertensive medication, cholesterol: HDL ratio, BMI, DM, smoking

Model 3: model 2 + history of CHD

* $P \leq 0.1$ indicates significant interaction

† indicates lower numbers for LVRI due to lower available numbers of LV mass. (n=879)

Abstract 3 Table 1 b) Associations between 3DE-derived LV volumes and all-cause mortality

	Unadjusted HR (95% CI), <i>p</i>	Model 1 HR (95% CI), <i>p</i>	Model 2 HR (95% CI), <i>p</i>	Model 3 HR (95% CI), <i>p</i>
EDV All (N=910; 120 deaths, SD=9.6ml/m ²)	1.20 (1.0, 1.42), 0.029	1.21 (1.02, 1.43), 0.021	1.21 (1.0-1.43), 0.027	1.16 (0.98-1.38), 0.079
EDV Interaction term for Sex*	0.43 (0.23, 0.84), 0.013	0.49 (0.25, 0.97), 0.042	0.50 (0.25, 1.0), 0.051	0.56 (0.29, 1.1), 0.088
EDV Men (N=707; 103 deaths, SD=9.9ml/m ²)	1.23 (1.0, 1.46), 0.017	1.26 (1.1 1.5), 0.006	1.26 (1.1, 1.5), 0.007	1.22 (1.0, 1.4), 0.026
EDV Women (N=203; 17 deaths, SD=7.3ml/m ²)	0.54 (0.29, 1.0), 0.056	0.63 (0.33, 1.2), 0.182	0.51 (0.25, 1.1), 0.076	0.55 (0.28, 1.1), 0.097
ESV All (N=910; 120 deaths, SD=5.8ml/m ²)	1.27 (1.1, 1.51), 0.007	1.27 (1.1, 1.53), 0.009	1.27 (1.1, 1.53), 0.008	1.21 (1.0, 1.45), 0.042
EDV Interaction term for Sex*	0.40 (0.22, 0.71), 0.002	0.44 (0.25, 0.81), 0.008	0.45 (0.24, 0.82), 0.009	0.49 (0.27, 0.89), 0.019
ESV Men (N=707; 103 deaths, SD=6.1ml/m ²)	1.34 (1.1, 1.6), 0.002	1.37 (1.13, 1.65), 0.001	1.37 (1.14, 1.66), 0.001	1.31 (1.1, 1.6), 0.006
ESV Women (N=203; 17 deaths, SD=3.7ml/m ²)	0.54 (0.32, 0.92), 0.026	0.62 (0.36, 1.1), 0.092	0.56 (0.31, 1.0), 0.048	0.59 (0.34, 1.0), 0.060

Model 1: adjusted for age, sex and ethnicity

Model 2: model 1 + SBP, antihypertensive medication, cholesterol: HDL ratio, BMI, DM, smoking

Model 3: model 2 + history of CHD

3D ESV is log transformed.

*P≤0.1 indicates significant interaction

Abstract 3 Figure 1

Results Over a median follow-up of 8 years, 120 participants (13.1%) died. Overall, lower 3D LVEF, and higher 3D iEDV and iESV were associated with increased risk of all-cause mortality (table 1a 1b). However, sex modified the associations between volume indices and LVRI with all-cause mortality; hence we present sex-specific results in table 1. After multi-variable adjustment for age, ethnicity, systolic blood pressure, antihypertensive medications, cholesterol: HDL ratio, diabetes, body mass index, smoking and history of coronary heart disease, per SD change, decrease in LVRI and LVEF, and increase in volumes were independently associated with increased risk of mortality in men (figure 1, table 1a 1b). However, associations between volume indices, LVRI and, to a lesser extent, LVEF, and mortality were reversed in women (figure 1, table 1a 1b): fully adjusted hazard ratios (95% CI): EDV: men: 1.22 (1.0, 1.4), women: 0.55(0.28, 1.1), ESV: men: 1.31(1.10, 1.60), women: 0.59(0.34, 1.0), LVRI: men: 0.79(0.65, 0.98), women: 1.40(0.88, 2.2), LVEF: men: 0.81(0.67, 0.97), women: 1.31(0.69, 2.5). Although numbers of women are small, effect sizes indicated that lower volumes and higher LVRI were associated with higher risk of all-cause mortality in women.

Conclusion In the general population, 3DE-derived LVEF, volumes and remodelling predict the long-term risk of all-mortality, independent of clinical confounders and cardiovascular risk factors strongly in men. However the direction of association for volumes and remodelling measures is reversed in women suggesting that sex-differences in cardiac structure and function seem to be associated differently with the risk of all-cause mortality.

Conflict of Interest N/A

4

18F-SODIUM FLUORIDE POSITRON EMISSION TOMOGRAPHY PREDICTS PROGRESSION OF CORONARY CALCIFICATION

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Introduction Combined positron emission tomography and computed tomography (PET-CT) using the radiotracer 18F-sodium fluoride (18F-NaF) to detect microcalcification provides imaging of both coronary artery anatomy and disease activity simultaneously. While recent studies have suggested that 18F-NaF activity may help identify high-risk coronary atherosclerosis, the role of 18F-NaF uptake in predicting progression of coronary atherosclerosis is unknown. In this study, we aimed to investigate the relationship between baseline coronary arterial 18F-NaF activity and the subsequent progression of coronary arterial calcification in patients with clinically stable coronary artery disease.

Methods Patients with clinically stable, multivessel coronary artery disease underwent combined 18F-NaF PET-CT and CT coronary calcium scoring at baseline with repeat CT coronary calcium scoring at one year. Coronary arterial PET uptake was analysed qualitatively and semi-quantitatively in diseased vessels by measuring maximum tissue-to-background ratio (TBRmax) – defined as the maximum standardised uptake value in a plaque divided by the mean blood pool activity

Abstract 4 Figure 1

Abstract 4 Table 1 Progression of coronary calcification in patients with and without increased ¹⁸F-NaF activity at baseline

PROGRESSION OF CALCIFICATION AT ONE YEAR	All patients (n=183)	PET positive (n=116)	PET negative (N=67)	P value
Change in Agatston Score (AU)	70[22-143]	98[39-166]	35[7-93]	<0.001
Change in Calcium Volume (mm ³)	60[16-121]	82[29-149]	33[3-82]	<0.001
Change in Calcium Mass (mg)	14[4-35]	22[7-42]	6[1-21]	<0.001
Change in Average Density (mg/mm ³)	0.01[-0.002-0.02]	0.01[0.001-0.02]	0.01[-0.004-0.12]	0.78