number may have had non cardiac chest pain. The CAD50 rate in the ACP, AP and AB groups (50% of total cases) was only 10% suggesting that CTA is largely being used to exclude significant CAD in low or intermediate risk groups. The rate of CAD 70 in group A was only 20%. It is likely that figure is artificially low as, while waiting lists for CTA remain longer than for ICA, clinicians will request ICA as the first test if CAD70 felt likely. Better case selection for CTA could free up capacity for PTs with AA and A. Without this or substantial further increases in CTA capacity, the number of ICAs performed not leading to revascularisation is unlikely to change.

Conflict of Interest None

Stable IHD/Prevention/Hypertension/Lipids

181 THE PREVALENCE OF CORONARY ARTERY DISEASE IN PATIENTS PRESENTING TO A RAPID ACCESS CHEST PAIN CLINIC WITH 'NON-ANGINAL CHEST PAIN'

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Introduction NICE define three key features of typical angina, and patients presenting with only one of these features are described as having 'non-anginal chest pain'. The latest NICE guidelines advise against routine testing for coronary artery disease (CAD) in this cohort. Despite this, the rapid access chest pain clinic at our institution have continued to investigate these patients. This study sought to establish the prevalence of CAD in this cohort, and evaluate the differences in cardiovascular risk factors between those with and without significant disease.

Methods Between 1st January 2017 and 1st December 2019, patients without prior history of cardiac disease presenting with non-anginal chest pains were referred for coronary computed tomography angiography (CCTA). Calcification scores were recorded using the Coronary Artery Calcium-Data and Reporting System (CAC-DRS) and luminal stenosis was recorded using the Coronary Artery Disease-Reporting and Data System (CAD-RADS). Patients unable to have luminal analysis due to calcium or high heart rates either had stress echocardiography (SE) or invasive coronary angiography (iCA). Patients were deemed as having significant CAD if they had any of the following: CAD-RADS score of 4 or more on CCTA, 4 segments of stress inducible ischaemia on SE or severe stenosis on iCA. Cardiovascular risk factors were then compared between those with and without CAD, to determine if there were any significant differences between the groups.

Results In total, 1078 patients presented with non-anginal chest pains. All patients had CAC-DRS scores and in 872 patients it was possible to record CAD-RADS scores (table 1). Of the 206 patients unable to have luminal analysis, 199 had SE and 7 had iCA. In total, 143 patients (13.3%) were found to have significant CAD on imaging. In total, 34 of these patients had revascularisation (9 patients had bypass grafting and 25 patients had percutaneous coronary intervention). Table 2 shows that patients with significant CAD were more likely to be hypertensive ($p=0.004$), older ($p<0.001$) and have a higher Qrisk2 score ($p<0.001$).

Abstract 181 Table 1 The CAD-RADS scores of patients with a CCTA allowing luminal analysis

CAD-RADS score	Number of patients
0	261
1	194
2	190
3	126
4A or 4B	90
5	11

Abstract 181 Table 2 The differences in cardiovascular risk factors in those with and without significant CAD

Cardiovascular risk factor	All (n=1078)	Patients without significant CAD (n=935)	Patients with significant CAD (n=143)	P value
Age (years), mean (SD)	60 (10.4)	59 (10.5)	63 (9.1)	<0.001
Qrisk2, median (IQR)	14.2 (8.1-22.5)	13.3 (7.8-21.2)	19.6 (12.4-26.2)	<0.001
Diabetes, n(%)	167 (15)	139 (15)	28 (20)	0.171
Hypertension, n(%)	416 (39)	345 (37)	71 (50)	0.004
Family History, n(%)	429 (40)	370 (40)	59 (41)	0.714
Hypercholesterolaemia, n(%)	168 (16)	147 (16)	21 (15)	0.806

Conclusion Following current NICE guidelines for 'non-anginal chest pain' may mean a considerable proportion of patients with underlying CAD are missed, including those with severe lesions requiring revascularisation. Our analysis suggests that age, Qrisk2 score and a history of hypertension are statistically significant predictors of significant CAD in this population.

Conflict of Interest None

182 TISSUE DOPPLER E' VELOCITY AND E/E' PREDICT 19-YEAR CARDIOVASCULAR MORTALITY IN HYPERTENSION

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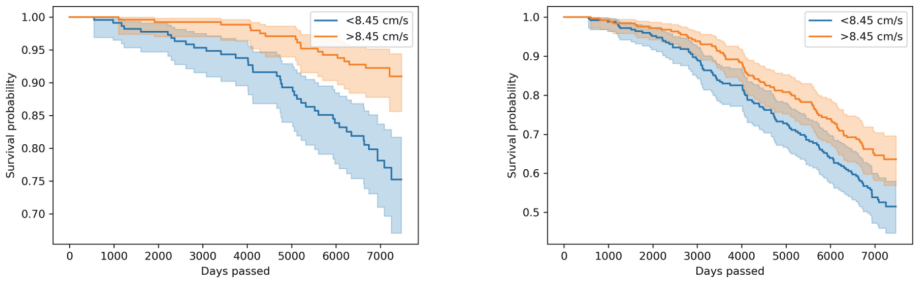
Background We have previously shown that tissue Doppler assessments of left ventricular (LV) diastolic function predict cardiac events in a hypertensive population over a period of 4 years. These out-performed traditional echocardiographic measures in a well-treated hypertensive population.

Purpose We aimed to test whether tissue Doppler assessment of LV diastolic function would predict cardiovascular (CV) mortality in the Hypertension Associated Cardiovascular Disease sub-study of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT).

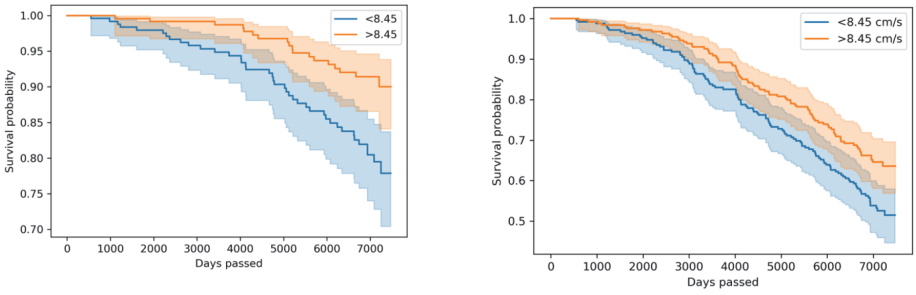
Methods ASCOT was a multicentre randomised trial with a 2x2 factorial design. Inclusion criteria for the study included hypertension and three other CV risk factors, including male sex and age over 55. Protocols, including for echocardiography, have been detailed previously.This study comprised the

519 patients recruited to the St Mary's Hospital site of the ASCOT study, who were followed for a median of 19 years with mortality flagged by the Office for National Statistics. We have used all reported deaths on or before 31st January 2019. CV deaths include deaths due to coronary heart disease (CHD), stroke and other CV causes. Echocardiography was performed one year after blood pressure control. mean tissue Doppler E' was calculated as the average of septal, lateral and inferior wall measurements over three cycles. The ratio of the transmitral Doppler E wave velocity and the composite mean of E' was used to calculate E/E' ratio. Statistical analysis was performed using Python including multivariable Cox proportional hazards regression. A two-sided P-value <0.05 was considered statistically significant.

Results After a median of 19 years (±5 years), 317 patients survived (mean age at baseline 60y, 38 female) and 202 did not (mean age 68y, 30 female). Twenty-three deaths were due to CHD, 11 were due to stroke, 27 were due to other CV causes, and 76 were due to cancer. Baseline characteristics were not significantly different between those who survived to follow



Abstract 182 Figure 1 Kaplan-Meier survival curves for mean tissue Doppler E' velocity (unadjusted)



Abstract 182 Figure 2 Kaplan-Meier survival curves by mean tissue Doppler E' velocity (unadjusted)

Abstract 182 Table 1 Multivariate analysis: hazard ratios for mean tissue Doppler E' velocity and mean tissue Doppler E/E'

		Model	Adjusted	Hazard ratio	Confidence interval	p-value
E'	Cardiovascular causes	Unadjusted		0.74	0.63-0.86	<0.005
		1	Age, sex	0.83	0.71-0.91	0.02
		2	Age, sex, systolic BP	0.83	0.71-0.98	0.03
	All causes	Unadjusted		0.86	0.80-0.94	<0.005
		1	Age, sex	0.98	0.68-1.54	0.64
		2	Age, sex, systolic BP	0.98	0.91-1.07	0.70
E/E'	Cardiovascular causes	Unadjusted		1.18	1.09-1.29	<0.005
		1	Age, sex	1.12	1.02-1.22	0.01
		2	Age, sex, systolic BP	1.11	1.00-1.22	0.04
	All causes	Unadjusted		1.06	1.00-1.13	0.04
		1	Age, sex	1.00	0.94-1.06	0.95
		2	Age, sex, systolic BP	0.98	0.92-1.04	0.53

Abstract 182 Table 2 Multivariate analysis: hazard ratios for mean TDI E/E' and mean TDI E' velocity

		Model	Adjusted	Hazard ratio	Confidence interval	p-value
E/E'	Cardiovascular causes	Unadjusted		1.18	1.09-1.29	<0.005
		1	Age, sex	1.12	1.02-1.22	0.01
		2	Age, sex, systolic BP	1.11	1.00-1.22	0.04
	All causes	Unadjusted		1.06	1.00-1.13	0.04
		1	Age, sex	1.00	0.94-1.06	0.95
		2	Age, sex, systolic BP	0.98	0.92-1.04	0.53
E'	Cardiovascular causes	Unadjusted		0.74	0.63-0.86	<0.005
		1	Age, sex	0.83	0.71-0.91	0.02
		2	Age, sex, systolic BP	0.83	0.71-0.98	0.03
	All causes	Unadjusted		0.86	0.80-0.94	<0.005
		1	Age, sex	0.98	0.68-1.54	0.64
		2	Age, sex, systolic BP	0.98	0.91-1.07	0.70

up and those who did not. Unadjusted analysis showed a strong association between CV mortality and E' (HR = 0.74, p <0.005) and E/E' (HR = 1.18, p<0.005) (table 1). The association between CV mortality and E' was attenuated slightly but persisted after adjusting for age and sex (HR = 0.83, p = 0.02) and after adjusting for age, sex and systolic BP (HR = 0.83, p = 0.03). The association between CV mortality and E/E' was attenuated but persisted after adjusting for age and sex (HR = 1.12, p = 0.01) and after adjusting for age, sex and systolic BP (HR = 1.11, p = 0.04). There was a weak association between all-cause mortality and both E/E' and E', which was null after adjusting for age and sex. Figure 1 shows unadjusted Kaplan Meier survival curves for E'.

Conclusions Tissue Doppler E' velocity and E/E' are simple echocardiographic indicators of diastolic function and predicted 19-year cardiovascular mortality in a hypertensive population independent of age, sex and systolic blood pressure.

Conflict of Interest None

183 LOW-GRADE HYPERTENSION AND CORONARY ATHEROSCLEROSIS IN MALE MASTER ENDURANCE ATHLETES

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Background Ostensibly healthy male master endurance athletes have a greater prevalence of high coronary calcium (CAC) scores and coronary artery plaque burden, compared to similar aged, healthy sedentary counterparts. A number of theories have been postulated but reasons remain unclear. Hypertension is the leading cardiovascular risk factor in the general population. The role of subclinical hypertension and the hypertensive response to exercise (HRE) in the pathophysiology of high CAC scores and other CT markers of high-risk coronary artery disease, has not previously been reported in the healthy master endurance athlete population.

Methods A cohort of 214 low Q-risk, male master (40-65 years) endurance athletes were prospectively evaluated between February 2018 and September 2019. All athletes were free from any cardiovascular risk factors, symptoms and any relevant health conditions. Clinical evaluation included cycle ergometer cardiopulmonary exercise test, ambulatory BP monitoring and coronary computed tomogram angiography (CCTA). The CCTA assessed CAC score, coronary stenosis, defined as >50%, plaque morphology (i.e. calcified, soft or mixed), and markers of plaque vulnerability including spotty calcification, ruptured plaque, positive remodelling, low attenuation plaque and napkin ring sign. Resting BP, HRE and ambulatory hypertension were defined in accordance with the latest European Society of Cardiology (ESC) guidelines and are detailed in table 1.

Results All athletes (mean age 51, SD 70.1) exercised for at least 6 hours per week (median 8.5 hours) for a median of 15 years of either cycling, running or swimming or any combination of the three. The median Q risk score was 3.5%. A quarter (26%) of athletes were hypertensive at rest. A significant proportion of athletes exhibited exercise (12%) and ambulatory hypertension (41%). A CAC score >100 Agatston units (AU) was present in (16%) of athletes, with 5% exhibiting significant coronary stenosis. A third (32%) of all plaque morphologies were non-calcified (27% mixed and 5% soft) and 13% had additional plaque vulnerability markers. The most prevalent marker was spotty calcification (11.3%), followed by positive remodelling (8.9%). Logistic regression identified the BP parameters predictive for a CAC score >100 AU, significant stenosis and plaque vulnerability markers. A 5 mmHg increase in resting systolic BP, maximal systolic exercise BP and 24 hour and nocturnal ambulatory systolic were associated with both significant stenosis and plaque vulnerability markers and a 5 mmHg increase in resting systolic BP (SBP) and 24 hour and nocturnal ambulatory SBP were associated with a CAC score >100 AU. The p values, odds ratios (OR) and respective 95% confidence intervals are detailed in table 3. BP parameters were not associated with calcified plaque by multinomial logistic regression, however for every 5mmHg increase in maximal exercise SBP, resting SBP and 24-hour and nocturnal ambulatory SBP, the relative risk (RR) of mixed morphology plaque was increased by 19.27% (p=0.001, CI

Abstract 183 Table 1 ESC definitions of hypertension[3]

Resting Blood Pressure	Systolic (mmHg)		Diastolic (mmHg)
High Normal	130-139	and/or	85-89
	Hypertension	≥140	and/or ≥90
- Grade 1	140-159	and/or	90-99
- Grade 2	160-179	and/or	100-109
- Grade 3	≥180	and/or	≥110
	Ambulatory Hypertension (any of following on 24hour monitoring)		
- 24 hour	≥130	and/or	≥80
or			
- Diurnal	≥140	and/or	≥90
or			
- Nocturnal HRE	≥120 ≥220	and/or	≥70

Abstract 183 Table 2 Prevalence of hypertension according to ESC guideline classifications

Grade of hypertension	Resting Hypertension
Total Abnormal i.e. ≥130/85 mmHg (%)	115 (53.74)
Total Hypertensive (%)	55 (25.71)
High Normal (%)	60 (28.04)
Grade 1 (%)	44 (20.56)
Grade 2 (%)	10 (4.67)
Grade 3 (%)	1 (0.47)