170 ETHNIC DIFFERENCES IN PHENOTYPIC EXPRESSION OF HYPERTROPHIC CARDIOMYOPATHY

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N Sheikh, M Papadakis, N Chandra, H Raju, A Zaidi, S Ghani, M Muggenthaler, S Gati, S Sharma. *St. George's University of London, London, UK*

Purpose Hypertrophic Cardiomyopathy is a heterogeneous condition with variable phenotypic expression. Current studies are based on predominantly Caucasian cohorts (white patients; WP), therefore the phenotypic manifestations of HCM in individuals of African/Afro-Caribbean origin (black patients; BP) are not fully realised. Data in athletes and hypertensive patients indicate that black ethnicity is associated with a greater prevalence of repolarisation abnormalities on the ECG as well as a greater magnitude of left ventricular hypertrophy (LVH), highlighting the importance of defining the HCM phenotype in this ethnic group.

Methods Between 2001 and 2010, 155 consecutive patients with HCM (52 BP, 103 WP) were assessed in 3 specialist cardiomyopathy clinics in South London. All individuals underwent comprehensive cardiac evaluation including 12-lead ECG and echocardiography. Patients subject to therapeutic interventions potentially affecting repolarisation patterns were excluded.

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	Black HCM (n=52)	white HCM (n=103)	p Value
Demographics			
Age of diagnosis (years)	48.1±17.1	50.5±16.5	0.552
Gender (males)	61.5%	62.1%	0.942
FH of HCM/SCD	34.6%	32.0%	0.747
NYHA functional class III or IV	7.7%	7.8%	0.987
Patients on treatment	55.8%	46.6%	0.281
B-blockers	28.8%	39.1%	0.445
Calcium antagonists	26.9%	12.6%	0.026
Amiodarone	7.7%	1.9%	0.080
Diuretics	13.5%	9.7%	0.480
Disopyramide	3.8%	9.7%	0.197
Intracardiac defibrillator in situ	5.8%	5.8%	0.989
Echocanliographic characteristics			
Ao (mm)	31.3±3.7	33.2±5.8	0.123
LA (mm)	40.9±7.3	39.9±7.3	0.593
LVED (mm)	44.0±6.1	44.4±6.1	0.787
mLVWTd (mm)	17.3±4.9	18.8±4.1	0.069
LV mass (g)	279.6±106.5	287.6±112.7	0.767
FS (%)	40.4±9.1	39.8±8.3	0.641
E wave (m/s)	0.70±0.18	0.74±0.20	0.443
A wave (m/s)	0.67±0.18	0.66±0.27	0.851
E/A	1.11±0.44	1.22±0.58	0.422
SAM	23.1%	37.9%	0.064
LVOT gradient = 30 mm Hg	23.1%	34.0%	0.163
LVH pattern			
ASH	25%	57.3%	0.004
Concentric	44.2%	30.1%	
Apical	28.8%	11.7%	
No hypertrophy	1.9%	1.0%	
Echocanliographic characteristics			
LVH (Sokolow & Lyon)	53.8%	35.9%	0.033
Left atrial enlargement	44.2%	49.5%	0.534
Pathological Q waves	9.6%	23.3%	0.039
Left axis deviation	11.5%	17.2%	0.270
Inverted T-waves	82.7%	69.9%	0.086
T-wave inversions in V1–V4	3.8%	3.9%	0.991
T-wave inversions in inferior leads	1.9%	5.8%	0.269
T-wave inversions in lateral leads	76.9%	60.2%	0.038
Deep T-wave inversions	69.2%	51.5%	0.035
ST-segment elevation	9.6%	9.7%	0.985
ST-segment depression	50%	35.0%	0.071

Results Black patients revealed significantly different echocardiographic patterns of LVH, with more concentric (44.2% vs 30.1%) and apical (28.8% vs 11.7%) hypertrophy compared to WP who exhibited more asymmetric septal hypertrophy (57.3% vs 25.0%) (p=0.004). Black patients exhibited a similar magnitude of LVH compared to WP (17.3±4.9 vs 18.8±4.1 mm, p=0.069). Relating to ECG repolarisation abnormalities, BP exhibited more T wave inversions in the lateral leads (76.9% vs 60.2%, p=0.038) and deep (≥−0.2 mV) T-wave inversions (69.2% vs 51.5%, p=0.035). Black patients also tended to display more ST segment depression (50.0% vs 35.0%, p=0.071), although this was not statistically significant. In contrast, WP had significantly more pathological Q waves (23.3% vs 9.6%, p=0.039).

Conclusions Ethnicity appears to exert a significant effect on ECG and echocardiographic patterns in patients with HCM. A significant proportion of black patients exhibit concentric LVH, highlighting the diagnostic challenges in distinguishing HCM from hypertensive heart disease and physiological adaptation to exercise in black individuals. The greater prevalence of deep T wave inversions and T wave inversions in the lateral leads underscores the importance of further evaluation of black individuals with such ECG repolarisation abnormalities, which may represent the initial expression of HCM.

171 THE RIGHT VENTRICLE OF THE ENDURANCE ATHLETE: THE RELATIONSHIP BETWEEN MORPHOLOGY AND DEFORMATION

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[1]D Oxborough, [2]R Shave, [3]G Whyte, [1]K Birch, [4]N Artis, [3]K George, [5]S Sharma. *[1]University of Leeds, Leeds, UK; [2]UWIC, Cardiff, UK; [3]Liverpool John Moores University, Liverpool, UK; [4]Leeds Teaching Hospitals NHS Trust, Leeds, UK; [5]St Georges University Hospital, London, UK*

Introduction It is well established that endurance exercise results in cardiac adaptation including eccentric hypertrophy of the left ventricle which can complicate the differential diagnosis of the athletic heart from some cardiac pathologies that may pre-dispose to sudden cardiac death. The impact of physiological conditioning on RV structure and function, and a similar diagnostic challenge with arrhythmogenic right ventricular cardiomyopathy (ARVC), has received less attention. A recent guideline paper from the American Society of Echocardiography (ASE) has provided a range for normal RV dimensions and functional deformation. These guidelines suggest the RV inflow (RVI) should be <42 mm while the proximal outflow tract (RVOT) <35 mm. A recent paper also suggested that an RVOT dimension >36 mm or 21 mm/m^2 is a major criterion for the diagnosis of ARVC and furthermore longitudinal RV deformation has been shown to be impaired in these patients. In view of this, the aims of this study are twofold:

1. To provide a range of absolute values for RV dimensions in 102 endurance athletes as well as providing a range of data indexed for body surface area (BSA).
2. To provide normal athlete data for indices of RV strain (ε) and strain rate (SR).

Methods and Results One hundred and two (102) endurance athletes (86 males and 16 females) with a broad age range (mean ± SD age (range)=36 ± 11 (21–71) years) volunteered and were consecutively enrolled in the study. All subjects were either endurance runners or cyclists and were scanned at peak condition. Echocardiography provided measurements of RVI, RV length, RVOT and RV diastolic area (RVDarea). A 2D strain technique was utilised to provide indices of RVε and systolic and diastolic SR. The values for RVI

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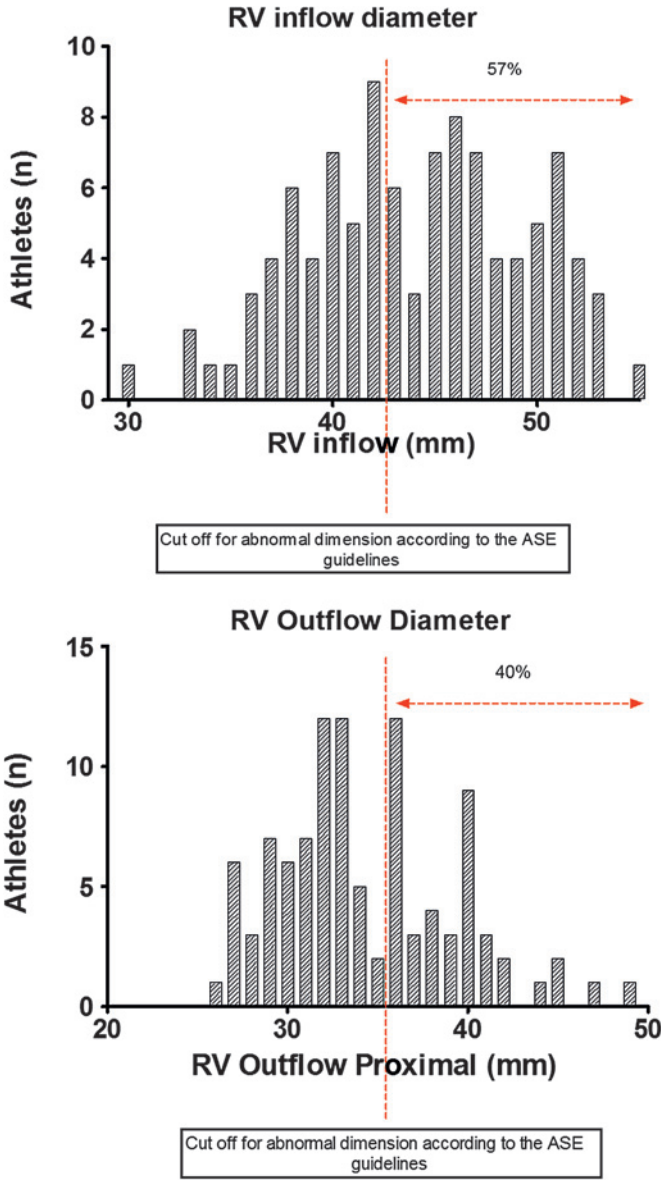
ranged from 30 to 55 mm with 57% of the population having values greater than the normal range. Proximal RVOT ranged from 26 to 49 mm with 40% of the population above the normal range. 28% of the population had RVOT values greater than the proposed "major criteria" for ARVC. RV length ranged from 70 to 110 mm and RVDarea from 13 to 38 cm^2 with values falling above ASE cut-offs in 69% and 59% of the population, respectively. When indexed (ratio scaling) for BSA proximal RVOT ranged from 13 to 25 mm/m^2 with 6% of the population meeting the major criteria for ARVC. Peak RVε ranged from −18 to −41% and peak RV SRS′ from −0.75 to −2.65 l/s, consistent with normal ranges proposed by the ASE. RV diastolic deformation indices displayed marked individual variability with a dominant SRE′ (mean ± SD=2.0±0.61 l/s) and smaller SRA′ (1.25±0.56 l/s).

Conclusion RV dimensions in endurance athletes are higher than those proposed as "normal" and likewise may be consistent with the criteria for ARVC. Despite this enlargement, RV function in endurance athletes is preserved and therefore the role of RV strain imaging may provide additional diagnostic value in differentiating physiological from pathological adaptation.



Abstract 171 Figure 1

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Parameter	Mean ± SD (range)	ASE Normal Values	Indexed (ratio scaling) for BSA
RVOT (mm)	34±5 (26 to 49)	<35	17±3 (13 to 25) mm/m^2
RVDI (mm)	44±5 (30 to 55)	<42	22±3 (15 to 30) mm/m^2
RV Length (mm)	92±9 (70 to 110)	<86	45±5 (32 to 61) mm/m^2
RVDarea (cm^2)	26±5 (13 to 38)	<25	12.8±2 (8.7 to 17.6)
RV ε (%)	−27±6 (−18 to −41)	−18 to −39	N/A
RVSRS′ (l/s)	−1.53±0.43 (−0.75 to −2.63)	0.7 to 2.54	N/A
RVSRE′ (l/s)	2.0±0.61 (0.87 to 3.76)	N/A	N/A
RVSRA′ (l/s)	1.25±0.56 (0.28 to 2.88)	N/A	N/A
RVSR E′/A′	1.89±1.03 (0.49 to 7.25)	N/A	N/A

172 INCREASING ANTI-OXIDANT CAPACITY REVERSES IRON OVERLOAD MEDIATED DYSFUNCTION IN CARDIOMYOCYTES

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[1]F R Millar, [1]D T Baptista-Hon, [2]S C O′Neill, [1]M E Díaz. *[1]University of Edinburgh, Edinburgh, UK; [2]University of Manchester, Manchester, UK*

Introduction Iron overload-cardiomyopathy (IOCM) is an increasing clinical problem worldwide. 70% of patients who receive compulsory blood transfusion die of IOCM, with increased susceptibility to arrhythmias and sudden death. We have previously found that iron exposure impairs cardiomyocyte Ca homeostasis. However, the cellular mechanisms responsible are unknown. Iron is known to participate in the Fenton reaction to produce reactive oxygen species (ROS), which mediate oxidative damage. We therefore tested the hypothesis that increasing the anti-oxidant capacity of cardiomyocytes, with the ROS scavenger Tempol, could be cardioprotective in the presence of iron.

Methods Single rat ventricular cardiomyocytes were loaded with fluo-3 to monitor intracellular Ca changes upon stimulation while bathed in control Tyrode solution and after adding ferrous iron (iron II). Sarcoplamic reticulum (SR) Ca load and sarcolemmal Ca extrusion rates were estimated during exposure to caffeine, which empties SR Ca stores. The ROS scavenger tempol was used to dissect ROS-mediated pathways from the direct effects of iron II on Ca handling. Data are provided as mean±SEM. Significance was tested using paired student t test and defined as $p<0.05$.

Results Iron II exposure significantly increases systolic Ca transient amplitude (mean increase 82.8±21.8%, n=9) and causes spontaneous arrhythmogenic Ca release events (SACRE). These changes corresponded with increased SR Ca content (mean increase 21.0±5.7%, n=8), which is known to impact on systolic Ca release and spontaneous activity in cardiomyocytes. Sarcolemmal Ca extrusion rate was also significantly reduced upon iron II exposure (measured as the rate of fall of the caffeine response; mean decrease 48.7±5.5%, n=8), consistent with an overall gain of Ca by the cardiomyocyte. The onset of these Ca disruptions was significantly delayed in the presence of the ROS scavenger tempol ($p<0.001$). Without tempol, SACRE onset occurred after 6.9±0.6 min (n=22) following iron II exposure. The same manoeuvre in in tempol delayed the onset of SACRE to 17.8±1.8 min (n=7). Furthermore, increasing ROS scavenging reversed the increase in systolic Ca transient amplitude, as well as SACRE upon washout of iron II. In contrast, in cardiomyocytes not exposed to tempol, the effects of iron II were irreversible.

Conclusions Our data show that iron II disrupts cardiomyocyte Ca handling. This is mediated via inhibition of sarcolemmal Ca extrusion, leading to SR Ca overload and SACRE. These are the initiators of most fatal non-reentrant arrhythmias and cardiac sudden death in

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