2 THE RELATIONSHIP BETWEEN LEFT VENTRICULAR OUTFLOW TRACT GRADIENT AND SUUDEN CARDIAC DEATH IN CHILDHOOD HYPERTROPHIC CARDIOMYOPATHY

Gabrielle Norrish. *Institute of Cardiovascular Science*

10.1136/heartjnl-2020-BCS.2

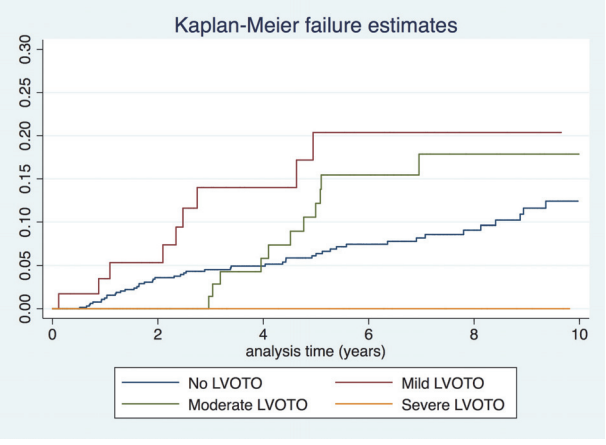
Introduction The most common mode of death in childhood hypertrophic cardiomyopathy (HCM) is sudden cardiac death (SCD). Left ventricular outflow tract (LVOT) obstruction is an established risk factor for SCD in adults with the disease. In contrast, the prognostic implications of left ventricular outflow tract obstruction (LVOTO) in childhood disease is unclear, with recent studies suggesting that it may have an inverse relationship with the risk of SCD. The aim of this study was to explore the role of LVOTO and the risk of SCD in childhood HCM.

Methods A multi-centre, retrospective, longitudinal cohort of 871 children (diagnosed with HCM <16 years of age) was used to explore the relationship between SCD and LVOTO (LVOT gradient ≥30mmHg).

Results 189 patients (23%) had LVOTO, which was mild (30-50mmHg), moderate (50-100mmHg) or severe (>100mmHg) in 58 (6.7%), 98 (11.3%) and 33 (3.8%), respectively. The risk of SCD showed an inverse relation to LVOT gradient severity compared to those with no obstruction: mild HR 1.75 (95% CI 0.89-3.44), moderate HR 1.04 (95% 0.55-1.98), and severe HR 0.7 (0.36-1.35) [figure 1]. On univariable analysis [table 1] LVOTO was associated with heart failure symptoms (NYHA>1) [p <0.001], maximal wall thickness (MWT) [p <0.001], left atrial (LA) diameter [p <0.001], and future myectomy occurring during follow up [p <0.001]. The inverse relationship observed was not altered by the presence or absence of other traditional risk factors.

Conclusions LVOT gradient has a complex relationship with the risk of SCD in childhood with multiple contributing factors. The pathophysiological mechanisms behind this observation need further exploration, which may be limited by low patient numbers.

Conflict of Interest Nil



Abstract 2 Figure 1

Abstract 2 Table 1 Demographics and clinical characteristics by LVOT gradient

	<30mmHg (n=682)	30-50mmHg (n=58)	50-100mmHg (n=98)	>=100mmHg (n=33)	P value
B Blocker therapy	288 (42.3%)	30 (51.7%)	50 (51.6%)	19 (57.6%)	0.085
NYHA>1	137 (20.5%)	17 (29.3%)	37 (37.8%)	14 (43.8%)	<0.001
NSVT	39 (6.8%)	5 (10.4%)	7 (8.5%)	2 (8.7%)	0.771
Unexplained syncope	70 (10.3%)	9 (15.5%)	6 (6.1%)	4 (12.1%)	0.296
Z score MWT	10.3 (+/-6.6)	15.1 (+/-7.6)	15 (+/-8.1)	16.6 (+/-8.3)	<0.001
Z score LA	1.7 (+/-2.3)	2.8 (+/-2.4)	3 (+/-2.4)	3.4 (+/-2.5)	<0.001
Myectomy during follow up	20 (2.9%)	9 (15.5%)	29 (29.9%)	12 (36.4%)	<0.001
SCD event	54 (7.9%)	11 (18.9%)	13 (13.2%)	1 (3.0%)	0.009
Incidence of SCD/100 pt years	1.40 (1.06-1.80)	3.5 (1.90-6.30)	2.08 (1.21-3.58)	0.42 (0.06-2.98)	0.219

3 RESIDENTIAL EXPOSURE TO FINE PARTICULATE MATTER AIR POLLUTION IS ASSOCIATED WITH IMPAIRED CARDIAC PHENOTYPES IN DILATED CARDIOMYOPATHY

[1]Upasana Tayal, [1]Daniela Fecht, [1]Marc Chadeau, [2]John Gulliver, [1]James Ware, [3]Stuart Cook, [1]Sanjay Prasad. *[1]Imperial College London; [2]University of Leicester; [3]National University of Singapore*

10.1136/heartjnl-2020-BCS.3

Background Air pollution might contribute to adverse ventricular remodelling in healthy populations. A recent study on a cohort of 500,000 participants (UK Biobank) showed that residential exposure to particulate matter with aerodynamic diameter <2.5 μm (PM2.5) and nitrogen dioxide (NO2) was associated with cardiac chamber dilatation and increased left ventricular mass.

Dilated cardiomyopathy (DCM) has marked structural and functional phenotypic heterogeneity. The biological basis for this is undefined, but environmental factors are plausible phenotypic modifiers.

Abstract 3 Table 1 Hazard ratio for primary composite outcome and exposure to small particle pollutants. Volumes and masses are indexed to body surface area. Hazard ratios presented with 95% confidence intervals

Exposure	Absolute left ventricular ejection fraction (%)	Indexed left ventricular mass (g/m^2)	Indexed left ventricular end diastolic volume (ml/m^2)	Absolute right ventricular ejection fraction (%)	Indexed right ventricular end diastolic volume (ml/m^2)
PM2.5 per 1 unit (μg/m^3)	-0.6 (-1.1 to 0.0), p=0.05	1.4 (0.18 to 2.60), p=0.02	0.39 (-1.3 to 2.1), p=0.64	-0.03 (-0.7 to 0.6), p=0.93	-1.2 (-2.3 to -0.03), p=0.04
NO2 per 1 unit (μg/m^3)	-0.1 (-0.2 to -0.04), p= 0.004	0.27 (0.10 to 0.43), p=0.001	0.20 (-0.03 to 0.4), p=0.09	-0.03 (-0.11 to -0.06), p=0.52	-0.06 (-0.22 to 0.09), p= 0.42

Heart: first published as 10.1136/heartjnl-2020-BCS.3 on 17 July 2020. Downloaded from http://heart.bmj.com/ on April 26, 2024 by guest. Protected by copyright.

We sought to evaluate whether air pollution could be an important environmental modifier in DCM.

Methods Prospectively recruited patients with DCM underwent advanced phenotyping by cardiac magnetic resonance. Patients were followed up for the primary composite end-point of cardiovascular mortality, major arrhythmic events and major heart failure events.

Long-term air pollution exposure estimates prior to the year of DCM diagnosis were assigned to each residential postcode centroid (on average 12 households). Annual average maps were available for NO2 concentrations in 2009 at 200m resolution and PM2.5 in 2010 at 100m resolution. Postcode centroids (x,y locations) were overlaid with each air pollution surface to obtain NO2 and PM2.5 estimates for each postcode and concentrations extrapolated to the year of diagnosis using information from the national air pollution monitoring network.

Results From the total cohort of 716 DCM patients enrolled to the study, 678 patients had postcodes which could be assigned a geographical location and air pollutant estimates.

The median PM2.5 concentration was 15.4 (14.3 – 16.3) µg/m3 and the median NO2 concentration was 32.4 (24.1 – 40.6) µg/m3.

Higher residential exposure to PM2.5 and NO2 was associated with increased left ventricular mass in DCM patients (table 1). Higher residential exposure to NO2 was associated with reduced left ventricular ejection fraction (Table 1).

There was no association between exposure to PM2.5 levels or NO2 levels and cardiovascular outcomes (NO2 Hazard ratio 0.99, 95% confidence intervals (CI) 0.98-1.01, p= 0.90; PM2.5 hazard ratio 1.0, 95% CI 0.89-1.25, p= 0.54).

Conclusion Fine particulate matter air pollution has an adverse effect on cardiovascular phenotypes amongst patients with DCM suggesting air pollutants could be an environmental modifier of DCM. There was no apparent effect of fine particulate matter on major cardiovascular outcomes in this cohort. Future studies should explore whether air pollution contributes to DCM amongst at risk individuals.

Conflict of Interest None

4 A PROSPECTIVE STUDY OF HYPERTROPHIC CARDIOMYOPATHY AND SLEEP DISORDERED BREATHING

Shahid Karim, Shreyas Venkataraman. *Mayo Clinic*

10.1136/heartjnl-2020-BCS.4

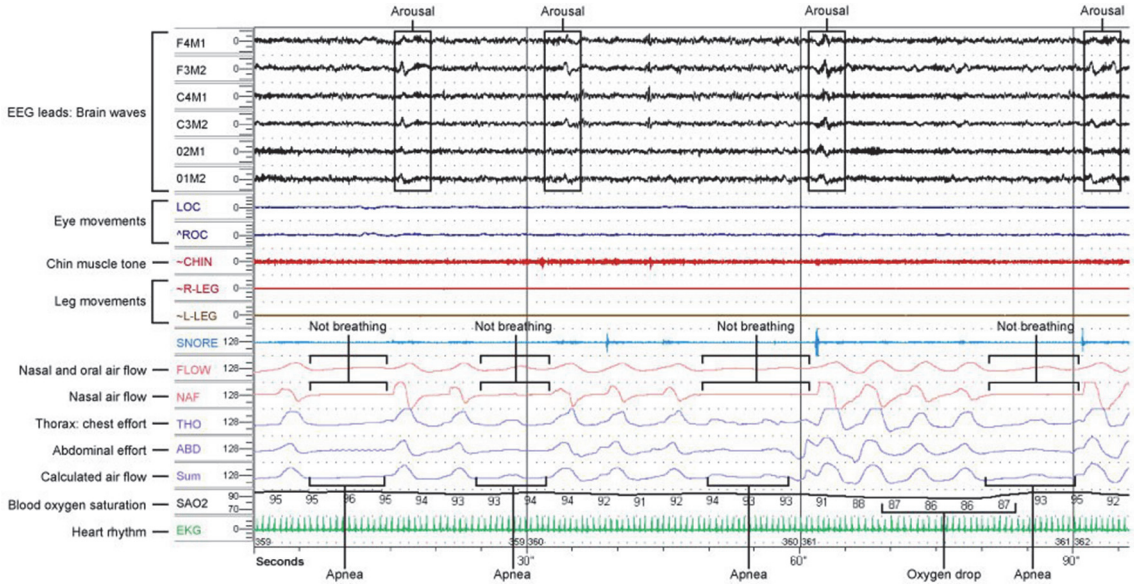
Background Hypertrophic cardiomyopathy (HCM) represents the most common inherited cardiomyopathy characterized by otherwise unexplained left ventricular hypertrophy. Sleep disordered breathing (SDB) (including both central and obstructive sleep apnea) is considered to be an important and potentially reversible cause of cardiovascular disease progression. This prospective trial aims to define the prevalence of SDB within patients diagnosed with HCM using gold standard polysomnography (PSG). Previous studies have suggested a prevalence of SDB in the general population of 25.5%.

Methods Previous trials have been retrospective studies and have used overnight oximetry analysis to find the prevalence of SDB in HCM using portable, at-home monitors, however overnight oximetry is unable to accurately delineate between central and obstructive sleep apnea – both of which vary significantly in terms of prognosis and management - and is not as sensitive nor specific in diagnosing or classifying SDB as polysomnography assessment.

An ongoing prospective analysis of 85 patients diagnosed with HCM was performed at Mayo Clinic using PSG. Apnea is defined as the absence of inspiratory airflow for at least 10 seconds. Hypopnea is defined as a decrease in airflow lasting 10 seconds or longer associated with a desaturation >4%. Apnea Hypopnea Index (AHI) – the number of events per hour was assessed using PSG. SDB is defined as an AHI>5/hour of sleep.

Results Of 85 HCM patients examined using PSG, 49 were found to have an AHI>5/hour. Average AHI was 20.0, Standard Deviation 23.3, Interquartile range 3.3-28.2.

15 patients had central sleep apnea defined as the absence of inspiratory effort for at least 10 seconds. 17 patients had obstructive apneas. 18 patients had severe SDB (AHI>30/hour).



Abstract 4 Figure 1

Heart: first published as 10.1136/heartjnl-2020-BCS.3 on 17 July 2020. Downloaded from http://heart.bmj.com/ on April 26, 2024 by guest. Protected by copyright.