259

ASYMMETRICAL SEPTAL HYPERTROPHY IS ASSOCIATED WITH MEAN ARTERIAL BLOOD PRESSURE IN HEALTHY ADULTS: DATA FROM HIGH RESOLUTION 3D CARDIAC MRI

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Background Left ventricular hypertrophy (LVH) is the result of complex interactions between genes and environmental factors, and an independent risk factor for all-cause mortality. Clinically, it is important to differentiate 'physiological' LVH, such as in hypertension, from pathological LVH as found in hypertrophic cardiomyopathy.

Standard methods of cardiac phenotyping such as echocardiography or 2D cardiac magnetic resonance (CMR) provide only macroscopic descriptors of IV mass (LVM) and function. These methods are underpowered for population wide study of regional wall thickness variability.

Methods and Results 187 healthy multi-ethnic volunteers (105 female, age range 18–72, mean age 40) were recruited into a sub-study of the UK GenScan project. Exclusion criteria included established history of cardiovascular disease, hypertension, diabetes or hypercholesterolemia.

Subjects were phenotyped by CMR using a 1.5T Philips Achieva system with a 32 element cardiac phased-array coil. High resolution 3D cine imaging of the LV was obtained with a voxel size of 2x2x4 mm and 20 cardiac phases.

Three blood pressure (BP) measurements were taken and averaged (in mmHg): Mean systolic BP 123 (range 90–186), mean diastolic BP 80.4 (range 47–109).

The high resolution 3D images were automatically segmented using training data from 10 healthy volunteers in which the myocardium had been manually labelled at end-diastole and end-systole (Figure 1). The 187 3D segmentations were exactly co-registered with each other so that corresponding points within the myocardium could be accurately compared within the cohort. Mean wall thickness was calculated at each point as the distance between the endocardial and epicardial surfaces. Automated measurements were validated against manual readings using standard clinical software (Philips Extended Workspace). LV end-diastolic volumes and LVM measurements were highly correlated ($R^2=.98$ and $R^2=0.93$ respectively).

Mass univariate analysis was performed on the co-registered 3D models with wall thickness as the dependent variable. Pooling data from all the subjects, we plotted wall thickness at each 3D coordinate as a linear function of Mean Arterial Pressures (MAP) with gender and age held constant. The 3D models showed that blood pressure associated LVH begins asymmetrically within the mid-septum and progressively extends towards the base and apex with rising MAP (Figure 2). The lateral wall and apex are relatively spared.

Conclusions Whole-heart high-resolution 3D CMR provides new opportunities for quantitative phenotyping of the heart and

understanding the genetic and environmental determinants of heart disease. In this study we used these techniques to show the pattern of progressive asymmetrical septal hypertrophy that is associated with increasing blood pressure even within the general population.

Figure 1

Figure 2