Novel insights into abnormal haemodynamics in hypertrophic cardiomyopathy from 4D flow cardiac magnetic resonance

Z. Ashkir[1], S. Myerson[1], A.J. Lewandowski[2], M. Mahmod[1], R. Ariga[1], C. Carhall[3], T. Ebbers[3], H. Watkins[4], S. Neubauer[1], B. Raman[1]

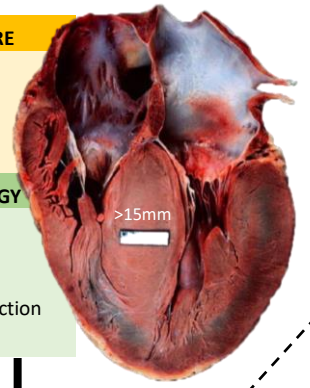
[1]Oxford Centre for Clinical Magnetic Resonance Research (OCMR), Division of Cardiovascular Medicine, Radcliffe Department of Medicine, University of Oxford. [2]Oxford Cardiovascular Clinical Research Facility (CCRF), Division of Cardiovascular Medicine, Radcliffe Department of Medicine, University of Oxford. [3]Division of Diagnostics and Specialist Medicine, Department of Health Medicine and Caring Sciences, Linköping University, Linköping, Sweden.
[4]Radcliffe Department of Medicine, Division of Cardiovascular Medicine, University of Oxford.

Rationale

Hypertrophic cardiomyopathy (HCM)

Abnormal STRUCTURE
- Hypertrophy
- Fibrosis
- Valve dysfunction
- Atrial dilatation

Abnormal PHYSIOLOGY
- Impaired energetics
- Hypercontractility
- Diastolic dysfunction
- Microvascular dysfunction
- Arrhythmias



Deranged blood flow haemodynamics

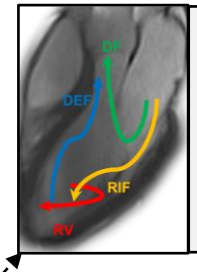
Heart failure
Exercise intolerance
Syncope

Aims

We aimed to:
1. characterise abnormalities in ventricular haemodynamics in HCM using 4D flow CMR
2. describe their relationship with phenotypic and metabolic perturbations that underpin HCM.

Methods

4D flow CMR: Sensitive to early changes in blood flow and cardiac function



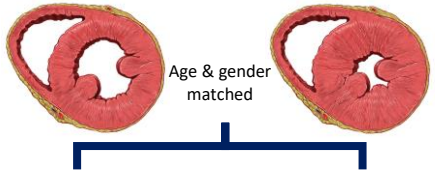
Direct Flow (DF)
Blood that transits the ventricle within one cardiac cycle
Retained Inflow (RIF)
Blood that enters the LV during diastole but is retained for at least one cycle
Delayed Ejection Flow (DEF)
Blood already in the LV during diastole that leaves during systole
Residual Volume (RV)
Blood that remains in the LV for at least two cycles

Ejected: DF+DEF

Non-Ejected: RIF+ RV

20 healthy controls

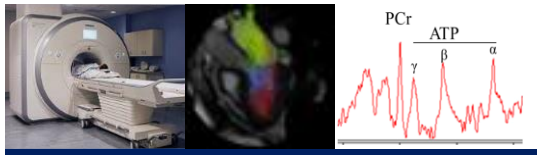
38 HCM patients



Age & gender matched

Echocardiography

Diastolic function



CMR (4D + ^{31}Phosphorus MRS)

Flow component analysis
Distribution , End-diastolic kinetic energy (ED KE)

Myocardial energetics
phosphocreatine-to-ATP concentration (PCr/ATP) ratio

Results

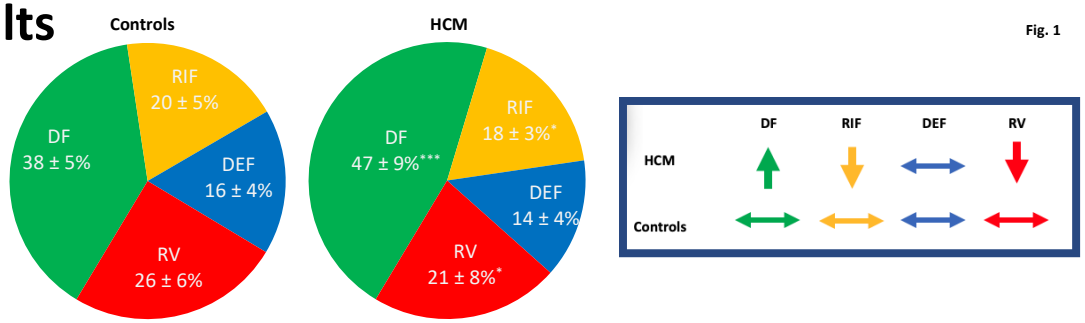


Fig. 1

Flow component distribution in healthy controls and HCM

- HCM patients - ↑ Direct Flow (DF) proportion (47±9% vs 38±5%, p<0.001)
  ↓ Retained Inflow (18±3% vs 20±5%, p=0.016) and Residual Volume (21±8% vs 26.6%, p=0.03) proportions. **(Fig. 1)**
- Direct Flow proportion moderately correlated with LV wall thickness (rho=.548, p<0.001) **(Fig. 2)** and LV mass index (rho=.477, p=<0.001).

Fig. 2

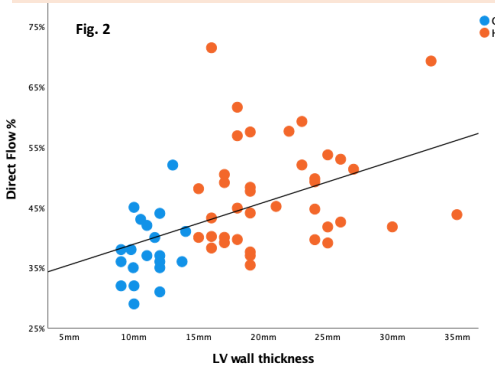
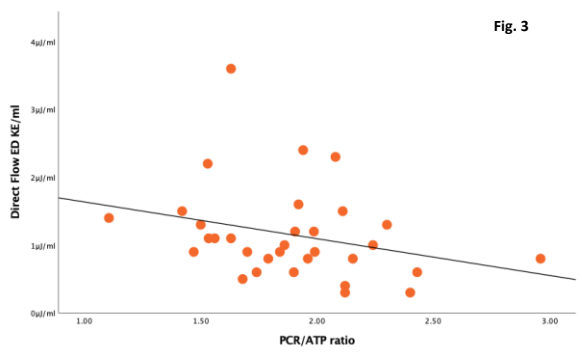


Fig. 3



End-diastolic kinetic energy and relationship with myocardial energetics

- NO difference in component end-diastolic kinetic energy between groups.
- The phosphocreatine-to-ATP ratio correlated inversely with all components of end-diastolic kinetic energy for both groups.
- In HCM patients, this was strongest for the ejection components - Direct Flow (rho -.362, p=0.04) **(fig 3)** and Delayed Ejection Flow (rho -.365, p=0.04).

What does this mean?

- HCM generates greater **Direct Flow** proportion at the expense of non-ejection components (**Retained Inflow** and **Residual Volume**) to maintain stroke volume.
- Correlation of **Direct Flow** with **LV wall thickness** & **mass index** suggests that component distribution changes are closely linked to disease severity in HCM.
- Inverse relationship between **component end-diastolic kinetic energy & PCr/ATP ratio** suggests that with greater myocardial energy depletion, less kinetic energy can be transferred into useful potential energy during active relaxation.