

ORAL PRESENTATION

Open Access

# Derangement of cardiac energy metabolism is acutely exacerbated during exercise in hypertrophic cardiomyopathy, independent of hypertrophy or late gadolinium burden

Sairia Dass<sup>1\*</sup>, Lowri E Cochlin<sup>2</sup>, Joseph Suttie<sup>1</sup>, Cameron Holloway<sup>1</sup>, Christopher T Rodgers<sup>1</sup>, Damian Tyler<sup>2</sup>, Theodoros Karamitsos<sup>1</sup>, Kieran Clarke<sup>2</sup>, Hugh Watkins<sup>1</sup>, Stefan Neubauer<sup>1</sup>

From 15th Annual SCMR Scientific Sessions  
Orlando, FL, USA. 2-5 February 2012

## Summary

This work demonstrates that cardiac energetics is further impaired during exercise in hypertrophic cardiomyopathy. This may be a possible reason for exercise related death in HCM.

## Background

In hypertrophic cardiomyopathy (HCM), sarcomere mutations increase the energy cost of contraction. Impaired resting cardiac energetics as measured by phosphocreatine/adenosine triphosphate (PCr/ATP) using <sup>31</sup>Phosphorus MR Spectroscopy (<sup>31</sup>P MRS) has been documented in animal models and patients.

We hypothesize that: 1. Cardiac energetics are further impaired acutely during exercise in HCM, which does not occur in normals or athletes (physiological hypertrophy); 2. This impairment is not related to the degree of hypertrophy or late gadolinium enhancement (LGE) burden.

## Methods

Cardiac <sup>31</sup>P MRS (3T) was performed in 35 HCM patients, 12 athletes and 20 normal controls (all age- and gender-matched) at rest and during 8 minutes of prone leg exercise with 2.5 kg weights attached to both legs. Cine and LGE images were also acquired.

## Results

Increases in rate pressure product with exercise were similar: normal  $72 \pm 44\%$ ; HCM  $73 \pm 38\%$ ; Athlete  $75 \pm 47\%$ .

There was no difference in resting PCr/ATP between normals ( $2.14 \pm 0.36$ ) and athletes ( $2.04 \pm 0.32$ ,  $P = 0.36$ ). Resting PCr/ATP was significantly reduced in HCM, ( $1.71 \pm 0.35$ ,  $P < 0.01$  compared to normal and athletes, figure 1).

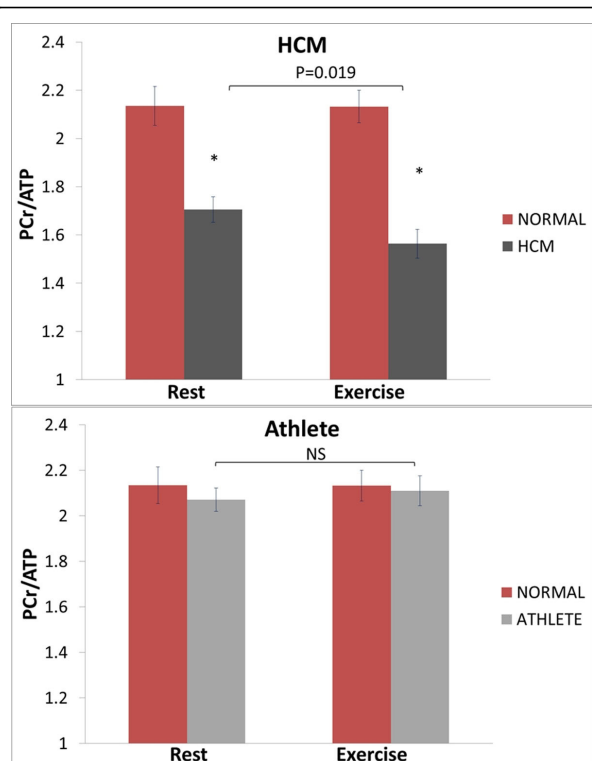
During exercise, there was a further reduction in PCr/ATP in HCM ( $1.56 \pm 0.31$ ,  $P < 0.05$ ), but no significant change in normals ( $2.13 \pm 0.34$ ,  $P = 0.98$ ), and athletes ( $2.09 \pm 0.50$ ,  $P = 0.63$ , figure 1). The change of PCr/ATP during exercise in HCM ( $-0.14 \pm 0.34$ ) was significantly different ( $P < 0.05$ ) from the change in normals ( $+0.05 \pm 0.27$ ).

LV mass index was higher in HCM ( $90 \pm 14 \text{ g/m}^2$ ) and athletes ( $92 \pm 19 \text{ g/m}^2$ ) compared to normal ( $65 \pm 10 \text{ g/m}^2$ )  $P > 0.05$ . There was no correlation between cardiac mass index and rest PCr/ATP ratios (HCM:  $R = 0.01$ ,  $P = \text{NS}$ ; athletes:  $R = 0.22$   $P = \text{NS}$ ) or change in PCr/ATP with exercise.

In HCM, average wall thickness at voxel placement for PCr/ATP was  $18 \pm 6 \text{ mm}$  (range 7.8-28.8 mm). This did not correlate with resting PCr/ATP or change in energetics with exercise. Wall thicknesses were normal in the athlete group,  $9 \pm 2 \text{ mm}$ .

Normals and athletes had no LGE. In HCM, the average LGE  $> 2\text{SD}$  in the mid ventricular septum was  $24 \pm 15\%$ . LGE correlated weakly with resting PCr/ATP ratio, ( $R = -0.35$ ,  $P = 0.04$ ), and did not correlate with absolute exercise or change in PCr/ATP with exercise.

<sup>1</sup>Department of Cardiovascular Medicine, University of Oxford, Oxford, UK  
Full list of author information is available at the end of the article



**Figure 1** The effect of exercise on PCr/ATP in normal controls, HCM, and Athletes, showing a reduction in the HCM group, but no change in athletes or normal.\* $p < 0.05$  vs normal.

## Conclusions

During exercise, the pre-existing energetic deficit in HCM is further exacerbated and is not influenced by the degree of hypertrophy or scar burden. Acute derangement of energy-dependent ion homeostasis, triggering  $Ca^{++}$  overload and ventricular arrhythmias, may be a possible explanation for the high incidence of exercise-related death in HCM. Treatments that optimize energetics may be protective.

## Funding

This research was funded by the British heart foundation.

## Author details

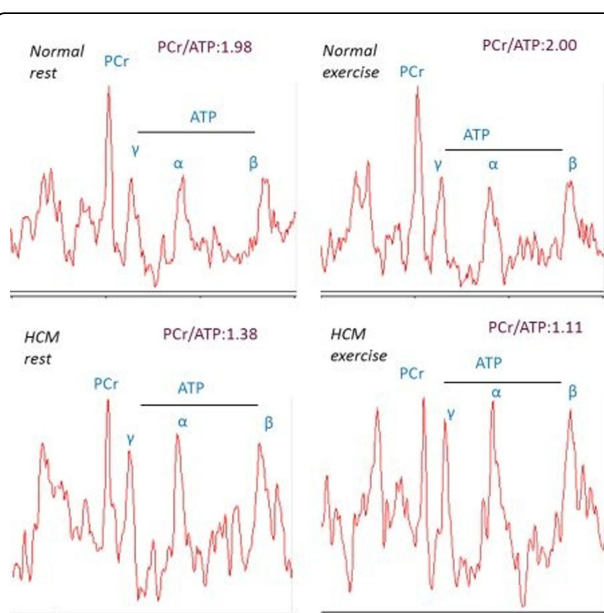
<sup>1</sup>Department of Cardiovascular Medicine, University of Oxford, Oxford, UK.

<sup>2</sup>Department of Physiology, Anatomy and Genetics, University of Oxford, Oxford, UK.

Published: 1 February 2012

doi:10.1186/1532-429X-14-S1-O75

**Cite this article as:** Dass et al.: Derangement of cardiac energy metabolism is acutely exacerbated during exercise in hypertrophic cardiomyopathy, independent of hypertrophy or late gadolinium burden. *Journal of Cardiovascular Magnetic Resonance* 2012 **14**(Suppl 1):O75.



**Figure 2** Examples of rest and exercise energetics in normal and HCM.

**Submit your next manuscript to BioMed Central and take full advantage of:**

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at  
www.biomedcentral.com/submit

