

Oral presentation

Open Access

Normobaric hypoxia elevates free fatty acids and impairs cardiac energetics and diastolic function in normal human volunteers

Cameron J Holloway*¹, Lowri E Cochlin¹, Ion Codreanu¹, Edward Bloch¹, Marzieh Fatemian², Cezary Szmigielski¹, Andrew Johnson¹, Jane M Francis¹, Peter Robbins¹, Stefan Neubauer¹ and Kieran Clarke¹

Address: ¹University of Oxford, Oxford, UK and ²The University of Oxford, Oxford, UK

* Corresponding author

from 13th Annual SCMR Scientific Sessions
Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, **12**(Suppl 1):O18 doi:10.1186/1532-429X-12-S1-O18

This abstract is available from: <http://jcmr-online.com/content/12/S1/O18>

© 2010 Holloway et al; licensee BioMed Central Ltd.

Introduction

In the first few days of hypoxic exposure, left ventricular dysfunction is consistently observed in human heart, yet the cellular mechanisms underlying the dysfunction are poorly understood.

Purpose

Our hypothesis was that normobaric hypoxia impairs cardiac energetics, leading to cardiac dysfunction.

Methods

Healthy males from the University of Oxford ($n = 12$, age 24 ± 2) underwent twenty hours of normobaric hypoxia in purpose-built hypoxia chambers. The partial pressure of oxygen during end tidal expiration ($P_{ET}O_2$) was kept between 50 and 60 mmHg, whilst keeping peripheral oxygen saturation (Sa_{O_2}) above 80%. Cardiac function was measured using magnetic resonance imaging (MRI) and echocardiography. High-energy phosphate metabolism was measured as the ratio of phosphocreatine to ATP (PCr/ATP) by ³¹Phosphorus magnetic resonance spectroscopy (MRS) before and after twenty hours of hypoxia. Additionally, four subjects had blood taken for biochemical analysis every four hours.

Results

During hypoxia, $P_{ET}O_2$ and Sa_{O_2} averaged 55 ± 1 mmHg and $83.6 \pm 0.4\%$, respectively. There was a 15% reduction in cardiac PCr/ATP, from 2.0 ± 0.1 to 1.7 ± 0.1 after

hypoxia ($p < 0.01$, Figure 1), reduced diastolic function, measured as E/E' , from 6.1 ± 0.4 to 7.5 ± 0.7 , ($p < 0.01$) and a three-fold elevation in plasma Free Fatty Acids (FFAs, $p < 0.05$).

Conclusion

Short term normobaric hypoxia led to rapid changes in FFAs, cardiac metabolism and alterations in diastolic function in normal human hearts. Elevated FFAs may lead to impaired high energy phosphate metabolism and cardiac dysfunction after hypoxic exposure.

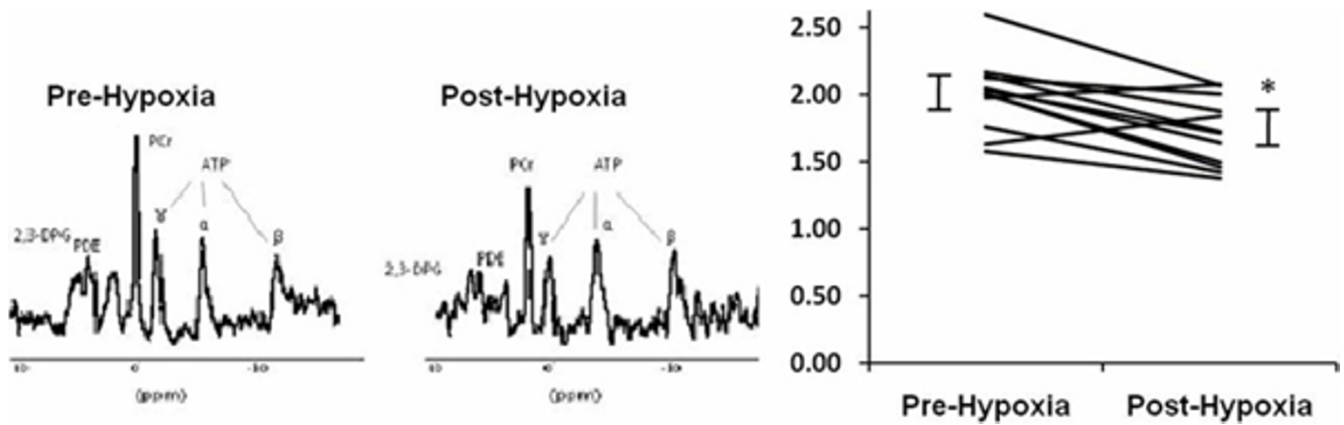


Figure 1

Publish with **BioMed Central** and every scientist can read your work free of charge

"BioMed Central will be the most significant development for disseminating the results of biomedical research in our lifetime."

Sir Paul Nurse, Cancer Research UK

Your research papers will be:

- available free of charge to the entire biomedical community
- peer reviewed and published immediately upon acceptance
- cited in PubMed and archived on PubMed Central
- yours — you keep the copyright

Submit your manuscript here:
http://www.biomedcentral.com/info/publishing_adv.asp

