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

Cameron J Holloway*[1], Lowri E Cochlin[1], Ion Codreanu[1], Edward Bloch[1], Marzieh Fatemianl[2], Cezary Szmigielski[1], Andrew Johnson[1], Jane M Francis[1], Peter Robbins[1], Stefan Neubauer[1] and Kieran Clarke[1]

Address: [1]University of Oxford, Oxford, UK and [2]The University of Oxford, Oxford, UK
* Corresponding author

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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_{O2}) 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 ^{31}Phosphorus 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 SaO_2 averaged 55 ± 1 mmHg and 83.6 ± 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.

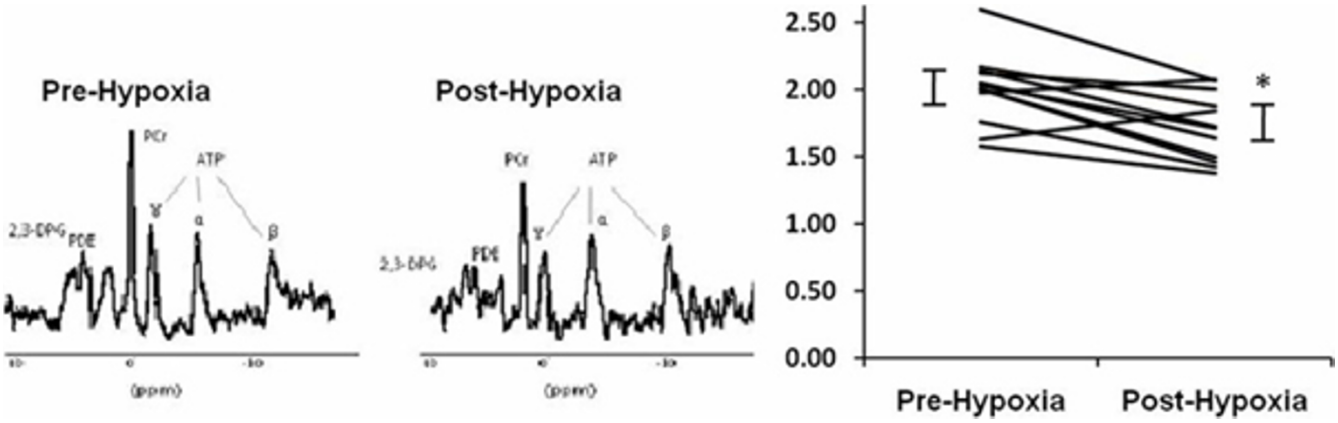

Figure 1