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The Effect of Fatty Acids and Isoflurane on Cardiomyocyte Respiration

Curtis R. Bradford [1]

1. Medical College of Wisconsin

Corresponding author: Curtis R. Bradford, cbradford@mcw.edu

Categories: Anesthesiology
Keywords:

How to cite this poster
Bradford C R (2012) The Effect of Fatty Acids and Isoflurane on Cardiomyocyte Respiration. Cureus 4(10): e319.

Abstract

Ischemia and reperfusion injury to cardiac tissue can be reduced by anesthetic-induced preconditioning (APC). Mitochondria are important mediators for APC whereby anesthetics trigger APC through mild inhibition of the electron transport chain. However, the cardioprotective benefits of APC are attenuated in diabetic patients, who are vulnerable to ischemic heart disease. In diabetes, cardiac mitochondria depend more on the utilization of fatty acids as substrate for energy production. How this substrate switch affects the isoflurane effect on mitochondrial electron transport chain is unknown. We hypothesized that in the presence of increased concentrations of the saturated fatty acid palmitate, the isoflurane-mediated effect may be less prevalent. We measured the effect of palmitate and isoflurane on the rate of mitochondrial oxygen consumption in a line of mouse atrial cardiomyocytes (HL-1 cells). We confirmed that isoflurane is able to attenuate oxygen consumption in HL-1 cells. Palmitate increases the oxygen consumption of cardiomyocytes in dose-dependent manner. Further, oligomycin-induced reduction of oxygen consumption is reversed by palmitate, indicating uncoupling of respiration. However, isoflurane inhibits oxygen consumption in the presence of palmitate. This suggests that the acute presence of palmitate does not attenuate the isoflurane-induced inhibition of the electron transport chain that triggers APC. Chronic exposure to palmitate should be tested in order to resemble the condition of the diabetic cardiomyocyte.

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Published 10/08/2012

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