

Oral presentation

Cardiovascular effects of inhaled oxygen assessed with magnetic resonance imaging

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Introduction

International practice guidelines prescribe the administration of 10–15 liters/min of O₂ to all critically ill patients, including to those who are initially normoxic. However, there are observations to suggest that inhaled supplemental O₂ may increase blood pressure and decrease cardiac output (CO) and coronary blood flow (CBF). The aim of this study was to establish the acute cardiovascular effects of oxygen inhalation in healthy subjects, using frontline cardiac magnetic resonance imaging (MRI).

Methods

16 healthy adults (34–54 years old, 8 females) inhaled O₂ at 1, 8 and 15 l/min through a bag-valve mask. A 1.5 T Philips Intera CV MRI Scanner was used to measure stroke volume (SV), CO and CBF. SV and CO were evaluated in the proximal aorta and CBF was measured as coronary sinus blood flow with a fast echo phase contrast image sequence. Left ventricular perfusion (LVP) was calculated as CBF divided by left ventricular mass. Arterial blood gases and hemoglobin was analyzed at each O₂ level. Statistical evaluation was performed by Friedman's test and Wilcoxon's signed ranks test.

Results

The response to O₂ was dose-dependent. 15 l O₂/min increased PaO₂ from an average 11.7 kPa to 51.0 kPa with no significant changes in PaCO₂ or arterial blood pressure. At the same dose, LVP decreased by 23% (P = 0.005) and CO by 10% (P = 0.003) with no significant changes

in SV or left ventricular dimensions. Because of the decreased CO and LVP, systemic and coronary O₂ delivery was lowered by some 4 and 11% at 8 l O₂/min, in spite of the increased blood oxygen content.

Conclusion

These data indicate that inhaled O₂ decreases systemic and coronary O₂ delivery in healthy subjects, and raise the possibility that myocardial ischemia may be increased by high-dose oxygen therapy in normoxic patients with acute ischemic heart disease.