Cardiovascular effects of inhaled oxygen assessed with magnetic resonance imaging

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Background

Oxygen is a cornerstone in the emergency treatment of all serious medical conditions, including acute ischemic heart disease. International guidelines and standard emergency care concepts like MedicALS and ATLS all prescribe the administration of 10-15 liters of O_2 to critically ill patients before anything else, including to the majority of the patients who are initially normoxic. However, there are observations to suggest that inhaled O_2 may increase blood pressure and systemic vascular resistance (SVR), decrease cardiac output (CO), and perhaps even decrease coronary blood flow, and that these effects are caused by hypocapnia resulting from hyperoxia-induced hyperventilation.
The methods used in these studies, however, have been less precise, indirect or invasive. Also, the amounts of O_2 in the blood often have not been measured directly, but instead only estimated by indirect techniques.

Aims

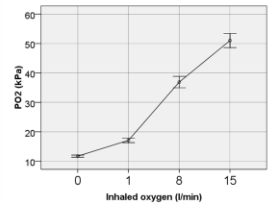
The aim was to establish the acute cardiovascular effects of graded inhalation of oxygen in healthy subjects, using frontline cardiac magnetic resonance imaging (MRI).

Methods

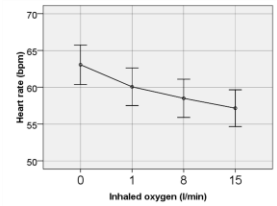
We evaluated the effects of inhaled O_2 at 0, 1, 8 and 15 l/min given through a bag-valve mask in 16 healthy adult subjects (8 females and 8 males; 26-65 years) without cardiovascular medication or pathological ECG findings. A 1.5 T Philips Intera CV MRI Scanner was used to measure stroke volume (SV) and coronary blood flow (CBF) after 10 min of O_2 inhalation at each level. SV were evaluated in the proximal aorta and CBF was measured as coronary sinus blood flow with a fast echo phase contrast image sequence For technical reasons, CBF was unavailable in three patients. Heart rate and blood pressure were measured conventionally and CO calculated as HR x SV. From a radial artery catheter, blood was analyzed before and after each MR scan for SaO_2, PaO_2 and hemoglobin, for O_2 delivery calculation.

Results

PaO_2

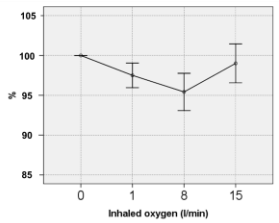


Heart rate

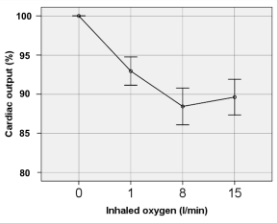


$PaCO_2$ (kPa)	4.9	5.1	5.0	4.8
MAP (mmHg)	92	91	93	94

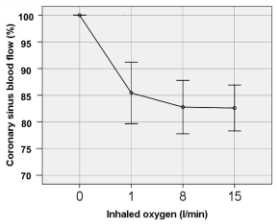
Stroke volume



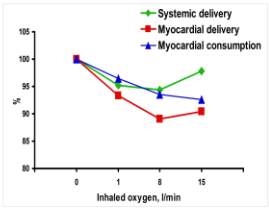
Cardiac output



Coronary blood flow



O_2 delivery and consumption



Systemic delivery: CO x arterial O2 content
Myocardial delivery: CBF x arterial O2 content
Myocardial consumption: HR x systolic blood pressure

Fifteen liters of O_2/min decreased HR by an average 9% (P=0.002), CO by 10% (P=0.007), and CBF by 18% (P=0.007). There was no significant increase in MAP or decrease in $PaCO_2$ suggesting hyperventilation. Because of the CO and CBF decrease, total O_2 delivery to the body and myocardium at 8 l/min O_2 was lowered by some 6 % and 11 %, in spite of the increased blood oxygen content.

Conclusion

These data indicate that inhaled O_2 decreases cardiac output and coronary blood flow, and decreases systemic and myocardial O_2 delivery in healthy subjects. The mechanism behind these effects is likely not hyperoxia-induced hypocapnia.

Our results raise the possibility that myocardial ischemia may be increased by high dose oxygen therapy in normoxic patients with acute ischemic heart disease.