

OXYGEN INHALATION REDUCES LEFT VENTRICULAR PERFUSION AND CARDIAC OUTPUT MEASURED BY MAGNETIC RESONANCE IMAGING

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INTRODUCTION: Oxygen (O₂) is routinely administered on broad indications to a wide variety of patients in the emergency setting. However, the effects of this therapy on the cardiac physiology have not been fully described, in part because of a lack of non-invasive methods of hemodynamic parameters. MRI offers the possibility to non-invasively measure several physiological and hemodynamical parameters such as cardiac output (CO), cardiac dimensions, left ventricular (LV) function and LV perfusion using the combination of flow measurements and cine imaging.

PURPOSE: To measure the effect of oxygen inhalation on LV perfusion, LV dimensions, LV function and CO in healthy subjects using MRI.

MATERIALS AND METHODS: The local ethics committee approved the study. Volunteers (n=16, age 43±3 range 25-65 years, 8 females) underwent MR-imaging (1.5T Philips Intera CV). MR images were obtained at baseline when subjects were breathing room air, during O₂ inhalation (1, 8 and 15 l/min) through a bag-valve mask and 15 minutes after stopping O₂ flow. The order of O₂ flow alternated, half of the patients started with 15 l/min and the other half with 1 l/min. There was a delay of 10 minutes at each level before imaging to obtain steady state. LV perfusion was determined by dividing the flow in the coronary sinus by LV mass. Flow in coronary sinus was quantified using a breath hold turbo field echo velocity encoded sequence with retrospective ECG triggering (TR 5 ms, TE 3 ms, α 15°, 35 phases through the cardiac cycle, number of acquisitions 1, SENSE factor 2, VENC 100 cm/s, in-plane spatial resolution 1.2x1.2 mm). Figure 1 demonstrates how the imaging plane of the coronary sinus flow measurement was planned. LV dimensions, function and mass was quantified on cine images encompassing the LV using a steady state free precession sequence in free breathing (TR 3 ms, TE 1.5 ms, α 60°, 30 phases through the cardiac cycle, number of acquisitions 1, SENSE factor 2, and spatial resolution 1.2x1.2x8 mm). Cardiac output was quantified on flow measurements in the aorta using a free breathing fast field echo velocity encoded sequence with retrospective ECG triggering (TR 9 ms, TE 5 ms, α 15°, 35 phases through the cardiac cycle, number of acquisitions 1, no parallel imaging, VENC 200 cm/s, in-plane spatial resolution 1.2x1.2 mm). Continuous variables are presented as mean±SEM and compared using Wilcoxon's matched paired test.

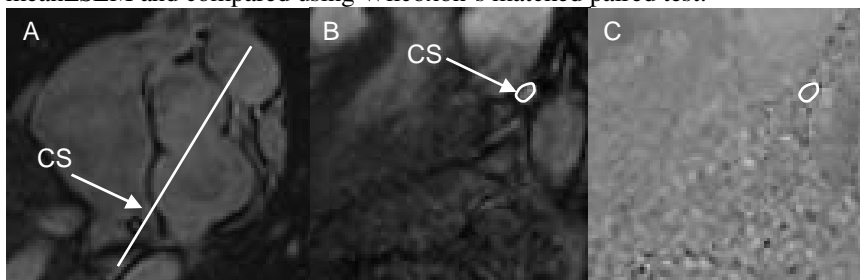


Figure 1. The CS was identified on short axis cine images (A). The imaging plane for the velocity encoded images was placed perpendicular to the flow in the CS (white line). The magnitude image (B) was used to identify the CS (circle) and the region of interest was copied to the phase image (C) where CS flow was measured.

RESULTS: LV perfusion was 1.2±0.1 ml/min/g at baseline, 1.1±0.1 ml/min/g at 1 l/min O₂ (p=0.04 vs. baseline), 1.0±0.1 ml/min/g at 8 l/min O₂ (p=0.0005 vs. baseline) and 0.9±0.1 ml/min/g at 15 l/min O₂ (p=0.0024 vs. baseline) (Figure 2A). There was a non-significant trend towards increased LV perfusion (1.0±0.2 ml/min/g) 15 minutes after cessation of O₂. LV dimensions did not significantly differ at baseline and 15 l/min O₂ (end diastolic volume 180±11 vs. 176±10, p=0.06 and end systolic volume 67±6 vs. 69±6, p=0.19). Ejection fraction was also unchanged (64±2 vs. 62±2, p=0.10). However, the stroke volume decreased from 113±6 ml at baseline to 108±6 ml at 15 l/min O₂ (p=0.008) and the heart rate decreased from 63±3/min at baseline to 61±3/min at 15 l/min O₂ (p=0.03). These changes resulted in a decrease of cardiac output from 7.0±0.3 l/min at baseline to 6.4±0.2 l/min at 15 l/min O₂ (8±2%, p=0.002) (Figure 2B).

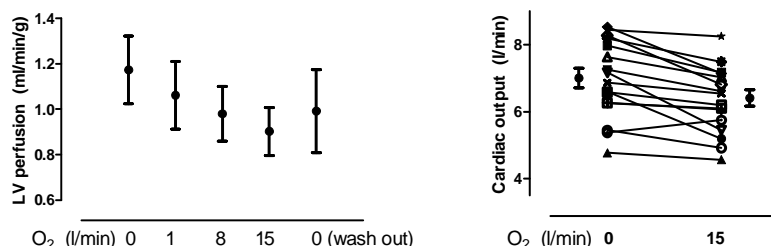


Figure 2. LV perfusion (left panel) decreased with increasing O₂ inhalation from baseline (0 l/min) to 15 l/min and 15 minutes after cessation of O₂ (wash out). Cardiac output (right panel) also decreased with oxygen inhalation. The figure shows the CO for each subject at baseline (0 l/min) and at maximum O₂ administration (15 l/min). Error bars denote mean±SEM.

CONCLUSION: This MR study have shown that LV perfusion decrease with increasing level of oxygen administration. Furthermore oxygen inhalation resulted in a decreased cardiac output due to a decrease in heart rate and stroke volume. Ejection fraction was not affected by oxygen administration. Further studies to investigate the effect of oxygen inhalation in patients are warranted. Flow quantification by MR imaging in the aorta and coronary sinus are sensitive techniques to detect physiological changes induced by therapy.