

ORAL PRESENTATION

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# Probing myocardial blood oxygenation reserve with controlled hypercapnia using BOLD CMR

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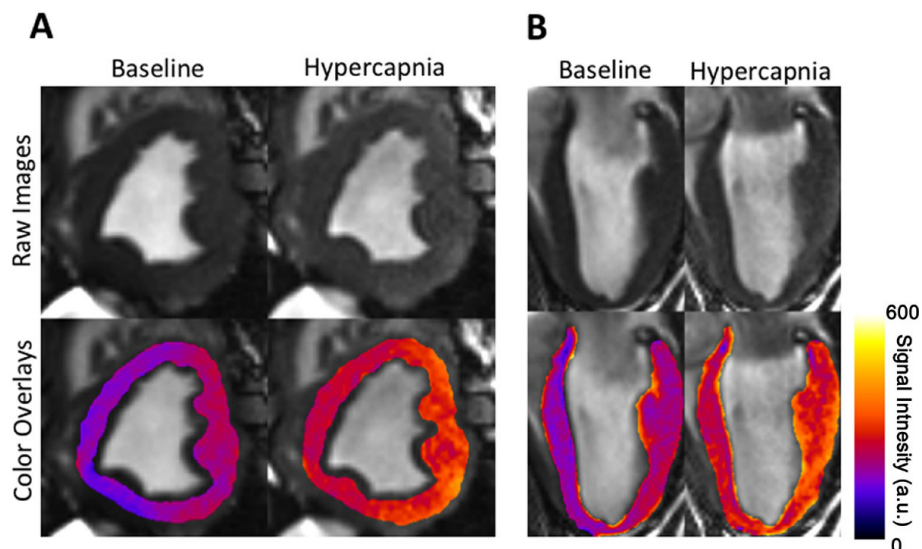
## Background

Background More than half of the cardiac stress tests require pharmacologic vasodilators for induction of hyperemia to assess myocardial perfusion, but carry the potential for side effects and are contraindicated in many patients considered for testing. We evaluated the feasibility of a non-invasive and safe stress-testing paradigm using a precisely targeted partial pressure of arter-

ial CO<sub>2</sub> (PaCO<sub>2</sub>) to induce myocardial hyperemia, and compared this response to intravenous adenosine.

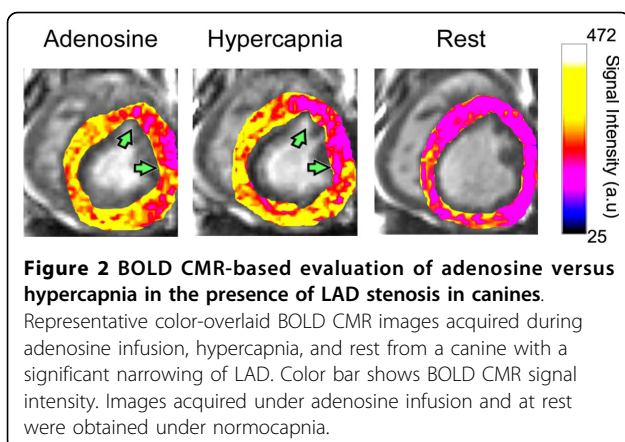
## Methods

Dose-response studies were performed on spontaneously breathing humans (n = 18), and canines (n = 18) with and without surgically implemented coronary stenosis to determine the optimal increase in PaCO<sub>2</sub> required to



**Figure 1 Effect of changing arterial CO<sub>2</sub> on BOLD CMR signal intensities.** Representative short (A) and long (B) axis BOLD CMR images collected from a canine from Group Ramp under baseline (PETCO<sub>2</sub> = 42 mmHg) and hypercapnia (PETCO<sub>2</sub> = 55 mmHg) are shown. Note the increase in signal intensity in images under hypercapnia relative to baseline.

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replicate the hyperemic response to intravenous adenosine (140  $\mu\text{g}/\text{kg}/\text{min}$ ). Blood-Oxygen-Level-Dependent (BOLD) CMR was used to determine the effects of hypercapnea.

## Results

In humans, an increase in  $\text{PaCO}_2$  of 10 mmHg was well tolerated, and the BOLD CMR responses were similar to those due to standard adenosine ( $p = 0.7$ ). In intact canines, the BOLD response to a mean increase in  $\text{PaCO}_2$  of 11 mmHg was similar to that of adenosine infusion (140  $\mu\text{g}/\text{kg}/\text{min}$ ,  $p = 0.4$ ); the responses were also similar in the territories subtended by stenotic ( $p = 0.7$ ) vessels.

## Conclusions

Conclusion Targeted increases in  $\text{PaCO}_2$  of 10 mmHg is well tolerated and has a myocardial vasodilating effect similar in extent to that of adenosine. These findings support continued investigation into the feasibility of inhaled  $\text{CO}_2$  as a vasodilator for cardiac stress testing.

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