

# Comparison of CE perfusion, $^3\text{He}$ ventilation and oxygen enhanced $^1\text{H}$ MRI for Imaging lung ventilation, perfusion and oxygen uptake

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**Introduction:** Contrast enhanced (CE) lung perfusion  $^1\text{H}$  MRI provides volumetric information on pulmonary perfusion. Hyperpolarised  $^3\text{He}$  MRI provides regional information on ventilation heterogeneity and is sensitive to alveolar oxygen partial pressure. Oxygen enhanced  $^1\text{H}$  MRI is also sensitive to ventilation and perfusion if it is assumed that the signal enhancement originates from oxygen dissolved in the pulmonary blood compartment. These three complementary functional lung MRI techniques have yet to be tested side by side in-vivo. In this study the methods are compared in a patient with Chronic Thromboembolic Pulmonary Hypertension (CTEPH) whose pulmonary vascular bed shows significant heterogeneity and V/Q mismatch.

**Methods:** A CTEPH patient was scanned on a 1.5T whole body MRI system (GE HDx) with ethics approval with the following techniques:

**$^3\text{He}$  Ventilation MRI:** A  $^3\text{He}$  transmit-receive vest coil (CMRS) was used. A mix of 300ml of hyperpolarised  $^3\text{He}$  (25% polarisation) and 700ml of  $\text{N}_2$  was inhaled, and  $^3\text{He}$  ventilation images were acquired at breathhold (full lung coverage,  $3\times 3\times 10\text{mm}$  spatial resolution,  $\theta=7^\circ$ ). 3D pO<sub>2</sub> mapping in a single breath-hold was also performed [1].

**Perfusion MRI:** Patients were repositioned in an 8-element  $^1\text{H}$  cardiac coil (GE) and CE perfusion data were acquired at inspiratory breath-hold with full lung coverage, using a time resolved spoiled gradient echo TRICKS sequence [2];  $2.4\times 6\times 10\text{mm}$  spatial resolution, 0.5s temporal resolution, 0.05ml/kg of Gadovist at 4ml/s, and 20ml saline flush at 4ml/s.

**Oxygen Enhanced  $^1\text{H}$  MRI:** was performed using a time resolved 2D Look-Locker T<sub>1</sub> mapping sequence as described in [3] with respiratory gating. The patient breathed room air followed by 100% oxygen from a tight fitting face-mask during the time course. All image reconstruction was performed in Matlab.

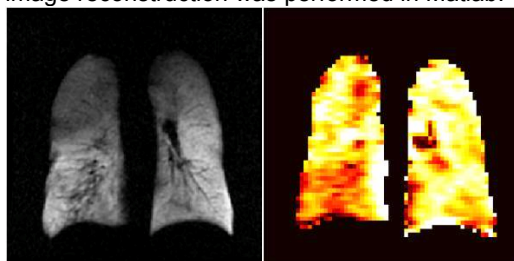


Fig.1  $^3\text{He}$  ventilation Fig. 2  $^3\text{He}$  pO<sub>2</sub> map  
Fig. 5 (below) time course of T<sub>1</sub> breathing air and oxygen

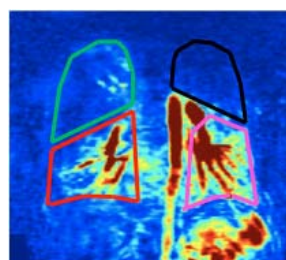


Fig.3 CE perfusion map

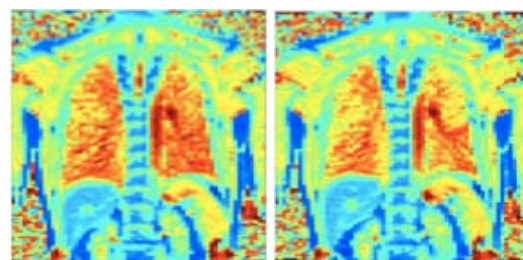
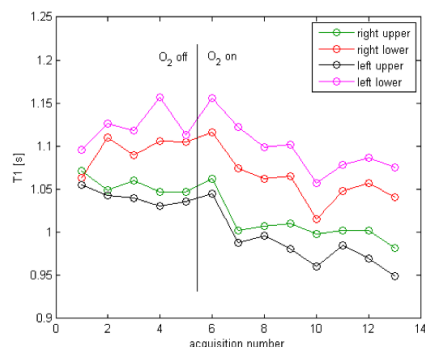


Fig. 4  $^1\text{H}$  T<sub>1</sub> map breathing air (l) and oxygen (r)



**Results and Discussion:** The  $^3\text{He}$  ventilation image in Fig.1 shows fairly homogeneous ventilation with some slight attenuation in the upper lobes which could be attributed to any of three mechanisms: (i) reduced ventilation (V/Q matching), (ii) T<sub>2</sub>\* dephasing from accumulation of stagnant blood in the distal vessels to the occlusion, or, (iii) elevated alveolar pO<sub>2</sub> from impaired perfusion. There is an elevated alveolar pO<sub>2</sub> seen in the upper lobes of the  $^3\text{He}$  pO<sub>2</sub> map (Fig. 2), which is also consistent with the CE perfusion image (Fig 3) showing obvious impaired perfusion in the upper lobes. Figure 4 shows example  $^1\text{H}$  T<sub>1</sub> maps from the time course when breathing air and oxygen respectively. The oxygen causes a global reduction in T<sub>1</sub> with a particularly noticeable drop in the upper left lobe, which is the least perfused. This is reflected in the time course of the T<sub>1</sub> map ROIs (Fig 5), which are delineated on the CE perfusion image of Fig. 3 for visualisation purposes alongside the perfusion. What is striking is the fact that the non-perfused lung regions (green and black) have a shorter baseline  $^1\text{H}$  T<sub>1</sub>. This is consistent with oxygen in inhaled

air not being removed across the alveolar/capillary interface due to perfusion impairment and this is also consistent with the elevated *alveolar* pO<sub>2</sub> seen in Fig. 2. What is difficult to reconcile from this data is the source of the oxygen-enhanced signal, assumptions in the oxygen-enhanced literature [3] assume the T<sub>1</sub> shortening effect to be due to dissolution of paramagnetic oxygen in the perfused pulmonary blood. In this example the upper lobes are not perfused but the T<sub>1</sub> maps still show substantial oxygen enhancement which suggests that the lung parenchyma  $^1\text{H}$  signal or else the stagnant blood/tissue in the distal vessels is causing the signal enhancement.

**Conclusion:** Three complementary techniques for imaging regional ventilation, perfusion and oxygen partial pressure have been compared in a patient with heterogeneous lung perfusion. The findings have common regional physiological explanations but some uncertainty is raised as to the source of signal change in the oxygen enhanced  $^1\text{H}$  MRI not being solely due to the perfused pulmonary blood pool enhancement in this pulmonary condition.

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**References:** [1] Proc ISMRM 2006, p 869 [2] Magn Reson Med. 2004;51(5):1009-16. [3] Magn Reson Med. 1996; 36(3):345-51