Comparison of CE perfusion, 3He ventilation and oxygen enhanced 1H MRI for Imaging lung ventilation, perfusion and oxygen uptake

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Introduction: Contrast enhanced (CE) lung perfusion 1H MRI provides volumetric information on pulmonary perfusion. Hyperpolarised 3He MRI provides regional information on ventilation heterogeneity and is sensitive to alveolar oxygen partial pressure. Oxygen enhanced 1H 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:

3He Ventilation MRI: A 3He transmit-receive vest coil (CMRS) was used. A mix of 300ml of hyperpolarised 3He (25% polarisation) and 700ml of N2 was inhaled, and 3He ventilation images were acquired at breathhold (full lung coverage, 3x3x10mm spatial resolution, θ=7°). 3D pO2 mapping in a single breath-hold was also performed [1].

Perfusion MRI: Patients were repositioned in an 8-element 1H 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]; 2.4x6x10mm spatial resolution, 0.5s temporal resolution, 0.05ml/kg of Gadovist at 4ml/s, and 20ml saline flush at 4ml/s.

Oxygen Enhanced 1H MRI: was performed using a time resolved 2D Look-Locker T1 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.

Results and Discussion: The 3He 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) T2* dephasing from accumulation of stagnant blood in the distal vessels to the occlusion, or, (iii) elevated alveolar pO2 from impaired perfusion. There is an elevated alveolar pO2 seen in the upper lobes of the 3He pO2 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 1H T1 maps from the time course when breathing air and oxygen respectively. The oxygen causes a global reduction in T1 with a particularly noticeable drop in the upper left lobe, which is the least perfused. This is reflected in the time course of the T1 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 1H T1. 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 pO2 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 T1 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 T1 maps still show substantial oxygen enhancement which suggests that the lung parenchyma 1H 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 1H MRI not being solely due to the perfused pulmonary blood pool enhancement in this pulmonary condition.

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