^{3}He pO_2 Mapping is Limited by Delayed-Ventilation and Diffusion in Chronic Obstructive Pulmonary Disease

Helen Marshall[1], Martin H Deppe[1], Juan Parra-Robles[1], and Jim M Wild[1]
[1]*Academic Radiology, University of Sheffield, Sheffield, South Yorkshire, United Kingdom*

Introduction: ^{3}He pO_2 mapping has been shown to provide quantitative measures of pO_2 in healthy volunteers [1]. pO_2 mapping assumes that all signal depletion during the breath-hold is due to RF depolarisation and oxygen-dependent T_1 effects [2], however the method is sensitive to other sources of signal change. Out-of-slice diffusion is a source of error in 2D pO_2 acquisitions leading to an underestimation of pO_2 values which can be mitigated by using a 3D sequence [3] where the whole lung experiences the same RF history. Here ^{3}He 3D pO_2 mapping was performed in patients with Chronic Obstructive Pulmonary Disease (COPD), a condition which is characterised by abnormalities of air-flow heterogeneity and regional gas apparent diffusion coefficient. Paradoxical findings in the measured regional pO_2 were observed confirming the technique is prone to error in lungs where ventilation is delayed or gas diffusion is spatially unconstrained.

Methods: Ten patients with moderate to severe COPD as defined by GOLD guidelines were scanned using a 1.5T whole body MRI system (GE HDx). Patients were positioned in a ^{3}He transmit-receive vest coil (CMRS). A mix of 200ml hyperpolarised ^{3}He (25% polarisation) and 800ml N_2 was inhaled, and ^{3}He pO_2 data were acquired using a single breath-hold sequence based on [4]. Sequence parameters were; 3D coronal spoiled gradient echo, full lung coverage, θ=1°, voxel size=5.5x10.9x20mm, 6 dynamic volumes and inter-image delay times τ_1=1.3s and τ_2=4.5s. A healthy volunteer was scanned with the same sequence after inhalation of 170ml ^{3}He and 830ml N_2. Data was masked according to the SNR of the final dynamic volume [5], and fit pixel by pixel in Matlab to calculate pO_2 values [3].

Results and Discussion: Figure 1 shows pO_2 maps from a healthy volunteer and from a COPD patient.

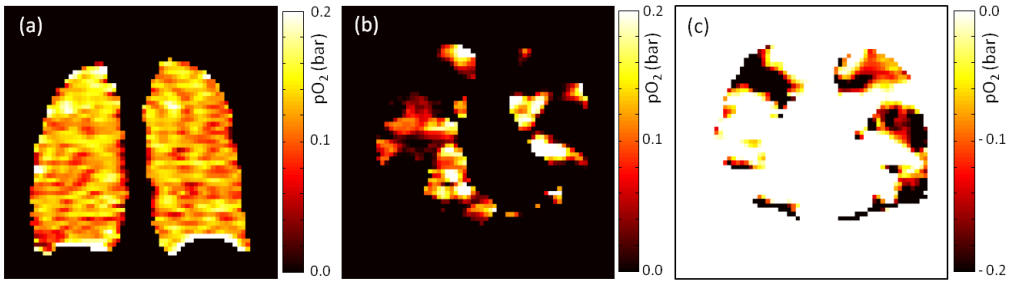

Figure 1 pO_2 maps from (a) a healthy volunteer, and (b, c) a COPD patient with gas movement in the lungs during breath-hold. (c) shows negative 'pO_2 values' where signal increases over time.

In the COPD patient, negative pO_2 values were returned from significant regions of the lung (fig. 1c). Analysis of ROI signal time-courses showed that signal increased over time in these regions (fig. 2d, magenta and green) which is clearly an unrealistic physical situation if $T_1(pO_2)$ and RF pulsing are the only sources of signal change during breath-hold. The source ^{3}He time-course images show gas moving into initially unventilated defects during the course of the static breath-hold. Signal ROI plots demonstrate that even regions away from obvious slow-filling ventilation defects can experience a delay before peak signal is reached (e.g. fig 2d, green). pO_2 values from regions of interest in fig. 2c were 0.17 (blue), 0.10 (red), -0.10 (green) and -0.40 bar (magenta). The pO_2 value measured from both ROIs in the healthy volunteer was 0.12 bar (fig. 2a, c), which is similar to values published previously for healthy volunteers [1, 3].

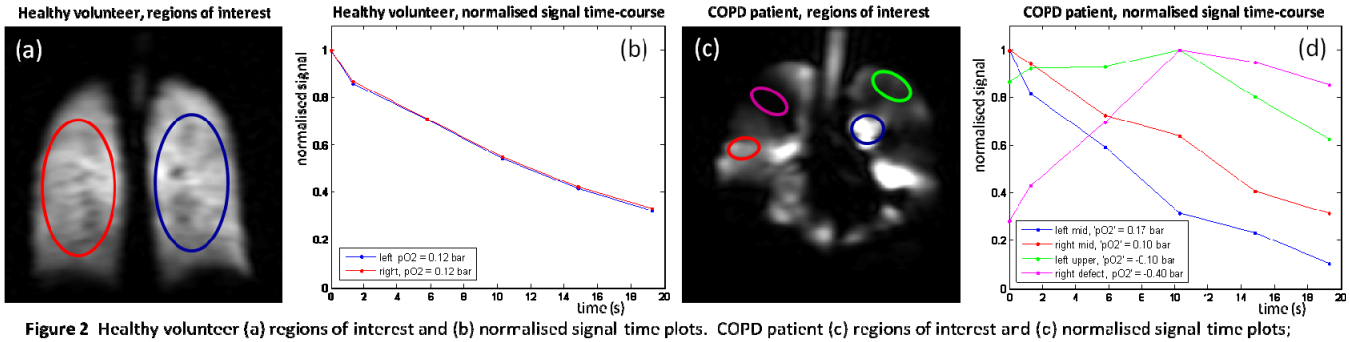

Figure 2 Healthy volunteer (a) regions of interest and (b) normalised signal time plots. COPD patient (c) regions of interest and (c) normalised signal time plots; signal increases over time within the right ventilation defect (magenta) and left upper lobe (green) ROIs.

Slow-filling ventilation defects of varying size and fill-rate were observed in 8 of the 10 COPD patients. Movement of gas within the lungs during breath-hold, either by unrestricted diffusion in bullous emphysematous regions or regions of delayed ventilation (as depicted here), can cause significant regional changes in signal over time which are not related to oxygen concentration. These changes can vary throughout the lung with pathology and cannot be disentangled from other signal decay mechanisms, leading to erroneous pO_2 measurements.

Conclusions: It has been demonstrated *in vivo* that delayed-ventilation and / or diffusion limits the effectiveness of pO_2 mapping in COPD, where movement of gas within the lungs during breath-hold invalidates the assumption that all signal decay is due to T_1 decay and RF depolarisation.

Acknowledgements: Funded by GlaxoSmithKline (RES111175) and UK EPSRC (EP/D070252/1). Polariser support from GE Healthcare.

References: [1] Deninger et al, JMR 141:207-16 (1999); [2] Saam et al, Phys Rev A52: 862-65 (1995); [3] Wild et al, Magn Reson Med 53:1055-1064 (2005); [4] Wild et al, 869 Proc. ISMRM (2006); [5] Woodhouse et al, JMRI 21:365-69 (2005);