Oxygen-Enhanced Ventilation Mapping of Whole Lungs using 3D UTE at 3T

Crystal E. Harrison[1], Masaya Takahashi[1], Robert E. Lenkinski[1], and Ananth J. Madhuranthakam[1]
[1]*Radiology and Advanced Imaging Research Center, UT Southwestern Medical Center, Dallas, TX, United States*

Introduction: Chronic obstructive pulmonary disease (COPD) affects lungs non-uniformly by disrupting pulmonary ventilation and perfusion. Oxygen-Enhanced (OE) MRI is a relatively inexpensive method for probing pulmonary ventilation with high spatial resolution.[1] Inhaled molecular oxygen (O_2) acts as a paramagnetic agent and reduces the T_1 of the lung parenchyma, which can be detected by MRI. A majority of the OE-MRI studies to date have been performed with 2D acquisitions[2,3], limiting the lung coverage to evaluate and monitor COPD.

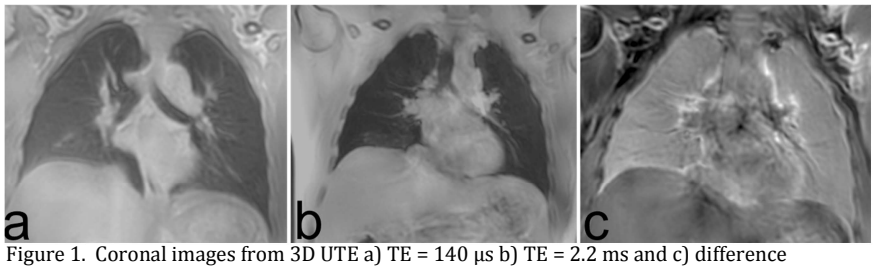

Figure 1. Coronal images from 3D UTE a) TE = 140 μs b) TE = 2.2 ms and c) difference

One of the primary challenges of lung imaging is the short T_2* of the lung tissue. In recent years, the implementation of 3D ultrashort echo time (UTE) radial k-space projections allow volumetric acquisitions while minimizing the signal loss due to short T_2* relaxation.[4] OE-MRI with 3D UTE was recently demonstrated in humans at 1.5 T.[5] For shorter TE values, it has been shown that the signal to noise ratio (SNR) at 3T is higher than at 1.5T in lung phantom models.[6] Moreover, the change in T_1 due to inhaled O_2 will be higher at 3T as compared to 1.5T. Hence, the purpose of the current study is to demonstrate the feasibility of OE-MRI with 3D UTE at 3 T for ventilation mapping in human subjects.

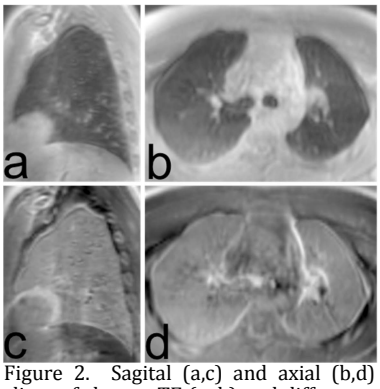

Figure 2. Sagital (a,c) and axial (b,d) slices of shorter TE (a, b) and difference images (c, d) from the same subject as seen in Fig 1.

Methods: With IRB approval and written informed consent, 5 healthy human volunteers were scanned on a 3 T Ingenia system (Philips Healthcare, The Netherlands) using a 24-channel phased array coil. Two subjects were scanned for protocol optimization without exogenous O_2, while three subjects were scanned twice: once with medical air (21% O_2) and once with 100% O_2. Medical air and O_2 were delivered with a rebreathing bag mask with a two-minute interval between successive scans. Images were acquired with respiratory triggering and without cardiac gating in a 5-6 min time period. 3D UTE was performed with a stack-of-stars trajectory (kx-ky radial projections with Cartesian slice-encoding) and the following scan parameters: 8° flip angle, $TE_1/TE_2/TR$ = 0.14/2.2/4 ms, 2.5 mm^3 isotropic resolution, FOV = 400 x 400 x 280 mm^3. Data were processed offline in Matlab. An overlay image was created by performing the percentage signal enhancement (PSE) calculation: $PSE = 100 * (S_{100\%} - S_{21\%})/S_{21\%}$. The PSE maps were smoothed with a 10x10 median filter and a threshold was applied to the original medical air image to mask the PSE maps within the lung volume for display.

Results : Figure 1 displays a coronal slice with shorter (TE = 140 μs) and longer TE (TE = 2.2 ms) and a difference image (a-b) demonstrating high signal in the lungs. Figure 2 contains the sagittal and axial views of the shorter TE and difference images in the same subject. Fig. 3 shows representative PSE maps in orthogonal orientations acquired with 3D UTE in a different subject. The average PSE for the whole lung was ~6.8 % and is slightly lower than the theoretically predicted 7.4 % based on literature values of T_1 in the lung at 3 T (1400 ms and 1260 ms with 21% and 100% O_2 respectively).[7]

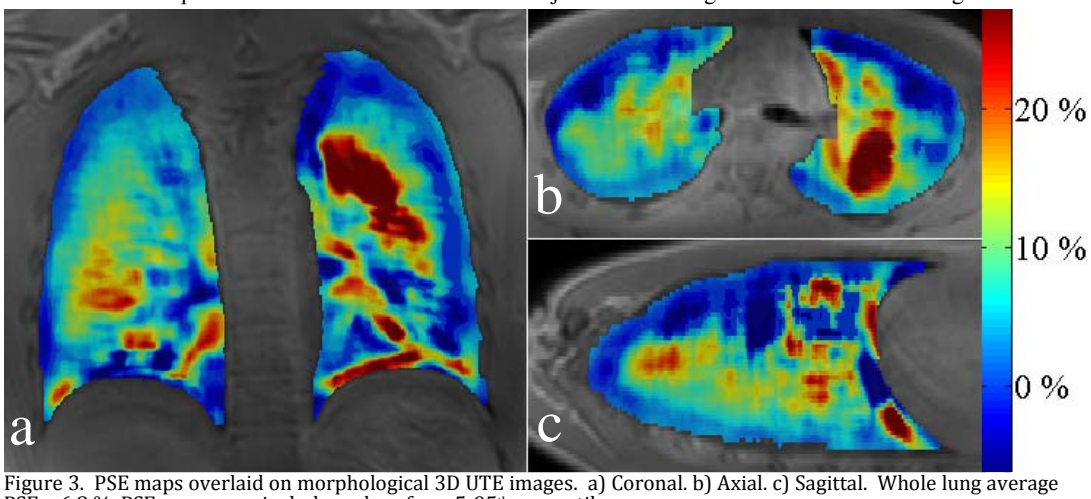

Figure 3. PSE maps overlaid on morphological 3D UTE images. a) Coronal. b) Axial. c) Sagittal. Whole lung average PSE = 6.8 %. PSE map range includes values from 5-95th percentile.

Conclusion: Oxygen enhanced volumetric ventilation mapping in humans using 3D UTE is feasible at 3 T. This approach enables volumetric ventilation mapping using standard hardware proton MRI and provides an alternative means to hyperpolarized gas imaging for broader dissemination to evaluate and monitor COPD.

References: 1. Edelman et. al. Nature Med. 2:1236-1239 (1996). 2. Jakob et. al., MRM 51:1009–1016 (2004). 3. Ohno et. al., EJR 77(1): 85–91 (2011). 4. Johnson et. al., MRM, 70: 1241–1250(2013). 5. Kruger et al., NMB (2014) doi: 10.1002/nbm.3158. 6. Molinari F. et. al., Diag. and Int. Rad. 20(1), 34(2014). 7. Nichols et. al., JMRI, 27: 224–228(2008).