Lung inflation state dominates over intrapulmonary pO2 regarding T2* of 3He in human lungs

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Introduction: Molecular oxygen (O2) is paramagnetic (\(\chi_{\text{vol}}=1.8\) ppm), and when present increases the magnetic susceptibility of lung gas. This effect has been demonstrated for investigation of pulmonary ventilation via the effective transverse relaxation time T2* of lung parenchyma [1]. In that method, T2* of the protons in lung tissue was mapped at baseline and after a few minutes of inhalation of pure O2. The elevated intrapulmonary partial oxygen pressure (pO2) results in an increased susceptibility gradient across the alveolar membranes, resulting in a shortening of the \(^1\text{H} T2^*\) by about 10% [1]. Thus, by mapping the change in T2*, an estimate of ventilation is obtained. A more direct approach for quantification of pulmonary ventilation is presented by the imaging of hyperpolarized 3He. Measurements of T2* of intrapulmonary 3He have attracted some interest in the past, as they are sensitive to lung microstructure [2]; however, other factors, such as the lung inflation state [3], diffusional motional narrowing and B0 field strength [4, 5] have an influence on T2* as well, and present potentially confounding factors. The aim of this work was to investigate the influence of intrapulmonary pO2 on T2* of 3He in human lungs.

Materials and Methods: T2* maps of 3He were obtained from the lungs of a healthy volunteer with ethics and regulatory approval. A double-interleaved 2D gradient-echo sequence, written in-house, was used on a GE 1.5T HDx scanner (GE, USA). Sequence parameters were: 64x64 matrix, FOV 38 cm, slice thickness 10 mm, 7 axial slices, TE1 = 3.8 ms, TE2 = 13.8 ms, TR = 20 ms, receiver bandwidth ±15.63 kHz, flip angle 7°. 3He was polarized to ~20% using a Helispin polarizer (GE, USA). During each scan, the volunteer inhaled a mixture of 200 ml 3He and 300 ml N2. In order to reproduce lung inflation state well, two scans were performed both at baseline and after the volunteer had been breathing pure O2 for about 4 minutes. For the first scan, the volunteer was asked to inhale the bag containing 3He from a state of full expiration. In the second scan, the volunteer was asked to top up her lungs to full inspiration, with room air or O2 respectively, after inhalation from the bag.

Results and Discussion: Figure 1 shows typical T2* maps obtained at full expiration and inspiration, at baseline and after O2 prewash. While maps at inspiration have regions of visibly longer T2*, no difference is apparent between maps acquired at air breathing baseline and after O2. Figure 2 shows the mean values of T2* for each acquisition as a function of slice number. Averaged over all slices, mean values at expiration of 16.3 ± 1.1 ms and 16.8 ± 1.0 ms are observed at baseline and after O2 respectively. At inspiration, the values are 27.5 ± 1.4 ms and 27.4 ± 1.2 ms respectively. It follows that while changes in the inhalation state can account for a change of ~60% in T2*, the influence of O2 appears to be negligible. If a small influence is present, it is likely to be masked by the effect of lung inflation, particularly as the reproduction of lung volumes in spontaneously breathing human volunteers and patients is not trivial. This behaviour differs from intrapulmonary \(^1\text{H}\), where a reduction of T2* by ~10% was observed after O2 prewash [1]. A possible explanation is the fact that \(^1\text{H}\) signal is detected from the alveolar walls, while 3He is present in the alveolar gas spaces. O2 in the gas spaces increases the susceptibility difference between gas and tissue but the resulting field inhomogeneities appear mainly close to the tissue-gas interfaces. Virtually all intrapulmonary \(^1\text{H}\) spins are located in the immediate vicinity of these interfaces. Whereas the \(^3\text{He}\) samples the whole alveolar space, and thus experiences less field inhomogeneity on average, with diffusional motional narrowing playing an additional role, making its T2* less sensitive to intrapulmonary pO2. While this work demonstrates that lung inflation state dominates over a potential pO2 effect, such an effect is still expected to be present via the known shortening of T2CPMG via dipole-dipole interaction [6]; however, at 1.5T T2* << T2CPMG, thus any effect on T2* appears to be too small to be convincingly detected and separated from the influence of the natural variation in lung inflation state. The findings of inflation state dependence are consistent with those measured previously at 1.5T [3], albeit at different echo times and voxel size. For the gradient echo sequences typically used for imaging of hyperpolarized noble gases, a long T2* is desirable. The finding presented here shows that inhalation of O2 has no negative effect on T2*, which might influence decisions in handling of patients who benefit from ventilation with O2. Nevertheless, the well-known shortening of T1 of \(^3\text{He}\) by increased pO2 [7] should still be taken into account when making those decisions.

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Fig. 1: \(^3\text{He} T2^*\) maps from a healthy volunteer at 1.5T, at expiration (a, c) and inspiration (b, d), at baseline (a, b) and after 4 min O2 (b, d).

Fig. 2: Mean T2* values at baseline (blue) and after O2 prewash (red). Upper curves: inspiration, lower curves: expiration.