Quantifying the effects of hyperoxia on abdominal tissue T_1

Eleanor F Cox[1], Emma L Hall[1], Paula L Croal[1], Ian D Driver[1], and Susan T Francis[1]
[1]*SPMMRC, School of Physics & Astronomy, University of Nottingham, Nottingham, Nottinghamshire, United Kingdom*

Background The longitudinal relaxation time (T_1) of tissues has been suggested to be a potential biomarker for tissue oxygenation. Previous studies have shown a reduction in T_1 or T_1-weighted signal in abdominal organs on inhalation of 100% oxygen at 1.5T [1-4] and 3T [5], hypothesised to be due to an elevated concentration in dissolved oxygen. However, there is large variability in findings for the renal cortex, which could be in part due to abdominal motion leading to poor cortical segmentation and assessment of changes in T_1 (or R_1). We have recently developed a modified respiratory-triggered free-breathing technique for mapping abdominal T_1, without the need for motion correction [6]. This study aims to use this modified respiratory-triggered method at 3T to assess changes in the longitudinal relaxation rate (ΔR_1) in the renal cortex, spleen and liver in response to hyperoxia.

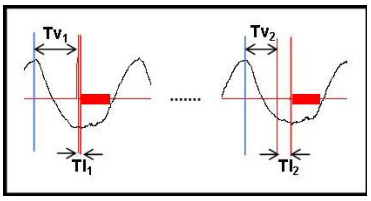

FIG 1: Modified respiratory-triggering T_1 method

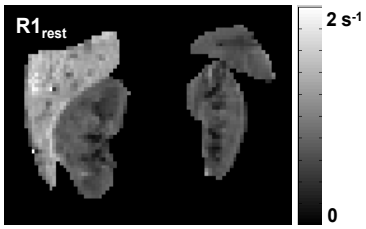

FIG 2: Example mean R1 map at normoxia

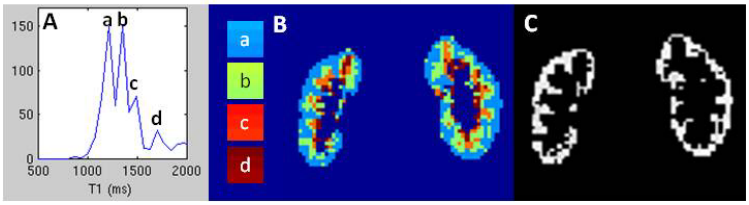

FIG 3: Example (A) histogram of single subject kidney T_1 values, (B) segmented T_1 map and (C) renal cortex mask

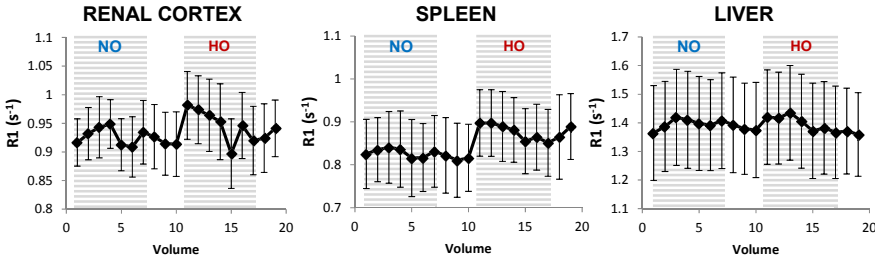

FIG 4: Example R_1 time courses (s^{-1}) for the renal cortex, spleen and liver, with periods of normoxia (NO) and hyperoxia (HO) indicated in grey (mean ± stdev)

	Renal cortex	Spleen	Liver
NO T_1	1132 ± 15 ms	1138 ± 34 ms	671 ± 53 ms
HO T_1	1111 ± 15 ms	1075 ± 23 ms	670 ± 47 ms
$\Delta R1_{(H0-NO)}$	0.016 ± 0.006 s^{-1}	0.049 ± 0.005 s^{-1}	0.004 ± 0.006 s^{-1}

TABLE 1: Mean T_1 (± sterr) and ΔR_1 (± sterr) across subjects in the renal cortex, spleen and liver during normoxia (NO) and hyperoxia (HO)

Methods The study was approved by the local Ethics committee and all volunteers gave informed written consent. Five healthy volunteers (3M/2F) were scanned (mean age 26y, range 22-31y) using a 3T Philips Achieva whole body scanner with a 6-element receive torso coil. A sequential gas delivery breathing circuit and a prospective, feed-forward gas delivery system (Respiract™, Thornhill Research Inc., Toronto, Canada) was used to control and monitor end-tidal O_2 ($P_{ET}O_2$) and CO_2 ($P_{ET}CO_2$) partial pressures. Normoxia (NO) was targeted at the subject's resting value ($P_{ET}O_2$ ~ 100 mmHg) and hyperoxia (HO) at $P_{ET}O_2$ ~ 500 mmHg. Isocapnia was maintained throughout at the subject's resting value ($P_{ET}CO_2$ ~ 40 mmHg). The gas challenge consisted of a 4 min period of normoxia, followed by a 2 min transition to isocapnic hyperoxia for 4 min and then a 2 min transition to return to normoxia. A modified IR-TrueFISP sequence [6] was used to measure T_1 in the kidneys, liver and spleen during normoxia and hyperoxia. This sequence uses an additional variable delay (T_v) to ensure that for each inversion delay (TI) in the inversion recovery (IR) data, slices are acquired at the same point during the 'stationary' near-end expiration period, in this study at a constant time T_v +TI=1100 ms after the respiratory trigger with a minimum TR of 5 s (Fig. 1) [6]. IR-data was repeatedly collected at 8 inversion times (TI = 300, 400, 550, 650, 800, 900, 1000, 1100 ms) over the 12 min gas challenge. Each IR data set was collected in ~40 s from which a T_1 (R_1) map was formed. As the modified respiratory triggered method [6] was used, volunteers breathed freely during the gas challenge, and the use of this sequence avoided the need for any image realignment. TrueFISP parameters: single coronal-oblique slice through long axis of kidney, SENSE 2, FA 60º, 3x3x8 mm^3 voxel, 288x288 mm FOV.

Data Analysis: Each set of IR data was fitted on a voxel-by-voxel basis to a 2 parameter model using Powell minimization to generate a time series (volume number) of T_1 (R_1) and M_0 maps. T_1 (R_1) maps were averaged across normoxia and hyperoxia periods (Fig. 2). A histogram of T_1 values across the kidney during normoxia was formed, and the peak of the histograms used to segment the kidney and form a mask of the renal cortex (Fig. 3). This mask was then used to calculate the mean T_1 for the renal cortex during normoxia and hyperoxia and the ΔR_1 for each subject. For each subject, masks were also drawn of the spleen and liver to calculate the mean T_1 and ΔR_1. T_1 and ΔR_1 values were averaged across subjects to obtain an estimate mean (± sterr) parameters for the renal cortex, spleen and liver.

Results $P_{ET}O_2$ increased to 464 ± 14 mmHg (mean ± sterr) on hyperoxia, while $P_{ET}CO_2$ varied by < 1 mmHg. Figure 2 shows an example mean R_1 map during normoxia. Figure 3 shows an example kidney histogram with a segmented T_1 map and the renal cortex mask. Figure 4 shows an example subject ΔR_1 time course for the renal cortex, spleen and liver. Table 1 shows the T_1 values for each organ during normoxia and hyperoxia, and the associated change in R_1 (hyperoxia-normoxia). There was significant increase in R_1 during hyperoxia in the spleen (*p = 0.043, Wilcoxon signed ranks), whilst the renal cortex showed a trend for R_1 to increase during hyperoxia (p = 0.225, Wilcoxon signed ranks). There was no change in the liver R_1 on hyperoxia (p = 0.686, Wilcoxon signed ranks).

Discussion and Conclusion Our T_1 values at normoxia are in line with those reported values at 3T [5,7]. Previous studies show a T_1 decrease in the renal cortex and spleen on breathing 100% O_2 and little or no change in the liver [2-4]. Here, we have independently controlled end-tidal concentrations of O_2 and CO_2 (constant to < 1 mmHg) and show similar results. Our controlled gas delivery is equivalent to breathing ~70 % O_2, which may lead to a smaller T_1 change in our data. Further, breathing 100 % O_2 can lead to hypocapnia, resulting in a reduction in flow, controlling $C0_2$ in our study prevents such a confound. The modified respiratory triggering method allowed us to form maps of T_1 and R_1 in the renal cortex, spleen and liver at 3T without the need for any image realignment, and assess the temporal change in R_1 to hyperoxia on a voxelwise level. This method will be used to assess the inter-subject variability in response to hyperoxia.

References: [1] Tadamura et al. JMRI 1997; **7**:220-5, [2] Jones et al. MRM 2002; **47**:728-35, [3] O'Connor et al. MRM 2007; **58**:490-6, [4] O'Connor et al. MRM 2009; **61**:75-83, [5] Ding et al. ISMRM 2011 P2951, [6] Cox et al. ISMRM 2011 P825, [7] De Bazelaire et al. Radiol. 2004; **230**:652-9.