

Imaging Water Content in the Lungs – Measuring and Correcting the Influence of Breathing

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Introduction: Diseases of heart, lung and kidneys, exposure to toxins and numerous other conditions can promote pulmonary edema, which refers to extravasation of fluid from the pulmonary vasculature into the interstitium and alveoli of the lung. Quantification of edema in the lungs is confounded by the large fractional air volume in lungs, which changes throughout breathing and strongly influences the water spin density^{1,2}. Breath-holding is an option for normalizing air volume in the lung, but this manoeuvre is notoriously inconsistent, highly variable from person to person and constrains acquisitions to breath-hold durations. The purpose of this study is to evaluate the dependence of lung spin density on respiration, and to develop an approach to correct for these variations, to allow free-breathing evaluation of lung water. Correction is appealing as opposed to respiratory gating to increase the acquisition duty cycle, shorten study duration and to account for phenomena such as drift in breathing patterns.

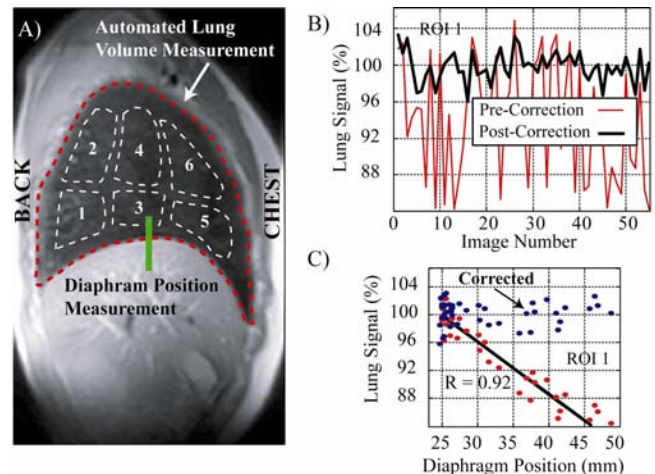
Methods: Changes in fractional air volume during breathing is assumed to be the primary mechanism for variability in measured lung signal intensity^{1,2}. Surrogates for total lung volume (cross-sectional area of the lung in a sagittal slice and the diaphragm position) were assessed as a metric for correcting the unwanted changes in MRI signal intensity. The sagittal view provides a good estimate of volume due to the prominence of diaphragm motion (head to foot) and chest wall motion (anterior/posterior) throughout breathing, and this view also provides a cross-section of the diaphragm. All cross-sectional areas and the diaphragm edge were calculated automatically with minimal user input.

Eight subjects were imaged with a single shot fast spin echo (HASTE) pulse sequence during free breathing. Because HASTE is a single shot technique it is not prone to breathing motion artifacts, common in segmented imaging sequences. Also, HASTE is predominantly T₂ weighted, so offers high signal yield in the lungs as compared to sequences with T₂* weighting³. Multiple repetitions were acquired for each of 5 to 8 slices throughout free-breathing, with slice interleaving to ensure full T₁ recovery for each acquisition (TR ~ 6-8 seconds). The signal intensity in each of six regions of interest was correlated with both the lung area and diaphragm position. The signal intensity in each region was corrected using a linear model of the relationship between the lung signal and the corresponding volume surrogate (Figure C). Typical parameters for HASTE lung studies: 360 x 270 mm FOV, 192 x 72 matrix, 3.5 ms echo spacing, 10 mm slice thickness, TE = 21 ms, 30-60 repetitions, 5 to 8 slices, 3-6 minute acquisition times. All studies were performed on a Siemens Sonata 1.5T system. Cardiac gating ensured all images were acquired at end-diastole.

Results: Figure A) shows a typical sagittal HASTE image with sample regions of interest as well as area and diaphragm measurements. Figure B) shows the signal intensity from 55 images acquired throughout free-breathing, before and after correction, *S* and *S*_{CORR} respectively. The correction procedure is illustrated in Figure C), where the relationship between the lung signal and diaphragm position are modeled with a straight line, which is subsequently used to normalize the data (blue points). The observed correlation value of R = 0.92 is typical for all slices and for all eight subjects studied, for both the area and diaphragm volume surrogates. Table 1 summarizes results for the 8 subjects, for a total of 150 regions of interest. **The variable *S* is the signal intensity from a region of interest for all repetitions, and *S*_{CORR} is the corrected data.** The rightmost columns show the average correlation between the signal intensity and both the diaphragm position or lung area.

Conclusion: We observed a strong correlation between our surrogates for lung volume (diaphragm position and lung area) and the measured lung signal intensity, with slightly better correlation for area measures. Assuming a linear correlation allowed a simple correction of lung signal intensity for changes in fractional air volume with breathing. The standard deviation of the measured signal (normalized to mean signal) was reduced by 2 to 4 times with correction, depending on the region, with the largest improvement at the back (ROI 1 and ROI 2). Practical application of this approach requires sufficient repetitions to allow the correction slope to be calculated, which is as few as 10 sample points, for a total acquisition time of 60 to 90 seconds for whole lung coverage. In combination with the correction methods outlined in this study, we are currently using surface coil intensity correction and calibration with an internal water standard (CSF in the spinal column) to derive absolute spin density in the lungs (not shown). Changes in lung volume during breathing are regional phenomena, but our assumption was that each region of interest differs from change in total lung volume primarily by a scaling factor. This is equivalent to changes in the slope of the correction curve if Figure C. The ratio of the slopes was typically [4:2:1] from the back to the middle to chest regions, showing a large regional variation in relative changes in lung volume with breathing.

References: [1] Mai VM, et al., Effect of Respiratory Phases on MR Lung Signal Intensity and Lung Conspicuity Using Segmented Multiple Inversion Recovery Turbo Spin Echo (MIR-TSE), *MRM* 43:760–763 (2000). [2] Bankier AA, et al., Gravity-Dependent Signal Gradients on MR Images of the Lung in Supine and Prone Positions: A Comparison With Isogravitational Signal Variability *JMRI* 23:115–122 (2006). [3] Hatabu H, et al., MR imaging of pulmonary parenchyma with a half-Fourier single-shot turbo spin-echo (HASTE) sequence. *EJR* 29, 152-159 (1999).



A) Sample sagittal HASTE image showing placement of 6 regions of interest. B) Sample signal intensities from 55 repetitions (ROI 1), before and after breathing correction. C) The same data in B) is shown as a correlation plot, comparing signal intensity and diaphragm position. The data is corrected by removing a linear component as shown in C).

Table 1 - Lung signal intensity before and after correction (*S* and *S*_{CORR}) and correlation of signal with lung volume surrogates.

	\bar{S}/\bar{S}_{CORR}	STD(<i>S</i>)/ \bar{S}_{CORR}	STD(<i>S</i> _{CORR})/ \bar{S}_{CORR}	Correlation (diaphragm)	Correlation (Area)
ROI 1	0.91	7.5%	2.2%	.92 (0.07)	.95 (0.04)
ROI 2	0.92	6.8%	2.2%	.90 (0.10)	.94 (0.05)
ROI 3	0.93	6.3%	2.3%	.81 (0.15)	.85 (0.12)
ROI 4	0.94	5.8%	2.4%	.83 (0.14)	.88 (0.09)
ROI 5	0.94	5.6%	2.8%	.80 (0.15)	.85 (0.08)
ROI 6	0.95	5.4%	2.9%	.75 (0.16)	.80 (0.12)