How volume affects the pulmonary MRI signal: Investigations with 3D ultra-fast balanced Steady-State Free Precession

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Target audience. Physicist and physicians interested in lung imaging methods and lung physiology.

Purpose.
Until recently, the application of MRI to study lung anatomy and physiology was limited due to the well-known physical and technical imaging difficulties relating to the lung parenchyma. However, constant progress in the MR technology in the last years has significantly improved pulmonary MRI. Specifically, rapid short echo time pulse sequences, such as ultra-short echo time (UTE)[1] or ultra-fast steady-state free precession (ufSSFP)[2] pulse sequences − with optimized data acquisition trajectories combined with multichannel phased array coils for parallel imaging − show promise to overcome present limitations of pulmonary MRI. Here, 3D ufSSFP imaging, recently proposed for improved morphological chest imaging and visualization of the lung parenchyma and vasculature at 1.5 T, was used to explore the functional dependence between the pulmonary MRI signal and volume.

Methods.
MR data acquisition: The study has been performed on three healthy volunteers. MR images of the lung were acquired in breath-hold at different lung volumes, ranging from forced expiration (i.e. residual volume) to forced inspiration (i.e. total lung capacity). All measurements were performed at 1.5T (Siemens MAGNETOM Avanto) using a 12-channel thorax and 24-channel spine coil. The parameters of the 3D ufSSFP pulse sequence were as follows: TE/TR = 0.47/1.19 ms, flip angle α = 15º, RF pulse length = 80 µs, 1563 Hz/pixel bandwidth, field-of-view = 400×400×250 mm^3, two averages, isotropic resolution = 3.1 mm^3, reconstruction matrix = 160^2×80, parallel imaging GRAPPA factor 2, total acquisition time = 17 s.
Image post-processing and analysis: In a first step, the 3D ufSSFP datasets were processed using a median filter (filter size 5×5×5 voxels) to reduce signal noise and remove the vasculature overlaying the pulmonary tissue[3]. Subsequently, lungs were segmented using a 3D fast-marching algorithm implemented in a stand-alone software (Medical Imaging Interaction Toolkit)[5]. The consistency of the segmentations was verified visually slice-by-slice in between datasets and, if needed, corrected manually. Finally, a corresponding mean signal intensity (SI) and its standard deviation was calculated for all segmented lung volumes.

Results. Figure 1 shows exemplary native images obtained using 3D ufSSFP pulse sequence for two different inspiratory volumes (i.e., 1.8L and 4.6L) with corresponding median filtered images. The different emanating signal levels for the lung parenchyma with respect to the two different lung volumes are clearly noticeable. The observed mean signal intensity in the lung parenchyma as function of the lung volume is presented in Figure 2, exemplarily for one volunteer. For the signal intensity (*SI*), empirically, a function of the form

$$SI(V) = \alpha / V + \beta \quad \text{[a.u.]} \qquad [1]$$

was assumed, where V represents the segmented lung volume (see Fig. 1), α a variable, i.e. individual, amplitude, and β the residual signal in the limit of infinite volumes, i.e., noise. From Fig. 2, excellent agreement between Eq. [1] and the experimental data is observed. In summary, for the three volunteers, the following parameters were derived by least-squares estimation: α_1=(274.9±1.8) [L], β_1 = (-13.4±0.6); α_2=(242.6±4.0) [L], β_2 = (-7.3±1.3) ; α_3=(300.0±5.7) [L], β_3 = (-10.8±1.7).

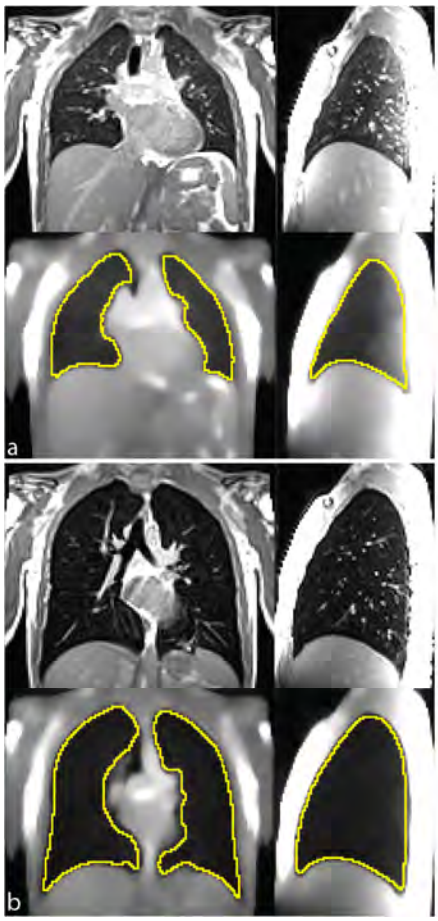

Figure 1. *Exemplary coronal and sagittal ufSSFP chest images at 1.5T for two different lung volumes: 1.8L (a), and 4.6L (b) with corresponding images after 3D median filtering and segmentation (yellow contour).*

Discussion and Conclusion.
We have investigated the dependence between the pulmonary signal and the lung volume in healthy volunteers. The high signal-to-noise ratio, as provided by 3D ufSSFP imaging, allowed a proper estimation of the residual signal intensity in the lung parenchyma even at very large lung volumes, where contemporary MR imaging techniques are typically dominated by noise. The signal intensity could be empirically described by a non-linear function that scales inversely proportional with the lung volume. For small volume variations, e.g. at the tidal volume range, the dependency of the SI can be approximated by a linear function (see detail-box of Figure 2). In this study, the respiratory phase signal intensity dependence was studied only in healthy volunteers. In future experiments it may be interesting to perform the measurement in patients with respiratory diseases, where the signal intensity dependence on the lung volume may reflect pathological changes in the lung parenchyma and airways.

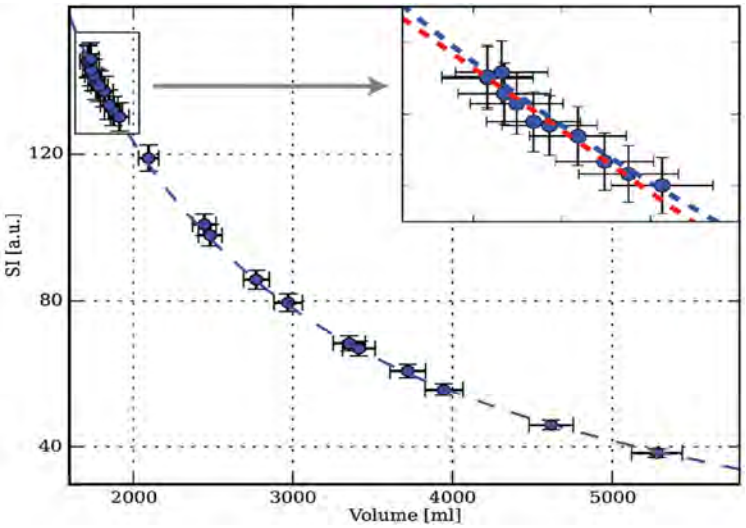

Figure 2. *(a) Signal intensity of the lung parenchyma as a function of lung volume. The dashed lines show a fit of the experimental data to Eq. [1].*
In the box (up-right), for tidal volumes it's also shown a linear fit (red-dashed line).

References:
[1] Johnson KM et al, MRM 2013;70:1241-1250;
[2] Bieri O, MRM2013;70:657-663; [3] Bieri O, ISMRM 2014;2300.