In vivo measurement of T2 relaxation of mouse lungs during inspiration and expiration

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Transverse magnetization, T2 relaxation time, has been a parameter of interest in animal models of lung damage induced by radiation, pulmonary edema and inflammation, however the dependence of T2 on inspiration/expiration has been neglected. Ex vivo it has been shown that T2 is dependent on lung inflation[1]. The effect is rather small and a difference was only found between inflated and degassed lungs. At present little is known about the effect of respiration on measured T2 in the lung in vivo. The aim of this study was to measure in vivo T2 relaxation time in mouse lung at inspiration and expiration.

Materials and Methods: Experiments were performed on a 4.7 T MR scanner (Bruker, Ettlingen, Germany). For the T2 measurement, ten mice were anaesthetized with isoflurane and scanned with a 2D multi spin echo sequence with inter echo time 3.5 ms and 8 echoes. The repetition time was 1000-1200 ms (depending on breathing rate), FOV30x30 mm^2, matrix 96x96, BW 100 kHz, and 6 averages. A coronal slice of 1.5 mm thickness was located centrally in the lungs. Images were acquired in either inspiration or expiration. The animals were freely breathing during image acquisition. Respiration was tracked by a small pressure sensitive pad on the abdomen connected to a computer controlled monitoring system (SA Instruments). For five of the mice the T2 measurement was repeated using an inter echo time of 4.0 ms keeping all other settings identical. T2-maps and relative proton density (PD) maps were calculated on a pixel-by-pixel basis. Theoretical calculation of the expected change in T2 was performed using the static dephasing regime model for mesoscopic inhomogeneities[2].

Results: T2 in the lungs was longer at end expiration than at end inspiration. Using an inter echo time of 3.5 ms the T2 was 9.7±0.7 and 9.0±0.8 ($p<0.01$), for expiration and inspiration, respectively (Fig. 1). The findings were supported by theoretical calculations, which resulted in a change in the same order of magnitude. The relative proton density (lung/muscle tissue) was higher during expiration than during inspiration. With an inter echo time of 3.5 ms the PD was 0.61±0.06 and 0.48±0.05 ($p<0.001$), for expiration and inspiration, respectively. The ratio between inspiration and expiration was 0.78±0.09, consistent with expected volume changes. T2 values were found to correlate with PD ($p<0.001$) (Fig. 2). The regression coefficient is $r^2=0.49$. The results for both T2 and PD were similar for inter echo time 4.0 ms

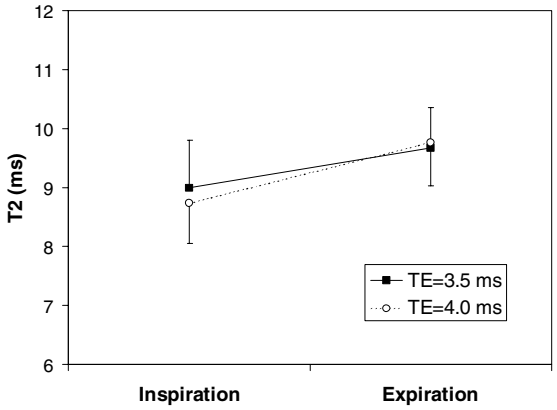

Figure 1. T2 measured during inspiration and expiration. The difference is significant for both inter echo times ($p<0.01$).

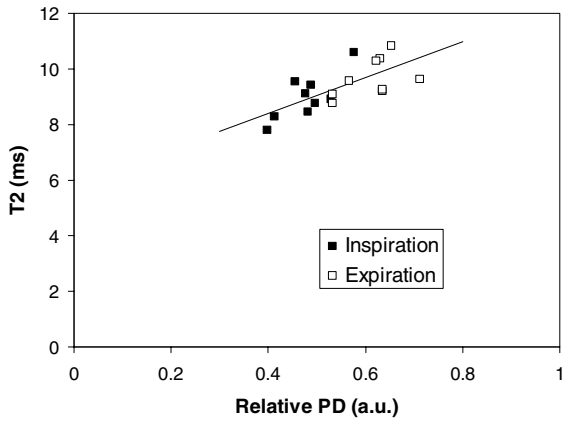

Figure 2. T2 as a function of the relative proton density. T2 increases with increasing proton density ($p<0.001$, r2=0.49)

Discussion: The effect of lung inflation on T2 can be attributed to water diffusion across the internal magnetic field gradients during the inter-echo time[3]. Changes in alveoli size during respiration will induce a change in the internal susceptibility gradients. The linear relationship between proton density and measured T2 in this study supports the argument that the changes obtained are a result of a change in alveolar size. Other factors such as the change in oxygen concentration in the lungs and in the blood during the breathing cycle were estimated and found to have a negligible effect on T2. In conclusion, T2 was found to be dependent on alveolar size. To our knowledge, this is the first time the phenomenon is observed in vivo and for the normal breathing cycle

References

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