Quantification of CPMG relaxation rate in MRI of lung tissue

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Target audience The present work targets investigators with an interest in quantitative CPMG measurements of capillary networks around lung alveoli and also addresses researchers with a focus on theoretical aspects of MR imaging.

Purpose Changes in lung tissue micromorphology are fundamental in evaluating (early stage) lung pathology as well as target-specific therapy strategies e.g. for lung cancer [1]. Here, an expression for transverse relaxation rate R_2 in dependence of interecho-time t_{180} for Carr-Purcell-Meiboom-Gill (CPMG) sequences is provided that allows to quantify microstructural parameters for dense capillary networks around lung alveoli.

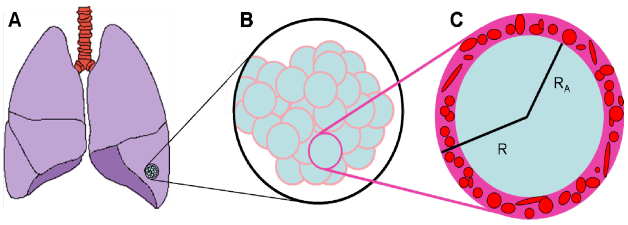

Fig.1: Schematic view of human lungs (A), lung tissue segment with numerous spherical alveoli (B) and cross section of a single alveolus (C). The alveolar tissue wall contains a dense capillary network.

Methods Peripheral lung tissue consists of densely packed alveoli that can be thought of air-filled spheres embedded in a network of closely spaced capillaries. In an external magnetic field B_0, the alveoli can be described as spherical magnetic field inhomogeneities generated by the susceptibility difference between air and blood [2]. In close analogy to Krogh's capillary model, symmetry arguments allow to consider a single sphere of radius R_A surrounded by a spherical spin dephasing volume R [3] with the volume fraction $\eta=R_A^3/R^3$. For $R_2=R_{2,0}+\Delta R_2$ with intrinsic and diffusion-related transverse relaxation rate $R_{2,0}$ and ΔR_2, respectively, ΔR_2 can be expressed within a weak field approximation in close analogy to [4] in terms of inter-echo time τ_{180}, correlation time $\tau = R_A^2/D$, susceptibility-dependent frequency shift $\delta\omega$:

$$\frac{\Delta R_2}{\tau\delta\omega^2}=\frac{8}{\pi^2}\sum_{m=0}^{\infty}\frac{1}{[2m+1]^2}\sum_{n=0}^{\infty}\frac{G_n\kappa_n^2}{\kappa_n^4+[\pi[2m+1]\tau/\tau_{180}]^2} \quad \text{with } G_n=\frac{216}{5\kappa_n^2}\frac{\eta}{1-\eta}\left[1-\eta^{\frac{4}{3}}\frac{j_2'(\kappa_n)}{j_2'(\kappa_n/\sqrt[3]{\eta})}\right]^2\left[\eta\left[\eta^{-\frac{2}{3}}\kappa_n^2-6\right]\left[\frac{j_2'(\kappa_n)}{j_2'(\kappa_n/\sqrt[3]{\eta})}\right]^2+6-\kappa_n^2\right]^{-1}$$

where parameters κ_n obey the equation $j_2'(\kappa_n)\, y_2'(\kappa_n\, \eta^{-1/3}) = y_2'(\kappa_n)\, j_2'(\kappa_n\, \eta^{-1/3})$ and j_2' and y_2' are the first derivatives of the spherical Bessel functions of the first and second kind, respectively.

Results In Fig. 2, theoretical results for ΔR_2 are compared with experimental data for excised lung samples of wistar rats [5]: for passively deflated lung tissue, τ= 3.79 ms, $\delta\omega$ = 276 Hz and consequently magnetic susceptibility follows as χ=0.12ppm (B_0,=2.1 T) which is close to that of blood (χ=0.18ppm [6]). For degassed lung tissue, τ= 2.79 ms, $\delta\omega$ = 6.33 kHz and, since alveoli are here reduced to the size of a capillary (R_A~2.5mm [7]), diffusion coefficient D = 2.24 µm^2/ms is in very good agreement with the expected value (D = 2.30 µm^2/ms) for water spin diffusion [8].

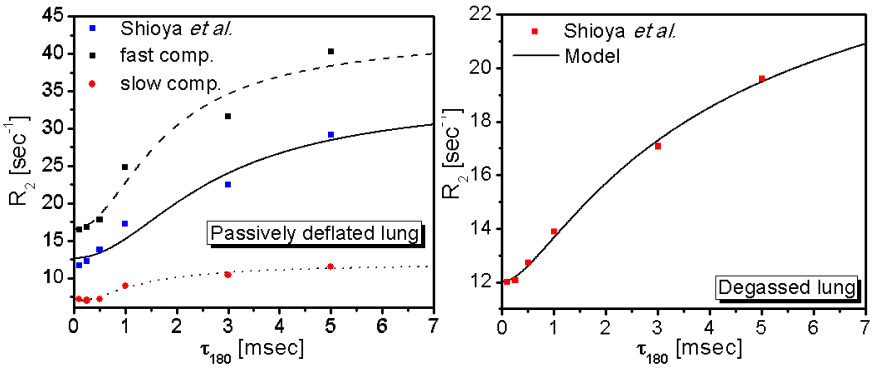

Fig.2: Model CPMG relaxation rate $R_2 = 1/T_2$ in dependence of inter-echo time τ_{180} for passively deflated and degassed lung tissue (continuous lines) in comparison with experimental data [5]. Also, in the left figure, model R_2 for fast and slow components of the biexponential inverse T_2 signal are shown and compared with experimental values (dashed and dotted lines, respectively) [5]. For small τ_{180}, R_2 increases quadratically with τ_{180} to reach a plateau for larger τ_{180}, though R_2 grows slower for degassed lung tissue due to the reduction of air-tissue interfaces.

Discussion & Conclusion Model signal behavior for R_2 agrees well with experimental data. A possible application might be the determination of pseudo-diffusion coefficients for in-vivo lung tissue measurements to evaluate lung perfusion in the intravoxel incoherent motion model [9].

References

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