The Distribution of Pulmonary Perfusion as Measured by FAIR MRI

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Introduction

Various pulmonary conditions such as pulmonary arterial hypertension are characterized by alterations in lung perfusion patterns[1]. MRI could be a useful tool for diagnosing and monitoring disease-dependent changes in perfusion distribution. This application requires knowledge of normal perfusion distribution, often thought to be linearly distributed according to gravity. Some non-MR studies have shown, however, that anatomic factors dominate over gravity in determining pulmonary perfusion. This study's goal was to use MRI to determine the distribution of pulmonary perfusion in healthy subjects.

Methods

Using a 3T Philips MR scanner with a six-channel torso coil, the arterial spin labeling technique of flow sensitive alternating inversion recovery[2] (FAIR) was used to acquire relative perfusion images of a sagittal slice though the right lung of eight informed and consenting volunteers. All subjects were imaged in both the prone and supine postures. In each image, a primary region of interest (PROI) isolating the lung was manually selected. After large blood vessels were removed by a simple thresholding technique, the perfusion data within the PROI was divided into ten secondary ROIs (SROIs) by three separate methods in order to investigate variations in relative perfusion along the radial, gravitational and isogravitational directions. For the radial analysis, ten concentric ring-shaped SROIs of equal width were defined about the lung's geometric center, while, for the gravitational and isogravitational analyses, ten bar-shaped SROIs of equal width were defined to segment the lung along the gravitational and isogravitational directions. Perfusion in each SROI was normalized to the mean for that subject and plotted versus SROI position.

Results

The radial perfusion pattern is characterized by a marked central-to-peripheral decline on the average of 50% (Figure 1, $p<0.005$ for slopes), a posture-independent result as evidenced by a strong correlation between prone and supine data ($R^2=0.93$). The data do not reveal a gravitational dependence to perfusion, as shown by the anatomically similar prone and supine blood flow distributions in Figure 2. The clear correlation between prone and supine gravitational direction data ($R^2=0.88$) directly contrast the results expected if gravity, rather than anatomic position, were the major determinant of perfusion. Along the isogravitational direction, perfusion is similar to the gravitational data in its non-uniformity, displaying higher values in the central-most regions. Isogravitational data were likewise well-correlated for both postures ($R^2=0.79$).

Figure 1

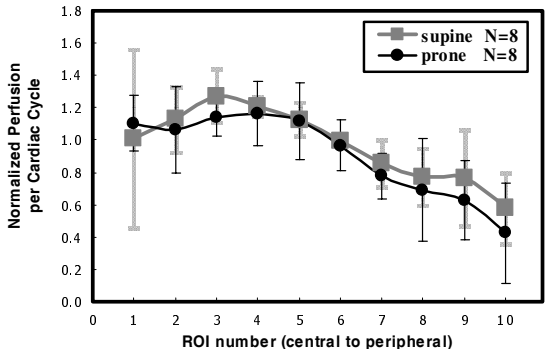
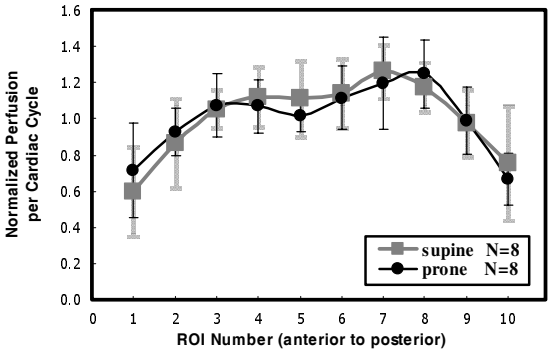


Figure 2



Discussion & Conclusions

Results indicate that the distribution of pulmonary perfusion is largely independent of gravity and instead dominated by radial anatomical position. While gravitational effects have been demonstrated in a variety of lung MR signals[3,4,5,6], no other MR study to date has examined perfusion in more than two ROI's along one direction per subject in a series of subjects. A limitation of the present study is that perfusion was only examined in one slice. Even so, the similarity of our results for supine and prone postures for all analysis directions (radial, gravitational, isogravitational) as well as the marked heterogeneity of blood flow in the isogravitational direction indicate that anatomically determined perfusion differences (such as the radial gradient) are more influential in determining the overall perfusion distribution than are gravitational effects. These results corroborate findings from major microsphere studies[7], human radionuclide imaging[8], and fractal vasculature modeling[9] and reveal the normal patterns of pulmonary perfusion distribution against which future patient data can be compared.

References: 1) Rubin LJ. Proc Am Thorac Soc 3:111-115, 2006. 2) Kim SG. Magn Reson Med 34: 293-301, 1995. 3) Mai *et al*. Magn Reson Imaging 17:355-361, 1999. 4) Stock *et al*. J Magn Reson Imaging 3:557-561, 1999. 5) Keilholz *et al*. Magn Reson Imaging 19:929-935, 2001. 6) Bankier *et al*. J Magn Reson Imaging 23:115-122, 2006. 7) Glenny RW *et al.* J Appl Physiol 71:620-629, 1991. 8) Hakim TS *et al.* J Appl Physiol 63:1114-21, 1987. 9) Glenny RW and Robertson HT. J Appl Physiol 70:1024-30, 1991.