

## MRI pulmonary perfusion imaging as a quantitative predictor of regional pulmonary vascular resistance in pulmonary hypertension

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### Introduction

The key diagnostic variables in the diagnosis of Pulmonary Hypertension (PH) are considered to be Cardiac Output (CO), Pulmonary Artery Pressures (PAP) and Pulmonary Vascular Resistance (PVR). At present the gold standard technique for the measurement of these variables is Right Heart Catheterisation (RHC). Well validated non-invasive alternative techniques using Echocardiography or MRI exist for estimating both CO and PAP (1-4), however currently no clinically validated method exists for the non invasive estimation of PVR. PVR is defined as;  $PVR = \Delta P/Q$  [1], where PVR is the pulmonary vascular resistance (fluid resistance),  $\Delta P$  is the pressure difference across the pulmonary circuit, and Q is the rate of blood flow through it (1). In this work we demonstrate a strong correlation between the mean transit time from gadolinium contrast enhanced lung perfusion MRI and PVR in patients with PH.

### Materials and Methods

21 patients were selected who had undergone RHC and contrast enhanced pulmonary MRI within 72 hours of each other. Patient mean age was 59 years, SD 11.4. 7 patients had Idiopathic PAH, 5 had Chronic Thrombo Embolic Pulmonary Hypertension, 3 patients had Systemic Sclerosis without PAH and 6 patients had Systemic Sclerosis with PAH.

Images were acquired on a 1.5T whole body MRI system (HDx, GE Healthcare, Milwaukee, USA) using a time resolved 3D spoiled gradient echo sequence with view sharing (TRICKS sequence (5)). An 8 channel cardiac array coil was used throughout. The imaging parameters were; coronal orientation, TE=1.1ms, TR=2.5ms, Flip angle 30°, FOV 48x48, Asset x2, in slice resolution 200x80, Bandwidth 125kHz, slice thickness 5mm, approximately 32 slices, 48 time points with an overall 3D frame rate of ~ 0.6 s. Acquisition time for the complete time resolved perfusion sequence was on average 29.5 secs, range 25-32 secs. MR data acquisition was preceded by contrast injection of a 0.05ml per kg patient weight of Gd-BT-D30A (Gadovist, Schering, Berlin, Germany) at a rate of 5ml per second with a 20ml saline flush via a cannula positioned in the antecubital fossa, injection rate was controlled using a remotely activated pump injector (Spectris, MedRad).

For each patient parametric hemodynamic maps (positive enhancement integral and mean transit time) were created using GE functool software from the passage of the contrast bolus through each pixel. ROIs were used to outline the complete lung field on each of three sample coronal slices (at the level of the aortic root, pulmonary artery bifurcation and descending aorta). For each patient the time of peak enhancement on each sample slice was noted and by analysis of the individual time frames, the time at which the contrast bolus reached: 1. Superior Vena Cava (SVC), 2.Right Atrium (RA), 3.Right Ventricle (RV) and 4.Pulmonary Artery (PA) was noted. The mean time to peak enhancement (TTP), defined as the time between the contrast bolus being detected at each of points 1-4 to the peak MR signal in each sample slice was then calculated and these values compared with the PVR values using Pearson's Rank Correlation.

### Results and Discussion

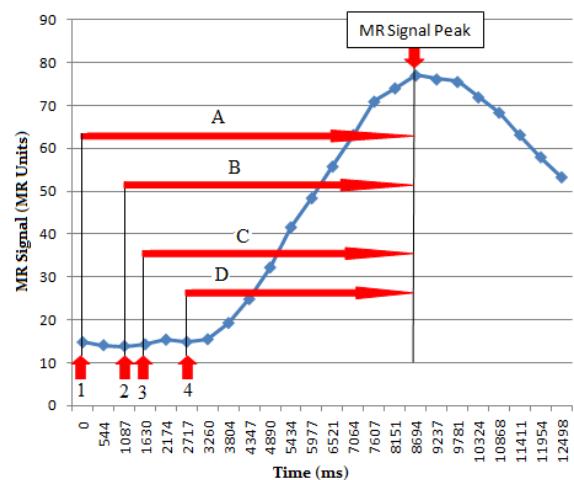
By using 4 possible anatomical locations for the initial detection of the contrast bolus and using 3 coronal sample slices for the peak MR units it was possible to calculate 12 correlation coefficients. These coefficients varied between 0.801 and 0.922 with all correlations highly statistically significant ( $P < 0.001$ ). The highest overall correlation coefficient was 0.922, when the TTP was defined from the SVC to the peak lung MR signal at the level of the Descending Aorta. It was found that correlation coefficients calculated from the most posterior sample slice (at the level of the Descending Aorta) were generally higher than those from the other sample slices as might be expected given the preferential anatomical perfusion in the supine position. From a physical perspective if we define flow rate as  $Q = dV/dt$  [2], then for a given pressure differential across the pulmonary vasculature of a fixed total blood volume (V), Eqs [1] and [2] would suggest a linear dependence between the PVR and  $dt$  (=TTP).

Conclusion Further studies on a larger and more diverse group of patients are needed to further validate this technique however the results of this study appear to indicate that Time to Peak could be an appropriate parameter as a non invasive estimator of PVR in PH.

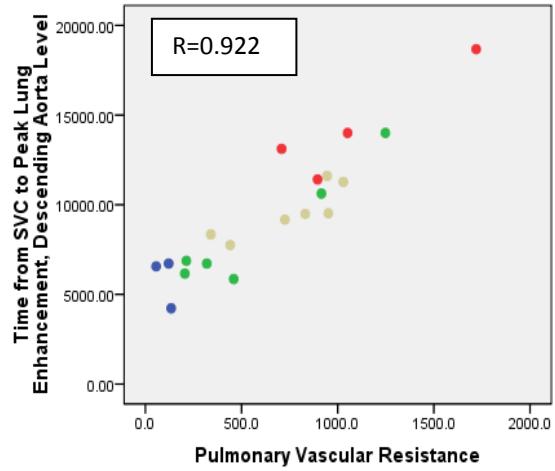
Acknowledgements: Bayer, UK EPSRC, Pfizer

References: 1)Ohno et al. AJR. 2007;188(1):48-56. 2)Ley et al. EJR. 2007;61(2):251-5. 3)Sergi et al. Radiology. 2010;254(2):601-8.

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Contrast in the: Time to Peak from  
1) SVC 2) RA 3) RV A) SVC to Peak Signal B) RA to Peak Signal  
4) PA C) RV to Peak Signal D) PA to Peak Signal



Correlation between PVR and TTP from the SVC at the level of the Descending Aorta