Introduction

Pulmonary hypertension (PH) is a progressive condition defined by elevated mean pulmonary arterial pressure, and is associated with a poor prognosis. Time-resolved 3D MR pulmonary angiography tracks the passage of a contrast bolus through the pulmonary vascular system in 3D. Previous studies assessing 3D MR angiography data from pulmonary artery [1] and lung [2, 3] regions of interest (ROIs) have shown that the time-resolved data correlates well with invasive haemodynamics in patients with pulmonary hypertension (PH). These studies have all used simple linear regression between contrast transit times and RHC metrics in order to investigate correlation of imaging and catheter haemodynamics. None have sought to understand the relationship of imaging based and catheter based haemodynamic parameters from the starting point of a physically realistic model of blood flow.

This study aims to define the relationship between time resolved 3D MR pulmonary perfusion contrast transit times and invasive haemodynamic indices measured at right heart catheterisation (RHC) using a basic physical model of blood flow in the pulmonary artery in patients with PH.

Methods

59 patients with PH and seven patients with ‘No PH’ underwent MR perfusion imaging at 1.5T using a 3D spoiled gradient echo sequence, RHC was performed within 48 hours of the MRI scan. Pulmonary artery rise time (PA-RT) and pulmonary artery time to peak (PA-TTP) values were generated from ROI’s placed in the main pulmonary artery (PA). Figure 1 (right) shows an Illustration of the anatomical locations where ROI analysis was performed in the SVC and the pulmonary artery. The SVC was used as the reference start time. In this simple model we assume a cylindrical pulmonary vessel of cross sectional area A, which has blood flow Q and an average velocity v. This model assumes a plug flow of an incompressible fluid, along a rigid (non-compliant) vessel, which is being driven by a cardiac output equal to Q. Figure 2 (right) illustrates plug flow with flow velocity (v) and cross-sectional area (A). Blood flow, Q = Av, assuming laminar flow of a non-compressible fluid and a non-compliant vessel wall.

Results

PA-RT and PA-TTP values were significantly prolonged in patients with PH compared to patients with ‘No PH’, p<0.0001. Based on a simple fluid dynamics model, regression curve fitting identified significant inverse proportional relationships between cardiac output (CO) and 3D MR perfusion contrast transit times of PA-RT (R²=0.37) and PA-TTP (R²=0.38), this relationship was improved when CO was corrected for vessel cross-sectional area, PA-RT (R²=0.51) and PA-TTP (R²=0.58). CI (CO/body surface area) also demonstrated a significant inverse proportional relationship with PA-RT (R²=0.56) and PA-TTP (R²=0.58). Figure 2 (right) shows the results of curve fitting analysis showing inverse relationships between 3D MR perfusion indices and CI (L/min/m²). Direct proportional relationships that have been previously reported were not found to be either physically realistic, or strong in correlation between 3D MR perfusion contrast transit times and invasive catheter measurements of pulmonary vascular resistance (PVR), and the pulmonary vascular driving pressure from our plug flow model.

Conclusions

CO and MR perfusion contrast transit times in the pulmonary artery show a strong inverse linear relation as predicted by the simple fluid dynamics model proposed.

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References


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Time-resolved 3D MR angiography transit times are inversely proportional to cardiac index