A Non-Invasive Assessment of Cardiopulmonary Hemodynamics with MRI

Octavia Bane[1,2], Sanjiv J Shah[3], Michael J Cuttica[4], Jeremy D Collins[1], Senthil Selvaraj[5], Christoph Guetter[6], James C Carr[1], and Timothy J Carroll[1,2]

[1]Feinberg School of Medicine, Radiology, Northwestern University, Chicago, IL, United States, [2]Biomedical Engineering, Northwestern University, Evanston, IL, United States, [3]Feinberg School of Medicine, Cardiology, Northwestern University, Chicago, IL, United States, [4]Feinberg School of Medicine, Pulmonary Care, Northwestern University, Chicago, IL, United States, [5]Feinberg School of Medicine, Northwestern University, Chicago, IL, United States, [6]Corporate Research, Siemens Corporation, Princeton, NJ, United States

Purpose: Pulmonary hypertension (PH) is a disease of the pulmonary vasculature, with subtypes pulmonary arterial (PAH) and pulmonary venous hypertension (PVH), usually diagnosed by right-heart catheterization (RHC). We propose a method to calculate PA pressure waveforms according to the two element windkessel model [1] from non-invasive measurements of pulmonary circulation parameters by magnetic resonance imaging (MRI).

Methods: We validated our approach in a study of 8 consecutive patients (8 females, average age 55 years, age range 32-70 years) with suspected PH (mean pulmonary arterial pressure, mPAP≥25 mmHg), who underwent RHC, Doppler echocardiography and cardiac MRI. Echocardiographic measurements of early diastolic mitral inflow and mitral annulus velocities were replicated by MRI in 7 patients, since not all sequences were performed in one patient. The flow and tissue velocities were used to estimate pulmonary capillary wedge pressure (PCWP) [2]. Pulmonary vascular resistance (PVR) was calculated as (mPAP-PCWP)/RV cardiac output, by RHC and MRI in this subset of 7 patients. Local area compliance, the ratio of changes in vessel cross section and pressure between systole and diastole (C_A=dA/dP), was estimated from MR images by a method previously used to measure compliance in the aorta [3]. Area compliance can be expressed independently of pressure change [3], in terms of known blood density and parameters measurable by MRI**,** as $C_A = (((\Delta A)^2 A)/((\Delta Q)^2 \rho)$, where ρ is the density of blood, A is vessel cross-section at diastole, ΔA change in area and ΔQ change in flow between systole and diastole. The cross-sectional area, as well as flow, was measured in the three proximal pulmonary branches at peak systole (maximum area) and end diastole (minimum area), using commercial post-processing software. Local area compliances were multiplied by typical vessel lengths (3 cm in main PA, and 2 cm in right and left PA) to obtain volume compliance (capacitance). Total lung vascular compliance (C) was estimated from the sum of proximal compliances [4].

Pulmonary pressure during systole and diastole was calculated from flow, PVR and C, as:

$$P_{systole}(t) = \frac{1}{C}(Q(t) \otimes e^{-\frac{t}{PVR \cdot C}}) + P(t_s)e^{-\frac{t}{PVR \cdot C}} \quad \text{Equation 1}$$

$$P_{diastole}(t) = P(t_{es})e^{-\frac{t}{PVR \cdot C}} \quad \text{Equation 2}$$

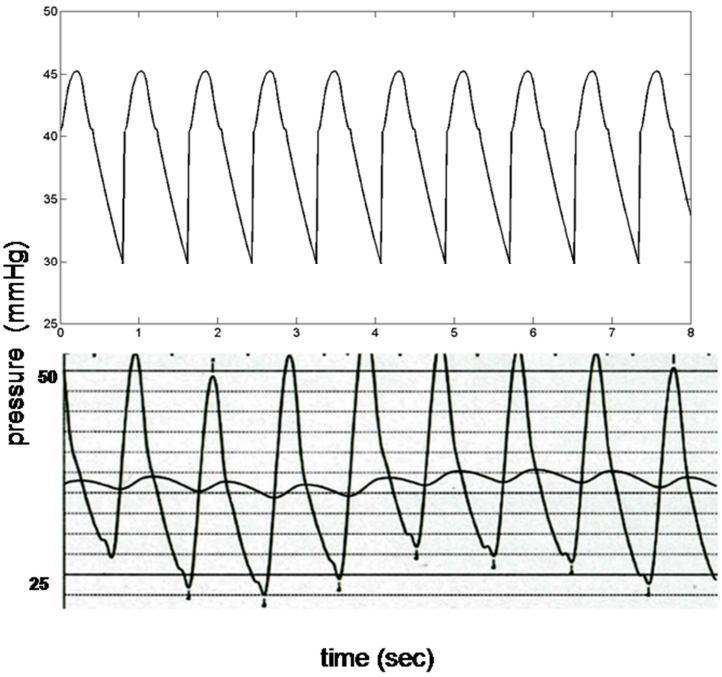

Figure 1: Comparison of MRI (top) and RHC down) pressure wave forms in representative patient with PAH.The MRI waveform is free of the variation due to breathing in RHC, and stable, although it results from a flow wave form averaged over 20 seconds

where $P(t_s)$ is the pressure at systole onset, and $P(t_{es})$ is the pressure at end systole, and $P_{end\ systole} = P_{beginning\ diastole}$, $P_{end\ diastole} = P_{beginning\ systole}$ from one cardiac cycle to the next. Furthermore, the time-averaged pressure curve must be equal to the mean arterial pressure (mPAP).

Results

There was no statistically significant difference (P<0.05) between parameters measured by MRI and by RHC. $mPAP^{RHC}$ and RV EDV/EF display a linear relationship with slope 1.85 and intercept 30.59 (R^2=0.52), which was used to predict $mPAP^{MRI}$; true $mPAP^{RHC}$ and $mPAP^{MRI}$ show a linear relationship (slope 1.21, intercept -8.7, and Spearman correlation of 0.86). In addition, linear regression of true RHC PVR on PVR predicted from MRI shows a linear dependence with slope 0.32 and intercept 2.84, Spearman correlation of 0.43. $PCWP^{MRI}$ distinguished PVH (PCWP > 15 mmHg) from PAH in 6 out of 7 cases. $PCWP^{MRI}$ under-estimated true $PCWP^{RHC}$ in 1 out of 7 cases, while echocardiography PCWP differed by 40-50% from $PCWP^{RHC}$ in 3 out of 7 cases. The pressure waveforms calculated from MRI parameters (Figure 1) agreed with those at RHC for patients with moderate PH.

Conclusions

We have obtained proof-of-principle results for an entirely non-invasive MRI-based method to construct pulmonary artery pressure waveforms and estimate pulmonary hemodynamics in patients with PH.

References: 1)Westerhof et al. Med Biol Eng Comput. 2009;47(2):131-41**;** 2)Nagueh et al. J Am Coll Cardiol 1997, **30**(6):1527-153 3) Vulliemoz et al., Magn Reson Med. 2002, 47(4):649-54; 4)Saouti et al. Am J Physiol Heart Circ Physiol. 2009,297(6):H2154-60