A Non-Invasive Assessment of Cardiopulmonary Hemodynamics with MRI

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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 (CΔ=ΔA/ΔP), 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 CΔ=(ΔA)2/((ΔQ)2ρ), 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:

\[
P_{\text{systole}}(t) = \frac{1}{C} \left( Q(t) \otimes e^{\frac{t}{PVR \cdot C}} + P(t_e)e^{\frac{t}{PVR \cdot C}} \right)
\]

Equation 1

\[
P_{\text{diastole}}(t) = P(t_e)e^{\frac{t}{PVR \cdot C}}
\]

Equation 2

where P(t_e) is the pressure at systole onset, and P(t_e) is the pressure at end systole, and P_{\text{end}}=P_{\text{beginning diastole}}. P_{\text{end}}=P_{\text{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 (R2=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 PH (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.