

Absolute Quantification of Pulmonary Pressure Waveforms with MRI in Pulmonary Hypertension Patients

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OBJECTIVE Pulmonary arterial hypertension, a disease of proliferation and remodeling of the pulmonary arteries, is currently diagnosed by a mean pulmonary arterial pressure greater than 25 mmHg upon right heart catheterization (RHC). We propose to develop an alternative, non-invasive diagnostic tool by which pulmonary artery pressure is estimated according to the Windkessel model [1] from non-invasive measurements of pulmonary artery compliance, resistance and flow by MRI.

BACKGROUND The two-element Windkessel is an adequate model for the estimation of ventricular after-load [1], particularly on short portions of the pulmonary artery branches, where flow and pressure wave reflection is negligible [2]. The Windkessel model allows expression of pulmonary pressure throughout the cardiac cycle in terms of blood flow, pulmonary arterial compliance and pulmonary vascular resistance (PVR). During diastole, pulmonary pressure can be estimated as an exponential decay from end systolic pressure, with the product of PVR and compliance as the decay constant. Systolic pulmonary pressure is modeled as the convolution of pulmonary arterial flow with the same exponential function (see Figure 3, where P_{es} end-systolic pressure, P_s peak systolic pressure, and $Q(t)$ main PA flow).

The model parameters compliance (ml/mmHg) and PVR (mmHg s/ml) can be measured non-invasively by MRI. Local area compliance, the ratio of changes in vessel cross section and pressure between systole and diastole ($C_A = \Delta A / \Delta P$), can be estimated from MR images by a method previously used to measure compliance in the aorta [2]. Area compliance can be expressed independently of pressure change [2], 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. Based on findings that show reduced volumetric parameters of right ventricle (RV) function in pulmonary hypertension patients [3], PVR can be estimated non-invasively based on pressure-volume loops and RV outflow.

MATERIALS AND METHODS

Data collection Cross-section views of the main, right and left pulmonary arteries acquired (orientation orthogonal to the pulmonary arteries, FOV 285 x 380, matrix size 101 x 192, TE 3.2 ms, TR 37.5 ms, slice thickness 6 mm), in one representative pulmonary hypertension patient on a 1.5 T Avanto scanner (Siemens Medical Systems, Erlangen, Germany) are shown in Figure 1 a). MR CINE flow short axis images of the heart (Figure 1b) were acquired on the same scanner (FOV 344 x 379, matrix size 174 x 192, TE 1.13 ms, TR 48.24 ms, slice thickness 6 mm) in 13 pulmonary hypertension patients. All patients had undergone RHC and were diagnosed with pulmonary hypertension.

Analysis

Compliance The cross-sectional area, as well as flow, was measured in all three proximal pulmonary branches (Figure 1 a) at peak systole (maximum area) and end diastole (minimum area), using the post-processing software Argus (Siemens). 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 was estimated from the sum of proximal compliances [4].

Pulmonary vascular resistance and mean pulmonary artery pressure (mPAP) were calculated from right ventricle volume measurements in Argus (Figure 1b). We have predicted mPAP from the linear relationship between catheter-measured mPAP and the MRI measured ratio of right ventricle end diastolic volume to ejection fraction (EDV/EF). Pulmonary vascular resistance (PVR) was calculated from the ratio of MRI-derived mPAP to flow across the pulmonary valve measured by MRI.

The pulmonary artery pressure wave was computed from the flow wave in the main PA measured by MRI and the parameters above, by convolution analysis according to the two-element Windkessel model, in MatlabR2009a (Mathworks, Natick, MA). The relative pressure obtained by convolution was normalized by systolic, diastolic and mean arterial pulmonary artery pressure measured by RHC, to obtain the absolute pulmonary artery pressure wave.

RESULTS

Proximal lung volume compliance measured in the representative pulmonary hypertension patient (main PA 0.687 ± 0.74 ml/mmHg, right PA 0.774 ± 0.91 ml/mmHg, left PA 0.594 ± 1.23 ml/mmHg) was in the range of values quoted in the literature for pulmonary hypertension patients [3]. However, the total lung vascular compliance calculated by MRI (2.055 ± 1.7 ml/mmHg) over-estimated the compliance calculated from mean pulmonary arterial pressure measured by catheterization.

PVR and mPAP measured by MRI in 12 patients diagnosed with pulmonary hypertension show moderate correlation with the values measured by RHC (Figure 2).

The PA pressure over the cardiac cycle (Figure 3) computed from our model displays the wave characteristics expected from the literature and the same mean value as MRI-measured mPAP (37.4 mmHg).

CONCLUSIONS

We have obtained proof-of-principle results for an entirely non-invasive, MRI based method to assess pulmonary pressure in pulmonary hypertension patients. The accuracy of flow, compliance, PVR and mPAP measurements will be increased by use of a phase contrast sequence with higher temporal resolution and acceleration. The two-element Windkessel model will be compared to the three and four-element Windkessel models, which take into account the characteristic impedance of the pulmonary artery, in order to evaluate the accuracy of pressure computation with each model.

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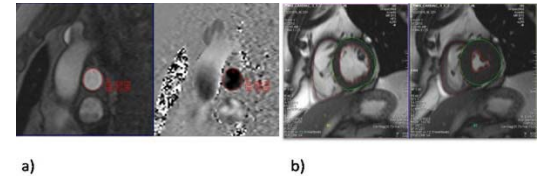


Figure 1 : a) Representative phase contrast (magnitude and phase) image of the right PA cross-section, used for measurement of flow and compliance; b) Representative end-diastole and end-systole CINE's for measurement of RV volumes, mPAP and PVR by MRI

MRI Can be Used to Determine Pulmonary Vascular Resistance (PVR) and Mean Pulmonary Artery Pressure (mPAP).

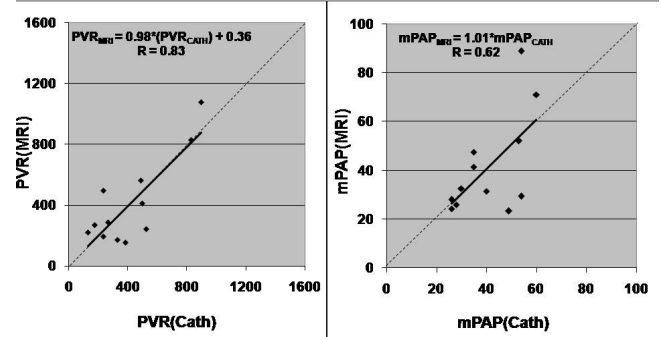


Figure 2. MRI-derived mPAP and PVR correlate with the RHC standard of reference.

Right Heart Catheter and MRI Measured mPAP Values Validate the Computed Pressure Wave

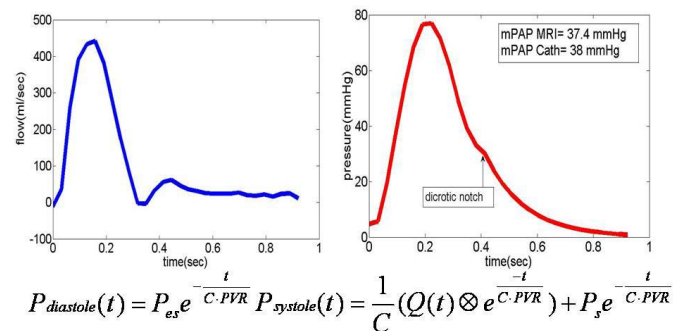


Figure 3. MRI-measured flow (left) and pressure wave form computed from flow (right) in a pulmonary hypertension patient