

A novel method of measuring PVR and total pulmonary arterial compliance using simultaneous pressure monitoring and phase contrast MR flow quantification

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Introduction

Calculation of PVR is essential in the management of patients with suspected pulmonary hypertension. However, arterial pressure and ventricular load are also dependant on total arterial compliance. Thus, complete hemodynamic assessment of patients with pulmonary hypertensive disease requires measurement of both PVR and compliance. Traditionally, due to difficulties in measuring compliance in the clinical environment, assessment of pulmonary hypertensive disease is made on measurement of PVR alone. This may not be sufficient to fully assess pulmonary hypertension and may explain instances where predicated treatment outcomes were incorrect. MR guided cardiac catheterisation allows acquisition of invasive pressure measurements and MR derived flow curves. Combining these measurements one can accurately calculate PVR and through parameter optimization of a 2 element Windkessel model calculate total arterial compliance. We believe that measuring both arterial resistance and compliance is a superior method of assessing pulmonary hypertensive disease and will significantly improve the management of these patients. The purpose of this study was to demonstrate the feasibility quantifying PVR and compliance by combining invasive pressure measurements and MR flow data, acquired during MR guided cardiac catheterization.

Methods

11 patients underwent MR guided cardiac catheterization, in an XMR suite (1.5T Intera I/T MRI scanner and BV Pulsera cardiac x-ray unit, Philips, The Netherlands). An interactive steady state free precession sequence (TE1.45 ms, TR 2.9ms, 10-14 frames per second, free breathing acquisition) was used to visualize the catheter balloon during manipulation within the heart and great vessels. Invasive pressure and MR flow was acquired at baseline (condition 1) and at 20ppm nitric oxide (condition 2). Vascular resistance, the mean pulmonary artery pressure divided by the mean pulmonary artery flow, was calculated in all patients at condition 1 and 2. MR flow data and calculated vascular resistance were inputted into a 2 element Windkessel model. Parameter optimization of the Windkessel model using systolic and diastolic pressure allowed calculation of compliance in all patients at condition 1 and 2. A two-tailed t-test was used to compare the hemodynamic responses to NO. Correlation coefficients were used to study the relationship between resistance and compliance. A p value of <0.05 was taken as statistically significant. Statistical analysis was performed using Matlab (Mathworks, USA).

Results

MR guided cardiac catheterisation with simultaneous acquisition of MR flow data and invasive pressure data was performed on all patients (Fig.2). In response to 20ppm of NO, there was a statistically significant fall in systolic pressure (53.8 ± 23.6 to 50.4 ± 24.1 mmHg, $p=0.002$) and pulse pressure (33.1 ± 12.5 to 30.5 ± 12.8 mmHg, $p=0.007$). This was associated with a significant fall in resistance (0.94 ± 0.61 to 0.87 ± 0.6 mmHg.mL⁻¹.s, $p=0.01$) and a significant increase in total arterial compliance (Fig.1, 0.56 ± 0.55 to 0.66 ± 0.62 mL/mmHg, $p=0.02$). There was a moderate/good correlation between resistance and 1/compliance ($r=0.75$, $p<0.0001$, regression coefficients, $m=2.21$ and $c=0.96$ for all data sets; $r=0.72$, $p<0.0001$ at condition 1; $r=0.77$, $p<0.0001$ at condition 2). In 5/11 patients, resistance decreased by $\geq 10\%$ in response to NO. In 4/5 of these patients the fall in resistance was associated with a significant increase in compliance.

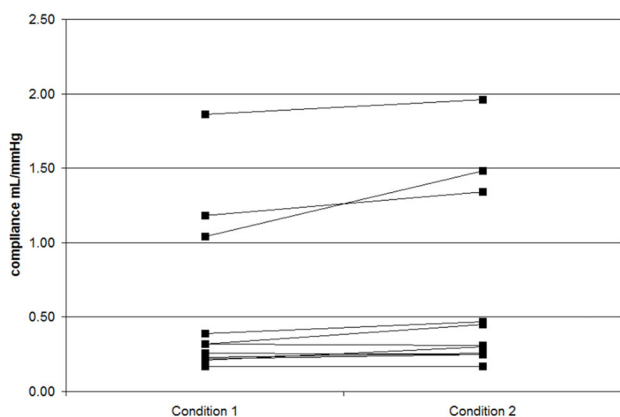


Fig. 1: Plot of total arterial compliance in 11 subjects at 35% oxygen (condition 1) and at 35% oxygen at 20ppm nitric oxide (condition 2)

Conclusion

We have demonstrated the feasibility of quantifying both PVR and total arterial compliance by combining invasive pressure measurements and MR flow data. Currently total arterial compliance is not measured routinely, due to difficulty in measuring it in the clinical environment. MR guided cardiac catheterization allows PVR and total arterial compliance to be measured easily and accurately. Using this technique we have demonstrated an increase in total arterial compliance and a decrease in PVR in response to NO, which is associated with a fall in pulmonary artery pressure. Thus reduction in pressure and ventricular load in response to pulmonary vasodilators maybe due to changes in both PVR and compliance. Further investigation using this technique is required to fully elucidate the relationship between PVR, compliance, and medical intervention. However, the implication is that for full and meaningful assessment of pulmonary hypertension both parameters must be measured. We believe that MR guided cardiac catheterization offers the opportunity to easily calculate pulmonary artery compliance and resistance giving new insights into the pathophysiology of pulmonary hypertension and improving assessment of this disease.

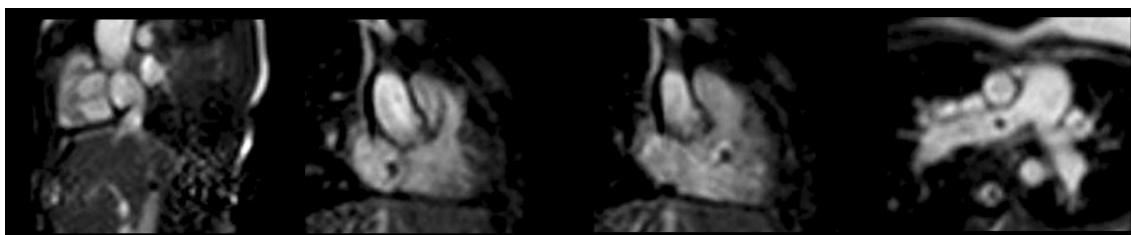


Fig. 2: Balloon tip catheter, filled with carbon dioxide, being manipulated through the; IVC, right heart and pulmonary arteries.