Introduction

Pulmonary hypertension is assessed at cardiac catheterization by measurement of pulmonary vascular resistance (PVR). However, there are risks attached to cardiac catheterization and therefore a non-invasive method of PVR quantification would be useful. Doppler echocardiography has been used to accurately assess systolic and diastolic pressure. Unfortunately, it is difficult to accurately measure mean pulmonary artery pressure and mean pulmonary artery flow using this technique, preventing the calculation of PVR.

Vascular compliance has previously been measured using windkessel modelling. The 2 element windkessel model considers the vasculature to be made up of single compliance and resistance components. These parameters can be measured if haemodynamic variables are inputted into the model. It has been shown that phase contrast magnetic resonance (MR) can accurately quantify blood flow (1). In addition, simultaneous acquisition of invasive pressure and MR flow data may provide a more accurate method of invasively measuring PVR (2).

We use MR flow data as an input to a simple windkessel model, allowing the non-invasive quantification of PVR.

Purpose

To demonstrate the feasibility of this potentially non-invasive method of PVR quantification

Methods

10 patients underwent cardiac catheterization, in an MR interventional suite (1.5T Intera I/T MRI scanner, Philips, The Netherlands) with x-ray back-up (BV Pulsera cardiac x-ray unit, Philips, Best, The Netherlands). PVR was calculated using invasive pressure and MR flow data, at baseline (condition 1) and at 20ppm nitric oxide (condition 2).

A 2 element windkessel model was created (fig 1) and MR flow data was used as the input. A range of resistance and compliance parameters were used, thus producing a series of modelled pressure curves. We assumed that one of these pressure curves was the same as the actual pressure curve, and that the resistance used to create equalled the actual resistance. Using a 2D error minimization protocol (based on the systolic and diastolic pressures) the correct pressure curve was selected and the PVR calculated.

All data is expressed as median (inter-quartile range) unless otherwise specified. Correlation coefficients, linear regression and Bland Altman analysis were used to compare the actual PVR and the modelled PVR.

Results

At condition 1 the median actual PVR was 7.56 WU.m² (3.23 - 12.1 WU.m²) and the modelled PVR was 7.32 WU.m² (3.41 - 11.4 WU.m²) which represents a difference of 3.1% between the 2 methods. At condition 2 the median actual PVR was 6.54 WU.m² (2.73 - 8.93 WU.m²) and the modelled PVR was 6.07 WU.m² (2.35 - 8.07 WU.m²) which represents a difference of 7.2% between the 2 methods.

The percentage change in the actual median PVR in response to nitric oxide was 13.5% and the percentage change using the modelled PVR was 17.1%.

The correlation coefficient between the actual PVR and the modelled PVR using all the data sets was 0.99 (p<0.05), the linear regression between the 2 methods revealed a gradient of 1.04 and an intercept of -0.47 WU.m².

Bland Altman analysis using all data sets revealed a bias of 0.12 WU.m², an upper limit of agreement of 2.04 WU.m² and a lower level of agreement of -1.8 WU.m².

Conclusion

We have demonstrated the feasibility of using a 2 element windkessel model and MR flow data to quantify PVR. This will hopefully form the basis of a wholly non-invasive method of PVR quantification.

Our model requires MR flow data and systolic and diastolic pressures (which can be accurately assessed using Doppler echocardiography) and correlate well with invasively calculated PVR. Furthermore new velocity mapping techniques will hopefully allows accurate quantification of regurgitant jet velocity, allowing all required input data to be acquired using MR. This should make MR a useful tool in the assessment of pulmonary hypertension.

Reference