A novel non-invasive method of pulmonary vascular resistence quantification

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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 has been difficult to accurately measure mean pulmonary artery pressure and mean pulmonary artery flow using this technique, preventing the calculation of PVR. Phase contrast magnetic resonance (MR) has been shown to provide accurate quantification of blood flow. 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). Invasive pressure and MR flow was acquired at baseline (condition 1) and at 20ppm nitric oxide (condition 2) allowing calculation of PVR. A series of pressure curves were calculated using MR flow data inputted into a 2 element windkessel model with a range of PVR's and vascular compliances. Using an error minimization protocol (based on the systolic and diastolic pressures) we were able to select the pressure curve that was most similar to the actual pressure wave and therefore quantify PVR using the set resistance. 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 $WU.m^2$ (3.23 – 12 .1 $WU.m^2$) and the modelled PVR was 7.32 $WU.m^2$ (3.41 – 11 .4 $WU.m^2$) which represents a difference of 3.1% between the 2 methods. At condition 2 the median actual PVR was 6.54 $WU.m^2$ (2.73 – 8.93 $WU.m^2$) and the modelled PVR was 6.07 $WU.m^2$ (2.35 – 8.07 $WU.m^2$) 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 $WU.m^2$. Bland Altman analysis using all data sets revealed a bias of 0.12 $WU.m^2$, an upper limit of agreement of 2.04 $WU.m^2$ and a lower level of agreement of -1.8 $WU.m^2$.

Conclusion

We have demonstrated the feasibility of using a 2 element winkessel model and MR flow data to quantify PVR. This will hopefully form the basis of a wholly non-invasive method of PVR quantification. Currently PVR is quantified at cardiac catheterisation using invasive pressure and flow measurements. We have previously demonstrated the feasibility of combining invasive pressure measurements of MR flow data to accurately invasively calculate PVR. Invasive catheterisation, however, is associated with significant morbidity and mortality, due to vascular damage, x-ray radiation exposure, and general anaesthetic. Therefore an accurate non invasive method would be useful, particularly in paediatric practice. 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.