4D Flow-Sensitive MRI Pulmonary Artery Pulse Wave Velocity in Pulmonary Arterial Hypertension

Christopher J François[1], Alejandro Roldan-Alzate[1], Andrew L Wentland[1], Heidi B Kellihan[2], Naomi C Chesler[3], and Oliver Wieben[1,4]
[1]*Radiology, University of Wisconsin, Madison, WI, United States,* [2]*Veterinary Medicine, University of Wisconsin, Madison, WI, United States,* [3]*Biomedical Engineering, University of Wisconsin, Madison, WI, United States,* [4]*Medical Physics, University of Wisconsin, Madison, WI, United States*

TARGET AUDIENCE: Those interested in cardiopulmonary physiology and pulmonary arterial hemodynamics.

PURPOSE: The rate at which the systolic wave propagates through the vasculature is known as the pulse wave velocity (PWV). PWV is a measure of the distensibility and compliance of vessels. The aims of this study were to (a) demonstrate feasibility of noninvasively assessing PWV using 4D flow-sensitive MRI in a canine model of acute embolic PAH, (b) assess changes in pulmonary PWV in acute PAH, and (c) correlate pulmonary PWV with mean pulmonary arterial pressure (mPAP) and RV function.

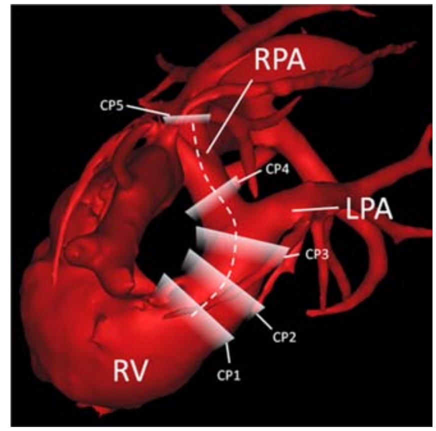

Figure 1 3D volume rendered image from complex difference dataset of PC VIPR acquisition indicating location of cutplanes (CP) in the main pulmonary artery (CP1-CP3) and the right pulmonary artery (CP4-CP5). Dashed line is centerline used to calculate distance between cutplanes.

METHODS: After IACUC approval, three female beagles were induced with propofol and maintained under anesthesia with isoflurane. Studies were performed prior to and following injecting micro-beads (150-500µm) into the right atrium. Pre- and post-embolization mPAP was measured from right heart catheterization (RHC). MRI was performed on a 3.0T clinical system (Discovery 750, GE Healthcare, Waukesha, WI). RV size (RV EDV and ESV) and function (RV EF) were quantified using CINE balanced SSFP acquisitions through the whole heart. 4D flow MRI (Phase Contrast with Vastly undersampled Isotropic Projection Reconstruction[1], PC VIPR) parameters were: FOV: 18-24cm, readout=256, TR=6.8-7.2ms, TE=2.4-2.7ms, acquired spatial resolution=1.3mm isotropic, VENC 150cm/s. PC VIPR was performed after injecting 0.1mmol/kg of gadobenate dimeglumine (Bracco Diagnostics, Inc., Princeton, NJ). Data were reconstructed to 19-34 time frames (depending upon heart rate) for post-processing using retrospective ECG gating and a temporal filter for radial view sharing. Calculation of PWV from 4D flow MRI data was performed using a previously described MATLAB tool[2]. For each 4D flow MRI dataset, cutplanes were placed in the MPA, RPA and LPA to measure PWV for the RPA and LPA, respectively (Figs. 1,2), resulting in 6 PWV calculations pre-embolization and 6 PWV calculations post-embolization. PWV was determined using time-to-upstroke (TTU) and time-to-foot (TTF) algorithms. Values are reported as mean ± standard deviation. Differences in PWV measurements were assessed with a paired Student's t-test. Linear regression analysis was used to assess the correlation between PWV and (a) RV EDV, ESV, SV, and EF; and (b) mPAP.

RESULTS: PWV, RV function, and mPAP data are summarized in Table 1. There was no significant difference between PWV measured from TTU or TTF (p=0.14) or in PWV between LPA and RPA (p>0.4). PWV significantly increased after embolization (p<0.05). Strong correlations were observed between PWV and mPAP. PWV was weakly correlated with indices of RV size and function.

DISCUSSION: <u>To our knowledge, this is the first demonstration of the use of 4D flow MRI to assess PWV in the pulmonary arteries and the first animal study to use MRI to calculate pulmonary artery PWV.</u> The values of pulmonary PWV prior to induction of PAH are similar to those reported previously using invasive measurements in healthy canine[3] and those reported in MRI studies of healthy human subjects using flow-area[4] and transit-time methods.[5] As expected, we observed a significant increase in pulmonary artery PWV after the induction of acute thromboembolic PAH and a strong correlation between PWV and mPAP. The lack of a strong correlation between PWV and RV EDV, ESV, and EF was not entirely unexpected because previous studies have shown preservation of RV EF following acute pulmonary arterial embolization.[6]

CONCLUSION: 4D flow-sensitive MRI offers a noninvasive means of calculating pulmonary artery PWV and assessing pulmonary arterial stiffness in an animal model of acute thromboembolic pulmonary arterial hypertension.

REFERENCES: [1]Johnson KM, et al. MRM 2008. [2]Wentland A, et al. ISMRM 2011. [3]Barainer JD. Circ Res 1967. [4]Peng H, et al. JMRI 2006. [5]Bradlow WM, et al. JMRI 2007. [6]Bellofiore A, et al. Ann Biomed Eng. 2012.

	Pre (N=6)	**Post (N=6)**	**R (PWV_{TTU})**	**R (PWV_{TTF})**
PWV_{TTU} (m/s)	2.3±0.6 (1.6-3.1)	5.5±2.8 (3.1-10.7)*		
PWV_{TTF} (m/s)	2.4±0.7 (1.5-3.5)	5.1±2.6 (2.8-9.6)*		
mPAP (mmHg)	12±2 (10-14)	29±1 (28-30)*	0.66	0.63
RVEDV (mL)	36.5±9.7 (25.5-44)	41.7±9.9 (32.1-51.9)	-0.23	-0.25
RVESV (mL)	17.8±6.9 (10-23)	24.9±7.4 (16.9-31.6)	-0.09	-0.12
RVEF (%)	52.5±7.2 (47.7-60.8)	41.0±5.7(36.5-47.4)	-0.16	-0.12

Table 1 Summary of PWV, mPAP, RVEDV, RVESV, and RVEF data pre and post pulmonary artery embolization. TTU: time-to-upstroke; TTF: time-to-foot. R: Pearson correlation coefficient. *p<0.05.

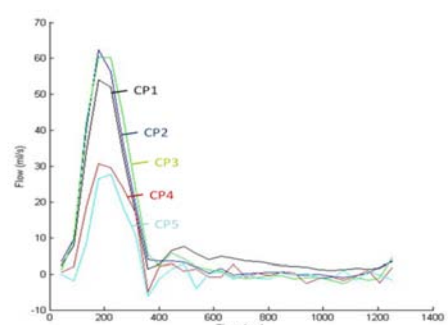

Figure 2 Flow-time curves for the five cutplanes (CP1-CP5) extending from the main pulmonary artery into the right pulmonary artery. These are used to calculate pulse wave velocity from the time-to-upstroke and time-to-foot algorithms.

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