Analysis of Right Ventricular Kinetic Energy in an Acute PAH Animal Model Using 4D Flow MRI.

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TARGET AUDIENCE: Those with an interest in cardiopulmonary physiology and pulmonary arterial hemodynamics.

PURPOSE: Pulmonary arterial hypertension (PAH) is a progressive disease of increased resistance to flow through the lungs. In PAH, capillary level changes lead to upstream remodeling of the pulmonary arteries (PA's) and right ventricle (RV), leading to RV failure in unmanaged cases [1]. RV kinetic energy is a parameter recently studied in humans [2] that provides information about the efficiency of the ventricle while pumping blood to the lungs. Prognostic, non-invasive metrics of ventricular efficiency and ventricular-vascular coupling hold potential to improve understanding of disease development. The purpose of this study was to demonstrate the feasibility of quantifying RV kinetic energy in a canine model of acute thromboembolic PH using time-resolved, three-dimensional, three-directional velocity encoding (4D-flow) MRI.

METHODS: After IACUC approval, three adult female beagles were induced with propofol and maintained under anesthesia with isoflurane. Measurements were performed prior to and following induction of acute PAH by injection of embolizing micro-beads (150-500μm) into the right atrium and ventricle. Pre- and post-embolization mPAP was measured with right heart catheterization (RHC). MRI studies were 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. 4D flow-sensitive MRI was acquired with a radially undersampled acquisition (Phase Contrast with Vastly undersampled Isotropic Projection Reconstruction, PC VIPR [3,4]):TR=6.8-7.2ms, TE=2.4-2.7, acquired spatial resolution=1.3mm isotropic. 4D flow MRI was performed following the administration of 0.1mmol/kg of Gd-based intravenous contrast (gadobenate dimeglumine, Bracco Diagnostics, Inc., Princeton, NJ). Data were reconstructed to 16 cardiac phases using retrospective ECG gating. Using dynamic magnitude images, ventricular volumes were segmented using MIMICS (Materialise, Ann Arbor, MI). These volumes were then utilized to calculate kinetic energy as the sum of the mass times velocity squared: $\sum \rho v^2$ where ρ is the density of the blood (ρ=1025 kg/m^3). These volumes were also used to calculate the RV ejection fraction. Values are reported as mean ± standard deviation in units of mJ. The differences between KE pre and post embolization were assessed with a paired Student t-test. Linear regression analysis was used to assess the correlation between KE and RV EDV, ESV and EF.
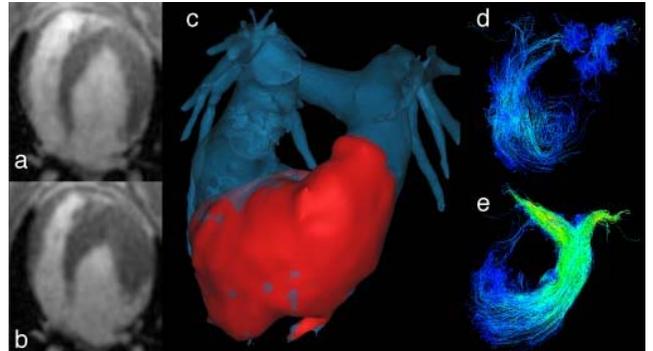

Figure 1 Schematic of the methodology for the calculation of right ventricular kinetic energy in an acute pulmonary arterial hypertension animal model. a. and b. show magnitude images of the heart from PCVIPR at end diastole and end systole respectively. c. shows the 3D volume rendered image from complex difference dataset of PC VIPR acquisition indicating the RV volume (red) used for the KE calculations. d. and e. show streamlines with the velocity distribution (from PC VIPR) in the RV and RVOT at end-diastole and peak systole, respectively.
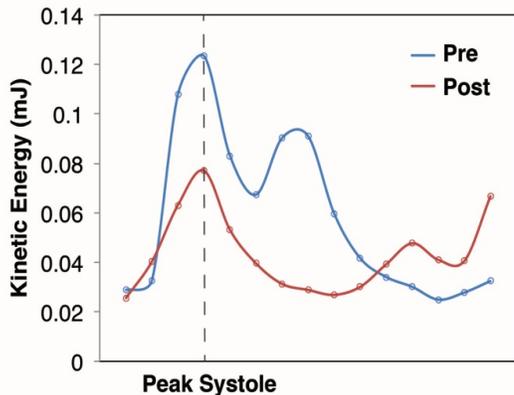

Figure 2. Right ventricular kinetic energy distribution in a complete cardiac cycle (16 time frames) before (blue) and after (red) pulmonary embolization in a dog model. Two main peaks are observed in both pre and post conditions, however the second peak is delayed in post with respect to pre.

RESULTS: Table 1. summarizes the results. Right ventricular peak systolic kinetic energy decreased with the induction of pulmonary hypertension (0.97±0.49mJ pre and 0.78±0.44mJ post), however this decrease was not statistically significant (p=0.32). Also, a reduction in RV ejection fraction was seen (38±7% pre and 30±10% post) but this was also not statistically significant.

DISCUSSION: In this pilot study we have demonstrated the feasibility of quantifying RV KE with 4D flow MRI in an animal model of acute thromboembolic PH. Our findings in pre-embolization canine studies are qualitatively similar to a previous study using 4D-flow MRI in healthy human subjects [2]. As expected we observed a decrease in RV KE after embolization and induction of acute PH. Interestingly, in dog 2, there was no change in RV EF, but there was a very large change in RV KE. This suggests that RV KE may be an earlier indicator of RV failure than RV EF. Future work is required to extent analysis to a larger group of animals and have statistical power. A correlation with pulmonary arterial dynamics would give important insight into the ventricular vascular coupling and more specifically how the pulmonary arterial changes affect the efficiency of the heart.

Table 1. Summary of the results

		EDV (ml)	ESV (ml)	EF (%)	KE (mJ)
Dog1	pre	27.32	15.80	42.17	0.40
	post	24.50	19.90	18.78	0.36
Dog2	pre	43.10	30.13	30.09	1.20
	post	32.70	22.80	30.28	0.77
Dog3	pre	47.10	26.90	42.89	1.30
	post	43.70	26.70	38.90	1.20

REFERENCES: [1] Bellofiore Ann Biomed Eng. 2012; [2] Carlsson Am J Physiol Heart Circ Physiol 2012; [3] Gu AJNR 2007; [4] Johnson MRM 2010;