Free breathing measurement of ventricular volumes from magnitude data of 4D flow-sensitive MRI in a canine model of acute pulmonary arterial hypertension

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BACKGROUND: Right ventricular (RV) function is a major determinant of functional capacity and prognosis in pulmonary arterial hypertension (PAH) [1]. MRI is increasingly used to measure RV volumes, using breath-hold CINE balanced steady-state free precession (bSSFP) sequences. However, in patients with PAH, dyspnea can be severe, and the multiple breath-holds required to scan the entire heart can be difficult, if not possible for many patients. An alternative approach is to use free-breathing methods of acquisition, with respiratory navigation or triggering to coordinate data acquisition with the same phase of the respiratory cycle. Using a three-dimensional approach enables quantification of RV and left ventricular (LV) volumes from the same dataset, which is important because left heart disease and pulmonary hypertension frequently occur together [2]. The purpose of this study was to quantify right (RV) and left (LV) ventricular volumes from the magnitude data of a time-resolved, three-dimensional, three-directional (4D) flow-sensitive, phase contrast (PC) MRI sequence (PC VIPR – Phase Contrast Vastly undersampled Isotropic Projection Reconstruction) [3] in a canine model of acute PAH. We hypothesized that differences in RV and LV volumes between PC VIPR and bSSFP would not be statistically significant, which would then permit simultaneous and co-registered acquisition of time-resolved volumetric and flow data.

METHODS: After IACUC approval, six adult female beagles were induced with propofol and maintained under anesthesia with isoflurane. MRI measurements were performed prior to and following induction of PAH by injecting micro-beads (150-500μm) into the right atrium and ventricle. MRI studies were performed on 3.0T clinical systems (GE Healthcare, Waukesha, WI). Contiguous, axial CINE bSSFP images were obtained through the entire heart and used as a standard of reference for quantification of RV and LV volumes. PC VIPR was performed following the administration of 0.1mmol/kg of Gd-based intravenous contrast (gadobenate dimeglumine, Bracco Diagnostics, Inc., Princeton, NJ).

PC VIPR parameters were FOV: 32x32x22cm, readout=256, TR/TE=6.7/2.4, spatial resolution=1.3mm isotropic. Data was reconstructed to 20 time frames for dynamic post-processing using retrospective ECG gating and a temporal filter for view sharing. Three contiguous axial slices were averaged to minimize the number of slices requiring segmentation of the RV. Images were reformatted into the short-axis (SA) orientation for LV segmentation.

RV and LV end-diastolic (EDV) and end-systolic (ESV) volumes were determined from manually segmented contours of end-diastolic and end-systolic bSSFP and PC VIPR images, respectively. Segmentation of bSSFP images was done using ReportCard (GE Healthcare, Waukesha, WI). Segmentation of PC VIPR magnitude images was done using Osirix (Pixmeo, Geneva, Switzerland). Stroke volume (SV) was calculated from EDV and ESV (SV=EDV-ESV). LV SV was also calculated from measurement of flow through the aorta from the PC VIPR velocity data. Differences between EDV, ESV, and SV measured using the two techniques were assessed using Bland-Altman analysis.

RESULTS:

RV volumes: The average (± standard deviation) RV EDV, ESV, and SV were 34.5±8.1mL, 19.6±5.8mL, and 14.8±3.1mL using bSSFP and 35.3±10.2mL (P=0.45), 21.22±6.8mL (P=0.10), and 14.1±4.1 (P=0.34) using PC VIPR. The mean differences (biases) for RV EDV, ESV, and SV were 0.8mL, 1.6mL, and -0.8mL, respectively.

LV volumes: The average (± standard deviation) LV EDV, ESV, and SV were 22.3±5.4mL, 9.1±3.6mL, and 13.1±3.6mL using bSSFP and 23.0±6.2mL (P=0.51), 13.1±3.8mL (P<0.05), and 9.9±3.4 (P<0.05) using PC VIPR. The average SV from quantification of flow in the aorta using PC VIPR was 11.6±4.0mL (P=0.06 with bSSFP and 0.08 with PC VIPR volumetry). The mean differences (biases) for LV EDV, ESV, and SV were 0.8mL, 4.0mL, and -3.2mL, respectively. The mean differences between PC VIPR LV SV and PC VIPR RV SV was -0.7mL and between PC VIPR LV SV using volumetry and flow quantification was -0.7mL.

SUMMARY: RV and LV volumes and, therefore, function can be accurately determined from the magnitude images of PC VIPR data. This is significant because it allows for the assessment of cardiac function during free breathing rather than a series of breath-holds, which can be quite difficult for patients with PAH. Although free-breathing three-dimensional bSSFP has been used to quantify ventricular volumes [4] and 4D flow-sensitive MRI has been used to study the PA flow patterns in PAH [5], accurate measurement of RV and LV volumes with a 4D flow-sensitive technique allows for the synthesis of time-resolved volumetric and flow data. This should result in shorter overall scan times needed to perform a comprehensive, non-invasive assessment of PA hemodynamics and cardiac function.

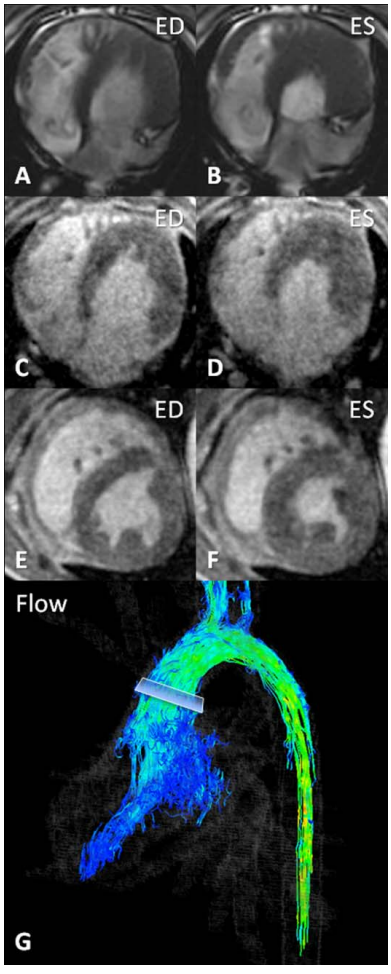

Figure 1 End-diastolic (ED) and end-systoic (ES) images from CINE bSSFP (A,B) and PC VIPR magnitude data reformatted in axial (C,D) and short axis (E,F) orientations. LV stroke volume calculated from PC VIPR short axis was also compared to flow volumes in the ascending aorta calculated from PC VIPR velocity data (G).

REFERENCES [1] Naije R & Huez S. Eur Heart J 2007;9:H5. [2] Haddad F, et al. Prog Cardiovasc Dis 2011. [3] Johnson KM, et al. Magn Reson Med 2008;60:1329. [4] Uribe S, et al. Magn Reson Med 2007;57:606. [5] Reiter G, et al. Circ Cardiovasc Imaging 2008;1:23.

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