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Right ventricular energetics and power in pulmonary regurgitation vs. stenosis using four dimensional phase contrast magnetic resonance☆

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ABSTRACT

Objective: We investigated a full energetic profile of pressure and volume loaded right ventricle (RV) in porcine models by evaluating kinetic energy (KE), stroke power, power output and power loss across pulmonary valves with stenosis (PS) or with regurgitation (PR).
Methods: Fifteen pigs (6 PS and 6 PR, 3 unoperated controls) were studied. Phase-contrast 4D-flow MRI was performed in models of PS and PR at baseline and at 10–12 weeks, in conjunction with cardiac catheterization. Phase contrast velocities over 1 cardiac cycle were registered with a dynamic mask of the RV segmented from cine images. Mean KE and KE curve profiles were measured, normalized for RV volumes and compared between groups. Right heart catheterization pressures were used to calculate RV stroke power and power output, from which pulmonary valve power loss and RV power output ratio were calculated, and compared between groups.
Results: PS and PR groups had similar KE pre procedure but significant changes in KE post procedure. The PR group had higher RV power output ratio and KE (72.1% ± 11.4%; 20.6 ± 6.1) than PS group (25.6% ± 4.7%; 13.8 ± 5.0) post procedure. Volume loaded RV from PR had higher KE and power output ratio compared to pressure load from PS.
Conclusions: In porcine models of PS and PR, the RV presents altered systolic and diastolic energetic profiles. Pulmonary valve efficiency appeared to decrease in the medium term with somatic growth, with increased power loss in all groups studied, and greatly within the PS group.

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1. Introduction

Large numbers of patients with congenital heart disease (CHD) of the right heart including pulmonary stenosis and tetralogy of fallot (TOF) are living with residual pulmonary stenosis and pulmonary regurgitation (PS and PR). In these patients, the right ventricle (RV) needs to cope with the burden of increased pressure and/or volume overload [1–4]. The RV in these clinical situations undergoes complex adaptation in response to the chronic load stresses, however traditional assessment of RV dimensions and function provide limited insights into this remodeling. Non-invasive methods for the determination of progression of RV remodeling arising from these load stresses are of importance in the clinical follow up of these patients.

It is known that the normal, highly ordered patterns of intracardiac flow may be disrupted with ventricular remodeling in CHD [3]. Four dimensional velocity encoded magnetic resonance imaging (4D-flow MRI) allows visualization of the changing, multidirectional flow fields through the entire heart and great vessels with full three-dimensional coverage, and enables collection of 4D velocity data needed to measure intracardiac flow and kinetic energy (KE) throughout the cardiac cycle [5–9]. KE is an inertial measure combining moving blood flow velocities and dynamic ventricular volume. Alterations of blood flow KE profiles, including mean and peak magnitude and duration, have been studied to provide new insights into cardiac pathophysiology [5–9]. 4D-flow MRI has been used in the assessment of flow dynamics within the left heart in aortic and left-heart diseases [9,10]. There has been limited

☆ New & noteworthy: The pathophysiology of pulmonary valve disease has been studied for long, but little is known about right ventricular kinetic energy and power output changes. We provide new energetic markers of right ventricular pathophysiological remodeling, and differentiate physiological and energetic adaptation of the ventricle to disease progression in stenosis vs. regurgitation.

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application of blood flow and KE in the study of right heart disease [11–15]. Also, the assessment of left ventricular power calculations has been done in CHD, but there is little knowledge of RV power in right-sided CHD [16–18].

The aim is of this experimental study was to quantify and evaluate indices of KE, power output and power loss as remodeling processes of the RV after PS and PR. Full characterisation of the detailed flow patterns through the right heart that mediate optimal movement of flow into the RV and pulmonary arteries were therefore investigated with the purpose of examining the differences in RV heart cycle KE profiles and power between PS and PR. Cine MRI and diagnostic cardiac catheterization were used in conjunction with 4D-flow MRI.

2. Materials and methods

This was a prospective single centre investigation. The study was approved by the Institutional Animal Care and Use Committee and was in compliance with the standards in the Guide for the Care and Use of Laboratory Animals. A total of 15 (12 models of disease and 3 controls) pigs were used in the study. Prior to each experiment, the animals were fasted overnight and pre-anesthetized with an intramuscular mixture of Telazol (4.4 mg/kg), ketamine (2.2 mg/kg), and xylazine (2.2 mg/kg). Intramuscular atropine (0.05 mg/kg) was used to dry oral-tracheal secretions and prevent bradycardia during the study. Following placement of a venous line in an ear vein, the animal was intubated and isoflurane anesthesia (induction at 4%, maintenance at 1.0 to 1.8%) was administered throughout the procedure. The animal was placed on a ventilator at a tidal volume of 10 cm^3/kg of body weight at a rate of 15 breaths per minute.

All animals had baseline 4D-flow MRI performed. Within 24 h of the baseline MRI, surgical PS or PR model creation was performed (see below). Introducer sheaths were placed in bilateral femoral vein and artery percutaneously for catheter access, pressure monitoring, and drug administration. Continuous monitoring of heart rate, arterial blood pressure, and systemic saturations by pulse oximetry was performed during the study.

2.1. PS and PR models creation

Under general endotracheal anesthesia and through a transverse left thoracotomy incision, the chest was entered through the 4th interspace and a rib spreader was placed. The pericardium overlying the pulmonary artery was incised and retracted, and the main pulmonary artery was circumferentially dissected. For creation of suprapulmonary stenosis, a Teflon tape was secured around the main pulmonary artery, and was progressively tightened with intermittent epicardial echocardiographic measurement of systolic flow velocity, until the maximum flow velocity possible was achieved. With this technique, excessive band stenosis caused RV dysfunction and decreased band velocity.

For creation of PR, a side-occluding clamp was applied partway across the pulmonary root, so that a valve commissure was centered within the clamp. Test application of the clamp established the amount of tissue that could be taken in the clamp while still allowing adequate RV output for a short period of hemodynamic stability. A longitudinal incision was made in the pulmonary root at the valve level. Valve leaflets were grasped and pulled through the clamp as far as possible, then excised. Typically, the majority of two leaflets could be removed. The incision was closed with running suture and the clamp was removed.

Epicardial echocardiography was employed to measure the final systolic flow velocity in the case of the banded pulmonary artery model, or to document PR in the pulmonary valvectomy model. The rib interspace, chest wall musculature, and subcutaneous fascia were closed over a tubular rubber drain. Suction was applied to the drain as it was withdrawn from the incision. The skin was closed.

2.2. Echocardiography

Epicardial echocardiograms were performed at the time of creation of the model and transthoracic echocardiograms were performed at the 12-week time point on the day of MRI and catheterization. A commercially available ultrasound system (iE33, Philips Medical Systems, Andover, MA) was used. Digital images were analyzed online by a single observer (SK). The average of 3 samples of each of the measurements was used for data analysis. RV outflow tract peak velocity was measured using continuous-wave Doppler. The duration, maximal velocity and velocity time integral were measured for main pulmonary artery Doppler. Velocities were reported in centimeters per second (cm/s) intervals in milliseconds (ms), and velocity time integral in cm. The modified Bernoulli equation was applied to estimate the RV systolic pressure from the tricuspid regurgitation velocity.

2.3. Cardiovascular magnetic resonance

Each pig underwent a MRI exam twice, once within 24 h prior to model creation and the second 10–12 weeks after model creation. MRI studies were performed on a 1.5 Tesla Philips Achieva scanner (Philips Medical Systems, Best, the Netherlands). Ventricular dimensions and function were assessed with an ECG-gated steady state free-precession cine MR pulse sequence during brief periods of breath-holding in the following planes: ventricular 2-chamber (vertical long-axis), 4-chamber (horizontal long-axis), and short-axis planes (perpendicular to the ventricular long-axis plane based on the previous 4-chamber images), with 8 to 12 equidistant slices (slice thickness 6–7 mm; interslice space 0–2 mm) completely covering both ventricles. The MRI data was analyzed using commercially available software packages (Q-MASS and QFLOW, Medis Inc., Leiden, the Netherlands). The end-diastolic and end-systolic volumes, mass at end diastole, stroke volumes, and ejection fractions were measured for the RV. Ventricular end-diastolic volumes, mass and pulmonary flows were indexed to body surface area. Velocity data was obtained from a high-resolution phase contrast velocity mapping flow-sensitive gradient-echo sequence. Three directional blood flow velocities and magnitude (vx, vy, vz) data over the full cardiac cycle were measured over the whole heart and pulmonary arteries. To avoid respiratory movement artifacts, respiratory gating was performed during the acquisition. Typical scan parameters were: FOV 26 × 26 × 26 cm, reconstruction matrix 128 × 128; reconstructed voxel 1.6 × 1.6 × 2.5 mm; flip angle 5°; repetition time 6.6 ms; echo time 2.5 ms; flip angle FA = 15°; nominal temporal resolution varying with heart rate for 25 time steps, and velocity encoding 250 cm/s. Scan time varied between 16 and 22 min, depending on the model chest size. Within 30 min of the 10–12 week interval MRI, the animal was brought back to the laboratory for diagnostic catheterization of the right heart. Right heart pressures were measured using a 7F Berman catheter introduced via the femoral venous sheath under X-ray fluoroscopy. After completion of the measurements, the animals were euthanized. The 3 non-operated pigs that served as controls were also studied at the 10–12 week time point with cardiac catheterization and 4D-flow MRI for characterisation of normal right heart flows.

2.4. 4D-flow MRI post-processing

All images were saved as DICOM files. Post-processing was carried out with MevisFlow, where 4D-flow MRI dataset was corrected from possible phase offset and phase wrap artifacts, and the 3D phase-contrast velocity data was extracted over a cardiac cycle (25 time-steps). CAIPI software was used for segmentation of a moving LV mask based on anatomical cine images, and an in-house developed MeVisLab module to register the segmented LV moving mask to the respective calculated 3D velocity vector field per time step and to calculate kinetic energy (KE) as described by Al-Wakeel et al. and Carlsson et al. [19,20] MevisFlow was further used for acquirement of mean pulmonary artery (MPA) cross-sectional flow rate and velocity. All three software were developed by Fraunhofer MEVIS, Bremen, Germany.

2.5. Data analysis

The pigs were divided in four groups: (1) baseline pre-intervention, (2) post-intervention pigs with PS, (3) post-intervention pigs with PR, and (4) unoperated controls. Characteristics of each pig were acquired: heart-rate (HR) and weight at each MRI scan, and volumetry from MRI short axis images including: end-systolic volume (ESV), end-diastolic volume (EDV), stroke volume (SV), and ejection fraction (EF). The mean KE and KE curve profiles resulting from MRI post-processing were considered for each MRI scan. For statistical analysis, systolic, early-diastolic and late-diastolic KE peaks were taken into account.

Pressures obtained from catheterization were used to calculate the RV stroke work (RVSW). The RV stroke power (RVSP) was calculated by dividing the RVSW by the contraction time (in seconds) including isovolumetric phase and time since pulmonary valve opens until maximal RV pressure is reached.

Systolic PA peak pressure (sPAP) was acquired in the MPA via catheterization and mean systolic ejection pressure (msePAP) was calculated from: msePAP = 0.80*sPAP [21]. Flow rate and velocity in MPA were acquired via MRI post-processing with MevisFlow. The RV power output was calculated according to Fogel et al.: $RVPO = (0.5 * \rho * v^2_{mean} + P_{mean}) * Q_{mean}$, where ρ is the blood density (1060 kg/m^3), v_{mean} is the mean systolic velocity acquired in MPA, P_{mean} is the msePAP and Q_{mean} is the mean systolic flow rate [22].

Power loss through the pulmonary valve is the difference between power generated in the RV and the power output to the PA. RV stroke power output (RVSPO) ratio is the ratio between the RV power output and RVSP and represents the amount of RVSP that is transferred to the MPA during systolic ejection of the blood from the RV. RVSP, RVPO, and RVSPO ratio were calculated for all groups.

2.6. Statistical analysis

Continuous variables were expressed as mean ± SD (if normally distributed) or as median and range where pertinent; the frequency and percent were summarized for categorical variables. Statistical analyses accounted for the fact that each pig had two MRI examinations (before and after intervention) for pre versus post-intervention analysis. Two-tailed t-students tests were made for the comparisons between pre-intervention with each of the post-intervention groups (PS, PR and Controls) separately, and between the two post-intervention groups (PS vs. PR). Analyses were carried out with SPSS version 21 (IBM Corporation, USA). Effects were considered significant for a significance level of 5% (p-value < 0.05).

3. Results

Animals showed no clinical signs of heart failure in the period between model creation and second MRI assessment. The results from right ventricular remodeling in terms of volumetry, energetics and

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