Pulmonary Valve Replacement Improves But Does Not Normalize Right Ventricular Mechanics in Repaired Congenital Heart Disease: A Comparative Assessment Using Velocity Vector Imaging

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Background: This study evaluated regional right ventricular (RV) mechanics before and after pulmonary valve replacement (PVR) by ultrasonic speckle tracking technology using velocity vector imaging (VVI).

Methods: Fifty-eight patients who underwent PVR (May 1999 to August 2007) were included. Two-dimensional Doppler indices included qualitative pulmonary incompetence, RV outflow tract peak gradient, RV systolic pressure estimate, indexed RV area, and fractional area change; VVI regional indices included peak systolic and diastolic velocities, peak systolic strain, maximal longitudinal displacement, and times to peak measure from 6 RV segments.

Results: PVR was performed at a median age of 12.1 years with echocardiographic analysis at median intervals of 2.8 months before and 30 months after PVR. Peak velocities and displacement increased in all 6 RV segments after PVR; peak systolic strain did not improve consistently. All indices remained significantly lower compared with normal values. There was a significant decrease in the degree of pulmonary incompetence, RV outflow tract gradient, RV systolic pressure, and indexed RV area (23.5 vs. 17.8 cm^2/m^2), but no significant change in the percentage of fractional area change (28.8% vs. 29.6%).

Conclusion: Regional RV mechanics using VVI in a relatively young cohort shows mild improvement after PVR, but the RV is not normalized despite physiologic improvement in loading conditions. This suggests intrinsic dysfunction or chronic myocardial injury that is nonmodifiable or requires earlier intervention to optimize physiology. VVI appears to be a potentially useful quantitative tool for follow-up evaluation of RV performance after congenital heart disease surgery. (J Am Soc Echocardiogr 2008;21:1216-1221.)

Keywords: Pediatric cardiology, Right ventricular function, Tetralogy of Fallot, Ultrasonic speckle tracking, Velocity vector imaging

The subpulmonary right ventricle (RV) in repaired congenital heart disease (CHD) is often subject to ongoing pressure or volume overload and therefore vulnerable to dysfunction. Pulmonary valve replacement (PVR) has been advocated to decrease load stresses to the RV in tetralogy of Fallot and other disease states in which residual pulmonary valve dysfunction is prevalent after repair; it is anticipated that this will improve RV performance and decrease the risk of RV failure and sudden death.[1,2] Assessment of the hemodynamic consequences of load stresses on regional RV function and the effects of PVR on subsequent RV performance is therefore important.

Quantification of RV function is limited using conventional echocardiographic techniques because its nongeometric shape makes mathematic modeling of volume analysis based on 2-dimensional slices through the chamber impossible. This is different than the left ventricle (LV), which is elliptical in shape and routinely assessed for volume change (as ejection fraction) using 2-dimensional echocardiography. Real-time 3-dimensional echocardiography was recently validated for assessment of RV ejection fraction in children[3] but has not become a routine clinical tool given the cumbersome postprocessing needed after image acquisition. Velocity vector imaging (VVI) is a new ultrasonic tool for deriving objective information about myocardial mechanics using speckle tracking analysis.[4,5] "Speckles" (natural acoustic markers) are tracked in the ultrasound image from frame to frame in 2 dimensions, and local tissue movement is determined from the geometric speckle shift. VVI uses a "feature

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tracking" algorithm that combines speckle tracking, atrioventricular valve annulus motion, myocardial blood interface, and myocardial structure.[6] Data regarding the application of this technology are rapidly accumulating.[4-7] The accuracy, validity, and interobserver and intraobserver variability of VVI in the assessment of regional myocardial function has been tested over a wide range of physiologic conditions in comparison with segment-length sonomicrometry.[6] Regional myocardial mechanics before and after PVR in repaired CHD with the RV in the subpulmonary position have not been assessed using this technology. The purpose of this study was to evaluate regional function of the volume- or pressure-loaded RV in this setting and to compare findings with previously published data in children with normal intracardiac anatomy.[7]

MATERIALS AND METHODS

The study protocol was approved by the institutional review board on human research. The cardiology database at the Herma Heart Center of Children's Hospital of Wisconsin and the Medical College of Wisconsin was reviewed for patients who had surgical repair of CHD with pulmonary outflow reconstruction that resulted in completely separated biventricular physiology. Any patient who subsequently required PVR after December of 1998 was eligible for inclusion in this study because this is the date when all echocardiographic imaging was captured and stored digitally using a digital imaging and communication in medicine (DICOM)-compatible protocol.[8] VVI analysis of previously captured echocardiography studies requires that the images be archived digitally using a DICOM-compatible protocol. Medical and operative reports were reviewed to obtain information about demographics and operative procedures.

Digital echocardiograms within 12 months before PVR and at the most recent follow-up after PVR were analyzed. Two-dimensional and Doppler measurements were obtained from standard views using tools on *syngo* US Workplace review stations (Siemens Medical Solutions USA Inc., Mountain View, CA). These included qualitative assessment of global RV systolic function and pulmonary incompetence, the peak instantaneous pressure gradient through the RV outflow tract, an estimate of RV systolic pressure from the tricuspid valve insufficiency jet (peak RV to right atrial pressure gradient plus estimated right atrial pressure of 10 mm Hg), indexed RV area, RV percentage of fractional area change (%FAC), and LV ejection fraction by biplane Simpson's. Pulmonary incompetence was described using color and pulsed-wave Doppler as mild if the regurgitant jet was seen in the RV outflow tract with no retrograde diastolic flow in the main or branch pulmonary arteries, moderate if retrograde diastolic flow was seen in the main pulmonary artery, and severe if retrograde diastolic flow was seen into the branch pulmonary arteries.[9] RV area was measured in diastole and systole from the apical 4-chamber image at the plane of the cardiac crux and indexed to body surface area. %FAC was calculated as (end-diastolic area−end-systolic area)/end diastolic area.

VVI Analysis

syngo VVI technology (Siemens Medical Solutions USA Inc.) was used to display and analyze global and regional myocardial motion. An apical 4-chamber image at the cardiac crux was used for analysis of RV parameters. Good-quality images with clear endocardial definition were selected. From a still frame image, manual tracing of the subendocardial border was performed in end systole and automatically software tracked with the frame of reference at the RV apex, as has been described.[7] Repeated tracing was performed to optimize accurate tracking of the endocardial border. Tracings were accepted only when the VVI visual display mode identified myocardial borders accurately throughout the cardiac cycle. Peak systolic and diastolic velocities, peak systolic strain, maximal longitudinal displacement, and time to peak for each measure were obtained from 6 RV segments: free wall base, mid, and apex; septal base, mid, and apex (Figure 1).

Data are described as mean with standard deviation or median with interquartile range. Differences between parameters before and after PVR were analyzed with the Wilcoxon signed-rank test. The Mann-Whitney test was used for comparative analysis of parameters in normal subjects. Subanalysis using a simultaneous regression method was done separating patients who had a native outflow tract (tetralogy of Fallot, double-outlet RV) from those who did not (pulmonary atresia, truncus). A P value of less than .001 was used for significance, in the light of multiple comparisons generated in the study. The statistical analysis was performed in SPSS version 14.0 (SPSS Inc., Chicago, IL).

RESULTS

There were 90 subjects who met our inclusion criteria and presented digitally stored images for offline analyses before and after PVR. Of these, we found 58 subjects (64%) who had adequate imaging of the RV endocardium for VVI analysis. Patient characteristics for those 58 patients are listed in Table 1. The primary diagnoses included tetralogy of Fallot, pulmonary atresia with ventricular septal defect, truncus arteriosus, and double-outlet RV. The median age at anatomic repair was 14 months (0.2-124 months). Twenty-six patients (45%) had undergone additional procedures to enhance pulmonary blood flow (Blalock-Taussig/Waterston shunt or pulmonary valvotomy) before anatomic repair. The time interval between the anatomic repair and the PVR was 12.7 years (2.1-24.7 years), and 41% had undergone previous PVR. Indications for PVR were at the discretion of the primary cardiologist and generally included significant pulmonary incompetence or RV outflow tract obstruction, frequently in association with varying severity of right heart dilation on noninvasive imaging or patient symptomatology. PVR was performed from May of 1999 to August of 2007 at a median age of 12.1 years (2-23.8 years). PVR used normothermic or moderately hypothermic cardiopulmonary bypass and cryopreserved pulmonary homografts in most patients. Patch enlargement of the intrapericardial pulmonary artery was performed concomitantly when necessary.

Echocardiographic analysis was performed at a median interval of 2.8 months (0.03-12 months) before and 30.1 months (0.07-72.7) after PVR. Two-dimensional Doppler indices measured before and after PVR are shown in Table 2. Qualitative assessment of RV function before PVR showed normal function in 19% of patients; mild and moderate dysfunction were present in 46% and 35% patients, respectively. Moderate to severe pulmonary incompetence was seen in 48 patients (83%). After PVR, there was a statistically significant decrease in the degree of pulmonary incompetence ($P <$ .001), with only 17% having greater than mild incompetence. The average peak RV outflow tract gradient from spectral Doppler decreased from 41 mm Hg (4-108 mm Hg) before PVR to 19 mm Hg (5-52 mm Hg) after PVR ($P <$ .001), and the average estimated RV systolic pressure decreased from 56 mm Hg (23-118 mm Hg) to 39 mm Hg (28-74 mm Hg). Assessment after PVR showed normal RV function in 31%, mild dysfunction in 57%, and moderate dysfunction in 12% of patients. Although a decrease was seen in indexed RV area (23.5 vs. 17.8 cm^2/m^2) after PVR, there was no statistically significant difference in the %FAC (28.8 vs. 29.6). There was also no significant change in LV ejection fraction after PVR.