Energy Transfer Ratio as a Metric of Right Ventricular Efficiency in Repaired Congenital Heart Disease

Namheon Lee, MS,* Ashish Das, MS,* Michael Taylor, MD,† Kan Hor, MD,† and Rupak K. Banerjee, PhD*

*Mechanical Engineering, School of Dynamic Systems, University of Cincinnati, Cincinnati, Ohio, USA; †The Heart Institute, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio, USA

ABSTRACT

Objective. With the success of early repair, continued functional assessment of repaired congenital heart disease is critical for improved long-term outcome. Pulmonary regurgitation, which is one of the main postoperative sequelae of congenital heart disease involved with the right ventricle (RV) such as tetralogy of Fallot and transposition of the great arteries, results in progressive RV dilatation coupled with pulmonary artery (PA) obstruction causing elevated RV pressures. The appropriate timing of intervention to correct these postoperative lesions remains largely subjective. In the present study, we evaluated an energy-based end point, namely energy transfer ratio (eMPA), to assess the degree of RV and PA inefficiency in a group of congenital heart disease patients with abnormal RV-PA physiology.

Methods. Eight patients with abnormal RV-PA physiology and six controls with normal RV-PA physiology were investigated using a previously validated technique that couples cardiac magnetic resonance imaging and invasive pressure measurements.

Results. The mean eMPA of the patient group (0.56 ± 0.33) was significantly lower (P < .04) than that of the control group (1.56 ± 0.85), despite the fact that the patient group had a significantly higher RV stroke work indexed to body surface area (RV SWI) than the control group (0.205 ± 0.093 J/m² vs. 0.090 ± 0.038 J/m²; P < .02).

Conclusion. We determined that the patients had inefficient RV-PA physiology due to a combination of RV dilatation with pulmonary regurgitation and RV outflow obstruction leading to an elevated end-systolic pressure. Using coupled magnetic resonance imaging and invasive pressure measurements, eMPA is determined to be a sensitive energy-based end point for measuring RV-PA efficiency. It may serve as a diagnostic end point to optimize timing of intervention.

Key Words. Congenital Heart Disease; Pulmonary Insufficiency, Energy-based End Point

Introduction

Congenital heart disease (CHD) is the most common birth defect. Approximately 2 million US children were born with CHD between 1940 and 2002.1,2 Particularly, CHD associated with the right ventricle (RV), such as tetralogy of Fallot (TOF; right ventricular outflow track [RVOT] obstruction, ventricular septal defect, aorta overriding, and RV hypertrophy), transposition of the great arteries (TGA, the aorta and pulmonary artery connected to the wrong ventricles), pulmonic stenosis, and aortic valve disease requiring Ross procedure (aortic autograft with RV-pulmonary artery [PA] homograft), accounts for 25–35% of the total CHD.3

With recent advances in diagnosing and surgical treatments for CHD, the early postoperative mortality rate for patients with previously listed defects is significantly reduced to less than 2%.4 However, the late postoperative mortality rate still remains high for these patients. In the third decade after the repair surgery, for example, it surges to approximately 30%.5,6 The primary cause of increased late postoperative mortality rate is the postoperative sequelae of pulmonary regurgitation involving RV dilatation and varying degrees of RV myocardial dysfunction.6,7

Further, a considerable number of patients with the aforementioned defects also develop RVOT obstruction; typically, either from PA conduit stenosis or native pulmonic stenosis itself.8,9 Irrespective of the etiology of obstruction, both

interventions. However, timing of intervention is difficult to determine due to lack of suitable quantifiable diagnostic parameters. In practice, clinicians use a variety of clinical and imaging measures, including RV cardiac indices, such as RV end-diastolic volume (EDV), end-systolic volume (ESV), end-systolic pressure (ESP), ejection fraction, and QRS duration to estimate the right timing of interventions. However, timing of interventions based on individual indices is inherently inaccurate because the RV-PA interaction is complex, and there is interdependence of the parameters.

Recently, energy-based end points have been investigated as metrics of cardiac pathophysiology. In particular, Das et al. from our research group described the body surface area (BSA)-indexed RV stroke work (RV SW) and energy transfer ratio ($e_{MPA}$) between the RV and main pulmonary artery (MPA) using measured RV volume (cardiac magnetic resonance imaging [CMR]) and pressure measurement (catheterization). Energy-based end points require simultaneously measured RV volume and pressure data, which are difficult to obtain in humans using MRI and catheterization. A novel method proposed by Das et al. computes energy-based indices from nonsimultaneously measured pressure and MRI cardiac volume and phase contrast flow data using electrocardiogram gating. With the methodology proposed by Das et al., we have recently published another study that confirmed that the repaired TOF patients ($n = 7$) had a significantly higher RV SW than the subjects with normal RV physiology ($n = 8$).

The present research is a natural extension of our recently published study. We evaluated $e_{MPA}$ between the RV and MPA for patients with RV-PA pathophysiology from TOF, TGA, pulmonic stenosis, and Ross procedure and control subjects with normal RV-PA physiology. The mean $e_{MPA}$ was compared between the two groups to test the hypothesis that $e_{MPA}$ for patients with RV pathophysiology would be significantly lower than that of controls with normal RV-PA physiology. We expect that $e_{MPA}$ may be useful to assess the progression of RV dysfunction in patients with repaired CHD and possibly assist with timing of intervention.

### Methods

#### Study Population

The details of demographics and clinical data of the subjects in our study are presented in Table 1. Patients who had undergone both CMR and right heart catheterization at the Cincinnati Children’s Hospital Medical Center from 2006 to 2010 were registered for the study. There were sixteen subjects, eight males and eight females, who met the inclusion criteria for the study.

Out of 16 subjects, 10 had abnormal RV and PA physiology. Two subjects out of 10 who had either moderate-to-severe tricuspid valve regurgitation or an extremely severe PA conduit stenosis were excluded from the study. As a result, eight subjects with RV pathophysiology were identified to be in the patient group. They included three males and five females with age: 13.3 ± 11.3 years and BSA: 1.2 ± 0.5 m². The patient group had a mean heart rate of 82.6 ± 20.6 beats/min and 78.3 ± 23.6 beats/min during CMR and catheterization, respectively. The difference in mean heart rate between CMR and catheterization was only 5.2% for the patient group. Seven patients had a PA conduit as a result of a surgical intervention during infancy or childhood. The remaining patient had an RVOT patch. Among the patients, there were varying levels of PA or conduit stenosis.

#### Table 1. Demographics and Clinical Data of Patients with RV-PA Pathophysiology and Controls with Normal RV-PA Physiology

<table>
<thead>
<tr>
<th></th>
<th>Control ($n = 8$)</th>
<th>Patient ($n = 8$)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>13.8 ± 8.5</td>
<td>13.3 ± 11.3</td>
<td>&gt;.9</td>
</tr>
<tr>
<td>Male/Female</td>
<td>3/3</td>
<td>3/5</td>
<td></td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.5 ± 0.6</td>
<td>1.2 ± 0.5</td>
<td>.4</td>
</tr>
<tr>
<td>Heart rate, CMR (bpm)</td>
<td>75.3 ± 12.8</td>
<td>82.6 ± 20.6</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Heart rate, catheterization (bpm)</td>
<td>73.5 ± 13.1</td>
<td>78.3 ± 23.6</td>
<td>&lt;.7</td>
</tr>
<tr>
<td>Heart rate change (%)</td>
<td>2.4</td>
<td>5.2</td>
<td></td>
</tr>
</tbody>
</table>

Note: Values are means and standard deviations. Heart rate change (%) = $(HRC_M - HRC_C)/(HRC_M \times 100)$.

BSA, body surface area; CMR, cardiac magnetic resonance imaging; PA, pulmonary artery; RV, right ventricle.
The patient group was again subdivided into two subgroups designated by group 1 and 2. Group 1 consisted of four patients who had TOF and had undergone TOF repair. Group 2 consisted of four patients who had other lesions requiring RV-PA conduits (Ross procedure or TGA with pulmonary stenosis). The details of demographics of the patient subgroup 1 and 2 are presented in the Appendix section with associated table.

Out of the total 16 subjects and excluding 10 patients mentioned previously, the remaining six subjects underwent diagnostic CMR and catheterization for conditions not related to the RV and PA. All six subjects were found to have normal RV structure and function and normal PA anatomy and physiology. Therefore, the RV performance and PA physiology was assumed to be normal. These subjects were considered as the control group for the study (three males and three females, age: 13.8 ± 8.5 years and BSA: 1.5 ± 0.6 m²). The subjects in the control group had mean heart rate of 75.3 ± 12.8 beats/min and 73.5 ± 13.1 beats/min during CMR and catheterization, respectively. The mean heart rate change between CMR and catheterization was only 2.4% for the control group. The difference in physical characteristics between the patient and control groups was insignificant (Table 1).

Data Acquisition
All patients and control subjects underwent CMR studies including functional analysis, phase contrast magnetic resonance (PC MR) imaging, and magnetic resonance angiography. Three different magnetic resonance imaging (MRI) scanners were used for CMR studies: (1) 1.5 Tesla GE Excite Magnet (General Electric, Milwaukee, WI, USA); (2) 3.0 Tesla Siemens Trio Magnet (Siemens Healthcare, Erlangen, Germany); and (3) 1.5 Tesla Achieva MRI scanner (Philips Healthcare, Best, The Netherlands).

CMR
In this study, CMR was performed under general anesthesia with breath holding or respiratory suspension technique. Standard CMR protocols were used. They included steady-state free precession in the 2-, 4- chamber and short axis planes.\(^{27,28}\) steady-state free precession parameters were 20–30 phases per cardiac cycle and with slice thickness of 5–8 mm.

PC MR Imaging
Free breathing fast cine PC MR images of all subjects were obtained on a plane positioned approximately halfway between pulmonary valve and MPA bifurcation, perpendicular to the predominant flow direction to measure MPA flow. For subjects who did not have pulmonary valve, the PC MR velocity measuring plane was positioned approximately halfway between the end of RVOT and MPA bifurcation. Typical PC imaging parameters include 30 phases per cardiac cycle, slice thickness of 5–6 mm, and velocity encoding of 150–450 cm/s.

Cardiac Catheterization
All patients and subjects underwent diagnostic right ventricular catheterization under general endotracheal anesthesia. The pressure in the RV and MPA were measured using a fluid-filled catheter (Cook Medical Inc., Bloomington, IN, USA) under fluoroscopic guidance. The best effort was made to measure MPA pressure at the similar location where MPA flow was measured. RV and MPA pressure curves were acquired in the standard fashion along with electrocardiogram gating. For each subject, typically, four to five waveforms were recorded over the cardiac cycle. The respiratory effects were present on the measured pressure waveforms, i.e., the RV pressure slightly increased with expiration and decreased with inspiration. To compensate for the respiratory effects, we used the pressure pulse (among measured waveforms) that had systolic and diastolic pressures approximately matching its weighted means for coregistration of RV pressure and volume.

Data Analysis

CMR Data for RV Volume
The short axis steady-state free precession stacks were analyzed by standard MRI planimetry techniques (QMASS; version 7.2, Medis Medical Imaging Systems, Leiden, The Netherlands). The RV EDV and ESV, stroke volume, ejection fraction, peak ejection rate, peak filling rate, and RV mass were assessed for each of our subjects.\(^{29}\) To facilitate comparisons, RV EDV, ESV, stroke volume, RV mass, and RV SW were indexed to BSA.

PC MR Image Data for Flow Rate at MPA
PC MR imaging data at the MPA were analyzed by standard techniques (QFLOW; version 5.2, Medis Medical Imaging Systems) to compute MPA flow rate over one cardiac cycle.

Volume-based RV Parameters
Volume-based RV parameters were EDV, ESV, regurgitation fraction, stroke volume, ejection fraction.
fraction, peak ejection rate, peak filling rate, and cardiac output. The regurgitation fraction was computed as a ratio of the backward flow volume and the forward flow volume at the MPA.

\[ \text{Regurgitation fraction} = \frac{\text{Backward flow volume}}{\text{Forward flow volume}} \times 100\% \]  

(1)

Stroke volume was calculated as the difference between EDV and ESV. The ejection fraction was calculated as a ratio of stroke volume and EDV.

\[ \text{Ejection fraction} = \frac{\text{Stroke volume}}{\text{End - diastolic volume}} \times 100\% \]  

(2)

The peak ejection rate and peak filling rate were calculated from the first derivative of RV volume vs. time curve.\(^{24,30–32}\) The peak negative derivative during RV systole was considered the peak ejection rate. Similarly, the peak positive derivative during RV diastole was considered the peak filling rate. These were normalized by the EDV. Cardiac output was computed by multiplying stroke volume with heart rate.

Coregistration of Volume and Pressure Data
The temporal coregistration of separately measured dynamic RV volume curves and RV pressure curves was required to calculate energy-based end-points. The coregistration process was done using each patient’s electrocardiogram gating with an in-house MATLAB program (MATLAB, Inc., Waltham, MA, USA).

Energy Transfer Ratio \(e_{\text{MPA}}\)
We define \(e_{\text{MPA}}\) as the ratio between the total energy of blood flow at the MPA and the RV SW. The RV SW was calculated by computing the area enclosed by the pressure–volume (P-V) loop by applying Gauss theorem.\(^{24,26}\) Therefore, RV SW is given by:

\[ \text{RV SW} = \iint_A p dV = \frac{1}{2} \oint_C (p dV - V dp) \]  

(3)

where \(p\) is the time-varying RV pressure measured by catheterization and \(V\) is the time varying RV volume measurement from CMR. \(C\) is the closed path of integration over the cardiac cycle. A representative P–V loop for a patient and a control subject is presented in the Appendix section. To facilitate intersubject comparisons, the RV SW was indexed to the BSA.

The energy transferred to the blood by the RV varies over the cardiac cycle, increasing rapidly during systole and decreasing during diastole. The rate of total energy transferred to the blood over one cardiac cycle at the MPA (\(\dot{E}_m\)) was calculated using\(^ {24}\)

\[ \dot{E}_m = \rho_m \cdot Q_m + \frac{1}{2} \rho_v m v_m^2 \cdot Q_m \]  

(4)

where \(\rho_m\) is the time-varying MPA pressure measured by catheterization, \(\rho\) is the density of blood (=1035 kg/m\(^3\)), \(v_m\) is the time-varying spatially averaged velocity of blood at the MPA, and \(Q_m\) is the time-varying blood flow rate at the MPA. The blood velocity and flow rate were measured by PC MR imaging. The detail of Eq. 4 in integral form and a representative \(\dot{E}_m\) plot for a patient and a control subject are given in the Appendix section. The first term on the right-hand side of Eq. 4 is the rate of the pressure-flow energy. The second term on the right-hand side is the rate of the kinetic energy associated with the velocity of blood at the MPA.

The quantity \(\dot{E}_m\) is the rate at which the RV is transferring the energy to the blood being transported into the MPA. The net energy transferred by RV (\(E_{\text{net}}\)) over one cardiac cycle (\(T\)) was computed by integrating the rate of total energy transferred to the MPA (\(\dot{E}_m\)) over \(T\).

\[ E_{\text{net}} = \int_0^T \dot{E}_m(t) \, dt \]  

(5)

Based on total RV SW and net energy transferred to the blood at the MPA (\(E_{\text{net}}\)), we define an index, \(e_{\text{MPA}}\) as

\[ e_{\text{MPA}} = \frac{E_{\text{net}}}{\text{RV SW}} \]  

(6)

It may be noted that \(e_{\text{MPA}}\) is a nondimensional number as both the numerator and denominator are in energy units. Further, \(e_{\text{MPA}}\) accounts for energy values at the RV and at the MPA. The RV energy condition is determined by RV SW that combines changes in pressure and volume in the RV, whereas MPA energy status is obtained from MPA pressure and velocity data.

Statistical Analysis
The Student’s \(t\)-test was performed to compare the statistical difference in RV pressure and volume, RV volume-based parameters such as regurgitation fraction and ejection fraction, RV SW\(_i\), and \(e_{\text{MPA}}\) between the two groups. Prior to performing the \(t\)-test, the normality test for all data was performed using both the Shapiro–Wilk test and Kolmogorow–Smirnov test. Instead of the

\[ \text{Congenit Heart Dis. 2013;8:328–342} \]
Table 2. RV Pressure, Volume Data, and Volume-Based Parameters for the Patient and Control Groups

<table>
<thead>
<tr>
<th>RV Pressure and Volume Characteristics</th>
<th>Control</th>
<th>Patient</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac MRI data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-diastolic volume index (mL/m²)</td>
<td>77.9 ± 18.3</td>
<td>102.2 ± 25.3</td>
<td>.07</td>
</tr>
<tr>
<td>End-systolic volume index (mL/m²)</td>
<td>31.6 ± 8.4</td>
<td>47.3 ± 21.0</td>
<td>&lt;.2</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>58.3 ± 7.8</td>
<td>53.5 ± 7.5</td>
<td>&lt;.3</td>
</tr>
<tr>
<td>Regurgitation fraction (%)</td>
<td>0.0 ± 0.0</td>
<td>32.1 ± 8.5</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Peak ejection rate (EDV/s)*</td>
<td>3.0 ± 0.8</td>
<td>2.2 ± 0.5</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>RV mass index (g/m²)*</td>
<td>19.8 ± 4.6</td>
<td>33.9 ± 11.4</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Stroke volume index (mL/beat/m²)</td>
<td>46.3 ± 13.9</td>
<td>54.9 ± 9.5</td>
<td>&lt;.2</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>4.8 ± 1.5</td>
<td>5.1 ± 1.3</td>
<td>&lt;.8</td>
</tr>
<tr>
<td>Cardiac catheterization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-systolic pressure (mmHg)*</td>
<td>24.6 ± 5.5</td>
<td>53.6 ± 7.1</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>End-diastolic pressure (mmHg)</td>
<td>3.2 ± 4.0</td>
<td>5.0 ± 2.4</td>
<td>&lt;.4</td>
</tr>
</tbody>
</table>

Values are means and standard deviations. The parameters with an asterisk (*) have a statistically significant difference (P < .05) between the two groups. EDV, end-diastolic volume; MRI, magnetic resonance imaging; RV, right ventricle.

Table 3. Total Energy at the MPA ($E_{\text{MPA}}$), RV SW, BSA Indexed RV SW (RV SWI), and Energy Transfer Ratio ($\theta_{\text{MPA}}$)

<table>
<thead>
<tr>
<th>Computed RV End Points</th>
<th>Control</th>
<th>Patient</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$E_{\text{MMPA}}$ (J)</td>
<td>0.19 ± 0.11</td>
<td>0.13 ± 0.07</td>
<td>.2</td>
</tr>
<tr>
<td>RV SW (J)*</td>
<td>0.12 ± 0.04</td>
<td>0.24 ± 0.12</td>
<td>&lt;.04</td>
</tr>
<tr>
<td>RV SWI (J/m²)*</td>
<td>0.090 ± 0.038</td>
<td>0.205 ± 0.095</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Energy transfer ratio, $\theta_{\text{MPA}}$ = ($E_{\text{MMPA}}$/RV SW)</td>
<td>1.56 ± 0.85</td>
<td>0.56 ± 0.33</td>
<td>&lt;.04</td>
</tr>
</tbody>
</table>

Values are means and standard deviations. The parameters with an asterisk (*) have a statistically significant difference (P < .05) between the two groups. BSA, body surface area; MPA, main pulmonary artery; RV, right ventricle; SW, stroke work.

Student's t-test, the two-tailed Wilcoxon test was used for RV mass index as it failed the normality test. All statistical analysis was done with SAS 9.2 (SAS Institute, Cary, NC, USA).

Results

Table 2 shows the measured RV pressures, volumes, and volume-based parameters (ejection fraction, regurgitation fraction, peak ejection and filling rates, RV mass index, stroke volume index, and cardiac output) of the patient and control group. The computed energy-based end points, $\theta_{\text{MPA}}$, and RV SWI, for both the patient and control group, are presented in Table 3 and Figure 1. The mean ± standard deviation of individual quantities are presented in both Tables 2 and 3. The quantities showing significant statistical difference (P < .05) between patients and controls are designated by an asterisk (*). The results for the patient subgroup 1 and 2 are detailed in the Appendix section with associated table.

RV Volume

The mean RV end-diastolic volume index (EDV$_{i}$) and end-systolic volume index (ESV$_{i}$) of the patient and control groups are presented in Table 2. The EDV$_{i}$ of the patient group was 102.2 ± 25.3 mL/m² and that of the control group was 77.9 ± 18.3 mL/m². The difference in EDV$_{i}$ between the two groups was marginally significant (P = .07). There was no difference in the ESV$_{i}$ between the two groups (P < .2). The ESV$_{i}$ for the patient group was found to be 47.3 ± 21.0 mL/m², whereas it was 31.6 ± 8.4 mL/m² for that of the control group. Despite the fact that both EDV$_{i}$ and ESV$_{i}$ of the patient group were larger than those of the control group by 31.2% (EDV$_{i}$) and 49.9% (ESV$_{i}$), respectively, the differences were not significant due to large variation among individual subjects in the group.

Volume-based RV Parameters

The patient group had a significantly higher regurgitation fraction (32.1 ± 8.5%) than the control group (0.0 ± 0.0 %; P < .01). The systolic function as measured by mean ejection fraction of...
the patient group (53.5 ± 7.5%) was not different than the control group (58.3 ± 7.8%; P < .3). The patient group had a significantly lower peak systolic ejection rate (2.2 ± 0.5 EDV/s) compared with the control group (3.0 ± 0.8 EDV/s; P < .05). However, the peak diastolic filling rate was not significantly different between the two groups (2.6 ± 0.8 EDV/s for the patient group and 2.8 ± 0.7 EDV/s for the control group, respectively; P < .8). Further, the patient group had a significantly higher RV mass index (33.9 ± 11.4 g/m²) than the control group (19.8 ± 4.6 g/m²; P < .02). In summary, the patient group had somewhat larger RVs with equivalent function, increased RV mass, and lower peak ejection rate.

The stroke volume index and cardiac output of the patient group were 39.3 ± 10.3 mL/beat/m² and 3.5 ± 1.2 L/min, respectively, and those of the control group were 46.3 ± 13.9 mL/beat/m² and 4.8 ± 1.5 L/min, respectively. Although no significant difference was found in stroke volume index and cardiac output between the two groups (P < .3 and P < .08, respectively), the mean values of both the indices were lower for the patient group compared with the control group. This is due to the flow regurgitation in the MPA which reduced stroke volume.

RV Pressure
The mean RV end-systolic pressure (ESP) and end-diastolic pressure (EDP) for the patient and control groups are presented in Table 2. The ESP of the patient group (53.6 ± 7.1 mmHg) was significantly higher (2.2 times; P < .01) than that of the control group (24.6 ± 5.5 mmHg). Conversely, the EDP of the patient group (5.0 ± 2.4 mmHg) and that of the control group (3.2 ± 4.0 mmHg) were not different (P < .4).

The Computed RV Energy-based End Points: Total energy at the MPA (E_{net}), RV SW, BSA-indexed RV SW (SWI), and Energy Transfer Ratio (e_{MPA} = E_{net}/RV SW)
The computed RV energy-based end points are shown in Table 3. The control group had a higher E_{net} (0.19 ± 0.11 J) compared with the patient group (0.13 ± 0.07 J; P = .2). As expected, the patient group had a significantly higher (P < .05) RV SW and RV SWI (0.24 ± 0.12 J and 0.205 ± 0.095 J/m², respectively) than the control group (0.12 ± 0.04 J and 0.090 ± 0.038 J/m², respectively). However, e_{MPA} was significantly lower in the patient group (0.56 ± 0.33) compared with that of the control group (1.56 ± 0.85; P < .04).

The Variations of e_{MPA} and RV SWI with RV Volume and Pressure Measurements
The variation of e_{MPA} with EDV for the subjects in our study is shown in Figure 2A. A negative correlation for the e_{MPA} was found with EDV for both the patient and control groups (r = −0.63 with P = .1 for the patient group and r = −0.80 with P < .06 for}

**Figure 2.** Correlations between (A) energy transfer ratio (e_{MPA}) and end-diastolic volume index (EDVI), (B) RV SW and EDVI. (A) Both the patient and control groups showed a negative correlation between e_{MPA} and EDVI (r = −0.63; P = .1 and r = −0.80; P < .06, for the patient and control group, respectively). (B) A positive correlation was shown between RV SW and EDVI in both the patient and control groups (r = 0.59; P < .2 and r = 0.87; P < .03, for the patient and control group, respectively). RV, right ventricle; SW, stroke work.
the control group). The negative trend of $e_{MPA}$ with EDVI was more pronounced in the control group. A positive correlation between RV SWI and EDVI was shown in both the groups ($r = 0.87$ with $P < .03$ and $r = 0.59$ with $P < .2$, for the control and the patient groups, respectively).

The variation of $e_{MPA}$ with ESVI showed a trend similar to the correlation between $e_{MPA}$ and EDVI (Figure 3A). A negative correlation was found between $e_{MPA}$ and ESVI of both the groups ($r = -0.69$ with $P < .07$ in the patient group and $r = -0.33$ with $P < .6$ in the control group). RV SWI showed a positive correlation with ESVI in both the groups ($r = 0.41$ with $P = .3$ for the patient group and $r = 0.25$ with $P < .7$ for the control group, respectively; Figure 3B).

The variation of $e_{MPA}$ with ESP is shown in Figure 4A. The control group showed a positive correlation between $e_{MPA}$ and ESP ($r = 0.74; P < .1$). However, the patient group had a negative correlation between $e_{MPA}$ and ESP ($r = -0.45; P < .3$). RV SWI had a significant positive correlation with ESP of the patient group ($r = 0.74; P < .04$), whereas the correlation between RV SWI and ESP was insignificant for the control group (Figure 4B).

Figure 5A shows the variation of $e_{MPA}$ with EDP. A significant positive correlation ($r = 0.94; P < .01$) was found between $e_{MPA}$ and EDP for the control group, whereas no significant correlation was found between $e_{MPA}$ and EDP in the patient group. The variation of RV SWI with EDP is shown in Figure 5B. The control group had a negative correlation between RV SWI and EDP ($r = -0.57; P < .3$), whereas there was no significant correlation between RV SWI and EDP for the patient group.

The variation between RV EDVI and ESP is shown in Figure 6A. The control group had a negative correlation between EDVI and ESP ($r = -0.46; P < .4$), whereas the patient group showed insignificant correlation between EDVI and ESP. Similarly, the control group had a significant negative correlation between EDVI and EDP ($r = -0.81; P < .05$), whereas the patient group showed insignificant correlation between EDVI and EDP (Figure 6B).

Discussion

Pulmonary regurgitation with or without obstruction is the primary long-term hemodynamic consequence of many CHDs, such as repaired TOF, TGA, pulmonic stenosis, and aortic valve disease requiring Ross procedure. The chronic pulmonary regurgitation may result in progressive RV dilatation leading to RV dysfunction, arrhythmias, exercise intolerance, and rarely sudden cardiac death. As expected, our patient group had higher mean RV volume than the control group (by 31.2% in EDVI and 49.9% in ESVI).

The majority of our patients had a PA conduit as a result of the surgical intervention performed in their childhood. They had various levels of the PA or the PA conduit obstruction, which also
adversely affect the RV hemodynamics eventually leading to increased RV pressure. As shown in Table 2, the mean RV ESP of the patient group (53.6 ± 7.1 mmHg) was significantly higher than that of the control group (24.6 ± 5.5 mmHg). Interestingly, the RV SWI had a positive correlation with ESP in the patient group (Figure 4B). Apparently, the increased RV ESP of the patient group provides the RV with additional momentum to the blood flow toward the PA. Although RV ESP increases, ε_{MPA} decreases in the patient group (Figure 4A).

The PA regurgitant flow and PA obstruction directly affect the RV performance and efficiency as confirmed in our previous work. It showed that a patient with repaired TOF physiology, including PA regurgitation and PA obstruction, had a significantly higher RV SWI compared with a subject with normal RV physiology. Thus, both RV pressure and volume overloading resulting

Congenit Heart Dis. 2013;8:328–342
from mainly PA regurgitation and PA obstruction need to be taken into account for long-term follow-up for patients after repair.

The majority of CHD patients who underwent the repair surgery may not have high pressure level but volume enlargement of the RV. However, the pressure level can gradually increase during the postoperative period due to RV hypertrophy and/or obstruction such as pulmonic stenosis. Thus, both the level of RV pressure and volume increase has to be closely monitored during the postoperative period. In this study, we observed that RV ESP for the patients did not correlate with RV EDV, \( r = 0.20 \). It may be indicating that independently using RV volume or pressure may not be adequate to delineate the RV abnormal physiology. Whereas, \( e_{MPA} \) showed better correlations with both RV ESP \( (r = -0.45) \) and RV EDV \( (r = -0.63) \) and ESV \( (r = -0.69) \) for the patients.

Although the \( P \) values for comparing the group mean between the patient and control groups for RV EDV and ESV \( (P = .7 \) and \( P < .2 \), respectively) were not significantly different, \( e_{MPA} \) was significantly lower \( (P < .04) \) in the patient group than the control group. This is because RV ESP for the patients was significantly higher \( (P < .01) \) than the control subjects. Considering this fact, \( e_{MPA} \) has an advantage of incorporating both RV volume and pressure data into one single index. Therefore, we believe that \( e_{MPA} \), as a longitudinal (time history) measure, could delineate abnormal RV-PA physiology for patients better than using current RV indices separately.

Present limitation of \( e_{MPA} \) is that it requires invasive cardiac catheterization for pressure measurement which is generally not a standard of care procedure for a typical CHD patient. However, with recent development in 4D PC MRI technique, the methodologies to assess relative pressure distribution in the heart chamber have been proposed. The pressure drop in the heart conduit, pulmonary artery and aorta, can also be obtained using the Bernoulli equation or Pressure Poisson equation. The pressure information obtained from above methodologies was validated with the pressure derived from Doppler ultrasound measurement or obtained from catheterization and showed a good agreement with them. We believe that it would be possible to assess RV pressure by applying those methodologies with 4D PC MRI data.

From an energy point of view, \( e_{MPA} \) accounts for pressure-flow and kinetic energy of the blood at MPA along with stroke work performed by the RV. Therefore, \( e_{MPA} \) may be useful to quantify the state of the RV-PA hemodynamics in terms of energy transition between the RV and PA. Specifically, as seen from Table 3 and Figure 1, \( e_{MPA} \) of the patient group \( (0.56 \pm 0.33) \) was significantly lower \( (2.8 \text{ times}; P < .04) \) than that of the control group \( (1.56 \pm 0.85) \), despite the fact that RV SW of the patient group

Figure 6. Correlations between (A) end-systolic pressure (ESP) and end-diastolic volume index (EDVI), (B) EDP and EDVI. (A) No correlation was observed between ESP and EDVI in the patient group, whereas the control group had a weak negative correlation between ESP and EDVI \( (r = -0.46; P < .4) \). (B) A significant negative correlation was observed between EDP and EDVI of the control group \( (r = -0.81; P < .05) \), whereas an insignificant correlation was shown between EDP and EDVI of the patient group. EDP, end-diastolic pressure.

Congenit Heart Dis. 2013;8:328–342
Energy Transfer Ratio in RV Efficiency

(0.205 ± 0.095 J/m²) was significantly higher (2.3 times; P < .02) as compared with that of the control group (0.090 ± 0.038 J/m²). These results show that the RV, in the presence of PA regurgitation and PA obstruction, is under an overloading condition, for maintaining blood flow through the PA. However, based on our empa values, the RV performance in patients was less efficient compared with that of normal subjects. The lower empa value of the patient group can be attributed to the combined effect of lower Enet_syst, caused by both PA regurgitation and PA obstruction, and higher RV SW, caused by relatively higher RV pressure and volume overloading.

The RV SW that we computed does not include all the energy components in the RV. It only includes boundary (or pressure) work in the RV. The elastic energy stored in RV tissues, kinetic and potential energy, internal energy, and viscous dissipation are not included in the energy components of the RV. Moreover, exit loss at the pulmonary valve and entry loss at the tricuspid valve are also not accounted for in determining the total energy of the RV. Consequently, the denominator of empa, RV SW, is lower than the energy that the RV possibly generates, resulting in empa ratio (= Enet/RV SW) greater than 1 (Table 3).

As an example, we have estimated the elastic energy stored in the RV of a representative control subject. The pressure-strain elastic modulus of the RV was computed using RV pressure, measured by catheterization, and RV hydraulic diameter, obtained from CMR imaging (Eq. 7; 40):

\[ E_p = \frac{(P_{systolic} - P_{diastolic}) \times D_{systolic} - D_{diastolic}}{D_{systolic} - D_{diastolic}} \]  

(7)

where \( p \) is the RV pressure and \( D \) is the hydraulic diameter of the RV. The first order approximation of elastic energy stored in the RV (Eq. 8) can be obtained from Eq. 8:

\[ \text{Elastic energy} [J] = -\frac{1}{2} \times E_p \times \Delta T^2 \]  

(8)

where \( \Delta T \) is the change in average RV wall thickness between the diastolic and systolic phases measured by CMR imaging. The elastic energy in the RV was calculated to be 0.027 J, which is 14.8% of RV SW.

Similarly, the elastic energy stored in the MPA was computed to be 0.0025 J, assuming the MPA to be a simplified cylindrical artery with 4 cm of length. This was found to be only 1% of Enet (the energy transferred to the MPA). As a result, with our elastic energy estimation in the RV and MPA, the empa can be modified to a ratio of the sum of Enet and elastic energy stored in the MPA to the sum of RV SW and elastic energy stored in the RV. Thus, the modified empa decreased from 1.32 to 1.16 for the control subject. The said estimation of elastic energy indicates that the elastic energy component in the RV is significantly higher (one order of magnitude) than that in the MPA. We believe that the significant amount of the elastic energy stored in the RV got converted in terms of pressure and kinetic energy in the MPA (Eq. 4).

It is not trivial to calculate all the energy terms that we previously mentioned under the clinical setting. The goal of our study was to use only the parameters measureable under the clinical setting, such that clinicians can assess the RV-MPA hemodynamic using empa ratio, without needs for any additional measurement. We believe that empa would become less than 1 if we are able to incorporate all the energy terms. It may be noted that the said calculation was possible for control subjects because the normal RV had well-defined geometric information for both the RV and MPA. However, in the case of patients, the elastic energy calculation may not be easy. The changes in diameter and wall thickness in the RV and MPA of the patients are often difficult to measure accurately from CMR imaging, due to geometric abnormalities in the RV and MPA.

Despite the limitation in our estimation of empa, the presented empa between the patient and control groups in the manuscript was significantly different and correlated well with current RV indices, RV EDV, and RV ESP.

In this study, spatially averaged velocity values from PC MRI measurement were used to calculate the total energy transferred at the MPA (Enet). We found that the kinetic energy contribution to Enet associated with the blood flow velocity at the MPA was insignificant compared with the contribution from the pressure-flow energy. For example, the mean kinetic energy for the patient group was 0.014 J compared with the mean pressure-flow energy of 0.112 J; whereas for the control group the mean kinetic energy was 0.023 J compared with the mean pressure-flow energy of 0.167 J. Therefore, the contribution of the kinetic energy was one order of magnitude lower than that of the pressure-flow energy. This significant difference between the kinetic energy to pressure-flow energy is attributed to the pressure change that occurs between the RV and MPA (or PA conduit)
due to appreciable change in area. Therefore, the contribution of the kinetic energy toward the total energy at the MPA is insignificant.

Calculation of energy-based indices requires simultaneous measurements of pressure and volume or flow rate. However, this is a limitation because simultaneous measurement of MRI and catheterization is not possible under clinical settings. Our approach to synchronize pressure and volume or flow measurements by electrocardiogram gating as reported by Das et al.\(^2\) is thus far the best alternative.

We do not expect our results to be significantly affected by variation in heart rate during CMR and catheterization. This is because most subjects (10 out of 14) had a gap of at most 1 month between CMR and catheterization. Only one subject had a gap of 7 months, whereas the remaining three subjects had a gap of no more than 3 months. As shown in Table 1, the mean heart rate change between CMR and catheterization was 2.4% for the control group and 5.2% for the patient group. Therefore, the RV-PA physiologic conditions for both the groups were comparable during CMR and catheterization measurements. Further, we do not observe any significant RV remodelling during such a short time span.

### Conclusions

We have calculated energy-based end points characterizing the efficiency of energy transfer between the RV and MPA, \(e_{\text{MPA}}\), using measured RV volume and pressure, as well as MPA pressure and flow rate for human subjects. Our data showed that the RV of patients with repaired CHD, such as TOF, TGA, and Ross procedure, was volume and pressure overloaded and the RV was less efficient due to a combination of pulmonary regurgitation and obstruction. Thus, we think that \(e_{\text{MPA}}\) can provide comprehensive hemodynamic assessment in terms of RV performance and its efficiency since \(e_{\text{MPA}}\) accounts for the RV pressure and volume, as well as the MPA pressure and flow rate. Furthermore, it correlates well with RV pressure and volume data for the patients in this study. Based on that, we conclude that \(e_{\text{MPA}}\) may be useful to assess the progression of the RV myocardial dysfunction of patients with CHD associated the RV in a long-term clinical follow-up.

### Appendix

#### The Demographic of the Patient Subgroups 1 and 2

The details of demographics and clinical data of subjects in the patient subgroup 1 and 2 are given in Table 4. The subgroup 1 consisted of one male and three females with mean age 17.8 ± 15.0 years, BSA 1.4 ± 0.7 m\(^2\), and heart rate during CMR 78.8 ± 21.7 beats/min. The subgroup 2 was comprised of two males and two females with mean age 8.8 ± 4.6 years, BSA 1.1 ± 0.3 m\(^2\), and heart rate during CMR 86.5 ± 21.9 beats/min.

#### Result for the Patient Subgroup 1 and 2

The result RV volume, volume-based parameters, and pressure for patient subjects in subgroup 1 and 2 are shown in Table 5. Both the subgroups had a significantly higher regurgitation fraction (30.0 ± 4.8% with \(P < .01\) and 34.2 ± 11.5% with \(P < .01\), for the subgroup 1 and 2, respectively) than that for the control group with no regurgitant flow to the RV. The increase of regurgitation fraction was more pronounced in the subgroup 2 than 1. RV mass index was significantly increased in subgroup 2 (32.3 ± 4.4 g/m\(^2\); \(P < .01\)) compared with that of control (19.8 ± 4.6 g/m\(^2\)), whereas the difference in RV mass index between the subgroup 1 and control groups was not significant due to the large variation. RV ESP in both the subgroups was significantly higher (50.6 ± 7.0 mmHg with \(P < .01\) and 56.7 ± 6.6 mmHg with \(P < .01\), for the subgroup 1 and 2, respectively) than the control group (24.6 ± 5.5 mmHg). Other parameters, RV EDP, EDV\(_{\text{i}}\), ESV\(_{\text{i}}\), ejection fraction, peak ejection/filling rate, stroke volume index, and cardiac

---

**Table 4. The Detail of Demographics and Clinical Data of Subjects in the Patient Subgroups 1 and 2**

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 6)</th>
<th>Group 1 (n = 4)</th>
<th>Group 2 (n = 4)</th>
<th>(P) Value 1 vs. Control 2 vs. Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>13.8 ± 8.5</td>
<td>17.8 ± 15.0</td>
<td>8.8 ± 4.6</td>
<td>.6</td>
</tr>
<tr>
<td>Male/Female</td>
<td>3/3</td>
<td>1/3</td>
<td>2/2</td>
<td>&lt;.4</td>
</tr>
<tr>
<td>BSA (m(^2))</td>
<td>1.5 ± 0.6</td>
<td>1.4 ± 0.7</td>
<td>1.1 ± 0.3</td>
<td>&lt;.9</td>
</tr>
<tr>
<td>Heart rate, CMR (bpm)</td>
<td>75.3 ± 12.8</td>
<td>78.8 ± 21.7</td>
<td>86.5 ± 21.9</td>
<td>&lt;.9</td>
</tr>
</tbody>
</table>

Values are means and standard deviations.
BSA, body surface area; CMR, cardiac magnetic resonance imaging.

---

\(^2\) Das et al.
is the blood density in kg/m$^3$.

The rate of energy transferred at the MPA can be expressed in integral form using fundamental fluid mechanics principles:

$$E_m = \int\int_A \left( p_m + \frac{1}{2} \rho \bar{v}_m \cdot \bar{v}_m \right) \bar{v}_m \cdot ndA_m \quad (9)$$

where $p_m$ is the time-varying MPA pressure measured during catheterization, $A$ is the cross-sectional area of the MPA plane, $\bar{v}_m(x,t)$ is the blood velocity at any point $x$ on the MPA plane and at any instant of time $t$, $n$ is the unit normal vector to the MPA plane, and $\rho$ is the blood density (=1035 kg/m$^3$). Equation 9 can be reduced to Eq. (4) by the MPA volumetric flow rate defined as

$$Q_m = \int_A \bar{v}_m \cdot ndA_m \quad (4)$$

output for both the subgroups were not different from those of the control group.

The control group had a higher $E_{\text{net}}$ (0.19 ± 0.11 J) compared with both the patient subgroups (0.15 ± 0.06 J and 0.10 ± 0.08 J for groups 1 and 2, respectively) as shown in Table 6. Whereas, the control group had a lower RV SW (0.12 ± 0.04 J) than both the patient subgroups (0.21 ± 0.12 J and 0.28 ± 0.13 J for the subgroups 1 and 2, respectively). The difference in $E_{\text{net}}$ and RV SW between the patient subgroups and control group were not statistically significant except RV SW between the patient subgroup 2 and the control group ($P < .02$). RV SW for both the patient subgroups was significantly higher (0.147 ± 0.035 J/m² with $P = .05$ and 0.262 ± 0.104 J/m² with $P < .01$, for the subgroup 1 and 2, respectively) than that of the control (0.090 ± 0.038 J/m²). However, only the subgroup 2 had a significantly lower $e_{\text{MPA}}$ (0.35 ± 0.24; $P < .02$) compared with that of the control group (1.56 ± 0.85).

### Integral form of the Equation for the Rate of Energy Transferred at the MPA ($E_m$)

The control group had a higher $E_{\text{net}}$ (0.19 ± 0.11 J) compared with both the patient subgroups (0.15 ± 0.06 J and 0.10 ± 0.08 J for groups 1 and 2, respectively) as shown in Table 6. Whereas, the control group had a lower RV SW (0.12 ± 0.04 J) than both the patient subgroups (0.21 ± 0.12 J and 0.28 ± 0.13 J for the subgroups 1 and 2, respectively). The difference in $E_{\text{net}}$ and RV SW between the patient subgroups and control group were not statistically significant except RV SW between the patient subgroup 2 and the control group ($P < .02$). RV SW for both the patient subgroups was significantly higher (0.147 ± 0.035 J/m² with $P = .05$ and 0.262 ± 0.104 J/m² with $P < .01$, for the subgroup 1 and 2, respectively) than that of the control (0.090 ± 0.038 J/m²). However, only the subgroup 2 had a significantly lower $e_{\text{MPA}}$ (0.35 ± 0.24; $P < .02$) compared with that of the control group (1.56 ± 0.85).

### Integral form of the Equation for the Rate of Energy Transferred at the MPA ($E_m$)

The rate of energy transferred at the MPA can be expressed in integral form using fundamental fluid mechanics principles:

$$E_m = \int\int_A \left( p_m + \frac{1}{2} \rho \bar{v}_m \cdot \bar{v}_m \right) \bar{v}_m \cdot ndA_m \quad (9)$$

where $p_m$ is the time-varying MPA pressure measured during catheterization, $A$ is the cross-sectional area of the MPA plane, $\bar{v}_m(x,t)$ is the blood velocity at any point $x$ on the MPA plane and at any instant of time $t$, $n$ is the unit normal vector to the MPA plane, and $\rho$ is the blood density (=1035 kg/m³). Equation 9 can be reduced to Eq. (4) by the MPA volumetric flow rate defined as

$$Q_m = \int_A \bar{v}_m \cdot ndA_m \quad (4)$$

### Table 5. RV Volume, Volume-based Parameters, and Pressure for Subjects in Patient Subgroups 1 and 2

<table>
<thead>
<tr>
<th>RV Pressure and Volume Characteristics</th>
<th>Control</th>
<th>Group 1</th>
<th>Group 2</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV mass index (g/m²)</td>
<td>19.8 ± 6</td>
<td>24.6 ± 5.5</td>
<td>56.7 ± 6.6</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>RV SWI (J/m²)</td>
<td>0.038 ± 0.147</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Stroke volume index (ml/m²)</td>
<td>46.3 ± 13.9</td>
<td>55.1 ± 5.5</td>
<td>54.7 ± 13.5</td>
<td>&lt;.3</td>
</tr>
<tr>
<td>RV SWI (J/m²)</td>
<td>0.038 ± 0.147</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Stroke volume index (ml/m²)</td>
<td>46.3 ± 13.9</td>
<td>55.1 ± 5.5</td>
<td>54.7 ± 13.5</td>
<td>&lt;.3</td>
</tr>
<tr>
<td>RV SWI (J/m²)</td>
<td>0.038 ± 0.147</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Stroke volume index (ml/m²)</td>
<td>46.3 ± 13.9</td>
<td>55.1 ± 5.5</td>
<td>54.7 ± 13.5</td>
<td>&lt;.3</td>
</tr>
<tr>
<td>RV SWI (J/m²)</td>
<td>0.038 ± 0.147</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>Stroke volume index (ml/m²)</td>
<td>46.3 ± 13.9</td>
<td>55.1 ± 5.5</td>
<td>54.7 ± 13.5</td>
<td>&lt;.3</td>
</tr>
</tbody>
</table>

### Table 6. Total Energy at the MPA ($E_{\text{net}}$), RV SW, BSA indexed RV SW (RV SWI), and Energy Transfer ratio ($e_{\text{MPA}}$) for Subjects in Patient Subgroups 1 and 2

<table>
<thead>
<tr>
<th>Computed RV End Points</th>
<th>Control</th>
<th>Group 1</th>
<th>Group 2</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$E_{\text{net}}$ (J)</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>0.10 ± 0.08</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>RV SW (J)</td>
<td>0.12 ± 0.04</td>
<td>0.21 ± 0.12</td>
<td>0.28 ± 0.13</td>
<td>&lt;.2</td>
</tr>
<tr>
<td>RV SW index (J/m²)</td>
<td>0.090 ± 0.038</td>
<td>0.147 ± 0.035</td>
<td>0.262 ± 0.104</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Energy transfer ratio, $e_{\text{MPA}}$</td>
<td>1.56 ± 0.85</td>
<td>0.78 ± 0.27</td>
<td>0.35 ± 0.24</td>
<td>&lt;.12</td>
</tr>
<tr>
<td>$E_{\text{net}}$ (J)</td>
<td>0.19 ± 0.11</td>
<td>0.15 ± 0.06</td>
<td>0.10 ± 0.08</td>
<td>&lt;.5</td>
</tr>
<tr>
<td>RV SW (J)</td>
<td>0.12 ± 0.04</td>
<td>0.21 ± 0.12</td>
<td>0.28 ± 0.13</td>
<td>&lt;.2</td>
</tr>
<tr>
<td>RV SW index (J/m²)</td>
<td>0.090 ± 0.038</td>
<td>0.147 ± 0.035</td>
<td>0.262 ± 0.104</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Energy transfer ratio, $e_{\text{MPA}}$</td>
<td>1.56 ± 0.85</td>
<td>0.78 ± 0.27</td>
<td>0.35 ± 0.24</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

Values are means and standard deviations. The parameters in italic have a statistically significant difference ($P < .05$) between either the group 1 and control or the group 2 and control.

BSA, body surface area; MPA, main pulmonary artery; RV, right ventricle; SW, stroke work.
Therefore, Eq. (9) is simplified to Eq. (4) by using the spatial average of MPA velocity, $v_m (= Q_m/A)$, approximated with $\bar{v}_m(x,t)$ at the MPA plane.

Pressure–Volume Loop and $E_m$

A representative RV pressure–volume loop for a rTOF and control subject is shown in Figure 7A. It was observed that the RV of a patient was under increased RV pressure as well as elevated RV volume compared with that of a subject in the control group. Figure 7B shows a representative plot of the rate of total energy transferred at the MPA ($E_m$) vs. time for a rTOF and control subject. For each subject, this curve was used to calculate the total energy at MPA ($E_{net}$) by integrating over the cardiac cycle (see Eq. 5). The large negative energy was observed during the diastole period for the patient. This is attributed to the regurgitant flow from the MPA to the RV which led to net reduction in $E_{net}$ value. In contrast, no negative energy was observed for the subject from the control group, causing increased net $E_{net}$ value.

Author Contributions

Namheon Lee, MS: Contributions include study design, data analysis and interpretation, and drafting of the manuscript.

Ashish Das, MS: Contributions include data analysis and review of the manuscript.

Kan N. Hor, MD: Contributions include CMR and catheterization data acquisition, participated in study design, data interpretation, and review of the manuscript.

Michael D. Taylor, MD: Contributions include CMR and catheterization data acquisition, participated in study design, data interpretation, and review of the manuscript.

Rupak K. Banerjee, PhD: Contributions include study design, data interpretation, and review and approval of the final manuscript.

Conflict of interest: None.

Accepted in final form: November 27, 2012.

References