

# Estimation of Pulmonary/Systemic Resistance Ratios From Echocardiographic Systolic Time Intervals in Young Patients With Congenital or Acquired Heart Disease

ERIC W. SPOONER, MD  
BURTON L. PERRY, MD  
AARON M. STERN, MD  
JOAN M. SIGMANN, MD

*Ann Arbor, Michigan*

Previous work has shown the positive correlation of echocardiographic right ventricular preejection period/right ventricular ejection time ratio (RPEP/RVET) with pulmonary vascular resistance and pulmonary arterial diastolic pressure obtained at cardiac catheterization. However, the correlation was insufficient to predict pulmonary arterial diastolic pressure or vascular resistance from a given RPEP/RVET ratio. In this study the RPEP/RVET ratio was compared with left ventricular preejection period/ejection time ratio (LVEP/LVET) in 25 patients undergoing cardiac catheterization, and a strong correlation was found between the ratio (RPEP/RVET)/(LVEP/LVET) = R/L and the ratio of pulmonary arteriolar resistance/systemic arteriolar resistance (PAR/RS), especially when R/L was correlated with  $\log_{10}$  PAR/RS ( $r = 0.902$ ). A very high correlation ( $r = 0.960$ ) was found between R/L and  $\log_{10}$  PAR/RS when the group was restricted to patients with a ventricular septal defect or a complete endocardial cushion defect. Regression equations for prediction of PAR/RS have been derived for the various groups.

Since its introduction to clinical use, echocardiography has been evolving from a qualitative descriptive tool of diagnosis to a procedure providing quantitative data. Pediatric and adult cardiologists alike are asking for quantitative measurements and estimates of various hemodynamic variables. Therefore, after the qualitative descriptions of pulmonary valve motion in pulmonary hypertension,<sup>1,2</sup> great interest was created by the quantitative correlation of right ventricular systolic time intervals with pulmonary arterial diastolic pressure and pulmonary vascular resistance reported by Hirschfeld et al.<sup>3</sup> However, although statistically significant, the correlation coefficients between right ventricular systolic time intervals and pulmonary arterial diastolic pressure or vascular resistance were inadequate to predict these values accurately.<sup>4</sup> Clearly, further investigation was needed to provide the data required for more accurate prediction. In this study we assessed whether one could more accurately predict the pulmonary vascular resistance/systemic vascular resistance ratio by comparing the patient's left and right ventricular systolic time intervals because the normal range of pulmonary/systemic vascular resistance ratio is well known.

## Case Material

Thirty patients undergoing diagnostic cardiac catheterization at the University of Michigan Medical Center for delineation of congenital or acquired heart disease underwent echocardiography within 12 hours of the catheterization; in most cases the procedure was performed in the catheterization laboratory immediately before the hemodynamic study. Patients were not studied in the first 6 hours after catheterization. Patients with a complete right or left bundle branch block pattern in the electrocardiogram, complete transposition of the great ar-

From the Department of Pediatrics, Section of Pediatric Cardiology, University of Michigan Medical Center, Ann Arbor, Michigan. Manuscript received December 12, 1977; revised manuscript received June 6, 1978, accepted June 7, 1978.

Address for reprints: Eric W. Spooner, MD, Department of Pediatric Cardiology, Albany Medical Center Hospital, 47 New Scotland Avenue, Albany, New York 12208.

TABLE I

Summary of Echocardiographic and Cardiac Catheterization Data in 25 Cases

Case no.	Diagnosis	RPEP RVET	LPEP LVET	R/L	PAR	TPR	RS	PAR RS	TPR RS	Log <sub>10</sub> PAR RS	Log <sub>10</sub> TPR RS
1	VSD	0.187	0.301	0.621	1.27	2.89	12.55	0.101	0.230	-0.996	-0.638
2	VSD	0.637	0.557	1.133	35.7	38.1	14.9	2.35	2.56	0.371	0.408
3	ASD	0.203	0.422	0.481	0.766	1.72	16.67	0.046	0.103	-1.337	-0.987
4	VSD	0.192	0.340	0.565	0.972	2.92	22.06	0.044	0.132	-1.356	-0.879
5	ASD	0.186	0.283	0.657	1.43	1.94	24.14	0.059	0.080	-1.229	-1.095
6	Pulm emboli	0.595	0.691	0.861	10.39	14.34	32.62	0.319	0.440	-0.496	-0.357
7	PS	0.112	0.154	0.727	1.65	3.32	16.82	0.098	0.197	-1.009	-0.706
8	VSD, PDA	0.199	0.236	0.843	4.02	5.85	19.78	0.203	0.296	-0.693	-0.529
9	Normal	0.201	0.297	0.676	1.77	2.74	14.18	0.125	0.193	-0.903	-0.714
10	ECD	0.220	0.254	0.866	5.52	6.18	18.94	0.292	0.326	-0.535	-0.487
11	AS, PS	0.096	0.164	0.588	1.39	5.03	27.42	0.051	0.184	-1.294	-0.736
12	VSD	0.170	0.291	0.584	2.56	3.62	24.34	0.105	0.1486	-0.979	-0.828
13	ECD	0.189	0.233	0.850	8.60	9.21	33.18	0.259	0.278	-0.587	-0.556
14	Inf PS	0.189	0.324	0.582	1.09	2.36	11.81	0.092	0.200	-1.034	-0.699
15	ECD	0.327	0.295	1.108	3.02	3.42	3.25*	0.929	1.053	-0.032	0.022
16	ASD	0.194	0.318	0.612	1.14	1.77	19.94	0.057	0.089	-1.243	-1.052
17	PS	0.212	0.252	0.838	2.47	4.63	23.46	0.105	0.197	-0.979	-0.706
18	PDA	0.159	0.298	0.533	1.29	1.90	13.53	0.095	0.140	-1.021	-0.854
19	VSD	0.142	0.250	0.568	0.91	2.55	26.11	0.035	0.098	-1.456	-1.009
20	AS	0.264	0.350	0.753	2.08	6.24	27.01	0.077	0.231	-1.114	-0.636
21	VSD	0.229	0.271	0.842	7.00	10.67	23.29	0.301	0.458	-0.521	-0.339
22	a-v fist;	0.306	0.343	0.891	2.73	3.23	5.93	0.461	0.544	-0.337	-0.265
23	AS	0.263	0.330	0.798	1.70	4.16	17.20	0.099	0.242	-1.005	-0.616
24	ASD	0.172	0.280	0.615	0.83	1.53	25.12	0.033	0.061	-1.482	-1.215
25	VSD	0.174	0.303	0.575	1.51	2.45	17.34	0.087	0.141	-1.061	-0.851

\* Hemoglobin 4.2 g/100 ml at time of echocardiography and cardiac catheterization.

PAR, TPR, RS are expressed in units of mm Hg/liter per min per m<sup>2</sup>.

AS = aortic stenosis; ASD = atrial septal defect; a-v fist = cerebral arteriovenous fistula; ECD = endocardial cushion defect; Inf PS = infundibular pulmonary stenosis; PAR = pulmonary arteriolar resistance (mm Hg/liter per min per m<sup>2</sup>); PDA = patent ductus arteriosus; PS = pulmonary stenosis; Pulm = pulmonary; RS = systemic resistance (mm Hg/liter per min per m<sup>2</sup>); TPR = total pulmonary resistance (mm Hg/liter per min per m<sup>2</sup>); VSD = ventricular septal defect.

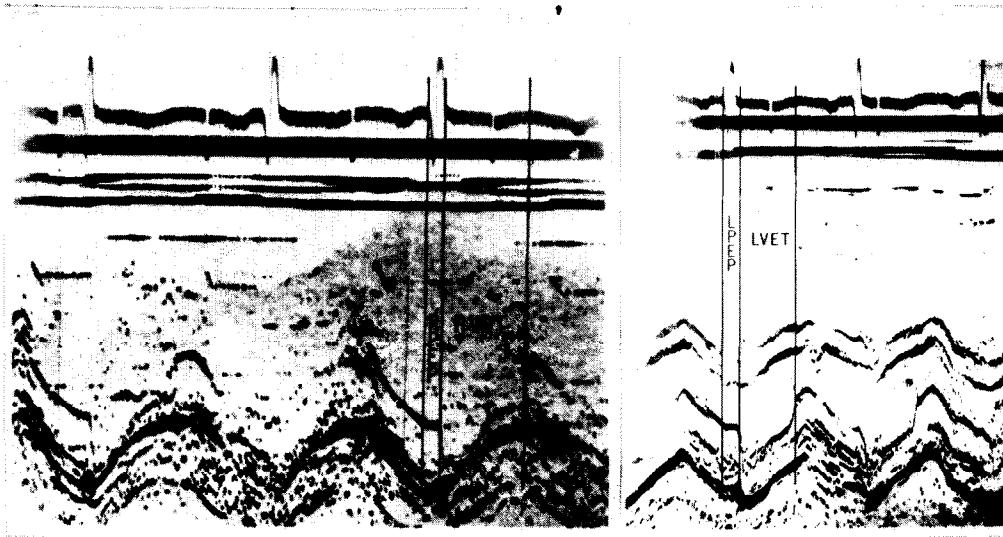
teries or a cardiac pacemaker were specifically excluded from the study, as were patients breathing a greater than 40 percent oxygen concentration (only Patient 15, breathing 35 percent oxygen, was not in room air). Patients with an incomplete right bundle branch block pattern with a QRS interval within normal limits for age<sup>5</sup> were included. Of these 30 patients, 25 had echocardiographic and cardiac catheterization data suitable for inclusion. Their diagnoses are included in Table I. All patients were under 10 years of age except Patients 2 (18 years) and 6 (15 years).

### Methods

**Echocardiograms:** These were obtained with 5.0, 3.5 or 2.25 megahertz nonfocused Aerotech transducers, and either a Picker model 104 ultrasonoscope with Polaroid® film recording or a Smith Kline model 20-A ultrasonoscope and Smith Kline model 21 fiberoptic strip chart recorder. Echocardiograms of the aortic and pulmonary valves were obtained with standard techniques and recorded so that one complete cardiac cycle was obtained per sweep on the Polaroid camera, or at a paper speed of 100 mm/sec on the Smith Kline instrument. Only complexes including both the opening and closing movements of the valve under study were included. The echocardiographic limb lead showing the earliest depolarization, usually lead II, was utilized and superimposed on the echocardiographic tracing. Measurements of preejection period and ejection period were made as previously described<sup>3</sup> (Fig. 1) with the additional technique of utilizing a dial caliper, accurate to ±0.05 mm, measuring the intervals to the nearest 0.1 mm and using a drafting triangle and stylus to make par-

allel rulings at the onset of the Q wave and the opening and closure of the valve in question. Measurements in millimeters were then converted to seconds (carried to three significant digits), using the highly accurate and stable interval time markers generated by each instrument. At least six cardiac cycles for each valve, during all phases of respiration, were measured and the values averaged. All measurements were carried out by one observer (E.S.).

**Cardiac catheterization:** This procedure was carried out with the patient under light morphine and diphenhydramine (Benadryl®) sedation, and samples for oxygen saturation and pressure measurements were obtained simultaneously or in rapid succession (less than 2 minutes) from the pulmonary artery, pulmonary vein, systemic artery and mixed venous return. When pulmonary venous return or pulmonary arterial wedge saturation could not be sampled (five cases) it was assumed to be the same as the systemic arterial or left ventricular saturation, provided there was no right to left shunting as shown with angiography or an arterial saturation level above 93 percent, or both. Samples were analyzed for oxygen saturation with an American Optical Corporation reflectance oximeter or Instrumentation Laboratories Company transmittance oximeter, or both. Values for assumed oxygen consumption were obtained from tables as reported by Cayler et al.<sup>6</sup> for infants, and LaFarge and Miettinen<sup>7</sup> for children over age 3 years. Pressures were obtained with no. 5, 6 and 7F National Institutes of Health or Cournand (USCI, Billerica, Massachusetts) or Gensini (Edwards Laboratories, Santa Ana, California) catheters of 50, 80 and 100 cm lengths coupled to Statham P23Db strain gauge transducers and recorded on an



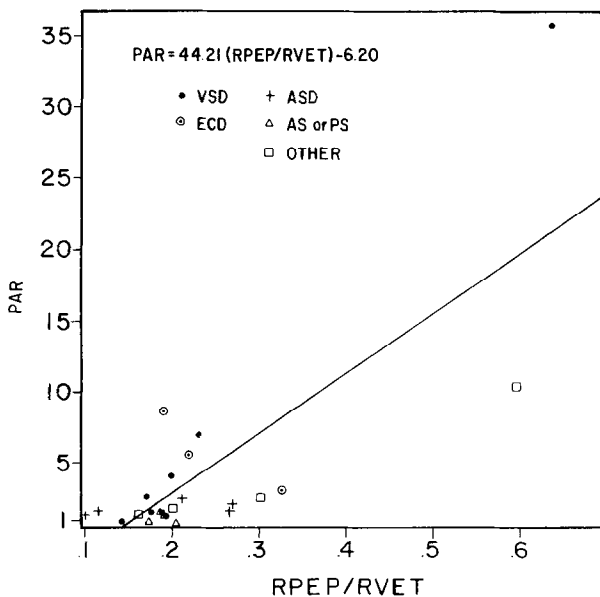
**FIGURE 1.** Patient 16. *Left*, echocardiographic tracing of pulmonary valve, showing method of measurement of right ventricular pre-ejection period (RPEP) and ejection time (RVET). RPEP/RVET = 0.203 in this patient, whose pulmonary arteriolar resistance = 1.14 mm Hg/liter per min per m<sup>2</sup>. *Right*, echocardiographic tracing of aortic valve, showing method of measurement of left ventricular pre-ejection period (LPEP) and ejection time (LVET). LPEP/LVET in this instance = 0.315.

Electronics for Medicine VR6 photo-optical recorder. Catheterization values thus obtained were entered into a Digital Equipment Corporation PDP-12 computer programmed to calculate flows by standard Fick principle and to report pulmonary arteriolar, total pulmonary and systemic arteriolar resistances in units of mm Hg/liter per min per m<sup>2</sup> of body surface area. So as to preclude observer bias this analysis was performed after analysis of the echocardiographic systolic time intervals.

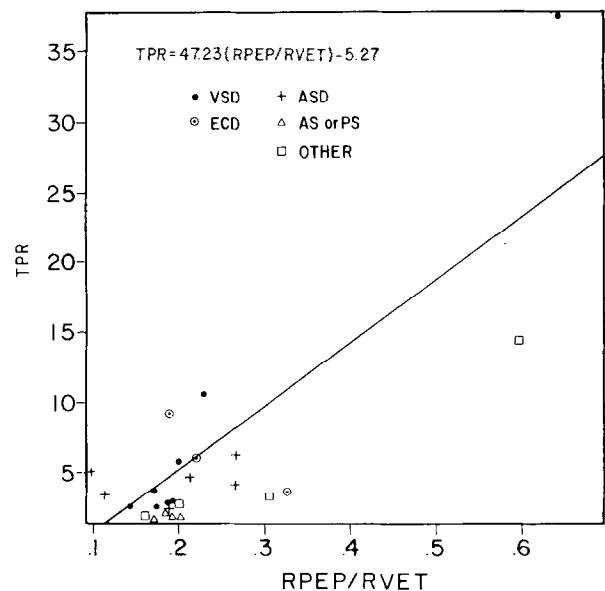
The resultant data points were entered into the University of Michigan Data Terminal System, and correlation matrices and linear regression analysis carried out by the method of least-squares using the MIDAS statistical research package.

**Results**

Table I lists the results of the echocardiographic systolic time intervals and cardiac catheterization data for the 25 patients. As might be expected, there was a



**FIGURE 2.** Right ventricular pre-ejection period/right ventricular ejection time ratio (RPEP/RVET) in 25 patients is plotted against pulmonary arteriolar resistance (PAR) in units of mm Hg/liter per min per m<sup>2</sup> together with the regression line. Although there is a fair degree of scatter, the correlation coefficient (*r*) is 0.787, and the linear regression equation is PAR = 44.21 (RPEP/RVET) - 6.20. AS = aortic stenosis; ASD = atrial septal defect; ECD = endocardial cushion defect; PS = pulmonary stenosis; VSD = ventricular septal defect.

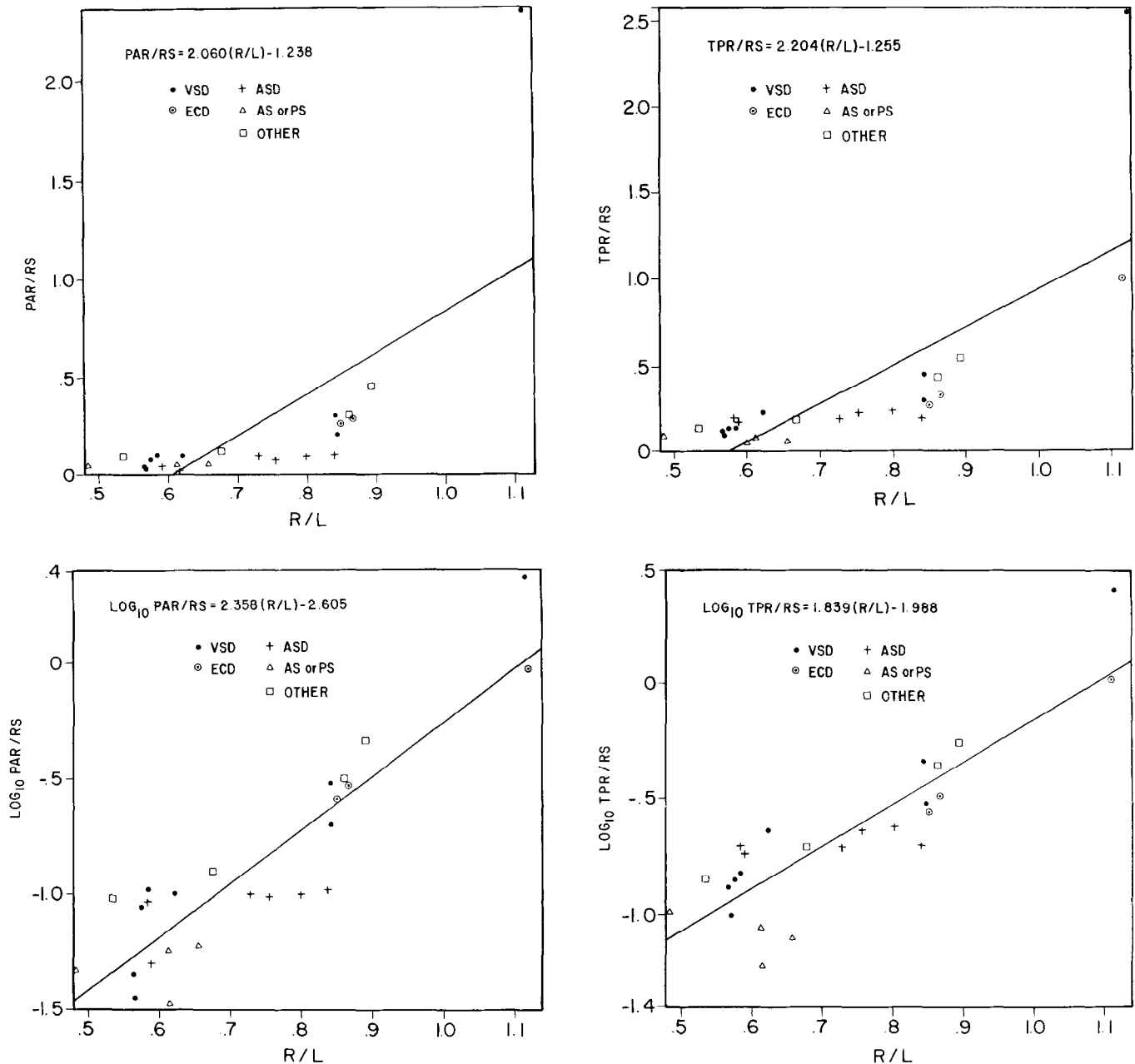


**FIGURE 3.** Right ventricular pre-ejection period/right ventricular ejection time ratio (RPEP/RVET) in 25 patients is plotted against total pulmonary resistance (TPR) in units of mm Hg/liter per min per m<sup>2</sup>, together with the regression line. Although there is a fair degree of scatter again, *r* = 0.800 and TPR = 47.23 (RPEP/RVET) - 5.27 by least-squares regression analysis. Abbreviations as in Figure 2.

preponderance of children with ventricular septal defect, atrial septal defect or endocardial cushion defect.

**Right/left systolic interval ratios compared with pulmonary/systemic resistance ratios:** The ratio of right ventricular preejection period/right ventricular

ejection time (RPEP/RVET) was compared with pulmonary arteriolar resistance and total pulmonary resistance using linear regression analysis (Fig. 2 and 3). There was a significant statistical correlation between these values, with the correlation coefficient of 0.787 ( $r^2 = 0.619$ ) for RPEP/RVET versus pulmonary arteriolar



**FIGURE 4.** R/L (ratio between right ventricular preejection period/right ventricular ejection time [RPEP/RVET]/[LPEP/LVET]) plotted against four variables. **Upper left,** R/L versus PAR/RS (pulmonary arteriolar/systemic resistance). The linear regression equation for this relation is  $PAR/RS = 2.060(R/L) - 1.238$ ,  $r = 0.743$ . There appears to be a significant curvilinear relation between the variables. **Upper right,** R/L is plotted against total pulmonary/systemic resistance (TPR/RS), with a resultant linear regression equation of  $TPR/RS = 2.204(R/L) - 1.245$ ,  $r = 0.749$ . There also appears to be a significant curvilinear relation between these variables. **Lower left,** R/L is plotted against  $log_{10}$  PAR/RS (pulmonary arteriolar/systemic resistance) with resultant improvement in the correlation coefficient to 0.902. The relation between the variables now appears more linear, and the linear regression equation is  $log_{10} PAR/RS = 2.358(R/L) - 2.605$ . **Lower right,** R/L is plotted against  $log_{10}$  TPR/RS (total pulmonary/systemic resistance) with a result that is also more linear. The regression equation is  $log_{10} TPR/RS = 1.839(R/L) - 1.988$ ,  $r = 0.889$ .

resistance comparing favorably with that obtained by Hirschfeld et al.<sup>3</sup> ( $r = 0.69$  for RPEP/RVET versus pulmonary vascular resistance for their data). Also, there was significant deviation from a straight line in the points with higher pulmonary arteriolar resistance and total pulmonary resistance, with the curve resembling a logarithmic function, as did the data points of Hirschfeld et al.<sup>3</sup> The regression equation is pulmonary arteriolar resistance =  $44.21$  (RPEP/RVET) -  $6.20$ . There was no significant correlation ( $r = 0.042$ ,  $r^2 = 0.0017$ ) between the ratio of left ventricular preejection period/left ventricular ejection time (LPEP/LVET) and systemic arteriolar resistance. When right ventricular systolic time intervals were compared with left ventricular systolic time intervals by the ratio of (RPEP/RVET)/(LPEP/LVET) = (R/L) and this ratio was compared with indexes of pulmonary/systemic resistance ratio by linear regression analysis, the correlation coefficients were also significant. Figure 4 (upper left) is the plot of right/left systolic time intervals (R/L) versus pulmonary/systemic resistance ratio (PAR/RS) with resultant correlation coefficient of  $0.743$  ( $r^2 = 0.552$ ). At upper right is a plot of right/left systolic time interval ratio (R/L) versus total pulmonary/systemic resistance ratio (TPR/RS) with a correlation coefficient of  $0.749$  ( $r^2 = 0.561$ ). Still it can be seen that there is a curvilinear shape to the data curve, resembling a logarithmic function.

For this reason, when R/L is compared with  $\log_{10}$  PAR/RS (Fig. 4, lower left), a marked improvement in correlation coefficient to  $0.902$  ( $r^2 = 0.814$ ) is noted, because the mathematical model takes into account the deviation of the data points from a straight line. The regression equation for this relation is:

$$\log_{10} \text{PAR/RS} = 2.358 (\text{R/L}) - 2.605$$

$$\text{PAR/RS} = \text{antilog}_{10} [2.358 (\text{R/L}) - 2.605]$$

and is significant at the  $P < 0.0001$  level. The antilog may then be found in a table of logarithms. When R/L is compared with  $\log_{10}$  TPR/RS (Fig. 4 lower right), the correlation coefficient is  $0.889$  ( $r^2 = 0.790$ ). The regression equation is:

$$\log_{10} \text{TRP/RS} = 1.839 (\text{R/L}) - 1.988$$

$$\text{TPR/RS} = \text{antilog}_{10} [1.839 (\text{R/L}) - 1.988]$$

and is also significant at the  $P < 0.0001$  level. As can be seen from the scatter plot, the data points now conform much more closely to a straight line.

**Effect of excluding patients with aortic or pulmonary stenosis:** Because severe aortic stenosis changes LPEP/LVET, and severe pulmonary stenosis changes RPEP/RVET, both by prolonging ejection time, it might be expected that excluding patients with a pulmonary or aortic valve gradient greater than 30 mm Hg might further improve the correlation coefficient because it would result in a more homogeneous group. When patients with aortic or pulmonary stenosis (gradient greater than 30 mm Hg) are excluded, regression analysis provides an improvement in correlation coefficient to  $0.939$  ( $r^2 = 0.882$ ) with the equation

$$\log_{10} \text{PAR/RS} = 2.468 (\text{R/L}) - 2.639.$$

When  $\log_{10}$  TPR/RS is compared, a correlation coefficient of  $0.909$  ( $r^2 = 0.826$ ) results from the equation

$$\log_{10} \text{TRS/RS} = 1.958 (\text{R/L}) - 2.071.$$

Nonetheless, even with inclusion of the patients with aortic stenosis and pulmonary stenosis, all patients with an R/L of less than 0.84 had a PAR/RS of less than 0.20, which would be considered normal.

**Group of patients with ventricular septal defect or complete endocardial cushion defect:** The highest correlation coefficient of  $0.960$  ( $r^2 = 0.923$ ), as expected, results from the most homogeneous group, consisting of those 11 patients with a ventricular septal defect or a complete endocardial cushion defect, all of whom had a high pressure left to right shunt. Coincidentally, this may be the group pediatric cardiologists would be most interested in, in relation to the development of increasing pulmonary vascular resistance. For this group, the regression equation is:

$$\log_{10} \text{PAR/RS} = 2.465 (\text{R/L}) - 2.630.$$

Similarly,

$$\log_{10} \text{TPR/RS} = 1.880 (\text{R/L}) - 1.979$$

for which the correlation coefficient is  $0.945$  ( $r^2 = 0.894$ ). From the patient data it is apparent that all patients with a ventricular septal defect or a complete endocardial cushion defect having an R/L of less than 0.85 had a PAR/RS of less than 0.30, which some consider the upper limit for safe operability.

*Example:* For Patient 21, with an R/L of 0.842, using the previous equation:

$$\log_{10} \text{PAR/RS} = 2.465 (0.842) - 2.630 = -0.525$$

$$\frac{1}{\log_{10} \text{PAR/RS}} = +0.525$$

$$\frac{1}{\text{PAR/RS}} = 3.350 \text{ (antilog found in table)}$$

$$\text{PAR/RS} = 1/3.35 = 0.299,$$

where, in fact, the measured PAR/RS = 0.301.

## Discussion

A systolic time interval is a phase of electromechanical systole whose duration is governed by four basic factors: (1) preload, indicated by end-diastolic pressure; (2) afterload, reflected by vascular resistance or arterial diastolic pressure; (3) contractile state of the myocardium; and (4) the rate and sequence of intraventricular electrical conduction.<sup>8,9</sup>

The clinical usefulness of left ventricular systolic time intervals as a bedside method of clinical evaluation has been documented by several investigators.<sup>10-12</sup> Extremely close correlation has been found between conventionally obtained systolic time intervals and those obtained from echocardiograms.<sup>13</sup> The correlation of right ventricular projection period/right ventricular ejection time ratio (RPEP/RVET) with pulmonary ventricular resistance and pulmonary arterial diastolic pressure (afterload) was documented by Hirschfeld et

al.<sup>3</sup> but, as Silverman and Hoffman<sup>4</sup> noted, the correlation was not strong enough to predict pulmonary resistance accurately, even with their modifications, at least without having determined right ventricular systolic time intervals and pulmonary resistance at a previous cardiac catheterization. Comparing their methods with those under discussion, several important observations may be made.

**Accuracy and limitations of echocardiographic systolic time interval measurements:** In determining the preejection period/ejection period ratio, measurements of intervals must be very exact because a small error in timing the opening of a valve will be added to the numerator of a ratio and subtracted from the denominator. Therefore, small errors, by their inclusion in a ratio, become large. For this reason, only complexes with well seen opening and closure movements were included, and they were measured at high paper speed with dial calipers, using scribed parallel rulings made with a drafting triangle. Even so, the choice of the point of onset of the QRS complex and of valve opening and closure is somewhat subjective and therefore prone to error. However, if the same criteria are always used for both the aortic and pulmonary valves, the identical error will be included in both the denominator and numerator of the right/left systolic time interval ratio (R/L), and the two errors will cancel out.

It has been shown that there is significant variation in the onset of the QRS complex in various leads, with the earliest depolarization generally noted in lead V<sub>2</sub>.<sup>14</sup> Even if this lead were used (and it was not used in our study or that of Hirschfeld et al.<sup>3</sup>), interpatient variation occurs, and therefore errors are introduced into the determination of the preejection period. Again, with the ratio R/L, the same error is introduced into both the numerator and the denominator, and it is canceled out.

Left ventricular systolic time intervals show little respiratory variation, but right ventricular systolic time intervals show rather large variations with differing phases of respiration.<sup>15</sup> Yet in the study by Hirschfeld et al.,<sup>3</sup> measurements were made only during expiration in older children, and in their infants with high respiratory rates only the shortest systolic time intervals were selected. All phases of respiration were included in our study, because we believed that error would result from exclusion of respiratory variation.

Importantly, in any estimation of pulmonary vascular resistance, very large errors may be made if actual oxygen consumption is not measured for use in the Fick principle flow determinations. Instead, if one uses oxygen consumption values obtained from tables, as Hirschfeld et al. and our laboratory do, an error of up to 40 percent may be made. However, the determination of pulmonary to systemic resistance ratio requires no assumed oxygen consumption values, yet normal limits are well known and frequently used by clinicians.

**Effect of left or right ventricular ejection fraction on systolic time interval ratios:** In our data and those of previous investigators,<sup>12</sup> there was insignificant

correlation between LPEP/LVET and systemic vascular resistance, but others<sup>11</sup> have shown a very strong correlation between this ratio and left ventricular ejection fraction. The effect of a decreasing right ventricular ejection fraction on right ventricular systolic time intervals is not known, but one would presume it also to be significant. As an index of left ventricular ejection fraction, the use of LPEP/LVET in the denominator of the ratio R/L would presumably correct for the effect of decreasing right ventricular ejection fraction on right ventricular systolic time intervals. In contrast to adults, most infants and children with congestive heart failure have failure of both ventricles, although in those with left to right shunt, there is usually high output failure and little diminution of ejection fraction.

For these reasons, while the plot of RPEP/RVET versus pulmonary arteriolar resistance shows increasing variance of the data points with increasing resistance, the plot of R/L versus PAR/RS and TPR/RS (pulmonary arteriolar resistance/systemic resistance and total pulmonary resistance/systemic resistance) shows little increase in variance with increasing PAR/RS or TPR/RS, as multiple sources of introduced error are eliminated or canceled out.

**Significance of results in patients with ventricular septal defect and complete endocardial cushion defect:** Our data have shown a highly significant correlation for log<sub>10</sub> PAR/RS versus R/L and log<sub>10</sub> TPR/RS versus R/L. Because stenosis of aortic or pulmonary valves has been shown to prolong ejection time,<sup>12</sup> the effect of excluding patients with a greater than 30 mm Hg peak systolic pulmonary or aortic gradient was investigated. This changed the slope and intercept of the regression line very little but increased the correlation coefficient significantly. Restricting the group to only those with a ventricular septal defect or a complete endocardial cushion defect resulted in the highest correlation coefficient of 0.96, which should have significant predictive value in following up these patients, who are at high risk for the development of pulmonary vascular obstructive disease. The patient data also indicate that all patients with a ventricular septal defect or an endocardial cushion defect having an R/L less than 0.85, have PAR/RS less than 0.30. An R/L greater than 1.0 would suggest a greatly increased PAR/RS. A much larger patient population would improve the confidence limits and will be reported on when patient data have been acquired and analyzed.

Patients with *d*-transposition of the great arteries were specifically excluded from our study at the outset because right and left ventricular activation times are not the same; therefore in these patients the RPEP/RVET ratio cannot be used interchangeably with the LPEP/LVET ratio in normal subjects. Also, in *d*-transposition of the great arteries before the Mustard procedure, estimation of pulmonary blood flow is often highly inaccurate because of contributions by bronchial collateral vessels and inaccuracies of most oximeters in the 95 to 100 percent saturation range. Investigation of the systolic time intervals of such pa-

tients is currently being undertaken and will be reported separately.

**Clinical implications:** Previously, serial cardiac catheterization was the only method for serial evaluation of the pulmonary vascular bed in patients at risk for development of pulmonary vascular obstructive disease. Although determination of echocardiographic right systolic time interval ratios at the time of catheterization may allow subsequent estimation of pulmonary ventricular resistance and pulmonary arterial diastolic pressure at later dates, no method previously described allowed accurate estimation of pulmonary

ventricular resistance or pulmonary arteriolar resistance/systemic arteriolar resistance ratio before catheterization. The use of the regression equations described should allow at least a rough estimation of pulmonary arteriolar resistance/systemic arteriolar resistance, and more accuracy can be expected if patients with aortic stenosis or pulmonary stenosis can be excluded, or if the patient has a ventricular septal defect or a complete endocardial cushion defect. This should prove useful to clinicians in the timing, or avoidance altogether, of cardiac catheterization with its attendant risk and expense.

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