

Right and Left Ventricular Volume Characteristics in Children with Pulmonary Stenosis and Intact Ventricular Septum

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SUMMARY Right (RV) and left ventricular (LV) volume variables were calculated in 27 patients with pulmonary stenosis (PS) during routine cardiac catheterization. These included 21 patients with isolated PS (group I) and seven studies in six patients (group II) with PS and right-to-left atrial shunt. Right and left ventricular volumes were calculated according to Simpson's rule and the area-length methods respectively.

In group I, right ventricular end-diastolic volume (RVEDV) was not different from normal, RVEF (0.70 ± 0.02) was significantly higher than normal, and right ventricular stroke index (RVSI) ($4.36 \text{ L/min/M}^2 \pm 0.23$) was normal. The RVEDV/LVEDV ratio was significantly less than normal ($P = 0.001$). Multiple regression

analysis indicated that RVEDV (% of normal) decreased with both age and severity of RV outflow obstruction ($r = 0.77$).

In group II, RVEDV and RVSI were both less than normal ($P < 0.001$), while RVEF was normal. LVEDV in the group was slightly higher than normal ($P = 0.026$) while LVEF was less than normal ($P = 0.027$) and resulted in normal LVSI.

The data suggest that RV and LV function in children with isolated PS are normal, and that knowledge of the RV volume variables is not essential for the management of these patients. In contrast, hearts of patients with PS and right-to-left interatrial shunt have evidence that suggest depressed ventricular function, and the quantitation of RV volume may be helpful in the management of these patients.

ALTHOUGH CONGESTIVE HEART FAILURE (CHF) is seen in infants and children with severe pulmonary stenosis,¹⁻³ these patients have low operative risk²⁻⁴ and become asymptomatic after successful correction. In patients with long-standing pulmonic stenosis, congestive heart failure is intractable. McIntosh reported a 36-year-old patient with valvular pulmonic stenosis who had CHF which persisted and led to death even though the obstruction had been relieved surgically.⁵ Hudson reported a 33-year-old patient with pulmonary stenosis who died of congestive heart failure preoperatively.⁶ The postmortem examination of the heart in the latter patients showed myocardial fibrosis. This raises important questions as to the time course of myocardial fibrosis and the age at which irreversible myocardial damage takes place. In patients with PS, RV pressure and pulmonic valve area measurements have been utilized to evaluate the severity of the lesion and the indication for surgery. Additional data describing left and right ventricular function might help in identifying patients with deteriorating myocardial function before it becomes apparent clinically and possibly irreversible, and in identifying small RV size in patients with PS and right-to-left atrial shunt.

Since the development of methods for determining right ventricular (RV) volume,⁷⁻⁹ they have been used to quantitate RV volume variables in children with a variety of congenital heart diseases affecting the right side of the heart such as transposition of the great vessels,^{10, 11} tetralogy of Fallot,^{9, 12, 13} and pulmonary atresia with intact ventricular septum.¹⁴

The purpose of this study was to: 1) quantitate left and right ventricular volume variables in children with isolated PS and in children with PS and right-to-left interatrial shunt, and 2) clarify the effect of RV pressure overload and patient's age on RV dimensions and function in these patients.

Methods

Right and left ventricular volumes were calculated in 27 patients with pulmonary stenosis. None of the patients had detectable left-to-right atrial shunt by oxygen saturation and cineangiocardiology. None of the patients had symptoms of congestive heart failure requiring digitalization. These were divided into two groups: Group I included 21 patients with isolated RV outflow obstruction and RV pressure > 55 mm Hg (valvular PS, $N = 19$; infundibular PS, $N = 1$; peripheral PS, $N = 1$). The right-to-left shunt in this group was less than 20% of systemic flow (insignificant shunt) and the decrease in systemic saturation was less than 5%. Eleven of the 21 patients had patent foramen ovale, but only four of them had decreased arterial saturation (#18, 19, 20 of 93% and #21 of 92%). The arterial saturation in all other patients in this group was 95%. Group 2 consisted of seven studies in six patients with valvular pulmonary stenosis and right-to-left interatrial shunt ranging from 25 to 59%. The arterial saturation in these patients were: #22-a = 80%, #22-b = 66%, #23 = 82%, #24 = 86%, #25 = 90%, #26 = 89%, #27 = 85%.

All data were obtained during diagnostic cardiac catheterization. Children more than one year of age were premedicated with meperidine (1 mg/kg), promethazine (0.5 mg/kg), and chlorpromazine (0.5 mg/kg). Infants less than 3 months of age were not premedicated and those between 3 and 12 months of age were given morphine (0.1 mg/kg) only. Before contrast medium injection, intracardiac pressures were recorded using Miller tip transducer catheter (Miller Instruments, Inc., P.O. Box 18227, Houston, Texas 77023) or a side hole catheter (NIH No. 6 or 7) connected to

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Supported by research grant HL-16329 from the NHLI and the American Heart Association, Greater Los Angeles, Affiliate, and a grant from the Children's Bureau.

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Received October 14, 1975; revision accepted for publication January 5, 1976.

a Statham P23GB transducer. Zero pressure was referenced to mid chest. Intra-cardiac shunt was calculated by the Fick principle in group II.

Left and right ventricular volumes were calculated from biplane cineangiocardigrams filmed in the anterior-posterior (AP) and lateral views at 60 frames/sec after injecting 1.25 ml/kg of body weight of 76% Renografin into the right atrium, RV and/or pulmonary artery. Only sinus beats were utilized in calculating volumes. Right ventricular volumes were calculated using Simpson's rule method and were fit to a regression line as shown previously.⁷ Left ventricular volumes were calculated by the area-length method and by applying a previously described regression equation.^{15, 16}

Right and left ventricular end-diastolic volumes (EDV) in each patient were expressed as a percentage of normal value. Normal values for RVEDV and LVEDV in all patients were derived from normal RV and LV end-diastolic volumes expressed as a function of body surface area in children with normal hearts. These equations are:

$$\text{LVEDV} = 72.5 (\text{BSA})^{1.43} \quad (r = 0.97, N = 87) \text{ fig. 1}$$

(Eq. 1)

$$\text{RVEDV} = 75.1 (\text{BSA})^{1.43} \quad (r = 0.97, N = 42) \text{ fig. 2}$$

(Eq. 2)

The following variables were calculated from EDV and end-systolic volume (ESV) for both RV and LV.

$$\text{Stroke volume (SV)} = \text{EDV} - \text{ESV} \quad (\text{Eq. 3})$$

$$\text{Ejection Fraction (EF)} = \text{SV} / \text{EDV} \quad (\text{Eq. 4})$$

$$\text{Systolic Output (SO)} = \text{SV} \times \text{Heart Rate} \quad (\text{Eq. 5})$$

$$\text{Systolic Index (SI)} = \text{SO} / \text{BSA} \quad (\text{Eq. 6})$$

RVEDV/LVEDV, RVEF/LVEF, and RVSI/LVSI were also calculated and compared with normal values. Pulmonic valve area was calculated from RV systolic index and mean RV outflow gradient according to the Gorlin formula.¹⁷

The data in PS patients were compared with normal values which were obtained in 42 children with "normal heart" in whom both RV and LV parameters were calculated. The age distribution of these patients was as follows: one day to six months = 8 patients, 6 months to one year = 4 patients, and one year to 16 years = 30 patients. In addition, left ventricular end-diastolic volumes (LVEDV) were obtained in 45 "normal" children. The diagnoses in the 87 patients were: normal heart, very mild PS, bicuspid aortic valve with no significant gradient, vascular ring and mediastinal mass. None of the 87 patients had detectable shunt, and the peak RV pressure was less than 45 mm Hg.

Results

Clinical and hemodynamic data for both normal and PS groups are shown in tables 1, 2. Heart rates (HR) are tabulated for RV and were not different from those of LV by more than 10%. Group I patients were not different from normal in terms of age, BSA, HR, hemoglobin concentration or peak LV pressure. Group II patients were younger than the normal group. The heart rates in the two infants (<1 year) and four children were normal for their ages. Hemoglobin concentration in this group (mean = $16.0 \pm 1.1\text{g\%}$) was higher than both normal ($P < 0.004$) and group I ($P = 0.04$). Peak RV pressure in both groups were significantly ($P < 0.001$) higher than normal. In addition,

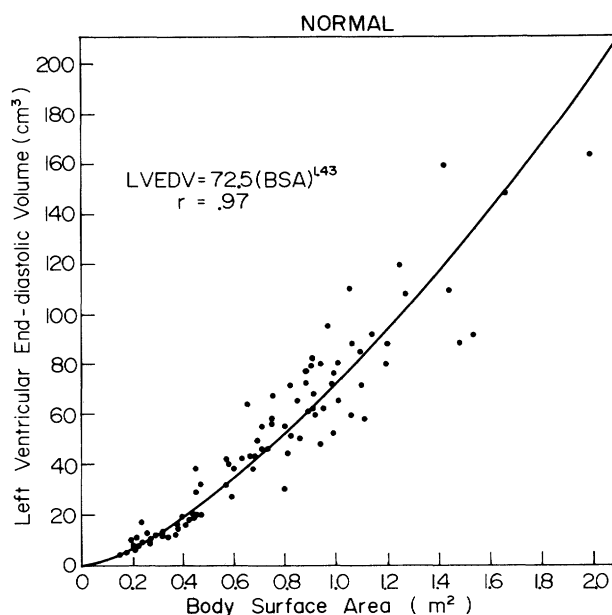


FIGURE 1. Left ventricular end-diastolic volume as a function of body surface area in patients with normal left heart. The equation and the solid line represents the best fit for this relationship.

RV end-diastolic pressure was higher than normal in group I (table 2). Pulmonic valve area in group I ranged from 0.30 to 0.79 cm²/M² (valve area/body surface area). In group II, pulmonary artery pressure was obtained only in two patients and the valve areas were 0.30 and 0.35 cm²/M² (table 2).

RVEDV in group I averaged $94\% \pm 4\%$ of normal (mean \pm SEM) and was not significantly different from normal (fig. 3). There was no statistically significant relationship between RVEDV and peak RV systolic

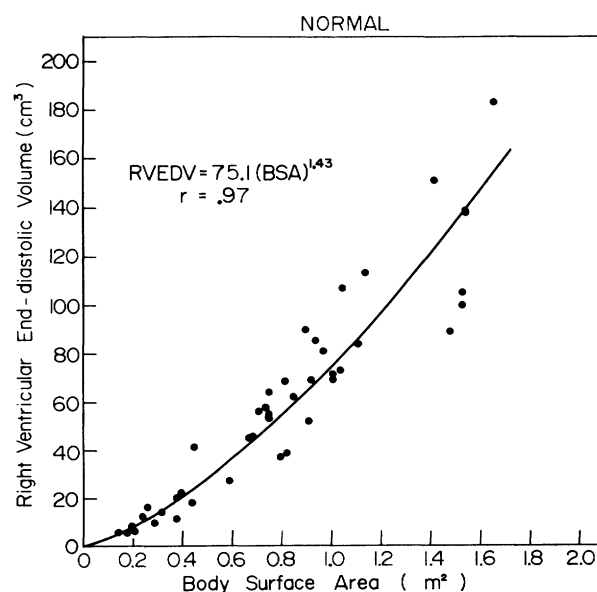


FIGURE 2. Right ventricular end-diastolic volume as a function of body surface area in patients with normal left heart. The equation and the solid line represents the best fit for this relationship. (Figure reprinted from *Circulation* 54: 555, 1976)

TABLE 1. Vital Statistics and Hemodynamic Variables

Group	BSA (m ²)	HR (beats/min)	Hgb (g%)	Peak LVP (mm Hg)
Normal				
N = 42	0.78 ± 0.06	112 ± 5	12.8 ± 0.03	98 ± 4
Gr. I (PS)				
N = 21	0.68 ± 0.05	110 ± 5	12.9 ± 0.2	114 ± 7
P: I vs normal	NS	NS	NS	NS
Gr. II (PS + R→L shunt)				
N = 7	0.47 ± 0.09	131 ± 8	16.0 ± 1.1	97 ± 11
P: II vs normal	NS	NS	0.04	NS
P: II vs. I	NS	NS	0.04	NS

All values are mean ± SEM.
Abbreviations: BSA = body surface area; HR = heart rate; Hgb = hemoglobin;
LVP = left ventricular pressure; NS = not significant.

pressure. RVEDV and pulmonary valve area were related (fig. 4). In addition, a multiple regression analysis of RVEDV (%) as a function of peak RV pressure (RVP), age in years and pulmonary valve area (PVA) showed good correlation and was expressed by:

$$\text{RVEDV (\%)} = 14 + 0.41 (\text{RVP}) - 1.6 (\text{age}) + 110 (\text{PVA});$$

$$r = 0.77 \quad (\text{Eq. 7})$$

RVEDV in group II (63% ± 9% of normal) was significantly ($P < 0.001$) less than normal. Two patients (B. S. and O. T.) in group II had very small RVs (34% and 23% of normal, respectively). One patient (not shown in table 2) with PS, right-to-left atrial shunt and tricuspid insufficiency had increased RVEDV (177% of normal) and increased RVSI (5.63 L/min/M²).

Right ventricular ejection fraction (RVEF) in group I (0.70 ± 0.02) was significantly ($P < 0.001$) higher than normal, but was not different from normal in group II.

Right ventricular systolic index (RVSI) was normal in group I (4.36 ± 0.23 L/min/M²), but was significantly ($P < 0.001$) less than normal in group II (table 2).

Left ventricular end-diastolic volume in group I was not different from normal, and was significantly ($P = 0.026$) greater than normal in group II (mean = 125% ± 6% of normal).

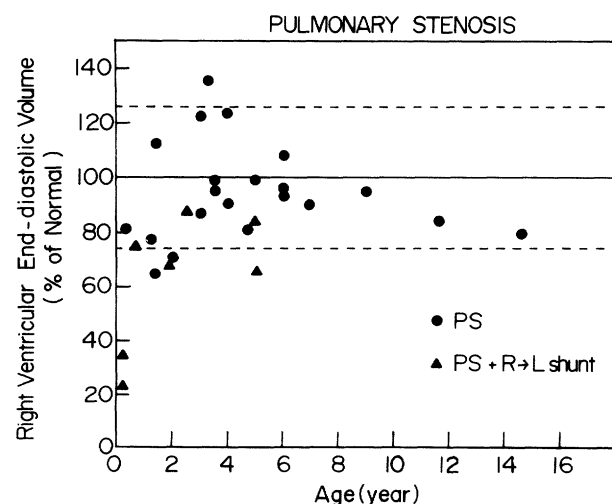


FIGURE 3. Right ventricular end-diastolic volume in pulmonary stenosis patients on the ordinate versus age on the abscissa. The solid and dashed lines represent the mean ± 1 SD in patients with normal right heart.

LVEF in group I was normal. This variable in group II was significantly less than normal ($P = 0.027$) and group I ($P = 0.004$). Left ventricular systolic output was not different from normal in both groups I and II (table 2).

The RVEDV/LVEDV ratio in group I (table 2, fig. 5) and group II was significantly ($P \leq 0.001$) less than normal. The RVSI/LVSI ratio was not different from normal in group I (0.94 ± 0.02), and was significantly ($P < 0.001$) less than normal in group II (0.53 ± 0.09).

Discussion

Simpson's rule method has been used with accuracy to calculate RV volume in patients with normal right hearts and more recently in a number of PS patients.^{7, 9, 18} However, further validation of the accuracy and applicability of this method is needed in patients with right ventricular pressure overload and hypertrophy. The area-length method of calculating LV volume has been widely accepted^{7, 8, 16, 19} and LVSO could serve as a reference for RVSO in patients with isolated PS (group I). The fact that the relation between RVSO and LVSO in group I patients with isolated PS was not different from normal (RVSO = 0.89 LVSO - 0.11; SEE = 0.32; $r = 0.96$, fig. 6) indicates that Simpson's rule method is quite accurate for calculating RV volumes in PS patients.

It is of interest that the nonlinear relation between normal LVEDV and BSA, shown in figure 1, was similar but not identical to that of RVEDV with BSA (fig. 2). It is important to note that LVEDV/BSA or RVEDV/BSA should not be used for normalization of end-diastolic volumes for patient size because the relationships between EDV and BSA were neither linear nor proportional in normal children, thus the ratio of EDV/BSA will change with BSA showing erroneous difference when comparing groups of different mean age.

The fact that RV volume in patients with isolated PS was normal or decreased from normal is in agreement with the previously reported pathological observations.^{2, 20, 21, 22} The decreased RVEDV/LVEDV ratio in group I is similar to that reported previously by Graham and co-workers.⁷ The reason that the RVEDV/LVEDV ratio in group I was significantly reduced from normal was due to slight but insignificant decrease in RVEDV and slight increase in LVEDV. Although RVEDV in group I was not significantly

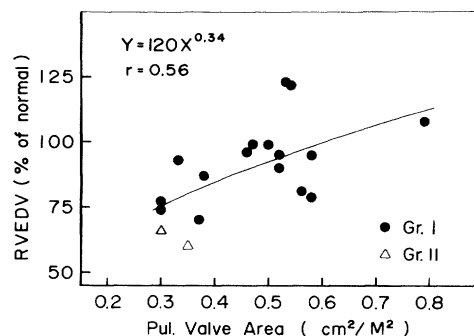


FIGURE 4. Right ventricular end-diastolic volume (% of normal) as a function of pulmonary valve area on the abscissa expressed as valve area/body surface area (cm²/M²).

TABLE 2. Right and Left Ventricular Volume Variables

Patient	Age (yrs)	RVP (mm Hg)	RVEDV (% of normal)	RVEF	RVSI (L/min/M ²)	LVEDV (% of normal)	LVEF	LVSI (L/min/M ²)	RVEDV LVEDV	RVEF LVEF	RVSI LVSI	PVA (cm ² /m ²)	R→L shunt (%)
<i>Normal (N = 42)</i>													
Mean	5.9	31/ 6	103	0.61	4.37	104	0.63	4.43	1.02	0.97	1.01		
SEM	0.7	2/0.4	4	0.01	0.17	4	0.01	0.19	0.02	0.02	0.02		
<i>Group I (P.S.)</i>													
1 B.C.	1.2	125/ 8	77	0.76	3.93	101	0.62	4.03	0.80	1.22	0.98	0.30	0
2 B.P.	4.0	100/ 8	74	0.65	3.11	126	0.56	4.42	0.62	1.16	0.70	0.30	0
3 B.R.	3.5	60/ 7	95	0.61	4.58	134	0.63	6.32	0.74	0.97	0.72	0.58	0
4 B.R.	6.0	120/12	93	0.67	3.63	114	0.52	3.82	0.86	1.29	0.95	0.33	0
5 C.T.	4.0	120/ 9	123	0.55	7.01	101	0.63	6.63	1.29	0.87	1.06	0.53	0
6 D.A.*	11.6	80/ 8	84	0.72	3.72	114	0.63	4.14	0.78	1.14	0.90	—	0
7 D.E.*	9.0	90/10	95	0.76	4.78	117	0.66	5.52	0.83	1.14	0.87	0.52	0
8 D.S.	6.9	90/ 8	90	0.82	4.38	118	0.68	4.62	0.78	1.22	0.95	0.52	0
9 F.C.*	6.0	68/ 9	108	0.81	6.63	130	0.75	7.11	0.86	1.08	0.93	0.79	0
10 G.L.	3.0	100/10	122	0.79	5.69	173	0.65	6.53	0.73	1.21	0.87	0.54	0
11 I.A.	4.7	60/ 9	81	0.80	3.99	108	0.62	3.96	0.78	1.28	1.01	0.56	0
12 M.J.	3.5	75/ 9	99	0.62	3.94	104	0.64	4.21	0.96	0.98	0.94	0.50	0
13 O.P.*	6.0	75/15	96	0.63	3.29	100	0.58	3.40	1.02	1.09	0.97	0.46	0
14 S.M.*	3.0	75/15	87	0.56	2.85	80	0.50	2.48	1.14	1.12	1.15	0.38	0
15 T.J.	14.6	68/ 6	79	0.71	4.44	108	0.64	4.85	0.78	1.10	0.92	0.58	0
16 T.W.*	5.0	80/10	99	0.67	3.94	119	0.57	3.99	0.87	1.17	0.99	0.47	0
17 W.H.*	3.3	200/ 7	135	0.63	4.95	131	0.71	5.14	1.08	0.89	0.96	—	0
18 D.J.*	0.4	120/ 8	82	0.77	3.87	115	0.58	4.06	0.73	1.33	0.96	—	10
19 D.J.*	1.4	113/12	64	0.87	4.42	103	0.62	4.47	0.65	1.40	0.99	—	10
20 M.G.*	2.0	90/ 7	70	0.71	4.16	104	0.66	5.34	0.90	1.08	0.78	0.37	8
21 M.T.*	1.4	67/ 6	112	0.67	4.30	99	0.69	4.03	1.15	0.97	1.07	—	12
Mean	4.8	94/ 9	94	0.70	4.36	114	0.63	4.72	0.87	1.13	0.94	0.48	
SEM	0.8	7/ 1	4	0.02	0.23	4	0.01	0.25	0.04	0.03	0.02	0.03	
P: I vs	NS	<0.001	NS	<0.001	NS	NS	NS	NS	=0.001	<0.001	NS		
<i>Normal</i>													
<i>Group II (PS + R→L shunt)</i>													
22-a B.S.	0.1	90/ 5	34	0.76	1.20	108	0.49	2.73	0.28	1.57	0.45	—	59
22-b B.S.	0.7	150/10	75	0.41	1.98	123	0.55	4.44	0.60	0.74	0.45	—	48
23 O.T.	0.1	170/10	23	0.52	0.84	143	0.65	5.53	0.19	0.84	0.15	—	50
24 C.J.	5.0	75/10	66	0.56	2.36	109	0.55	3.75	0.64	1.02	0.63	0.30	35
25 C.R.	2.5	94/ 8	88	0.55	4.08	120	0.58	5.61	0.76	0.95	0.73	—	25
26 D.J.	5.0	90/ 5	84	0.58	3.98	117	0.52	4.71	0.75	1.11	0.84	0.35	31
27 W.S.	1.9	112/10	68	0.54	2.34	152	0.53	4.94	0.47	1.02	0.47	—	40
Mean	2.2	112/ 8	63	0.56	2.40	125	0.55	4.53	0.53	1.03	0.53	—	
SEM	0.8	13/ 1	9	0.04	0.47	6	0.02	0.38	0.08	0.10	0.09	—	
P: II vs	=0.003	<0.001	<0.001	NS	<0.001	=0.026	=0.027	NS	<0.001	NS	<0.001		
<i>Normal</i>													
P: II vs I	NS	NS/NS	=0.001	=0.002	<0.001	NS	=0.004	NS	<0.001	NS	=0.004		

Abbreviations: PS = pulmonic stenosis; RVP = right ventricular pressure; RVEDV = RV end-diastolic volume; RVEF = RV ejection fraction; RVSI = RV systolic index; LV = left ventricle; PVA = pulmonary valve area; R→L shunt = right-to-left shunt; * = patent foramen ovale; NS = not significant ($P > 0.05$).

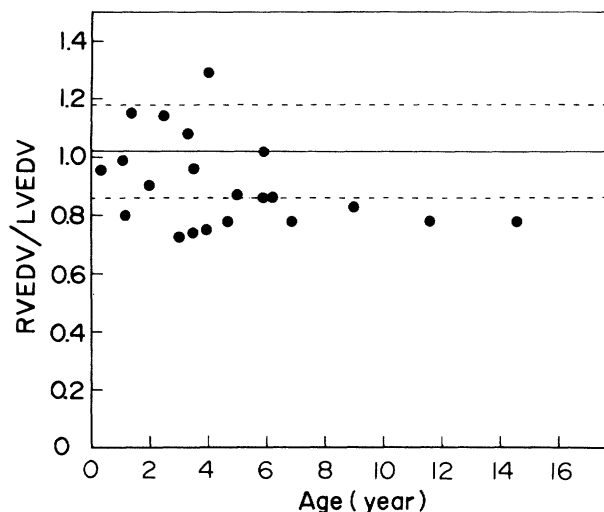


FIGURE 5. The right ventricular end-diastolic volume/left ventricular end-diastolic volume ratio (RVEDV/LVEDV) on the ordinate as a function of age on the abscissa in group I. The solid and dashed line represents the mean \pm 1 SD for normal values.

decreased from normal, the decrease in RVEDV with age and severity of obstruction (equation 7, fig. 4) suggests a gradual decrease in RV size in these patients. This observation is similar to the decreased LVEDV in patients with aortic stenosis shown by Graham and associates.²³ Some patients with severe RV outflow obstruction are exceptions to the rule (patients #5, 10, 17). All three patients showed no tricuspid regurgitation on RV cineangiogram to account for the increased RVEDV. Furthermore, patient #17 was studied three months after valvulotomy and showed: RV pressure = 48/4 mm Hg, RVEDV = 88% of normal, and RVEF = 0.75. These values, in addition to the equal LV and RV output preoperatively, suggest that the increased preoperative RVEDV was due to increased afterload and not to valvular or myocardial damage.

Fisher and coworkers⁹ showed that RVEDV normalized

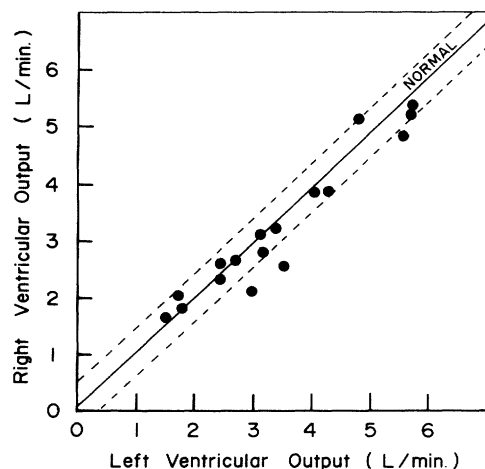


FIGURE 6. Right ventricular output (L/min) as a function of left ventricular output (L/min) in patients with pulmonary stenosis. The solid and dashed lines represent the regression line \pm SE.

for body surface area in PS patients was significantly less than normal. RVEDV/BSA in group I averaged $62 \text{ cm}^3/\text{M}^2 \pm 13 \text{ cm}^3/\text{M}^2$ (SD) and was also significantly ($P < 0.001$) less than normal. Although our data agrees with those reported by Fisher, we feel that this difference is due to an error introduced by dividing RVEDV by BSA as outlined above. Figure 3 in Fisher's paper shows that RVEDV in older PS patients was below normal but was in the normal range for younger patients, which is consistent with the decreasing RVEDV with age seen in our patients (suggested by Eq. 7).

The markedly reduced RVEDV in group II is in agreement with a previous report by Freed and coworkers.²⁴ The small RV in these patients is probably due to decreased RV filling which in turn could be explained by the following factors: 1) concentric RV hypertrophy and decreased RV compliance associated with increased RV filling pressure, 2) small and/or stenotic tricuspid valve, 3) the interatrial communication with right-to-left shunt. Pressure pullback from the RV to the right atrium in group II patients showed no significant diastolic gradient, and the difference between the "a" wave and RV end-diastolic pressure was ≤ 2 mm Hg, thus excluding significant tricuspid stenosis. All patients were in normal sinus rhythm. It can be assumed, however, that very small RVs have small tricuspid valves. The very small RV size in two patients (BS = 34% and OT = 23%) might be due to an earlier development of a critical pulmonary stenosis leading to a significant decrease in RV filling during intrauterine life. Two patients in this group (CR, DJ) and some patients in group I had normal RVEDV and normal or increased ejection fractions. These patients probably did not have severe obstruction during the intrauterine period to alter their flow dynamics thus leading to a small RV at birth.

Although RVEF was significantly higher than normal, RV output in group I was normal and not different from LV output. This was due to the slightly (but not significantly) decreased RVEDV and decreased RVEDV/LVEDV ratio.

The increased RVEF in children with isolated PS (group I) is in agreement with Fisher's data.⁹ This observation is similar to the increased LVEF in patients with isolated aortic stenosis,²³ and is in contrast to the decreased LVEF in response to the acute increase in afterload in the experimental animal.²⁵ This difference is probably related to an increase in ventricular muscle mass in patients with chronically elevated ventricular pressure. The fact that RVEF in group I was higher than both normal and group II could be explained by: 1) RVEDV (preload) in group II was significantly less than in group I; 2) peak RV pressure (RVP) in group II was slightly higher than RVP in group I; 3) the relative hypoxia in group II might play an additional role in suppressing RVEF. It is interesting to note that RVEF was not decreased in the three oldest patients in group I which suggests that RV function remains normal in the pediatric age group. In contrast, Gentzler et al.¹⁸ reported that only two out of 17 adult patients had increased RVEF with prolonged RV pressure overload (all patients had noncyanotic cardiac lesions, however only three had PS) while eight of the 17 had decreased RVEF. Furthermore, RVEF was decreased in patients with increased RVEDV (in-

creased preload) but had no relationship to peak RV pressure (afterload), thus suggesting depression in myocardial contractility in adult patients which might be related to myocardial fibrosis. The high RVEF in PS (group I) is also in contrast to low RVEF in children with transposition of the great vessels^{10, 11} and tetralogy of Fallot.^{12, 13} Peak RV pressure is increased in patients with transposition or tetralogy and RVEDV in transposition patients is larger than in PS patients. This indicates that chronic pressure overload usually increases ejection fraction in noncyanotic patients and does not suppress cardiac function in the pediatric age group. The decreased RVEF in transposition and tetralogy patients suggests that arterial desaturation enhances the development of myocardial fibrosis and cardiac depression in children.

Although RVEDV was decreased in many patients with isolated PS, RV output in these patients remained normal due to an increase in RVEF. Furthermore, the data shows no evidence of deteriorating RV performance in these patients and therefore RV volume variables are not essential in the management of children with isolated pulmonary stenosis. In contrast two (#22, #23) of the six patients in group II had significantly reduced RVEDV and RVSI. The very small RV in these patients could not maintain a normal forward cardiac output and the initial surgical procedure should include pulmonary valvulotomy only, leaving the closure of the interatrial communication for a later date and after a significant increase in RV size. Graham et al.¹⁴ showed that small RVs increased significantly after pulmonary valvulotomy. In addition, Freed and coworkers²⁴ reported ten cyanotic patients preoperatively, who became acyanotic and asymptomatic after valvulotomy, thus suggesting significant increase in RV size and output. It is interesting that the small RV (34% of normal) in patient 22 increased significantly (75% of normal) with increasing RV pressure and was associated with a decrease in RVEF. This suggests that RV size may increase without valvulotomy. The normal tricuspid gradient in the patient ruled out tricuspid valvular stenosis. Although an increase in afterload depresses ejection fraction, RVEF in this patient was well below the lower limits of normal, suggesting a definite decrease in RV function during this period. This is suggestive evidence that pressure overload should not be left alone, if there is a possibility for a relief of the stenosis. It is clear that RV volume determination in this group of patients with right-to-left interatrial shunt can be used to identify patients with small RV and is helpful in patient management.

Left ventricular pressure was normal in both groups. Left ventricular end-diastolic volume was normal (group I) or slightly higher than normal (group II). Left ventricular ejection fraction was normal in group I, thus suggesting that LV function is normal in these patients. The significantly ($P = 0.027$) decreased LVEF in group II in the presence of normal LV pressure (normal afterload) and increased LVEDV (increased preload) strongly suggest depression in LV function. This provides additional evidence that chronic hypoxia depresses ventricular function. The normal RVEDV in group I with increased RVEF in the presence of increased RV pressure suggests that RV function is not

depressed from normal and could possibly be higher than normal. Since preload and afterload can best be expressed in terms of end-diastolic and systolic stress and these parameters cannot be calculated for the RV, the data presented here cannot be used as evidence of increased contractility in these patients. The data, however, strongly suggest that RV function in group I is not depressed from normal.

In summary the data suggest that RV function in patients with isolated pulmonary stenosis is normal and determination of RV volume variables is not essential in the management of these patients. On the other hand, RV volume variables in PS patients with right-to-left interatrial shunt are helpful in patient management.

Acknowledgments

We wish to express our appreciation to the valuable technical assistance of Ms. Jean Gordon, Mr. George Oku, and Mrs. Kay Graetz and to Mrs. Cathy Heteniak for typing the manuscript.

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