JACC Vol 2, No. 2
August 1983 225–32

Right Ventricular Function in Valvular Heart Disease: Relation to Pulmonary Artery Pressure

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Right ventricular angiography was performed in 46 patients with acquired valvular heart disease and 8 normal subjects. Right ventricular ejection fraction (RVEF) correlated highly only with right ventricular peak systolic pressure (RVPSP) and mean pulmonary artery pressure, both in patients with and without tricuspid insufficiency. For the group, RVEF = −0.33 RVPSP + 63 (correlation coefficient [r] = −0.76, probability [p]<0.001). Of 20 patients with moderate or severe elevation of pulmonary artery pressure, 17 (85%) had an abnormally low ejection fraction (<47%), while 19 (73%) of 26 patients with normal or mildly elevated pulmonary artery pressure had a normal right ventricular ejection fraction.

In seven patients with elevated pulmonary artery pressure, a second ventriculogram was performed during intravenous nitroglycerin administration. Nitroglycerin produced a significant decrease in right ventricular peak systolic pressure (59 ± 22 to 49 ± 18 mm Hg, mean ± standard deviation) ($p<0.05$) and in end-systolic volume (71 ± 16 to 59 ± 11 ml/m^2) ($p<0.05$), and an increase in ejection fraction (43 ± 9 to 48 ± 7%) ($p<0.05$). Thus, at least part of the depression of ejection fraction in patients with elevated pulmonary pressure is reversible with a decrease in pulmonary artery pressure.

The relation of right ventricular pump function to afterload in adults with valvular heart disease has not been definitely established. Previous angiographic studies (1,2) failed to demonstrate a relation between right ventricular afterload or peak systolic pressure and pump performance. However, in other investigations using radionuclide techniques, decreased ejection fraction was demonstrated in patients with increased pulmonary artery pressure due to valvular heart disease (3) or chronic pulmonary disease (4,5). Additionally, a recent study (6) using gated radionuclide ventriculography demonstrated a significant correlation between right ventricular ejection fraction and both pulmonary artery and right atrial pressures. Although studies by Maughan et al. (7) suggest that right ventricular end-systolic volume is affected by acute changes in afterload, other acute and chronic animal studies (8–11) have yielded conflicting results.

Thus, to further define the relation of right ventricular pump function to hemodynamic variables in patients with acquired valvular heart disease, this investigation was performed during cardiac catheterization using biplane right cineventriculography to determine right ventricular volume and ejection fraction.

Methods

Study patients. The study group consisted of 46 patients with acquired valvular heart disease and 8 normal subjects who underwent diagnostic cardiac catheterization. There were 13 men and 33 women with an average age of 52 years (range 24 to 76). No patient had regional wall motion abnormalities of either the right or left ventricle and 41 had normal coronary arteries. Twenty-one patients had normal sinus rhythm and 25 had atrial fibrillation or flutter. Eighteen patients had isolated mitral valve disease, 10 with dominant mitral stenosis and 8 with either mixed stenosis and insufficiency or pure mitral insufficiency. Ten patients had both mitral and aortic valve disease, 4 had isolated aortic valve disease and 14 (most of whom had aortic and mitral disease) were studied after cardiac surgery because of recurrent symptoms. The cause of the valve disease was rheumatic in 37 patients, ruptured chordae tendineae in 4, bicuspid aortic valve in 2 and unknown in 3.

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0735-1097/83/$3.00

JACC Vol 2, No. 2
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Tricuspid regurgitation was assessed angiographically as absent, mild (small amount of contrast medium within the right atrium), moderate (contrast medium filling the right atrium less densely than the right ventricle) or severe (contrast medium filling the right atrium as densely than the right ventricle within two or three systoles). A significant amount of tricuspid regurgitation (moderate or severe angiographically) was present in 17 patients.

Eight subjects with normal coronary arteries, normal pressure in both the left and right ventricles and qualitatively normal left and right ventriculograms were used for comparison as normal controls. All patients signed informed consent. This study was approved by this institution's research committee and institutional review board.

Procedures and recordings. Left heart catheterization was accomplished using the Seldinger technique (12) and Judkins (13) type 7 or 8F catheter passed retrograde from the right femoral artery. Initial right heart catheterization was performed via the right femoral vein with a 7F Cournand catheter. Right ventriculography was done with a 7 or 8F specially designed pigtail catheter inserted via the right femoral vein. Pressures were measured with Statham P23Db transducers and recorded simultaneously with a standard limb lead of the electrocardiogram on an Electronics for Medicine VR12 recorder. Cardiac output was determined by use of the Fick principle from measurements of oxygen consumption and systemic and pulmonary artery oxygen content. Biplane left and right ventriculogram in the right and left anterior oblique views were recorded at 50 frames/s using Renografin-76, 40 to 50 ml, injected at a rate of 10 to 14 ml/s. Nine inch (22.9 cm) image intensification was used in both planes for right ventriculography to ensure inclusion of the entire right ventricular cavity. In seven of the study patients with elevated pulmonary artery pressure secondary to mitral valve disease and no significant tricuspid regurgitation, a second right ventriculogram was performed during administration of intravenous nitroglycerin titrated to decrease right ventricular peak systolic pressure by approximately 20% (14). In two of these patients, specially designed micromanometer-tipped catheters were used for ventriculography. At least 15 minutes elapsed between ventriculograms to allow for dissipation of the effects of the contrast medium (15). In each patient, selective left and right coronary arteriograms were recorded in at least two projections. Intracardiac pressures and cardiac output were recorded before angiography.

Cases were excluded from analysis if right ventricular systolic pressure just before right ventriculography was not within 5 mm Hg of the recording during initial hemodynamic evaluation, if there were not at least two well opacified conducted beats not following a premature ventricular contraction or if there was catheter-induced tricuspid regurgitation (defined as regurgitation associated with catheter interference with the tricuspid leaflets).

Ventricular volumes. Left ventricular volumes and ejection fraction were calculated using a light-pen computer system (Electronics for Medicine) that employs the single plane, right anterior oblique area-length method of Sandler and Dodge (16). Right ventricular volumes and ejection fraction were calculated using a biplane area-length method (17) from area and length measurements generated with a special modification of the computer system used for left ventricular volumes. All right ventricular volumes were measured in duplicate by one of us (R.G.) and are reported as an average of at least two beats (three beats for most patients with atrial fibrillation). The initial 15 cases were reanalyzed at least 3 months after the initial measurements and the reproducibility of ejection fraction was ± 3.2 ejection fraction units.

To confirm the validity of our angiographic determination of right ventricular volume, stroke volume determined from the right ventriculogram (RVSV) was compared with stroke volume determined from the left ventriculogram (LVSV) for the normal subjects. There was no difference between right (98 ± 18 ml) and left (93 ± 20 ml) ventricular stroke volume by paired *t* tests and using linear regression: RVSV = 0.76 LVSV + 27, (r = 0.88). Additionally, right ventricular end-systolic volume index and ejection fraction for normal subjects in our laboratory (98 ± 13 ml/m^2 and 55 ± 4%, respectively, were similar to published values obtained with similar techniques (95 ± 11 ml/m^2 and 49 ± 2%) (2).

Statistical analysis. The relation of each measured hemodynamic and ventriculographic variable to right ventricular ejection fraction and end-systolic volume (as measures of right ventricular pump performance) was analyzed using linear regression. A high degree of correlation was defined as a correlation coefficient (r) greater than 0.7 (18). Differences between the normal group and the groups with and without significant tricuspid regurgitation were first determined using analysis of variance. If a difference was found, then a modified *t* test was used to determine where the differences occured (19). In the patients in whom a second ventriculogram was obtained after administration of nitroglycerin, differences were determined using Student's paired *t* test. A probability level of 0.05 was considered significant.

Results

Hemodynamic and ventriculographic results for the study patients and control subjects are listed in Table 1. Among the 46 patients studied, there was a wide range of both left and right ventricular function. Left ventricular end-diastolic pressure ranged from 3 to 28 mm Hg and ejection fraction from 22 to 86%. Right ventricular end-diastolic pressure ranged from 3 to 32 mm Hg and ejection fraction from 23 to 68%. Persons with a right ventricular systolic pressure

Table 1. Hemodynamic and Ventriculographic Measurements

	Patients (n = 46)		Control Subjects (n = 8)	
	Range	Mean ± SD	Range	Mean ± SD
LVPSP (mm Hg)	100 to 225	140 ± 32	100 to 145	113 ± 14
LVEDP (mm Hg)	3 to 28	15 ± 7	11 to 15	13 ± 1
LVESV (ml/m^2)	12 to 91	36 ± 17	9 to 33	20 ± 7
LVEDV (ml/m^2)	50 to 198	97 ± 32	56 to 80	70 ± 9
LVEF (%)	22 to 86	62 ± 14	59 to 84	71 ± 8
$\overline{PCW}$ (mm Hg)	8 to 40	22 ± 9	4 to 12	8 ± 3
PASP (mm Hg	22 to 110	56 ± 24	17 to 25	21 ± 3
PADP (mm Hg)	10 to 50	24 ± 10	7 to 11	10 ± 2
$\overline{PA}$ (mm Hg)	16 to 50	35 ± 15	13 to 16	15 ± 2
RVSP (mm Hg)	22 to 105	54 ± 21	18 to 30	24 ± 4
RVEDP (mm Hg)	3 to 32	12 ± 6	4 to 8	6 ± 2
$\overline{RA}$ (mm Hg)	3 to 30	11 ± 6	4 to 7	6 ± 1
RVESV (ml/m^2)	32 to 132	68 ± 23	30 to 58	44 ± 9
RVEDV (ml/m^2)	76 to 234	123 ± 33	70 to 115	98 ± 13
RVEF (%)	23 to 64	45 ± 9	50 to 64	55 ± 4
SVI (ml/m^2)	14 to 68	31 ± 11	41 to 51	47 ± 4

LVEDP = left ventricular end-diastolic pressure; LVEDV = left ventricular end-diastolic volume index; LVEF = left ventricular ejection fraction; LVESV = left ventricular end-systolic volume index; LVPSP = left ventricular peak systolic pressure; $\overline{PA}$ = mean pulmonary artery pressure; PADP = pulmonary artery diastolic pressure; PASP = pulmonary artery peak systolic pressure; $\overline{PCW}$ = mean pulmonary capillary wedge pressure; RA = mean right atrial pressure; RVEDP = right ventricular end-diastolic pressure; RVEDV = right ventricular end-diastolic volume index; RVEF = right ventricular ejection fraction; RVESV = right ventricular end-systolic volume index; RVSP = right ventricular peak systolic pressure; SD = standard deviation; SVI = stroke volume index from the Fick cardiac output

of 22 to 105 mm Hg are represented, with a peak systolic pressure of 54 ± 21 mm Hg (mean ± standard deviation).

Correlation with loading conditions. The only variables listed in Table 1 that correlated highly ($r > 0.7$) with right ventricular ejection fraction (RVEF) were right ventricular (or pulmonary artery) peak systolic pressure (RVPSP) and mean pulmonary artery pressure ($\overline{PA}$). For right ventricular peak systolic pressure, the relation in 46 patients was $RVEF = -0.33\,RVPSP + 63$ ($r = 0.76$, $p < 0.001$). For mean pulmonary artery pressure the relation was $RVEF = -0.46\,\overline{PA} + 60$ ($r = 0.75$, $p < 0.001$). For the group, 17 (85%) of 20 patients with moderate to severe elevation of pulmonary artery pressure (peak systolic pressure ≥ 50 mm Hg) had an abnormally low ejection fraction (< 47%) but only 7 (27%) of 26 with normal or mildly elevated pulmonary artery pressure had a low ejection fraction. The strong correlation between right ventricular ejection fraction and peak systolic pressure (PSP) was present in the subgroup of 25 patients with atrial fibrillation ($RVEF = 0.43\,PSP + 69$)($r = 0.81$, $p < 0.001$) and the 25 in sinus rhythm ($RVEF = -0.26\,PSP + 60$)($r = 0.75$, $p < 0.001$). Although, no variable met the definition of a high correlation ($r > 0.7$) with right ventricular end-systolic volume index (RVESV), again right ventricular peak systolic pressure in the 46 patients had the highest correlation with end-systolic volume ($RVESV = 0.61\,RVPSP + 35$)($r = 0.58$, $p < 0.001$).

The correlation between right ventricular ejection frac-

Table 2. Right Ventricular Pressure, Volumes and Ejection Fraction in Three Groups of Patients

	EDP (mm Hg)	PSP (mm Hg)	EDV (ml/m^2)	ESV (ml/m^2)	EF (%)
Group 1 (n = 8)	6 ± 2	24 ± 4	98 ± 13	44 ± 9	55 ± 4
Group 2 (n = 29)	12 ± 5	51 ± 18	112 ± 21	61 ± 18	46 ± 8
Group 3 (n = 17)	14 ± 7	60 ± 27	143 ± 39	80 ± 25	44 ± 11
1 vs. 2	*	*	NS	*	*
1 vs 3	*	*	*	*	*
2 vs. 3	NS	NS	*	*	NS

*$p < 0.05$. Values are mean ± standard deviation.

Group 1 = normal subjects; Group 2 = patients without tricuspid insufficiency; Group 3 = patients with tricuspid insufficiency, EDP = right ventricular end-diastolic pressure; EDV = right ventricular end-diastolic volume index; EF = right ventricular ejection fraction, ESV = right ventricular end-systolic volume index; NS = not significant; PSP = peak right ventricular systolic pressure

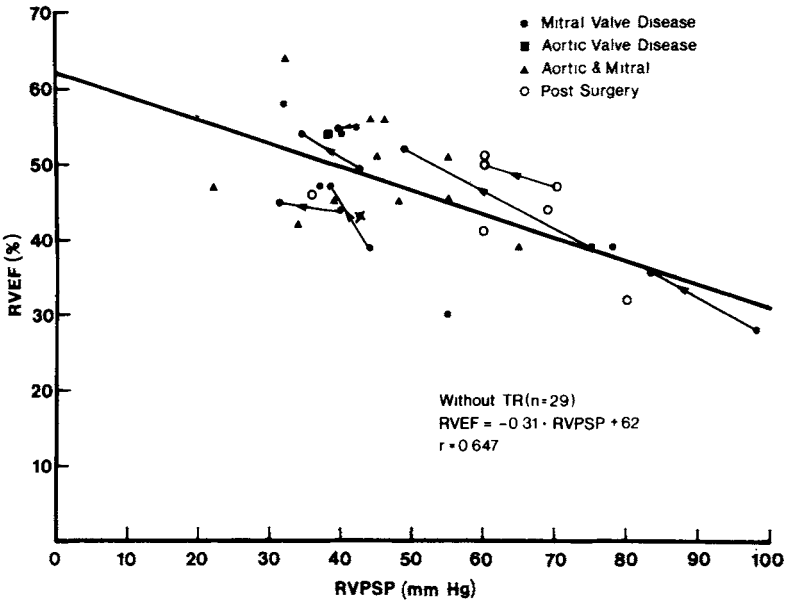

Figure 1. Relation of right ventricular ejection fraction (RVEF) and right ventricular peak systolic pressure (RVPSP) in 29 patients without tricuspid regurgitation (TR). There is a strong negative correlation between right ventricular ejection fraction and right ventricular peak systolic pressure. Patients in whom a second right ventriculogram was performed after intravenous nitroglycerin have two points connected by a **line,** with the **arrow** pointing toward the intervention study. In six of these seven patients, there was a dramatic decrease in right ventricular peak systolic pressure and increase in right ventricular ejection fraction with nitroglycerin. r = correlation coefficient.

tion and variables of right ventricular filling pressure was weak. A correlation greater than 0.5 was present only between right ventricular ejection fraction and mean right atrial pressure ($\overline{RA}$) (RVEF = $-0.91\ \overline{RA} + 55$) ($r = 0.56$, $p < 0.001$). No significant correlation was found between right ventricular ejection fraction and any left ventricular hemodynamic or ventriculographic variables, and only a weak correlation ($r < 0.5$) was found between right ventricular ejection fraction and mean pulmonary capillary wedge pressure.

Effect of tricuspid regurgitation. Table 2 includes the pressure, volume and ejection fraction data for the normal subjects (group 1), patients without tricuspid regurgitation (group 2) and those with tricuspid regurgitation (group 3). Patients with and without tricuspid regurgitation had higher peak systolic and end-diastolic right ventricular pressures than did normal subjects, but there was no significant difference between patients with and without tricuspid regurgitation. As expected, patients with tricuspid regurgitation had a larger end-diastolic volume (143 ± 39 ml/m^2) than did the patients without tricuspid regurgitation (112 ± 21 ml/m^2) and the normal subjects (98 ± 13 ml/m^2), but there was no statistical difference between patients without tricuspid regurgitation and normal subjects. End-systolic volume was also larger in patients with (80 ± 25 ml/m^2) than in those without (61 ml/m^2) tricuspid insufficiency and normal subjects (44 ± 9 ml/m^2), and was larger in the patients without tricuspid regurgitation than in normal subjects.

There was no difference in ejection fraction in patients with (44 ± 11%) and without (46 ± 8%) tricuspid re-

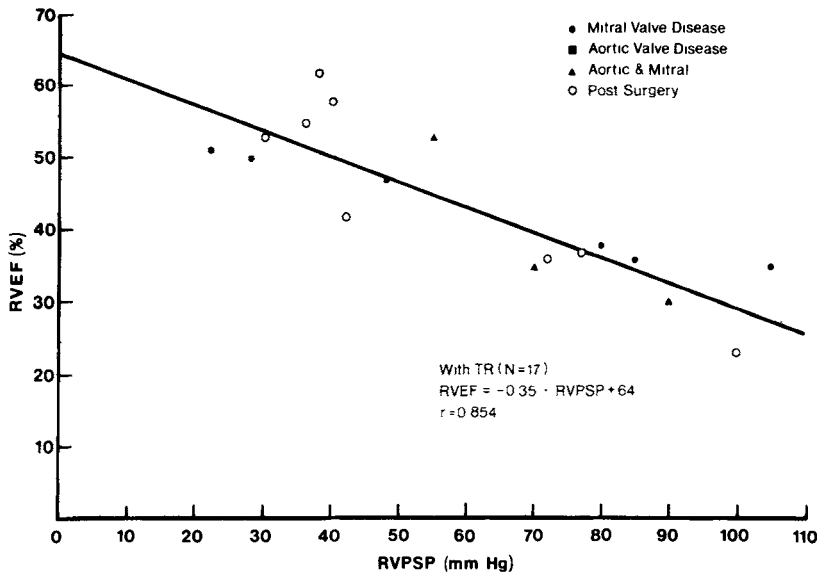

Figure 2. Relation of right ventricular ejection fraction and peak systolic pressure in 17 patients with tricuspid regurgitation. There is also a strong correlation between these two variables in this patient group. Abbreviations as in Figure 1.

grugitation, but mean ejection fraction was lower in both groups of patients than in the normal group (55 ± 4%). Although patients with tricuspid regurgitation had a larger right ventricular volume, a similar inverse correlation between right ventricular peak systolic pressure and ejection fraction was found in the groups without (RVEF = −0.31 RVPSP + 62)(r = 0.647, $p < 0.001$) (Fig. 1) and with (RVEF = −0.35 RVPSP + 64) tricuspid insufficiency (r = 0.854, $p < 0.001$) (Fig. 2). Correlation between end-systolic volume and peak systolic pressure was also similar in both groups: (RVESV = 0.56 RVPSP + 32) (r = 0.540, $p < 0.001$) for those without tricuspid regurgitation (Fig. 3) and (RVESV = 0.54 RVPSP + 48) (r = 0.572, $p < 0.001$) for the group with regurgitation (Fig. 4).

Nitroglycerin effect. Seven patients with mitral valve disease and elevated right ventricular peak systolic pressure had a second right ventriculogram performed during administration of intravenous nitroglycerin (14) (Table 3, Fig. 1 and 3). Nitroglycerin administration was associated with a statistically insignificant decrease in both end-diastolic pressure and volume in the right ventricle but with a statistically significant decrease in peak systolic pressure from 59 ± 22 to 49 ± 18 mm Hg and in end-systolic volume from 71 ± 16 to 59 ± 11 ml/m^2. Average right ventricular ejection fraction increased from 43 ± 9 to 48 ± 7%. There was no change in the ratio of peak systolic pressure to end-systolic volume for the group. The most dramatic increases in ejection fraction (from 28 to 36% and 39 to 52%, respectively) occurred in the two patients with the highest peak systolic pressure at rest (98 and 75 mm Hg, respectively, Fig. 2).

Micromanometer-tipped catheters were used for right ventriculography in two of the patients who were studied at baseline and with nitroglycerin. The data from one of these patients are shown in Figure 5. It is apparent that the pressure-volume relation during nitroglycerin therapy is shifted down and to the left. Ejection fraction increased, end-systolic volume decreased and the ratio of peak systolic pressure to end-systolic volume remained unchanged.

Discussion

Response of right versus left ventricle to increased systolic pressure. Patients with acquired valvular heart disease often have chronic elevation of pulmonary artery pressure. The adaptation of the low pressure right ventricle to this acquired increase in pulmonary artery pressure has not been definitively established. The left ventricle responds to an acquired increase in systolic pressure (as in valvular aortic stenosis) with an increase in muscle mass, while left ventricular wall stress and ejection fraction are often normal (20–22). When decreased ejection performance in the afterloaded left ventricle occurs, it may or may not be associated with an increase in wall stress (20). Thus, it appears that both decreased contractility and inadequate hypertrophy in response to increased stress may contribute to decreased ventricular function in the afterloaded left ventricle.

Our data suggest that the right ventricle responds differently than the left ventricle to an acquired chronic increase in systolic pressure. We found that most patients with significant elevation in right ventricular systolic pressure had decreased ejection fraction and that a high negative correlation was present between right ventricular peak systolic pressure and ejection fraction. Whether the abnormally low ejection fraction and increased end-systolic volume are secondary to a decrease in myocardial contractility or to inadequate hypertrophy in response to an increased pressure is difficult to determine without measuring wall stress. Unfortunately, the complex geometry of the right ventricle and the absence of an angiographic method of measuring right ventricular wall thickness preclude analysis of systolic wall

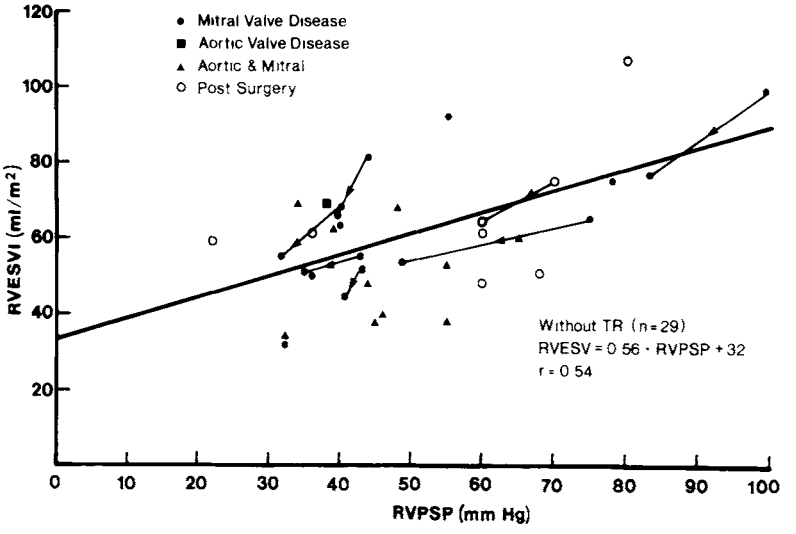

Figure 3. Relation of right ventricular end-systolic volume index (RVESVI) and peak systolic pressure in 29 patients without tricuspid regurgitation. There is a direct correlation between these two variables. In all patients studied before and after nitroglycerin, there is a decline in end-systolic volume and peak systolic pressure with nitroglycerin. Abbreviations as in Figure 1.

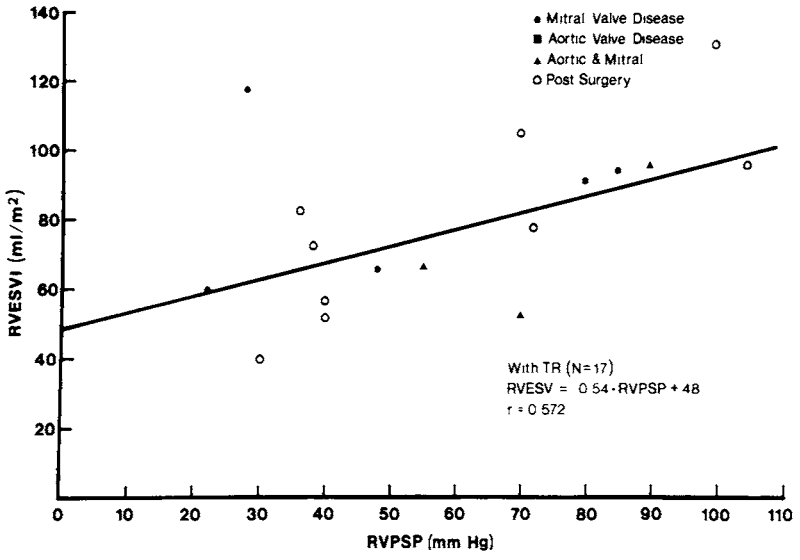

Figure 4. Relation of right ventricular end-systolic volume index and peak systolic pressure in patients with tricuspid regurgitation. Similar to findings in the patients without tricuspid regurgitation, there is a positive correlation between right ventricular end-systolic volume and peak systolic pressure. However, in the patients with tricuspid regurgitation, right ventricular end-systolic volume index tend to be larger. Abbreviations as in Figures 1 and 3.

stress or afterload. One can speculate that afterload is increased in patients with high pulmonary artery pressure and elevated end-systolic volume, although it is possible that with severe hypertrophy, afterload could be normal. Thus, it is likely that right ventricular ejection fraction in acquired heart disease is also inversely related to afterload. The depressed ejection performance found in our patients with elevated pulmonary artery pressure does not appear to be totally irreversible, because ejection fraction normalized when pulmonary artery pressure was acutely lowered with intravenous nitroglycerin (14). The right ventricle appears to respond differently to a congenital increase in pulmonary artery pressure; for example, in patients with congenital pulmonary stenosis the right ventricle remains small, with a normal or increased ejection fraction (1).

Role of tricuspid insufficiency. The effect on right ventricular performance of an additional volume load caused by tricuspid insufficiency has also not been well established. As might be expected, in this study tricuspid insufficiency was associated with increased end-diastolic and end-systolic volumes, but ejection fraction was similar in patients with and without tricuspid regurgitation. The relation of ejection fraction to afterload was also remarkably similar in patients with and without tricuspid regurgitation. Thus, it appears that although tricuspid regurgitation resulted in ventricular dilation, the response to elevated systolic pressure was in-

Table 3. Right Ventricular Function Before and After Nitroglycerin in Seven Patients

Patient	EDP (mm Hg)	PSP (mm Hg)	EDV (ml/m²)	ESV (ml/m²)	EF (%)	PSP/ESV (mm Hg/ml per m²)	AoP (mm Hg)	HR (beats/min)
1 C	18	98	139	99	28	0.99	100	73
N	14	83	120	77	36	1.08	102	68
2 C	17	75	107	66	39	1.14	185	81
N	13	49	114	54	52	0.91	170	81
3 C	6	40	120	68	44	0.59	135	67
N	4	32	99	55	45	0.58	115	80
4 C	15	70	141	75	47	0.93	140	66
N	11	60	129	65	50	0.92	100	65
5 C	8	45	132	81	39	0.56	120	90
N	9	40	129	66	47	0.61	110	92
6 C	10	43	116	52	55	0.83	87	62
N	10	41	98	45	54	0.91	85	79
7 C	4	43	108	55	49	1.28	130	68
N	4	35	112	51	54	1.46	115	75
C	11 ± 6	59 ± 22	123 ± 14	71 ± 16	43 ± 9	0.90 ± 0.25	128 ± 32	72 ± 10
N	9 ± 4	49 ± 18*	114 ± 13	59 ± 11*	48 ± 7 *	0.92 ± 0.27	114 ± 27	77 ± 9

*$p < 0.05$. Values are mean ± standard deviation.

AoP = peak aortic systolic pressure; C = control; EDP = right ventricular end-diastolic pressure; EDV = right ventricular end-diastolic volume; EF = right ventricular ejection fraction; ESV = right ventricular end-systolic volume index; HR = heart rate, N = nitroglycerin; PSP = right ventricular peak systolic pressure.

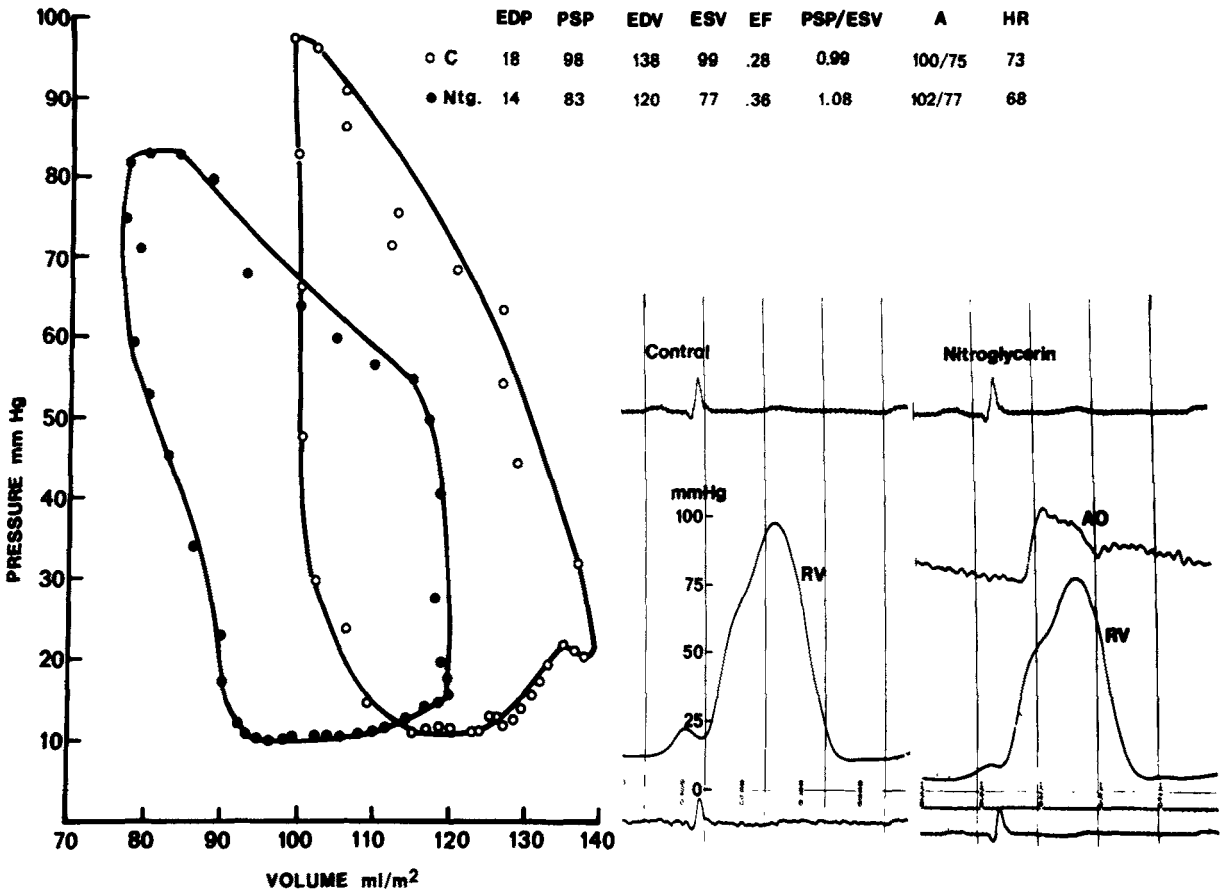

Figure 5. Right ventricular (RV) micromanometer pressures **(right panel)** and pressure-volume loops **(left panel)** for one patient during control conditions (C) and during intravenous nitroglycerin (Ntg.) administration. Hemodynamic and ventriculographic data are listed at the **top**. It is apparent that during nitroglycerin administration the pressure-volume relation in the right ventricle is shifted down and to the left. A = systemic arterial pressure (mm Hg); AO = aorta; EDP = right ventricular end-diastolic pressure (mm Hg); EDV = right ventricular end-diastolic volume index (ml/m^3); EF = right ventricular ejection fraction; ESV = right ventricular end-systolic volume index (ml/m^2); HR = heart rate (beats/min); PSP = right ventricular peak systolic pressure (mm Hg).

dependent of the volume load. End-systolic volume was higher in patients with than in those without tricuspid regurgitation for all levels of pulmonary artery pressure, implying greater myocardial impairment in those with tricuspid regurgitation.

Comparison with previous studies. A relation between right ventricular ejection fraction and pulmonary artery pressure was present in one previous study (6) of right ventricular function in adults, but not in others (1,2). Korr et al. (6), using gated radionuclide ventriculography, studied 24 patients with acquired heart disease, 6 with atrial septal defect and 20 normal subjects. Despite differences in technique, they also found a high negative correlation between right ventricular ejection fraction and pulmonary artery pressure in their patients with acquired heart disease. Gentzler et al. (1) studied 33 patients at catheterization using contrast ventriculography and came to very different conclusions. They found no consistent relation between right ventricular ejection fraction and pulmonary artery pressure, but a strong negative correlation between right ventricular end-diastolic volume and ejection fraction. However, they studied a very heterogeneous group of patients. Examination of only the patients with acquired valvular disease in their report (excluding those with congenital heart disease, coronary disease and primary myocardial disease) shows a relation between right ventricular ejection fraction (RVEF) and peak systolic pressure (RVPSP) quite similar to that demonstrated in our current investigation (RVEF = -0.40 RVPSP + 61, r = 0.583). Wroblewski et al. (2) examined seven patients with isolated mitral stenosis and seven normal subjects with contrast ventriculography. They found no relation between right ventricular ejection fraction and afterload. The discrepancy between their findings and our data may in part be explained by the fact that they studied a relatively small group of patients, who appear to have been earlier in the course of their disease than those in our study group.

End-systolic pressure-volume relation in the right ventricle. In the left ventricle, there appears to be a unique

end-systolic pressure-volume relation for any given inotropic state both in experimental animals (20) and in human beings (21). In human beings the peak systolic left ventricular pressure can be substituted for end-systolic pressure with a small but consistent error (22). There is experimental evidence that a similar end-systolic pressure-volume relation exists for the right ventricle (7). Our findings with control and nitroglycerin ventriculograms suggest, but do not prove, the presence of a similar relation in human beings. In our patients, end-systolic volume decreased with the decline in peak systolic pressure, but there was no change in the ratio of peak-systolic pressure to end-systolic volume. More definitive data could not be obtained because micromanometer-tipped catheters were used in only two of the patients because of the difficulty in obtaining high quality ventriculograms with the micromanometer catheters.

Conclusion. There is a strong negative correlation between right ventricular ejection fraction and pulmonary artery systolic pressure in patients with acquired valvular heart disease. In patients with decreased ejection fraction and elevated pulmonary artery pressure, decreasing pulmonary artery pressure with intravenous nitroglycerin resulted in improved ejection performance, implying that ventricular function would improve with a permanent decrease in afterload.

We thank James Scheuer, MD, and Hiltrud Mueller, MD, for their review of this manuscript, Norman Soloman, MD, and Robert Rosenblum, MD, for their advice and patience and Ms. Janet Holwell for secretarial assistance.

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