



## Characterization of the Human Right Ventricular Pressure-Volume Relation: Effect of Dobutamine and Right Coronary Artery Stenosis

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Right ventricular function was assessed in 15 patients using right ventricular pressure-volume loops. Right ventricular pressure using a micromanometer-tipped catheter, thermodilution cardiac output and gated blood pool scintigrams were simultaneously obtained. To help isolate the right ventricle, a slant hole collimator was used. The measurements were repeated during dobutamine infusion, which was titrated so there was minimal change in systemic pressure and heart rate.

The right ventricular pressure-volume loop resembles the usual left ventricular loop except that the isovolumic contraction phase is often not as distinct, and right-sided ejection may continue well beyond right ventricular peak systolic pressure. Systolic but not diastolic function improved with dobutamine administration. There was no significant difference in right ventricular systolic function (ejection fraction, stroke work index, stroke volume

index and cardiac index) or in end-diastolic volume index between patients without (Group I) and with (Group II) significant right coronary artery stenosis. However, there was a small but significant difference in right ventricular end-diastolic pressure ( $5.3 \pm 2.5$  and  $8.1 \pm 1.8$  mm Hg [ $p < 0.05$ ]) for Group I and II, respectively.

Thus, the right ventricular pressure-volume loop can be used to graphically display right ventricular function and improvement in contractility with dobutamine. The right ventricular isovolumic contraction phase and ejection phase differ from those in the usual left ventricular loop. Although there was a small difference in right ventricular end-diastolic pressure in patients with and without right coronary artery stenosis, the right ventricular pressure-volume loop did not provide additional discriminatory information between these two groups of patients.

Little quantitative information is available regarding right ventricular function in human beings, in large part because the complex geometric shape of the right ventricle is an impediment to volumetric determinations (1). In addition, right ventricular infarction and the importance of right ventricular function have only recently been recognized (2). Right ventricular dysfunction to varying degrees is common in acute myocardial infarction (2,3), especially with inferior left ventricular infarction. However, there has been relatively little work on right ventricular performance in the short- or long-term setting of ischemic heart disease.

The importance of left ventricular pressure-volume diagrams has long been recognized. Pressure-volume loops graphically describe the pressure and volume relations

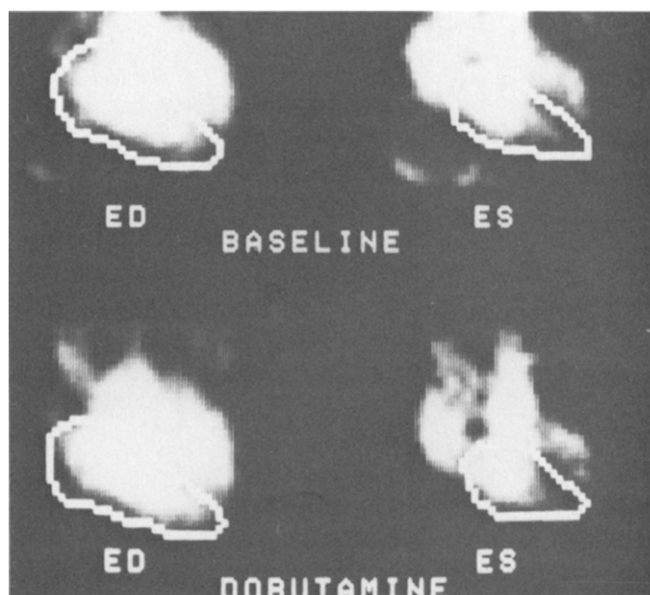
throughout the cardiac cycle and illustrate disturbances in left ventricular function. Characteristic changes in the left ventricular pressure-volume loop occur with various disease states (4). To make available similar information for the right ventricle, right ventricular volume from gated blood pool scintigrams and right ventricular pressure were measured simultaneously and used to construct a right ventricular pressure-volume loop. To help validate the technique, right ventricular pressure-volume loops were reconstructed during low dose dobutamine infusion to increase the contractile state. In addition, patients with right coronary artery stenosis were studied to determine if there were persistent changes in right ventricular systolic or diastolic function in patients with impaired blood flow to the right ventricle.

### Methods

**Patient characteristics.** Fifteen patients referred for cardiac catheterization with known or suspected coronary artery disease were studied. No patients with unstable angina, acute myocardial infarction or clinical evidence of

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**Figure 1.** Right ventricular regions of interest at end-diastole (ED) and end-systole (ES) in the baseline study (**upper panels**) and after dobutamine administration (**lower panels**).

congestive heart failure were included. Informed written consent was obtained from all patients. In one patient, right ventricular volume could not be determined from the gated blood pool study because of inability to isolate the right ventricle.

There were 12 men and 3 women with an average age of 53 years (range 37 to 74). Fourteen patients had coronary artery disease; the remaining patient had hypertensive heart disease and angina with normal epicardial vessels. Group I consisted of seven patients without significant (>70%) right coronary artery stenosis (three with previous anterior infarction). Group II consisted of eight patients with significant right coronary artery stenosis (six with previous inferior infarction). Previous infarction was documented by electrocardiogram, serum enzyme elevation and wall motion abnormalities.

All patients were being treated medically with various combinations of drugs including beta-receptor blocking agents, calcium channel blocking agents and nitrates. None of the patients had previous cardiac surgery. There was no significant difference in left ventricular function, as assessed by left ventricular ejection fraction and pulmonary capillary wedge pressure, between Groups I and II. Left ventricular ejection fraction was  $58 \pm 10\%$  (range 45 to 70) and  $57 \pm 10\%$  (range 41 to 74) for Group I and II, respectively. Pulmonary capillary wedge pressure was  $10 \pm 3$  mm Hg (range 6 to 17) and  $9 \pm 2$  mm Hg (range 7 to 12) for Group I and II, respectively.

**Cardiac catheterization.** After premedication with diazepam, 10 mg orally, each patient underwent right heart catheterization and coronary angiography using a percuta-

neous femoral approach. After coronary angiography, the right coronary catheter was pulled into the descending aorta to record arterial pressure. A balloon-tipped thermodilution catheter was introduced into the femoral vein and right heart catheterization was performed. This catheter was left in the pulmonary artery and a micromanometer-tipped catheter (Millar Instruments) was inserted percutaneously through a second puncture in the femoral vein and advanced into the right ventricle. It was calibrated in the right atrium to match the zero of the fluid-filled catheter at mid chest. Right ventricular pressure during several cardiac cycles was recorded using the micromanometer-tipped catheter in the early, mid and late portions of the gated blood pool scintigram.

**Cardiac output** was recorded with a miniature analog computer (Edwards Company). Multiple thermodilution cardiac output measurements were made during the gated blood pool scintigram, and the average of three to five determinations was used for the final cardiac output. For the 15 patients, the variability of thermodilution cardiac output measurements was  $1.9 \pm 1.3\%$ . The stroke volume was calculated by dividing the cardiac output by simultaneous heart rate.

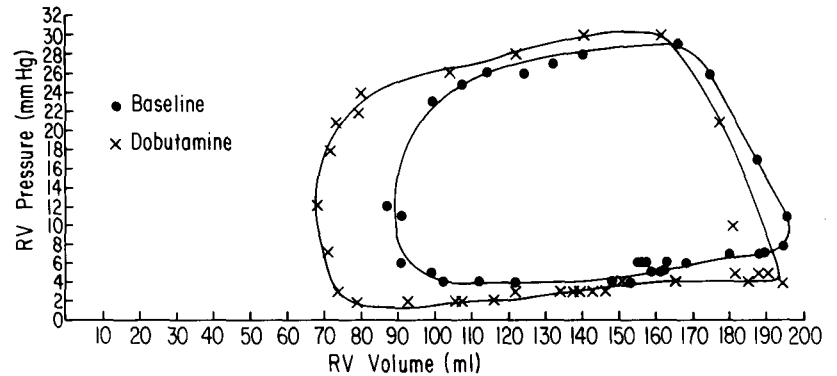
**Radionuclide data acquisition.** Modified in vivo red blood cell labeling (5) was performed with 25 mCi of technetium-99m pertechnetate. Studies were recorded with a portable Anger scintillation camera with a high resolution,  $30^\circ$  slant hole collimator. Routine anterior, left anterior oblique and left posterior oblique projections were obtained before transfer of the patient to the cardiac catheterization laboratory.

After completion of coronary angiography and right heart catheterization, the slant hole collimator was positioned in a  $10$  to  $15^\circ$  left anterior oblique projection with the collimator slant directed toward the cardiac apex along the axis of the ventricular septum. This was modified in each patient to best isolate the right ventricle from the right atrium and left ventricle (Fig. 1) (6). During data collection, the right atrial, right ventricular, pulmonary artery and systemic arterial pressures were recorded. Thermodilution cardiac output was measured as described at the midpoint of the data collection.

After completion of the baseline scintigram, without repositioning of the patient or camera, dobutamine (0.5 mg/ml) was infused through a sheath in the femoral vein. The dose was titrated so there was less than a 5% change in systemic arterial pressure and heart rate. Dobutamine was withheld after the baseline study from two patients, one with chest pain requiring sublingual nitroglycerin and the other with urinary retention. After the dobutamine infusion reached a steady state, the gated blood pool scintigram, hemodynamic measurements and cardiac output determinations were repeated.

**Data processing.** Images were collected and stored in frame mode on a digital computer (series 560, Technicare). The cardiac cycle was divided into 32 frames and counts

**Figure 2.** Patient from Group II. Right ventricular (RV) pressure-volume loops in baseline and dobutamine studies.



were collected for 100% of the cardiac cycle. Any RR interval exceeding  $\pm 20\%$  of the baseline RR interval resulted in rejection of the following cardiac cycles until the RR interval returned to the preselected range. Image sets were acquired for a predetermined information density of 150 counts/pixel over the right ventricle. The total acquisition time ranged from 5 to 10 minutes. All data were stored on magnetic discs for analysis. The scintigram was gated using standard electrocardiographic lead I.

To correct for respiratory-induced variations in the RR interval, a low intensity technetium-99m marker was placed in the corner of the field of view. This was used to normalize the last several (end-diastolic) frames if they had significantly ( $>10\%$ ) fewer counts in the region of interest surrounding the technetium-99m marker. Using the end-diastolic frame, a weighted interpolated background (7) image was constructed and subtracted from all 32 frames. For each background-corrected frame, a region of interest was drawn around the right ventricle and the number of counts in that region was determined.

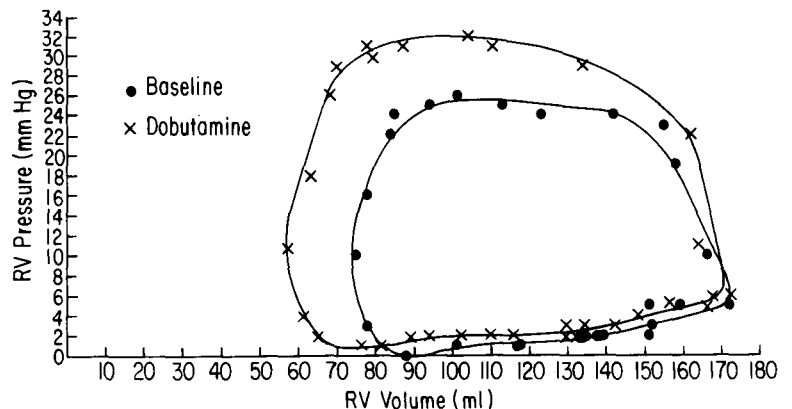
Count data derived from gated blood pool scintigraphy have been used to determine absolute left and right ventricular volumes (6,8,9). These volumes have been validated by correlations with angiographic end-diastolic and end-systolic volumes (8), as well as thermodilution-deter-

mined stroke volume (6,9). It has been shown that counts are directly proportional to blood volume (10). Thus, the number of right ventricular stroke counts, defined as the number of counts ejected during right ventricular systole (background-corrected end-diastolic minus background-corrected end-systolic counts) is proportional to the stroke volume (end-diastolic minus end-systolic volume). The stroke volume may be determined by dividing thermodilution cardiac output by the heart rate. Equating the stroke counts and stroke volume provides a proportionality constant or conversion factor to convert counts to volume (ml). The assumption that this factor is constant over the cardiac cycle allows the conversion of right ventricular counts to right ventricular volume for each of the 32 frames.

Acquisition of data into the first frame of each RR cycle of the gated study was initiated when the R wave reached two-thirds of its peak amplitude. The pressure recording was similarly divided into 32 equal segments between the two-thirds maximal point of sequential R waves of a simultaneously recorded lead I. Right ventricular pressure in each of the 32 segments from representative (early, mid and late) portions of the gated acquisition were averaged together.

The simultaneous pressure and volume measurements from each of the 32 frames were then plotted. Right ventricular stroke work index was obtained by planimetry of

**Figure 3.** Patient from Group I. Right ventricular (RV) pressure-volume loops in baseline and dobutamine studies.



the pressure-volume loop and dividing the value obtained by body surface area. Ejection fraction was defined as stroke counts divided by background-corrected end-diastolic counts.

**Statistics.** To determine the effect of dobutamine on systolic variables (including end-systolic volume index, ejection fraction and stroke work index) and diastolic variables (end-diastolic pressure and end-diastolic volume index), a paired *t* test was used in the seven patients in Group I (no significant right coronary artery stenosis) and independently in the eight patients in Group II (with significant right coronary artery stenosis). To compare the systolic and diastolic variables of Groups I and II, a one-tailed *t* test was used. A probability value less than 0.05 was considered significant. The average variability of the conversion factor (counts to volume) for the baseline and dobutamine studies was calculated for each patient.

**Validation of scintigraphic ejection fraction measurement.** The validity of right ventricular ejection fraction measurement by this technique has been established (6). To validate the technique in our laboratory, we compared the gated equilibrium right ventricular ejection fraction with that determined by a gated first pass technique (11) in 16 patients. These patients were first given unlabeled stannous pyrophosphate, followed 20 minutes later by a bolus injection of 25 mCi of technetium-99m pertechnetate. The volume of pertechnetate was less than 0.5 ml, and the delivery syringe was flushed with 20 ml of saline solution. Stannous pyrophosphate is required for the technetium-99m to bind to the red blood cells. Acquisition in the right anterior oblique projection was started when the persistence scope showed activity in the lower portion of the superior vena cava. Data were collected for 6 seconds at 15 frames/s and analyzed using commercially available software. After 10 minutes, the routine gated equilibrium study was performed in multiple projections including the 10 to 15° left anterior oblique view with the slant hole collimator directed toward the apex to isolate the right ventricle.

## Results

**Right ventricular pressure-volume diagram.** The correlation of the right ventricular ejection fraction determined by the equilibrium method with that derived from the gated first pass study was statistically significant ( $r = 0.85$ ,  $p < 0.001$ ). The absolute mean difference between the two measurements was 0.04. Figures 2 and 3 show two typical right ventricular pressure-volume diagrams and the response to dobutamine administration. These figures demonstrate that the right ventricular pressure-volume loop does not have an isovolumic contraction phase, that right ventricular volume begins to decrease early after the onset of the QRS while right ventricular pressure is still increasing and that right ventricular ejection (systole) continues well past right ventricular peak systolic pressure. Table 1 shows the mean

**Table 1.** Baseline Right Ventricular Systolic and Diastolic Variables in 15 Patients

	Mean $\pm$ SD	Range
End-diastolic volume (ml)	138 $\pm$ 34	101 to 213
End-diastolic volume index (ml/m <sup>2</sup> )	71 $\pm$ 16	47 to 110
End-systolic volume (ml)	57 $\pm$ 20	32 to 108
End-systolic volume index (ml/m <sup>2</sup> )	29 $\pm$ 10	17 to 56
Stroke index (ml/m <sup>2</sup> )	42 $\pm$ 7	27 to 54
Ejection fraction (%)	59 $\pm$ 5	49 to 73
Stroke work index (g·m/m <sup>2</sup> )	9.8 $\pm$ 1.9	6.7 to 13.1
Cardiac index (liters/min per m <sup>2</sup> )	2.3 $\pm$ 0.4	1.6 to 3.2

SD = standard deviation.

values and range of the systolic and diastolic variables of the 15 patients studied.

**Effect of dobutamine (Table 2).** The dose of dobutamine was  $4.6 \pm 2.2$   $\mu$ g/kg per min (mean  $\pm$  SD) (range 1.1 to 9.0). Figures 2 and 3 illustrate the typical increase in stroke volume and stroke work, decrease in end-systolic volume and lack of significant change in end-diastolic volume in patients who respond to dobutamine. In both Groups I and II, dobutamine administration resulted in a significant improvement in systolic function, including cardiac index, ejection fraction and stroke work index, without significantly changing diastolic variables. Pulmonary vascular resistance (difference between the mean pulmonary artery and capillary wedge pressures divided by cardiac output) decreased with dobutamine infusion in all five patients in whom it was measured. The average variability of the conversion factor from baseline to dobutamine was  $9 \pm 6\%$ .

**Effects of right coronary stenosis (Table 3).** There was no significant difference between Groups I and II in systolic variables, including end-systolic volume index, ejection fraction and stroke work index. There was no significant difference in end-diastolic volume index; however, there was a small but significant ( $p < 0.05$ ) difference in end-diastolic pressure ( $5.3 \pm 2.5$  and  $8.1 \pm 1.8$  mm Hg for Group I and II, respectively). No difference in the shape of the right ventricular pressure-volume diagrams for Group I versus Group II was demonstrated.

## Discussion

The complex geometric shape of the right ventricle and the long held belief that it was a passive conduit are two factors largely responsible for the scarcity of quantitative information on right ventricular function. Radionuclide techniques and, more recently, echocardiography have provided new means to investigate right ventricular function. Investigations have primarily involved right ventricular ejection fraction, and many of these investigations have shown right ventricular ejection fraction to be largely de-

**Table 2.** Effect of Dobutamine Administration on Right Ventricular Function in Groups I and II\*

	Group I (baseline/dobutamine)	Group II (baseline/dobutamine)
Heart rate	58 ± 11/59 ± 14 (NS)	55 ± 7/55 ± 7 (NS)
Cardiac output (liters/min)	4.3 ± 0.7/5.1 ± 1.3 (p<0.05)	4.6 ± 0.8/5.2 ± 0.9 (p<0.05)
Ejection fraction (%)	60 ± 3/67 ± 9 (p<0.05)	57 ± 4/65 ± 7 (p<0.05)
End-systolic volume index (ml/m <sup>2</sup> )	26 ± 7/21 ± 6 (p<0.05)	33 ± 12/26 ± 11 (p<0.05)
Stroke work index (g·m/m <sup>2</sup> )	9.6 ± 2.5/14.0 ± 4.7 (p<0.05)	10.1 ± 1.8/14.5 ± 3.2 (p<0.05)
End-diastolic volume index (ml/m <sup>2</sup> )	66 ± 13/64 ± 13 (NS)	75 ± 18/75 ± 18 (NS)
End-diastolic pressure (mm Hg)	5 ± 2/6 ± 3 (NS)	8 ± 2/9 ± 3 (NS)
Right atrial mean pressure (mm Hg)	6 ± 3/6 ± 2 (NS)	5 ± 2/5 ± 2 (NS)
Pulmonary artery systolic pressure (mm Hg)	28 ± 8/27 ± 5 (NS)	26 ± 4/29 ± 7 (NS)
Pulmonary artery mean pressure (mm Hg)	17 ± 3/15 ± 3 (NS)	15 ± 3/15 ± 4 (NS)

\*Group I = seven patients without significant right coronary artery stenosis; Group II = eight patients with significant right coronary artery stenosis. NS = not significant.

pendent on right ventricular afterload and, thus, not a reliable indicator of right ventricular contractility (12,13). Our aim was to study whether the pressure-volume diagram of the right ventricle provides quantitative and useful information regarding right ventricular function.

**Characteristics of the right ventricular pressure-volume loop.** The right ventricular pressure-volume diagram has a different shape from the rectangular left ventricular pressure-volume loop. The right ventricular loop is more trapezoidal with right ventricular ejection continuing past peak right ventricular systolic pressure, a phenomenon previously observed (14) in cross-circulated isolated canine hearts. We also have demonstrated that, unlike the left ventricular pressure-volume loop, the right ventricular pressure-volume loop lacks a distinct isovolumic contraction phase.

These findings may be explained by the low pressure, low impedance and high compliance of the pulmonary vasculature (14,15). The normal pulmonary gradient is positive in early to mid-systole, allowing the rapid onset of ejection of blood from the right ventricle. In later systole, even though the impulse gradient (right ventricular pressure - pulmonary artery pressure) becomes negative, the ejection of blood continues (16,17), implying that momentum of the blood coupled with the low resistance of the pulmonary vasculature is an important factor during late systole (18).

**Table 3.** Comparison of Right Ventricular Function in Patients Without Significant (Group I) and With Significant (Group II) Right Coronary Stenosis

	Group I	Group II
End-diastolic volume index (ml/m <sup>2</sup> )	66 ± 13	75 ± 18 (NS)
End-diastolic pressure (mm Hg)	5 ± 2	8 ± 2 (p<0.05)
Cardiac output (liters/min)	4.3 ± 0.7	4.7 ± 0.9 (NS)
Ejection fraction (%)	59 ± 4	59 ± 7 (NS)
End-systolic volume index (ml/m <sup>2</sup> )	27 ± 7	32 ± 11 (NS)
Stroke work index (g·m/m <sup>2</sup> )	9.5 ± 2.3	10.4 ± 17 (NS)

These right-sided characteristics represent a potential source of error if one uses pulmonary artery systolic pressure, as has been done previously (12), instead of right ventricular end-systolic pressure to study end-systolic right ventricular pressure-volume relations.

**Comparison with other methods.** Many approaches have been used to evaluate right ventricular performance. Early attempts (19,20) with indicator-dilution techniques overestimated right ventricular volumes. Angiographic methods for determining right ventricular volumes and ejection fraction have relied on various techniques, including Simpson's rule, area-length methods and different geometric models, as a basis for volume calculations. Echocardiographic methods are also being developed for volumetric determinations (21). Although a number of these methods have been validated with postmortem casts, the normal ranges have a wide variation (19,22-24). Our measurements of ejection fraction and end-diastolic, end-systolic and stroke volumes are consistent with values in these previous reports.

**Response to dobutamine.** The most prominent action of dobutamine, at moderate dose levels, is augmentation of myocardial contractility. This property has made it useful as an inotropic agent to improve left ventricular function in patients with heart failure (25). At the doses used, cardiac output is increased by an increase in stroke volume without a significant change in heart rate. The increase in stroke volume is secondary to improved contractility, with a decrease in end-systolic volume without a significant change in end-diastolic volume (26).

In evaluating the right ventricle of patients in this study, a similar response to dobutamine was demonstrated. Dobutamine caused an increase in contractility manifested by an increase in cardiac output, right ventricular ejection fraction and right ventricular stroke work. Right ventricular end-systolic volume and pulmonary vascular resistance decreased, and there was no significant change in right ventricular end-diastolic pressure or volume.

**Ventricular diastolic and systolic function with coronary disease.** Decreased left ventricular compliance accompanies both chronic ischemic heart disease and acute myocardial infarction (27). Compliance is a major determinant of left ventricular diastolic pressure and of the relation between right and left heart filling pressures. In patients with myocardial infarction, left atrial pressure measurements by transeptal catheterization do not correlate well with left ventricular end-diastolic volume. This lack of correlation is not surprising since an increase in left ventricular diastolic pressure may be secondary to decreased left ventricular compliance without any increase in left ventricular volume (28).

It has been reported (29) that ischemic right ventricular dysfunction is associated with an elevated right atrial pressure with a noncompliant pattern. Our findings suggest that a decrease in right ventricular compliance, as manifested by an increase in right ventricular end-diastolic pressure without a significant change in end-diastolic volume, may be a manifestation of ischemic disease of the right ventricle.

After an acute inferior infarction, right ventricular ejection fraction may return to normal within 10 days (30). Other patients may have persistent right ventricular systolic impairment (3). In the two groups of patients we studied with chronic ischemic heart disease (with or without left coronary artery stenosis), all had normal right ventricular systolic function and could only be distinguished on the basis of a small difference in their right ventricular end-diastolic pressure. These findings are compatible with previous studies (30,31) that showed no difference in right ventricular ejection fraction at rest between normal patients and those with right coronary artery stenosis. There are data suggesting that patients with right coronary artery stenosis may have diminished right ventricular systolic function during exercise (32,33).

**Reliability and sources of error.** The accuracy for the thermodilution technique for cardiac output determination is well established (34). Right ventricular stroke volume using this method may be in error in the presence of right-sided regurgitation or intracardiac shunting. Auscultation, intracardiac oximetry and hemodynamic measurements excluded shunts and right-sided regurgitant lesions in our series.

For the optimal determination of right ventricular volumes, it is important to maximize the separation of the right ventricle from surrounding structures. The projection used in our study was shown to be the best of several studied (6). Regardless, in some patients it was more difficult to completely isolate the right ventricle and in one patient the right ventricle was not successfully isolated. An arbitrary assumption of all radionuclide quantitation is background selection. As noted, we used a weighted interpolative background (7). If the right ventricle is well isolated and background properly selected, the radionuclide technique may be used to view the right ventricle three-dimensionally and

to accurately assess changes in right ventricular blood volume. The limited average variability of the conversion factor (counts/ml) from the baseline to the dobutamine study suggests that this procedure for determination of volumes is reliable and reproducible. Correction for radioactive decay between the baseline and dobutamine studies should reduce the variability further.

*Slight variations in the RR interval due to respiratory-induced changes in the heart rate* during the 5 to 10 minute collection of the gated study necessitates making some minor approximations regarding the end-diastolic portion of the volume curve. In most patients, occasional RR intervals were slightly shorter than the mean RR interval, resulting in fewer counts in the last 2 to 5 frames (frames 28 to 32). These few frames were normalized, as described, with the low activity radioactive marker. This does result in some estimation of the exact counts and, thus, volume in these end-diastolic frames.

**Clinical utility.** In the clinical setting it is often important to quantitate the basal level of right ventricular contractility and to assess the effect on contractility of various therapeutic interventions. In the isolated muscle preparation, it is possible to sort out the individual influences of preload, afterload and contractility on cardiac performance by holding any two of these three variables constant and varying the third. However, in patients, preload and afterload are not as easily controlled, making it difficult to assess contractility. This is particularly true in the right ventricle where ejection fraction is extremely afterload-dependent.

The ability of the pressure-volume loop to sort out changes in preload and afterload and define left ventricular contractility by an end-systolic pressure-volume line has been documented (35). We have shown that a radionuclide assessment of right ventricular volumes using gated blood pool scintigraphy at the time of cardiac catheterization may be used to construct right ventricular pressure-volume loops and graphically display right ventricular performance. We have not evaluated the use of the end-systolic pressure-volume relation to assess right ventricular contractility, and the utility of this measurement in the right ventricle remains a question. To study the end-systolic pressure-volume relation, two or more interventions (either a change in preload or afterload) is necessary for each contractile state. The time necessary for these multiple interventions in this group of patients was prohibitive. However, a different experimental design may be used to study the right ventricular end-systolic pressure-volume relation.

**Conclusion.** The right ventricular pressure-volume diagram provides a graphic means of comparing right ventricular function and therapeutic interventions. All of our patients were in stable condition at the time of evaluation without evidence of severe right ventricular dysfunction or other abnormalities of right-sided circulation. Additional studies of patients with more significant right ventricular

dysfunction will be needed to determine the clinical utility of this technique.

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