Angiographic Estimation of Right Ventricular Volume in Man

By Richard D. Gentzler, II, M.D., Michael F. Briselli, B.A.,
And James H. Gault, M.D.

SUMMARY

Although the feasibility of angiographic estimation of right ventricular (RV) volume (V) has been demonstrated, no method has been validated by comparison with independent measurements of volume, and none has been applied to the systematic analysis of RVV characteristics in man. In the present study, postmortem casts of the human RV were used to determine a regression analysis for RVV for biplane frontal and lateral films by the Simpson's rule technique (true volume = 0.749 calculated volume, r = 0.99, see 3.7 ml, for casts ranging in volume from 20 to 115 ml). This method was then employed to estimate RVV from biplane cineangiograms of 32 patients. The validity of RVV measurements in vivo was corroborated by close correlation of stroke volumes estimated independently by angiographic and indicator dilution methods (r = 0.98, see 4.1 ml).

In nine patients with normal RV hemodynamics RV end-diastolic volume (EDV) averaged 81 ml/m² (range 63 to 101 ml/m²); ejection fraction (EF) averaged 0.51 (range 0.40 to 0.66), values generally lower than the normal left ventricular EF. Of 14 patients with elevated RV systolic pressure (> 45 mm Hg), normal RV function was evidenced in eight by normal levels of EF and EDV; in the remaining six patients, EF was reduced and EDV was elevated, suggesting that dilatation occurred in these patients as a function of depressed RV myocardial performance. In six patients with left-to-right shunts RV EDV was also increased; however, in these patients normal levels of EF were observed, indicating that the accommodation to volume overload was accomplished through the Starling mechanism. Finally, in six of 13 patients with reduced levels of cardiac output, RV EDV was increased and EF reduced, signifying that impaired RV myocardial function contributed to depression of over-all cardiac performance. It is concluded that the biplane angiographic method provides a practical, accurate means of quantitating RVV characteristics, including the magnitude of volume overload as well as the level of RV myocardial performance.

Additional Indexing Words:
Cineangiograms Myocardial performance End-diastolic volume

VOLUME ESTIMATION by angiography provides the only method by which it is possible to measure accurately ventricular end-diastolic volume (EDV) and total stroke volume (SV), and thus to quantitate the magnitude of volume overload and valvular regurgitation, as well as to characterize ventricular pump performance. While this technique is now employed widely in the assessment of left ventricular function, the feasibility of angiographic measurements of right ventricular volume has only recently been established. Indeed, no systematic examination of the alterations in right ventricular (RV) performance resulting from mechanical overloading of various types has been previously reported in adults.

The present studies were undertaken first to examine rigorously the accuracy of a biplane angiographic method of right ventricular volume (RVV) measurement in vivo, and second, using this method, to determine the effects on RVV characteristics of mechanical overloading and of depressed RV myocardial function.

Methods

A regression analysis for estimation of RVV from angiographic silhouettes was derived in the following manner: silicone rubber casts* of the human RV cavity were made in 12 subjects at the time of postmortem examination. Roentgenograms of the casts were then exposed in the frontal and lateral projections. Chamber silhouettes were then drawn to outline each view. In so doing, and subsequently in obtaining angiographic silhouettes, no effort was made to follow the pattern of trabeculation precisely. Instead, the silhouette was drawn as a smoothed line to include all con-

*RTV-11, General Electric Company.
RIGHT VENTRICULAR VOLUME

In all studies, 75% sodium diatrizoate (Hypaque) 1 ml/kg was injected into the right atrium over a period of 2 sec. Right atrial injection was used in preference to RV injection of contrast material to avoid premature ventricular contractions. Biplane cineangiograms were exposed at 60 or 100 frames/sec in the frontal and lateral projections. While some difficulty in determining the ventricular silhouette was encountered in an occasional patient because of superimposition of the opacified right atrium, this problem proved to be a minor one. In preliminary studies the injection of contrast material into the RV chamber was virtually always accompanied by ventricular extrasystoles, which impose serious limitations on the physiologic validity of the volume measurements. During the angiogram, the instant of each radiographic exposure was recorded with the arterial pressure pulse and electrocardiogram on an oscillographic recorder which permitted precise correlation of angiographic data and electrical and pressure events. RV silhouettes were drawn in the frontal and lateral projections at end-diastole, determined by using the time interval between the Q wave of the electrocardiogram and the inscruma of the RV pressure pulse recorded immediately prior to the cineangiogram, and at end-systole, taken as the smallest RV chamber size during contraction. Corresponding frames were used in the two projections. Stroke volume was calculated as the difference between EDV and end-systolic volume (ESV), and ejection fraction (EF) was calculated as the ratio SV/EDV. Effective RV SV was derived for each patient from an indicator dilution (In-

Contrast material, following the perimeter of the trabeculae. Using the Simpson's rule method of analysis, the respective frontal and lateral silhouettes were divided transversely into an equal number of segments or slices one-half centimeter in thickness (fig. 1). Each segment was represented as an elliptical cylinder and its volume computed using diameters measured directly from the silhouettes, corrected for X-ray magnification as described for X-ray magnification. Total RVV was then derived as the sum of the volumes of all segments

\[ V_{RV} = \frac{\pi h}{4} \sum V_{segment} = \frac{\pi h}{4} \sum (D_{AP} + D_{LAT}) \]

The regression analysis relating the cast volume calculated by this method to the true volume determined by water displacement, is illustrated in figure 2. This analysis demonstrated a close correlation between calculated and true volumes \((r = 0.99)\), with a small standard error \((\text{SEE} = 3.7 \text{ml})\) for casts ranging in volume from 20 to 115 ml. Volumes were calculated from angiographic studies then corrected by this regression equation \((V_{true} = 0.749V_{calc})\) to derive true volumes.

Biplane cineangiograms were then employed to estimate RV volumes in 32 patients (33 studies) undergoing diagnostic cardiac catheterization, using the method outlined above. All patients were in sinus rhythm at the time of study. These included two patients with rheumatic mitral valve disease, one patient with aortic stenosis, and one patient with coronary artery disease, in whom this additional study could not be regarded as part of the diagnostic procedure. In these patients, and in the eight patients with rheumatic mitral valve disease who were not suspected clinically of having tricuspid regurgitation, informed consent was obtained for the additional RV angiographic study. In the remaining 21 patients the RV angiogram was considered part of the diagnostic evaluation. The clinical diagnoses of these patients are shown in table 1.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

The right ventricular cavity silhouette is shown in frontal and lateral radiographic projections to illustrate the method of dividing the chamber into horizontal segments for calculation of volume by the Simpson's rule method. A representative slice, 0.5 cm in thickness \((h)\), is shown at the lower left with its major and minor diameters \((D)\).

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

The calculated volume from 12 human postmortem chamber casts of the right ventricle is plotted on the vertical axis, with corresponding measurements of true volume determined by water displacement on the horizontal axis. The regression analysis relating these measurements is shown at the lower right with correlation coefficient \((r)\), standard error of the estimate \((\text{SEE})\) and is also indicated by the oblique dashed line.

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**Table 1**

**Hemodynamic and Volumetric Data in All Patients**

**Abbreviations:**
- Asy. H = asynergic hypertrophy
- Atp. MR = atrophic mitral regurgitation
- AS = aortic stenosis
- CAD = coronary artery disease
- TV = tricuspid valve
- P.M.D. = pulmonic stenosis
- T.P.M.D. = tricuspid insufficiency
- RV = right ventricular
- LV = left ventricular
- E.D.V. = end-diastolic volume
- E.S.V. = end-systolic volume
- SV = stroke volume
- EF = ejection fraction
- SV = stroke volume
- EF = ejection fraction
- Stroke volume = (end-diastolic volume - end-systolic volume)
docyanine green) measurement of cardiac output obtained immediately (within 30 sec) prior to the cineangiogram. In three patients with left-to-right shunt, those with atrial septal defect, pulmonary blood flow was calculated from paired pulmonary arterial indicator dilution curves using the forward triangle method. Right ventricular stroke volume was then derived as pulmonary blood flow divided by heart rate, and this measurement was compared with the angiographic estimate of SV.

**Results**

Hemodynamic and volume data in all patients are shown in table 1.

The accuracy of in vivo estimates of RVV was examined by comparing measurements of RV SV derived angiographically with those obtained by the indicator dilution method (fig. 3) in 23 patients in whom a left-to-right shunt and tricuspid regurgitation could be excluded. The accuracy of the angiographic estimates of SV is indicated by the close correlation between these measurements (r = 0.98, SEE = ± 4.1 ml).

Nine patients with normal pulmonary arterial and right heart pressures were considered to have normal RV function. In these individuals EDV normalized for body surface area ranged from 63 to 102 ml/m² (mean = 81 ± 12.3) and EF ranged from 0.40 to 0.66 (mean 0.51 ± 0.08). These measurements are contrasted with the normal ranges of left ventricular volume measurements after Kennedy et al.² in figure 4.

Thirteen patients had RV pressure overload (RV systolic pressure > 45 mm Hg); in 11, pulmonary arterial, and thus RV systolic, hypertension was related to rheumatic mitral valve disease, while in two patients, one of whom had two RV angiograms, there was congenital pulmonary stenosis (PS). Neither RV EDV or EF bore any relation to the level of RV systolic pressure (fig. 5). However, in seven (5 mitral stenosis, 2 PS) of the eight patients with normal levels of EF, EDV was normal. In the remaining six patients, in whom EF was reduced, EDV was increased in five (fig. 6). Four patients in the latter group had tricuspid regurgitation as well, with regurgitant fractions of 29 to 44%. These observations suggested that RV dilatation resulted from depression of RV myocardial function and perhaps associated tricuspid regurgitation (fig. 6), not from RV pressure overload.

In five of six patients with a large left-to-right shunt, RV EDV and SV were substantially increased. In the three patients with atrial septal defect, measurements of RV SV derived from pulmonary arterial dye curves correlated closely with angiographic estimates of SV, the maximum

![Figure 3](http://circ.ahajournals.org/)

Angiographic (angio) measurements of right ventricular stroke volume (SV) are plotted on the vertical axis with corresponding measurements derived by the indicator dilution method (IND) on the horizontal axis in 23 patients without tricuspid regurgitation or left-to-right shunt. The oblique line indicates identity of measurements, while the equation of the lower right expresses the mathematical relation between these measurements derived by the least squares regression analysis.

![Figure 4](http://circ.ahajournals.org/)

Values for end-diastolic volume, normalized for BSA (m²), (left panel), and ejection fraction (right panel) are indicated by the closed circles for nine patients in whom performance of right ventricle (RV) was considered to be normal. The normal range of measurements for the left ventricle (LV) is shown by the cross-hatched area on the right of each panel.
difference between measurements being 6%. In each instance the increase in SV was accompanied by a parallel increase in EDV, which indicated that the requirement for an increased RV SV was met through the Starling mechanism (fig. 7). Levels of EF in these patients, ranging from 0.45 to 0.64, were comparable to the normal group. In six patients in whom RVV overload resulted from tricuspid regurgitation (regurgitant fractions 29–44%), EDV was also significantly increased (94–201 ml/m²). However, as indicated above, and in contrast to patients with left-to-right shunt, EF was reduced in these patients, which suggests that RV dilatation was not the result of tricuspid regurgitation alone but reflected the presence of associated impaired RV myocardial performance as well.

In many forms of acquired heart disease selective left heart abnormalities are responsible for reduced cardiac output. Because RV SV is dependent on the level of venous return, which is determined by SV of the left ventricle, it is necessary to distinguish the changes in RV pump characteristics where a reduced RV stroke volume is the result of left heart disease, from those associated with a primary abnormality of the RV myocardium. Accordingly, the relationship between EF and EDV was examined in 13 patients in whom cardiac output, and thus RV SV, was reduced. In seven patients, RV function was presumably normal, as evidenced by levels of EF exceeding 0.40 (range 0.40 to 0.66). Each of these patients had a normal EDV (range 54 to 95 ml/m²) (fig. 8). In five patients in whom impaired RV function was suggested by reduced levels of EF (range 0.06 to 0.40), EDV was increased in all but one patient (range 94 to 201 ml), while in one patient with an EF of 0.40, the RV EDV was elevated to 133 ml/m². This patient exhibited significant tricuspid regurgitation. The association of reduced levels of EF with ventricular dilatation in the latter group suggested that a primary abnormality of the RV myocardium contributed to the reduced levels of SV, whereas in the former group, reduced RV myocardial function appeared to result from limited preload and therefore is presumably secondary to left heart disease.

Discussion

The use of angiography as a means of estimating volume of the left ventricle in man was originally
based on the assumption that this chamber could be represented as a conventional geometrical form, specifically, an ellipsoid. Indeed, the shape of the left ventricular chamber has proven to be sufficiently predictable so that its volume can be estimated with reasonable accuracy from silhouettes obtained in one radiographic projection. Conversely, when single plane volume estimates are in error it can be shown that these errors are the consequence of deviations from this predicted chamber morphology. In contrast, the cavity silhouette of the right ventricle does not correspond to a conventional geometric form. Accordingly, rather than attempting to model the cavity per se for purposes of volume calculation, it is appropriate to employ the Simpson’s rule method of analysis, where slices of the chamber are made perpendicular to a long axis which is common or parallel to both frontal and lateral planes. The slices or segments of the chamber are then assigned a geometric form, in this instance that of elliptical cylinders, the major and minor diameters of which can be measured directly from frontal and lateral films of biplane cineangiograms. The validity of this approach has been proven in previous studies in canine right ventricular casts as well as in postmortem casts of the human RV chamber by Graham et al. It will be noted that the regression analysis relating true volume to calculated volume of RV casts in this study differs from that reported by Graham et al. This difference appears to be explained by the assumption of an ellipsoidal form for ventricular slices by Graham et al., as compared to the elliptical cylinder model employed in this study.

No angiographic method of ventricular volume estimation is susceptible to direct validation in vivo. Previous investigators have attempted to assess the accuracy of angiographic estimates by a comparison of the closeness of left and right ventricular stroke volume measurements. While these investigators have demonstrated agreement between measurements of the respective ventricular SVs, inaccuracies could result from small amounts of left-sided valvular regurgitation, and perhaps changes in heart rate and blood pressure between the two measurements. In the present study, angiographic estimates of RV SV were compared with those derived independently using the indicator dilution method. The close correlation demonstrated between these measurements provides further substantive evidence for the high degree of accuracy and reproducibility of the angiographic method for RVV estimation.

Other investigators have suggested that the area-length method provides a simpler means of deriving RVV which is more readily employed in manual calculation than the more cumbersome Simpson’s rule method. While these observers have demonstrated a correlation between calculated and true chamber volume using this method, it has been shown to be significantly less accurate than the Simpson’s rule method. In addition, this approach lacks a sound mathematical basis. The area-length method was employed initially by Dodge et al. in the estimation of left ventricular volume because of the empirical observation that, by deriving values for the minor semiaxis of the cavity from its area and length rather than attempting to measure this semiaxis directly, the reproducibility of angiographic volume estimates was greatly improved. Underlying this area-length method is the assumption of an ellipsoidal form for the ventricular cavity, an assumption which would appear invalid when applied to the calculation of RVV.

The findings of the present study suggest that the normal right ventricle operates at a slightly greater EDV (81 ± 12 ml/m²) than has been reported for the normal left ventricle (70 ± 20 ml/m²), while EF is somewhat lower in the right than in the left ventricle (0.51 ± 0.08 vs 0.67 ± 0.16). Similar observations were made in children by Thilenius and Arcilla. However, measurements in a larger group of patients will be required in order to establish the range of normal values for this chamber with certainty.

It was observed that in patients with pulmonary arterial hypertension, RV EDV remained within the normal range when EF was normal (fig. 6). Only when EF fell below levels observed in patients with normal

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**Figure 8**

Right ventricular end-diastolic volume is plotted on the vertical axis with measurements of cardiac index on the horizontal axis in nine patients with reduced levels of ejection fraction (open circles) and in 15 patients in whom ejection fraction was normal (closed circles). The vertical dashed line indicates the lower limit of normal for cardiac index.

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right ventricles did EDV increase. This suggests that dilatation occurred as a function of depressed RV myocardial performance rather than because of pressure overload per se. Thus, while the right ventricle has been considered primarily a volume pump, it appears to be capable of accommodating a substantial chronic pressure overload without dilatation. These observations are also consistent with those of the studies of children with RV pressure overload related to pulmonary arterial hypertension by Graham et al.\(^7\) However, in patients in previous studies with acute right heart overload, severe RV failure has been shown to occur at relatively low levels of pulmonary arterial hypertension, suggesting that the right ventricle may not be able to accommodate acute pressure overload as readily.\(^10\)

In contrast to the findings in RV pressure overloading in this study, substantial RV dilatation occurred in each patient in whom RVV overload resulted from a left-to-right shunt. Furthermore, in this relatively small group of patients, it was apparent that the degree of RV dilatation, i.e., the level of EDV, bore a close relation to the magnitude of volume overload, as estimated from the level of angiographically estimated RV SV. In each of those patients normal levels of EF were observed.

Finally, while the role of beat-to-beat changes in resistance to emptying as a determinant of RV performance must be further characterized, it is apparent that angiographic volume measurements provide a potentially useful clinical tool in assessing the adequacy of RV myocardial performance. It is apparent that RV SV must be reduced in some patients as the consequence of isolated left heart abnormalities with reduced systemic blood flow and systemic venous return. It might be anticipated under these circumstances that the Starling mechanism would operate to account for reduced RV SV through reduced RV preload, i.e., EDV. In the present study, patients in whom depressed SVs were associated with normal EDVs, the levels of EDV were generally the lowest observed, and were associated with normal levels of EF. This suggests that reduced preload was, indeed, responsible for reduced RV SV. In contrast, patients with similarly reduced levels of systemic cardiac output, in whom RV SV was reduced while EDV was increased, always exhibited reduced levels of EF, which suggests that impaired RV myocardial performance contributed to reduction of cardiac output. Observations in the present study indicate that substantial reductions in RV myocardial function, as evidenced by depressed levels of EF, may occur, for example, in patients with rheumatic mitral valve disease, sufficient to limit the improvement in over-all cardiac pump performance anticipated with correction of the mechanical defect. These findings suggest that depressed RV myocardial function may be an important determinant both of the clinical features and of the outcome of surgical treatment in many forms of acquired and congenital heart disease.

References

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RICHARD D. GENTZLER II, MICHAEL F. BRISCELLI and JAMES H. GAULT

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