Right Ventricular Performance in Patients with Coronary Artery Disease

By Jack Ferlinz, M.D., Richard Gorlin, M.D., Peter F. Cohn, M.D., and Michael V. Herman, M.D.

SUMMARY
While left ventricular (LV) performance in patients with coronary artery disease (CAD) has been extensively investigated, little attention has been given to right ventricular (RV) function in this disease. For this purpose, a new geometric model for RV volume has been developed and RV end-diastolic volume index (EDVI), end-systolic volume index (ESVI), stroke volume index (SVI) and ejection fraction (EF) have been determined from biplane RV cineangiograms in 26 patients.

Eight patients served as normal (control) subjects (group I). Eighteen patients with obstructive CAD comprised two other groups: six who had no significant disease of the right coronary artery (RCA) (group II) and 12 who had a high grade RCA lesion (group III). The mean values for EDVI, SVI and EF in group I were 76 ± 11 ml/m², 50 ± 6 ml/m², and 66 ± 6%. The only significant difference between groups I and II was that SVI was lower in group II than in group I (P < 0.01). No measurements in groups II and III were statistically different from each other. However, markedly subnormal values were found in group III (EDVI: 61 ± 16 ml/m², SVI: 33 ± 10 ml/m² and EF: 52 ± 7%); all values being significantly lower (SVI and EF: P < 0.001; EDVI: P < 0.05) than in group I. RV end-diastolic pressure was normal in all patients. These findings may be related to 1) reduced RV compliance, 2) distorted LV geometry, 3) possible RV ischemia or 4) reduced Frank-Starling effect.

LEFT VENTRICULAR GEOMETRY, volume and contractile parameters in normal hearts have been analyzed in great detail within the last 15 years. In addition, the effects of coronary artery disease on left ventricular performance have also been extensively studied. In contrast, much less attention has been given to the performance of the right ventricle in either normal subjects or patients with coronary artery disease. This has been due partially to the fact that the left ventricle was always considered the more important chamber of the heart. The relative neglect of right ventricular performance has been further compounded by difficulties in analyzing the geometry of the right ventricular chamber: while the left ventricular configuration approximately resembles an ellipsoid of revolution (and therefore renders itself to relatively simple mathematical analysis), the right ventricle has always been considered a somewhat freeform shell that does not yield to simple geometric manipulations. Whenever routine geometric analyses were attempted, relatively poor correlations between the actual and mathematically derived volumes were obtained. Attempts by various investigators to overcome this problem have involved the use of Simpson’s rule which is sufficiently laborious to virtually necessitate computer analysis. Such facilities often are not available in the standard cardiac diagnostic laboratory.

In the present report a new geometric model for the right ventricle is proposed. With this model, relatively simple, reproducible and accurate measurements and calculations of ventricular volume from the biplane right ventriculograms can be obtained, and normal subjects can be compared with patients with coronary artery disease.

Materials and Methods

Casts of Right Ventricle

Autopsied intact human hearts of various sizes without any significant gross abnormalities were used to prepare casts. A silicone disk, fitted with a cannula for injection of plastic compound, was sutured into the tricuspid valve orifice. The pulmonary artery was clamped just above the pulmonary valve. Dow-Corning Silastic A-RTV Mouldmaking Rubber treated with RTV-4 catalyst was then injected through the cannula. Filling pressure was maintained at approximately 15 cm H₂O. The filled heart was then suspended by sutures from a small stand, and the compound inside the right ventricle allowed to harden in this suspended state in order to minimize any potential distortion of the right ventricular chamber.

From the Cardiovascular Division, Department of Medicine, Peter Bent Brigham Hospital and Harvard Medical School, Boston, Massachusetts.

Supported by USPHS Grants PO1 HL 11306 and IT1 HL 5679.

Dr. Herman is presently Chief, Division of Cardiology, and Dr. Gorlin Chairman, Department of Medicine, Mt. Sinai School of Medicine, New York, New York.


Address for reprints: Jack Ferlinz, M.D., Chief, Cardiac Catheterization Laboratory, Veterans Administration Hospital, 5901 East Seventh Street, Long Beach, California 90801.

Received December 6, 1974; revision accepted for publication May 21, 1975.
Nine right ventricular (RV) casts were obtained (fig. 1). Actual volume (Va) was determined by immersing each cast into a graduated beaker five times, establishing the displaced volume, and averaging the results. Thirty degree right anterior oblique (RAO) and sixty degree left anterior oblique (LAO) cineangiograms of each densely radiopaque cast were then obtained (fig. 2). These projections allow for the interventricular septum to be parallel to the film in the 30 degree RAO projection, while in the LAO projection the septum is perpendicular to the film and is viewed end on. Films of the casts were then projected on a screen and their outlines traced. A 1 × 1 cm grid (filmed at the mid-level of each ventricular cast) was used for calibration purposes. RAO and LAO outlines for each ventricular cast were then planimeted and the long axis in the RAO projection was determined.

The mathematical model for the right ventricular volume is predicated on the assumption that the right ventricular chamber resembles a pyramid with a triangular base (fig. 3). Area_{abc} (A_{abc}) closely approximates the 30 degree RAO projection of the right ventricular casts, and area_{aoe} (A_{aoe}) the simultaneous 60 degree LAO projection. Therefore,

\[ A_{abc} = A_{RAO} \]
\[ A_{aoe} = A_{LAO} \]
\[ ab = d_{RAO} \]
\[ ad = d_{LAO} \approx \text{height (h\textsubscript{aoe}) of the area of base (A}_{aoe} \]
\[ h' = l_{RAO} \]
\[ h'' = l_{LAO} \approx h^* \]

As the area of the triangle is given as (length\textsubscript{base}) × (height)/2, area of base (A_{abd}), d_{RAO} and d_{LAO} can be approximately expressed as

\[ A_{abd} = \frac{d_{RAO} \times d_{LAO}}{2} \quad (1) \]
\[ d_{RAO} = \frac{2 \times A_{RAO}}{l_{RAO}} \quad \text{and} \]
\[ d_{LAO} = \frac{2 \times A_{LAO}}{l_{LAO}} \quad \text{respectively.} \quad (3) \]

Substituting:

\[
\text{volume (V)} = \frac{A_{abd} \times h}{3} = \frac{d_{RAO} \times d_{LAO}}{3} \times l_{LAO} = \\
\frac{2 \times A_{RAO} \times A_{LAO}}{l_{RAO} \times l_{LAO}} \times l_{LAO};
\]

and the expression for the right ventricular volume reduces to

\[ V = \frac{2}{3} \times \frac{A_{RAO} \times A_{LAO}}{l_{RAO}} \quad (4) \]

Right ventricular volume can thus be simply derived by only the following three actual measurements: the planimetry of the areas of RAO and LAO projections, and the assessment of the length of RAO (fig. 4). l_{RAO} is obtained by dividing the projected RAO image with a connecting line between the bisected pulmonic valve and the bisected base.

Calculated right ventricular volumes (V\textsubscript{c}) were then com-
pared to the actual volumes ($V_a$); and correlation coefficient ($r$), regression equation for corrected volume ($V_{corr}$), standard error of estimate (SEE), mean percent error ($E$), where percent error ($E$) is given as

$$E = \frac{V_{corr} - V_a}{V_a} \times 100,$$

and standard deviation of error (SDE) were determined.

**Patient Population**

Biplane cine right ventriculograms in 30° RAO and 60° LAO projections were performed in 26 patients. All gave informed consent and underwent diagnostic catheterization including complete left and right heart hemodynamic evaluation, selective left ventriculography, and coronary arteriography. None had systemic hypertension, overt congestive heart failure or significant valvular or congenital lesions. All patients were suspected of having coronary artery disease.

**Figure 3**

Geometric model (a pyramid) of right ventricular chamber.

 Fifty-five to 65 mL of meglumine sodium diatrizoate (Renografin-76) dye was injected directly over a 3 to 4 second interval into the RV chamber through a No. 8 Eppendorf catheter in each case, and cineangiograms exposed at 100 frames/sec. Although premature ventricular contractions (PVCs) were frequently encountered, there were always a sufficient number of normal sinus beats (NSBs) for adequate analysis. At least two successive NSBs (following a PVC) had to occur before a frame was selected for volumetric analysis. A typical frame (patient KG) is presented in figure 5.

Right ventricular cineangiograms were first carefully screened for any evidence of asynomy, or any other abnormality of right ventricular contraction. Optimal end-systolic and end-diastolic RV images were then selected and calibrated with a 1 × 1 cm grid filmed at the right ventricular level for each individual patient. Outermost trabeculations were included in the tracing of the projected images. The end-systolic shape was generally an almost identical smaller version of its end-diastolic counterpart. The exact pulmonary valve position was usually easy to identify in both views. End-diastolic volume index (EDVI), end-systolic volume index (ESVI), stroke volume index (SVI), and ejection fraction (EF = SV1/EDVI) were then calculated from the traced outlines for each patient.

When left ventricular (LV) cineangiograms were performed in the standard manner, at least 60 minutes were allowed to elapse between the two injections in order to minimize the effects of the contrast material on myocardial contractility and peripheral circulation. A modification of the area-length method of Dodge was used in analysis of left ventricular volumes.

Coronary artery anatomy was evaluated in all 26 patients studied. Significant coronary artery disease (CAD) was considered to be present only when there was at least 75% occlusion of at least one of the three coronary arteries, and when this observation was confirmed by at least three independent observers.

**Results**

**Casts of Right Ventricle**

An extremely close correlation between the calculated ($V_c$) and the actual ($V_a$) volumes was obtained even before the adjustment of the calculated volumes by the regression equation ($V_{corr}$). The calculated uncorrected volumes ranged from 38 mL to 162 mL; the largest deviation from the actual volumes was an overestimation of 23 mL. The error, usually involving an overestimation, increased with larger volumes while smaller calculated volumes correlated exceptionally well with their actual counterparts. The correlation coefficient for the uncorrected volumes was 0.98.

A plot of actual volumes against the calculated volumes is presented in figure 6. A regression equation was calculated from this plot to obtain the optimal correlation and to correct for the minor approximations used in the derivation of the RV model.

$V_{corr}$ is then given by

$$V_{corr} = 0.893 V_a + 3.862; \quad (5)$$

by approximating and substituting into expression (4); the right ventricular volume ($V_{RV}$) becomes

![Figure 4](https://example.com/figure4.png)

RAO and LAO tracings of the cineangiograms presented in figure 2.
RV PERFORMANCE IN CAD

The corrected volumes now range from 38 ml to 149 ml and the correlation coefficient is $r = 0.99$. Standard error of estimate (SEE) is 5.7 ml, mean percent error (E) 0.4%, and standard deviation of error (SDE) ±6.5%.

Patient Studies

Of the 26 patients studied, eight had no significant CAD and served as "normal" or control subjects (group I). Eighteen patients had obstructive CAD. Of these, six (group II) had no significant disease of the right coronary artery (RCA). The remaining 12 patients (group III) had a significant obstruction of the RCA, with or without two or three vessel disease.

The complete results for all three groups are found in tables 1, 2 and 3; their mean values (± 1 sd) are illustrated in figure 7. Frame by frame motion analysis of RV revealed no areas of obvious asynergy in any patient within the three groups studied.

The highest mean values for RV EDVI, SVI and EF were found in group I and the lowest in group III. Thus in the subgroup with CAD and RCA involvement, the RV EDV, SV and EF were significantly reduced compared with the control group ($P < 0.05$, $<0.001$ and $<0.001$, respectively). ESVI was essentially the same for all three groups, as was the filling pressure of the right ventricle (RVEDP). The only significant difference between groups I and II was found in SVI ($P < 0.01$). None of the measurements in groups II and III differed significantly. Poor resolution during the levophase part of the right ventricular injection prevented accurate analysis of simultaneous left ventricular volumes and ejection fraction.

Discussion

The early attempts to estimate right ventricular EDV, ESV and SV were made by employing the indicator-dilution method with indocyanine green.23

---

**Figure 5**

_Biplane right ventricular cineangiograms._

**Figure 6**

_Actual versus calculated right ventricular cast volumes._

**Figure 7**

_Mean right ventricular values (± 1 sd) for groups I, II and III._

---

Circulation, Volume 52, October 1975
or the thermal or radioisotope indicators. These, as well as some of the later efforts using indicator-dilution methodology, grossly overestimate the ventricular volumes.

It is by now well established that the error of the angiographic method (with the appropriate regression equation) falls within the ±20% range in the LV volume analysis. No comparable firm standards are yet available for the RV cineangiographic volume indices, however, with a great variability in values published to date. The relatively simple mathematical model developed in our laboratory does away with the need for the use of Simpson's rule, yet possesses a high degree of accuracy (r = 0.99). Concern has been expressed that injections directly into the RV 1) will "unnaturally" distend the chamber and therefore increase the SV by the Frank-Starling mechanism, 2) may alter myocardial contractility because radiopaque contrast material is pharmacologically active, and 3) will cause so many PVCs that a valid analysis will be impossible. These objections have been answered by previous investigators. Finally, uncontrolled groups of PVCs can be avoided by careful positioning of the RV catheter midway in the RV outflow tract.

The RV volumes for our control (normal) group were similar to the normal values described for the LV by Dodge. While it can be argued that normal RV EDV, ESV and EF do not have to be identical to their LV counterparts, the SV of both ventriciles, aside from minor transient readjustments, must be the same. RV SVI of group I (normal) patients was 50 ± 6 ml/m², correlating closely with the value that Dodge established for the LV SVI (45 ± 13 ml/m²).

In comparing the RV angiographic stroke volume to its LV counterpart, the levophase analysis of the RV injection would theoretically appear to be an ideal approach because it almost instantaneously reflects the performance of both ventricular chambers, and so minimizes the variations in the constantly changing physiological parameters during cardiac catheterization. Unfortunately, resolution of the levophase is often suboptimal and volumes calculated from it differ from volumes calculated following direct LV injection of contrast material.

Little is known about the extent of permanent or even transient RV dysfunction that can be induced by CAD, and whether there are alterations similar to those described for the LV. The common changes in LV function, usually encountered in the early stages

### Table 1

**Summary of Clinical Findings — Group I (Controls)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>MI</th>
<th>Coronary obstruction</th>
<th>RV volumes (ml/m²)</th>
<th>EF (%)</th>
<th>RVEDP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LZ</td>
<td>57</td>
<td>F</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>99 34 65</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>DM</td>
<td>41</td>
<td>F</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>79 29 63</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>EC</td>
<td>53</td>
<td>F</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>71 24 66</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>RB</td>
<td>33</td>
<td>F</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>79 27 66</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>RF</td>
<td>32</td>
<td>M</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>72 29 41</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>50</td>
<td>M</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>63 13 79</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>TS</td>
<td>53</td>
<td>M</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>75 25 66</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>FF</td>
<td>42</td>
<td>M</td>
<td>no</td>
<td>RCA LAD LCF</td>
<td>76 ± 11 26 ± 6 50 ± 6 66 ± 6</td>
<td>3.4 ± 1.8</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: MI = myocardial infarction; RCA = right coronary artery; LAD = left anterior descending coronary artery; LCF = left circumflex coronary artery; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; SVI = stroke volume index; EF = ejection fraction (SVI/EDVI); RVEDP = right ventricular end-diastolic pressure.

### Table 2

**Summary of Clinical Findings — Group II (CAD without RCA Disease)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>MI</th>
<th>Coronary obstruction</th>
<th>RV volumes (ml/m²)</th>
<th>EF (%)</th>
<th>RVEDP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BM</td>
<td>49</td>
<td>M</td>
<td>AS</td>
<td>no yes no</td>
<td>75 30 45</td>
<td>60</td>
<td>1</td>
</tr>
<tr>
<td>VM</td>
<td>48</td>
<td>F</td>
<td>AS</td>
<td>no yes no</td>
<td>65 26 38</td>
<td>59</td>
<td>1</td>
</tr>
<tr>
<td>WB</td>
<td>50</td>
<td>M</td>
<td>AS</td>
<td>no yes yes</td>
<td>62 24 38</td>
<td>61</td>
<td>1</td>
</tr>
<tr>
<td>AF</td>
<td>51</td>
<td>M</td>
<td>no</td>
<td>no yes yes</td>
<td>41 13 28</td>
<td>68</td>
<td>2</td>
</tr>
<tr>
<td>RD</td>
<td>46</td>
<td>M</td>
<td>no</td>
<td>no yes no</td>
<td>72 30 42</td>
<td>38</td>
<td>4</td>
</tr>
<tr>
<td>KC</td>
<td>49</td>
<td>M</td>
<td>AS</td>
<td>no yes yes</td>
<td>66 ± 12 25 ± 6 39 ± 6 61 ± 4</td>
<td>2.0 ± 1.3</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AS = anteroseptal.
of CAD, are an elevated EDP with a normal EDV. and minimal (if any) decrease in EF and SV. With more extensive focal damage or ischemia, LV asynergy supervenes, EDV almost invariably increases and SV decreases. Finally, a grossly deteriorated LV generally exhibits a greatly augmented EDV, a more extensive focal damage LV in one case in the patients with CAD (RVEDP — group II: 2.0 ± 1.3 mm Hg; group III: 3.4 ± 2.1 mm Hg). Unfortunately, data pertaining to LV compliance cannot be adapted a priori to RV studies, especially when RVEDP measurements alone are subject to so many variables (e.g., changes in intrathoracic pressures, catheter whip, status of pulmonary circulation, etc.). Furthermore, a low end-diastolic volume (as seen in group III) per se may be associated with a low filling pressure regardless of ventricular compliance, solely as a function of the intrinsic volume-pressure relationship of the ventricle. The interplay of all these factors is so complex that the exact status of RV compliance in patients with CAD remains unanswered.

Second, it is possible that a diseased LV may adversely affect RV performance and bring about changes in RV volume-pressure characteristics. This influence may be exerted not only by the interventricular septum which is comprised of a thin right and a relatively thick left muscle mass, but also by the circular and spiral bundles of muscle fibers which encircle both ventricles. Oboler et al. attempted to show that the RV pumping function is aided by an inferior left ventricle. They found that RV pacing (i.e., induced LBBB) produced an early peak in right ventricular dP/dt followed by a second peak which occurred at approximately the same time as peak left ventricular dP/dt — which presumably was the LV contribution to the pumping action. Unfortunately, the work of Wallace et al. demonstrated that ventricular pacing itself intrinsically affected dP/dt, thus making interpretation of these findings difficult.

Many investigators have shown that volume-pressure relationships are highly dependent upon the state of filling of the contralateral chamber. Some of our patients in group III (with the most extensive CAD) had greatly increased left ventricular EDV. The RV adjusts to such a grossly distended LV in one of two ways either 1) the RV volume and sarcomere length necessary to maintain the original level of RV function is associated with an abnormally elevated RVEDP, or 2) (as it appears to be the case in this study) the RV volume at normal filling pressures is decreased.

Third, it is possible that CAD (and especially RCA lesion) significantly impairs RV function directly through ischemia or prior infarction. This appears to be relatively unlikely in the patients we studied because no obvious RV asynergy was detected, and diminished EF and SV were accompanied by low filling volumes and normal filling pressures. It is remotely possible, however, that the impaired coronary blood supply to the RV myocardium depresses
over-all RV contractility to a degree, but not markedly
enough to activate the Frank-Starling mechanism
with augmentation of RV EDV and EDP. This
hypothesis is based on the fact that the nonstressed
RV performs mostly "shortening" (external)\(^6\) work
which requires relatively little energy expenditure.
Brooks et al\(^6\) have shown that a total occlusion of
RCA in dogs, while causing a drop in RV contractility,
elicits no change in right or left ventricular pressures
or aortic flow as long as the RV is not further stressed
by marked pulmonary hypertension.

Fourth, decreased RV EF and SV in group III may
be due to a decreased venous return\(^7\) and therefore an
attenuation of the RV Frank-Starling effect.\(^6\)\(^8\) Such
an adjustment may occur secondary to reduced LV
output when its function is severely impaired. It is
concluded that in most cases CAD probably does not
intrinsically influence RV function per se in the
resting state. With advanced degrees of CAD, par-
ticularly when there is involvement of RCA, reduction
in RV EDV, SV and EF occurs.

Acknowledgment

The critical comments of Dr. E. H. Sonnenblick are gratefully
acknowledged. Further, the authors wish to acknowledge the
assistance of Dr. J. A. Levine and Mr. D. Bechtel for their con-
tributions in preparation of many of the cineangiograms and right
ventricular casts, Ms. M. Holder for her efforts in the assembly
of graphic material, and Ms. J. Richardson and S. Savoy for their ex-
tensive secretarial help.

References

1. Chapman CB, Baker O, Beynolds J, Bondy FJ: Use of blip-
eneurofluorography for measurement of ventricular volume.
Circulation 18: 1105, 1958

2. Dodge HT, Sandler H, Ballweh DW, Lord JD: The use of
blipeneuroangiography for the measurement of left ventric-

3. Chapman CB, Baker O, Mitchell JH, Collier RG: Ex-
periences with cinefluorographic method for measuring ven-
tricular volume. Am J Cardiol 18: 25, 1966

4. Dodge HT, Sandler H, Baxley WA, Hawley RR: Usefulness
and limitations of radiographic methods for determining left
ventricular volume. Am J Cardiol 18: 10, 1966

5. Davila JC, Sanmarco ME: An analysis of the fit of
mathematical models applicable to the measurement of left
ventricular volume. Am J Cardiol 18: 31, 1966

6. Sandler H, Dodge HT: The use of single plane angi-
cardiograms for the calculation of left ventricular volume in

7. Dodge HT, Baxley WA: Left ventricular volume and mass
and their significance in heart disease. Am J Cardiol 23: 528,
1969

8. Kennedy JW, Trenholme SE, Kasner LS: Left ventricu-
lar volume and mass from single plane cineangiogram. A com-
parison of anteroposterior and right anterior oblique
methods. Am Heart J 80: 343, 1970

9. Dodge HT: Determination of left ventricular volume and mass.

10. Gorlin R, Klein MD, Sullivan JM: Prospective correla-

11. Herman MV, Heisler RA, Klein MD, Gorlin R: Localized
disorders in myocardial contraction. N Engl J Med 227: 222,
1967

12. Herman MV, Gorlin R: Implications of left ventricular
asymmetry. Am J Cardiol 23: 538, 1969

13. Baxley WA, Reeves TJ: Abnormal regional myocardial
performance in coronary artery disease. Prog Cardiovasc Dis
13: 405, 1971

angiography in ischemic heart disease. Circulation 40:
1065, 1972

15. Cohn PF, Gorlin R, Adams DF, Chahine RA, Voronov PS,
Herman MV: Comparison of biplane and single plane left
ventriculograms in patients with coronary artery disease. Am J
Cardiol 33: 1, 1974

method for volume estimation of right and left ventricles.
Chest 60: 446, 1971

17. Reddy T, Chapman CB: Measurement of right ventricular
volume by cineangiography. Am Heart J 66: 221, 1963

18. Godke R, Carlsson E: Calculations of right and left cardiac
ventricular volumes. Invest Radiol 2: 560, 1967

19. Graham TP, Jarmakani JM, Atwood GF, Canent RV: Right
ventricular volume determinations in children. Circulation
47: 144, 1973

20. Gentzler RD, Briselli MF, Gault JH: Angiographic
estimation of right ventricular volume in man. Circulation
50: 324, 1974

21. Deslens DT, Kadell BM, Ruttenberg HD, Goldberg SJ,
Macalpin RN: Angiographic determination of the ventricu-

22. Mullins CB, Leshin SJ, Mierzwa DS, Alsobrook HD,
Mitchell JH: Changes in left ventricular function produced
by the injection of contrast media. Am Heart J 83: 373, 1972

23. Freid ED, Rivara GL, Gilmore BL: Estimation of residual
diastolic volumes of the right ventricle of men without
heart disease, using the dye-dilution method. Am Heart J
60: 898, 1960

BD: Right ventricular volumes in patients with and without

25. Lewis ML, Giunti C, Donato L, Harvey RM, Courand A:
Quantitative radiocardiography. III. Results and validations

26. Lee SJ, McClelland AR, Zaragoza AJ: The right ventricular
function during exercise in patients with and without right

27. Swan HJC: Problems in the measurement of ventricular

volume correlation of the two cardiac ventricles in man.
Invest Radiol 6: 44, 1971

29. Gentzler RD, Gault JH, Briselli MF: Angiographic
estimation of right ventricular volume (RVV) in man. (abstr)
Am J Cardiol 31: 134, 1973

30. Minsky I, Ghita DN, Sandler H: Cardiac mechanics:
Physiological, clinical, and mathematical considerations.
New York, John Wiley, 1974, p 145

31. Hallerman FJ, Rastelli GC, Swan HJC: Effects of rapid
injection of heparinized blood into right and left ventricles
in dogs. Radiology 83: 647, 1964

32. Hammermeister KE, Warbaas J: Immediate hemodynamic
effects of cardiac angiography in man. Am J Cardiol 31: 307,
1973

33. Vine DL, Dodge HT, Stewart DK, Murbay JA: Immediate
effect of contrast injection on the left ventricular volume.
(abstr) Am J Cardiol 33: 175, 1974
42. Wade WG: The pathogenesis of infarction of the right ventricle. Br Heart J 21: 545, 1959
45. Ruckle CE, Russell RO: Right ventricular function in acute myocardial infarction. Am J Cardiol 33: 927, 1974
51. Smith M, Ratshin RA, Hambell FE, Russell RO, Ruckley CE: Early sequential changes in left ventricular dimensions and filling pressure in patients after myocardial infarction. Am J Cardiol 33: 363, 1974
58. Oboier AA, Keefe GF, Gaasch WH, Banas JS, Levine HJ: Influence of left ventricular isovolumic pressure upon right ventricular pressure transients. Cardiology 58: 32, 1973
Right ventricular performance in patients with coronary artery disease.
J Ferlinz, R Gorlin, P F Cohn and M V Herman

Circulation. 1975;52:608-615
doi: 10.1161/01.CIR.52.4.608
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/52/4/608

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/