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 < 0.01; 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
Castst 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.

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Nine right ventricular (RV) casts were obtained (fig. 1). Actual volume ($V_a$) 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.\(^{21}\)

Films of the casts were then projected on a screen and their outlines traced. A $1 \times 1$ 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 planimetered 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$_{abcd}$ ($A_{abcd}$) the simultaneous 60 degree LAO projection. Therefore,

\[
A_{abcd} = A_{RAO} \\
A_{abcd} = A_{LAO} \\
d_{RAO} = d_{LAO} \\
d = d_{LAO} \approx \text{height (} h_{ab}\text{) of the area of base (} A_{abcd}\text{)} \\
h' = l_{RAO} \\
h'' = l_{LAO} \approx h
\]

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

\[
A_{abcd} = \frac{d_{RAO} \times d_{LAO}}{2} \\
d_{RAO} = \frac{2 \times A_{RAO}}{l_{RAO}}; \text{ and} \\
d_{LAO} = \frac{2 \times A_{LAO}}{l_{LAO}} \text{ respectively.}
\]

Substituting:

\[
V = \frac{A_{abcd} \times h}{3} = \frac{d_{RAO} \times d_{LAO}}{3} \times l_{LAO} = \frac{2 \times A_{RAO} \times 2 \times A_{LAO}}{l_{RAO} \times l_{LAO}} \times l_{LAO};
\]

and the expression for the right ventricular volume reduces to

\[
V = \frac{2}{3} \frac{A_{RAO} \times A_{LAO}}{l_{RAO}}
\]

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 $l_{RAO}$ (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 ($V_c$) were then com-

\footnote{h'' (or $l_{LAO}$) is substituted as an approximation of h or true height of the pyramid. Due to the convex curvature of base and greater height than diameter of the pyramid, the difference between the two is trivial.}

Figure 1

Right ventricular cast.

Figure 2

Cineangiograms of the cast in figure 1 in the RAO (left panel) and LAO (right panel) projections.
pared to the actual volumes ($V_a$); and correlation coefficient ($r$), regression equation for corrected volume ($V_{corr}$), standard error of estimate ($s_e$), 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 ($s_dE$) 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 asynery, or any other abnormality of right ventricular contraction. Optimal end-systolic and end-diastolic RV images were then selected and calibrated with a $1 \times 1$ 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 = SVI/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 Ventricule

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.

$$V_{corr} = 0.893 V_e + 3.862; \quad \text{or} \quad (5)$$

by approximating and substituting into expression (4); the right ventricular volume ($V_{RV}$) becomes
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.$^{23}$
or the thermal\(^4\) or radioisotope\(^5\) indicators. These, as well as some of the later efforts using indicator-dilution methodology,\(^5\) grossly overestimate the ventricular volumes.\(^27\)

It is by now well established that the error of the angiographic method (with the appropriate regression equation) falls within the \(\pm 20\%\) range in the LV volume analysis.\(^27\) No comparable firm standards are yet available for the RV cineangiographic volume indices, however, with a great variability in values published to date.\(^19, 28, 29\)

The relatively simple mathematical model developed in our laboratory does away with the need for the use of Simpson's rule,\(^30\) 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.\(^31, 33\) 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.\(^9\) 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 ventricles, aside from minor transient readjustments, must be the same.\(^17, 34, 35\) RV SVI of group I (normal) patients was \(50 \pm 6 \text{ ml/m}^2\), correlating closely with the value that Dodge established for the LV SVI \((45 \pm 13 \text{ ml/m}^2)\).\(^9\)

In comparing the RV angiographic stroke volume to its LV counterpart, the levophase analysis\(^6\) 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.\(^19, 28\) Unfortunately, resolution of the levophase is often suboptimal\(^37, 48\) and volumes calculated from it differ from volumes calculated following direct LV injection of contrast material.\(^39, 40\)

Little is known about the extent of permanent or even transient RV dysfunction that can be induced by CAD,\(^41, 46\) 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 (\text{ml/m}^2)</th>
<th>EF ((%))</th>
<th>RVEDP ((\text{mm Hg}))</th>
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<tr>
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<td>F</td>
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<tr>
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<td>75 25 50</td>
<td>66 5</td>
<td></td>
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</tbody>
</table>

Mean \((\pm 1 \text{ sd})\) 45 \(\pm 10\) 76 \(\pm 11\) 26 \(\pm 6\) 50 \(\pm 6\) 66 \(\pm 6\) 3.4 \(\pm 1.8\)

**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 \((\text{SVI/EDVI})\); 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 (\text{ml/m}^2)</th>
<th>EF ((%))</th>
<th>RVEDP ((\text{mm Hg}))</th>
</tr>
</thead>
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<tr>
<td>KC</td>
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<td>AS</td>
<td>no yes yes yes</td>
<td>66 27 40</td>
<td>60 3</td>
<td></td>
</tr>
</tbody>
</table>

Mean \((\pm 1 \text{ sd})\) 49 \(\pm 2\) 64 \(\pm 12\) 25 \(\pm 4\) 39 \(\pm 6\) 61 \(\pm 4\) 2.0 \(\pm 1.3\)

**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, variably elevated EDP, and severely depressed EF and SV.

Such a pattern was not observed in the RV. Patients in group III with the lowest mean values for EF and SV had, contrary to expectations, a decreased EDV and a normal RVEDP.

Several possible explanations can be offered for these findings. First, decreased EDV could be attributed to a poorly distensible (noncompliant) RV, secondary to either ischemia or fibrosis resulting from the myocardial underperfusion distal to the RCA lesions. Various methods and parameters have been proposed to measure (LV) compliance. In virtually all cases, a noncompliant ventricle was associated with elevated diastolic filling pressure — a finding that was not observed in either of our two groups of 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, 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 insidious 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)84 work which requires relatively little energy expenditure. Brooks et al.65 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 return66 and therefore an attenuation of the RV Frank-Starling effect.67, 68 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, particularly when there is involvement of RCA, reduction in RV EDV, SV and EF occurs.

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