Assessment of Right Ventricular Anatomy and Function by Quantitative Radionuclide Ventriculography

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Determination of right ventricular ejection fraction and volumes from radionuclide studies is cumbersome and is subject to considerable methodologic error. Further, assessment of regional wall motion has only infrequently been approached in a systematic way. A system of right ventricular ejection fraction and volume measurements is described that utilizes the previously validated single plane geometric method applied to first pass radionuclide angiocardiograms. Five right ventricular chords were defined and used to assess regional wall motion; normal values were obtained from 14 patients who were without demonstrable cardiac disease.

Among 23 patients with anterior myocardial infarction, the right ventricular ejection fraction was within 2 SD of normal in 16; however, 3 of these patients showed regional wall motion abnormalities in the right ventricle. Among 23 patients with anterior myocardial infarction, the right ventricular ejection fraction was within 2 SD of normal in 16; however, 3 of these patients showed regional wall motion abnormalities in the right ventricle. Of 21 patients with inferior myocardial infarction, right ventricular ejection fraction was reduced in 15; of the 6 with normal values, 3 had regional wall motion abnormalities as demonstrated by the chord shortening method. Of 21 patients with dilated cardiomyopathy, right ventricular function was abnormal in 20; the presence of a wall motion abnormality in the conus segment separated these patients from patients with right ventricular dysfunction after recent myocardial infarction. Thus: 1) right ventricular ejection fraction, volumes and wall motion can be assessed by a simple, geometric technique; 2) analysis of chord shortening by this method provides information unavailable from global ejection fraction data alone; and 3) the clinical correlates of these data will require further investigation.

Methods

Study patients. Seventy-nine patients were randomly selected from a group referred to our laboratory for first pass radionuclide studies for routine clinical indications. Each gave consent for presentation of data in collective form. Group I comprised 14 normal subjects who were referred for radionuclide studies for a variety of clinical indications but in whom extensive evaluation failed to demonstrate the presence of cardiac disease.
**Group II comprised 44 patients with a first Q wave acute myocardial infarction.** The infarction was anterior in 23 patients and inferior in 21, as defined by standard electrocardiographic (ECG) criteria. Radionuclide studies in this group were obtained 2 to 18 days after hospital admission. Group III comprised 21 patients who had stable dilated cardiomyopathy in association with cardiomegaly, typical symptoms and signs of congestive heart failure, left ventricular ejection fraction <40% and no clinical or ECG evidence of recent myocardial infarction. The mean age of the 79 patients was 58 ± 15 years (range 25 to 80) and did not differ among groups.

**Radionuclide data collection and analysis.** First pass studies were obtained in the 30° right anterior oblique view after the administration of 15 to 25 mCi of technetium-99m-labeled human serum albumin through a large proximal vein. Data acquisition was gated to the R wave of the ECG. Precordial activity was monitored using an Anger camera (Searle LEM) equipped with a medium sensitivity collimator. A ventriculogram was reconstructed by summing appropriate 20 ms frames from two to four cardiac cycles, with registration of both end-diastolic and end-systolic time points, as has been previously described for the left ventricle (5,6). After completion of the first pass study, a gated equilibrium study was acquired in the best septal left anterior oblique view with 10° caudal angulation. Sixteen frames per cycle were obtained with approximately 350,000 counts per frame. Both studies were displayed in endless loop cine format.

*The quality of these images has been described in detail previously (8,9)* with use of a count profile across the data matrix to define background and edge characteristics. The absence of significant radiotracer in other thoracic regions during right ventricular transit and the steepness of the profile made edge definition during first pass studies highly satisfactory for this chamber. Intra- and interobserver variability in measurements of volumes was assessed retrospectively in a random subset of patients and were similar, with r values approximating 0.9 and a coefficient of variation of <8%.

**Regional wall motion analysis.** Regional wall motion was studied in all patients by manual definition of ventricular outlines from a video monitor calibrated by use of point sources 10 and 20 cm apart. Figure 1 illustrates the right ventricular segments and chords that were analyzed. In the right anterior oblique view, the walls were anterior (between the pulmonary valve plane and the apex), inferior (between the apex and the tricuspid valve plane), and crista (between the tricuspid and pulmonary valve planes). Five segments were defined: cristal, anterior, apical, inferior and tricuspid. These and the conus segment were used to ascertain the extent of regional wall motion. On both end-diastolic and end-systolic silhouettes, five chords were plotted, as follows: A, from the mid point of the cristal segment to the apex; B, from the mid-pulmonary valve to the mid-inferior wall; C, from the mid-tricuspid valve to the superior aspect of the apical one-third of the anterior wall; D, defined in the discussion to follow; and E, bisecting and perpendicular to the interventricular septum in the left anterior oblique view. The segments in the latter view were defined as lateral, septal and inferior (Fig. 1). Percent shortening was computed for each chord, assuming a fixed frame of reference between end-diastole and end-systole. Chord D was a conus chord utilized to assess the extent of wall motion in the outflow tract of the right ventricle. This chord is parallel to and 1 cm (life-size) below the pulmonary valve plane. Circumferential contraction of the distal outflow tract was assessed from the percent shortening of this chord.

*The end-diastolic and end-systolic volume indexes of the left ventricle were computed with standard single plane right anterior oblique angiographic formulas (10). Right ventricular volumes were similarly measured with the method of Ferlinz (3). Ejection fraction was calculated as stroke volume divided by end-diastolic volume.*

**Statistical methods.** Data are expressed as mean values ± SD. The significance of differences between groups was assessed by analysis of variance and Bonferroni t statistics with use of BMDP software (11).

**Results**

**Ventricular volumes and ejection fraction (Table 1). In normal subjects, left ventricular ejection fraction averaged 71 ± 6%; end-diastolic and end-systolic volumes were 60 ± 17 and 18 ± 6 ml/m², respectively. Right ventricular ejection fraction averaged 67 ± 5%; corresponding end-diastolic and end-systolic volumes were 63 ± 15 and 21 ± 6 ml/m², respectively.**
Table 1. Ventricular Volumes and Ejection Fractions in Clinical Groups (n = 79)

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Patients</th>
<th>Heart Rate (beats/min)</th>
<th>End-Diastolic Volume (ml/m²)</th>
<th>End-Systolic Volume (ml/m²)</th>
<th>Ejection Fraction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>RV</td>
<td>LV</td>
<td>RV</td>
<td>LV</td>
</tr>
<tr>
<td>Group I:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal subjects</td>
<td>14</td>
<td>75 ± 14</td>
<td>63 ± 15</td>
<td>60 ± 17</td>
<td>21 ± 6</td>
</tr>
<tr>
<td>Group II:</td>
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</tr>
<tr>
<td>Anterior M.I.</td>
<td>23</td>
<td>80 ± 12</td>
<td>52 ± 15</td>
<td>70 ± 23</td>
<td>21 ± 8</td>
</tr>
<tr>
<td>Inferior M.I.</td>
<td>21</td>
<td>73 ± 17</td>
<td>64 ± 17</td>
<td>67 ± 22</td>
<td>30 ± 9</td>
</tr>
<tr>
<td>Group III:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>21</td>
<td>90 ± 19*</td>
<td>73 ± 31</td>
<td>118 ± 40†</td>
<td>42 ± 21†</td>
</tr>
</tbody>
</table>

*p < 0.05; †p < 0.01 compared with normal subjects. Data are expressed as mean ± SD. LV = left ventricle; M1 = myocardial infarction; RV = right ventricle.

In patients with acute anterior myocardial infarction, left ventricular ejection fraction was markedly reduced (37 ± 11%, p < 0.01) largely because of increased end-systolic volume (45 ± 21 ml/m², p < 0.05). In this group of patients, there was a nonstatistically significant trend toward reduction in right ventricular end-diastolic volume and ejection fraction.

In patients with inferior myocardial infarction, however, left ventricular ejection fraction was less profoundly reduced (53 ± 10%, p < 0.01) because of a lesser increase in its end-systolic volume. Right ventricular function was significantly affected, with a decrease in ejection fraction to 53 ± 11% (p < 0.05). This change appeared to be due to increased right ventricular end-systolic volume from 18 ± 6 to 31 ± 12 ml/m².

In patients with dilated cardiomyopathy, there was, as expected, a profound reduction in biventricular function with marked increases in left ventricular volumes. Right ventricular end-systolic volume, but not end-diastolic volume, was markedly greater than in normal subjects.

Right ventricular wall motion data (Table 2). In normal subjects, the greatest relative motion occurred in the tricuspid valve plane and the least in the anterior and inferior segments. In patients with acute anterior myocardial infarction, diminished contraction was noted only in the inferior segment; this reduction was small (10.8 ± 4.5% versus 14.2 ± 3.1%) but significant at the p < 0.05 level. In patients with inferior myocardial infarction, reduced contraction was seen only in the inferior segment as well, but the magnitude of the reduction was much greater (3.1 ± 4.2% versus 14.2 ± 3.1%, p < 0.01). A trend for reduction in chord shortening was noted in the anterior, apical, lateral and tricuspid segments as well, but did not reach statistical significance for any of these. In patients with dilated cardiomyopathy, significant reductions in segmental wall motion were found in the cristal, anterior, apical and inferior segments. A significant decline in chord shortening in the conus portion of the right ventricle was also found.

Abnormal chord shortening, defined as >2 SD below the mean value of the normal subjects (Group I), was found in the cristal and conus segments, principally in patients with dilated cardiomyopathy (Group III: 24 and 57% of patients, respectively); only rarely were these segments affected by either anterior or inferior myocardial infarction (Group II: 0 and 4.5%, respectively). The inferior segment was involved much more frequently in inferior (81%) than anterior (13%) segments.

Table 2. Right Ventricular Percent Chord Shortening in Clinical Groups (n = 79)

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Patients</th>
<th>Cristal</th>
<th>Anterior</th>
<th>Apical</th>
<th>Inferior</th>
<th>Tricuspid</th>
<th>Lateral</th>
<th>Conus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I:</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Normal subjects</td>
<td>14</td>
<td>17.5 ± 4.6</td>
<td>13.6 ± 4.3</td>
<td>19.6 ± 8.1</td>
<td>14.2 ± 3.1</td>
<td>22.4 ± 5.3</td>
<td>18.9 ± 4.8</td>
<td>38.9 ± 6.9</td>
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<tr>
<td>Group II:</td>
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<tr>
<td>Anterior M.I.</td>
<td>23</td>
<td>16.5 ± 3.7</td>
<td>14.5 ± 9.2</td>
<td>20.6 ± 8.8</td>
<td>10.8 ± 4.5†</td>
<td>19.8 ± 5.7</td>
<td>19.7 ± 7.1</td>
<td>36.5 ± 8.5</td>
</tr>
<tr>
<td>Inferior M.I.</td>
<td>21</td>
<td>16.4 ± 4.5</td>
<td>11.4 ± 5.7</td>
<td>11.8 ± 8.0</td>
<td>3.1 ± 4.3†</td>
<td>16.1 ± 7.4</td>
<td>19.8 ± 9.0</td>
<td>41.9 ± 6.6</td>
</tr>
<tr>
<td>Group III:</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>21</td>
<td>13.0 ± 5.2†</td>
<td>4.1 ± 4.9‡</td>
<td>8.0 ± 5.4‡</td>
<td>5.8 ± 3.3‡</td>
<td>16.9 ± 5.7</td>
<td>15.1 ± 5.9</td>
<td>24.3 ± 10.4‡</td>
</tr>
</tbody>
</table>

*p < 0.05; †p < 0.01 compared with normal subjects. Data are expressed as mean ± SD. Abbreviations as in Table 1.
myocardial infarction; the tricuspid and lateral segments were occasionally affected by inferior infarction (each in 38% of patients) but only rarely by anterior infarction (each in 4%).

Of the 65 patients with cardiac disease (Groups II and III), 23 had a right ventricular ejection fraction >57% (a value 2 SD below the mean of the normal group). Of these, however, seven patients (30%) demonstrated abnormal regional right ventricular wall motion, analyzed by individual segment. In these seven, the abnormalities were in the inferior segment in five, the lateral segment in three and the conus and tricuspid regions in one each. By contrast, of the 42 patients with an abnormal right ventricular ejection fraction, only 2 (5%) manifested normal regional wall motion. These two patients had a right ventricular ejection fraction of 54 and 55%, respectively. Thus, regional wall motion measurements showed many more abnormalities than did global ejection fraction measurement alone in our study group.

Discussion

Structure of the right ventricle. The right ventricular chamber is bounded by a free wall and a thick walled septum that occupies most of the posterior and medial surface of the chamber. It may be appreciated as a tetrahedron or bellows whose base lies between the superomedially oriented pulmonary conus and the laterally lying tricuspid valve apparatus (12). Across the base of the bellows is the crista supraventricularis, a thick muscle bundle that closes the bellows during normal contraction. The remaining walls are thin and highly trabeculated, with the trabeculae acting as chords to assist in bellows closure during systole. Thus, unlike findings in the more spherical left ventricle, wall motion observed in one region may be the result of muscle contraction occurring at some distance from that wall. Additionally, motion of the interventricular septum is the complex result of many influences, including the volume of blood in each chamber, relative ventricular contractility and the attachments of the heart by the great vessels and pericardium.

Methodologic considerations in radionuclide angiography. Contrast angiography, although cumbersome and subject to the difficulties mentioned earlier, remains the reference standard for measurement of right ventricular ejection fraction and volumes. The use of radionuclide techniques for determination of right ventricular ejection fraction and volumes was recently reviewed by Marving et al. (13). There are clear-cut limitations of radionuclide assessment of right ventricular ejection fraction by counts-based methodologies (5, 13). With the multigated equilibrium technique, high background and overlap of the right atrium and right ventricle in the left anterior oblique view result in systematic underestimation of ejection fraction when compared with values obtained with contrast angiographic techniques. Variations in region of interest determination, collimation, angulation and filtering and temporal variation of background activity may cause imprecision in measurement. Absolute volume measurements require blood sampling as well, and are cumbersome to perform. These limitations have led one group (13) to doubt the accuracy of right ventricular ejection fraction determinations obtained with the multigated equilibrium technique.

First pass measurement of right ventricular ejection fraction is also subject to significant methodologic variation, largely dependent on the region of interest and background choices and method of generation of time-activity curves (13). Methodology to correct for some of these has been proposed but is largely arbitrary (13, 14); thus, we believe that there is a role for geometric measurement of right ventricular ejection fraction from first pass images. The upper limit to the precision of radionuclide studies with use of the geometric approach proposed in this report is the accuracy with which the chamber edge can be defined. In first pass studies of the right ventricle, cross talk problems are far less severe than in studies of the left ventricle because the absence of tracer in adjacent chambers leaves only the problem of removing the Compton out-scatter halo effect from the apparent chamber edge. For the left ventricle, which is more difficult to assess by this approach, we obtained correlation coefficients of 0.83 and 0.91 for measurement of end-diastolic volume and ejection fraction, respectively (8). Corresponding data are unavailable for the right ventricle. The ease with which our approach could be adapted to edge-detection algorithms and digital analysis is apparent.

Right ventricular ejection fraction and volumes in normal subjects. Using a geometric, single plane right anterior oblique method, we obtained a mean right ventricular end-diastolic volume of 63 ± 15 ml/m² and an end-systolic volume of 21 ± 6 ml/m². These values are similar to contrast angiographic data previously reported (2-6, 15); Ferlinz (6) reported values of 69 ± 13 and 22 ± 5 ml/m², respectively. The right ventricular ejection fraction in our normal group was 67%, compared with 68% reported by Ferlinz (3). Using rigorous first pass methodology, Marving et al. (13) reported that the mean right ventricular ejection fraction in normal subjects was 65%. Although we do not have corroborating cineangiographic or postmortem validations, there is a striking similarity between our measurements and those made by previous investigators in normal adult patients.

Right ventricular regional wall motion in normal subjects. Ferlinz (6) has reported that regional wall motion abnormalities are absent in normal persons and that free wall asynchrony is absent in patients with coronary disease not involving the right coronary artery, as assessed by biplane cineangiography. Our method confirms the visual absence of wall motion abnormalities in normal subjects. The definition of chords,
Right ventricular function in acute myocardial infarction. Depression of right ventricular function was evident in the patients with inferior myocardial infarction and was noticeable only in the inferior segment. Fully, 81% of these patients demonstrated the latter abnormality, compared with 13% of patients with anterior infarction. Among six patients with inferior infarction and normal right ventricular ejection fraction (Table 2), three (50%) demonstrated abnormal right ventricular regional wall motion. All 15 patients with abnormal right ventricular ejection fraction had concomitant wall motion abnormalities. Among 16 patients with anterior infarction and normal right ventricular ejection fraction, only 3 (19%) had right ventricular wall motion abnormalities, but among 7 with reduced ejection fraction, 6 (83%) demonstrated this finding. Thus, right ventricular functional abnormalities can be detected in some patients with Q wave myocardial infarction at the inferior or anterior wall by analysis of wall motion even when global function is normal. It has previously been found that regional right ventricular akinesia or dyskinesia, as assessed visually from multigated equilibrium studies, is diagnostically useful in determining the presence or absence of right ventricular infarction in the presence of inferior left ventricular infarction (17); the role of assessment of quantitative abnormalities using the methodology described will require further study. The mechanisms by which right ventricular wall motion abnormalities may occur in anterior myocardial infarction have been previously elucidated (6).

Involvement of the conus segment was rare in patients with acute myocardial infarction, perhaps because of the rarity of atherosclerotic disease in the conus branch of the right coronary artery. If such findings persisted beyond the acute phase, they might serve to differentiate ischemic from dilated cardiomyopathy, when other methods are not helpful.

Right ventricular function in dilated cardiomyopathy. Left ventricular volumes were significantly greater in patients with dilated cardiomyopathy than in normal subjects (Table 1). Right ventricular end-diastolic volume was not increased, but right ventricular ejection fraction was reduced to 43 ± 11%. The lesser degree of right ventricular dilation may be due to the predominance of ischemic rather than nonischemic dilated cardiomyopathy, with preferential left ventricular dysfunction in our study group. Regional wall motion was reduced in all segments save for the tricuspid and lateral, and was particularly profound in the conus, anterior and inferior segments.

Limitations of the current study. Several limitations are evident. There was no independent validation of right ventricular volumes or ejection fraction in our patients. We believe that right heart cineangiography is unwarranted and has potentially deleterious side effects in the patient with impaired cardiac function or recent myocardial infarction. The similarities between measurements of right ventricular volumes and ejection fraction in our normal subjects and those obtained by previous investigators suggests the validity of our results. Another limitation in this study was our inability to separate patients with inferior infarction according to the presence or absence of right ventricular involvement. Because only a minority of patients had invasive hemodynamic monitoring and right precordial ECG mapping, clinical definition of right ventricular infarction was not possible and the use of a radionuclide definition, based on right ventricular ejection fraction as is commonly used in practice, would be circuitous (18). The role of radionuclide angiography in assisting in the diagnosis of right ventricular infarction is now clear, however (17). Methodologic limitations are as described.

Conclusions. The frequency with which right ventricular wall motion abnormalities can be detected in patients with cardiac disease and normal right ventricular volume and global ejection fraction suggests that subclinical dysfunction of this chamber could be assessed readily by this radionuclide angiographic technique. The significance of such abnormalities will require further study. However, the method is applicable to serial first pass radionuclide studies and can be performed with a modicum of training.

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References


