Diagnostic Value of Visualization of the Right Ventricle Using Thallium-201 Myocardial Imaging

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SUMMARY The diagnostic significance of visualizing the right ventricle on thallium-201 myocardial perfusion scans (T-scan) at rest was studied in 53 patients. In 33 patients the right ventricle was visualized clearly on the T-scan (group A). Hemodynamic evidence of right ventricular hypertrophy with systolic pressure $\geq 30$ mm Hg was present in 28 of 33 (85%) of these patients. Right ventricular volume overload with left-to-right shunt $> 2:1$ was present in three patients. Other tests were diagnostic for right ventricular enlargement and or pulmonary hypertension as follows: chest x-ray (58%), echocardiogram (36%) and electrocardiogram (15%). In an unselected group of 20 patients (group B) where resting T-scan did not show visualization of the right ventricle, the right ventricular systolic pressure was $< 30$ mm Hg in all. The other noninvasive tests did not reveal presence of right ventricular hypertrophy or enlargement. T-scan appears to be a useful and sensitive test in detecting right ventricular pressure or volume overload compared with other noninvasive tests. This may be useful in detection of patients with right ventricular hypertrophy or enlargement secondary to pulmonary hypertension or other causes.

MANY NONINVASIVE TECHNIQUES are being used to evaluate right ventricular hypertrophy. Echocardiography is helpful in identifying some patients with pulmonary hypertension, but the assessment of right ventricular hypertrophy with noninvasive techniques has variable reliability. Although the right ventricle is usually not well-visualized during thallium-201 myocardial perfusion scanning, Cohen et al. observed that this technique was more useful than the electrocardiographic criteria for the determination of right ventricular hypertrophy in patients with chronic pulmonary hypertension. This report emphasizes the value of the thallium scan in the diagnosis of right ventricular pressure or volume overload compared with echocardiographic and electrocardiographic techniques in 53 patients with documented hemodynamic studies.

Material and Methods

Fifty-three patients with known or suspected heart disease were studied. Each had a complete clinical evaluation, including ECG, chest x-ray, echocardiogram, right and left heart catheterization, and resting thallium-201 myocardial images. There were 32 males and 21 females. The mean age for the whole group was 50.1 years (range 23–72 years). Informed consent for performing thallium-201 imaging was obtained in all patients. Myocardial images were obtained in the resting state 20 minutes after intravenous administration of 2 mCi of thallium-201. Images were obtained in the anterior, 30° and 60° left anterior oblique (LAO) and left lateral projections. A Picker gamma camera 4/10 with parallel hole collimator and a 20° window was used. A 1-mm lead ring ‘mask’ was placed on the collimator to limit the field of view. Each image contained 300,000 total counts, and computer views were made with 30% background subtraction. The 30° LAO projection provided the best separation of the right and left ventricles and was used for evaluation of the right ventricular images. The scans were interpreted by three observers without knowledge of the hemodynamic, electrocardiographic or echocardiographic findings. Visualization of the right ventricle was considered to be present when at least two of the
three observers were able to identify clearly the right ventricular myocardium. The images were graded for
the tracer uptake in the right ventricular free wall as follows: 0 = no uptake of tracer; 1+ = definite uptake but less than in the left ventricular free wall; 2+ = uptake equal to that in the left ventricular free wall; 3+ = uptake greater than in the left ventricular free wall. The right ventricular chamber size was con-
sidered increased when the right ventricular cavity appeared to be equal to or greater than the left ventric-
ular cavity in size. Increased wall thickness was qualitatively judged to be present when the right ven-
tricular free wall appeared to be equal to or greater than the left ventricular wall. Cardiac catheterization
was carried out for diagnostic reasons using standard
techniques within 1 week of isotopic study. This in-
cluded left and right ventricular hemodynamics and
left ventricular and coronary angiography. Pressure
measurements were obtained using Hewlett Packard
transducers kept at midsievel level. Standard chest
x-rays in the posteroanterior and lateral projections
were obtained in each patient. Right ventricular
enlargement was considered to be present on plain
chest x-rays when in the lateral view, the right vi-
tricle approximated the retrosternal space by greater
than one-third its length. Milner's electrocardio-
graphic criteria of right ventricular hypertrophy were
used, which consisted of QRS duration of less than
0.12 sec plus either a mean frontal plane axis of
±110° to ±180° or −90° to ±180° or an R/S or
R/S ratio in V1 greater than 1.0 with R or R1 greater
than 0.5 mV.4 Echocardiographic evidence of pul-
monary hypertension was considered present when
the pulmonic valve showed absence of an a wave in
patients with normal sinus rhythm and/or systolic
notching and flat E-F slope.1,7
The right ventricle was visualized by resting thallium-201 scan in 33 patients (group A), 14 of
whom had acquired valvular heart disease, including
mitral stenosis and regurgitation or associated
tricuspid or aortic regurgitation. One patient had
isolated pulmonic stenosis. Nine patients had cor-

Table 1. Clinical Diagnoses

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>33</td>
<td>20</td>
</tr>
<tr>
<td>Normal heart</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary heart disease</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>ASHD* with severe congestive heart failure</td>
<td>9</td>
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</tr>
<tr>
<td>ASHD without congestive heart failure</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Idiopathic congestive cardiomyopathy</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Group A = patients in whom the right ventricle was visualized on a resting thallium-201 scan; group B = patients in whom the right ventricle was not visualized on a resting thallium-201 scan.

Abbreviation: ASHD = arteriosclerotic heart disease.

tric vein disease, 30% with left ventricular failure, and
ventricular tachycardia. The right ventricle was
carried out for diagnostic reasons using standard
left ventricular and coronary angiography. Pressure
measurements were obtained using Hewlett Packard
transducers kept at midsievel level. Standard chest
x-rays in the posteroanterior and lateral projections
were obtained in each patient. Right ventricular
enlargement was considered to be present on plain
chest x-rays when in the lateral view, the right vi-
tricle approximated the retrosternal space by greater
than one-third its length. Milner's electrocardio-
graphic criteria of right ventricular hypertrophy were
used, which consisted of QRS duration of less than
0.12 sec plus either a mean frontal plane axis of
±110° to ±180° or −90° to ±180° or an R/S or
R/S ratio in V1 greater than 1.0 with R or R1 greater
than 0.5 mV.4 Echocardiographic evidence of pul-
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patients with normal sinus rhythm and/or systolic
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whom had acquired valvular heart disease, including
mitral stenosis and regurgitation or associated
tricuspid or aortic regurgitation. One patient had
isolated pulmonic stenosis. Nine patients had cor-

Results

The clinical diagnosis, electrocardiographic, echocardiographic, chest x-ray and thallium-201 scan
findings on each of 33 patients of group A in whom the right ventricle was visualized are shown in table 2. A
summary of these findings along with hemodynamic
data is presented in table 3. Electrocardiographic
criteria of right ventricular hypertrophy was present
in five of 33 (15%) and roentgenographic criteria in 19 of
33 (58%) patients in group A. The pulmonic valve was
visualized in 22 of 30 patients in whom an echocardi-
gram was available and echocardiographic evidence of
pulmonary hypertension was present in eight of these
22 (36%) patients. In group A the right ventricular
systolic pressure at rest was ≥ 30 mm Hg in 28 of 33
(85%) patients. In the remaining five patients the right
ventricular systolic pressure was < 30 mm Hg. Atrial
septal defect was present in two patients. Mitral
stenosis and aortic regurgitation with a pulmonary
artery pressure at rest of 28 mm Hg, rising to 50 mm
Hg on exercise, was recorded in one patient. In one of
these five patients with severe coronary artery disease
and mitral regurgitation, the right ventricular systolic
pressure was 28 mm Hg when his systemic pressure
was 84/66 mm Hg and the cardiac index was severely
decreased. One additional patient with chronic
obstructive pulmonary disease and documented cor-

The patients in group B in whom the right ventricle
was not visualized by thallium-201 scan had no
evidence of right ventricular hypertrophy by electro-
cardiogram or x-ray, and the echocardiogram
<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>ECG Axis</th>
<th>RVH</th>
<th>Chest x-ray</th>
<th>ECG Right ventricle</th>
<th>Echo Right ventricle</th>
<th>Right ventricular thallium-201 scan</th>
<th>Increased wall thickness</th>
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<tr>
<td>1</td>
<td>62/F</td>
<td>MS, MR</td>
<td>+60</td>
<td>No</td>
<td>LAE, LVE</td>
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<td>+</td>
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<tr>
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<td>+90</td>
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<td>Reduced</td>
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<td>+40</td>
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<td>10</td>
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<td>-80</td>
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<td>Reduced</td>
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<tr>
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<td>MR</td>
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<td>14</td>
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<td>MVP, Obstruction</td>
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<td>Reduced</td>
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<tr>
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<td>18 Present</td>
<td>Normal</td>
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<tr>
<td>20</td>
<td>43/M</td>
<td>ASHD, MR</td>
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<td>-</td>
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<td>21</td>
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<td>+</td>
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<td>22</td>
<td>32/M</td>
<td>ASHD, MR</td>
<td>+30</td>
<td>No</td>
<td>LVE</td>
<td>18 PV not seen</td>
<td>+</td>
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<td>23</td>
<td>43/F</td>
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<td>Reduced</td>
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<td>ASD</td>
<td>+100</td>
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<td>RVE</td>
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<td>38/M</td>
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<td>+80</td>
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<td>RVE</td>
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<tr>
<td>28</td>
<td>45/M</td>
<td>Cardiomyopathy</td>
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<td>No</td>
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<td>Reduced</td>
<td>+</td>
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<td>29</td>
<td>51/M</td>
<td>Cardiomyopathy</td>
<td>-65</td>
<td>No</td>
<td>LVE, LAE</td>
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<td>30</td>
<td>57/M</td>
<td>COPD, ASHD</td>
<td>+100</td>
<td>No</td>
<td>No chamber enlargement</td>
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<td>33</td>
<td>49/F</td>
<td>Primary PH</td>
<td>+100</td>
<td>Yes</td>
<td>RVE</td>
<td>18 Absent</td>
<td>Reduced</td>
<td>+</td>
<td>No*</td>
</tr>
</tbody>
</table>

For tracer uptake, + = definite uptake, but less than in the left ventricular (LV) free wall; ++ = uptake equal to that in the LV free wall; +++ = uptake greater than in the LV free wall.

*The right ventricular chamber size was less than the left, but the left ventricle itself was significantly enlarged.

Abbreviations: RVH = right ventricular hypertrophy; RVD = right ventricular diameter; a = pulmonic valve a wave; Syst = systolic; PH = pulmonary hypertension; MS = mitral stenosis; MR = mitral regurgitation; TR = tricuspid regurgitation; AR = aortic regurgitation; MVP = mitral valve prosthesis; ASHD = arteriosclerotic heart disease; Vent = ventricular; ASD = atrial septal defect; COPD = chronic obstructive pulmonary disease; SLE = systemic lupus erythematosus; LAE = left atrial enlargement; LVE = left ventricular enlargement; RVE = right ventricular enlargement; AF = atrial fibrillation.
available in five showed no evidence of pulmonary hypertension. The right ventricular systolic pressure did not exceed 28 mm Hg in any patient in this group and ranged from 12–28 mm Hg (mean of 21.2 ± 4.1 mm Hg) (fig. 1). The right ventricular end-diastolic pressure did not exceed 6 mm Hg in any patient and averaged 3.9 ± 1.7 mm Hg for this group. The right ventricular pressures were significantly higher in group A patients compared with group B patients (p < 0.001, unpaired t test).

Three basic patterns of right ventricular images were observed in our study of the thallium-201 scan:

1) Right ventricular hypertrophy with a thickened ventricular wall that tended to encroach on the ventricular chamber was observed in patients in whom pulmonary hypertension was not secondary to mitral valve or left ventricular dysfunction. Dense uptake of the radionuclide in the right ventricular myocardium indicated severe hypertrophy of this chamber. This can be seen in the patient with primary pulmonary hypertension (fig. 2). The left ventricle in this patient is relatively small.

2) Right ventricular dilatation without increase in wall thickness was seen in patients with volume overload. The patients with tricuspid regurgitation or atrial septal defect typified this group (fig. 3). The left ventricle in these patients was relatively small.

3) Dilatation of the chamber and thickening of the right ventricular wall was seen in patients with pulmonary hypertension secondary to left ventricular

### Table 3. ECG, X-ray, Hemodynamic and Echocardiographic Findings in Patients with Right Ventricular Image on Resting Thallium-201 Scan (Group A)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No.</th>
<th>ECG-RVH</th>
<th>Echo PH</th>
<th>X-ray RVE</th>
<th>RVS Pr. ≥30 (mm Hg)</th>
<th>RVED ≥6 (mm Hg)</th>
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<tr>
<td>Valvular disease</td>
<td>15</td>
<td>1</td>
<td>5</td>
<td>9</td>
<td>14</td>
<td>10</td>
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<tr>
<td>ASHD</td>
<td>9</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Other*</td>
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<td>2</td>
<td>6</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>5</td>
<td>8</td>
<td>19</td>
<td>28</td>
<td>21</td>
</tr>
</tbody>
</table>

*See table 1 for diagnosis.
†Echocardiogram available in 30 patients, but pulmonic valve seen only in 22 patients.

Abbreviations: ECG RVH = electrocardiographic evidence of right ventricular hypertrophy by Milner’s criteria; Echo PH = echocardiographic evidence of pulmonary hypertension; X-ray RVE = x-ray evidence of right ventricular enlargement; RVS Pr. = right ventricular systolic pressure at rest; RVED = right ventricular end-diastolic pressure at rest; ASHD = arteriosclerotic heart disease.

**Figure 1.** The average right ventricular systolic pressure in group A was 57.4 mm Hg compared to 21.2 mm Hg in group B (p < 0.001). In none of the patients of group B did the pressure exceed 30 mm Hg. T-scan = thallium-201 scan.
dysfunction and patients with congestive cardiomyopathy. Dilatation of both right and left ventricles was often observed. These findings can be seen in patients with severe aortic regurgitation (fig. 4, top), severe coronary artery disease and mitral regurgitation (fig. 4, middle) and congestive cardiomyopathy (fig. 4, bottom). Although the right ventricular chamber is large in these patients, it is still smaller than the left ventricle. The patient with coronary disease also shows a perfusion defect in the apical region of the left ventricle and decreased uptake in the septum secondary to previous myocardial infarction.

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

**Figure 2.** Thallium-201 scan in a patient with primary pulmonary hypertension. The right ventricle (RV) shows dense uptake of the radionuclide and appears hypertrophied. The left ventricle (LV) is relatively small. The scan on the right is a computer image. The arrows indicate the right and left ventricular free walls.

Discussion

Myocardial thallium-201 imaging has been widely used to assess left ventricular perfusion in patients with ischemic heart disease because of its characteristic uptake by myocardial cells.\(^5\)\(^-\)\(^12\) Since the left ventricular myocardium has a much greater mass and consequent blood flow, there is usually little uptake of the radionuclide by the right ventricle in normal resting persons and the left ventricle saturates the image. If more counts were collected for imaging, the right ventricle could be visualized but the left ventricular image would be too intense and not acceptable for clinical diagnostic interpretation. The right ventricle is frequently seen, however, in exercise thallium-201 scans.

Bulkley et al. reported right ventricular dilatation in two of 13 patients with ischemic cardiomyopathy using combined thallium-201 and gated cardiac blood pool scan techniques.\(^13\) The thallium scan in one of our patients (fig. 4) with ischemic cardiomyopathy is similar to the patient in their report. Right ventricular dilatation was also seen in six of their eight patients with idiopathic congestive cardiomyopathy. Although Bulkley et al. did not provide hemodynamic data on all patients, it appears that those patients were in severe congestive heart failure and probably had elevated pulmonary artery pressures. Both right and left ventricular chambers were dilated and in some patients also hypertrophied when pulmonary hypertension was secondary to left ventricular failure or mitral valve disease in 26 of our 33 patients in group A. Patients with right ventricular volume overload, in contrast, showed thin-walled and dilated right ventricles. The findings were confirmed in two patients with right ventricular angiographic studies.

Cohen et al. reported visualization of the right ventricle on resting thallium-201 scans in 18 patients with pulmonary hypertension resulting from a variety of causes and noted that the right ventricular wall thickness was \(\geq 1.1\) cm.\(^5\) The right ventricle was also visualized in two of these patients with normal

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

**Figure 3.** Thallium-201 scan in a patient with tricuspid regurgitation (TR) (top) and a patient with atrial septal defect (ASD) (bottom). These volume overloaded right ventricles (RV) are dilated and the chamber wall is not thickened. The left ventricle (LV) is smaller than the RV.
Our data suggest that the electrocardiogram is rather insensitive in establishing the presence of right ventricular volume or pressure overload. Chest x-ray and echocardiogram were diagnostic in only 58% and 36%, respectively. In contrast, thallium-201 visualization of the right ventricle characterized the presence of systolic and/or diastolic overloading of the right ventricle in 93% of our patients (31 of 33 patients of group A). Two patients in whom the right ventricle was visualized, had normal right ventricular pressures at rest. This was due to hypotension at the time of cardiac catheterization in one patient. In the other patient with severe pulmonary disease, it is possible that the thallium-201 scan was more sensitive than hemodynamics at rest. This is further supported by the fact that one additional patient in whom the right ventricle was visualized had a normal resting pulmonary artery pressure, but pulmonary hypertension during exercise.

Regarding the structural nature of the right ventricular abnormality in these studies, the presence of dilatation and hypertrophy correlates closely with clinical findings in these patients and the pathophysiology of underlying disease. Whether regression of these findings can be correlated with improvement of the disease state has not been shown.

Based on these observations, we conclude: 1) The right ventricular chamber is not visualized on a resting thallium-201 scan in normal persons; its presence suggests pressure and or volume overload of the chamber. 2) When right ventricular visualization is observed in association with normal resting right ventricular systolic pressure, it is possible that hypertension may be induced during exercise. 3) When volume overload and obstruction of the pulmonic valve are excluded, visualization of the right ventricle by resting thallium-201 scan indicates the presence of pulmonary hypertension. These observations should aid in identifying occult right ventricular loading lesions and in assessing the extent of pulmonary heart disease. Our studies suggest that thallium-201 myocardial imaging is useful for detecting right ventricular pressure or volume overload and more sensitive than electrocardiography, echocardiography or chest x-ray.

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