Differentiation of Restrictive Cardiomyopathy From Pericardial Constriction: Assessment of Diastolic Function by Radionuclide Angiography

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Diastolic filling variables were studied in 12 patients with the hemodynamic features of constriction, of whom 5 had restrictive cardiomyopathy, 5 had pericardial constriction and 2 had combined pericardial constriction and restrictive cardiomyopathy. The values were compared with those in 10 normal subjects of comparable age. The filling fractions between 10% and 70% of the diastolic time interval were greater in patients with pericardial constriction than in those with restrictive cardiomyopathy (p < 0.01 between 20% and 50%, p < 0.05 at 10%, 60% and 70%), with no overlap. The filling fractions in patients with pericardial constriction were also greater than those in normal subjects between 10% and 60% of the diastolic time interval. The filling fraction was lower in patients with restrictive cardiomyopathy than in normal subjects at 40% of the diastolic time interval (p < 0.05).

The time to peak filling rate in patients with pericardial constriction was shorter (110 ± 14 ms) than in those with restrictive cardiomyopathy (195 ± 45 ms, p < 0.01) or in normal subjects (173 ± 32 ms, p < 0.01). The percent of atrial contribution to left ventricular filling was higher in those with restrictive cardiomyopathy (4.5 ± 17%) than in those with pericardial constriction (21 ± 6%, p < 0.05) or in normal subjects (24 ± 9%, p < 0.01). Peak filling rate in stroke volume/s, but not in end-diastolic volume/s, was significantly greater in patients with pericardial constriction (5.09 ± 0.97) than in those with restrictive cardiomyopathy (3.52 ± 0.43, p < 0.01) or in normal subjects (3.98 ± 0.70, p < 0.05).

The pattern of left ventricular filling by radionuclide angiography helps differentiate restrictive cardiomyopathy from pericardial constriction.

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Patients presenting with the hemodynamic features of constriction (constrictive physiology*) (1) at cardiac catheterization represent an important diagnostic dilemma in cardiology. The differentiation of pericardial constriction from restrictive cardiomyopathy in this situation is critical because management and prognosis of these two conditions are different. The separation of such patients on clinical grounds is difficult, and noninvasive techniques have been unreliable (1–6). Cardiac catheterization and, in particular, right ventricular biopsy can be helpful in establishing the diagnosis (1,4,5,7–10) however, the hemodynamic features of restrictive cardiomyopathy may mimic precisely those of restrictive cardiomyopathy, and exploratory thoracotomy may be required to examine the pericardium for definitive diagnosis (1). Accordingly, a noninvasive method that reliably distinguishes pericardial constriction from restrictive cardiomyopathy would be useful. The pattern of left ventricular filling in patients with pericardial constriction may differ from that of patients with restrictive cardiomyopathy, with the constraint on filling occurring earlier in diastole with restrictive cardiomyopathy and later in diastole with pericardial constriction.
We studied 12 patients with constrictive physiology, and analyzed their diastolic filling patterns at rest, as measured by radionuclide angiography, to determine whether pericardial constriction could be differentiated from restrictive cardiomyopathy. The results were also compared with those of 10 normal subjects of comparable age.

**Methods**

**Study patients.** Over a 30 month period, all 12 patients whose hemodynamic profile met the criteria for constrictive physiology underwent first pass radionuclide angiography at rest. This group included three patients in whom leg raising or fluid challenge was performed and diastolic equalization was maintained. Ten normal subjects were also studied. The constrictive physiology group included five patients with pericardial constriction, five with restrictive cardiomyopathy and two with a combination of myocardial fibrosis and pericardial constriction. The diagnosis in these 12 patients was made by right ventricular endomyocardial biopsy and pericardial constriction by thoracotomy (Table 1). There were seven men and five women with a mean age of 58 ± 11 years (range 37 to 74). Age, gender, diagnosis, result of endomyocardial biopsy and hemodynamic data are shown in Table 1. Nine of the 12 patients had sinus rhythm, and 3 had atrial fibrillation (Patients 3, 9 and 11) with a heart rate at rest <110 beats/min. Symptoms were rated according to the New York Heart Association functional classification for heart failure.

The normal group included five men and five women with a mean age of 50 ± 10 years (range 35 to 64) who had normal findings on physical examination, electrocardiogram (ECG) and rest and exercise first pass radionuclide ventriculogram and no history of cardiovascular disease. There was no difference in age and heart rate between the constrictive physiology and normal groups (Table 2).

**Pericardial Constriction**

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>NYHA</th>
<th>RV Biopsy</th>
<th>Pericardium</th>
<th>RVEDP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>CI (liters/min per m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>58/M</td>
<td>III</td>
<td>Normal</td>
<td>+ Thor/Echo</td>
<td>11</td>
<td>12</td>
<td>3.20</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>71/M</td>
<td>III</td>
<td>Normal</td>
<td>+ Thor/Echo/NMR</td>
<td>20</td>
<td>22</td>
<td>2.30</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>62/M</td>
<td>III</td>
<td>Normal</td>
<td>+ Thor/Echo/NMR</td>
<td>20</td>
<td>20</td>
<td>1.80</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>52/M</td>
<td>IV</td>
<td>Normal</td>
<td>+ Thor/Ca²⁺</td>
<td>11</td>
<td>11</td>
<td>2.50</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>74/M</td>
<td>IV</td>
<td>Normal</td>
<td>Echo</td>
<td>30</td>
<td>30</td>
<td>7.70</td>
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</table>

<table>
<thead>
<tr>
<th>Combined Constriction/Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
</tr>
<tr>
<td>12</td>
</tr>
</tbody>
</table>

Ca²⁺ = calcified pericardium on chest X-ray film; CI = cardiac index; Echo = two-dimensional echocardiography (pericardial thickening); F = female; IF = interstitial fibrosis; LVEDP = left ventricular end-diastolic pressure; M = male; MH = myocyte hypertrophy; NMR = nuclear magnetic resonance imaging; NYHA = New York Heart Association functional class; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure; Rad = radiation changes; RF = replacement fibrosis; RV = right ventricular; RVEDP = right ventricular end-diastolic pressure; Thor = thoracotomy; + = positive.
on chest X-ray film and an abnormal thickened pericardium (>4 mm) using magnetic resonance imaging (Table 1) (11).

Restrictive cardiomyopathy group. The diagnosis of restrictive cardiomyopathy required hemodynamic evidence of constrictive physiology and either a specific form of heart muscle disease demonstrated by endomyocardial biopsy or no pericardial constraint at thoracotomy. A specific form of heart muscle disease was demonstrated in three of the five patients (amyloid heart disease in two and acute myocarditis in one). Of the remaining two patients, one (Patient 1) had radiation changes including myocyte hypertrophy and interstitial fibrosis secondary to inadequately shielded radiotherapy (12) for breast carcinoma, and one (Patient 4) had nonspecific myocyte hypertrophy and interstitial fibrosis. No evidence of pericardial constriction was present in these two patients at thoracotomy. The patient with myocarditis on biopsy study (Patient 2) had complete symptomatic and histologic recovery and normal findings on repeat cardiac catheterization after 2 months, precluding a diagnosis of pericardial constriction.

Combined pericardial constriction and restrictive cardiomyopathy. Two patients had mixed features, with both pericardial constriction at thoracotomy and an abnormal endomyocardial biopsy. Patient 11 had dense pericardial adhesions causing marked myocardial scarring evident at thoracotomy, and large areas of interstitial and replacement fibrosis on endomyocardial biopsy. Patient 12 had a history of mediastinal radiation for Hodgkin's disease. In addition to pericardial constriction, marked epicardial fibrosis was evident at thoracotomy, and fibrosis was also present in the endomyocardial biopsy sample. These patients had pathologic features common to both restrictive cardiomyopathy and pericardial constriction, and were considered separately from the other groups.

Data collection and analysis. First pass radionuclide angiography was performed with a multicrystal gamma camera (Baird-Atomic System 77) with the patient at rest. All studies were performed with the patient upright in the anterior projection. An indwelling catheter was placed in the antecubital vein for injection of the radionuclide. Twenty minutes after pretreatment with stannous pyrophosphate, 20 mCi of technetium-99m pertechnetate dissolved <1 ml of normal saline solution was injected and immediately flushed with 20 ml of normal saline solution. Data were accumulated at 25 ms intervals as the radionuclide bolus entered the central circulation. The first transit of the bolus through the major vessels and heart chambers was stored on a minicomputer system associated with the camera.

The data were analyzed with use of the software of the Baird-Atomic System 77, as previously described (13–15). The entire study was completed within 50 s. A region of interest was manually placed over the left ventricle, and a time-activity curve generated. Left ventricular peak activity was considered to be end-diastole, and the activity minimum was considered to be end-systole. Only cycles with ≥70% of the maximal end-diastolic activity in the end-diastolic frame were included for analysis. Background was the activity within the left ventricular region of interest before the first left ventricular beat. The background-subtracted left ventricular beats were summed to generate a single representative cardiac cycle (13,16). There was no bad beat rejection because of the few number of cycles included (four to eight). Left ventricular ejection fraction was derived from left ventricular time-activity curves by the usual method, and expressed as a percent of end-diastole. In addition, the end-diastolic outline was analyzed for end-diastolic volume with use of a single plane area-length method (13,17).

The filling fraction curves were calculated by dividing the diastolic time interval for each patient into 10 equal periods and plotting each interval against the simultaneous cumulative filling fraction ([counts at that time – end-systolic counts] × 100/[end-diastolic counts – end-systolic counts]).

### Table 2. Standard Left Ventricular Systolic and Filling Indexes in 12 Patients and 10 Normal Subjects

<table>
<thead>
<tr>
<th></th>
<th>KCM</th>
<th>PC</th>
<th>Norm</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>5</td>
<td>5</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>57 ± 11</td>
<td>63 ± 9</td>
<td>50 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>86 ± 19</td>
<td>76 ± 6</td>
<td>81 ± 14</td>
<td>NS</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>46 ± 14</td>
<td>55 ± 6</td>
<td>66 ± 8</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDV1 (ml/m²)</td>
<td>58 ± 17</td>
<td>80 ± 17</td>
<td>71 ± 16</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TTPFR (ms)</td>
<td>195 ± 45</td>
<td>110 ± 14</td>
<td>173 ± 32</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>AFC (%)</td>
<td>45 ± 17</td>
<td>21 ± 6</td>
<td>24 ± 9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>PFR</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

AFC = atrial contribution to filling; EDV = end-diastolic volumes; HR = heart rate; LVEDV1 = left ventricular volume index; LVEF = left ventricular ejection fraction; Norm = normal subjects; NS = not significant; PC = pericardial constriction; PFR = peak filling rate; RCM = restrictive cardiomyopathy; SV = stroke volumes; TTPFR = time to peak filling rate.

The filling fraction curves were calculated by dividing the diastolic time interval for each patient into 10 equal periods and plotting each interval against the simultaneous cumulative filling fraction ([counts at that time – end-systolic counts] × 100/[end-diastolic counts – end-systolic counts]).
Linear interpolation was performed between points in the raw data curve to determine filling at each 10% period. This technique has been used to assess filling using a cineangiographic method (10), but has not previously been applied to radionuclide data.

The peak filling rates were calculated by an algebraic method similar to a method described previously (18), except that the derivative of the time-activity curve was calculated using a wrap around digital filter with coefficients \((-0.2, -0.1, 0, 0.1, 0.2)\). This method assumes that the data can be described by a third order polynomial function, but does not require actual fitting of the data with a polynomial function. The peak filling rates were normalized by end-diastolic counts and by stroke counts expressed in end-diastolic volumes/s and stroke volumes/s (19). The time to peak filling rate was calculated as the time at peak filling rate – time at end-systole, and was expressed in milliseconds (ms) (Fig. 1). Time to peak filling rate was also normalized to the RR interval (time to peak filling rate/RR interval) (20).

The atrial contribution to filling was derived from time-activity curves, and was defined as the percent of diastolic filling occurring with atrial systole. The beginning of atrial filling was obtained by using the total PR interval from the electrocardiogram + 40 ms (electromechanical delay) (21). The percent of atrial filling contribution was defined as atrial filling contribution \(\times \frac{100}{\text{filling volume}}\) (Fig. 1).

Statistics. Group data are expressed as mean values \(\pm SD\). Statistical analysis was done using multivariate one-way analysis of variance, and comparisons of group means was by Newman-Keuls test (22). The differences were considered significant at the level of \(p < 0.05\).

Results

Table 1 compares the clinical, histologic and hemodynamic data of the patient groups.

Systolic function. Table 2 compares left ventricular ejection fraction and end-diastolic volume index in patients with constrictive physiology and normal subjects. There was no difference in ejection fraction between patients with pericardial constriction (53 \(\pm 6\%\)) and those with restrictive cardiomyopathy (48 \(\pm 14\%\)). However, normal subjects had a greater ejection fraction (66 \(\pm 8\%\)) than did either patient group. There was no difference in end-diastolic volume index among groups.

Diastolic function. Figure 1 demonstrates representative raw time-activity curves for restrictive cardiomyopathy (Patient 1) and pericardial constriction (Patient 7), and Figure 2 shows the filling curves for the 10 individual patients normalized to the diastolic filling period. The averaged filling curves normalized to the diastolic filling period for those with pericardial constriction or restrictive cardiomyopathy and for normal subjects are shown in Figure 3. The filling in patients with pericardial constriction was greater than that in patients with restrictive cardiomyopathy at all intervals between 10% and 70% of the diastolic filling interval, with no overlap between groups. This difference was greatest between 20% and 50% (\(p < 0.01\)) of the diastolic filling interval (peak separation at 40%), with a lesser separation at 10%, 60% and 70% (\(p < 0.05\)). The filling in patients with pericardial constriction was greater than that in normal subjects between 10% and 60% of the diastolic time interval (\(p < 0.01\) between 10% and 40%, and \(p < 0.05\) at 50% and 60% of the diastolic time interval). The filling fraction of patients with restrictive cardiomyopathy was lower than that of normal subjects at 40% of the diastolic time interval (\(p < 0.05\)); therefore, at 40% of this interval, there was significant separation of cumulative filling fractions in all three groups. Flattening of the filling curve in normal subjects (diastasis) occurred at about 75% of the diastolic time interval, whereas in those with pericardial constriction (where it represents the cessation of rapid filling caused by the constraints of the
pericardium), it occurred earlier at about 50% of the diastolic time interval (Fig. 3).

Table 2 and Figure 4 show the results of other variables used to assess left ventricular filling. The time to peak filling rate in those with pericardial constriction (110 ± 14 ms) was shorter than in those with restrictive cardiomyopathy (195 ± 45 ms, p < 0.01) and in normal subjects (173 ± 32 ms, p < 0.01), and this difference was maintained after correction for the RR interval. There was no overlap between time to peak filling rate for patients with pericardial constriction (100 to 125 ms) and restrictive cardiomyopathy (175 to 275 ms). The atrial contribution to filling was greater in those with restrictive cardiomyopathy (45 ± 17%, range 33% to 70%) than in those with pericardial constriction (21 ± 6%, range 12% to 27%, p < 0.05) or in normal subjects (24 ± 9%, p < 0.01). The peak filling rate normalized by end-diastolic volume was not different among the three groups, although there was a trend to a higher rate in patients with pericardial constriction (2.5 ± 0.5) than in those with restrictive cardiomyopathy (1.90 ± 0.44), and no significant difference from normal subjects (2.65 ± 0.63). However, when the peak filling rate was normalized by stroke volume, it was significantly higher in those with pericardial constriction (5.09 ± 0.97) than in those with restrictive cardiomyopathy (3.52 ± 0.43, p < 0.01) and in normal subjects (3.98 ± 0.70, p < 0.05).

Thus, the extent of early filling was consistently increased in patients with pericardial constriction, with no overlap between those with pericardial constriction and those with restrictive cardiomyopathy from 10% to 70% of the diastolic time interval. In addition, in patients with pericardial constriction, the time to peak filling occurred earlier and the peak filling rate (in stroke volumes/s) was greater than in patients with restrictive cardiomyopathy or in normal subjects.

Combined restrictive cardiomyopathy and pericardial constriction. Two patients had both myocardial fibrosis on endomyocardial biopsy and pericardial constriction at thoracotomy (Patients 11 and 12, Table 1), including one (Patient 11) with pericardial calcification. In Patient 11, the filling curve was similar to that in those with restrictive cardiomyopathy, suggesting that the dominant component contributing to reduced filling in this patient was restrictive cardiomyopathy. In Patient 12, the extent of early filling was intermediate compared with that in patients with pericardial constriction or restrictive cardiomyopathy.

**Discussion**

The differentiation of pericardial constriction from restrictive cardiomyopathy is very difficult and often relies on thoracotomy for definitive exclusion of pericardial constriction. The risks of such surgery in patients with restrictive cardiomyopathy, particularly those with cardiac amyloidosis, have been well documented (23,24). Pericardial stripping
also carries a mortality risk, which could be reduced if it were possible to identify that subset of patients with co-
comitant pericardial constriction and restrictive cardiomy-
opathy who may not benefit from surgery.

**Limitations of other techniques.** Endomyocardial biopsy 
(25,26) may confirm the presence of specific infiltrative 
disease, provide prognostic information and even suggest 
therapy. A normal biopsy examination excludes restrictive 
cardiomyopathy (25). However, a biopsy showing nonspe-
cific changes such as hypertrophy and interstitial or replace-
ment fibrosis, or both, does not exclude concomitant peri-
cardial constriction (Patients 11 and 12) or identify whether 
this or restrictive cardiomyopathy is the major determinant 
of constrictive physiology (25,27). Echocardiography may 
allow for qualitative discrimination of normal from thickened 
pericardium (28) but does not quantify pericardial thickness 
accurately (29,30). Abnormal septal motion (31–33), flat-
tening of left ventricular posterior wall diastolic movement 
(29,31,33,34) and increased left ventricular wall thickness 
(2,34,35) have been described in restrictive cardiomyopathy. 
Computed tomographic scanning has been proposed to iden-
tify pericardial thickening and tumor deposition (36,37). 
Nuclear magnetic resonance imaging appears to be an accu-
rate technique for assessing pericardial thickness, and a 
thickness >4 mm has been considered abnormal (11).

**Hemodynamic data have not proved to be reliable in differentiating pericardial constriction from restrictive cardiomyopathy** in patients with constrictive physiology (3–6,10,30,38). Patients with restrictive cardiomyopathy with both equal (constrictive physiology) and unequal ventricular filling pressures have been assessed as a homogeneous group (10), whereas other studies of restrictive cardiomyopathy have not reported individual hemodynamic data (6,30). Therefore, a disparate variety of abnormalities of diastolic function have been described in restrictive cardiomyopathy.

**Diastolic filling curves.** Left ventricular filling curves de-
erived from the fraction of the diastolic filling volume from 
left ventricular angiography or digitized M-mode echocar-
diography and plotted against the fractionated diastolic time 
interval (10,30) have provided the most insight in character-
izing left ventricular filling in pericardial constriction and 
restrictive cardiomyopathy. These techniques support our 
findings of divergent early filling patterns in pericardial 
constriction and restrictive cardiomyopathy. However, they 
call for geometric assumptions about the shape of the left 
ventricle, and require frame by frame tracing of multiple left 
ventricular silhouettes, which precludes their use in routine 
clinical practice. Furthermore, regional differences in seg-
mental wall thickening, excursion and rates of change are 
important limitations of these techniques (39,40). The 
present study applied this concept to radionuclide methods, 
thereby avoiding geometric assumptions and planimetry. It 
also showed, with use of a standard noninvasive method, a 
clear separation of patients with pericardial constriction and 
restrictive cardiomyopathy on the basis of their diastolic 
filling curves. There was no overlap of patients with pericar-
dial constriction and those with restrictive cardiomyopathy 
between 10% and 70% of the diastolic time interval, and of 
patients with pericardial constriction and normal subjects 
between 10% and 60% of the diastolic time interval.

**Restrictive cardiomyopathy and pericardial constriction may coexist.** In both our patients with combined disease, severe myocardial involvement was present, and in one 
(Patient 11), a restrictive pattern of filling dominated and 
concealed the coexistence of pericardial constriction. More 
information is required in patients with combined pericardial 
constriction and restrictive cardiomyopathy, but this tech-
nique may 1) identify severe associated restrictive cardio-
myopathy, which may reduce the chances of a successful 
surgical outcome; or 2) identify pericardial constriction as 
the major determinant of reduced filling, suggesting that 
pericardial stripping will be beneficial.

**Traditional radionuclide filling indexes.** The differences in 
filling curves were corroborated by a significant shortening 
of the time to peak filling rate in patients with pericardial 
constriction compared with that in normal subjects and

![Figure 4. Values for time to peak filling rate (ms), peak filling rate (stroke volume [SV]/s) and atrial filling contribution (%) are shown for each group, including mean ± SD. C = pericardial constriction; NORM = normal subjects; R-CM = restrictive cardiomyopathy.](image-url)
patients with restrictive cardiomyopathy. This shortening was maintained even when time to peak filling rate was corrected for RR interval (20). The percent of atrial filling contribution, a strong inverse correlate of early filling (21), is increased in patients with restrictive cardiomyopathy compared with those with pericardial constriction and normal subjects, confirming the marked reduction in early filling in restrictive cardiomyopathy and a greater reliance on atrial systole. The percent atrial filling contribution may be more sensitive than the diastolic filling curves in separating patients with restrictive cardiomyopathy from normal subjects because the significance level was greater ($p < 0.01$ without overlap) than with diastolic filling fraction at 40% of the diastolic time interval ($p < 0.05$). However, atrial fibrillation may occur with restrictive cardiomyopathy, precluding the use of percent atrial filling contribution.

The peak rate of ventricular filling has been shown to be preload dependent (41), and approaches to normalizing this index have included dividing by instantaneous or end-diastolic volume. These techniques adjust for variation in ventricular size, but will conceal elevations in filling when there are subtle elevations in end-diastolic volume (as with the patients with pericardial constriction in this study). Angiographically determined filling rate has been shown to correlate with stroke volume in normal patients (42), which suggests normalization of filling rate by dividing by stroke volume. Just as we employed the fraction of filling volume for describing the pattern of filling, so we employed filling volume to normalize peak filling rate (19). This variable distinguished pericardial constriction from restrictive cardiomyopathy without overlap, whereas peak filling rate corrected by end-diastolic volume did not.

Limitations of the study. All patients with pericardial constriction and restrictive cardiomyopathy in this study had overt and symptomatic disease. With a moderate or marked elevation and equalization of ventricular filling pressures. This technique has not been validated in patients with occult disease, who do not have elevation of ventricular filling pressure at rest. Although the number of patients studied is small, the differences are statistically significant. We consider that the lack of overlap of filling curves, time to peak filling rate, peak filling rate and percent atrial filling contribution indicates the merit of this technique. The usefulness of peak filling rate may be reduced by its preload dependence (41), although this index provided a clear separation of groups when expressed as stroke volumes/s.

This study was performed with first pass radionuclide angiography, which ensures that adequate counts are obtained with few beats and without the need for gating. The data are acquired in list mode and then reformatted, taking every end-diastole with $\geq 70\%$ of peak activity. These concepts could theoretically be applied to R wave-gated blood pool scanning, but this procedure would require high counts, high time resolution and rejection of bad beats, probably using a list mode acquisition with reformattting (21).

This study includes patients with atrial fibrillation, whose abnormal filling characteristics may have confounded the influence of pericardial constriction or restrictive cardiomyopathy on left ventricular diastolic function. However, our three patients with constrictive physiology and atrial fibrillation had filling characteristics similar to those of patients in sinus rhythm (Fig. 2) because they did not have unusual heart rates or wide variations in RR intervals. Information is required to determine the filling pattern in patients with restrictive cardiomyopathy who do not have constrictive physiology, although their differentiation from those with pericardial constriction on hemodynamic grounds is clear.

Conclusions. First pass radionuclide angiography in patients with constrictive physiology allows differentiation of pericardial constriction from restrictive cardiomyopathy. The extent of early filling from 10% to 70% of diastole is greater in pericardial constriction than in restrictive cardiomyopathy. This observation is confirmed by the atrial contribution to filling, the time to peak filling rate and the peak filling rate (stroke volumes/s), each of which separates the patient groups without overlap. The demonstration of early rapid filling with this technique indicates that pericardial constriction is likely. Reduced or normal early filling is demonstrated in patients with restrictive cardiomyopathy or with combined disease. We suggest that patients with constrictive physiology syndrome could be evaluated by a combination of endomyocardial biopsy and first pass radionuclide angiography to differentiate pericardial constriction from restrictive cardiomyopathy, and to select candidates who may benefit from thoracotomy and pericardiectomy.

References


