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Right Ventricular Ejection Fraction: An Indicator of Increased Mortality in Patients With Congestive Heart Failure Associated With Coronary Artery Disease

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The predictive value of radionuclide ventriculography was studied in 34 patients with depressed left ventricular ejection fraction (< 40%) and clinically evident congestive heart failure secondary to atherosclerotic coronary artery disease. In addition to left ventricular ejection fraction, right ventricular ejection fraction and extent of left ventricular paradox were obtained in an attempt to identify a subgroup at increased risk of mortality during the ensuing months. The 16 patients who were alive after a 2 year follow-up period had a higher right ventricular ejection fraction and less extensive left ventricular dyskinesia. When a right ventricular ejection fraction of less than 35% was used as a discriminant, mortality was significantly greater among the 21 patients with a depressed right ventricular ejection fraction (71 versus 23%), a finding confirmed by a life table analysis. Depressed right ventricular function was further linked to more severely compromised left ventricular function, as confirmed by a greater reduction in left ventricular ejection fraction and by an increased extent of left ventricular dyskinesia. These patients had a greater prevalence of chronic obstructive pulmonary disease and previous inferior myocardial infarction but the differences between groups were not statistically significant. It appears that the multiple factors contributing to the reduction in right ventricular ejection fraction make it a useful index not only for assessing biventricular function, but also for predicting patient outcome.

Chronic congestive heart failure is a common late sequela of myocardial infarction in patients with atherosclerotic heart disease. Not only are these patients easily identified by their symptoms and certain clinical findings but also, as a group, they manifest certain disorders in myocardial function that lend themselves to quantification. Contrast ventriculographic studies will characteristically reveal reductions in left ventricular function, as measured by ejection fraction, paralleling the extent of abnormal segmental contraction of the left ventricle (1–3).

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Previous investigators have noted that these measurements can, in part, help to identify those patients at higher risk of cardiac death. Thus, a depressed left ventricular ejection fraction (< 40%) appears to be an indicator of increased mortality in either the early or the late postmyocardial infarction period (4,5). It has also become apparent that our modern therapeutic approaches and their great reliance on diuretic drugs have made the evaluation of ventricular function more difficult clinically and thus more heavily dependent on invasive hemodynamic measurements.

Radionuclide ventriculography is an easily implemented, noninvasive means of assessing ventricular function. Thus, measurements of ventricular ejection fraction correlate well with those obtained from contrast ventriculography (6–8). The amount and extent of contractile abnormalities of the left ventricle can also be ascertained and, in some cases, quantitated (9,10).

The present study was designed to determine whether certain indexes of biventricular function would be helpful for identifying a subgroup at increased risk of death among patients with depressed left ventricular ejection fraction. Specifically, it attempts to determine whether right ventric-

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ular ejection fraction is an important determinant for the survival of patients suffering from chronic congestive cardiomyopathy secondary to atherosclerotic heart disease.

Methods

Study patients. All patients referred to the clinical nuclear medicine unit between April 1978 and July 1979 were considered for admission to the study. A group of 36 patients with severely depressed biventricular function was selected first by means of ejection fraction values determined noninvasively by radionuclide ventriculography (Group I); the limits were set at 35% (3 standard deviations below our mean value for normal subjects) for the right ventricle and at 40% for the left ventricle. A matched control group consisted of 36 patients whose left ventricular function was severely depressed (< 40%) but whose right ventricular function was relatively preserved (> 35%) (Group II).

To further characterize a cohort whose survival would reflect progressive myocardial decompensation associated with ischemic heart disease, those patients with recent myocardial infarction (< 3 months) and significant valvular disease, and those without documentation of atherosclerotic heart disease (by previous history, electrocardiography or coronary angiography) were excluded. Furthermore, patients with a history of severe alcoholism were also excluded. After this elimination process, Group I consisted of 21 patients and Group II of 13; 2 patients previously had valvular replacement, 1 was the recipient of aortocoronary bypass surgery and 3 had undergone both procedures.

Confirmation of ischemic heart disease was obtained by combined historical and electrocardiographic criteria. Patient histories included documentation of previous myocardial infarction using serum creatine kinase elevation and typical serial evolution of electrocardiographic signs. Evidence of previous myocardial infarction was further characterized using the following electrocardiographic criteria: significant Q waves (≥ 0.04 second) seen in leads III and aVF (inferior) or in leads I, II, aVL or V_1 to V_4 (anterior).

Seventeen patients manifested electrocardiographic evidence of old anterior infarction and one patient had significant changes in the inferior leads; concomitant evidence of damage to both myocardial regions was seen in six patients. In 10 cases, the electrocardiogram could not be interpreted because of conduction abnormalities. Significant atherosclerotic heart disease was confirmed by coronary angiography in 13 cases, using the presence of significant narrowing of the luminal diameter (> 50%) of any one or more of the major coronary artery branches as a criterion (one case of one vessel disease, four cases of two vessel and eight cases of three vessel disease).

Radionuclide ventriculography. This procedure was performed subsequent to the in vivo labeling of red blood cells with technetium-99m (as pertechnetate) after the injection of a tin (Sn-pyrophosphate) preparation (11).

Table 1. Clinical Characteristics of Survivors and Nonsurvivors

	Survivors (n = 16)	Nonsurvivors (n = 18)	p Value§
Age (years)	58.1 ± 11.9	57.7 ± 6.9	
Sex			
Male	14	13	
Female	2	5	
Digoxin	16 (100%)	18 (100%)	NS
Diuretic drugs	16 (100%)	18 (100%)	NS
Vasodilators	8 (50%)	11 (61%)	NS
Abnormal chest X-ray*	15 (94%)	16 (89%)	NS
Right-sided failure†	4 (25%)	8 (44%)	NS
Duration of symptoms			
> 1 year	8 (62%)	5 (33%)	
< 1 year	5 (38%)	10 (67%)	
Unavailable	3	3	
COPD	1 (6%)	6 (33%)	NS
Smoker (pack-years)	38.0 ± 21.3 (10)	45.5 ± 31.8 (11)	NS
Diabetes	3 (19%)	4 (22%)	NS
Hypertension	3 (19%)	6 (33%)	NS
Follow-up (months)	19.9	6.7	

*Radiograph showing at least two of the following: cardiomegaly, pulmonary vascular redistribution or interstitial edema. †Either jugular venous distension or peripheral edema, or both. §p ≥ 0.5 is not significant.

Values in this and succeeding tables are mean values and, where applicable, ± standard deviation.

COPD = respiratory impairment necessitating treatment with bronchodilators (chronic obstructive pulmonary disease); NS = not significant; p = probability value.

Electrocardiographic synchronization was performed so that individual cardiac cycles could be acquired from a standard gamma camera and superimposed with the help of a dedicated computer system (Gamma-11, Digital Equipment Corporation). Images were obtained from the left anterior oblique projection with the help of a slant hole collimator (30° caudal angulation). Left and right ventricular ejection fraction values were determined according to previously validated techniques (8–10). A noninvasive index of the extent of myocardial dyskinesia was obtained by mathematical subtraction of the end-diastolic frame of the individual studies from the end-systolic frame (12).

Follow-up. Follow-up information was obtained by soliciting the treating physicians and reviewing available medical records; when necessary, family members were interviewed by telephone. The reviewer had no knowledge of whether the patient was in Group I or Group II. Death was considered to be due to cardiac causes whenever rapid cardiopulmonary decompensation leading to death occurred. One patient with pancreatic carcinoma was considered to have migrated alive from the cohort (Group I). In another patient, concomitant cerebrovascular disease may have contributed to sudden death.

Analysis of data. Pertinent historical and clinical findings and other confirmatory evidence of congestive heart failure (chest radiographs) were obtained by review of the patients' charts for the interval of time closest to the day of radionuclide ventriculography. In no case was this longer than 1 week. The reviewer was unaware of the results of radionuclide ventriculography.

Right-sided cardiac catheterization had also been performed within 2 weeks of radionuclide ventriculography in 13 patients. Individual measurements of pulmonary artery and pulmonary capillary wedge pressure and pulmonary vascular resistance were available for review in eight patients in Group I and five patients in Group II.

Statistical analysis. Comparison between means was carried out with the use of the Student's *t* test, and differences between proportions were made with the help of chi-square analysis with Yates' correction for continuity. Cardiac catheterization data were analyzed with the Mann-Whitney U test.

A life table analysis for both groups was conducted according to the method of Kaplan and Meier (13). Statistical comparison was performed according to the Mantel-Haenszel test for survival data (14). All means are expressed with 1 standard deviation interval unless otherwise specified.

Results

Findings Linked to Survivorship

Clinical evidence of congestive heart failure. All of the 34 patients followed up had clinical evidence of congestive heart failure (Table 1). Chest radiography revealed cardiomegaly in all but three patients; the presence of either pulmonary vascular redistribution or interstitial edema, or both, was also confirmed in these patients. According to the New York Heart Association criteria, the level of impairment in physical activity secondary to dyspnea was more severe among nonsurvivors than survivors (mean 3.6 ± 0.5 versus 3.0 ± 0.7, respectively; probability [p] < 0.01). No significant differences emerged when the average age of nonsurvivors (57.7 ± 6.9 years; range 43 to 68) was compared with that of survivors (58.1 ± 11.9 years; range 37 to 74). The therapeutic regimens used for both groups were similar. Clinical signs of right-sided failure, although more common among nonsurvivors, did not differ significantly in the two groups. Thus, 2 of the 16 survivors had direct clinical evidence of elevated right-sided pressures (jugular venous distension) and 2 had pedal edema; jugular venous distension was seen in 4 of the 18 nonsurvivors, pedal edema in 1 and both findings in 3.

Table 2. Survival as Related to the Noninvasive Assessment of the Extent of Myocardial Dysfunction

	Survivors (n = 16)	Nonsurvivors (n = 18)	p Value
LVEF	18.7 ± 6.8%	18.1 ± 10.3%	NS
RVEF	41.4 ± 23 0%	23 9 ± 10.2%	< 0.01
LV dyskinesia	20.9 ± 11.8%	29.7 ± 15.0%	< 0.05
Ventricular arrhythmia	8 (50%)	8 (44%)	NS
Time since first MI (years)	4 4 ± 3.5 (14)	6.9 ± 4.9 (12)	NS
Number of infarcts	1.8 ± 1.3 (16)	1.7 ± 0.8 (16)	NS
Electrocardiographic location of previous MI			
Anterior	10	7	
Inferior	0	1	
Both	2	4	
Not determined	4	6	
Functional class (NYHA)	3.0 ± 0.7	3.6 ± 0.5	< 0.01

LV = left ventricular; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NS = not significant; NYHA = New York Heart Association, RVEF = right ventricular ejection fraction

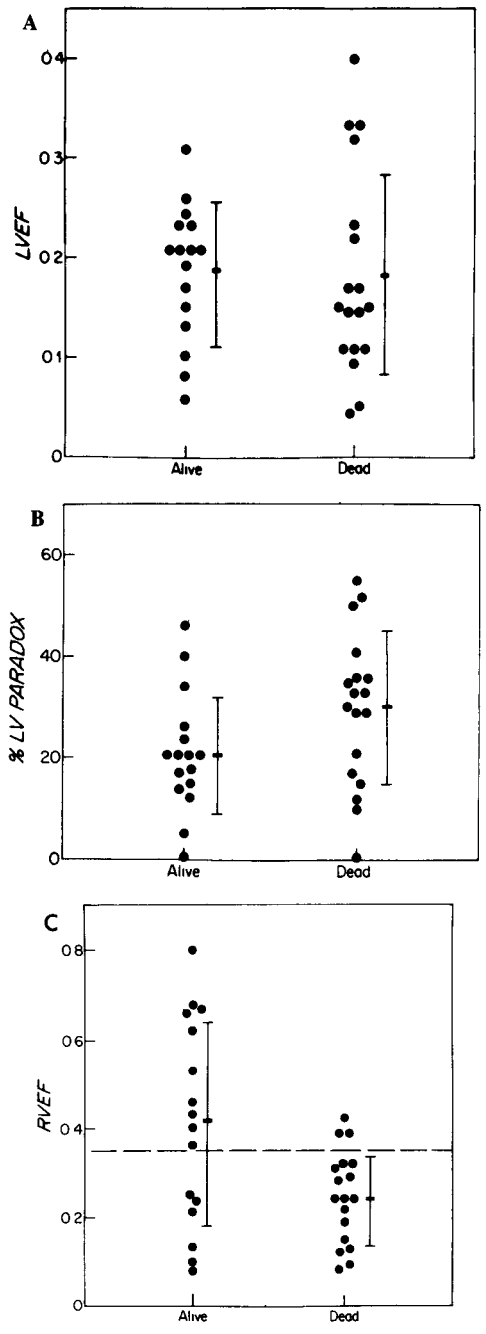


Figure 1. Left and right ventricular ejection fraction and survival. Although no significant difference exists between the left ventricular ejection fraction (LVEF) in survivors and nonsurvivors **(A)**, the extent of left ventricular (LV) paradox is greater among nonsurvivors **(B)**. The right ventricular ejection fraction (RVEF) values for nonsurvivors are markedly depressed when compared with those of survivors **(C)**.

Other risk factors. Similarly, a greater number of nonsurvivors had enough evidence of chronic obstructive pulmonary disease to warrant treatment with bronchodilators (33%, nonsurvivors versus 6%, survivors). Survival did not appear to be linked to the presence of hypertension (33%, nonsurvivors versus 19%, survivors), the incidence of adult onset diabetes (22%, nonsurvivors versus 19%, survivors) or previous exposure to cigarette smoke (45.5 ± 31.8 pack-year nonsurvivors versus 38.0 ± 21.3 pack-year survivors).

Previous myocardial infarction and left ventricular dysfunction. The average number of previous myocardial infarctions was similar (1.7 ± 0.8, nonsurvivors versus 1.8 ± 1.3, survivors), whereas a trend toward a longer interval between the first documented myocardial infarction and the radionuclide ventriculogram (6.9 ± 4.9 years, nonsurvivors versus 4.4 ± 3.5 years, survivors) was evident although failing to reach statistical significance (Table 2). There was a higher incidence of electrocardiographically identifiable inferior myocardial infarction among nonsurvivors (5 of 12 versus 2 of 12 survivors; p = not significant) and, on the average, right ventricular function as measured by the right ventricular ejection fraction was more severely depressed in nonsurvivors (23.9 ± 10.2%, nonsurvivors versus 41.4 ± 23.0%, survivors; $p < 0.01$) (Fig. 1). The extent of left ventricular dysfunction as determined from the ejection fraction values was similar for both groups (18.1 ± 10.3%, nonsurvivors versus 18.7 ± 6.8%, survivors; p = not significant), although the amount of left ventricular dyskinesia obtained from the paradox index was larger among nonsurvivors (29.7 ± 15%, nonsurvivors versus 20.9 ± 11.8%, survivors; $p < 0.05$) (Fig. 1).

The prevalence of atrial fibrillation was 17% (6 of 34). Ventricular arrhythmia was present to an equal amount among nonsurvivors (8 of 18; 44%) and survivors (8 of 16; 50%). The incidence of previous valvular or coronary bypass surgery, or both, was similar for both survivors (3 of 16; 19%) and nonsurvivors (3 of 18; 17%).

Clinical Relevance of Right Ventricular Ejection Fraction

Left ventricular dysfunction. Comparison of the group with depressed right ventricular function (Group I) and the group with normal right ventricular function confirmed a trend toward more severe left ventricular dysfunction in Group I (Table 3; Fig. 2). This is reflected by the greater reduction in left ventricular ejection fraction (15.8 ± 8.6% versus 22.5 ± 7.4%; $p < 0.025$) and the increased extent of left ventricular dyskinesia (30.1 ± 13.5% versus 18.2 ± 12.3%; $p < 0.025$) in the Group I patients compared with the Group II patients. There was also an increase in mortality in patients with significant depression of right ventricular function compared with those without (71 versus 23%, respectively; $p < 0.02$).

Correlation with other clinical features. Severe depression of right ventricular ejection fraction correlated with greater symptomatic impairment due to dyspnea (functional stage, New York Heart Association: 3.0 ± 0.7, Group II versus 3.5 ± 0.6, Group I; $p < 0.01$). There was no significant association of a more depressed right ventricular

Table 3. Relevance of Right Ventricular Ejection Fraction to Extent of Myocardial Dysfunction

	RVEF (< 35%) (n = 21)	RVEF (≥ 35%) (n = 13)	p Value
LVEF	15.8 ± 8.6%	22 5 ± 7 4%	< 0 025
LV dyskinesia	30.1 ± 13.5%	18 2 ± 12 3%	< 0.025
Deaths	15 (71%)	3 (23%)	< 0.02
Ventricular arrhythmia	9 (43%)	7 (54%)	NS
Time since first MI (years)	5.8 ± 4 6 (15)	5 2 ± 4 1 (11)	NS
Number of infarcts	1 6 ± 0 8 (19)	1 9 ± 1.4 (13)	NS
Electrocardiographic location of previous MI			
Anterior	11	6	
Inferior	1	0	
Both	5	1	
Not determined	4	6	
Functional stage (NYHA)	3.5 ± 0.6	3 0 ± 0 7	< 0.01

Abbreviations as in Table 2

ejection fraction with the prevalence of signs of right-sided heart failure, the number of myocardial infarctions, elapsed time since the first infarction, a history of either diabetes or hypertension and the presence of either obstructive lung disease or ventricular arrhythmia (Table 4). Although electrocardiographic evidence of inferior left ventricular wall damage was more common in Group I (35%, Group I versus 14%, Group II; p = not significant), this was not statistically significant.

Correlation with survival. Actuarial analysis of the survival curves for patients in Groups I and II confirmed the lower likelihood of survival in patients with a depressed right ventricular ejection fraction (Fig. 3). This difference was seen at 6 months (52%, Group I versus 84%, Group II; $p < 0.05$), 12 months (37%, Group I versus 84%, Group II; $p < 0.05$) and 24 months (20%, Group I versus 58%, Group II; $p < 0.05$); after that time, only six patients remained in the follow-up group (two in Group I; four in Group II). Combined survival for our study population is depicted in Figure 4; by 12 months, the probability of survival was 53%, decreasing to 34% by 24 months.

Correlation with cardiac catheterization data. Analysis of the available right-sided cardiac catheterization data (Table 5) revealed greater pulmonary capillar wedge pressures among Group I patients (31.0 ± 4.4 mm Hg) than in Group II patients (17.2 ± 8.9 mm Hg). The eight patients with depressed right ventricular ejection fraction also had a significantly greater mean pulmonary artery pressure (40.8 ± 7.1 versus 23.8 ± 6.8 mm Hg; $p < 0.005$) and larger pulmonary vascular resistances (400.3 ± 206.7 versus 127.6 ± 78.2 dynes·s·cm^{-5}; $p < 0.01$) than the five patients from Group II.

Discussion

Role of radionuclide ventriculography in assessing ventricular function. Recent studies have emphasized the relevance of determining left ventricular ejection fraction at rest in patients with coronary artery disease. Whether such measurements are obtained during acutely evolving damage to the myocardium or as part of the assessment of myocardial impairment in the basal state, survival rates tend to worsen and parallel decreases in ejection fraction values (1–2,4–5,15). Until recently, determination of this index of myocardial contractility has been obtained from contrast ventriculography performed during catheterization procedures. The advent of radionuclide ventriculography has increased the ease with which ventriculography is performed without sacrificing either the accuracy of quantitative measures of global left ventricular function or the reliability of qualitative scoring of the extent of impaired left ventricular wall motion

Figure 2. Individual values of right and left ventricular ejection fraction values are plotted against each other for both survivors (△) and nonsurvivors (●). A right ventricular ejection fraction (RVEF) of 35% is indicated by the **horizontal line.** The subgroup of patients with an ejection fraction above this value is mostly composed of survivors. LVEF = left ventricular ejection fraction.

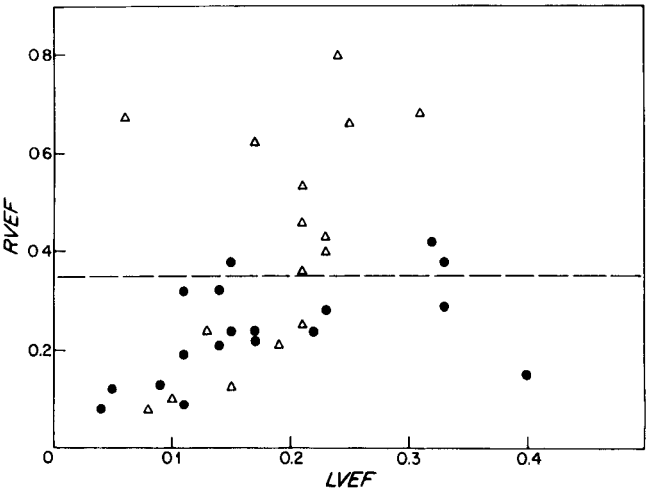


Table 4. Clinical Characteristics of Patients With and Without Right Ventricular Dysfunction

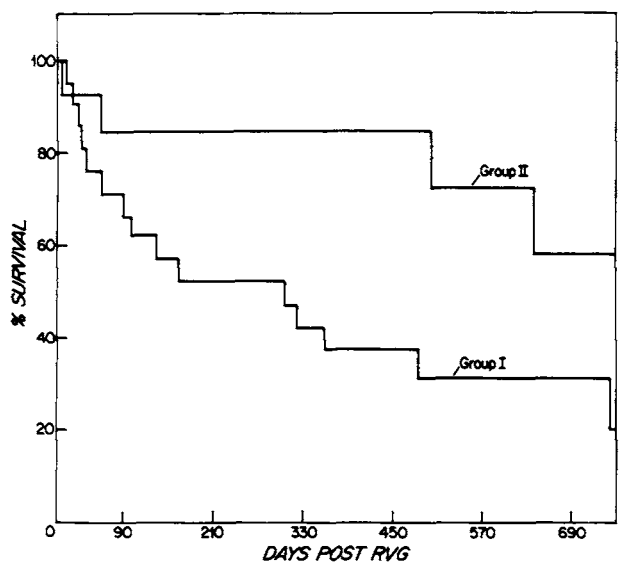
	RVEF < 35% (n = 21)	RVEF ≥ 35% (n = 13)	p Value
Age (years)	57.9 ± 10.0	56.4 ± 11.1	NS
Sex			
Male	17	10	
Female	4	3	
Vasodilators	13 (62%)	6 (46%)	NS
Abnormal chest X-ray*	20 (95%)	11 (85%)	NS
Right-sided failure†	8 (38%)	4 (31%)	NS
COPD	5 (24%)	2 (15%)	NS
Smoker (pack-years)	42.3 ± 32.4 (11)	41.5 ± 21.0 (10)	NS
Diabetes	5 (24%)	2 (15%)	NS
Hypertension	5 (24%)	4 (31%)	NS

*Radiograph showing at least two of the following: cardiomegaly, pulmonary vascular redistribution or interstitial edema. †Either jugular venous distension or peripheral edema, or both.

Abbreviations as in Tables 1 to 3.

(6,9,10). In addition to the noninvasive character of radionuclide ventriculography, the quantitative assessment of biventricular function is also possible if the right ventricular contours are carefully delineated. We recently showed the ease of determining right ventricular ejection fraction using a modified left anterior oblique projection (8). We also demonstrated that the amount of left ventricular dyskinesia can be quantitated as a ratio of the portion of the left ventricle showing paradoxical increases in volume (as measured by counts issuing from an isotopic label of the blood pool) at end-systole to the total area of the ventricle. This paradox index not only correlates with the amount of paradoxical movement seen during contrast ventriculography but has an inverse relation to left ventricular ejection fraction at rest (12).

Figure 3. Cumulative probability of survival for those patients with a right ventricular ejection fraction either above (Group 2) or below (Group 1) 35%. These remain significantly different ($p < 0.05$) at 6, 12 and 24 months after radionuclide ventriculography (RVG).



The observation that radionuclide ventriculography not only aids in the diagnosis of congestive heart failure secondary to atherosclerotic disease (16) but also offers prognostic information prompted us to utilize this technique to identify patients with a significant reduction in left ventricular ejection fraction (< 40%) and consider them for inclusion in our study. Further segregation of our cohort into a group with normal or mildly reduced right ventricular ejection fraction and a matched group with significant impairment of right ventricular function was made on the basis of an ejection fraction value of 35%. This value had been chosen at the 99% confidence limit for our laboratory in order to maintain consistency with the left ventricular ejection fraction value of 40%, which is also at the 99% confidence limit for our normal subjects. The subsequent elimination of patients in the early postmyocardial infarction period was done to exclude patients with transient depression in right ventricular function (17). We excluded patients with a history of ethanol abuse or of uncorrected valvular disease so that the observed congestive cardiomyopathy was limited to a single etiology.

Prognostic role of right ventricular ejection fraction. The probability of survival for our patients (Fig. 4) was similar to that presented by other groups. For a similar cohort, Bruschke et al. (1) showed a 53% mortality at 12 months, which increased to 65% by 24 months compared with our values of 47 and 66%, respectively. Our survivors tended to have a shorter history from the time of the first myocardial infarction to the time of the radionuclide ventriculography and fewer signs or symptoms referable to right ventricular dysfunction. Even among nonsurvivors, right-sided failure was clinically evident in only 44% of cases. This finding is not surprising given that the therapeutic utilization of diuretic drugs is expected to reduce preload significantly, thereby obscuring the clinical signs of right-sided failure.

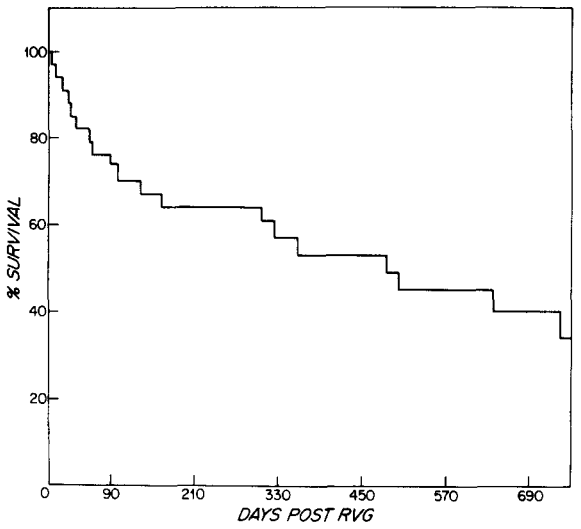


Figure 4. Cumulative probability of survival for the members of both groups. By 12 months, this has reached 53%, decreasing to 34% by 24 months after radionuclide ventriculography (RVG).

Although there was a higher prevalence of patients with chronic obstructive pulmonary disease among nonsurvivors, it was not statistically significant; the extent of cigarette use was also not significantly different for both groups. No history of previous pulmonary embolism was elicited in any of the members of our cohort; however, the presence of undetected episodes cannot be excluded.

The more significant depression of right ventricular function in the group of nonsurvivors, despite a similar depression in left ventricular function for both groups might, at first glance, appear contradictory. The electrocardiographic records for both groups demonstrate a higher prevalence of inferior infarction in the group of nonsurvivors, but in an amount insufficient to account for the group differences observed. Therefore, the possibility remains that the extent of left ventricular dysfunction may, in certain patients, express itself not only by the amount of left ventricular dyskinesia present, but also as an associated depression in right ventricular function.

Factors responsible for reduced right ventricular ejection fraction. Reduction in right ventricular ejection fraction is now thought to occur in association either with primary damage to the right ventricular wall (17–19) or with either chronic or acute elevations in right ventricular afterload (20,21). The first possibility cannot account for all the cases of right ventricular dysfunction because the prevalence of inferior infarction identifiable on the electrocardiogram in Group 1 was 35% and not statistically different from that of Group 2. Similarly, a high prevalence of right ventricular damage does not appear in large autopsy series of patients dying from the sequelae of coronary artery disease. It could also be argued that chronic elevations in afterload could be accounted for by either obstructive pulmonary disease or recurrent pulmonary embolism. The lack of any acute decompensation at the time of our evaluation makes it quite unlikely that an acute event is responsible for a transient elevation in right ventricular afterload. The last possibility would require that the left ventricle is at least partly responsible for the right ventricular dysfunction we observed.

Such an interaction has recently been shown during the acute phases of myocardial infarction. Marmor et al. (22) reported that patients in Killip classes II and III show a reduced right ventricular ejection fraction even when myocardial infarction is limited to the anterior wall of the left ventricle. This was not observed for patients in Killip class I. Furthermore, the extent of right ventricular dysfunction paralleled the extent of left ventricular dysfunction.

Such an effect, if present chronically, could account for our findings. This hypothesis becomes more acceptable if we examine the effect of segregating our patients into a cohort with or without depression of right ventricular ejection fraction. The extent of left ventricular dysfunction and the severity of the reduction in left ventricular ejection fraction are greater in patients with reduced right ventricular ejection fraction. It is unclear whether the mechanism by which the left ventricle affects the right ventricle is mediated by an increased right ventricular afterload or by direct mechanical interaction mediated by way of the left ventricular septum and the pericardium. Recent observations on the inverse correlation between right ventricular ejection fraction and pulmonary artery pressure favor the former possibility (23,24). The results of right-sided catheterization in 13 of our patients (Table 5) support this hypothesis. On the

Table 5. Right-Sided Catheterization Data for Patients With and Without Right Ventricular Dysfunction

	Depressed Right Ventricular Function (n = 8)	Preserved Right Ventricular Function (n = 5)	p Value
Pulmondary capillary wedge pressure (normal < 10 mm Hg)	31 0 ± 4 4	17.2 ± 8.9	≤ 0.01
Mean pulmonary artery pressure (normal 9 to 16 mm Hg)	40 8 ± 7.1	23 8 ± 6.8	≤ 0.005
End-systolic pulmonary artery pressure (normal 15 to 30 mm Hg)	58 8 ± 12.2	34.8 ± 9 4	≤ 0.005
Pulmonary vascular resistance (normal 20 to 120 dynes·s·cm $^{-5}$)	400 3 ± 206.7	127.6 ± 78 2	≤ 0.01

average, pulmonary capillary wedge pressure tended to be larger among patients with depressed right ventricular function. These patients also had a significant increase in both indexes of right ventricular overload, pulmonary artery pressures and pulmonary vascular resistances.

We conclude that a depressed right ventricular ejection fraction is an indicator of increased mortality among patients suffering from chronic congestive heart failure secondary to atherosclerotic heart disease. The decrease in left ventricular ejection fraction and the increase in the extent of left ventricular dyskinesia seen in these patients suggest that increased right ventricular afterload is the underlying physiologic process.

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