Arrhythmogenic right ventricular dysplasia: a generalized cardiomyopathy?

DANTE E. MANYARI, M.D., GEORGE J. KLEIN, M.D., SAJAD GULAMHUSEIN, M.D., DEREK BOUGHNER, M.D., GERARD M. GUIRAUDON, M.D., GEORGE WYSE, M.D., L. BRENT MITCHELL, M.D., WILLIAM J. KOSTUK, M.D.

With the technical assistance of Paul Purves, R.T.N.M.

ABSTRACT Arrhythmogenic right ventricular dysplasia (ARVD) is a recently described entity characterized by right ventricular myopathic changes and right ventricular tachycardia. The presence or extent of left ventricular dysfunction in ARVD is not known. We assessed right ventricular and left ventricular function and size in six patients with ARVD by echocardiography and radionuclide angiography done in patients at rest and during exercise. All patients had recurrent ventricular tachycardia of left bundle branch block morphology, and right ventricular origin of the ventricular tachycardia was confirmed by endocardial mapping in four patients. The results were compared with those of 10 normal subjects and five patients with Wolff-Parkinson-White syndrome taking amiodarone. The latter group was a control group, since we did not withhold amiodarone therapy in four patients with ARVD. Mean (± SD) right ventricular ejection fraction (EF) in patients with ARVD was 25 ± 11% at rest and 26 ± 12% during exercise. In normal subjects right ventricular EF was 51 ± 4% at rest and 59 ± 6% during exercise (p < .001). The right ventricular/left ventricular end-diastolic diameter ratio was 0.60 ± 0.24 in patients with ARVD and 0.37 ± 0.10 in normal subjects (p < .05). Right ventricular/left ventricular end-diastolic volume ratio was 2.41 ± 1.05 in patients with ARVD and 1.16 ± 0.21 in normal subjects (p < .001). Measured in patients at rest, a subnormal left ventricular EF was present in two patients with ARVD but an abnormal left ventricular EF was present in all six patients during exercise. Mean left ventricular EF in patients with ARVD was 57 ± 8% at rest and 55 ± 10% during exercise (p > .05). In normal subjects, left ventricular EF was 61 ± 4% at rest and 72 ± 5% during exercise (p < .001). New left ventricular wall motion abnormalities were seen during exercise in all but one patient with ARVD. At rest and exercise, left ventricular and right ventricular EF in patients with Wolff-Parkinson-White syndrome were similar (p > .05) to those of normal subjects. We conclude that right ventricular dysfunction predominates in patients with ARVD but latent left ventricular dysfunction is present more often than is commonly recognized. These findings may have important diagnostic and therapeutic implications.


RIGHT VENTRICULAR DYSPLASIA is a pathologic condition in which the right ventricular myocardium is totally or partially replaced by fatty and fibrous tissue.1–11 The clinical expression of this condition may depend on the extent of the pathologic changes. If involvement of the right ventricle is extensive, cardiomegaly and congestive cardiac failure may be present.5, 7, 11, 12 When dysplasia is limited to portions of the right ventricle, depression of cardiac function may be minimal or absent and clinically nonsignificant.3, 10 Patients may present with premature ventricular complexes or ventricular tachycardia of right ventricular origin independent of the extent of pathologic change and of the degree of diminished ventricular function.1, 2, 4, 8, 10, 13–21 When ventricular tachycardia is the principal manifestation, this condition is known as arrhythmogenic right ventricular dysplasia (ARVD). ARVD is recognized clinically by recurrent ventricular
tachycardia of right ventricular origin and by segmental or generalized right ventricular cardiomyopathy. 10, 18, 20

Isolated cases have been reported that indicate that areas of the heart other than the right ventricle may also be involved to a lesser extent. 4, 5, 8, 10, 22 However, the incidence of anatomic and functional left ventricular involvement in ARVD remains unknown. Our objective was to prospectively assess both right and left ventricular systolic function at rest and during exercise in patients with ARVD with ECG-gated blood cardiac scintigraphy23, 24 and echocardiography. 25, 26

Material and methods

Study population. Six consecutive patients with ARVD referred to the participating electrophysiologic laboratories constituted the study group (group 1). There were four men and two women with a mean age of 41 years (range 21 to 60). The diagnosis of ARVD was based on recurrent ventricular tachycardia of left bundle branch block morphology in the absence of clinical or angiographic evidence of coronary artery disease or other organic heart disease. All patients had segmental or global right ventricular wall motion abnormalities documented angiographically. Right ventricular origin of the tachycardia was confirmed at electrophysiologic study in four patients. Medications could not be safely discontinued in all patients; at the time of this investigation, four patients were receiving amiodarone (400 to 800 mg/day) for the control of ventricular tachycardia.

A control group (group 2) consisted of 10 asymptomatic subjects, 28 to 56 years of age (mean 38), who underwent an identical study protocol to establish the limits of normality. There were six men and four women. None had clinical, electrocardiographic, or radiographic evidence of heart disease or conditions known to affect cardiac structure and/or function.

Another control group (group 3) consisted of five patients who were taking 400 to 800 mg of amiodarone per day. In this group there were four men and one woman with a mean age of 45 years (range 36 to 53). These patients had Wolff-Parkinson-White syndrome but no clinical, electrocardiographic, echocardiographic, or angiographic evidence of associated cardiovascular disease. This group was selected to compare the effects of amiodarone on ejection fractions (EFs) determined at rest and during exercise, since four patients with ARVD were taking amiodarone and the effect of this medication on rest and exercise ventricular function is not known.

Methods. In the first two groups (patients with ARVD and normal subjects) right and left ventricular function and size were assessed with M mode echocardiography performed at rest and radionuclide angiography performed at rest and during exercise. In the third group (patients with the Wolff-Parkinson-White syndrome) cardiac function was assessed with radionuclide angiography performed at rest and during exercise.

Standard equipment and techniques26 were used to record echocardiograms. Echocardiographic measurements of cavity size and wall thickness were made according to the recommendations of the American Echocardiographic Society. 26 Since paradoxic septal motion was observed in several patients with ARVD, the posterior wall velocity27 was used to quantify left ventricular function on M mode echocardiograms. This measurement was taken directly from the posterior left ventricular endocardium as the amplitude of endocardial motion divided by the ejection time.27

Radionuclide angiography was carried out with the ECG-multigated blood pool method. Equipment and techniques have been described previously. 23 Briefly, after in vivo labeling of the red blood cells with technetium-99m, blood pool cardiac scintigraphy was performed in the modified left anterior oblique projection that best separated both ventricles and atria. Acquisitions of data were performed in duplicate in patients at rest and during maximal supine bicycle ergometer exercise. Left ventricular global EF was calculated with the time-activity curve generated by an automated edge-detection program that defined new left ventricular regions of interest for each frame of the cardiac cycle. Right ventricular EF was calculated by similar methods with manually defined right ventricular regions of interest at end-diastole and end-systole. 28 Right ventricular/left ventricular end-diasstolic volume ratio was calculated in each patient as the ratio between the normalized (that is, corrected for frame duration and number of beats processed) right ventricular end-diastolic counts/left ventricular end-diastolic counts. Segmental right and left ventricular function was analyzed by subjective assessment of the endless-loop movie format24 and such computer image manipulations as the stroke-volume image. 29, 30 EF image, 29 and superimposition of end-diastolic and end-systolic perimeters. 31 Five segments (figure 1) were classified as normal, hypokinetic, akinetic, or dyskinetic (scored 0, 1, 2, and 3, respectively). Segment scores were then added and the wall motion of each ventricle was expressed numerically (with a minimum of 0 and a maximum of 15).

In our laboratory, radionuclide left ventricular EF and wall motion score (WMS) correlate closely with contrast studies (r = .87); the standard error of the estimate for EF is 0.05. 30 Interobserver and intraobserver variability of EF measurements showed correlation coefficients of .92 and .97, respectively. 23 A radionuclide ventriculogram taken in a patient at rest was considered normal if left ventricular EF was at least 55%. Right ventricular EF was at least 45%, and the WMS was equal to zero. Failure of EF to increase by at least 7% (absolute value) and/or an increase in WMS from resting values defined an abnormal ventricular function response to exercise. The 95% confidence limits for repeated individual EF was ± 6.9%. EF changes of less than 7% may be attributed to physiologic and statistical noise. 23.

Statistical methods. All echocardiographic and radionuclide measurements were performed independently by two observers blinded to the clinical status of the patients. The two measurements were averaged and the mean value was used in statistical analysis. Analysis of variance determined the significance of differences in EF and WMS among the three groups. Post hoc analysis was performed with Tukey’s multiple comparison procedure when appropriate. Echocardiographic measurements of patients with ARVD and those of normal subjects were compared by the Student’s t test. Probability of 5% or less was used to reject the null hypothesis.

Results

Exercise studies were completed without complication in all participants. Although the mean exercise duration was slightly lower in patients with ARVD, no significant differences in mean exercise duration were noted among the three groups. Patients with ARVD exercised for 8.5 ± 3.5 min (mean ± SD), normal subjects exercised for 11.1 ± 2.9 min, and patients with Wolff-Parkinson-White (W-P-W) syndrome exercised for 12.0 ± 2.1 min. The mean heart rate, systolic blood pressure, and rate-pressure product in
patients at rest as well as during peak exercise were similar in the three groups. All subjects and patients terminated exercise because of fatigue; no significant arrhythmias were observed during or after exercise.

**Right ventricular function and size.** In patients with ARVD, end-diastolic diameter determined by echocardiography, right ventricular/left ventricular end-diastolic diameter ratio, and right ventricular/left ventricular end-diastolic volume ratio were larger than those in normal subjects (table 1). Five of the six patients with ARVD had right ventricular end-diastolic diameters greater than our upper limit of normal (26 mm).

In patients at rest, the mean EF was markedly lower in patients with ARVD than in normal subjects or patients with W-P-W syndrome (table 1). Five of the six patients with ARVD, but none of the other subjects, had an EF lower than 45% (figures 2 and 3). Similarly, the average WMS during rest was significantly higher in patients with ARVD. An abnormal WMS was present in each patient with ARVD (figure 4), although in the patient with a normal EF during rest (46%), the WMS was nearly normal (1). All segments of the right ventricle demonstrated abnormal motion in patients at rest in two patients with ARVD. The right ventricular WMS was normal or nearly normal in all normal subjects and patients with W-P-W syndrome.

At peak exercise, patients with ARVD had significantly lower mean EF and significantly higher mean WMS than normal subjects or patients with W-P-W syndrome (table 1). Mean EF increased significantly from rest to exercise in normal subjects and in patients with W-P-W syndrome (p < .001) but not in patients

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**TABLE 1**

Summary of the results in the three groups studied

<table>
<thead>
<tr>
<th></th>
<th>ARVD</th>
<th>Normal subjects</th>
<th>W-P-W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-diastolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>diameter (mm)</td>
<td>32.0 ± 11.0°</td>
<td>17.4 ± 4.2</td>
<td>—</td>
</tr>
<tr>
<td>EF-R (%)</td>
<td>25.3 ± 11.4°</td>
<td>50.7 ± 4.3</td>
<td>51.8 ± 4.3</td>
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<tr>
<td>EF-Ex (%)</td>
<td>26.0 ± 11.8°</td>
<td>59.2 ± 5.6</td>
<td>61.0 ± 4.1</td>
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<tr>
<td>WMS-R</td>
<td>4.33 ± 2.34°</td>
<td>0.20 ± 0.42</td>
<td>0.20 ± 0.45</td>
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<tr>
<td>WMS-Ex</td>
<td>5.67 ± 2.07°</td>
<td>0.10 ± 0.32</td>
<td>0.00 ± 0.00</td>
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<tr>
<td>Left ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-diastolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>diameter (mm)</td>
<td>52.5 ± 7.3</td>
<td>47.2 ± 5.0</td>
<td>—</td>
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<tr>
<td>End-systolic</td>
<td>37.3 ± 6.6</td>
<td>33.4 ± 4.6</td>
<td>—</td>
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<tr>
<td>diameter (mm)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Posterior wall</td>
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<td></td>
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<tr>
<td>velocity (mm/sec)</td>
<td>32.8 ± 4.9</td>
<td>36.2 ± 4.1</td>
<td>—</td>
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<tr>
<td>EF-R (%)</td>
<td>57.5 ± 7.8</td>
<td>61.3 ± 4.3</td>
<td>61.2 ± 7.3</td>
</tr>
<tr>
<td>EF-Ex (%)</td>
<td>55.0 ± 9.9°</td>
<td>72.1 ± 5.4</td>
<td>71.8 ± 5.5</td>
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<tr>
<td>WMS-R</td>
<td>0.50 ± 0.84</td>
<td>0.20 ± 0.42</td>
<td>0.00 ± 0.00</td>
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<tr>
<td>WMS-Ex</td>
<td>2.00 ± 1.41°</td>
<td>0.00 ± 0.00</td>
<td>0.20 ± 0.45</td>
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<tr>
<td>Ratios</td>
<td></td>
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</tr>
<tr>
<td>RV/LV end-diastolic</td>
<td>0.60 ± 0.24°</td>
<td>0.37 ± 0.10</td>
<td>—</td>
</tr>
<tr>
<td>RV/LV end-diastolic volume</td>
<td>2.41 ± 1.05°</td>
<td>1.16 ± 0.21</td>
<td>1.09 ± 0.16</td>
</tr>
</tbody>
</table>

All values are mean ± SD.

RV = right ventricular; LV = left ventricular; R = rest; Ex = peak exercise.

Statistical comparisons: *p < .05; †p < .01; ‡p < .001 vs normal subjects.
with ARVD. No patient with ARVD responded to exercise with an adequate rise of the EF, although all patients with W-P-W syndrome and all but one normal subject had an increase in EF by at least 7% (figures 2 and 3). Mean WMS increased significantly from rest to exercise only in patients with ARVD (p < .01), but not in normal subjects or patients with W-P-W syndrome. Individual results of WMS in patients with ARVD are shown in figure 4. Three patients with ARVD had abnormal wall motion detected in all segments of the right ventricle during exercise. The only normal subject whose EF failed to increase by at least 7% (46% at rest and 52% at peak exercise) had a normal WMS (0) at rest and during exercise.

Five of the six patients with ARVD showed obvious right ventricular dysfunction at rest, whereas global and segmental right ventricular dysfunction was present in all six patients during peak exercise.

**Left ventricular function and size.** Mean left ventricular end-diastolic and end-systolic diameters determined by echocardiography were somewhat larger in patients with ARVD than in normal subjects, but the differences did not reach statistical significance (table 1). Two patients with ARVD, but no other subject, had left ventricular end-diastolic diameters larger than the upper limit of normal for adults in our laboratory (55 mm). These two individuals were those with generalized right ventricular dysfunction and paradoxical septal motion.

In patients at rest, mean EF was lower in patients with ARVD than in normal subjects and patients with W-P-W syndrome; mean posterior wall velocity was lower in patients with ARVD than in normal subjects, but the differences were not statistically significant (table 1). Left ventricular EF was lower than 55% in two patients with ARVD (51% and 49%) and in one patient with W-P-W syndrome (53%). All normal subjects had left ventricular EF greater than 55% (figures 3 and 5). Similarly, although mean WMS was slightly higher in patients with ARVD than in the other two groups, no significant differences were noted. Individual results of WMS in patients with ARVD are shown in figure 4. Those with WMS of 1 and 2 were the same patients who had an EF of 49% and 51%, respectively.

During peak exercise, patients with ARVD had significantly lower mean EF and significantly higher mean WMS than normal subjects or patients with W-P-W syndrome (table 1). Mean EF increased significantly from rest to exercise in normal subjects and in patients with W-P-W syndrome (p < .001) but not in patients with ARVD. All normal subjects but one and all patients with W-P-W syndrome had an increase in EF by at least 7%, although no patient with ARVD

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

**FIGURE 2.** Group and individual results of right ventricular (RV) EF at rest (R) and during exercise (Ex) in patients with arrhythmogenic right ventricular dysplasia (ARVD) and in normal subjects. *p < .05 vs patients with ARVD.

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

**FIGURE 3.** Group and individual EF values in patients with W-P-W syndrome taking 400 to 800 mg/day of amiodarone, at rest (R) and during peak exercise (Ex). In all patients, EF in the right ventricle (RV) and left ventricle (LV) increased normally with exercise. *p < .05 vs rest.

![Figure 4](http://circ.ahajournals.org/)

**FIGURE 4.** Individual and group results of WMS in the right ventricle (RV) and left ventricle (LV) in patients with arrhythmogenic right ventricular dysplasia at rest (R) and during exercise (Ex). *p < .05 vs rest.
had a normal EF response to exercise (figures 3 and 5). Mean WMS increased significantly from rest to exercise in patients with ARVD (p < .001) but not in the other two groups. Individual values of WMS in patients with ARVD are depicted in figure 4. Left ventricular WMS was normal or nearly so in every normal subject and in every patient with W-P-W syndrome. The only normal subject who did not have an increase in EF of at least 7% (64% at rest and 68% during exercise) had a normal WMS when at rest and during peak exercise.

Two of the six patients with ARVD exhibited segmental left ventricular dysfunction (hypokinesis) in measurements taken while at rest, with the dysfunction confined to the interventricular septum. During exercise, five patients with ARVD had segmental left ventricular dysfunction (hypokinesis and akinesis) involving the interventricular septum in all five patients and the inferoapical segment in four patients. In only one patient with ARVD, the WMS was 0 at rest as well as during peak exercise, but in this patient, left ventricular EF failed to increase significantly from rest (68%) to exercise (71%).

Discussion

It is generally believed that the myopathic abnormalities in ARVD are confined to the right ventricular myocardium. Although involvement of the left ventricle has been described in isolated cases, the frequency of left ventricular dysfunction in ARVD is not known. In this study, we found that latent left ventricular dysfunction, uncovered during exercise studies, was present in all patients with ARVD.

To avoid selection bias, this investigation was carried out prospectively in consecutive patients with ARVD referred to the participating institutions. Accepted criteria were used to make the diagnosis of ARVD. Ventricular tachycardia of right ventricular origin was documented in all patients who underwent electrophysiologic studies. As in previous studies, there was a preponderance of men in our series of patients.

Since we considered it ethically unacceptable to withhold antiarrhythmic therapy from patients with life-threatening arrhythmias, we used alternative ways to verify that the results obtained in patients with ARVD were not due to the antiarrhythmic drug (amiodarone) taken. Five patients with W-P-W syndrome who had tachyarrhythmias uncontrolled by conventional therapy were taking comparable doses of amiodarone. They had no evidence of associated cardiovascular disease and their mean age was similar to that of patients with ARVD. The normal right and left ventricular function at rest and during exercise in these patients argue against any significant effect of amiodarone.

Right ventricular function and size. Our findings are in accord with previous descriptions of ARVD. The increased right ventricular dimensions and the presence of global or segmental right ventricular dysfunction during resting studies, in the absence of cardiorespiratory conditions known to affect right ventricular anatomy and/or function, constitute diagnostic landmarks of ARVD. Definite right ventricular dysfunction was not present at rest in only one of our patients with ARVD. However, all showed segmental or global right ventricular dysfunction at peak exercise. Therefore radionuclide ventriculography performed in patients at rest and during exercise may be useful as a noninvasive test to support the diagnosis of ARVD.

Global or segmental functional impairment of the right ventricle in patients with ARVD is presumably due to the absence of or decrease in normal myocardial fibers in the segments involved. In patients with ARVD, normal myocardium is replaced by fatty tissue and a variable amount of intramyocardial fibrosis; fatty tissue and the intramyocardial fibrosis are noncontractile tissue. Previous pathologic studies and observations made during surgical treatment of recurrent ventricular tachycardia have found the most frequent areas of dysplasia to be the anterior infundibulum, the right ventricular apex, and the inferior wall of the right ventricle. Our findings confirmed this pattern of distribution. Our six patients with ARVD had hypokinesis (one patient), akinesis (four patients), or dyskinesis (one patient) of the apical and inferior segments of the right ventricle. All right ventricular segments showed wall motion abnormalities in two patients at
rest and in three patients during exercise. Although an enlarged right ventricle was found in all 22 patients with ARVD reported by Marcus et al.,10 generalized right ventricular dysplasia and/or dysfunction was not described in any of their patients. This discrepancy could be explained because Marcus et al.10 did not perform exercise studies. The stress of exercise was an important part of our investigation; it revealed new wall motion abnormalities not readily apparent during studies done in patients at rest. It can be postulated that segments with early dysplasia may show normal ventricular function in patients at rest, whereas exercise studies may be needed to uncover the actual extent of right ventricular dysplasia. This possibility is important when surgical treatment is performed for recurrent ventricular tachycardia unresponsive to drug therapy. Most surgical procedures are designed to electrically isolate or ablate all possible sites of origin of the ventricular tachycardia.10, 13–19, 33, 34 Knowledge of all involved segments is therefore not only of academic interest but also has important clinical implications.

Left ventricular function and size. Group values of end-diastolic or end-systolic diameter in our patients with ARVD were not increased, although end-diastolic diameter was slightly larger than normal in two patients. Interestingly, these two patients exhibited generalized dysfunction of the right ventricle.

Contrary to previous publications, our investigation documented evidence of left ventricular dysfunction in all patients with ARVD. Segmental contraction abnormalities were located in the interventricular septum in five patients and in the inferoapical region in four patients. In the largest series published to date,10 only one of 22 patients showed left ventricular contraction abnormalities. The greater frequency of left ventricular systolic dysfunction observed in our study is probably related to the method used to assess left ventricular function. Marcus et al.10 used contrast ventricular angiography; in some individuals the contrast injection was made into the right ventricle and followed to the levophase. In our study, we used radionuclide ventriculography taken in patients at rest and during supine exercise. Exercise studies have allowed investigators to uncover latent ventricular dysfunction in patients with coronary artery disease and other heart diseases when ventricular dysfunction was not apparent in patients at rest.33, 34, 35 Resting studies showed normal left ventricular function in the majority of our patients with ARVD, whereas evidence of latent left ventricular dysfunction was uncovered only during exercise studies.

Three possible mechanisms could account for the abnormal left ventricular systolic function of patients with ARVD. First, the left ventricular myocardium may be involved with the same dysplasia that affects the right ventricle. We observed that the two individuals with the lowest left ventricular EF values during exercise also had slightly enlarged left ventricular end-diastolic diameters and generalized right ventricular involvement; this supports the first mechanism by suggesting that left ventricular involvement is more likely (or more extensive) in patients with widespread right ventricular dysplasia. Second, left ventricular dysfunction may be secondary to the enlarged and poorly functioning right ventricle. There is evidence of a close interrelation between the functions of the right and left ventricles,56 and marked dysfunction of one chamber could lead to apparent dysfunction in the other. Although such an argument may be an appropriate explanation for decreased global left ventricular function (that is, EF), it is a less tenable hypothesis to explain regional left ventricular dysfunction, particularly if segments not contiguous to the right ventricle are involved. Third, it is conceivable that recurrent ventricular tachycardia in these patients could have produced recurrent episodes of hypotension that lead to subendocardial hypoxia and subclinical left ventricular "damage." Detailed pathologic studies will be needed to establish which mechanisms are operational and to improve our understanding of the left ventricular dysfunction observed in these patients.

The results of our study are relevant to the recently described surgical technique known as "right ventricular free wall disconnection,"33, 37 which treats recurrent ventricular tachycardia unresponsive to conventional therapy in patients with ARVD. In this technique, the right ventricular free wall is disconnected from the interventricular septum and the left ventricle to confine any arrhythmic activity arising from the right ventricle to that chamber. When the arrhythmogenic segments are confined to the right ventricular free wall, this surgical technique should prove effective. However, uniform involvement of the left ventricle in ARVD raises the possibility of recurrence of ventricular tachycardia with progression of the disease process.

In summary, rest–exercise studies of ventricular function in our patients with ARVD have confirmed global and segmental right ventricular dysfunction to be present in all cases. Obvious abnormalities of right ventricular function were detected in most patients at rest. Latent left ventricular dysfunction was detected in all patients with ARVD during exercise. Left ventricular function was normal in most patients at rest.
Whether or not ARVD represents a generalized cardiomyopathy with predominant right ventricular involvement is not clear from our data.

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References

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