Findings on Magnetic Resonance Imaging of Idiopathic Right Ventricular Outflow Tachycardia

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We evaluated 20 patients with idiopathic ventricular tachycardia for structural abnormalities using magnetic resonance imaging (MRI) and compared them with 20 controls. Two experienced observers interpreted the MRIs. There were no differences in incidence of qualitative MRI findings in patients compared with controls. These findings do not favor an association between anatomic abnormalities and arrhythmia in these patients. ©2004 by Excerpta Medica Inc.

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In this study, we used black-blood, double inversion-recovery magnetic resonance imaging (MRI), and cine MRI to screen a homogeneous population of patients with idiopathic ventricular tachycardia (VT). The purpose of the study was threefold: (1) to determine the incidence of intramyocardial hyperintense signals and wall thinning in idiopathic VT, (2) to determine the effect of blinded versus unblinded interpretation, and (3) to quantitatively evaluate right ventricular (RV) global and regional function in this patient group.

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The study group consisted of 20 consecutive patients with idiopathic VT arising from the RV outflow tract evaluated at The Johns Hopkins Hospital. Patients were prospectively enrolled if they met the following inclusion criteria: (1) a normal 12-lead electrocardiogram at rest, (2) no evidence of structural heart disease on a 2-dimensional echocardiogram, (3) a normal signal-averaged electrocardiogram, and (4) origin of the arrhythmia to the RV outflow tract localized on electrophysiologic study. MRI was performed according to a standard protocol that included fast-spin echo and gradient echo cine sequences. Twenty age- and gender-matched healthy volunteers underwent MRI with the same protocol and served as the control group. The study was approved by the institutional review board, and all subjects gave informed consent.

TABLE 1 Baseline Characteristics of Patients With Idiopathic Ventricular Tachycardia (VT) and Control Subjects

Variable	Patients (n = 20)	Controls (n = 20)
Gender		
Men (n)	5 (25%)	8 (40%)
Women (n)	15 (75%)	12 (60%)
Mean age (yrs)	38 ± 12	33 ± 8
Range	14–61	23–46
Symptoms		
Palpitations	13 (65%)	0
Chest pain	2 (10%)	0
None	3 (15%)	100 (100%)
History of sustained VT	5 (25%)	0
Systemic hypertension	2 (10%)	0
Electrocardiogram (T-wave inversions)		
V_1 only	10 (50%)	—
V_1 and V_2	0	—
$>V_2$	0	—
Signal-averaged electrocardiographic abnormalities	0	—
Echocardiographic abnormalities	0	—
Holter monitoring		
Nonsustained VT	10 (50%)	—
Frequent ventricular premature contractions	10 (50%)	—
Exercise treadmill test		
Ectopy suppressed during exercise	15 (75%)	—
Nonsustained VT	5 (25%)	—

MRI was performed using a 1.5-T scanner (CV/i, General Electric Medical Systems, Waukesha, Wisconsin) using electrocardiographic gating and a 4-element cardiac phased-array surface coil. Double inversion recovery (blood suppression) fast-spin echo MRI was acquired in the transaxial and short-axis planes with and without fat suppression (time to repitition [TR] 1-RR interval, time to echo [TE] 5 ms, slice thickness 5 mm, interslice gap 5 mm, and field-of-view 24 to 28 cm). Bright blood cine imaging in the axial and short-axis planes was acquired by a breath-hold, steady-state free precession pulse sequence (TR 3.5, TE 1.2 ms, flip angle 45°, slice thickness 8 mm, interslice gap 4 mm, and field-of-view 36 to 40 cm [20 views/segment], temporal resolution ≤50 ms). MRI datasets were transferred to an Advantage Workstation (General Electric Medical System) for analysis. The images were qualitatively and independently evaluated by 2 experienced MRI physicians. One of the physicians (DAB) reported the images in a clinical setting (unblinded) and the other physician (VAF) was blinded to all clinical histories. MRI readers filled out a questionnaire that included qualitative reporting of

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TABLE 2 Magnetic Resonance Imaging Abnormalities in the Study Group

	Blinded		Unblinded		
	Patients	Controls	Patients	Controls	Agreement (%)
Qualitative analyses					
RV abnormalities					
Intramyocardial fat	0	0	2 (10%)	0	38/40 (95%)
Wall thinning	0	0	0	0	40/40 (100%)
Global dysfunction	0	0	0	0	40/40 (100%)
Cavity dilation	0	2 (10%)	1 (5%)	0	37/40 (92%)
Regional dysfunction	1 (5%)	0	1 (5%)	0	38/40 (95%)
Outflow tract enlargement	1 (5%)	0	3 (15%)	0	38/40 (95%)
Total abnormalities	2 (10%)	2 (10%)	7 (35%)	0	33/40 (82%)
					Correlation Coeffecient
Quantitative analyses					
Right ventricle					
End-diastolic volume (ml)	152 ± 21	160 ± 20	156 ± 22	168 ± 24	97%
Ejection fraction (%)	57 ± 7	59 ± 6	56 ± 7	57 ± 4	95%
Outflow tract area (cm^2)	9.2 ± 1.5	9.0 ± 1.7	9.0 ± 1.5	8.9 ± 1.5	90%
End-diastolic diameter (mm)	3.6 ± 0.6	3.9 ± 0.3	3.9 ± 0.4	3.7 ± 0.4	92%
Left ventricle					
End-diastolic volume (ml)	146 ± 21	153 ± 20	142 ± 23	149 ± 22	95%
Ejection fraction (%)	63 ± 9	62 ± 6	62 ± 8	60 ± 7	93%

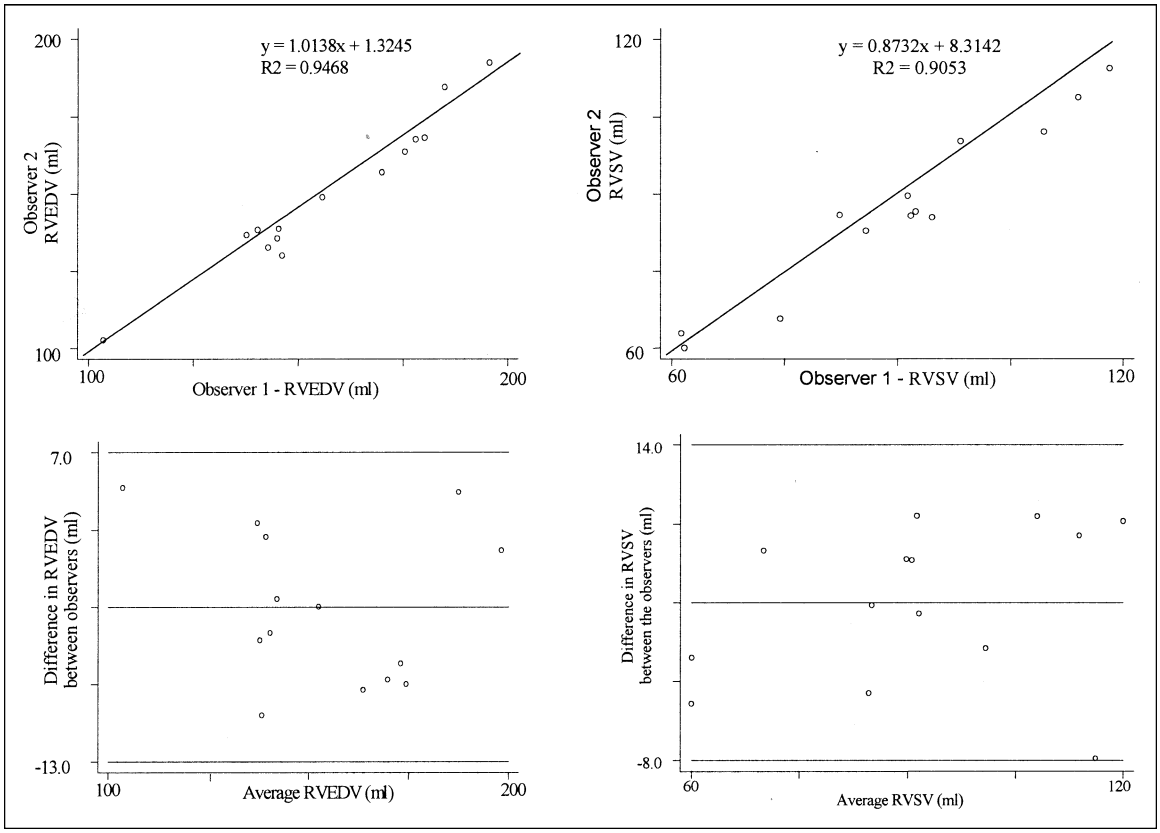

FIGURE 1. Reproducibility for quantitative analysis. *(A)* Correlation of the measurements between the 2 observers for RV end-diastolic volume and RV stroke volume. *(B)* Bland-Altman analysis showing excellent agreement between the 2 observers for RV end-diastolic volume (RVEDV). Limits of agreement are displayed as ±2 SDs. Difference, −3.4 ± 3 ml; upper and lower limits of agreement, 6.5 ml and −13 ml, respectively; RV stroke volume (RVSV) difference, −3.1 ± 3 ml; upper and lower limits of agreement, 7 ml and −14 ml, respectively.

intramyocardial fat, wall thinning, and RV global and regional function. The right ventricle was divided into 3 regions on the axial black blood images (RV free wall, RV apex, and RV outflow tract). Each segment was individually assessed for the presence or absence of fat by a graded scale of 1 to 5, (1 = definitely absent and 5 = definitely present). Wall thinning was also assessed on the axial black-blood images. Qual-

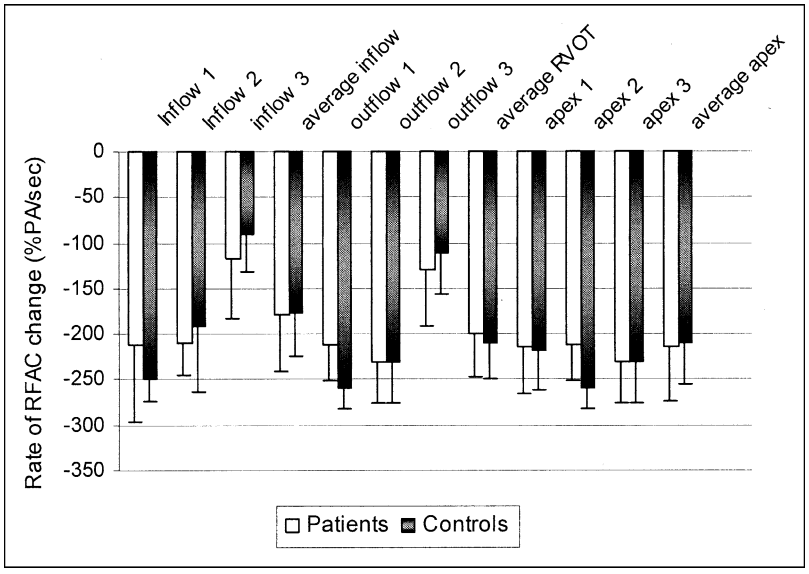

FIGURE 2. Results of regional function analysis. Rate of regional fractional area change was similar in patients and controls in all segments assessed at the level of the RV inflow, the outflow tract, and the apex. PA = percent area change; RFAC = regional fractional area change; RVOT = RV outflow tract.

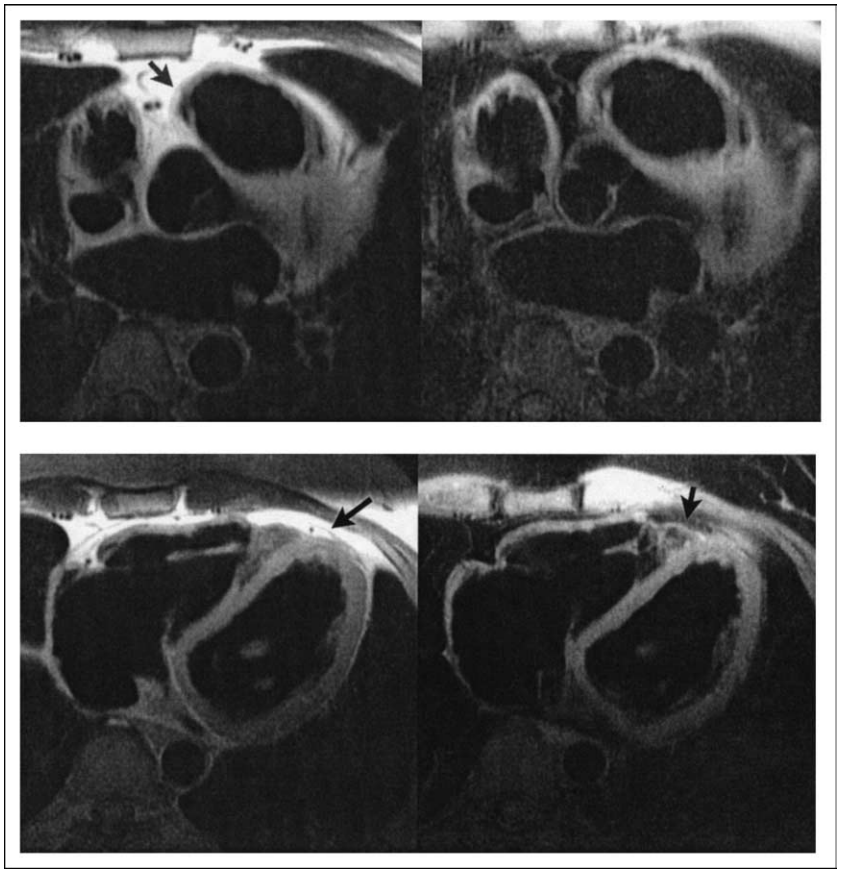

FIGURE 3. *Top left,* double-inversion recovery fast-spin echo RV outflow tract of a patient with idiopathic VT who was categorized as indeterminate after the first reading (without fat suppression). Epicardial border of RV wall is obscured by fat, mimicking partial fat replacement *(black arrow). Top right,* fat-suppressed image from the same subject showing a normal RV wall. *Bottom left,* black-blood image from a control subject showing normal RV wall and normal fat distribution at the RV apex *(black arrow). Bottom right,* fat-suppressed image from the same control subject. Note the extremely thin RV wall close to the apex, which is normal *(black arrow).*

itative RV global and regional function was evaluated using the cine images.

Quantitative analysis was performed using the software program MASS (Medis, The Netherlands) by 2 other observers (HT and CB) independently. RV dimensions were measured on 4-chamber cine images. Endocardial margins of the ventricles were manually contoured, and volumes were calculated using a summation of the disks method ("Simpson's Rule"). For regional function analysis, the endocardial border of the right ventricle was manually contoured frame by frame in all phases of the cardiac cycle on the axial gradient echo images at 3 levels (RV apex, RV inflow level, and RV outflow tract). The RV anterior wall in all levels was then divided into 3 equal segments. For each segment, cavity area was measured frame by frame during each cardiac phase, and the rate of regional fractional area change was calculated.

Data were analyzed using STATA version 7 (Stata Corp. College Station, Texas). Data are expressed as mean ± SD. Fisher's exact test was used to assess differences in the frequency of positive findings between groups. Agreement for qualitative measures was assessed by percent agreement and for quantitative measures by correlation coefficients. In addition, a Bland-Altman analysis was performed to determine agreement between the 2 observers measuring the same quantity.

To assess the accuracy of the readers in detecting intramyocardial fat and wall thinning, black blood MRI of 9 additional patients with idiopathic VT (not included in the study) was compared with 9 patients with arrhythmogenic RV dysplasia (ARVD) (diagnosed by Task Force criteria) in a blinded fashion. Intramyocardial fat on MRI was identified in 8 of the 9 patients with ARVD (88%) and in none of the patients with idiopathic VT. Wall thinning was observed in 1 patient with ARVD and in none of the patients with idiopathic VT (100% agreement). The overall agreement between the observers

FIGURE 4. Utility of fat suppression. Results of blinded reading for patients are shown. Assessment of fat infiltration was performed twice, once without and once with inspection of fat-suppressed images. The addition of the fat-suppressed version decreased the number of intermediate or nondiagnostic results and improved observer confidence in reporting absence of intramyocardial fat.

was 88%. The specific agreement for the presence of intramyocardial fat was 85% and for the absence of intramyocardial fat was 91%.

The baseline characteristics of the study population are listed in Table 1. Sustained monomorphic VT was induced in 2 patients, and nonsustained VT was induced in 6 patients. VT was induced by atrial and/or ventricular overdrive pacing within a range of cycle lengths consistent with a triggered or automatic mechanism. The remaining 12 patients had only frequent premature ventricular contractions induced. Isoproterenol was required for VT induction in 12 patients. The morphology of VT was left bundle inferior axis consistent with an origin in the RV outflow tract.

Table 2 lists the results of qualitative evaluation of the MRI. A blinded analysis of the images revealed no evidence of fat infiltration, wall thinning or global RV dysfunction in either the patients or the control subjects. The unblinded reader reported a higher frequency of abnormal MRI findings in patients compared with interpretations of the blinded reader (7 of 20 vs 2 of 20, $p < 0.01$). In contrast, the total number of abnormalities reported in the controls was not different between blinded and unblinded interpretations (2 of 20 vs 0 of 20, p = NS). The interobserver agreement was good for all qualitative parameters evaluated.

Results of blinded and unblinded quantitative MRI analysis in patients and controls are shown in Table 2. No differences were observed in the results of quantitative MRI analysis between the blinded and unblinded readers. There was excellent correlation between the observers (3 and 4) for quantitative analyses (Figure 1). Bland-Altman plots for interobserver agreement for quantitative analyses are shown in Figure 1.

Quantitative analysis of the RV regional function was performed at 3 levels: the RV inflow, RV outflow, and the apex. The RV anterior wall at each level was further divided into 3 equal segments, resulting in a total of 9 segments analyzed for the entire right ventricle. Rate of regional fractional area change for each of the 9 segments assessed is shown in Figure 2. No difference was observed in regional RV function among patients with idiopathic VT and controls.

Transaxial inversion recovery fast-spin echo images of a patient at the level of the RV outflow tract are shown in Figure 3. In the non–fat-suppressed image (Figure 3), the anterior wall of the right ventricle is partially obscured by epicardial fat. The improved contrast from simultaneously nulling the blood signal and the epicardial fat (Figure 3) at the same level delineates the epicardial border of the right ventricle. Figure 3 was obtained from a control subject. The RV wall is obscured by fat at the RV apex in Figure 3, mimicking apical fat infiltration. The fat-suppressed image at the same level (Figure 3), delineates the true epicardial border. Diagnostic benefit of fat suppression was assessed by reporting of fat infiltration first with the non–fat-suppressed image, and the second time with both the non–fat-suppressed image and its fat-suppressed version by the blinded reviewer. Results of the readings for the patients are shown in Figure 4. Five patients who had an indeterminate, nondiagnostic report after the first reading became diagnostic with the second reading, as noted by a change to "definitely absent" in 3 patients, and to "possibly absent" in the remaining 2 patients. Compared with non–fat-suppressed images only, the total number of indeterminate results was decreased by adding fat suppression (5 of 20 vs 0 of 20, $p < 0.001$).

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The results of this study reveal that patients with idiopathic VT who have a normal surface electrocardiogram and signal-averaged electrocardiogram do not have evidence of structural heart disease on MRI, and are indistinguishable from controls. Quantitative evaluation of the right ventricle revealed no differences in the global and regional RV function in idiopathic VT compared with control subjects. These results argue against the hypothesis that idiopathic VT may represent an early form of ARVD.

The findings of this study confirm and extend the results of a previous study by Grimm et al,[1] but are in contrast to the results of several other clinical studies that have reported structural abnormalities limited to the right ventricle in patients with idiopathic VT.[2–6] Carlson et al[3] were the first to report MRI abnormalities in 21 of 22 patients (95%) with idiopathic VT. The abnormalities were limited to the cine MRI, and no patient had intramyocardial hyperintense signals in this study. Using a comparable imaging protocol and patient selection criteria, White et al[4] reported MRI abnormalities in 32 of 53 patients (60%). In addition to cine MRI abnormalities, 25% of the patients also had intramyocardial hyperintense signals indicative of fat infiltration. Similarly, Markowitz et al[2] reported intramyocardial hyperintense signals in 4 of 14 patients (29%) with adenosine-sensitive VT. These studies did not exclude patients with 12-lead electrocardiographic abnormalities, and the signal-averaged electrocardiograms were not recorded on any of the

patients. Grimm et al[1] evaluated idiopathic VT patients with the breath-hold electrocardiographic-gated turbo spin-echo technique and found no abnormalities on static black-blood images, similar to the results of the present study. However, cine imaging was not used in this study and the readers were not blinded.

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Upper Limit of Vulnerability Determination During Implantable Cardioverter-Defibrillator Placement to Minimize Ventricular Fibrillation Inductions*

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The defibrillation threshold (DFT) and upper limit of vulnerability (ULV) were determined using step-down protocols in 50 patients who underwent implantable cardioverter-defibrillator placement or testing. The sensitivity and specificity of each ULV energy level was assessed for detecting an increased DFT, correlation of the DFT and ULV, and optimal shock timing for ULV determination. A ULV <10 or 11 J (failure to induce ventricular fibrillation with 10- to 11-J shocks) was 100% predictive of an acceptable DFT and may be sufficient to exclude unacceptable DFTs in 60% of implantable cardioverter-defibrillator recipients. All 4 shocks used to scan the peak of the T wave during ULV testing were necessary for accurate ULV determination.

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Upper limit of vulnerability (ULV) testing has not been broadly adopted outside research protocols because of time-consuming methods that require multiple shocks. Using a single-energy level to determine vulnerability may be sufficient to guide implantable cardioverter-defibrillator placement and could shorten the preimplantation testing protocol. We hypothesized that the vulnerability safety margin could substitute for the longer but more accurate determination of the ULV, in the

TABLE 1 Patient Characteristics and Hardware Data (n = 50)*

Characteristic	Value
Age (mean ± SD) (yrs)	64 ± 13
Men	44 (88%)
Ejection fraction, mean ± SD (%)	35 ± 13
Procedure, implantation:check	20:30
Underlying heart disease	
Coronary disease	36 (72%)
Idiopathic dilated cardiomyopathy	7 (14%)
Other	6 (12%)
HCMP	3 (6%)
Valvular	2 (4%)
Myocarditis	1 (2%)
Primary electrical heart disease (LQTS)	1 (2%)
Implantable cardioverter-defibrillator	
Medtronic single chamber	20 (40%)
Medtronic dual chamber	14 (28%)
Guidant single chamber	8 (16%)
Guidant dual chamber	7 (14%)
St. Jude single chamber	1 (2%)
Implantable cardioverter-defibrillator system	
Single-coil lead	13 (26%)
Dual-coil lead	37 (74%)
Right-sided implant	4 (8%)
Left-sided implant	46 (92%)
Antiarrhythmic medications	
Amiodarone	9 (18%)
Other	4 (8%)

*Values are given as numbers of patients (percentages) unless otherwise stated.
HCMP = hypertrophic cardiomyopathy; LQTS = long-QT syndrome.

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