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Repetitive Monomorphic Tachycardia From the Left Ventricular Outflow Tract: Electrocardiographic Patterns Consistent With a Left Ventricular Site of Origin

DAVID J. CALLANS, MD, FACC, VOLKER MENZ, MD, DAVID SCHWARTZMAN, MD, FACC, CHARLES D. GOTTLIEB, MD, FACC, FRANCIS E. MARCHLINSKI, MD, FACC

Philadelphia, Pennsylvania

Objectives. **This study sought to characterize the electrocardiographic patterns predictive of left ventricular sites of origin of repetitive monomorphic ventricular tachycardia (RMVT).**

Background. **RMVT typically arises from the right ventricular outflow tract (RVOT) in patients without structural heart disease. The incidence of left ventricular sites of origin in this syndrome is unknown.**

Methods. **Detailed endocardial mapping of the RVOT was performed in 33 consecutive patients with RMVT during attempted radiofrequency ablation. Left ventricular mapping was also performed if pace maps obtained from the RVOT did not reproduce the configuration of the induced tachycardia.**

Results. **Pace maps identical in configuration to the induced tachycardia were obtained from the RVOT in 29 of 33 patients. Application of radiofrequency energy at sites guided by pace mapping resulted in elimination of RMVT in 24 (83%) of 29 patients. In four patients (12%), pace maps obtained from the RVOT did not match the induced tachycardia. All four patients had a QRS configuration during RMVT with precordial R wave transitions at or before lead V_2. In two patients, RMVT was mapped to the mediosuperior aspect of the mitral valve annulus, near the left fibrous trigone; catheter ablation at that site was successful in both. In two patients, RMVT was mapped to the basal aspect of the superior left ventricular septum. Catheter ablation was not attempted because His bundle deflections were recorded from this site during sinus rhythm.**

Conclusions. **RMVT can arise from the outflow tract of both the right and left ventricles. RMVTs with a precordial R wave transition at or before lead V_2 are consistent with a left ventricular origin.**

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Repetitive monomorphic ventricular tachycardia (RMVT) is a clinical syndrome characterized by "salvos" of nonsustained ventricular tachycardia (VT) or paroxysmal sustained VT often exacerbated by physical or emotional exertion (1–8). This syndrome almost exclusively occurs in young patients without identifiable structural heart disease (1–8), although several investigators (9,10) have reported subtle abnormalities in local anatomy or autonomic innervation. The tachycardia typically originates from the superior septal aspect of the right ventricular outflow tract (RVOT) (11–15); this location results in a characteristic electrocardiographic (ECG) "signature" of a left bundle branch block configuration—inferior-axis tachycardia.

Previous series of RMVT have focused on several intriguing aspects of the syndrome, including its benign clinical course (4,5,7,8) and its mechanism as assessed by response to programmed stimulation and antiarrhythmic drugs (4–8,16–19). More recently, the efficacy of catheter ablation has been demonstrated, particularly when the tachycardia is mapped to a typical RVOT location (10–15). Several investigators (6,8) have described patients with RMVT and atypical ECG manifestations. Although the possibility of left ventricular origin was considered, mapping was not performed in these series. The purpose of the present study was to assess the incidence of left ventricular sites of origin in patients with the clinical syndrome of RMVT and to identify ECG patterns consistent with a left ventricular origin.

Methods

Patients (Table 1). The study included 33 consecutive patients with RMVT referred for mapping and radiofrequency catheter ablation (August 1992 to October 1995; 21 women, 12 men; mean [±SD] age 42 ± 13 years, range 29 to 69). For inclusion in the study, all patients had normal sinus rhythm on electrocardiography and normal echocardiographic findings. Individual patients underwent other diagnostic testing as dictated by clinical necessity. The clinical arrhythmia was always nonsustained VT in 25 patients; one or more episodes of sustained VT were documented in 8. When available, ECG recordings during the clinical VT demonstrated large-voltage monophasic R waves in the inferior leads.

From the Philadelphia Heart Institute and the Sidney Kimmel Cardiovascular Research Center, Philadelphia Pennsylvania.

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Address for correspondence: Dr. David J. Callans, Allegheny University Hospitals, MCP Division, Division of Cardiology, Suite 803, 3300 Henry Avenue, Philadelphia, Pennsylvania 19129.

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Abbreviations and Acronyms

ECG = electrocardiogram, electrocardiographic
LVOT = left ventricular outflow tract
RMVT = repetitive monomorphic ventricular tachycardia
RVA = right ventricular apex
RVOT = right ventricular outflow tract
VT = ventricular tachycardia

Electrophysiologic study. All patients provided written informed consent to the study protocol, which was approved by the Institutional Review Board of Presbyterian Medical Center. Electrophysiologic studies were performed after withdrawal of antiarrhythmic drugs under mild intravenous sedation. Catheters were inserted through the right or left femoral vein, or both, under fluoroscopic guidance, to the high right atrium, right ventricular apex (RVA), and RVOT. When necessary, left ventricular mapping was performed through the retrograde aortic approach. The stimulation protocol consisted of incremental atrial and ventricular pacing, ventricular programmed stimulation from the RVA at two drive cycle lengths and burst pacing from the RVA and RVOT at cycle lengths from 350 to 250 ms. If VT was not induced during the baseline state, stimulation was repeated during isoproterenol infusion (1 to 5 μg/min intravenously). Twelve surface ECG leads were continuously monitored, as well as intracardiac recordings from the RVA and RVOT and the left ventricle in selected patients.

Mapping and ablation procedure. After induction of VT, pace mapping of the RVOT was performed using a previously described nine-site schema (20). Pace mapping was performed during sinus rhythm with bipolar cathodal stimulation (catheter poles 1 and 2) at an output just greater than the diastolic threshold. Mapping of the left ventricle, with particular reference to the left ventricular outflow tract (LVOT), was performed when pace mapping from the right ventricle failed to match the induced arrhythmia. Localization of the VT site of origin and selection of target sites for ablation were based on the site corresponding to the best available pace map (i.e., the pace map that precisely matched the greatest number of surface ECG leads) (11,13).

Modulated radiofrequency energy was delivered using a 4-mm tip deflectable catheter (EP Technologies). The power output was adjusted to provide a 10-ohm decrease in impedance from baseline or an electrode tip temperature of 50° to 70°C, or both, and was maintained for up to 120 s. *Procedural success* was defined as the absence of all configurations of spontaneous and inducible VT (either sustained or nonsustained), both with and without isoproterenol infusion.

Table 1. Characteristics of Patients With Repetitive Monomorphic Ventricular Tachycardia Originating From Right and Left Ventricles

	RVOT (n = 29)	LVOT (n = 4)
Women/men	20/9	1/3
Age (yr)	43 ± 12	46 ± 15
Presenting symptoms		
Palpitations	14 (48%)	4
Near syncope	8 (28%)	0
Syncope	7 (24%)	0
Clinical sustained VT	6 (21%)	2 (50%)
Sustained VT on isoproterenol	17 (59%)	4 (100%)

Data presented are mean value ± SD or number (%) of patients. LVOT = left ventricular outflow tract; RVOT = right ventricular outflow tract; VT = ventricular tachycardia.

Results

Sustained or nonsustained VT of sufficient duration to obtain recordings in all 12 surface ECG leads occurred spontaneously or was induced in all 33 patients. Detailed pace mapping of the RVOT resulted in an identical or nearly identical match of the induced VT in 29 of 33 patients. All 29 patients had an inferiorly directed VT configuration, with a precordial R wave transition equal to or later than lead V_3. Catheter ablation attempts based on this mapping information were immediately successful in 24 (83%) of 29 patients.

Pace mapping in the RVOT did not reproduce the induced VT configuration in 4 patients. The clinical characteristics of these four patients did not differ significantly from those with RVOT tachycardias (Table 1), except that the usual female predominance was not observed. All four patients had clinical symptoms of palpitations, exacerbated by exercise; all had inducible sustained VT on isoproterenol administration in the electrophysiology laboratory. Two distinct ECG patterns were demonstrated. In two patients, the VT had a right bundle, inferior axis, with a dominant R wave in lead V_1 (Fig. 1). This VT configuration was reproduced with pace maps performed within the LVOT at the mediosuperior aspect of the mitral valve annulus. Application of radiofrequency energy at this site resulted in successful elimination of RMVT in both patients. The second pattern, also observed in two patients, was a left bundle, inferior-axis VT with a precordial R wave transition at lead V_2 (Fig. 2). Pacing at the basal aspect of the superior left ventricular septum resulted in a QRS configuration similar to that of spontaneous VT, particularly with reference to the precordial R wave transition. In both patients, His bundle activation was evident in recordings performed in sinus rhythm at the sites from which the closest pace map was obtained. Catheter ablation was not attempted in either patients because of concern over producing atrioventricular (AV) block.

Discussion

Atypical ECG manifestations of RMVT. Although a left bundle, inferior-axis VT configuration is almost pathognomonic of RMVT, patients with less common QRS configurations have been demonstrated to experience identical clinical syndromes (6,8). The possibility of a left ventricular site of

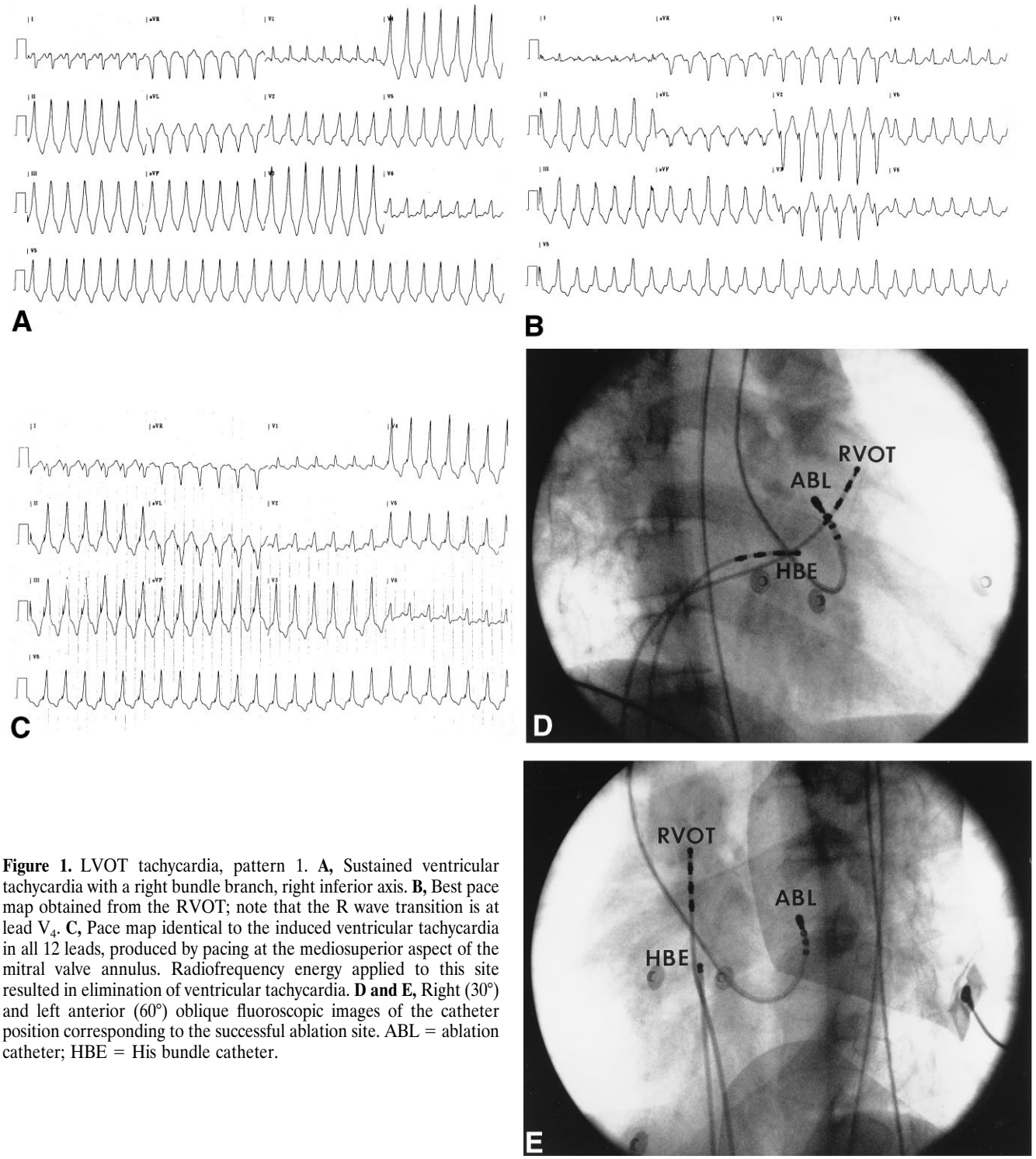

Figure 1. LVOT tachycardia, pattern 1. **A,** Sustained ventricular tachycardia with a right bundle branch, right inferior axis. **B,** Best pace map obtained from the RVOT; note that the R wave transition is at lead V_4. **C,** Pace map identical to the induced ventricular tachycardia in all 12 leads, produced by pacing at the mediosuperior aspect of the mitral valve annulus. Radiofrequency energy applied to this site resulted in elimination of ventricular tachycardia. **D and E,** Right (30°) and left anterior (60°) oblique fluoroscopic images of the catheter position corresponding to the successful ablation site. ABL = ablation catheter; HBE = His bundle catheter.

origin of RMVT has been previously considered (6,8,19). In a series of 70 patients with RMVT, Coumel et al. (6) described 14 patients with ECG patterns compatible with the two LVOT patterns demonstrated in the present study (7 patients with each pattern). After excluding patients with midseptal left ventricular idiopathic VT, Mont et al. (8) observed that 9 of 53 patients with RMVT had a right bundle, inferior-axis VT configuration, consistent with our LVOT pattern 1. Endocardial mapping was not performed in either series. Several series of catheter ablation have demonstrated left ventricular sites of origin in patients with a variety of idiopathic VT syndromes (12–14). These studies did not provide a clinical description of the arrhythmia syndromes and did not characterize the ECG patterns in patients with left ventricular sites of origin.

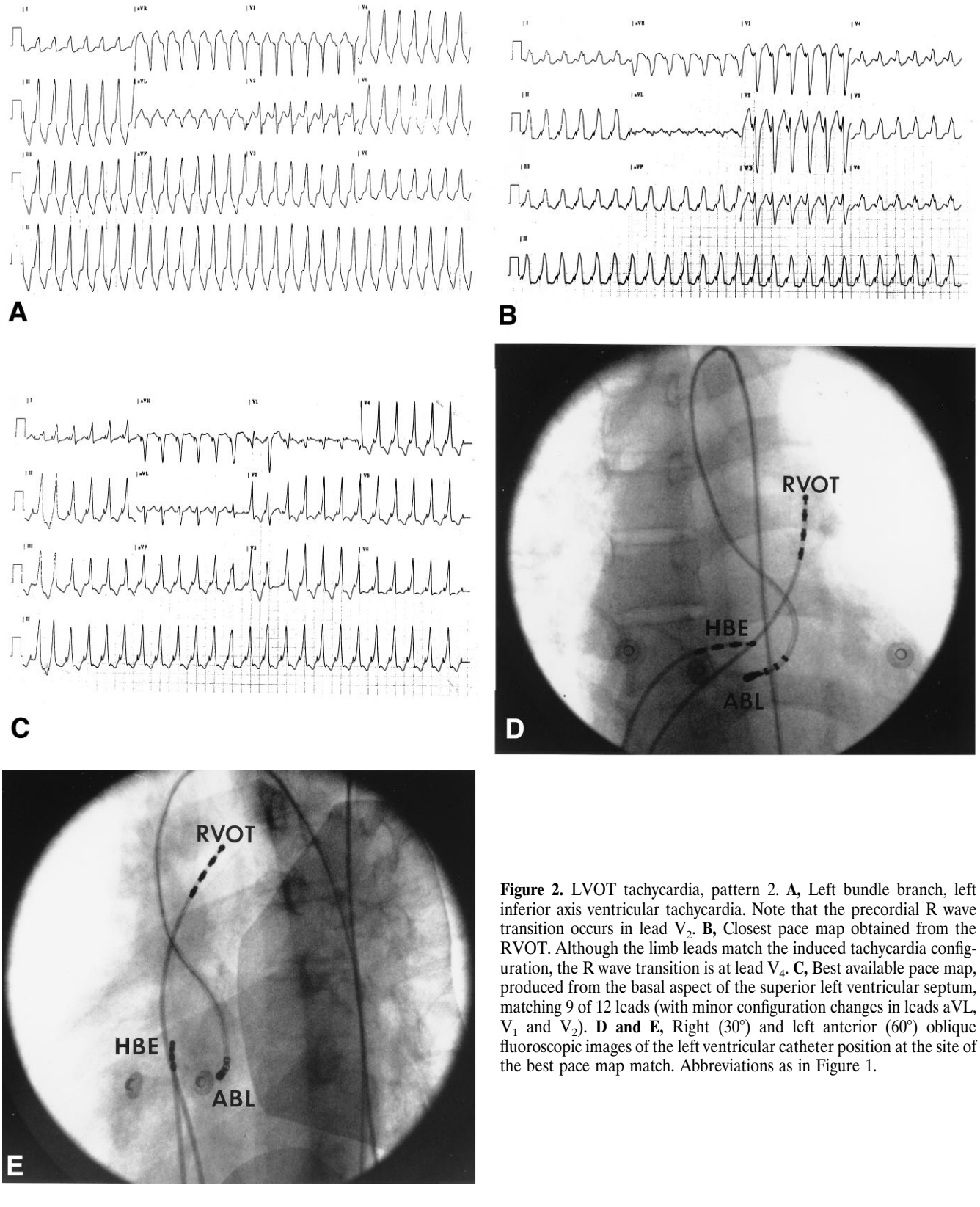

Figure 2. LVOT tachycardia, pattern 2. **A,** Left bundle branch, left inferior axis ventricular tachycardia. Note that the precordial R wave transition occurs in lead V_2. **B,** Closest pace map obtained from the RVOT. Although the limb leads match the induced tachycardia configuration, the R wave transition is at lead V_4. **C,** Best available pace map, produced from the basal aspect of the superior left ventricular septum, matching 9 of 12 leads (with minor configuration changes in leads aVL, V_1 and V_2). **D and E,** Right (30°) and left anterior (60°) oblique fluoroscopic images of the left ventricular catheter position at the site of the best pace map match. Abbreviations as in Figure 1.

ECG patterns predictive of LVOT origin. To our knowledge, the present study is the first to provide an assessment of the incidence (12%) of an left ventricular origin in patients with the clinical syndrome of RMVT. More important, two distinct ECG configurations consistent with a LVOT site of origin were characterized in the present study. This and

previous studies from our laboratory (15) suggest that tachycardias from sites on the septal aspect of the RVOT consistently have a precordial R wave transition at lead V_3 or later. Two distinct patterns inconsistent with typical RVOT origin are presented in this study. Inferiorly directed VTs with a dominant R wave in lead V_1 (pattern 1) were localized to the area of the aortomitral continuity (left fibrous trigone). Inferiorly directed VTs with a left bundle branch block configuration and a precordial R wave transition at lead V_2 (pattern 2) arose from the basal aspect of the superior left ventricular septum just inferior to the aortic valve. Because the pace maps obtained at this site closely approximated but did not exactly reproduce the clinical VT configuration, an intraseptal site of origin cannot be excluded. Recognition of these ECG patterns may have considerable clinical significance, both in terms of procedural planning for left ventricular mapping and in counseling patients about the risk of inability to attempt ablation or the potential for heart block with LVOT pattern 2.

Study limitations. This small series admits to several limitations: 1) LVOT pattern 2 tachycardias may have been suboptimally localized in that the closest pace map did not provide a precise match and because successful ablation, an admitted reference standard for localization, was not achieved because of concern over producing AV block. A different location of LVOT pattern 2 VT cannot be excluded. Nonetheless, it is clinically significant that this ECG configuration is incompatible with a RVOT origin and that mapping of this ECG pattern will require access to the left ventricle and careful assessment of the risk of AV block. 2) It is impossible to determine the true sensitivity and specificity of the ECG patterns for the diagnosis of LVOT sites of origin, given the relatively small number of patients studied. However, given the consistent failure to reproduce ECG configurations with an R wave transition earlier than lead V_3 by pacing from any site within the RVOT, we believe that earlier precordial transitions strongly suggest an left ventricular site of origin. 3) The definition of RMVT used in this study is based on clinical characteristics; although all VTs were facilitated by isoproterenol, formal drug testing to investigate tachycardia mechanism was not performed systematically.

Conclusions. The clinical syndrome of RMVT can originate from the LVOT as well as from the RVOT. RMVTs from the LVOT have broad, monophasic R waves in the inferior leads, with a precordial wave transition at or before lead V_2. The possibility of a site of origin close to the His bundle should be considered if the VT has a left bundle branch, inferior configuration with a precordial transition at lead V_2.

References

1. Gallavardin L. Extrasystolie ventriculaire à paroxysmes tachycardiques prolongés. Arch Mal Coeur 1922;15:298–312.
2. Parkinson J, Papp C. Repetitive paroxysmal tachycardia. Br Heart J 1947; 9:241–52.
3. Froment R, Gallavardin L, Cahen P. Paroxysmal ventricular tachycardia: a clinical classification. Br Heart J 1953;15:172–84.
4. Rahilly GT, Prystowsky EN, Zipes DP, Naccarelli GV, Jackman WM, Heger JJ. Clinical and electrophysiologic findings in patients with repetitive monomorphic ventricular tachycardia and an otherwise normal electrocardiogram. Am J Cardiol 1982;50:459–68.
5. Buxton AE, Waxman HL, Marchlinski FE, Simson MB, Cassidy D, Josephson ME. Right ventricular tachycardia: clinical and electrophysiologic characteristics. Circulation 1983;68:917–27.
6. Coumel P, LeClerq JP, Slama R. Repetitive monomorphic idiopathic ventricular tachycardia. In Zipes DP, Jalife J, editors. Cardiac Electrophysiology and Arrhythmias. Orlando (FL): Grune and Stratton, 1985:455–66.
7. Lemery R, Brugada P, Della Bella P, Dugernier T, van den Dool A, Wellens HJJ. Nonischemic ventricular tachycardia: clinical course and long-term follow-up in patients without clinically overt heart disease. Circulation 1989;5:990–8.
8. Mont L, Seixas T, Brugada P, et al. The electrocardiographic, clinical and electrophysiologic spectrum of idiopathic monomorphic ventricular tachycardia. Am Heart J 1992;124:746–53.
9. Carlson MD, White RD, Trohman RG, et al. Right ventricular outflow tract ventricular tachycardia: detection of previously unrecognized anatomic abnormalities using cine magnetic resonance imaging. J Am Coll Cardiol 1994;24:720–7.
10. Mitrani RD, Klein LS, Miles WM, et al. Regional cardiac sympathetic denervation in patients with ventricular tachycardia in the absence of coronary artery disease. J Am Coll Cardiol 1993;22:1344–53.
11. Morady F, Kadish AH, DiCarlo L, et al. Long-term results of catheter ablation of idiopathic right ventricular tachycardia. Circulation 1990;82: 2093–9.
12. Klein LS, Shih HT, Hackett FK, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. Circulation 1992;85:1666–74.
13. Calkins H, Kalbfleisch SJ, El-Atassi R, et al. Relation between efficacy of radiofrequency catheter ablation and site of origin of idiopathic ventricular tachycardia. Am J Cardiol 1993;71:827–33.
14. Coggins DL, Lee RJ, Sweeney J, et al. Radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. J Am Coll Cardiol 1994;23:1333–41.
15. Movsowitz C, Schwartzman D, Callans DJ, et al. Idiopathic right ventricular outflow tract tachycardia: narrowing the anatomical location for successful ablation. Am Heart J 1996;131:930–6.
16. Sung RJ, Shapiro WA, Shen EN, et al. Effects of verapamil on ventricular tachycardias possibly caused by reentry, automaticity, and triggered activity. J Clin Invest 1983;72:350–60.
17. Lerman BB, Belardinelli L, West GA, Berne RM, DiMarco JP. Adenosine-sensitive ventricular tachycardia: evidence suggesting cyclic AMP-mediated triggered activity. Circulation 1986;74:271–80.
18. Sung RJ, Keung EC, Nguyen NX, et al. Effects of β-adrenergic blockade on verapamil-responsive and verapamil-irresponsive sustained ventricular tachycardia. J Clin Invest 1988;81:688–99.
19. Lerman BB, Stein K, Engelstein ED, et al. Mechanism of repetitive monomorphic ventricular tachycardia. Circulation 1995;92:421–9.
20. Jadonath RL, Schwartzman D, Preminger MW, Gottlieb CD, Marchlinski FE. The utility of the 12 lead electrocardiogram in localizing the site of origin of right ventricular outflow tract tachycardia. Am Heart J 1995;130:1107–13.