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Repetitive Monomorphic Ventricular Tachycardia Originating From the Aortic Sinus Cusp

Electrocardiographic Characterization for Guiding Catheter Ablation

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OBJECTIVES We sought to investigate the electrocardiographic (ECG) characteristics for guiding catheter ablation in patients with repetitive monomorphic ventricular tachycardia (RMVT) originating from the aortic sinus cusp (ASC).

BACKGROUND Repetitive monomorphic ventricular tachycardia can originate from the right ventricular outflow tract (RVOT) and ASC in patients with a left bundle branch block (LBBB) morphology and an inferior axis.

METHODS Activation mapping and ECG analysis was performed in 15 patients with RMVT or ventricular premature contractions. The left main coronary artery (LMCA) was cannulated as a marker and for protection during radiofrequency delivery if RMVT originated from the left coronary ASC.

RESULTS During arrhythmia, the earliest ventricular activation was recorded from the superior septal RVOT in eight patients (group 1) and from the ASC in the remaining seven patients (group 2). The indexes of R-wave duration and R/S-wave amplitude were significantly lower in group 1 than in group 2 (31.8 ± 13.5% vs. 58.3 ± 12.1% and 14.9 ± 9.9% vs. 56.7 ± 29.5%, respectively; $p < 0.01$), despite similar QRS morphology. In five patients from group 2, RMVT originated from the left ASC, with a mean distance of 12.2 ± 3.2 mm (range 7.3 to 16.1) below the ostium of the LMCA. In the remaining two patients, the RMVT origin was in the right ASC. All arrhythmias were successfully abolished. None of the patients had recurrence or complications during 9 ± 3 months of follow-up.

CONCLUSIONS On the surface ECG, RMVT from the ASC has a QRS morphology similar to that of RVOT arrhythmias. The indexes of R-wave duration and R/S-wave amplitude can be used to differentiate between the two origins. Radiofrequency ablation can be safely performed within the left ASC with a catheter cannulating the LMCA. (J Am Coll Cardiol 2002;39: 500–8) © 2002 by the American College of Cardiology

Catheter ablation has increasingly been used for ablation of repetitive monomorphic ventricular tachycardia (RMVT) (1–5) and symptomatic monomorphic ventricular premature contraction (VPCs) (6). These arrhythmias typically originate from the so-called "superior septal" aspect of the right ventricular outflow tract (RVOT) (1–5) and are often provoked by physical or emotional stress (7–10). The origin of the arrhythmia from the RVOT results in a typical electrocardiograph (ECG) with a left bundle branch block (LBBB) morphology and an inferior axis. Most patients with this ECG pattern and structurally normal heart are amenable to radiofrequency (RF) ablation, with a high success rate (1–5,11,12). In patients with failed ablation in the RVOT, a left ventricular origin of the RMVT should be considered (11–15). Although recent studies have described that RMVT can originate from the aortic sinus cusp (ASC), as confirmed by pace mapping and successful RF catheter ablation (14–16), the ECG characteristics were not quantitatively analyzed. The aim of this study was to investigate whether ECG characteristics can be used to identify the arrhythmia origin and guide catheter ablation in the ASC, with a note on anatomy.

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METHODS

Patient group. Between May 2000 and February 2001, 15 patients (8 men and 7 women; mean age 36.6 ± 20.3 years [range 11 to 78 years]) with symptomatic RMVT or frequent monomorphic VPCs were referred to our center. During clinical arrhythmia, the surface ECG showed a typical LBBB morphology with an inferior axis in all patients. All patients were refractory to anti-arrhythmic drugs (mean 3 ± 1 drugs), and six patients had previously failed ablation in the RVOT. All patients had structurally normal hearts and a normal ECG during sinus rhythm.

Electrophysiologic study. After giving written, informed consent and after withdrawal of anti-arrhythmic drugs, all patients underwent an electrophysiologic evaluation under

Abbreviations and Acronyms

ASC = aortic sinus cusp
ECG = electrocardiogram or electrocardiographic
LBBB = left bundle branch block
LMCA = left main coronary artery
RCA = right coronary artery
RF = radiofrequency
RMVT = repetitive monomorphic ventricular tachycardia
RVOT = right ventricular outflow tract
VPC = ventricular premature contraction

intravenous sedation. Catheters were introduced to the right ventricular apex, RVOT and His bundle region under fluoroscopy through the femoral veins. The stimulation protocol consisted of programmed ventricular stimulation from the right ventricular apex and RVOT at two drive cycle lengths with up to three extrastimuli and incremental burst pacing at a cycle length up to 250 ms. If the clinical arrhythmia did not occur spontaneously and was not inducible during the baseline state, intravenous isoproterenol infusion (2 to 5 μg/min) was administered to provoke the clinical arrhythmia.

Electrocardiographic analysis. Spontaneous or provoked RMVT or VPCs allowed analysis of the QRS complex morphology, independent of the P and T waves. During clinical arrhythmias, analysis of the surface ECG was focused on the following (Fig. 1): 1) total QRS duration; 2) R-wave duration in leads V_1 and V_2, determined from the onset of the QRS complex to the transition point between the R-wave and the isoelectric line; 3) R-wave duration index, calculated as a percentage by dividing the QRS complex duration by the longer R-wave duration in lead V_1 or V_2; 4) R/S-wave amplitude ratio in leads V_1 and V_2, measured from the QRS complex peak or nadir to the isoelectric line, expressed as a percentage; and 5) R/S-wave amplitude index, calculated from the greater percentage of the R/S-wave amplitude ratio in lead V_1 or V_2.

Mapping and RF ablation. With spontaneous or induced RMVT or VPCs, activation mapping was performed with a 7F, 4-mm-tipped ablation catheter (Cordis-Webster, Inc., Baldwin Park, California). Activation mapping identified bipolar ventricular electrograms preceding the onset of the QRS complex. If RMVT or VPCs were infrequent, pace mapping was performed at 2 to 10 mA, and a pulse width of 0.5 to 2 ms was used to identify the site for RF catheter ablation. In all patients, initial mapping was performed in the RVOT. Whenever RVOT mapping failed to identify an early endocardial activation site, mapping of the left ventricle and ASC was performed retrogradely through the right femoral artery. The arrhythmia origin and ablation target were defined by the earliest activation. Bipolar and unipolar electrograms were filtered at 30 to 400 Hz and 0.05 to 400 Hz, respectively. The data were stored on optical disks for further analysis.

When the earliest ventricular activation was identified in the ASC, a 5F pigtail catheter was inserted into the aortic root through the left femoral artery. The aortic root and the ostia of the right coronary artery (RCA) and left main

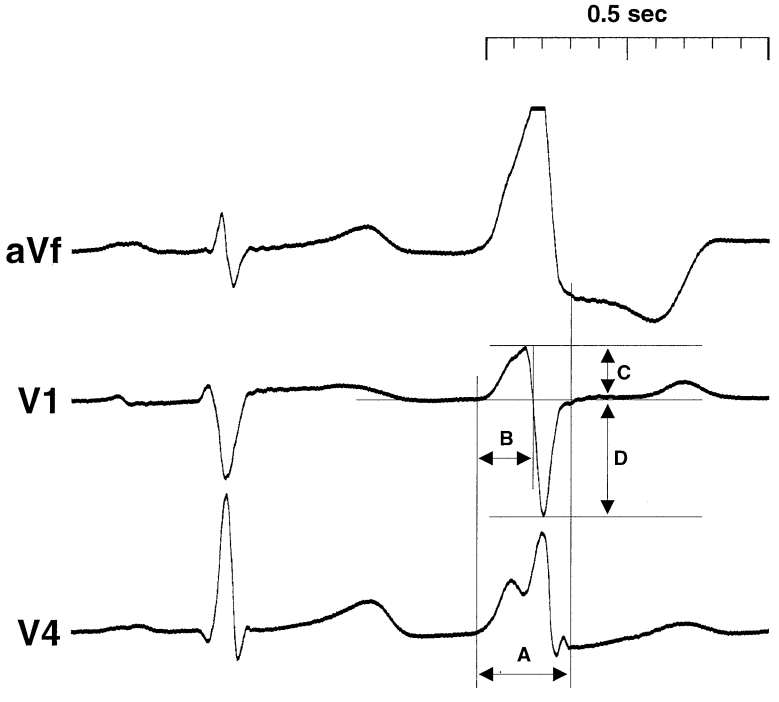

Figure 1. Example of electrocardiographic analysis of clinical arrhythmias: leads aVF, V_1 and V_4 of a normal sinus beat, followed by the first beat of repetitive monomorphic ventricular tachycardia. **A** = total QRS duration, measured from the earliest onset in lead V_4 to the latest activation in lead aVF (ms); **B** = R-wave duration, determined in lead V_1 from the QRS onset to the R-wave transaction point of the R-wave with the isoelectric line (ms); **C** = R-wave amplitude, measured from the peak to the isoelectric line (mV); **D** = S-wave amplitude measured from the cusp to the isoelectric line (mV).

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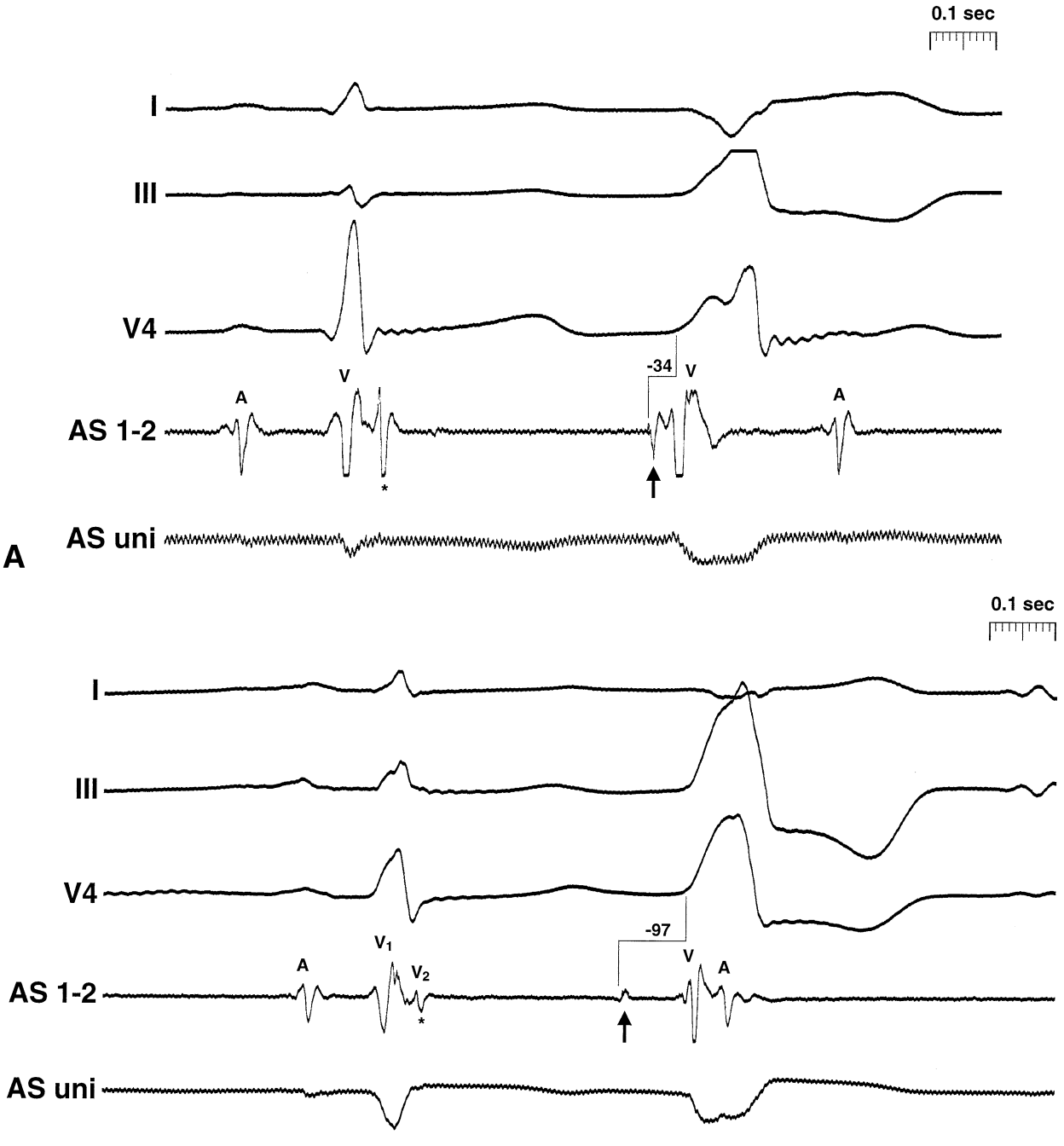

Figure 2. Mapping catheter recordings of a sinus beat and the first beat of repetitive monomorphic ventricular tachycardia (RMVT) at the successful ablation sites in two different patients. Each panel shows surface electrocardiographic leads I, III and V_4, as well as the bipolar and unipolar signals recorded from the mapping catheter. **(A)** Earliest ventricular activation preceding onset of the QRS complex by 34 ms in a patient with RMVT originating from the left coronary aortic sinus. The unipolar signal (AS uni) also has a QS morphology, but is activated later than the bipolar signals (AS 1–2). Please note that a low-amplitude presystolic potential **(arrow)** appears during RMVT, although this potential is the second component of the "ventricular signal" **(asterisk)** during sinus rhythm. **(B)** Earliest low-amplitude late-diastolic ventricular potential **(arrow)** preceding onset of the QRS complex by 97 ms in a patient with RMVT originating from the left coronary aortic sinus cusp. As in part A, this potential is also seen as the second component of the "ventricular signal" during sinus rhythm **(asterisk)**. A = atrial potential; V = ventricular potential.

coronary artery (LMCA) were visualized by angiography. If the origin was in the left ASC, the LMCA was cannulated with a 5F left Judkin's catheter. This was done, as a marker and for protection of the LMCA, in case of ablation catheter dislodgment during RF application.

Radiofrequency energy was delivered at the distal electrode of the thermocouple catheter and maintained for 120 s with a preselected temperature of 60°C in the RVOT or 55°C in the ASC. Radiofrequency energy was started at 30 W and increased up to 50 W in the RVOT and from 15 to

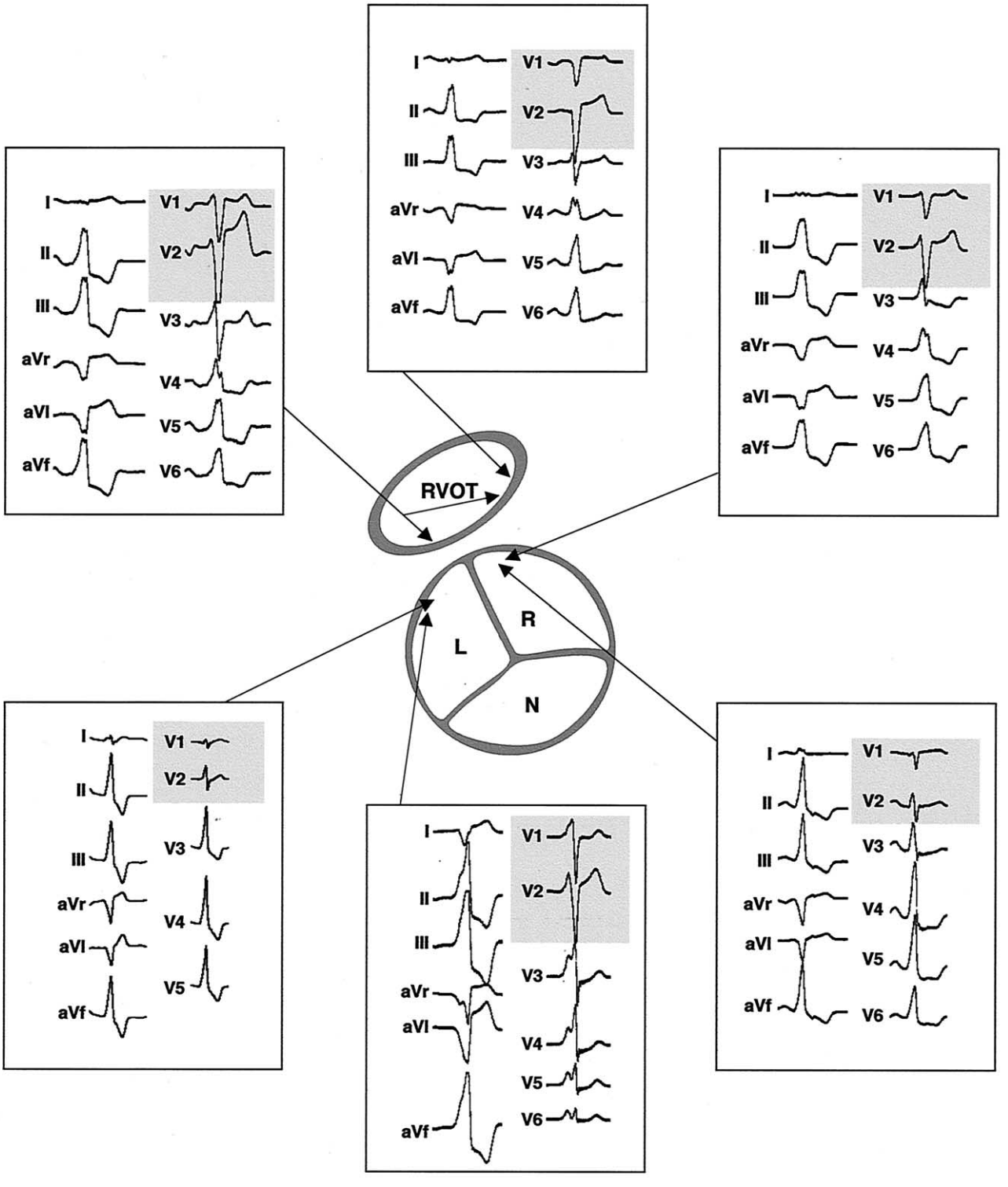

Figure 3. Anatomic location of the origin of the arrhythmia, with corresponding 12-lead electrocardiographic morphology. Note the different morphology in leads V_1 and V_2 with respect to the anatomic origin. L = left coronary aortic sinus; N = noncoronary aortic sinus; R = right coronary aortic sinus; RVOT = right ventricular outflow tract.

30 W in the ASC, under continuous fluoroscopy, to reach the target temperature. Radiofrequency application was immediately stopped if we observed catheter dislodgment.

Ablation success was defined as: 1) the absence of spontaneous or inducible clinical RMVT and/or VPCs at the end of the procedure; and 2) the absence of these arrhythmias after 48 h of continuous ECG monitoring and the absence of exercise stress after ablation, without anti-

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Table 1. Patient Characteristics and Electrocardiographic Procedural Data

Patients	Group 1	Group 2
Patients with RMVT and VPC (n)	4	7
Patients with VPCs only (n)	4	0
Patients with previously failed RF procedures (n)	1	5
Total QRS duration (ms)	165 ± 16	160 ± 24
R-wave duration in lead V_1 (ms)	46.1 ± 28.8	81.9 ± 42.4*
R-wave duration in lead V_2 (ms)	45.9 ± 20.4	86.0 ± 31.0†
R/S-wave amplitude ratio in lead V_1 (%)	11.1 ± 8.1	28.0 ± 17.3*
R/S-wave amplitude ratio in lead V_2 (%)	13.7 ± 9.5	55.3 ± 30.4†
RF applications (n)	4.8 ± 4.4	2.4 ± 2.3

*$p < 0.05$. †$p < 0.01$.
Data are expressed as the mean value ± SD.
RF = radiofrequency; RMVT = repetitive monomorphic ventricular tachycardia; RVOT = right ventricular outflow tract; VPCs = ventricular premature contractions.

arrhythmic drugs. Transthoracic echocardiography was performed before hospital discharge in all patients. Transesophageal echocardiography was also performed after the procedure in the patients with RMVT originating in the ASC. Follow-up information was obtained from either the referring physicians or direct follow-up in our clinic. Transthoracic echocardiography was performed in the patients with RMVT originating in the ASC at three and six months after ablation.

Statistical analysis. Data are expressed as the mean value ± SD. The Mann-Whitney *U* and Wilcoxon tests were used for comparisons. A p value <0.05 was considered significant.

RESULTS

No clinical ventricular arrhythmias were induced by ventricular stimulation. At baseline, the clinical arrhythmia with LBBB morphology and an inferior axis occurred spontaneously in nine patients. During isoproterenol infusion, the clinical arrhythmia could be provoked or aggravated in all 15 patients. Eleven patients had RMVT and frequent monomorphic VPCs, with an identical morphology, whereas four patients had only frequent monomorphic VPCs.

Mapping. In eight patients (group 1), the earliest ventricular activation was localized in the superior septal RVOT, preceding the onset of the QRS complex by 29.3 ± 5.5 ms (range 19 to 35 ms), with simultaneous activation in the bipolar and unipolar recordings. In this group, no presystolic or late diastolic potentials were recorded. Additional pace mapping was performed in six of these eight patients and resulted in a perfect match in all six patients.

In the remaining seven patients (group 2), the earliest ventricular activation was recorded from the ASC preceding the onset of the QRS complex by 39.3 ± 24.2 ms (range 23 to 97 ms). Two ventricular activation components were recorded at the earliest site in all patients from group 2. The first component was either presystolic or late diastolic, of high frequency and low amplitude and only present in the ASC of origin, whereas the second component was coincident with the QRS complex (Fig. 2A and 2B). The ventricular activation on the unipolar recording was always simultaneous with the second component on the bipolar recording. Interestingly, during sinus rhythm, two ventricular activation components were also recorded in the left ASC in patients with RMVT origin, but the sequence of the two components was reversed, as compared with RMVT. Pace mapping was performed at the site of the earliest ventricular activation in three patients, but in only one patient, a perfect pace map was achieved. In addition, in all patients, an atrial potential was recorded from the ASC.

Electrocardiographic characteristics. Seven of eight patients from group 1 presented with rS morphology in lead V_1 or V_2, or both, and an R/S-wave transition between leads V_3 and V_4. In six of these patients, the earliest ventricular activation was located at the posterior part of the superior septal RVOT; in one patient, the site of earliest activation was in the anterior part of the superior septal RVOT (Fig. 3). In the remaining patient with QS morphology in leads V_1 and V_2 and R/S-wave transition in lead V_4, the earliest endocardial activation was located in the anterior part of the superior septal RVOT (Fig. 3).

In group 2, six patients presented with rS morphology in lead V_1 or V_2, or both, and R/S-wave transition between leads V_2 and V_3. In these six patients, the earliest ventricular activation was recorded at the right ASC in two patients and at the left ASC in the other four patients (Fig. 3). The remaining patient from group 2 showed QRS morphology in lead V_1 and R/S-wave transition in lead V_2. In this patient, the earliest ventricular activation was located in the left ASC (Fig. 3).

The total QRS duration, R-wave duration and R/S-wave amplitude ratio in leads V_1 and V_2, for both groups, are shown in Table 1. No difference in the total QRS duration was seen between the two groups. In contrast, the R-wave duration and R/S-wave amplitude ratio in leads V_1 and V_2 were significantly greater in group 2, which resulted in significantly higher indexes of R-wave duration and R/S-wave amplitude (Fig. 4). Cut-off values for an R-wave duration index ≥50% and for an R/S-wave amplitude index ≥30% allowed us to identify six of seven patients with RMVT origin from the ASC. One patient with RMVT originating from the right ASC had an R-wave duration of

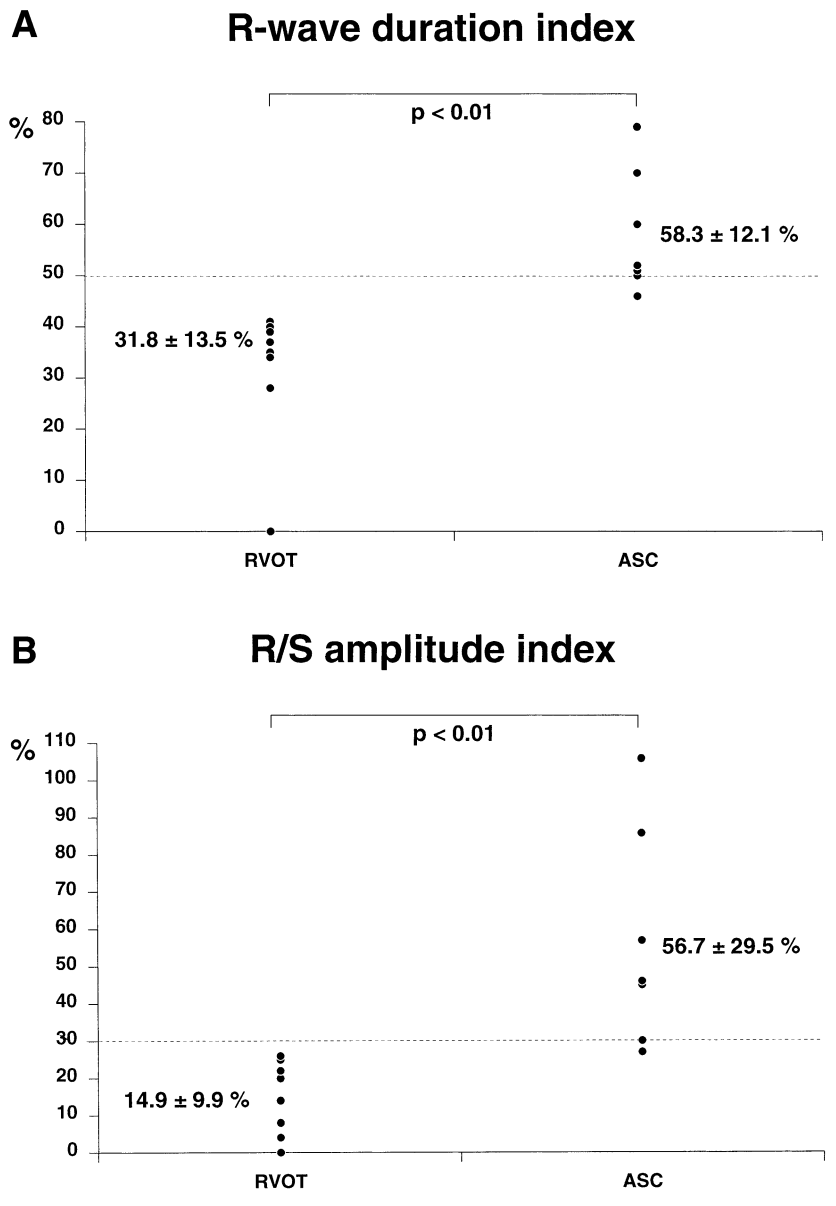

Figure 4. Plot of the indexes of R-wave duration **(A)** and R/S amplitude **(B)** in patients with repetitive monomorphic ventricular tachycardia originating from the superior septal right ventricular outflow tract (RVOT) and aortic sinus cusp (ASC).

46% and an R/S-wave amplitude index of 27%. No overlap of R-wave duration and R/S-wave amplitude indexes was noted between the two groups.

Radiofrequency ablation. Five of seven patients with RMVT originating from the ASC, compared with only one of eight patients with RVOT originating from the ASC, had a previously failed ablation in the RVOT. The clinical arrhythmia was successfully ablated in all 15 patients. No significant difference was seen in the number of RF applications between the two groups (Table 1). In group 1, frequent VPCs and/or short runs of ventricular tachycardia with a very similar QRS morphology were observed during RF application. All ventricular arrhythmias were successfully abolished by RF applications.

In five patients from group 2, RF energy was applied at the left ASC below the LMCA ostium (Fig. 5). The distance from the tip of the ablation catheter to the LMCA ostium was 12.2 ± 3.2 mm (range 7.3 to 16.1 mm). In the remaining two patients, RF energy was applied in the right ASC, 8.8 and 11.1 mm antero-inferior to the RCA ostium. Radiofrequency current terminated RMVT in <3 s in 6 patients and within 8 s in one patient. No increase of ventricular ectopy was observed during RF delivery in group 2.

In all 15 patients, no procedure-related complications occurred, and no damage of the aortic valve was found by echocardiography. In addition, all 15 patients were free of arrhythmias, without anti-arrhythmic drugs, during a follow-up period of 9 ± 3 months.

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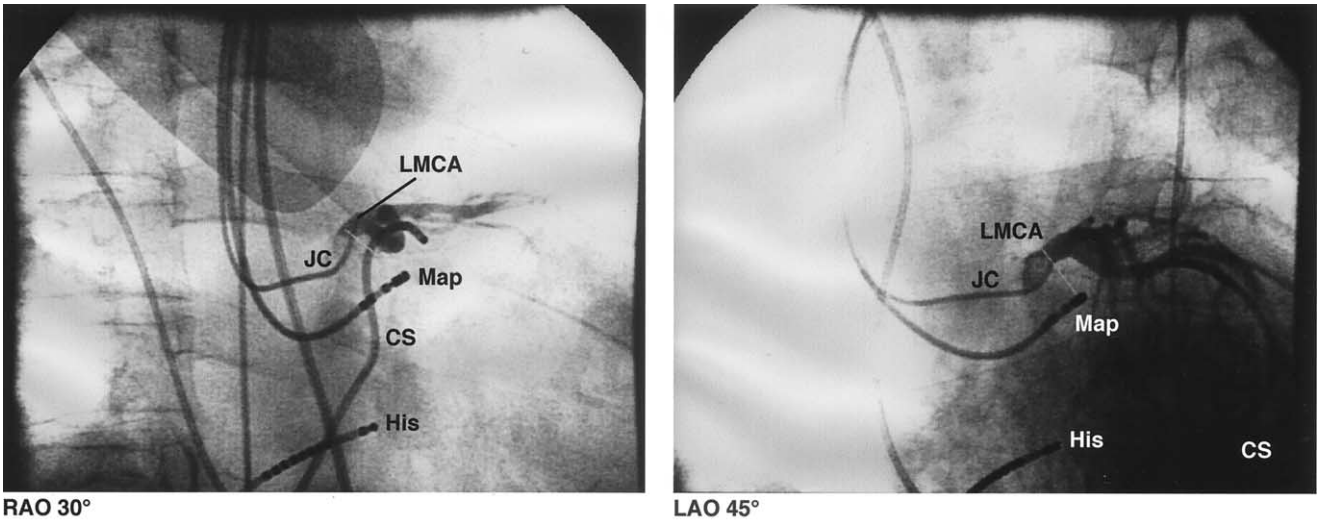

Figure 5. Right (30°) and left (45°) anterior oblique (RAO and LAO, respectively) radiographic views of the mapping catheter (Map) at the successful ablation site in the left coronary sinus cusp. The mapping catheter was located below the ostium of the left main coronary artery (LMCA). CS = decapolar catheter inside the coronary sinus, with the distal electrode inside the great cardiac vein; His = decapolar catheter at the His bundle region; JC = 5F left Judkin's catheter.

DISCUSSION

Electrocardiographic characteristics and arrhythmia origin. Using quantitative ECG analysis, we could differentiate between RMVT originating from the RVOT and ASC. In the present study, the arrhythmia origin was in the superior septal RVOT in eight patients and in the ASC in seven patients. The indexes of R-wave duration and R/S-wave amplitude, calculated from lead V_1 or V_2 during arrhythmia, were significantly different between these two origins. These differences can be explained by their anatomic location. The arrhythmia originating from the ASC produces a slightly different vector, because the anatomic location is more posterior and rightward, which results in a longer R-wave duration and higher R/S-wave amplitude ratio in leads V_1 and V_2. However, the right ASC is in close anatomic proximity to the so-called superior septal RVOT. This may explain why one patient with RMVT originating from the right ASC had lower indexes of R-wave duration and R/S-wave amplitude. Furthermore, the value for an R-wave duration index $\geq$50% and for an R/S-wave amplitude index $\geq$30% strongly suggest the ASC origin in patients with a typical LBBB morphology and an inferior axis.

In addition to the significant difference in the indexes of R-wave duration and R/S-wave amplitude, a low-amplitude, high-frequency potential was always found in patients with the ASC origin of RMVT. In patients with RMVT originating from the left ASC during tachycardia, this potential presented as either presystolic or late diastolic activation, whereas during sinus rhythm, it was the second ventricular activation component, which suggests that there was a slow conduction area between the ventricle and the left ASC. Again, this finding can be explained by the anatomic data. Myocardium from the septum and left ventricle is present in the right and left ASCs (17). In some patients, such myocardium can be arrhythmogenic and produce RMVT.

Anatomic considerations. For a better understanding of the origins of arrhythmias and approaches for catheter ablation, the anatomic arrangement between the RVOT and aortic sinus is relevant. Spatially, the aortic root occupies a central location within the heart, with the left and right coronary aortic sinuses adjacent to the left and right atrial appendages, respectively (Fig. 6a and 6b). The anteriorly situated RVOT passes slightly superior to and leftward of the aortic valve. The conical-shaped infundibulum of the right ventricular muscle is like a free-standing sleeve that elevates the pulmonary valve above the ventricular septum and superior to the aortic valve. Although commonly referred to as the "superior septal RVOT," the posterior wall of the infundibulum is not septal, because an extracardiac tissue plane interposes between its epicardial surface and the right and left coronary aortic sinuses, as clearly seen on simulated long-axis sections of the heart (Fig. 6c and 6d). The circular ventriculo-arterial junction between the muscle of the infundibulum and the wall of the pulmonary trunk is crossed by the semilunar hinge lines of the leaflets of the pulmonary valve. This arrangement leaves three semilunar areas of muscle in the troughs of the sinuses (18). The left ventricular outflow tract, in contrast, has part muscular and part fibrous walls (Fig. 6c). The fibrous continuity between the aortic and mitral valves lies between the noncoronary leaflet and the posterior part of the left coronary leaflet, to a greater or lesser extent. The larger part of the right coronary and a portion of the left coronary aortic leaflets are related to the ventricular septum and the free wall of the left ventricle, respectively (17). Therefore, in these areas, the semilunar leaflets are hinged superior to the

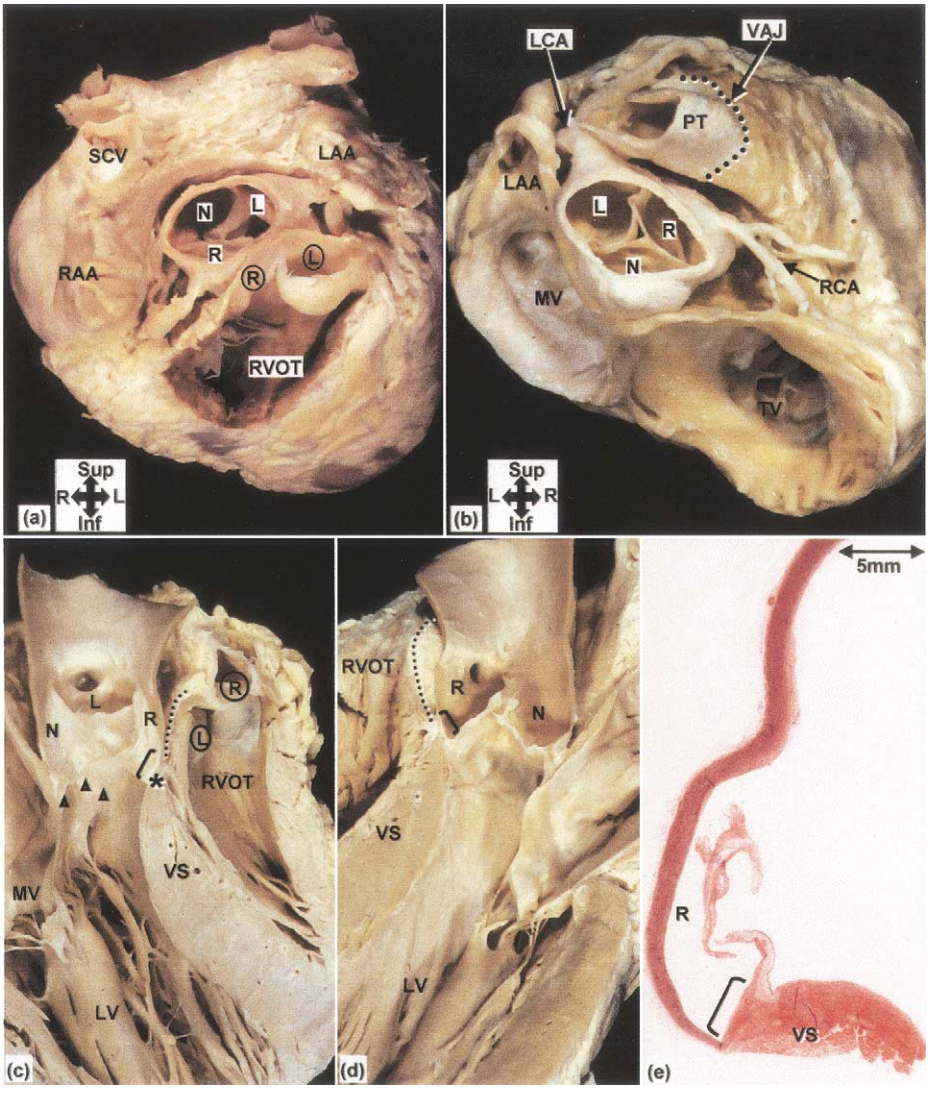

Figure 6. Heart specimens illustrating the anatomic arrangement between the right ventricular outflow tract (RVOT) and the aortic sinuses. **(a)** Viewed anteriorly, the RVOT passes leftward and superior to the aortic valve. **(b)** The posterior view shows the left (L) and right (R) coronary aortic sinuses adjacent to the pulmonary infundibulum. The noncoronary (N) aortic sinus is remote from the RVOT, but is related to the mitral valve (MV) and central fibrous body. The **dotted line** marks the ventriculo-arterial junction (VAJ) between the wall of the pulmonary trunk (PT) and right ventricular muscle. Note the cleavage plane behind the pulmonary infundibulum and in front of the aortic root. **(c and d)** These simulated parasternal long-axis sections show two halves of the same heart and display the left and right coronary orifices. The right- and left-facing pulmonary sinuses (R and L in circles, respectively) are situated superior to the aortic sinuses. The **dotted line** marks the epicardial aspect of the subpulmonary infundibulum in the so-called "septal" area. LAA = left atrial appendage; LCA = left coronary artery; LV = left ventricle; RAA = right atrial appendage; RCA = right coronary artery; SCV = superior vena cava; TV = tricuspid valve; VS = ventricular septum.

aortic wall but inferior to the muscle, enclosing ventricular muscle at the cusps of the sinuses (Fig. 6c to 6e). The posterior part of the right ASC is adjacent to the central fibrous body, which carries within it the penetrating His bundle. Anteriorly, the right ASC is related to the bifurcating atrioventricular bundle and the origin of the left bundle branch (Fig. 6d). The complex spatial relationships between the RVOT and ASC may explain the arrhythmia's ECG characteristics.

Radiofrequency ablation. Previous studies have shown that RMVT originating from the ASC can be successfully ablated (14–16), although aortic valve and coronary artery damage represent potential risks. In our patients, RF current was applied in the ASC 7.3 to 16.1 mm below the coronary artery ostium, and RMVT was terminated within 3 s in six patients and within 8 s in one patient. The immediate termination of tachycardia, in conjunction with the findings of autopsied hearts, is highly supportive of the arrhythmogenic substrate located superficially within the ASC. On the basis of these data, we suggest that RF application should be stopped if the RMVT cannot be terminated after 10 s. In addition, continuously visualizing the mapping catheter by fluoroscopy, with an angiographic catheter in the LMCA as a marker and for protection, was used in our patients to decrease the risk of complications during RF application in the ASC.

Study limitations. This small series has several limitations: 1) our patients did not represent the natural incidence of

RMVT or VPCs, with LBBB morphology and an inferior axis, because patients with complex arrhythmias were usually referred to our center. However, quantitative ECG analysis from this study provides practical criteria for distinguishing between RVOT and ASC origins; and 2) the pace mapping used in this study was not systematically performed, although all clinical arrhythmias were identified by activation mapping under isoproterenol infusion.

Conclusions. In this study, all 15 patients with LBBB morphology and an inferior axis were referred for ablation of "RVOT" arrhythmias. Our data suggest that in some patients, RMVT can originate from the left and right ASC 7.3 to 16.1 mm below the coronary artery ostium. Repetitive monomorphic ventricular tachycardia originating from the ASC shows a QRS morphology very similar to that of RVOT arrhythmias on the surface ECG. However, the indexes of R-wave duration and R/S-wave amplitude can distinguish between the two arrhythmias. Mapping within the ASC is mandatory in patients with higher indexes of R-wave duration and R/S-wave amplitude, especially in those with previously failed RF ablation in the RVOT. If the RMVT origin is located in the left coronary ASC, RF ablation can be safely performed with a catheter used to cannulate the LMCA as a marker and for protection.

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REFERENCES

1. Morady F, Kadish AH, Dicarlo L, et al. Long-term results of catheter ablation of idiopathic right ventricular tachycardia. Circulation 1990; 82:2093–9.
2. Klein LS, Shih HT, Hackett F, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. Circulation 1992;85:1666–74.
3. 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.
4. 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.
5. 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.
6. Zhu DW, Maloney JD, Simmons TW, et al. Radiofrequency catheter ablation for management of symptomatic ventricular ectopic activity. J Am Coll Cardiol 1995;26:843–9.
7. Rahilly GT, Prystowsky EN, Zipes DP, Naccarelli GV, Jackman WM, Heger JJ. Clinical and electrophysiologic findings in patients with repetitive monomorphic ventricular tachycardia and otherwise normal electrogram. Am J Cardiol 1982;50:459–68.
8. Buxton AE, Waxman HL, Marchlinski FE, Simson MB, Cassidy D, Josephson ME. Right ventricular tachycardia: clinical and electrophysiologic characterization. Circulation 1983;68:917–27.
9. Coumel P, LeClerq JP, Slamm R. Repetitive monomorphic ventricular tachycardia. Zipes DP, Jalife J, editors. Cardiac Electrophysiology and Arrhythmias. Orlando, FL: Grune and Stratton, 1985.
10. 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;79:990–9.
11. Callans DJ, Menz V, Schwartzman D, Gottlieb CD, Marchlinski FE. Repetitive monomorphic ventricular tachycardia from the left ventricular outflow tract: electrocardiographic patterns consistent with a left ventricular site of origin. J Am Coll Cardiol 1997;29:1023–7.
12. Krebs ME, Krause PC, Engelstein ED, Zipes DP, Miles WM. Ventricular tachycardia mimicking those arising from the right ventricular outflow tract. J Cardiovasc Electrophysiol 2000;11:45–51.
13. Frey B, Kreiner G, Fritsch S, Veit F, Gössinger HD. Successful treatment of idiopathic left ventricular outflow tract tachycardia by catheter ablation or minimally invasive surgical cryoablation. Pacing Clin Electrophysiol 2000;23:870–6.
14. Shimoike E, Ohnishi Y, Ueda N, Maruyama T, Kaji Y. Radiofrequency catheter ablation of the left ventricular outflow tachycardia from the coronary cusp: a new approach to the tachycardia focus. J Cardiovasc Electrophysiol 1999;10:1005–9.
15. Sadanaga T, Saeki K, Yoshimoto T, Funatsu Y, Miyazaki T. Repetitive monomorphic ventricular tachycardia of the left coronary cusp origin. Pacing Clin Electrophysiol 1999;22:1553–6.
16. Kanagaratnam L, Tomassoni G, Schweikert R, et al. Ventricular tachycardia arising from the aortic sinus of Valsava: an under-recognized variant of left outflow tract ventricular tachycardia. J Am Coll Cardiol 2001;37:1408–14.
17. Sutton JP III, Ho SY, Anderson RH. The forgotten interleaflet triangles: a review of the surgical anatomy of the aortic valve. Ann Thorac Surg 1995;59:419–27.
18. Stamm C, Anderson RH, Ho SY. Clinical anatomy of the normal pulmonary root compared with that in isolated pulmonary valvular stenosis. J Am Coll Cardiol 1998;31:1420–5.