Radiofrequency catheter ablation for idiopathic right ventricular tachycardia with special reference to morphological variation and long term outcome

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Abstract

Objective—To assess the long term outcome of radiofrequency (RF) catheter ablation for idiopathic ventricular tachycardia (VT) originating from the outflow tract of the right ventricle, with special reference to the morphological variation in the VT-QRS complexes.

Patients—13 patients whose ventricular tachycardia was treated with RF ablation were followed up more than 18 months after RF ablation.

Results—Endocardial mapping revealed the various extensions of ventricular tachycardia origin (from 0.5×0.5 cm to 2.0×2.0 cm) in which the earliest local electrogram was recorded during ventricular tachycardia. In all five tachycardias from a relatively wider origin (more than 0.5×0.5 cm) and in four of eight from a narrow origin ($< 0.5 \times 0.5$ cm), subtle morphological variation in the VT-QRS complexes was observed. In tachycardias with morphological variation, the local electrogram at the tachycardia origin also showed concomitant variation in morphology and activation sequence. Ventricular tachycardia from a narrow site was eliminated by RF ablation to the confined site, but a larger number of RF applications was required in tachycardias from a wider origin. All 13 tachycardias were successfully ablated by RF current, and during the follow up period of 28.2 (SD 7.2) months, recurrence was observed in only one patient who had a wider origin.

Conclusions—Long term efficacy of RF ablation was excellent in idiopathic ventricular tachycardia originating from the outflow tract of the right ventricle. Subtle morphological variations were frequently observed in this type of ventricular tachycardia, and about half of them represented a relatively wider arrhythmogenic area.

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Keywords: radiofrequency ablation; idiopathic ventricular tachycardia; right ventricle; morphological variation

Radiofrequency (RF) catheter ablation has been used for treatment of ventricular tachycardia (VT), and excellent short term results have been reported in some types.¹⁻³ However, little information is available on the long term

efficacy of RF ablation in ventricular tachycardia.^{4,5} The purpose of this study was to assess the long term results of RF ablation for idiopathic ventricular tachycardia originating from the outflow tract of the right ventricle, with special reference to subtle morphological variation in VT-QRS complexes. Electrophysiological characteristics at the site of ventricular tachycardia origin and their implications were also assessed through the findings of RF ablation.

Methods

Thirteen consecutive patients were selected for this study. The criteria of patient selection were as follows: (1) there were recurrent episodes of symptomatic monomorphic ventricular tachycardia; (2) VT-QRS morphology showed a left bundle branch block pattern (QS or rS in V1) and inferior axis deviation; (3) RF catheter ablation was employed for the treatment of ventricular tachycardia, and the patients were followed up for 18 months or longer after ablation; and (4) underlying heart disease could not be detected by conventional examination. Cardiac catheterisation and magnetic resonance imaging were also normal in six patients.

There were two male and 11 female patients, and their ages ranged from 19 to 65 years, mean (SD) 48 (13) years (table). Twelve-lead electrocardiograms were normal, and neither ST-T abnormality nor epsilon waves were detected. Seven patients had both sustained (> 30 seconds) and non-sustained (three beats to 30 seconds) ventricular tachycardia, and the other six patients showed incessant non-sustained ventricular tachycardia. Non-sustained ventricular tachycardia was observed during an exercise test on a treadmill in five patients. In all patients the ventricular tachycardia was of single morphology, and the mean (SD) cycle length was 265 (48) ms (range 180 to 360 ms). Before admission to our hospital, one to three conventional antiarrhythmic drug treatments failed to prevent the recurrence of ventricular tachycardia.

ELECTROPHYSIOLOGICAL STUDY

Informed consent was obtained after explanation of the procedure, results, and possible risks. An electrophysiological study was performed in the non-sedated and postabsorptive state. All antiarrhythmic drugs were discontinued for at least five half-lives before the study. Three to four electrode catheters were introduced through the femoral vein to stimulate

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Characteristics of patients and ventricular tachycardia (VT)

Case	Age/sex	VT	VTCL	Aggravation by ISP	QRS	EAS	Size (cm)	Watts	No of applications		Result	Follow up (months)
									Ineffective	Effective		
1	39F	Sustained VT/NSVT	280	Yes	Variable	-30~-40	< 0.5 × 0.5	30-40	0	3	Success	39
2	19M	Sustained VT/NSVT	180	Yes	Variable	-30~-50	1.0 × 2.0	30	0	5	Success	38
3	64M	NSVT	240	Yes	Variable	-20~-35	< 0.5 × 0.5	30-40	1	4	Success	34
4	48F	NSVT	340	Yes	Variable	-30~-40	1.0 × 2.0	30-40	5	11	Success	33
5	42F	Sustained VT/NSVT	250	No	Variable	-10~-20	< 0.5 × 0.5	40	1	4	Success	31
6	57F	NSVT	300	Yes	Variable	-15~-25	1.0 × 2.0	40	2	9	Success	30
7	63F	Sustained VT/NSVT	360	Yes	Variable	-20~-30	< 0.5 × 0.5	20	1	2	Success	30
8	47F	Sustained VT/NSVT	240	Yes	Mono	-30	< 0.5 × 0.5	40	0	2	Success	30
9	53F	Sustained VT/NSVT	250	No	Mono	-25	< 0.5 × 0.5	20-30	12	8	Success	23
10	39F	NSVT	260	Yes	Mono	-30	< 0.5 × 0.5	20-30	4	3	Success	22
11	49F	NSVT	230	Yes	Variable	-10~-20	1.0 × 1.5	20-30	2	5	Success	20
12	36F	Sustained VT/NSVT	240	Yes	Variable	-15~-25	2.0 × 2.0	20	5	9	Recurrence	18
13	65F	NSVT	280	NP	Mono	-20	< 0.5 × 0.5	20-30	4	4	Success	18

NSVT, non-sustained VT; VTCL, cycle length of VT (ms); ISP, isoprenaline; NP, not performed; mono, monomorphic; EAS, earliest activation site.

the heart and to record the intracardiac electrograms. Band pass filter settings of 50 and 500 Hz were used to record the intracardiac electrograms.

To examine the inducibility of ventricular tachycardia,^{6,7} one to three extrastimuli were delivered after eight basic stimuli at two different basic cycle lengths (400 and 600 ms), followed by rapid pacing at cycle lengths up to 286 ms. Stimulation was attempted at two sites of the right ventricle (apex and outflow tract). If ventricular tachycardia was not inducible, isoprenaline was infused to increase the sinus rate by 20% and stimulation with the same protocol was repeated. The endpoint was the completion of the protocol or the induction of monomorphic sustained ventricular tachycardia. If sustained ventricular tachycardia was induced, rapid pacing was attempted to entrain the tachycardia from one or two sites of the right ventricle. The criteria of the transient entrainment, either classic or concealed, were the same as those reported by earlier workers.^{8,9}

ENDOCARDIAL MAPPING AND ABLATION SITE

Using a steerable 7F quadripolar catheter (Mansfield, Boston Scientific International, Watertown, Massachusetts, USA), the site of origin of ventricular tachycardia was mapped as the earliest activation site during ventricular tachycardia. It was mandatory to confirm that the local electrograms—recorded approximately 0.5 cm apart from the earliest activation site in four directions—should be delayed compared to the earliest activation site. If an isochronal presystolic electrocardiogram of the earliest activation site was confined within a site of dimensions 0.5 × 0.5 cm, ventricular tachycardia was considered to originate from a narrow site; otherwise it was considered to have a wide origin.¹⁰ Pace mapping was also used to facilitate mapping the tachycardia origin, but the site of origin was not based on pace mapping alone.

ABLATION PROCEDURE

RF currents were provided by a generator (HAT 200 Osypka, Grenzae-Wyhlen, Germany; 520 kHz), and delivered at 20-40 W to the distal large (4 mm) tip of the electrode, which was placed at the earliest activation site or within the area where the earliest isochronal electrograms were recorded. The site was considered to be appropriate if sus-

tained ventricular tachycardia was terminated or incessant non-sustained ventricular tachycardias or frequent premature ventricular complexes, or both, disappeared. In such instances, RF current was applied for 30 seconds. Otherwise, RF current was turned off after 10 seconds. The former RF application was considered to be effective and the latter ineffective. If impedance began to rise, the current was turned off immediately.

Complete disappearance of spontaneous ventricular tachycardia after ablation as assessed by electrocardiographic monitoring was the criterion of success. When ventricular tachycardia was induced by programmed electrical stimulation, the efficacy was judged by electrophysiological study at the end of ablation and two weeks later. For ventricular tachycardia induced by isoprenaline infusion or a treadmill exercise test before ablation, or both, ablation was judged to be successful when ventricular tachycardia was rendered non-inducible by these interventions.

After ablation, intravenous administration of heparin sodium was continued for one day at a dose of 10 000-15 000 units. A cross sectional echocardiogram was recorded immediately after the procedure and one to two weeks later.

ANALYSIS OF DATA

The patients were followed up in the outpatient clinic at least once one month in a drug free state. Since all patients were aware of each episode of ventricular tachycardia, recurrence of tachycardia was suspected on the basis of symptoms and confirmed by Holter electrocardiograms.

Special reference was given to ventricular tachycardia that showed a small change in the amplitude or configuration of the QRS complex (notch, slur, etc), or in its electrical axis (less than 45°), without any change in the bundle branch block pattern. The incidence of such subtle changes in tachycardia morphology was recorded. Great care was taken to exclude the possibility of respiratory variation or fusion with supraventricular beats as the cause of the morphological variation. To avoid catheter induced artefactual change in VT-QRS morphology, 12-lead electrocardiograms which were obtained during mapping at the outflow tract of the right ventricle were not used in the assessment of morphological variation. The cases were divided into two groups

Figure 1 Twelve-lead electrocardiogram.
(A) Ventricular tachycardia (VT) with a subtle morphological variation (case 4). Beat to beat variation in the morphology is evident.
(B) Monomorphic VT without morphological variation from a different case (case 8). All QRS complexes are identical.

according to whether the earliest local electrogram was confined to a narrow site or a wide area. The number of RF applications was compared between the two groups. Finally, the short and long term follow up data on RF ablation were analysed.

STATISTICAL ANALYSIS

Ventricular tachycardias with a narrow origin and those with a wide origin were compared using the Student *t* test and Fisher's exact probability test. Data are expressed as mean (SD) and differences are considered statistically significant at $P < 0.05$.

Results

INDUCTION AND MORPHOLOGICAL VARIATION OF VENTRICULAR TACHYCARDIA

Clinical ventricular tachycardia was induced

by programmed electrical stimulation in two patients (cases 1 and 2) and by constant pacing in another two patients (cases 4 and 11). Non-sustained ventricular tachycardia or premature ventricular complexes, or both, were increased during administration of isoprenaline in six patients, and sustained ventricular tachycardia was induced by isoprenaline in another two patients (table).

Ventricular tachycardia was monomorphic in all patients, but subtle morphological variation in the VT-QRS complex was observed in nine of the 13 patients (69%) (fig 1). The cycle length of the ventricular tachycardia did not differ between cases with and without subtle morphological variation: 269 (57) *v* 258 (17) ms, NS. During ventricular tachycardia, no ventriculoatrial conduction was observed in any patient, and the morphological variation was independent of atrial contraction. In the

Figure 2 Twelve-lead electrocardiogram. The patient is the same as in fig 1, panel A (case 4). Premature ventricular complexes (PVCs) in sinus rhythm also showed variation in the QRS complex, and these morphologies were included in the VT-QRS complex. Neither QRS nor ST-T abnormalities were noticed during sinus rhythm.

Figure 3 Electrophysiological findings. During ventricular tachycardia (VT), the earliest activation site was determined within a narrow site (0.5×0.5 cm) at the outflow tract of the right ventricle (case 1). However, the local electrogram at the site of VT origin showed variation in morphology and activation sequence (panel A). The 12-lead electrocardiogram of this case is shown in fig 5 (panel A). RF current of 40 W was delivered to the site, and VT was terminated within a few seconds (panel B). RVA, apex of the right ventricle; RVOT, outflow tract of the right ventricle; RF, radiofrequency current.

nine patients with subtle morphological variation, premature ventricular complexes also showed morphological variation, and two to four configurations were observed in each patient (fig 2).

RESULTS OF MAPPING

The site of ventricular tachycardia origin was determined at the outflow tract of the right ventricle in all patients (free wall in nine and interventricular septum in four). The mean interval from the local electrogram to the onset of the QRS complex at the site of ventricular tachycardia origin was 30 (9) ms (range 10 to 50 ms). During sinus rhythm and ventricular tachycardia, no abnormal local electrogram was recorded in any segment of the right ventricle except in one case (case 2) in which ventricular tachycardia was induced by programmed stimulation but transient entrainment was not observed by rapid pacing from two sites of the right ventricle. A slow pathway was not determined in any patient.

The extension of the area with the earliest

local electrogram during ventricular tachycardia was different for each patient and varied from 0.5×0.5 cm to 2.0×2.0 cm (table). In eight of 13 patients, the earliest activation was confined to a relatively narrow site ($< 0.5 \times 0.5$ cm), and morphological variation was observed in four of the eight patients. The earliest isochronal electrograms were obtained from a wider area (more than 0.5×0.5 cm) in the other five patients, in whom ventricular tachycardia consistently showed subtle morphological variation in VT-QRS complexes. The local electrogram at the origin showed variation in morphology and activation sequence when VT-QRS complexes showed morphological variation (figs 3 and 4). Pace mapping at the site of ventricular tachycardia origin showed almost identical morphology to that of clinical ventricular tachycardia (fig 5), but the morphological variation of the clinical arrhythmia could not be reproduced during pace mapping.

RESULTS OF RF ABLATION

In seven patients, RF current was delivered during ventricular tachycardia (sustained ventricular tachycardia in three, incessant non-sustained ventricular tachycardia in four) and the tachycardia was terminated by RF current in all patients (figs 3 and 4). In the other six patients, RF current abolished the frequent premature ventricular complexes, which showed an identical QRS morphology to that of ventricular tachycardia.

With between 2 and 11 effective RF applications (mean 5.3 (3.0)), all ventricular tachycardia was successfully ablated. RF application to narrow sites of origin was rapidly effective in terminating the tachycardia (fig 3). However, in cases with a wide origin, tachycardia of the same morphology remained inducible at the end of each RF application, and three to seven separate RF lesions were needed over the area of origin until the tachycardia was completely suppressed (fig 4). Significantly more RF applications were needed when the tachycardia originated from a relatively wider area than from a narrow site: 7.8 (2.7) *v* 3.8 (1.9), $P < 0.01$.

Two weeks after RF ablation, no ventricular tachycardia could be induced by electrical stimulation, treadmill exercise test, or intravenous administration of isoprenaline. Continuous electrocardiographic monitoring (> 10 days) showed no ventricular tachycardia. There were no major complications during or after RF ablation. Therefore the success rate of RF ablation was 100% (13/13 patients).

LONG TERM FOLLOW UP

After discharge, all patients were followed up at our outpatient clinic at least once a month without antiarrhythmic drugs. During the follow up period of 28.2 (7.2) months (range 18 to 39), neither recurrences of ventricular tachycardia nor symptoms suggesting tachyarrhythmia were observed in any of the patients, with one exception. This patient (case 12) had a wider site of origin ($2.0 \times$

Figure 4 Electrophysiological findings. This case is the same as in fig 1 (panel A) (case 4). The earliest activation site of the ventricular tachycardia (VT) was determined at the outflow tract of the right ventricle, but isochronal presystolic local electrograms were obtained from an area extending 1.0×2.0 cm. Local electrograms obtained from two different sites are shown in panels A and B, and these sites were separated 1.5 cm from one another. These local electrograms show variation in morphology and activation sequence. RF ablation to each site terminated the VT within a few seconds, but VT with the same morphology occurred until the area with isochronal presystolic electrogram was covered by several RF lesions. HBE, His bundle electrogram; RVA, apex of the right ventricle; RVOT, outflow tract of the right ventricle; RF, radiofrequency current.

Figure 5 Twelve-lead electrocardiogram of ventricular tachycardia (VT) with subtle morphological variation (case 1) in panel A, and pace mapping obtained from the earliest activation site of VT in panel B. The paced morphology was almost identical to the VT morphology.

2.0 cm), and ventricular tachycardia with the same QRS morphology recurred three months after RF ablation. However, the tachycardia was easily suppressed with mexiletine (300 mg per day), and thereafter it had not recurred up to the time of writing (15 months). Therefore, long term efficacy of RF ablation was observed

in 12 of 13 patients (92%), and clinical efficacy was observed in all patients.

Discussion

MECHANISM OF VENTRICULAR TACHYCARDIA

In this study, programmed electrical stimula-

tion failed to induce clinical ventricular tachycardia in 11 of 13 patients, but isoprenaline or exercise testing on a treadmill induced non-sustained ventricular tachycardia or premature ventricular complexes in eight of the 11 patients. In the remaining two patients (cases 1 and 2), clinical ventricular tachycardia was induced by programmed stimulation, but no criterion of transient entrainment was fulfilled. Therefore, a non-reentrant mechanism—automaticity or a triggered activity—is the likely cause of these ventricular tachycardias,^{11,12} though microreentry could not be excluded in two patients.

FINDINGS ON MAPPING

Endocardial mapping of the site of origin of the ventricular tachycardia revealed the various extensions of earliest activation site (from 0.5×0.5 to 2.0×2.0 cm), and tachycardias from a relatively wide origin consistently showed subtle morphological variation. Such variation was observed in only 50% of cases with a narrow origin. This morphological variation was not due to respiration, fusion with supraventricular beats, or a change of R-R intervals during ventricular tachycardia. Shorter tachycardia cycle lengths or different degrees of ventricular filling may be related to the morphological variation,¹³ but the tachycardia cycle length did not differ depending on whether there was morphological variation, and the variation was independent of atrial activation or blood pressure. Therefore, the subtle morphological variation in VT-QRS complexes was considered to be an essential electrophysiological characteristic of this type of ventricular tachycardia.

The concomitant variation in configuration and activation sequence in the local electrogram at the site of origin of the ventricular tachycardia suggests that there are multiple sites of origin or multiple exits from a relatively wide origin,¹⁴ or multiple exits or different activation patterns distal to the exit in cases with a narrow origin.¹⁵ It is unlikely that the wide origin represents multiple breakouts from an origin deep within the myocardium. Because each RF application terminated the ventricular tachycardia, the thermal effect has to reach the essential arrhythmogenic substrate and injure a specific site of origin. On the other hand, a small number of RF applications was sufficient to ablate ventricular tachycardia in cases with a narrow origin, though morphological variation of VT-QRS complexes could occur (fig 3).

The arrhythmogenic substrate of idiopathic ventricular tachycardia of right ventricular outflow tract origin seems to be non-uniform, and the relation between the morphological variations in VT-QRS complex and the mapping data at the site of ventricular tachycardia origin needs to be studied further.

RESULTS AND COMPLICATIONS OF RF ABLATION

RF ablation was highly effective and safe in treating idiopathic ventricular tachycardia originating from the outflow tract of the right ventricle; it was successful in 92% of the cases

(clinically in 100%). Therefore, catheter ablation with direct shock will no longer be recommended for this type of ventricular tachycardia. However, a larger number of RF applications needs to be delivered to ablate tachycardias with a wide origin, and one of these cases was reported previously.¹⁶ During the follow up period of 28.2 (7.2) months, ventricular tachycardia recurred in one patient; however, it responded to mexiletine satisfactorily.

LIMITATIONS

Variation in VT-QRS morphology seems to be frequent in ventricular tachycardia originating from the outflow tract of the right ventricle, but this finding has not been addressed so far by other investigators. Ablation of a specific type of QRS morphology at a specific site suggests that the variation is not an artefact,¹⁷ and data from a larger number of patients will provide more information. Underlying subclinical heart disease, such as arrhythmogenic right ventricular dysplasia,^{18,19} cannot be excluded completely.

CONCLUSIONS

RF ablation was effective and safe—and excellent long term efficacy was confirmed—in idiopathic ventricular tachycardia of right ventricular origin. Subtle morphological variation of VT-QRS was often found and it was associated with a wider arrhythmogenic area.

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