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 x 0.5 cm to 2.0 x 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 x 0.5 cm) and in four of eight from a narrow origin (< 0.5 x 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.

(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
Characteristics of patients and ventricular tachycardia (VT)

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<th>QRS</th>
<th>EAS</th>
<th>Size (cm)</th>
<th>Watts</th>
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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, 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 1.2 times the basic cycle length for 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.

ENDOCARDIAL MAPPING AND ABLATION SITE Using a steerable 7F quadrupolar 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 x 0.5 cm, ventricular tachycardia was considered to originate from a narrow site; otherwise it was considered to have a wide origin. 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, Grenzne-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 sustained 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 tachycardia began to rise, the current was turned off immediately.

Complete disappearance of spontaneous ventricular tachycardia after ablation was 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 a 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.) 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.
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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.
local electrogram during ventricular tachycardia was different for each patient and varied from $0.5 \times 0.5$ cm to $2.0 \times 2.0$ 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 $0.5 \times 0.5$ cm) in the other five patients, in whom ventricular tachycardia consistently showed subtle morphological variation in VT-QRS complexes. The local electrograms at the VT 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) vs 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 isoproterenol. 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 ($2.0 \times$
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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, 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 30% 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, 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, or multiple exits or different activation patterns distal to the exit in cases with a narrow origin. It is unlikely that the wide origin represents multiple breakthroughs 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. 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, and data from a larger number of patients will provide more information. Underlying subclinical heart disease, such as arrhythmogenic right ventricular dysplasia, 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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