RADIOFREQUENCY CURRENT CATHETER ABLATION FOR VENTRICULAR TACHYCARDIA

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Radiofrequency current catheter ablation was attempted for 17 morphologies of ventricular tachycardia (VT) in 14 patients. Five patients had underlying heart disease. The site of VT origin was determined as the earliest site of ventricular activation, or by pacing within the area of slow conduction. In 15 VTs, ablation was performed during VT, and 12 VTs (80%) were terminated within an average of 5.4 ± 4.2 seconds. After ablation, 14 VTs ($14/17=82\%$) of 11 patients ($11/14=79\%$) could not be induced by electrical stimulation. Radiofrequency ablation appeared to be more effective in VTs without underlying heart disease (91%), and in VTs originating from the right ventricle (100%). Successful ablation sites usually showed a normal local electrograms during VT. Ablation in the slow conduction area was attempted in 3 VTs, and 2 VTs became noninducible. The mean number of applications of radiofrequency current for each VT origin was 7.7 ± 6.4 at 20–50 Watts. In 4 patients, application of radiofrequency current was required 10 or more times because of a possible large arrhythmogenic area, or because of reinduction of VT, even though VT was terminated by radiofrequency current. No major complication was observed except for complete right bundle branch block in 1 patient. In conclusion: (1) Radiofrequency catheter ablation was considered to be effective and safe, especially for VT without underlying heart disease or VT originating from the right ventricle. (2) Ablation during VT was considered to be useful for identifying the proper ablation site and to avoid creating an unnecessary lesion.

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RADIOFREQUENCY (RF) current catheter ablation has been widely used for the treatment of Wolff-Parkinson-White syndrome^{1,2} and atrioventricular nodal reentrant tachycardia^{3,4}. The clinical efficacy and safety of this therapy has been established. On the other hand, the results of RF ablation in sustained ventricular tachycardia (VT) have been less satisfactory⁵, and this may be related to the relatively smaller lesion produced by the RF current^{6,7}. Recently, the number of VT cases successfully

ablated by RF current has grown^{8–12}.

In the present study, we applied RF ablation to 17 monomorphic VTs in 14 consecutive patients, and evaluated the clinical efficacy and safety of RF ablation for VT. Some characteristics related to successful ablation were also analyzed.

SUBJECT AND METHODS

Patients

From October 1991 through December 1992, RF catheter ablation was attempted in 14 patients with symptomatic, drug refrac-

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TABLE I PATIENTS' CHARACTERISTICS

	Case	Type	UHD	Morphology	VTCL	Origin	Induction	Symptom
①	R.K. 60M	SVT	idio	LBBB+NAD	270	RV (His)	EPS	syncope
②	Y.I. 53M	SVT	ARVD	LBBB+NAD	220	RVOT	ISP	palpitation/dizziness
③	H.S. 66M	SVT	idio	RBBB+NAD	340	RV (His)	EPS (ISP)	palpitation/dizziness
		SVT		LBBB+NAD	380	RV (His)	EPS (ISP)	
④	S.Y. 55F	SVT	(PSS)	LBBB+LAD	270	RVIT	EPS	palpitation/dizziness
		SVT		LBBB+LAD	255	RV (His)	EPS	
⑤	B.N. 69M	SVT	(CRF)	LBBB+NAD	260	RVOT	ISP	palpitation/dizziness
⑥	M.T. 19M	SVT	idio	LBBB+NAD	180	RVOT	EPS	syncope
⑦	T.S. 39F	SVT	idio	LBBB+LAD	280	RVOT	ISP	palpitation/dizziness
⑧	S.A. 36M	SVT	DCM	LBBB+LAD	300	RVOT	EPS (ISP)	palpitation/dizziness
⑨	S.I. 64M	NSVT	idio	LBBB+LAD	240	RVOT	ISP	palpitation/dizziness
⑩	M.Y. 48F	NSVT	idio	LBBB+LAD	340	RVOT	EPS (ISP)	syncope
⑪	T.K. 21M	SVT	idio	RBBB+LAD	360	LV	EPS	palpitation/dizziness
⑫	S.E. 45M	SVT	OMI	RBBB+RAD	380	LV	EPS	palpitation/dizziness
⑬	K.O. 51F	SVT	DCM	LBBB+RAD	380	RVOT	EPS	palpitation
⑭	K.K. 68M	SVT	DCM	RBBB+LAD	260	LV	EPS	palpitation/dizziness
		SVT		RBBB+RAD	300	LV	EPS	

ARVD=arrhythmogenic right ventricular dysplasia, CRF=chronic renal failure, DCM=idiopathic dilated cardiomyopathy, EPS=electrophysiologic study, idio=idiopathic, Induction=the methods used to induce VT, ISP=isoproterenol, LAD=left axis deviation, LBBB=left bundle branch block pattern, LV=left ventricle, NAD=normal axis, NSVT=nonsustained ventricular tachycardia, Origin=origin of VT, PSS=progressive systemic sclerosis, OMI=old myocardial infarction, RAD=right axis deviation, RBBB=right bundle branch block pattern, RV=right ventricle, RVIN=right ventricular inflow tract, RVOT=right ventricular outflow tract, SVT=sustained ventricular tachycardia, Symptom=symptoms during spontaneous VT, UHD=underlying heart disease, VTCL=ventricular tachycardia cycle length, Cases 1–7 were previously reported in *Am Heart J*¹⁰

tory, and monomorphic VTs; sustained VT in 12 patients, and incessant nonsustained VT in 2 patients. Since 3 patients had 2 morphologically distinct VTs, ablation was attempted in a total of 17 VT morphologies. As shown in Table I, 10 patients were male and 4 were female. The ages ranged from 19–69 years (mean $\pm$ SD; 50 ± 16 years). Five patients had underlying heart diseases: remote myocardial infarction in 1, arrhythmogenic right ventricular dysplasia in 1, and idiopathic dilated cardiomyopathy in 3. In the other 9 patients, underlying heart diseases could not be detected by conventional cardiac examinations. Of these 9, 1 patient had been receiving hemodialytic therapy for chronic renal failure. Another female patient had progressive systemic sclerosis.

Before admission to our hospital, a history of syncope was observed in 3 patients, and palpitation and/or dizziness was observed in the remaining 11 patients. The first episode of VT occurred 10 to 540 days (mean 165 ± 167 days) before the catheter ablation, and

the mean number of spontaneous VT attacks was 3.2 ± 3.4 per patient.

Before the ablation, pharmacologic therapy with conventional drugs failed to prevent the recurrence or induction of VT in an electrophysiologic study, and the mean number of drug tests was 2.5 ± 1.9 per patient. None of the patients was tested with amiodarone.

Electrophysiologic Study (EPS)

Informed consent was obtained after the procedure, and possible results and complications were explained to the patients. The electrophysiologic study was performed in the non-sedated and postabsorptive state. Three 6F quadripolar catheters with interelectrode distances of 5 mm were positioned within the heart under fluoroscopic guidance. Stimulations were performed using the distal and third electrodes, and the intracardiac electrogram was recorded using the second and fourth electrodes of the same catheter. Otherwise, the local electrogram was recorded using a pair of distal elec-

Fig.1. Schematic presentation of how to determine the area with the earliest ventricular activation.

When the local electrogram, at 5 mm from the site of the earliest ventricular activation, showed an equivocal activation sequence, as compared to that at the earliest activation site, the mapping catheter was moved in 5 mm steps, and the margin of the arrhythmogenic area was determined. At that time, radiofrequency ablation was applied to produce linear lesions to cover the possible arrhythmogenic area.

The shaded circle represents the possible arrhythmogenic area, and dark shaded circles show the lesions produced by radiofrequency current.

trodes. To record the His-bundle electrogram, a 6F tripolar catheter with an interelectrode distance of 10 mm was used. Intracardiac electrograms were recorded on an ink-jet recorder (Siemens Elema Co. Ltd. Mingograf 7) with 3 surface leads, I, II, and V1, at a paper speed of either 100 or 200 mm/sec. The band-pass filter was set at 50 to 500 Hz.

Protocol for VT Induction

Programmed electrical stimuli were delivered at twice the late diastolic threshold of 2 milliseconds using a cardiac stimulator (Fukuda Denshi Co, Cardiac Stimulator BC02). Our standard stimulation protocol consisted of programmed electrical stimulations; 1 to 3 extrastimuli delivered after 8 basic stimuli at 2 different basic cycle lengths (400 and 600 milliseconds), and rapid pacings at cycle lengths between 667 and 286 milliseconds for 5 to 15 sec. Stimulations were first attempted at the apex and then at the outflow tract of the right ventricle if VT

was not induced at the first site. If this protocol did not induce VT, isoproterenol was infused to increase the sinus rate by 20 percent, and stimulation with the same protocol was repeated at 2 sites in the right ventricle and at one site in the left ventricle. In 8 patients, a treadmill exercise test was attempted before the ablation to determine whether VT was provoked by the test.

Mapping the Site of VT Origin

In this study, the site of VT origin was defined as follows. First, the earliest site of ventricular activation^{13,14} was determined by extensive endocardial mapping during VT. Great care was taken to ensure that the local electrograms recorded 5 mm from the site of earliest activation were delayed versus that at the earliest activation site. When the difference in the activation sequence was equivocal (less than 10 milliseconds difference), the arrhythmogenic site was considered to extend as an area. At that time, the mapping catheter was moved away from the ear-

liest activation site in 5 mm steps, and the border of the arrhythmogenic area was determined. Since the site of VT origin in most of the patients in this study was at the out-flow tract of the right ventricle, the arrhythmogenic area could be determined by this approach (Fig. 1). Second, when an abnormal local electrogram, such as mid-diastolic potential¹⁵ or continuous activity¹⁶ was recorded during VT, rapid pacing was performed to determine the slow conduction zone.¹⁷ The following criteria were used as evidence of a critical slow conduction area: (1) A distinct local electrogram prior to the surface QRS complex, usually mid-diastolic, was obtained during VT; (2) The interval between the pacing stimulus and the onset of the surface QRS complex during pacing was identical to the interval between the local electrogram and the onset of the surface QRS complex during VT; and (3) Rapid pacing entrained VT without changing the QRS morphology, i.e., concealed entrainment.^{17,18} In some patients, pacemapping during sinus rhythm was used to help indicate the site of VT origin.

RF Ablation

A 7F quadripolar electrode catheter with a 4 mm tip electrode (Mansfield/Webster polaris) was positioned against the VT origin or at the slow conduction area within the reentry circuit.

An RF current was delivered at 20–40 Watts between the distal tip of the electrode catheter and a skin patch on the back. Whether or not RF was delivered to a proper site was judged by observing either the termination of VT within 10 sec, or a reduction in the rate of VT.^{10,19} In such instances, the current was delivered to the site for 20–30 sec. Otherwise, the current was discontinued at 10 sec, and the position and stability of the catheter was checked. When the impedance rose promptly, the current was turned off immediately. Radiofrequency current was provided through a generator (HAT 200 OSYPKA GmbH 520 KHz Germany). When the effect was transient, the energy was increased to 50 Watts.

After the ablation, intravenous administration of heparin sodium was continued for a day at a dosage of 15000 units. The electrocardiogram was monitored daily until the

patient was discharged. Serum CPK or CPK-MB was measured at 4–6 h and 12–24 h after the ablation. A cross-sectional echocardiogram was obtained both immediately and 1–2 weeks after the procedure.

Efficacy of RF Ablation

For VTs that were induced by programmed stimulations before ablation, the efficacy of ablation was evaluated by programmed stimulation of the heart at the end of the procedure and at 2 weeks after the ablation. Ablation was judged to be successful when VT was no longer induced after the induction protocol was complete. For VTs that were induced by either isoproterenol infusion or a treadmill exercise test before the ablation, the same procedures were performed 1–2 weeks after the ablation. VTs were no longer induced in cases that were judged to be successful. In such cases, programmed stimulations were also applied in an attempt to induce VT after ablation.

Definitions

Fractionated electrograms were defined as those showing a duration of 100 milliseconds or longer with a low peak to peak amplitude of 1.0 mV or less.²⁰ An electrogram that spanned the entire cardiac cycle was defined as continuous activity.¹⁶ Mid-diastolic potential¹⁵ was defined as a discrete potential separated by an isoelectric line between VT QRS complexes.

A sustained VT was defined as VT that lasted for more than 30 sec, or that required emergency intervention for termination within 30 sec. A non-sustained VT was defined as VT of 6 beats to 30 sec in duration.

Statistical analysis

Statistical analysis between 2 groups was performed with Student's t-test and Fisher's exact probability test. Multiple comparisons of proportion were tested by Ryan's method. A p value of less than 0.05 was considered significant. Values are presented as the mean $\pm$ SD.

RESULTS

(1) Characteristics and Induction of VT

We investigated 17 VT morphologies in 14 patients (12 VTs with a left bundle branch

Fig. 2. Ablation with radiofrequency (RF) current.

The patient was a 39-year-old woman (case 7) without underlying heart disease. The VT origin was determined to be at the outflow tract of the right ventricle and the local electrogram at this site was 30 milliseconds prior to the onset of QRS activation. RF current was delivered to that site at 30 Watts. VT was terminated 4.5 sec after delivery of RF current (downward arrow). The same VT was reinduced immediately after ablation, and 2 additional applications near the first ablation site were required to prevent induction of VT.

RF=radiofrequency current. RVA=apex of the right ventricle. RVOT=outflow tract of the right ventricle. S=seconds.

block pattern and 5 VTs with a right bundle branch block pattern) (Table I).

VT was repeatedly induced and terminated by programmed stimulation in 9 VTs of 7 patients, and transient entrainment was confirmed in 7 of these VTs. VT was induced by stimulations only after the administration of isoproterenol in 4 VTs of 3 patients. VT occurred spontaneously during administration of isoproterenol in 4 other VTs of 4 patients, and was considered to be catecholamine-sensitive VT²¹ In 2 patients, VTs that showed identical QRS morphologies to that of a clinical VT were induced by the treadmill exercise test.

(2) Mapping of the Site of VT Origin

The VT origin was determined as the site of earliest activation during VT in all of the VTs. In 3 VTs of 3 patients, a slow conduction area was also demonstrated by rapid pacing, but the areas were 1 to 2 cm away from the earliest activation site during VT. In some patients, pacemapping was also used to help determine the site of VT origin. However, there was a slight difference in the QRS morphology between VT and paced QRS, and the site of origin was not deter-

mined solely on the basis of pacemapping.

VT originated from the outflow tract of the right ventricle in 8 VTs, from the inflow tract of the right ventricle in 1 VT, and from the free wall of the left ventricle in 4 VTs. In the other 4 cases, VT arose from the right ventricular side of the interventricular septum close to the site of the His-electrogram recording area (Table I).

(3) RF Ablation

In 15 VTs, RF current was delivered during VT, and terminated soon after initiation of the ablation in 12/15 VTs at a mean time of 5.4 ± 4.2 sec (Fig. 2). In 2 VTs, ablation was performed during sinus rhythm because VT was not sustained. However, the site of earliest activation was determined during nonsustained VT.

Two to 26 applications (mean 7.7 ± 6.4) were delivered to the VT origin. When VT was terminated within 10 sec during the application of radiofrequency current, 1 to 5 additional applications of RF current were administered to sites within 5 mm from the VT terminating site. In 4 VTs, RF current was applied more than 10 times because although VT was terminated within a few

Fig.3. Electrophysiologic findings.

The patient was a 36-year-old man with idiopathic dilated cardiomyopathy (case 8). As shown in panels A to D, an arrhythmogenic area was determined within an area of 2.0 cm×2.0 cm, and each local electrogram from within this area was 50 milliseconds before the onset of QRS activation. However, the morphology of each local electrogram was different. Ablation of each of these sites terminated the VT, but VTs with the same morphology occurred until the area was crossed by RF lesions in the form of 2 perpendicular lines. The duration of the application required to terminate the VT is shown at the bottom of each panel. HBE=His electrogram recording area, MAP=mapping catheter. Other abbreviations are the same as in Fig. 1.

seconds, it remained inducible immediately after the ablation in 1 VT, or the arrhythmogenic area extended a few square cm in 3 VTs (Fig. 3). In the latter cases, RF ablation was applied systematically to create 2 intersecting lines to cover the area, and 2 of these 3 VTs became noninducible after such ablation (Figs. 1 and 4).

In another patient with incessant non-sustained VT, the surface ECG showed slightly changing QRS configurations, and the interval between the local electrogram and each QRS configuration varied slightly (Fig. 5). However, the earliest activation site for each QRS configuration was found within 1.0 cm, and RF ablation to each of the earliest ventricular activation sites successfully suppressed VT.

In 3 VTs, the slow conduction area in the reentry circuit was determined and radiofrequency current was applied to the site. Two of these 3 VTs were terminated during this application.

The mean number of ablation sessions per

patient was 1.8 ± 1.2 (range 1 to 5), and each procedure required from 2 to 5 h. The mean energy required per VT ablation was 6517 ± 5362 joules (from 1800 to 21800).

(4) Acute and Subacute Testings

Twenty minutes after the ablation, 14 of 17 VTs, in 11 of 14 patients, could not be induced by electrical stimulations before or after the administration of isoproterenol. In 7 patients, the electrophysiologic study was repeated 2 weeks later, but VT could still not be induced. After the ablation, a treadmill exercise test and/or intravenous administration of isoproterenol were performed in 5 patients in which VT had been induced by these procedures before the ablation, but no VTs were observed after the ablation. Therefore, radiofrequency ablation was effective in 82% of the VTs and 79% of the patients.

In the 3 other VTs in the 3 other patients, radiofrequency ablation could not prevent the induction of VT. One of these patients

Fig. 4. Application of radiofrequency current.

This case is the same as in Fig. 3 (case 8). Radiofrequency current at 40 Watts was applied to the area of the earliest ventricular activation (B in Fig. 3). VT was terminated 3.3 sec after the initiation of ablation, but the same VT was reinduced until systematic ablation was complete (see text).

Abbreviations are the same as in Fig. 3.

underwent catheter ablation using direct current shock (150 joules). However, a permanent pacemaker had to be implanted because of complete atrioventricular block. The efficacy of amiodarone was confirmed in another patient who had had an old myocardial infarction. While the third patient was waiting for the second trial, the site with the earliest ventricular activation during VT grew to 2.0 cm×2.0 cm with a fragmented local electrogram.

(5) Factors for Successful Outcomes

Although the success rate tended to be higher in VT without underlying heart disease, the difference between VT with and without underlying heart disease was not statistically significant (10/11 VTs=91% vs 4/6 VTs=67%, $P>0.1$). RF ablation was successful in only 6 of the 9 sites (67%) which showed fragmented activities, but was successful in 100% of the 8 sites with normal local electrograms. The interval between the local electrogram and the onset of the VT QRS complex was similar in the successful and unsuccessful ablation sites (38 ± 11 msec

in successful sites vs 30 ± 17 msec in unsuccessful sites, $P>0.1$).

The success rate for ablation of VT from the free wall of the right ventricle was 100% (9/9 VTs). In contrast, the success rate for ablation of VT from the septum of the right ventricle was 75% (3/4 VTs), and that for ablation of VT from the free wall of the left ventricle was 50% (2/4 VTs). However, no significant differences were observed.

(6) Complications

There was no rise in the serum creatine kinase level after ablation; 88.8 ± 66.2 IU/ml before vs 89.9 ± 69.0 IU/ml after. In one patient, complete right bundle branch block was produced by ablation to a site close to the His bundle area. Nonsustained VT that showed a different QRS morphology from that of clinical VT was induced during ablation in 2 cases, but never recurred. Three other patients had a tolerable sensation of heat in the chest during the application of RF current, but this disappeared quickly after the current was discontinued.

Fig. 5. Electrophysiologic findings.

This case was a 64-year-old man with symptomatic incessant non-sustained VT (case 9). During non-sustained VTs, endocardial mapping was performed, and the area that showed the earliest ventricular activation was determined within 1.0 cm $\times$ 1.0 cm. The VT morphology and the time interval from the local electrogram to the onset of the QRS activation was different in each complex. Nonsustained VT disappeared after ablations within this area, and 4 other lesions were produced near the site in a linear form. No recurrence of VT was observed after this procedure.

Abbreviations are the same as in Figs. 1 and 3.

(7) Follow-up

During the follow-up periods of from 2 to 13 months (mean 8.4 ± 3.7 months), VT did not recur in any of the successful cases.

DISCUSSION

The size of the lesion made by RF current is smaller than that made by direct current shock^{6,7} and the lesion produced by the RF current is affected by the delivered energy²² contact between the electrode and target myocardium²³ the size of the electrode²⁴ and the characteristics of the target myocardium²⁵ The maximal volume of the lesion produced by appropriate RF energy at 20–50 Watts is about 350 mm³ and is limited to 5 mm in depth²⁶ In contrast, direct current shock at 250 joules can produce a lesion

of about 3.0 cm³.²⁷ This may be one of the reasons why ablation by RF current is safer.

Efficacy of RF Ablation

RF catheter ablation was once considered to be less effective for ablation of VT: some of the origins of VT are considered to be located in deep layers of myocardium²⁸ and myocardium at the ablation site is replaced by fibrosis, calcification, or fatty infiltration.^{29,30}

Recently, the number of cases in which VT has been successfully ablated with RF current has grown,^{9–12} most often in VTs originating from the right ventricle, or in VTs without underlying heart diseases. More recently, Morady et al¹⁹ reported that this current therapy was also effective for treating VTs associated with old myocardial infarct-

TABLE II ELECTROPHYSIOLOGIC FINDINGS

	morphology	ablation site	LEG	Watt×numbers	effect	follow-up
①	LBBB+NAD	EAS (−30 ms)	140* ms	40 W×2, 50 W×1	Failure	/
②	LBBB+NAD	EAS (−40 ms)	70 ms	30 W×4	Success	11 months
③	RBBB+NAD	EAS (−30 ms)	170* ms	30 W×6	Success	13 months
	LBBB+NAD	EAS (−25 ms)	190* ms	30 W×2	Success	
④	LBBB+LAD	EAS (−30 ms)	90 ms	30 W×2	Success	12 months
	LBBB+LAD	EAS (−50 ms)	110* ms	30 W×3	Success	
⑤	LBBB+NAD	EAS (−30 ms)	80 ms	30 W×5	Success	10 months
⑥	LBBB+NAD	EAS (−50 ms)	150* ms	30 W×5	Success	10 months
⑦	LBBB+LAD	EAS (−30 ms)	60 ms	40 W×3	Success	11 months
⑧	LBBB+LAD	EAS (−50 ms)	60 ms	40 W×16, 50 W×2	Success	7 months
⑨	LBBB+LAD	EAS (−35 ms)	40 ms	30 W×3, 40 W×2	Success	6 months
⑩	LBBB+LAD	EAS (−40 ms)	80 ms	30 W×5, 40 W×11	Success	5 months
⑪	RBBB+LAD	EAS (−50 ms)	60 ms	30 W×3, 40 W×5	Success	7 months
⑫	RBBB+RAD	Slow Pathway→EAS (−25 ms)	120* ms	40 W×3, 50 W×2	Failure	/
⑬	LBBB+RAD	EAS (−50 ms)→Slow Pathway	200* ms	40 W×21, 50 W×5	Success	3 months
⑭	RBBB+LAD	Slow Pathway	120* ms	40 W×4, 50 W×4	Success	2 months
	RBBB+RAD	EAS (−30 ms)	140* ms	40 W×7, 50 W×5	Failure	

An asterisk (*) indicates a site with fragmentation, EAS=earliest activation site (endocardial activation time relative to earliest onset of QRS complex is shown in parentheses), LEG=the duration of the local electrogram at the ablation site, W=Watt, Other abbreviations are the same as in Table I.

tion. In the present study, the success rate tended to be higher in VTs that were not associated with underlying heart diseases (10/11 VTs), and in VTs that originated in the free wall of the right ventricle (9/9 VTs). However, the population in this study was too small to provide clear information regarding the clinical and electrophysiologic characteristics for successful RF ablation.

Ablation of VT origins that showed normal local electrograms was highly successful. This may have been due to the fact that the site of ablation consisted of normal myocardium. Conversely, when the VT origin showed fragmentation, the site may have involved electrophysiologically abnormal myocardium²⁰ and RF current might be less effective in such diseased myocardium²⁵

Feasible Ablation Site

In direct current catheter ablation, the shock is applied to the site of the earliest ventricular activation, or to the area of slow conduction, with comparable success rates^{14,17,19}. In radiofrequency ablation, Klein et al⁸ and Gonska et al⁹ used the site of earliest ventricular activation as the target site of ablation with good results. Kuck et al³¹ reported a successful case in which VT

was terminated by ablation of the area of slow conduction.

In the present study, the site of earliest ventricular activation was used as the ablation site in 16 of 17 VTs and satisfactory results were obtained in 12/16 VTs. The interval from the local electrogram to the QRS complex varied from case to case, but the interval in the successful sites was not longer than that in the unsuccessful sites (Table II).

Delivery of RF current to the slow conduction area in the reentry circuit was successful in 2 of 3 VTs. In these patients, the termination of VT by RF current did not necessarily produce non-inducibility after ablation. A relatively extended arrhythmogenic area, as compared to the RF lesion, or alternative pathways³² within the slow conduction area might be related to this result.

In this study, RF current was delivered during VT, and VT was terminated within a few seconds. This early termination may be helpful in determining whether the site is a critical for VT. We believe that RF application during VT is useful for determining the critical site of VT, and for avoiding lesions at sites that are inappropriate.

RF Ablation for Nonsustained VT

Application of RF ablation for nonsustained VT is controversial, because such tachycardia has been considered benign; especially when they originated from the outflow tract of the right ventricle or were in hearts without underlying heart diseases.³³ However, in some patients, incessant nonsustained VT has resulted in syncope or fainting³⁴ and appropriate therapy has been required. Two patients with symptomatic nonsustained VT were included in this study and pharmacological therapy with 2–5 antiarrhythmic drugs failed to prevent spontaneous attacks. These nonsustained VTs disappeared after application of RF ablation. However, more studies and long-term follow-ups will be needed to establish the usefulness of radiofrequency catheter ablation for treating nonsustained VT.

Complications

Although RF current catheter ablation is considered to be safer than direct current ablation, a few complications have been reported!^{–4,6,10} The only major complication observed in this study was complete right bundle branch block in 1 patient.

Limitations

The present study had certain limitations. For example, the follow-up period was short, and long-term results have not yet been evaluated. Few patients were included in the study and most had VTs of right ventricular origin, and did not have apparent underlying heart diseases. The study of a large number of patients, and the success rate in VTs originating from the left ventricle, or in VTs associated with diseased myocardium, are required to establish the efficacy and safety of RF ablation for the treatment of VTs.

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