Radiofrequency catheter ablation in recurrent ventricular tachycardia

B. D. Gonska, S. Brune, K. P. Bethge and H. Kreuzer

Department of Cardiology, University of Goettingen, Germany

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Catheter ablation by radiofrequency energy was carried out in 10 patients with one type of recurrent monomorphic sustained ventricular tachycardia resistant to medical antiarrhythmic management. Electrophysiological studies before ablation included activation and pace-mapping. In all patients, the origin of the tachycardia was localized in the left ventricle: in the septum in six, at the posterolateral wall in three and anterobasal in one. The earliest onset of endocardial activation preceding the QRS complex during ventricular tachycardia ranged between — 45 and — 90 ms. Transcatheter ablation was performed with a bipolar or quadripolar catheter using a radiofrequency generator (HAT 100, Osypka). No complications occurred during the ablation procedure. Thereafter, in all patients, the clinical tachycardia was no longer inducible by programmed stimulation. During a follow-up period of 22 to 32 months including eight patients, the tachycardia recurred in two; one of these patients subsequently died suddenly. A third patient had one episode of a new type of sustained ventricular tachycardia some hours after catheter ablation. In the remaining patients, there was no recurrence of symptomatic tachycardia under maintenance of the antiarrhythmic management which, prior to ablation had been ineffective.

Thus, our preliminary results suggest that radiofrequency catheter ablation might be beneficial for these high risk patients.

Introduction

Catheter ablation is an accepted technique in the non-pharmacological treatment of recurrent supraventricular and ventricular tachycardia. To date, the largest experience is in the ablation or fulguration of the atrioventricular junction[1-6], but ablation procedures in the atrium[7,8] and of accessory pathways[9-11] have been described as well. In 1983, Hartzler[12] was the first to report attempted catheter ablation of ventricular tachycardia. In the meantime, a number of reports have been published[13-24].

Different ablation techniques have been applied. Most often, direct current via a cardioverter/defibrillator system is used. Radiofrequency energy has been shown to be safe and effective in ablation of the atrioventricular junction[25-29]. Recent studies reported successful radiofrequency ablation in patients with accessory pathways[30,31]. However, there is little experience with this technique in the treatment of ventricular tachycardia[32]. The application of laser technology to ablate arrhythmogenic myocardiun is still in the experimental stage and has been confined to open heart surgery[33-35].

The aim of this study was to prove the feasibility and long-term effectiveness of radiofrequency catheter ablation in patients with recurrent, sustained monomorphic ventricular tachycardia resistant to antiarrhythmic drugs.

Methods

PATIENTS

Between August 1987 and July 1988, catheter ablation of ventricular tachycardia was performed in 10 patients, seven men and three women with a mean age of 58 (range 43 to 67) years. The clinical characteristics are listed in Table 1. The underlying heart disease was confirmed in all patients by cardiac catheterization and coronary angiography. Nine had coronary artery disease, eight of them with a history of myocardial infarction (seven anterior, one inferior) 3 months to 15 years prior to catheter ablation. Six of these patients had an aneurysm of the anterior wall, two of them (patients 1 and 9) had already undergone aneurysmectomy, and ventricular tachycardia had occurred later. The underlying heart disease in the remaining patient was idiopathic dilated cardiomyopathy. The left ventricular ejection fraction ranged from 20 to 45% (mean 32%).

Recurrent sustained ventricular tachycardia had been present in all patients for a period ranging from one month to 8 years. All of them had undergone external cardioversion to terminate ventricular tachycardia, one to more than 15 times with up to 40 direct current countershocks. In none of the patients was the tachycardia due to acute myocardial infarction. A 12-lead electrocardiogram of the tachycardia was available, in all cases showing a monomorphic pattern. No more than one type of tachycardia had been registered clinically.

In all patients, the arrhythmia had been resistant to antiarrhythmic drugs as confirmed by serial electrophysiological testing (10 patients) and clinical recurrence (eight patients). Four to six drugs had been tested including mexiletine (median 600 mg), tocainide (median 1200 mg),
propafenone (median 750 mg), flecainide (median 300 mg), disopyramide (median 400 mg), amiodarone (median 400 mg), amiodarone plus mexiletine and propafenone plus sotalol. The last antiarrhythmic medication before catheter ablation was amiodarone for three patients, amiodarone plus mexiletine for three, propafenone plus sotalol for two, propafenone for one and mexiletine for one patient.

**Electrophysiological Studies**

**Programmed Ventricular Stimulation**

Programmed ventricular stimulation was performed in the right ventricular apex. The stimulation protocol utilized before and after catheter ablation included the application of one to three premature extrastimuli during sinus rhythm and paced cycle lengths ($S_1 S_2 = 600, 500, 428, 375$ ms). The endpoint of testing was the induction of stable sustained clinical ventricular tachycardia. The initial programmed stimulation was performed in the absence of antiarrhythmic drugs, except for four patients who were already on long-term amiodarone treatment on admission. In the course of serial testing, stimulation was repeated under antiarrhythmic therapy which was considered to be ineffective if the clinical tachycardia was still inducible and/or recurred spontaneously. Differences in heart rate of up to 20 beats $\text{min}^{-1}$ were accepted if morphology, axis and R-wave progression were identical.

**Catheter Mapping**

All patients had been informed in detail about the mapping and ablation procedure and had given their written consent. Two quadripolar catheters (6F USCI) were inserted percutaneously via the femoral vein and advanced to the right ventricular apex and outflow tract. A bipolar or quadripolar catheter (7F USCI or 6F Mansfield) was introduced via the brachial or femoral artery to the left ventricular cavity. Endocardial electrograms were recorded from the right ventricular apex and outflow tract and from the left ventricle on a modified seven channel ink-jet recorder (Mingograf 7, Siemens Elema, Sweden) at filter settings of 50 to 500 Hz.

The mapping procedure to detect the origin of ventricular tachycardia included activation and pace-mapping. The mapping scheme used has recently been described. Endocardial electrograms from seven to 12 different ventricular sites were recorded per patient. Activation mapping was carried out during induced or spontaneous ventricular tachycardia in order to record the earliest endocardial activation preceding the QRS complex of the surface electrogram (Fig. 1). This site was assumed to belong to the area of origin of the tachycardia especially if stimulated complexes at a rate close to that of the tachycardia showed little or no changes in axis and morphology as compared to the 12-lead electrogram of the clinical tachycardia (Figs 2 and 3).

**Ablation Procedure**

Catheter ablation was performed with a conventional catheter, used for the mapping procedure. A stable catheter position was confirmed by biplane fluoroscopy. The distal electrode was attached to a radiofrequency generator (HAT 100, Osypka, Grenzach, Germany) and a passive plate electrode was placed under the left shoulder of the patient.

The generator delivered a 500 KHz continuous unmodulated sinusoidal waveform in the unipolar output mode. In this mode, a large current density is present at the small surface area of the tip electrode. The output power had 10 different settings between 2.5 and 50 W at an assumed tissue resistance of about $500 \Omega$. The duration of each radiofrequency application was controlled automatically as a function of the impedance changes of the myocardial tissue.

The ablation procedure was performed during ventricular tachycardia (Fig. 4). The energy delivered was increased stepwise. When, after application of radiofrequency energy, the tachycardia stopped, programmed ventricular stimulation was carried out using the stimulation protocol described above. If the tachycardia was still inducible, the ablation procedure was continued. In two patients who underwent catheter ablation during sinus rhythm (patient 4 and second ablation in patient 1), programmed
Radiofrequency catheter ablation in ventricular tachycardia

Figure 1  Activation mapping in a 57-year-old patient with coronary artery disease. During ventricular tachycardia, the earliest onset of activation preceding the QRS complex (-85 ms) was recorded in the septoapical area. (LV = left ventricle, RVOT = right ventricular outflow tract, RVA = right ventricular apex).

Figure 2  Clinical tachycardia in this patient with a cycle length of 413 ms, superior axis and right bundle branch block configuration.

stimulation was carried out after every second application of radiofrequency energy.

Arterial pressure and cardiac rhythm were monitored continuously. No general anaesthesia was applied. Sedation was not required. During catheter ablation and for the following 48 h, all patients were fully anticoagulated by intravenous heparin. Serum creatine kinase and the kinase MB fraction were measured at 6-h intervals during the first day after catheter ablation. Two-dimensional echocardiography was carried out before and immediately after ablation and 5 to 7 days later.

FOLLOW-UP

After catheter ablation, the patients were monitored in the coronary care unit for 24 h. Five to 7 days later, programmed stimulation and 24 h ambulatory monitoring...
were carried out again. After their release, the patients were seen at individual intervals not exceeding 2 months in our outpatient clinic and underwent 24 h electrocardiography. Finally, they were contacted by telephone in May 1990.

Results

Electrophysiological Studies

In all 10 patients, only one type of monomorphic sustained ventricular tachycardia was documented clinically and reproduced by programmed ventricular stimulation from the right ventricular apex. Before ablation, the tachycardia was induced with a basic cycle length of $S_1S_2 = 600$ ms and one extrastimulus in four patients; with two extrastimuli in four, at a basic cycle length of $S_1S_2 = 375$ ms, and one extrastimulus in one patient and with two extrastimuli in another. In eight patients, the tachycardia had a right bundle branch block pattern and in two a left bundle branch block morphology. The mean cycle length of the tachycardia was 366 ms, ranging from 250 to 500 ms. A superior axis of the QRS complexes was seen in seven patients and three had a normal axis (Table 2).

The earliest endocardial activation preceding the QRS complex of the surface electrogram during ventricular tachycardia was detected between $-45$ and $-90$ ms (mean $-70$ ms). Pace-mapping showed little or no changes in axis or morphology as compared to the QRS complexes in a 12-lead electrogram in eight of the 10 patients.

Endocardial catheter mapping revealed a left ventricular origin of the arrhythmia in all patients. In six of them, it was found to be in the septum (four midseptum and two septoapical), in another two in the posterolateral-medial area and in the remaining two patients in the posterolateral-apical and anterobasal areas, respectively.

Catheter Ablation and Follow-up

A total of 11 ablation procedures were carried out in the 10 patients. Catheter ablation was repeated on the following day in only one patient, patient 1. On average, six (three to 12) applications of radiofrequency energy per patient were carried out in the region assumed to be the origin of the arrhythmia. The duration of one ablation ranged from 1 to 90 s (median 8 s). The maximal output power varied between 25 and 50 W (Table 2).

During catheter ablation, no complications occurred. No patient complained of discomfort. The procedure was performed without haemodynamic deterioration or thromboembolic events. Thrombi, however, were apparent at the tip of the ablation catheter when it was withdrawn. At two-dimensional echocardiography, no intracardiac thrombi were visible. Serum creatine kinase remained in the normal range.

The results of electrophysiological testing immediately after catheter ablation and 5 to 7 days later, as well as the later outcome of the patients, are shown in Table 3.

In patient 1, programmed stimulation after catheter ablation induced ventricular fibrillation necessitating direct current countershock for termination. Sixteen hours later, one episode of monomorphic non-sustained tachycardia with the same cycle length as before, and 20 consecutive QRS complexes, was registered at the coronary care unit; it was reproducible by means of programmed stimulation. Catheter ablation was repeated in the same area. Thereafter the response to stimulation was negative. During the following 4 weeks, the patient was well, showing only single ventricular extrasystoles during repeated 24 h electrograms. Then, she suffered from syncope.

Although she remained non-inducible at programmed stimulation, a recurrence of the tachycardia was suspected. Flecainide (200 mg) was added to amiodarone. Two weeks later she died from sudden cardiac death due to ventricular fibrillation, presumably following a recurrence of the tachycardia. Post-mortem histological examinations revealed no specific morphological changes in the ablated area which had bordered the zone of the aneurysm. Small fibrotic areas found in this region did not differ from other fibrotic areas found in the myocardium caused by chronic coronary artery disease.

The next patient (Case 2) also had inducible ventricular fibrillation immediately after catheter ablation, but only a low number of monomorphic single ventricular extrasystoles clinically. Five days later, he underwent coronary artery bypass grafting and aneurysmectomy including endocardial incision in the ablated area, although intraoperatively; ventricular tachycardia could not be induced by programmed stimulation. The haemodynamic indications for surgery were already present at the time of catheter ablation; the indication for ablation was incessant non-sustained ventricular tachycardia. Now, after a 32-month follow-up, he is well without antiarrhythmic drugs.
Radiofrequency catheter ablation in ventricular tachycardia

Table 2. Characteristics of ventricular tachycardia, results of endocardial mapping and catheter ablation

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Characteristics of VT</th>
<th>Catheter mapping</th>
<th>Catheter ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Morphology</td>
<td>QRS axis</td>
<td>Cycle length</td>
</tr>
<tr>
<td>1</td>
<td>LBBB</td>
<td>−87°</td>
<td>250 ms</td>
</tr>
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<td>2</td>
<td>RBBB</td>
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<td>400 ms</td>
</tr>
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<td>3</td>
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<td>350 ms</td>
</tr>
<tr>
<td>4</td>
<td>RBBB</td>
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<td>273 ms</td>
</tr>
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<tr>
<td>10</td>
<td>RBBB</td>
<td>+67°</td>
<td>332 ms</td>
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</tbody>
</table>

LBBB = left bundle branch block, RBBB = right bundle branch block, other abbreviations see Table 1.

Table 3. Electrophysiological results after catheter ablation and follow-up

<table>
<thead>
<tr>
<th>Patient No</th>
<th>After ablation</th>
<th>5–7 days later</th>
<th>Follow-up</th>
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<tr>
<td></td>
<td>PVS 24 h-ECG</td>
<td>PVS 24 h-ECG (Lown)</td>
<td>Period</td>
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<tr>
<td>1</td>
<td>VF mnsVT (20)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>VF VES</td>
<td>0</td>
<td>I</td>
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<tr>
<td>3</td>
<td>0 pnsVT (3)</td>
<td>mnsVT (10)</td>
<td>I</td>
</tr>
<tr>
<td>4</td>
<td>0 VES</td>
<td>n.d.</td>
<td>I</td>
</tr>
<tr>
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<td>VES</td>
<td>pnsVT (9)</td>
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<td>6</td>
<td>pnsVT (12)</td>
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<td>0</td>
</tr>
<tr>
<td>7</td>
<td>0 pnsVT (5)</td>
<td>0</td>
<td>IVb (8)</td>
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<tr>
<td>8</td>
<td>0 VP</td>
<td>0</td>
<td>IVa</td>
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<tr>
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<td>msVT</td>
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<tr>
<td>10</td>
<td>0 VES</td>
<td>0</td>
<td>sVT</td>
</tr>
</tbody>
</table>

PVS = programmed ventricular stimulation, p = polymorphic, m = monomorphic, ns = non-sustained, VES = ventricular extrasystoles, VP = ventricular pairs, n.d. = not done, SCD = sudden cardiac death, CABG = coronary artery bypass grafting, AE + EI = aneurysmectomy and endocardial incision, PF = pump failure, Am = amiodarone, Mex = mexiletine, Fle = flecainide, Prop = propafenone, Sot = sotalol; other abbreviations see Table 1.

In patient 3, the response to programmed stimulation was negative immediately after catheter ablation, but 6 days later, a monomorphic non-sustained ventricular tachycardia could be induced which was different from the clinical and inducible one before ablation, with respect to axis and cycle length. Spontaneous arrhythmias included three episodes of non-sustained tachycardia with three consecutive extrasystoles during the first day after ablation and one such episode at 24 h electrocardiography one week later. In the meantime, the patient has had no recurrence of the tachycardia and only single monomorphic ventricular extrasystoles clinically; the antiarrhythmic management has not been altered.

Patient 4 had no inducible tachycardia after catheter ablation and only single ventricular extrasystoles clinically. One week later he underwent coronary artery bypass grafting because of severe three-vessel disease. This patient already had a global impairment of the left ventricular function with an ejection fraction of 27% and thus was no candidate for antitachycardia surgery; he died a few hours after the operation from pump failure without having had a recurrence of tachycardia. No autopsy could be performed because of relatives refusal.

The next patient (Case 5) had polymorphic non-sustained ventricular tachycardia with 15 consecutive beats induced immediately following catheter ablation and with nine beats one week later. However, there were only a small number of monomorphic single ventricular extrasystoles on the monitor and during 24 h electrograms.

In patient 6, programmed stimulation after catheter ablation induced polymorphic non-sustained ventricular tachycardia with a cycle length of 300 ms which was shorter than before ablation. Four hours later, he had one spontaneous episode of sustained tachycardia at this
cycle length. During control stimulation 5 days later, the response was negative. 24 h electrocardiographic recording showed 16 single extrasystoles and one ventricular pair. The medication was not altered and during follow-up the patient only had polymorphic single ventricular extrasystoles.

Patient 7 had a negative response to stimulation immediately after catheter ablation as well as one week later. Clinically, two episodes of non-sustained ventricular tachycardia with five consecutive extrasystoles were recorded during the first day following the ablation procedure. 24 h electrocardiographic recording one week later showed one episode of polymorphic non-sustained tachycardia with eight consecutive ventricular extrasystoles. In the meantime, the patient has been feeling well with only a few polymorphic single extrasystoles.

In patient 8, programmed stimulation did not induce ventricular tachycardia after catheter ablation. 24 h electrocardiographic recording one week later showed 11 ventricular pairs. During the follow-up, one to ten ventricular pairs were recorded during 24 h monitoring.

Patient 9 had inducible non-clinical monomorphic sustained ventricular tachycardia with a shorter cycle length immediately after catheter ablation. Six days later, programmed stimulation induced polymorphic non-sustained tachycardia with seven consecutive beats. Clinically, the patient had complete AV block 6 h after the ablation procedure, necessitating external pacing for 5 h. Thereafter, normal sinus rhythm returned and persisted for 6 months. Since that time, however, intermittent first-degree AV block has been recorded. During the first week after catheter ablation, only single ventricular extrasystoles were recorded; they disappeared during the later follow-up.

In patient 10, programmed stimulation neither induced ventricular tachycardia immediately after catheter ablation nor at control 5 days later. 24 h electrocardiographic recording during the first day showed only single monomorphic extrasystoles. Three hours after the second stimulation, however, she had a recurrence of the tachycardia requiring external cardioversion for termination. The medication was changed to amiodarone. Thereafter, no more than single ventricular extrasystoles occurred during the 22-month follow-up.

Discussion

Catheter Ablation

Catheter ablation is considered to be a valuable non-pharmacological therapeutic tool for the treatment of recurrent ventricular tachycardia. The results suggest it is a promising technique. Belhassen et al. described five out of eight patients without recurrence of ventricular tachycardia during a follow-up period of 7 to 17 months. Huang et al. reported their results in five patients with a single type of monomorphic tachycardia due to coronary artery disease. Four of them were released without drug medication. However, two of these suffered a recurrence of the tachycardia within 7 months after the ablation procedure and were then treated with drugs, effectively.

Borggreve et al. recently described 24 patients, of whom 17 had no recurrence of ventricular tachycardia after one to three ablation procedures. The greatest number of ablations at one centre has been reported by Fontaine et al. who applied catheter ablation in 38 patients with single and multiple morphologies of tachycardia. The patients in their study underwent up to four ablation procedures with an increasing final rate of success. In 56%, the first ablation session had been effective. In contrast, Touboul et al. described only 36% of patients without recurrence of tachycardia in a study population of 30 patients during a mean follow-up of 14 months and Morady et al. recently considered 45% out of 31 patients to have been successfully treated.

The overall rate of successfully treated patients as listed in the Percutaneous Cardiac Mapping and Ablation Registry is about 70%. One third of these patients did not require further antiarrhythmic medication, while the others had no recurrence of tachycardia under a drug regimen which had been ineffective before ablation. In the remaining patients, the technique failed to suppress the clinical tachycardia. The best results have been reported in patients with bundle branch reentrant ventricular tachycardia.

In all of the studies mentioned above, direct current energy was used for catheter ablation. Radiofrequency energy for ablation of cardiac tissue, however, is a rather new method, but has been shown to be as effective as direct current in the ablation of atrioventricular junction. Experience with ventricular tachycardia has been limited to a small number of patients. Borggreve et al. reported successful radiofrequency ablation in three out of five patients, which is comparable to our results and to those achieved with direct current.

The main problem of catheter ablation is the exact localization of the arrhythmogenic focus i.e. the reentry circuit of the tachycardia. Usually, activation and pace-mapping are carried out for this purpose. In our patients, the earliest endocardial activation preceding the QRS complex of the surface electrogram during ventricular tachycardia was noted from −45 to −90 ms and pace-mapping revealed little or no differences in eight out of 10 patients. In the present study group, however, the results of activation and pace-mapping were not clearly predictive of a favourable outcome. This was in accordance with the data published by Borggreve et al. and Morady et al.

These findings indicate that the methods conventionally applied to determine an adequate ablation site are not sufficient and require further refinement. A recent retrospective study revealed that a stimulus to QRS latency > 100 ms during ventricular tachycardia pace-mapping, in addition to early endocardial activation, might be predictive of successful catheter ablation. Furthermore, Kuck et al. have demonstrated that in a small number of slow ventricular tachycardias discrete presystolic potentials may be visible for the duration of the diastole, possibly indicating a more selective site of electrical energy application.

It seems reasonable to assume that the success rate is related to the number of foci to be ablated. We therefore...
have to emphasize that the patients in this report represent a highly selective group. Like Huang et al.\textsuperscript{39} and Morady et al.\textsuperscript{24}, we only performed ablation in patients with a single type of monomorphic tachycardia, nine out of 10 with coronary artery disease. On the other hand, the number of ablation procedures performed in one patient has to be taken into account. In our 10 patients, 11 procedures were carried out.

During programmed stimulation, the clinical tachycardia was no longer inducible in any of the patients after the ablation procedure, which has been considered a sign of good prognosis\textsuperscript{10}. However, two of our patients with a negative response to programmed stimulation immediately following catheter ablation as well as 5 to 7 days later, had a recurrence of their tachycardia, which was documented in one (patient 10) and suspected in the other (patient 1). The clinical significance of inducible primary fibrillation after the ablation procedure (patient 1 after first ablation and patient 2) could not clearly be established, whereas the inducibility of polymorphic non-sustained tachycardia and even of non-clinical sustained tachycardia does not seem to be highly predictive with respect to the clinical efficacy of the method.

**FOLLOW-UP**

Our follow-up data are limited to eight patients. One (Case 4) died of causes unrelated to catheter ablation, in another (patient 2), 5 days after catheter ablation, endocardial incision in the ablated area was performed in the course of aneurysmectomy; thus we cannot prove that the good long-term success was only due to the ablation procedure.

Of the remaining eight patients, one died 6 weeks after catheter ablation from sudden cardiac death, presumably following a recurrence of ventricular tachycardia. Another patient (Case 10) had a recurrence of the arrhythmia some hours after control stimulation and was then treated with amiodarone successfully; this drug had not been administered before ablation. Patient 6 had a new type of tachycardia induced immediately after catheter ablation and had one spontaneous episode of this arrhythmia 4 hours later; he was non-inducible one week later and had no recurrence during the subsequent 29 months under maintenance of the antiarrhythmic management that had been ineffective before ablation.

The remaining five patients did not have a recurrence of sustained ventricular tachycardia during a follow-up period ranging between 22 and 32 months. The antiarrhythmic management set up before the ablation procedure was maintained in each case, since 5 to 7 days after catheter ablation, either programmed stimulation induced non-sustained ventricular tachycardia or 24 h electrocardiographic recording documented short runs of ventricular tachycardia or ventricular pairs.

The clinical importance of the antiarrhythmic drug regimen after successful catheter ablation is not quite clear. In larger study groups\textsuperscript{19,23,24}, about half the patients were receiving antiarrhythmics after successful ablation, even if programmed stimulation did not induce ventricular tachycardia.

A recurrence of the arrhythmia most often occurs during the first days and weeks. Borggreve et al.\textsuperscript{23} reported 14 patients with a relapse of ventricular tachycardia after the first ablation session, which occurred within 1-7-3-2 weeks. In our small study group, the latest life-threatening arrhythmia occurred 6 weeks after catheter ablation. In patients who have ventricular tachycardia during long-term follow-up, there is often no information available on whether the tachycardia was of the same type as before or whether it was a new one that had developed in the course of chronic myocardial disease.

Therefore it might seem reasonable to withdraw the antiarrhythmic drugs 4 to 6 weeks after successful catheter ablation, then to repeat programmed stimulation in the drug-free state and only continue medication if sustained ventricular tachycardia is inducible; the proarrhythmic potency of these drugs should indeed be kept in mind.

**SIDE EFFECTS**

A number of disadvantages and complications associated with the use of direct current for ablation such as haemodynamic deterioration, proarrhythmic effects, conduction disturbances, perforation, arterial embolism and even death have been reported\textsuperscript{5,9,19,23,27,42-44}. Whether this can also be found for the application of direct current is currently a matter of discussion. While Davis et al.\textsuperscript{43} and Hauer et al.\textsuperscript{44} reported a good correlation between the energy delivered and the volume of the myocardium desiccated has been established for in vitro application\textsuperscript{26,49}. Whether this can also be found for the application of direct current is currently a matter of discussion. While Davis et al.\textsuperscript{43} and Hauer et al.\textsuperscript{44} reported a good correlation between the energy used and the enlargement of the ablated myocardium — transmural lesions were only seen at 250 Joule — Lerman et al.\textsuperscript{43} published opposite results.

The mechanism of radiofrequency ablation probably accounts for the lower complication rate suggested by a recent comparative study\textsuperscript{39}. Complications during the application of radiofrequency energy were not seen in our patients. In one patient (Case 6), however, a proarrhythmic effect of the technique has to be presumed. In patient 1, such an effect cannot completely be excluded either since immediately after the first ablation session, programmed stimulation induced ventricular fibrillation, and 6 weeks later the patient died suddenly from ventricular fibrillation. On the other hand, a proarrhythmic drug effect also has to be envisaged since this patient had received flecainide in addition to amiodarone in the final 2 weeks; this combination had not been tried before ablation. The transient AV block occurring some hours after catheter ablation in another patient (Case 9) should be considered as a complication of the procedure, but not necessarily caused by the application of radiofrequency energy since the ablation site was septoapical.

One clear advantage of radiofrequency, as compared to direct current, is that no general anaesthesia is required.

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\text{Radiofrequency catheter ablation in ventricular tachycardia} & \quad 1263
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which makes the application easier and the whole procedure more suitable.

The technique of radiofrequency application is currently being refined. In a recently published animal study, Hindricks et al. demonstrated that monitoring of catheter tip temperature improved the prediction of lesion size.

Conclusion

In conclusion, we have reported our preliminary experiences with radiofrequency catheter ablation in 10 patients with a single type of recurrent sustained monomorphic ventricular tachycardia resistant to antiarrhythmic drugs. The results are limited to a small number of patients. Although further studies are required, a selected group of high risk patients might benefit from this method.

References


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