

Radiofrequency ablation of haemodynamically unstable ventricular tachycardia after myocardial infarction

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Abstract

Objective—To determine whether radiofrequency (RF) ablation might have a role in haemodynamically unstable ventricular tachycardia.

Methods—10 patients with a history of ventricular tachycardia producing haemodynamic collapse in whom drug treatment had failed and device therapy was rejected underwent RF ablation of ventricular tachycardia in sinus rhythm. The arrhythmogenic zone was defined on the basis of abnormal systolic movement, the presence of fragmentation (low amplitude, prolonged multiphasic electrograms), and pace mapping. RF lesions were delivered in power mode in linear fashion within the defined arrhythmogenic zone.

Results—Success (no ventricular tachycardia inducible postablation or at retest) was achieved in six patients, possible success (a different ventricular tachycardia inducible at more aggressive stimulation) in three. In one patient, the procedure was abandoned because of poor catheter stability. There were no clinical events during a mean (SD) follow up period of 23 (10) months in any of the nine patients defined as definite or possible successes.

Conclusions—RF ablation for addressing haemodynamically unstable ventricular tachycardia opens the door for the wider use of catheter ablation for treating this arrhythmia.

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Keywords: tachycardia; catheter ablation; sudden death

Radiofrequency (RF) ablation has become the treatment of choice for most supraventricular tachycardias. Focal ventricular tachycardia, particularly in the absence of structural heart disease, is also eminently ablatable.¹ Ventricular tachycardia arising in the context of structural heart disease, particularly old myocardial infarction, presents several problems for ablation, however. Advances in our understanding and mapping of arrhythmia circuits combined with significant improvements in catheter and energy technology have resulted in growing success of RF ablation of ventricular tachycardia in selected groups of patients.^{2–7}

One major hurdle remains. Most series on ablation of ventricular tachycardia involve mapping of the arrhythmia after its induction and hence require the arrhythmia to be well tolerated. Well tolerated ventricular tachycardia, however, rarely affects prognosis and, more importantly, accounts for less than 10% of the total ventricular tachycardia population.^{8–9}

Our study aimed to determine whether RF ablation could be performed in patients with poorly tolerated ventricular tachycardia using a sinus rhythm mapping strategy that did not require prolonged periods of tachycardia.

Methods

Of the 91 patients who underwent RF ablation of ventricular tachycardia between 1992 and December 1997, 10 had haemodynamically unstable ventricular tachycardia at the time of ablation. The clinical characteristics are shown in table 1. The mean (SD) age of the patients was 61 (3) years. All the patients had ventricular tachycardia secondary to ischaemic heart disease (mean ejection fraction 28%). All had

documented clinical episodes of ventricular tachycardia associated with haemodynamic collapse and had previously undergone programmed ventricular stimulation when ventricular tachycardia was induced, which required urgent termination. All had failed to respond to conventional antiarrhythmic treatment, including amiodarone, but had refused to have implanted cardioverter devices.

ELECTROPHYSIOLOGICAL STUDY AND MAPPING STRATEGY

Antiarrhythmic drugs other than amiodarone were discontinued one week before the study. All patients underwent echocardiography to exclude left ventricular thrombus. Programmed ventricular stimulation was performed from the right ventricular apex. Ventricular tachycardia was induced and rapidly terminated by overdrive pacing or cardioversion because of hypotension (blood pressure < 60 mm Hg) or loss of consciousness. After stabilisation and sedation following cardioversion, catheters were advanced to the left ventricle. In four patients, trans-septal puncture was performed to gain antegrade access to the left ventricle. The patient was fully heparinised and an activated clotting time (ACT) > 250 s was ensured by titrating heparin dosage to serial ACT measurements. The position of the catheters was initially guided anatomically by the scar defined at left ventriculography. Using a modified x ray system (Freeman Hospital, Medical Physics Department) that allowed single frames to be acquired at particular points in the cardiac cycle, an R wave gated ventriculogram outline was overlain on R wave gated images of catheter positions. Two extrastimuli

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Table 1 Clinical profile of patients and characteristics of ventricular tachycardia induced

No	Age (years)	Sex	Diagnosis	No of episodes	No of EF	No of extrastimuli	Cycle length (ms)	Termination of VT	Site of VT	Catheter used	Result of repeat VT stimulations	Success definition	Further treatment	Clinical recurrence	Follow up (months)
1	68	M	Inf MI	3	20%	1	360	DC	Inferobasal	8 mm	Polymorphic NSVT at 3 extras	+	—	—	47
2	66	M	Inf MI	4	31%	1	320	ODP	Inferobasal	4 mm	Different VT CL 300 ms at 3 extras	±	Amio	—	30
3	63	F	Inf MI	2	39%	1	460	ODP	Inferobasal	4 mm	No VT at 3 extras	+	—	—	26
4	54	M	Inf MI, CABG	6	40%	2	300	ODP	Inferobasal	4 mm	Different VT morphology CL 215 ms at 4 extras. No VT inducible at 4 extras stim 2	+	—	—	25
5	57	M	Ant MI, CABG	3	30%	1	250	ODP	Apicoseptal	8 mm	No VT at 3 extras	+	—	—	22
6	74	M	Ant MI	1	24%	2	320	ODP	Anterior	4 mm	Different VT CL 270 ms at 3 extras	±	Amio	—	21
7	65	F	Inf MI, CABG	1	28%	3	330	DC	Inferobasal	4 mm	Different VT morphology CL 300 ms at 4 extras stim 1	±	ICD	NSVT (8 beats)	18
8	47	M	Ant MI	6	13%	1	240	DC	Inferobasal	8 mm	VT at 3 extras	—	VT surgery	—	18
9	46	M	Inf MI	3	35%	1	337	DC	Inferobasal	8 mm	No VT at 4 extras	+	—	—	17
10	65	M	Ant MI	3	24%	2	280	DC	Apicoseptal	8 mm	No VT at 4 extras	+	—	—	8

Amio, amiodarone; Ant MI, anterior myocardial infarction; CABG, coronary artery bypass grafting; CL, cycle length; DC, direct current cardioversion; extras, extrastimuli; F, female; Inf MI, inferior myocardial infarction; ICD, implantable cardioverter defibrillator; IHD, ischaemic heart disease; M, male; NSVT, non-sustained ventricular tachycardia; OPD, overdrive pacing; VT, ventricular tachycardia. Success definition: +definite; ± possible; — failure.

at coupling intervals comparable to the tachycardia cycling were delivered from the roving catheters in sinus rhythm. The site which gave the closest match to the clinical ventricular tachycardia was taken as the exit zone for that ventricular tachycardia. Areas of fragmentation (low amplitude, multiphasic, prolonged electrograms) were noted and taken as indicative of scar.¹⁰ The extent of the scar based upon fragmentation and pacing was measured, using interelectrode spacing as a reference. Pacing stimulus to surface QRS onset delay was taken as indicative of slow conduction in that region.

ABLATION PROCEDURE

Radiofrequency current was delivered using an RF Radionics 3D generator (Radionics Inc, Burlington, Massachusetts, USA) in unipolar, power only mode between a distal pole (4–8 mm) and a reference backplate. Six to 20 burns (mean 14) were given at 40 watts for up to 60 seconds. Lesions were applied in a linear fashion from the presumed exit across the zone of abnormal electrograms. Energy application was continued at one site rather than performing drag lesions. In inferior infarction, special attention was given to the submitral isthmus such that the energy delivery was continued up to the mitral valve ring. In anterior infarction, a mesh of linear lesions was attempted in order to produce lines running from the exit zone to an area of “dead” myocardium with no pacing capture and very low amplitude electrograms.

Programmed ventricular stimulation was repeated only at the end of the sequence of RF applications. Inducibility of clinical ventricular tachycardia or any other ventricular tachycardia was noted. Repeat programmed RV stimulation was performed after approximately five days. Patients were anticoagulated for at least three months after the procedure.

OUTCOME DEFINITIONS

- **Success**—no ventricular tachycardia inducible at end of procedure or at retest five days later.

- **Possible success**—non-clinical ventricular tachycardia inducible at least two extrastimuli more than at baseline at end or at repeat testing.
- **Failure**—ventricular tachycardia inducibility not significantly altered.

Results

Patients were followed for a period of 6–47 months (mean follow up 23 months). The induced ventricular tachycardia had to be terminated by overdrive pacing in four patients and by cardioversion in five. Systolic blood pressure fell to less than 60 mm Hg in all patients. Mean ventricular tachycardia cycle was 330 ms (range 240–460). Details of the arrhythmias and ablation procedures are described in table 1.

No ventricular tachycardia was inducible (definite success) either at the end of the ablation procedure or at repeat programmed ventricular stimulation in six patients. There were no clinical events during mean follow up of 23 months in any of these patients. In one patient (patient 7), ventricular tachycardia similar to the preablation arrhythmia could be induced at a more aggressive level of stimulation (four extrastimuli). This patient underwent defibrillator implantation and has remained free of clinical treatment and symptoms during 18 months of follow up. At the end of the ablation procedure, two patients (2 and 6) still had poorly tolerated ventricular tachycardia (but of different morphology to that ablated), inducible by more aggressive stimulation. Both these patients have been maintained on amiodarone and have had no clinical events or ventricular tachycardia recorded on Holter monitoring during follow up of 30 and 21 months, respectively. Patient 8 had ventricular tachycardia as a result of a large anterior infarct. The induced arrhythmia could not be ablated because a stable catheter position in the apicoseptal region could not be obtained. Two RF lesions were delivered in

sinus rhythm but the procedure was abandoned and the patient subsequently underwent successful arrhythmia surgery. Thus of the 10 patients, nine were deemed a definite or possible success. Of the three defined as possible successes, one has received device therapy and two have been continued on amiodarone, without clinical recurrences in any of the three. None of the 10 patients has had further ventricular tachycardia or died.

Discussion

Radiofrequency ablation of ventricular tachycardia is now well established in the management of patients with focal ventricular tachycardia occurring without structural heart disease.^{1 2 11} Although high success rates have been reported in this context and much has been learnt about mapping and ablation, they form a small proportion of the total population of patients with ventricular tachycardia.^{8 9} Ischaemic heart disease is the dominant cause of ventricular tachycardia, and RF ablation in this setting is beset with problems. The traditional approach has been to induce ventricular tachycardia and then map the induced rhythm and perform ablation during the tachycardia, seeking to terminate it.

The conventional approach to mapping arrhythmias has been to identify the earliest ventricular activation, which is assumed to be at, or close, to the "ventricular tachycardia origin". This approach works well in focal ventricular tachycardia or at surgery where a large endocardial resection is performed, but in postinfarction ventricular tachycardia it may only identify the exit into normal myocardium. The contributions of Waldo and Henthorn,¹² Stevenson and colleagues,¹³ Downar and associates,^{14 15} and others to our understanding of arrhythmia circuits and entrainment mapping have been crucial advances in arrhythmic mapping and have enabled us to identify different components—entrance, exit, central common pathway, and so on—within the arrhythmia circuit itself.^{16 17} The linkage of this mapping information to efficacy of ablation has identified vulnerable components critical to the maintenance of sustained ventricular tachycardia.¹⁸ Entrainment mapping is invaluable but is time consuming and may at times be misleading, as areas demonstrating concealed entrainment are not always associated with ablation success. It also requires—as with conventional activation mapping—a sustained, well tolerated arrhythmia.

What then are the options if ventricular tachycardia is poorly tolerated and cardioversion is essential? Some operators are willing to perform repeated DC version on patients under general anaesthesia. One option that is useful in some patients is administration of procainamide or amiodarone to slow the arrhythmia so that it becomes haemodynamically tolerated and can be mapped conventionally. In very occasional patients, haemodynamic support using the intra-aortic balloon pump is adequate to allow mapping of ventricular tachycardia.

Multipoint mapping with high speed analysis of single beats is an attractive option for mapping of poorly tolerated but readily terminable ventricular tachycardia, and systems such as the EnSite non-contact mapping system and contact basket systems are becoming available.

We sought to map and ablate ventricular tachycardia without prolonged episodes of ventricular tachycardia and used a combination of three mapping approaches to localise the ventricular tachycardia "generator", as follows.

- *Anatomical*—Careful assessment was made of the infarct zone from echocardiography and angiography. In four patients, R wave gated fluoroscopy was used to remove heart motion changes, and the left ventricular contours were overlain on a fluoroscopy screen to guide catheter positioning.
- *Fragmentation*—We have previously demonstrated that the presence of abnormal, low amplitude, prolonged electrograms—an abnormality we have termed "fragmentation"—can be used to guide ventricular tachycardia surgery.¹⁰ These signals reflect scarred myocardium and hence potential ventricular tachycardia substrate.¹⁹⁻²¹ RF ablation was avoided in zones with normal electrograms unless at a clear scar border zone.
- *Pace mapping*—The resolution of pace mapping in ventricular tachycardia localisation in focal outflow tract tachycardia is good and, in our institution, is the preferred mapping technique.²² The resolution is not high in patients with ventricular tachycardia after myocardial infarction but may still be of great value.^{16 23} Pacing at the "exit" of the ventricular tachycardia into normal myocardium is usually associated with a normal electrogram in sinus rhythm, an anatomical catheter position at the scar edge, and no delay from pacing. Movement of the catheter into the scar zone demonstrates abnormal electrograms in sinus rhythm and a pacing morphology similar to the ventricular tachycardia morphology, often with delay from the spike to the QRS inscription.

For the purpose of this study, we have assumed that delays in conduction are caused by an infarct related scar and that functional conduction block does not play a major role in determining ventricular tachycardia circuits. Thus the same block to conduction is assumed to be present in ventricular tachycardia both in sinus rhythm and with pacing.⁹

INFARCT SITE

Wilber and others^{24 25} have shown that in inferior infarction there is often a critical submitral isthmus of conduction in patients with ventricular tachycardia. In our series, five of the patients had inferior infarction and in each of these the submitral isthmus was targeted successfully. The anatomical basis for the critical isthmus is not clear, but may relate to preserved blood supply and anisotropy. This feature alone makes the approach to inferior ventricular tachycardia foci much easier. We sought to ensure that ablation lesions in

inferior scars were continuous and extended up to the mitral valve ring. In the initial three patients, a mesh of lesions was attempted. Although this zone is important in some inferior wall scars, it is not critical in every patient, and in our larger series of patients we have found the region of the papillary muscles to be important in several of them.

The success rate for RF ablation of ventricular tachycardia following anterior infarction is likely to be lower than in inferior infarction. Although the most common sites are anteroapical and involve the septum, the zone of myocardium is much larger. We attempted to draw lines of burns using failure to capture as an indicator of an anatomical barrier. We have an active surgical programme and patients with large anteroapical aneurysms and ventricular tachycardia are often treated surgically when aneurysmectomy and revascularisation can be combined with the antiarrhythmic procedure. It is important, however, that many surgical series report greater difficulty in performing endocardial resection in inferior wall scars, and these patients may be the group most suited to RF ablation.

CATHETER TIP SIZE

Because of the desire to produce a complete lesion, particularly in inferior scars where epicardial components of ventricular tachycardia circuits may be more common,²⁶ we used an 8 mm tip catheter whenever it was available. Whether this contributed significantly to the results is unclear but it is likely to have been helpful. Unfortunately, the spatial resolution of information from such catheters is reduced but we feel that this disadvantage is offset by the production of a larger lesion,²⁷ particularly adopting our ablation strategy which assumes that most patients have several potential ventricular tachycardias and that single targeted lesions are unlikely to be successful. Production of larger, deeper lesions is more likely to ablate other ventricular tachycardias that may share components of the circuit.

DEFINITION OF SUCCESS AND OUTCOME

We used an aggressive stimulation at the end of the ablation procedure and this was repeated after several days. Successful ablation was defined as no ventricular tachycardia inducible at two more extrastimuli than at baseline, both immediately and one week after the ablation. The uncertain outcome that we defined as "possible success" was associated with induction of a different ventricular tachycardia at more aggressive stimulation and was also followed by a good clinical outcome in this small series. If, however, ablation is used as an adjunct to device therapy, then the criteria for success can be relaxed because the aim may merely be reduction in the number of shocks.²⁸ We sought "arrhythmia cure" and hence we felt it was important to try to avoid a false negative electrophysiological study reflecting normal variability in induction or temporary changes in inducibility of induced by the ablation.

All the patients in this group had clinical or personal reasons for not considering device

therapy in the first instance. Important questions remain concerning the role of RF ablation in patients with postinfarction ventricular tachycardia. What level of risk will be acceptable when considering ventricular tachycardia ablation? Will it only be relevant in patients who have already received a device for rescue? If a device is to be considered in every patient, then the pursuit of complete ventricular tachycardia ablation may be hard to justify because of the extra procedure length and uncertain risk of recurrence. The additional cost of a complicated and potentially risky ablation procedure will then be reserved for patients with very frequent shocks. However, the implication of device therapy for patients and for funding agencies are considerable, and RF ablation may be a better form of palliation in many patients whose outcome is determined predominantly by their left ventricular function rather than their arrhythmia.

CONCLUSIONS

We believe that the traditional maxim "if ventricular tachycardia is not tolerated, it cannot be mapped" should be abandoned. Mapping and catheter ablation of patients with haemodynamically unstable ventricular tachycardia is feasible and relies on a combination of features including anatomical scar site, sinus rhythm electrogram morphology, and pace mapping. Although an implanted cardioverter defibrillator is appropriate in most patients with poorly tolerated ventricular tachycardia after myocardial infarction, we believe that RF ablation should be considered in some patients, particularly if there are frequent shocks or if device implantation is contraindicated.

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IMAGES IN CARDIOLOGY

Recurrent myxoma of the right ventricle

A 34 year old man had three episodes of pleuritic chest pain over an 18 month period. Ventilation/perfusion scanning confirmed pulmonary emboli and he was anticoagulated. In February 1999 he presented with pleuritic chest pain again. A transthoracic echocardiogram (TTE) revealed a large, well circumscribed, echogenic mass in the right ventricle (below left and middle). At operation, the mass was seen to be densely adherent to the wall of the right ventricle but was resected. Subsequent histology confirmed the tumour to be a cardiac myxoma.

One year later the patient was readmitted with pleuritic chest pain. He had not continued with anticoagulation. Repeat TTE (below right) revealed a bright, pedunculated lesion identified on the septal wall of the right ventricle.

The mass was thought to represent a recurrence of the previous myxoma. Rather than reoperate to remove the mass, the patient was anticoagulated again with warfarin.

He is to be monitored closely with regular echocardiography.

Right ventricular myxoma is a rare cardiac tumour. Limited data exists on recurrence of myxomata at any site. Regrowth following incomplete excision seems the likely mechanism for recurrence in this case. Recurrence of right ventricular myxoma has been reported once before, although it has occurred following left and right atrial myxoma.

This rare case illustrates the difficulties associated with the management of ventricular tumours. Recurrence is likely to be high unless extensive endocardial resection is performed. Secondly, the case re-emphasises the need to investigate the source of pulmonary emboli, particularly in young patients with no known risk factors.

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