Radiofrequency ablation of haemodynamically unstable ventricular tachycardia after myocardial infarction

S Furniss, R Anil-Kumar, J P Bourke, R Behulova, E Simeonidou

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. (Heart 2000;84:648–652)

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. 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, however, rarely a

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. Programme 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
Radiofrequency ablation of haemodynamically unstable VT

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

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

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 tachycardias; ODP, overdrive pacing; VT, ventricular tachycardia. Success definition: + definite; ± possible; – failure.

Possible success—non-clinical ventricular tachycardia inducible at least two extrastimuli more than at baseline 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.

At the end of the ablation procedure, two patients (2 and 6) still had poorly tolerated 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. 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. 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, Stevenson and colleagues, 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. The linkage of this mapping information to efficacy of ablation has identified vulnerable components critical to the maintenance of sustained ventricular tachycardia. 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. These signals reflect scarred myocardium and hence potential ventricular tachycardia substrate. RF ablation was avoided in territories 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. The resolution is not high in patients with ventricular tachycardia after myocardial infarction but may still be of great value. 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 pacemapping 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.

**INFARCT SITE**

Wilber and others 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
Radiofrequency ablation of haemodynamically unstable VT

651

... 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 anter-...
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.

OLIVER R SEGAL
NICHOLAS M ROBINSON
ADAM D TIMMIS
Radiofrequency ablation of haemodynamically unstable ventricular tachycardia after myocardial infarction
S Furniss, R Anil-Kumar, J P Bourke, R Behulova and E Simeonidou

Heart 2000 84: 648-652
doi: 10.1136/heart.84.6.648

Updated information and services can be found at:
http://heart.bmj.com/content/84/6/648

These include:

References
This article cites 25 articles, 9 of which you can access for free at:
http://heart.bmj.com/content/84/6/648#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections
Articles on similar topics can be found in the following collections

- Drugs: cardiovascular system (8842)
- Clinical diagnostic tests (4779)
- Acute coronary syndromes (2742)

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/