Radiofrequency Catheter Ablation of Ventricular Tachycardia After Myocardial Infarction

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Background—Patients with ventricular tachycardia (VT) after myocardial infarction often have multiple morphologies of inducible VT, which complicates mapping and is viewed by some as a relative contraindication to ablation. Attempting to identify and target a single “clinical” VT is often limited by inability to obtain 12-lead ECGs of VTs that are terminated emergently or by defibrillators. This study assesses the feasibility of ablation in patients selected without regard to the presence of multiple VTs by targeting all VTs that allow mapping.

Methods and Results—Radiofrequency catheter ablation targeting all inducible monomorphic VTs that allowed mapping was performed in 52 patients with prior myocardial infarction. Antiarrhythmic drug therapy had failed in 41 (79%) patients including amiodarone in 36 (69%) patients. An average of 3.6±2 morphologies of VT were induced per patient. More than 1 ablation session was required in 16 (31%) patients. Complications occurred in 5 (10%) patients, including 1 (2%) death caused by acute myocardial infarction. During follow-up 59% of patients continued to receive amiodarone; 23 (45%) had implantable defibrillators. During a mean follow-up of 18±15 months (range 0 to 51 months) 1 patient died suddenly, 2 died from uncontrollable VT, and 5 died from heart failure. Three-year survival rate was 70±10%, and rate for risk of VT recurrence was 33±7%.

Conclusions—Radiofrequency catheter ablation controls VT that is sufficiently stable to allow mapping in 67% of patients despite failure of antiarrhythmic drug therapy and multiple inducible VTs. However, ablation was largely adjunctive to amiodarone and defibrillators in this referral population. (*Circulation*. 1998;98:308-314.)

Key Words: catheter ablation ■ tachycardia ■ myocardial infarction

Catheter ablation of sustained monomorphic ventricular tachycardia (VT) late after myocardial infarction has been challenging. These arrhythmias arise from reentry circuits that can be large and complex, with broad paths and narrow isthmuses, and that may traverse subendocardial, intramural, and epicardial regions of the myocardium.1,2

Mapping and ablation are further complicated by the frequent presence of multiple reentry circuits, giving rise to several morphologically different VTs.2–7 In some cases different reentry circuits form in the same abnormal region. In other cases reentry circuits form at disparate sites in the infarct. The presence of multiple morphologies of inducible or spontaneously VT has been associated with antiarrhythmic drug inefficacy8 and failure of surgical ablation.9

Several investigators have reported series of patients selected for having 1 predominant morphology of VT, who were treated with radiofrequency (RF) catheter ablation.8–10 It is likely that this represents <10% of the total population of patients with VT.10 Ablation that focused on the “clinical tachycardia” but did not target other inducible VTs successfully abolished the “clinical” VT in 71% to 76% of cases. However, during follow-up, up to 31% of patients with acutely successful ablation of the “clinical” VT had arrhythmia recurrences, some of which were due to a VT different from that initially targeted for ablation.

There are several difficulties with selecting a dominant, “clinical” VT for ablation. Often it is not possible to determine which VT is in fact the one that has occurred spontaneously. Only a limited recording of 1 or a few ECG leads may be available. In patients with implantable defibrillators VT is typically terminated by the device before an ECG lead is obtained. Even if 1 VT is identified as predominant, other VTs that are inducible may subsequently occur spontaneously.

An alternative approach is not to consider the number of VT morphologies in determining eligibility for catheter ablation but rather to attempt ablation of all inducible VTs that are sufficiently tolerated to allow mapping. The purpose of...
### Patient Characteristics and Comparison of Patients With and Without Arrhythmia Recurrences

<table>
<thead>
<tr>
<th></th>
<th>Total (Range)</th>
<th>Recurrent Arrhythmias</th>
<th>No Recurrence</th>
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<tr>
<td>n</td>
<td>52</td>
<td>17</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>65±10 (38–85)</td>
<td>64±9</td>
<td>65±10</td>
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<tr>
<td>Male/female</td>
<td>44/8</td>
<td>14/3</td>
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<tr>
<td>LV ejection fraction</td>
<td>0.33±0.11 (0.15–0.55)</td>
<td>0.27±0.09</td>
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<td>Myocardial infarction</td>
<td></td>
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<tr>
<td>Anterior</td>
<td>19 (37)</td>
<td>5 (29)</td>
<td>14 (40)</td>
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<tr>
<td>Inferior</td>
<td>26 (50)</td>
<td>12 (71)</td>
<td>14 (40)</td>
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<tr>
<td>Anterior and inferior</td>
<td>7 (13)</td>
<td>0</td>
<td>7 (20)</td>
<td></td>
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<tr>
<td>Spontaneous VT episodes</td>
<td></td>
<td></td>
<td></td>
<td>0.33</td>
</tr>
<tr>
<td>1–5</td>
<td>18 (35)</td>
<td>5 (29)</td>
<td>13 (37)</td>
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<td>&gt;5</td>
<td>27 (52)</td>
<td>8 (47)</td>
<td>19 (54)</td>
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<tr>
<td>Incessant</td>
<td>7 (13)</td>
<td>4 (24)</td>
<td>3 (9)</td>
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<tr>
<td>Failed AAD</td>
<td>41 (79)</td>
<td>16 (94)</td>
<td>25 (71)</td>
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<tr>
<td>Failed amiodarone</td>
<td>36 (69)</td>
<td>15 (88)</td>
<td>21 (60)</td>
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</tr>
<tr>
<td>ICD (initial)</td>
<td>16 (31)</td>
<td>5 (29)</td>
<td>11 (31)</td>
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</tr>
<tr>
<td>History of VF</td>
<td>11 (21)</td>
<td>4 (24)</td>
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<tr>
<td>Time from MI to VT (n=42)</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>3–12 mo</td>
<td>5 (12)</td>
<td>2 (20)</td>
<td>3 (9)</td>
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<tr>
<td>&gt;12 mo</td>
<td>37 (88)</td>
<td>8 (80)</td>
<td>29 (91)</td>
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<td>CAD (n=51)</td>
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<tr>
<td>1 Vessel</td>
<td>9 (11)</td>
<td>4 (24)</td>
<td>5 (14)</td>
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<tr>
<td>2 Vessels</td>
<td>7 (14)</td>
<td>3 (18)</td>
<td>4 (11)</td>
<td></td>
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<tr>
<td>3 Vessels</td>
<td>35 (68)</td>
<td>9 (53)</td>
<td>26 (74)</td>
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<td>Antiarrhythmic drug at ablation</td>
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<td></td>
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<tr>
<td>None</td>
<td>16 (31)</td>
<td>2 (12)</td>
<td>14 (40)</td>
<td></td>
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<tr>
<td>Amiodarone</td>
<td>30 (58)</td>
<td>12 (71)</td>
<td>18 (51)</td>
<td></td>
</tr>
<tr>
<td>Other†</td>
<td>9 (11)</td>
<td>3 (18)</td>
<td>3 (9)</td>
<td></td>
</tr>
<tr>
<td>Drug combinations</td>
<td>8 (15)</td>
<td>5 (29)</td>
<td>3 (9)</td>
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<tr>
<td>VT morphologies</td>
<td>3.6±2</td>
<td>4.2±2</td>
<td>3.3±1.7</td>
<td>0.09</td>
</tr>
<tr>
<td>&gt;3</td>
<td>20 (38)</td>
<td>9 (53)</td>
<td>11 (31)</td>
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<tr>
<td>Cycle length of slowest VT, ms</td>
<td>423±108</td>
<td>499±119</td>
<td>389±88</td>
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<tr>
<td></td>
<td>(260–720)</td>
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<td>Follow-up EP (n=41)*</td>
<td>41</td>
<td>10</td>
<td>31</td>
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<tr>
<td>No VT*</td>
<td>14 (46)</td>
<td>3 (30)</td>
<td>16 (52)</td>
<td>0.001</td>
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<tr>
<td>Modified*</td>
<td>16 (38)</td>
<td>2 (20)</td>
<td>14 (45)</td>
<td></td>
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<tr>
<td>Inducible VT unchanged*</td>
<td>6 (15)</td>
<td>5 (50)</td>
<td>1 (3)</td>
<td></td>
</tr>
<tr>
<td>Recurred spontaneously in hospital</td>
<td>7 (13)</td>
<td>7 (41)</td>
<td>0</td>
<td></td>
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<tr>
<td>AAD during follow-up (n=51)</td>
<td></td>
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<td></td>
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<tr>
<td>Amiodarone</td>
<td>30 (58)</td>
<td>10 (59)</td>
<td>20 (57)</td>
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<td>Sotalol</td>
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<td>1 (6)</td>
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<td>19 (37)</td>
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</tr>
<tr>
<td>ICD</td>
<td>23 (45)</td>
<td>9 (53)</td>
<td>14 (40)</td>
<td>0.55</td>
</tr>
</tbody>
</table>

1 LV indicates left ventricular; VT, ventricular tachycardia; AAD, antiarrhythmic drug; ICD, implantable cardioverter defibrillator; VF, ventricular fibrillation; MI, myocardial infarction; CAD, coronary artery disease; and EP, electrophysiology study.

Numbers in parentheses are percentages for the column or ranges.

*Percentages are relative to the 41 patients who had a follow-up EP.

†Class I drugs or sotalol.
ventricle was achieved retrogradely across the aortic valve or, in 6
Webster Laboratories) that had a 4 mm distal tip electrode and 2 to
formed with 6F or 7F steerable catheters (EP Technologies or
of each respective institution. Left ventricular mapping was per-
according to a protocol approved by the human research committee
informed consent, mapping and RF catheter ablation were performed
4000 U every 2 hours. Sedation was achieved with intermittent doses
heparin, 5000 U intravenously followed by 1000 U every hour or
Our methods have been described previously. 7,11,12 After obtaining
monomorphic VT late after myocardial infarction referred to
Brigham and Women’s Hospital, in Boston, Mass, UCLA Medical
Center or Wadsworth VA Medical Center in Los Angeles, Calif, and
the Hospital of the University of Pennsylvania, in Philadelphia.
Catheter ablation was offered to all patients who had sustained VT
that was sufficiently tolerated hemodynamically to allow catheter
mapping, provided that access to the left ventricle was possible and
that there was no left ventricular pedunculated thrombus present on
the transthoracic echocardiogram. Initially, catheter ablation was
offered only if spontaneous VT recurred despite antiarrhythmic drug
therapy. After the initial 21 patients, ablation was also offered to
patients with spontaneous VT without cardiac arrest, who had not
failed drug therapy (11 of the subsequent 21 patients). Patient
characteristics are shown in the Table.

Mapping and Radiofrequency Ablation
Our methods have been described previously. 7,11,12 After obtaining
informed consent, mapping and RF catheter ablation were performed
according to a protocol approved by the human research committee
each respective institution. Left ventricular mapping was per-
formed with 6F or 7F steerable catheters (EP Technologies or
Webster Laboratories) that had a 4 mm distal tip electrode and 2 to
2.5 mm spacing between the distal 2 electrodes. Access to the left
ventricle was achieved retrogradely across the aortic valve or, in 6
procedures, through transatrial septal puncture. Catheter position was
assessed by fluoroscopy and in 5 patients also by transesophageal
echocardiography. 13 Systemic anticoagulation was achieved with
heparin, 5000 U intravenously followed by 1000 U every hour or
2000 U every 2 hours. Sedation was achieved with intermittent doses
of midazolam, diazepam, meperidine HCl, and/or fentanyl.

Mapping data from the first 37 patients have been previously
reported. 7,11 The general approach to mapping and ablation is shown
in Figure 1. If VT was not incessant, fractionated electrograms were
sought during sinus rhythm and pace mapping was performed at
these sites to locate regions of slow conduction. 11 VT was then
initiated by programmed stimulation from the right ventricular apex.
Unipolar pacing was performed at the mapping site during VT to
reset or entrain the VT, followed immediately by RF current
application at selected sites to determine whether or not heating
would terminate VT. In the first 15 patients RF current was applied
if the site had low amplitude, fractionated electrograms and if pacing
from the site entrained VT. On the basis of analysis of the data in
these initial 15 patients, 7 priority for ablation sites in subsequent
patients was given to exit, central, or proximal sites as defined by
entrainment 11 or if such sites could not be found, sites with isolated
potentials or entrainment features suggesting an outer or inner loop, 11
and finally if none of these were present at sites with abnormal
presystolic electrograms. If a previously tolerated VT did not allow
mapping because of hemodynamic compromise despite adequate
cardiac filling pressures, administration of dopamine for hemody-
namic support and/or intravenous procainamide to slow the
tachycardia was used.

RF current (250 or 500 Hz) was applied between the distal
electrode on the mapping catheter and a cutaneous adhesive elec-
trode at 15 to 45 W for 20 to 40 seconds during VT. In the first 29
patients power was usually adjusted to produce a fall in impedance,
which is an indication of heating. 14 In the subsequent patients
body temperature monitored from the electrode tip was adjusted to
~60°C or a maximum RF current output of 45 to 50 W. If VT terminated,
the application was continued for a total of 60 to 120 seconds or until
a rise in impedance or boiling at the electrode tip on transesophageal
ehochiographic imaging was observed. 13 At sites where RF
current terminated VT, an attempt was made to enlarge the initial
lesion by applying RF current for 60 to 120 seconds during sinus
rhythm to sites within ~5 mm of the initial lesion. RF ablation at this
region was considered adequate when unipolar pacing stimuli at the
amplitude that had captured before ablation (usually 10 mA, 2 to 9
ms pulse width) failed to capture after ablation. If any hemodynam-
ically tolerated sustained monomorphic VT was still inducible, the
mapping procedure was continued. The procedure was ended when
either no hemodynamically tolerated VTs were inducible or when no
diagnostic target sites critical to reentry, as assessed from entrain-
ment and interruption of VT by RF current, could be found.

Management After Ablation
After ablation, patients were monitored on a cardiac step-down unit
or intensive care unit. If amiodarone had been administered long
before the procedure, this medication was continued unless
potentially necessary stopping. Other antiarrhythmic drugs were
discontinued unless the ablation procedure had been clearly unsuc-
cessful; that is, the same VTs that had predominated at the beginning
of the procedure remained inducible at the end of the procedure.
Aspirin (325 mg) was administered daily. Patients treated with
warfarin before the procedure for known or presumed increased risk
of systemic emboli received a continuous intravenous infusion of
heparin until resumption of warfarin. If sustained VT recurred
spontaneously in the hospital after an apparently successful ablation,
a repeat ablation attempt was recommended.

If the patient remained free of VT for the following 3 to 7 days, a
follow-up electrophysiology study was recommended with addi-
tional ablation attempts if VTs had been terminated at the previous
session and hemodynamically tolerated VT was again inducible.
This follow-up study was performed a mean of 7±14 days (range 3
to 57 days) after the last ablation in 41 of the 44 in whom VT did not
recur spontaneously in hospital. In the remaining 3 patients the last
electrophysiologic study was performed immediately after ablation.
Programmed stimulation included 1, 2, and 3 extrastimuli during
pacing at 100 and 150 bpm from the right ventricular apex and right
ventricular outflow tract. In 5 patients with VT that had been
repeatedly inducible from the right ventricular apex, noninvasive
programmed stimulation from an implanted defibrillator was per-
formed, with pacing at only 1 site. The end points for stimulation
were reproducible initiation of a tolerated VT, initiation of VT
producing syncope, or the third extrastimulus reaching refractori-
ness. Effects of ablation were defined as (1) no inducible VT:
monomorphic VT with a duration of ≥30 seconds or requiring
earlier termination due to hemodynamic compromise is not induc-
able; (2) modified: a sustained monomorphic VT is inducible but is

Figure 1. Flow diagram of the general approach to mapping
and ablation of ventricular tachycardia (VT). See text for discus-
sion. RF indicates radiofrequency catheter ablation.

this report is to assess the feasibility and summarize the initial
experience with this approach.
different in QRS morphology (frontal plane axis by >30 degrees or precordial transition zone ≥1 lead or a different dominant deflection in >1 precordial lead) or cycle length (>100 ms for VTs with a similar morphology) than the VTs for which ablation could be attempted; and (3) inducible VT: a sustained monomorphic VT observed or induced before attempted ablation remains inducible.

If any sustained monomorphic VT remained inducible at the predischARGE study, therapy with amiodarone and/or an implantable defibrillator was recommended. Prior amiodarone therapy was continued regardless of the results of electrophysiologic testing unless toxicity warranted stopping. If amiodarone was discontinued, we recommended a repeat electrophysiologic study after 6 to 12 weeks.

Statistics
Continuous data are expressed as mean±1 SD. Groups were compared with the Student’s t test, χ² tests, or Fisher’s exact test as appropriate. Multivariable stepwise logistic regression was used to assess predictors of arrhythmia recurrence. Variables used were ejection fraction, failure of amiodarone, failure of any drug, slowest cycle length of induced VT, and number of inducible VTs. Variables with P<0.1 were entered stepwise into the model. Continuous variables were dichotomized at the median (BMDP statistical software, 1992). Survival and arrhythmia recurrences were determined from the date of last ablation with the Kaplan-Meier method (BMDP statistical software, 1992). Arrhythmia recurrences were spontaneous sustained VT or a VT termination by an implantable defibrillator. Patients undergoing cardiac transplantation were censored from the analysis on the day of surgery.

Results
RF catheter ablation was attempted in 52 patients (Table). Antiarrhythmic drug therapy had failed to prevent spontaneous, sustained VT in 41 (79%) patients. Long-term oral amiodarone therapy had failed to suppress spontaneous VT in 33 (63%) patients and had to be withdrawn because of toxicity in 3 (6%) patients. Two patients had only 1 spontaneous episode of sustained monomorphic VT and 50 had had multiple episodes. VT was incessant in 7 (13%) patients. An implantable defibrillator was present in 16 (31%) patients before referral.

Catheter Mapping and Ablation
The 52 patients underwent 69 catheter mapping and ablation sessions; 1 session in 36 patients, 2 sessions in 15 patients, and 3 sessions in 1 patient. At the initial session, 36 patients (69%) were not receiving antiarrhythmic medications. During ablation an average of 3.6±2 different morphologies of monomorphic VT were observed per patient (range 1 to 10). In 5 patients (10%) only 1 VT was induced during the procedure. The average VT cycle length was 423±108 ms (range 260 to 720 ms). Pacing was performed to attempt to entrain 132 VTs in the 52 patients. Reentry circuit exit, central, or proximal regions were identified for 67 VTs in 40 patients. RF current was applied during 124 VTs and acutely terminated 74 VTs in 48 patients. In 8 patients entrainment mapping and RF application during VT were limited because pacing or catheter manipulation in the target region terminated VT or hemodynamic intolerance developed during VT.

The average number of RF applications per procedure, including those that failed to terminate VT was 23±18 (range 1 to 98, median 19). For all procedures, the average procedure time was 328±136 minutes (range 100 to 725 minutes) and the average fluoroscopy time was 60±32 minutes (range 5 to 135 minutes, median 56 minutes).

Procedure-related complications occurred in 5 (10%) patients. There was 1 (2%) procedure-related death from acute inferior wall myocardial infarction with cardiac rupture 12 hours after a second ablation procedure performed for in-hospital VT recurrence that has previously been reported in detail. This patient is included as a death and arrhythmia recurrence in the hospital for all follow-up analyses. This patient also had transient atrioventricular (AV) block observed during the first ablation procedure and developed Staphylococcus epidermidis sepsis from a temporary pacing catheter. One patient had a transient cerebral ischemic event 36 hours after ablation. This patient had a history of identical events before ablation attributed to carotid artery disease. Two patients who had chronic obstructive lung disease developed progressive hypventilation and required assisted ventilation during the procedure. The remaining complication was a femoral artery pseudoaneurysm. Implantable defibrillators, present in 16 patients, were programmed off during the procedure, and all functioned normally afterward.

Early Effects of Ablation on Inducible and Spontaneous Ventricular Tachycardias
In 10 patients, repeat ablation procedures were performed for VT that recurred spontaneously in the hospital before follow-up study (5 patients) or that was inducible at the pre–hospital discharge study (5 patients). In addition, 5 patients had late repeat procedures. Two patients had a repeat ablation attempt after VT recurred 21 and 18 days, respectively, after hospital discharge, despite no VT inducible before hospital discharge. In 3 patients, amiodarone was discontinued after initial ablation. In 2 of these patients, programmed stimulation after 7 and 10 weeks, respectively, induced a new VT in 1 patient and a VT similar to 1 of 3 previously observed VTs in the other patient. Both underwent an additional ablation procedure with no VT inducible before discharge. The third patient who was seen with incessant VT and had VT modified at the initial ablation session had a new morphology of VT occur spontaneously 6 months after amiodarone withdrawal. After repeat ablation, this VT was no longer inducible, but other faster VTs that did not allow mapping remained inducible.

Early after the final ablation procedure, 21 patients (40%) had no spontaneous or inducible sustained monomorphic VT. These patients had a total of 70 monomorphic VTs previously inducible. In 16 (31%) patients VT was inducible but modified and did not recur spontaneously before hospital discharge. The cycle length of the slowest inducible VT had shortened by 97 ms, from 425±15 to 328±18 ms. A total of 60 VTs had been previously inducible in these patients. Further ablation attempts were not performed because VT was poorly tolerated in 11 patients and because no endocardial target sites could be identified in 5 patients.

In 15 patients (29%), ablation was judged to be ineffective. In 8 patients (15%) VT recurred in the hospital; 5 of these had persistently inducible VT at the end of the ablation procedure. In 7 patients (13%), VT did not recur in the hospital but a VT similar to that initiated before ablation remained inducible at the last electrophysiology study. Further ablation was not attempted in these 15 patients because inducible VTs were
poorly tolerated in the electrophysiology laboratory in 6 patients, no target sites could be identified in 5 patients, previously frequent or incessant VT was now controlled with drug therapy in 3 patients, and 1 patient died, as noted above.

**Survival and Ventricular Tachycardia Recurrences**

After ablation, antiarrhythmic drugs continued to be administered to 32 (63%) of 51 patients; amiodarone in 30 (59%) patients and sotalol in 2 (4%) patients. Implantable defibrillators were present in 23 (45%) patients, 7 of which were implanted after ablation. Among the 23 patients with implantable defibrillators 14 also continued to be treated with antiarrhythmic drugs for atrial fibrillation (2 patients) or inducible VT after ablation. The mean follow-up after the final ablation session was 18±15 months (range 0 to 59 months).

Ten patients died (19%): 1 was procedure related, 2 were in the hospital from VT that was not controlled by ablation, 5 died of heart failure, and 1 died of end-stage liver disease. One patient died suddenly after 15 months. This patient had no VT inducible before discharge; amiodarone (200 mg/d) had been started 6 months later for atrial fibrillation. Three patients underwent cardiac transplantation, all had severe ventricular dysfunction before ablation (left ventricular ejection fractions of 0.15, 0.18, and 0.27, respectively) and unsuccessful ablations with multiple VT recurrences afterward. Of the 5 patients who died of congestive heart failure, all had symptomatic heart failure before ablation. Left ventricular ejection fraction was <0.20 in 3 patients, 2 of whom had class IV symptoms before ablation. In the 2 patients with better left ventricular ejection fractions (0.3 and 0.4, respectively), death occurred after 4.2 and 2.4 years of follow-up and followed coronary artery bypass surgery and aneurysmectomy in 1 patient. Three-year survival for all 52 patients was 70±10% (Figure 2A).

Sustained VT that was not fatal recurred in 16 (31%) patients either in the hospital before discharge (7 patients) or during follow-up (9 patients). For all 52 patients, the 3-year risk of recurrent ventricular arrhythmia, including the 1 sudden death, was 33±7% (Figure 2B). Thus 67% of patients remained free of VT during follow-up.

When ablation was not successful, it was evident soon after the procedures. In 76% of patients with recurrences, the first VT recurrence occurred within 4 weeks of ablation. For the patients who did not recur spontaneously in the hospital after ablation, predischarge electrophysiologic testing was predictive of outcome (Table). Arrhythmia recurrences (including 1 sudden death) occurred in 3 of 19 (16%) patients without inducible VT, 2 of 16 (13%) patients in whom inducible arrhythmias were modified, and 5 of 6 (83%) patients in whom inducible VT was not changed (P=0.001).

Patients with spontaneous arrhythmia recurrences (VT or sudden death) are compared with those remaining free of recurrences in the Table. Patients with recurrent arrhythmias had worse ventricular function. The number of morphologies of inducible VT was not significantly different between the groups. Prior antiarrhythmic drug failure was associated with a greater risk of arrhythmia recurrence (Table and Figure 3). Of the 11 patients who had not failed antiarrhythmic drugs, 10 (91%) were free of VT recurrences during follow-up. However, 3 were treated with amiodarone (2 patients) or sotalol (1 patient) for inducible modified VT at follow-up study (1 patient) or at the referring physician’s request (2 patients). One of these patients received an implanted defibrillator for modified but inducible VT. Patients with recurrences had slower VTs inducible at initial study, probably because of frequent amiodarone use before ablation in this group. By multivariate logistic regression, failed therapy with amiodarone (odds ratio 5.9, 95% confidence interval 1.1 to 33) and a slowest VT cycle length >405 ms (odds ratio 4.0, 95% confidence intervals 0.97 to 16.5) were independent predictors of arrhythmia recurrence. When failed amiodarone therapy was excluded from the model, left ventricular ejection fraction <0.34 was a predictor of recurrences with

![Figure 2](http://circ.ahajournals.org/Download/)

**Figure 2.** Survival after catheter ablation (A) and cumulative arrhythmia recurrences (B), including 1 sudden death, are shown for all 52 patients. Number of patients at each point in time is shown above the abscissa. VT indicates ventricular tachycardia.

![Figure 3](http://circ.ahajournals.org/Download/)

**Figure 3.** Arrhythmia recurrences are shown for the 41 patients who had previously failed antiarrhythmic therapy (dashed line) and 11 patients who had not previously failed antiarrhythmic drug therapy (solid line). Number of patients remaining at each point in time is shown below the curves.
borderline statistical significance (odds ratio 3.3, 95% confidence interval 0.85 to 13) independent of VT cycle length (odds ratio 5.14, 95% confidence interval 1.3 to 21).

Discussion
Ablation of VT after myocardial infarction is often more difficult than ablation of supraventricular tachycardia. The presence of multiple morphologies of inducible monomorphic VT is a frequent complicating factor. Recent studies of RF catheter ablation for postinfarct VT have generally attempted to exclude patients with more than 1 morphology of VT, offering the procedure to a small minority of patients with sustained monomorphic VT. Our study, as well as a recent study by Rothman and coworkers, suggests that RF catheter ablation that targets any stable VT may be useful even if multiple VTs are inducible. Initially all of our patients had failed antiarrhythmic drug therapy because of spontaneous recurrences of VT. Later in the study, for 11 patients, drug failure was not required. Despite the predominance of patients with drug-refractory VT and multiple morphologies of VT, two thirds were rendered free of recurrent VT during follow-up with a low incidence of complications. When ablation was not successful, 76% of patients had recurrent VT within 4 weeks. However, these procedures are technically challenging, the procedure times are relatively long, and many patients required multiple procedures.

This study, although not randomized or controlled, provides useful information regarding management of patients after ablation. Modification of the reentry substrate was a common outcome of ablation. Modification was defined as persistence of inducible VT that was clearly different than the previously inducible tachycardias. These VTs were usually faster and often not tolerated sufficiently to allow mapping. On average, the VT cycle length shortened by ≈100 ms, which suggests that ablation damaged a portion of the slowly conducting tissue in the reentry circuit, such that the remaining circuits had faster revolution times. We cannot be certain that these faster VTs were absent before ablation, however, because slower tachycardias are usually induced more easily and earlier in the stimulation protocol. In any case, modification was associated with a favorable outcome and relatively low risk of arrhythmia recurrence. However, these patients generally continued to receive amiodarone or received an implantable defibrillator. Defibrillator implantation is a reasonable strategy, supported by the study of Rothman and coworkers, who observed VT recurrences in 9 of 19 patients who had a “clinical” VT ablated but other VTs still inducible. Management of the patient who is receiving amiodarone at the time of ablation is a particularly difficult issue. Even if VT is not inducible after ablation, amiodarone may be suppressing other VTs. Because of the long half-life of elimination of the drug, withdrawal exposes the patient to a risk of arrhythmia recurrence long after hospital discharge. Therefore we continued amiodarone unless an implantable defibrillator was present or amiodarone toxicity precluded continued therapy. Our experience in 3 patients in whom amiodarone was discontinued is instructive. All had inducible or spontaneous VT at follow-up electrophysiologic studies 7 to 42 weeks later. However, all had successful ablation of these VTs and remained free of VT during subsequent follow-up. For patients who had not previously failed antiarrhythmic drug therapy, catheter ablation was quite effective: 91% were free of arrhythmia recurrences during follow-up. This was a select group of patients, however, who had hemodynamically tolerated VT without cardiac arrest in the absence of antiarrhythmic drug therapy.

In targeting multiple VTs, we probably applied ablation to larger regions of the infarct, accounting for the greater number of RF applications compared with previous series. In prior reports, surgical ablation of VT removed 8 cm² to >40 cm² of subendocardial tissue. Saksena and coworkers observed that focal intraoperative laser ablation of 2 to 25 cm² was required for interruption of VT. We performed ablation with conventional RF catheters, which create lesions that are typically 5 to 10 mm in diameter, and often substantially smaller because of vagaries of tissue contact, electrode-tissue orientation, and cooling from the surrounding blood pool. With present RF systems it is likely that multiple RF applications are required to remotely approach the effect achieved with surgery. This raises important safety concerns because damage to contracting myocardium could further depress ventricular function. We are careful to confine RF applications to regions that have abnormal electrogams, with the intention of avoiding damage to contractile muscle. In our single patient who died of myocardial infarction after ablation, the RF lesions were confined to the infarct region. Although we did not observe clinical exacerbations of heart failure after ablation, mild alterations may have been difficult to appreciate in patients with frequent episodes of VT. The 10% incidence of death from heart failure during follow-up is not unexpected in this patient population and is similar to that observed by Gonska and coworkers, who targeted 1 VT for ablation. Assessment of the impact of ablation on left ventricular function will be important in future studies.

Limitations
Antiarrhythmic drug management after ablation is a difficult issue, and many of our patients continued to receive amiodarone during follow-up. Continued amiodarone therapy is unlikely to explain the long-term success after ablation because this drug previously failed to prevent spontaneous VT. Our approach to mapping limited our ability to characterize all of the potential ventricular reentry circuits and to determine how frequently multiple VTs originated from common or separate regions. Ablation with enlargement of the initial lesion was performed at 1 of the initial reentry circuit sites identified, regardless of whether that VT had been previously identified as occurring spontaneously or whether other VTs had been mapped. Ablation at 1 region often appeared to abolish several VTs, as has been reported by others. For example, 1 patient had several episodes of VT with a right bundle-branch block configuration. Programmed electrical stimulation repeatedly induced a VT with a left bundle-branch block configuration. After ablation of this VT, only rapid “ventricular flutter” was provokable. During follow-up of 19 months there have been no arrhythm-
Ventricular Tachycardia Ablation

Clinical Implications
RF catheter ablation of VT after myocardial infarction abolishes spontaneous episodes of VT in two thirds of patients regardless of the presence of multiple morphologies of inducible VT. It provides excellent palliation for many patients who have repeated episodes of spontaneous VT. The procedure is, however, technically demanding. Further studies are justified to determine how catheter ablation should fit into the therapeutic strategy for patients with hemodynamically tolerated VT.

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