Conclusions—In patients with difficult-to-control VT in whom RFCA fails, TCEA prevents all VT recurrences in 36% and or incessant ventricular tachycardia (VT).1,2 Although percutaneous ethanol ablation (TCEA) was first performed experimentally3,4 and used to ablate postinfarction VT in the early 1990s.5,6 Subsequently, significant improvement and experience in interventional techniques for subselective targeting of coronary vessels have occurred. In 2008, we reported encouraging results with transcoronary ethanol ablation (TCEA), we began offering TCEA routinely when endocardial and epicardial RFCA failed or a deep intramural substrate was likely.

Methods and Results—Among 274 consecutive patients who underwent 408 ventricular tachycardia (VT) ablation procedures, 27 patients (21 men; age, 63±13 years; left ventricular ejection fraction, 30±11%; ischemic cardiomyopathy, 14) had 29 TCEA procedures attempted. In 5 patients, TCEA was abandoned because of unfavorable anatomy. In 22 patients, a mean of 1.3±0.6 arteries (range, 1–3 arteries) were targeted for TCEA. After ablation, the targeted VT was no longer inducible in 18 of 22 (82%) patients. Complete heart block occurred in 5 patients, and 3 patients with advanced heart failure died within 30 days of the procedure. After the last TCEA procedure, a VT recurred in 64% of patients, and overall, 32% of patients died. Of 11 patients with prior VT storm, 9 were free of VT storm. At repeat study in 8 patients who had a recurrence, 7 had a new QRS morphology of VT originating from the same general substrate region as the prior VT.

Conclusions—In patients with difficult-to-control VT in whom RFCA fails, TCEA prevents all VT recurrences in 36% and improves arrhythmia control in an additional 27%. Inadequate target vessels, collaterals, and recurrence of modified VTs limit efficacy, but TCEA continues to play an important role for difficult VTs in these high-risk patients. (Circ Arrhythm Electrophysiol. 2011;4:889-896.)

Key Words: ventricular tachycardia ■ catheter ablation ■ ethanol ■ coronary artery ■ outcome ■ complication

Radiofrequency catheter ablation (RFCA) plays an important and often life-saving role for patients with frequent or incessant ventricular tachycardia (VT).1,2 Although percutaneous and surgical approaches to the pericardial space now allow RFCA to target epicardial VTs, there are still a number of patients in whom VT cannot be eliminated with both the endocardial and the epicardial approach. Intramural ablation appears to be required in some patients. Transcoronary ethanol ablation (TCEA) was first performed experimentally3,4 and used to ablate postinfarction VT in the early 1990s.5,6 Subsequently, significant improvement and experience in interventional techniques for subselective targeting of coronary vessels have occurred. In 2008, we reported encouraging results in 9 patients who had undergone TCEA between January 1999 and May 2007.7 Based on this experience, we began offering TCEA routinely when endocardial and epicardial ablation failed or when endocardial ablation failed and a deep intramural VT, such as in the septum, was believe to be likely. This update provides the largest reported experience of TCEA for VT to our knowledge and further clarifies its limitations with present techniques.

Clinical Perspective on p 896

Methods

Patient Selection From June 2007 to September 2010, 274 consecutive patients (217 men; age, 62±13 years) with structural heart disease underwent 408 VT ablations (1.4±0.7 times) at our institution and were included in this analysis. Patients who had at least 1 RFCA for symptomatic monomorphic VTs that were refractory to endocardial ablation, epicardial ablation, or both were considered for TCEA. VT storm was defined as ≥3 separate VT episodes in a 24-hour period before
ablation. Written informed consent was obtained from all patients. Procedures and review of medical records were conducted under protocols approved by the institutional human subject protection committee.

Transcatheter Ethanol Ablation

Transesophageal echocardiography was performed before and after the procedure in all patients to assess cardiac disease and function. Patients were studied in a fasted state with all antiarrhythmics except amiodarone stopped for >5 half lives. Patients receiving warfarin were transitioned to heparin, which was then discontinued 6 hours before arrival in the electrophysiology laboratory. The details of the initial mapping, RFCA, and TCEA procedure have been described previously.7,8 Surface ECG leads and intracardiac electrograms were stored in digital format (Prucka CardioLab EP System; GE Healthcare; Waukesha, WI). Nonfluoroscopic electroanatomic mapping was performed using a 3D mapping system (CARTO; Biosense Webster, Inc; Diamond Bar, CA). Morphological analysis of VT, location of scar from low-voltage areas on electroanatomical mapping, pace mapping, and entrainment mapping were used to identify ablation target locations.

At least 1 quadripolar electrode catheter was positioned through a femoral vein in the right ventricle. When VT was not inducible, programmed stimulation using up to 3 extrastimuli from 2 right ventricular sites was performed for VT induction. The operators for each procedure included both an electrophysiologist and an interventional cardiac surgeon experienced in the TCEA. After clinical VT was induced and terminated, selective coronary angiography was performed using a femoral arterial sheath. Patients were heparinized, and the activated clotting time was maintained at >250s. The ostium of the relevant coronary artery was selectively intubated using an angioplasty wire (Graphix Intermediate; Boston Scientific Corporation; Natick, MA). An 8-mm over-the-wire balloon sized to be slightly larger than the angiographic diameter of the target vessel (HighSail; Guidant Corporation; Santa Clara, CA) was deployed in the ostium of the target branch vessel and fully inflated to prevent backwash of ethanol into other coronary vessels, and occlusion was verified using contrast injection after guidewire removal. A sonography contrast agent (Optison; Amersham Health; Buckinghamshire, UK) was injected in the targeted coronary artery, and its territory was verified by echocardiography in some patients. Next, the VT was reinduced, and with the balloon fully inflated, iced saline (2–3 mL) was injected through the central lumen in an attempt to terminate VT. Alcohol injection was performed only if VT terminated during iced saline infusion in the vessel (19 patients) or if VT that had been inducible before selective cannulation of the vessel was no longer inducible when blood flow was interrupted in the vessel by the cannulation and balloon inflation (3 patients). If the VT continued, another branch was targeted. In addition, the territory of perfusion was assessed, seeking arterial branches believed to be sufficiently distal and without supplying collateral flow to adjacent areas such that they were deemed unlikely to cause significant collateral vascular damage. Once an appropriate target site had been identified, 1 mL of sterile absolute alcohol was injected with the balloon inflated for 10 minutes. After deflation of the balloon, contrast was injected to assess target vessel patency. If perfusion was present, a second 1 mL of additional ethanol was infused, and the balloon inflation was maintained for 10 minutes after that infusion. This could be repeated up to a maximum of 5 mL of ethanol in 1 artery.

After TCEA, programmed right ventricular stimulation as described previously was performed. If the targeted VT remained inducible, we sought an adjacent target vessel using the same method.

Serum creatine kinase (CK), CK-MB, and troponin I were measured at least daily after each procedure until they peaked and declined. Renal function was assessed from serum creatinine within 24 hours before and 24 and 72 hours after the TCEA. Estimated glomerular filtration rate was calculated using a modified equation to predict the glomerular filtration rate from the serum creatinine value.9 Contrast-induced acute kidney injury was defined as an increase from the baseline serum creatinine concentration of at least 0.5 mg/dL or at least 25% within 48 to 72 hours after angiography.10

Patients were seen by the investigator or referring physician after discharge, and implantable cardioverter-defibrillator interrogation was obtained. Survival was assessed from referring physicians and the Social Security Death Index. VT improvement was defined as the absence of sustained VT or no recurrence of preexisting VT storm or incessant VT.

Statistical Analysis

Continuous variables are expressed as mean±SD or median. Student t test was chosen for continuous variables, and Mann-Whitney U test was chosen for ordinal outcome variables. Categorical variables, expressed as numbers or percentages, were analyzed using χ² tests (Table 1), unless the number of values in any cell was <5, in which case, Fisher exact test was used. Left ventricular ejection fractions before and after the TCEA procedure were compared with the paired t test. All tests were 2-tailed, and P<0.05 was considered to be statistically significant. Statistical analysis were performed using SPSS version 18.0.0 (SPSS Inc; Chicago, IL) software. The authors had full access to the data and take full responsibility for the integrity of the data.

Results

Patient Characteristics

A total of 29 TCEA procedures were performed for 27 patients (21 men; age, 63±13 years) at our institution from

Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>TCEA (−) (n=247)</th>
<th>TCEA (+) (n=27)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>62±13</td>
<td>63±13</td>
<td>0.87*</td>
</tr>
<tr>
<td>Male sex</td>
<td>196 (79)</td>
<td>21 (78)</td>
<td>0.51†</td>
</tr>
<tr>
<td>Ischemic cardiomyopathy</td>
<td>149 (60)</td>
<td>14 (52)</td>
<td>0.41†</td>
</tr>
<tr>
<td>Major coronary vessels stenosed</td>
<td>1.3±1.3</td>
<td>1.2±1.3</td>
<td>0.69*</td>
</tr>
<tr>
<td>1</td>
<td>68 (28)</td>
<td>4 (15)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>38 (15)</td>
<td>3 (11)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>30 (12)</td>
<td>7 (26)</td>
<td></td>
</tr>
<tr>
<td>History of cardiac surgery</td>
<td>94 (38)</td>
<td>13 (48)</td>
<td>0.31†</td>
</tr>
<tr>
<td>History of CABG</td>
<td>78 (32)</td>
<td>9 (33)</td>
<td>0.83†</td>
</tr>
<tr>
<td>No. of previous ablations</td>
<td>0.5±0.7</td>
<td>2.0±1.7</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>≥2</td>
<td>25 (10)</td>
<td>13 (48)</td>
<td>&lt;0.001†</td>
</tr>
<tr>
<td>NYHA class</td>
<td>0.58‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>117 (47)</td>
<td>7 (26)</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>52 (21)</td>
<td>15 (56)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>65 (26)</td>
<td>3 (11)</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>13 (5)</td>
<td>2 (7)</td>
<td></td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>34±15</td>
<td>30±11</td>
<td>0.23*</td>
</tr>
<tr>
<td>VT storm</td>
<td>49 (20)</td>
<td>11 (41)</td>
<td>0.02†</td>
</tr>
<tr>
<td>Failed AAD</td>
<td>2.0±1.3</td>
<td>2.8±1.3</td>
<td>0.003*</td>
</tr>
<tr>
<td>β-blocker</td>
<td>207 (84)</td>
<td>25 (93)</td>
<td>0.39†</td>
</tr>
<tr>
<td>ICD</td>
<td>210 (85)</td>
<td>27 (100)</td>
<td>0.03†</td>
</tr>
<tr>
<td>CRT</td>
<td>63 (26)</td>
<td>9 (33)</td>
<td>0.37†</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or n (%). AAD indicates antiarrhythmic drug; CABG, coronary artery bypass graft; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter-defibrillator; LV, left ventricular; NYHA, New York Heart Association; TCEA, transcoronary ethanol ablation; VT, ventricular tachycardia.

*Student t test. †Chi-square test. ‡Mann-Whitney U test.
June 2007 to September 2010. Two patients had 2 TCEA procedures. All 27 patients had structural heart disease as follows: ischemic cardiomyopathy, 14 (52%); dilated cardiomyopathy, 10 (37%); hypertrophic cardiomyopathy, 2 (7%) (previously reported); and valvular heart disease, 1 (4%).

Prior cardiac and coronary artery bypass graft surgery had been performed in 48% and 33% of these patients, respectively. All patients had VT attributed to scar-related reentry, and 20 of the 22 had a mean of 2.0 ± 1.7 failed RFCA attempts.

Prior endocardial ablation was not attempted in 1 patient with left ventricular thrombus and 1 patient with recent biventricular assist device placement because of concerns about potential embolization of thrombus from the recent surgery in the latter (patients 1 and 3, online-only Data Supplement). Epicardial ablation had been attempted in 13 (48%) patients. Initial RF ablation was attempted for all 20 patients with an initially failed ablation elsewhere. RFCA was performed first in 18 patients; in 2 patients, mapping without ablation was performed because a desirable endocardial target region was not identified. The median duration from the last RF ablation to the first TCEA was 32 days. All patients had an implantable cardioverter-defibrillator had been implanted; 9 (33%) had cardiac resynchronization therapy. One patient had a biventricular assist device. In Table 1, patient characteristics are compared with those of the 247 patients with structural heart disease who underwent RFCA but did not undergo attempted TCEA during the same period.

That the TCEA group had VT that was more difficult to control is supported by the fact that more had VT storm (41% versus 20%, \(P=0.02\)) and had failed a greater number of antiarrhythmic drugs (2.8 ± 1.3 versus 2.0 ± 1.3, \(P=0.003\)) and previous ablation attempts (2.0 ± 1.7 versus 0.5 ± 0.7, \(P<0.001\)). First-degree atrioventricular (AV) block was present in 13 (48%) patients, and 3 (11%) patients had complete heart block with ventricular pacing. Of 24 patients with intact AV conduction, 8 had complete left bundle branch block, and 4 had complete right bundle branch block. There were a mean of 3.2 ± 3.7 VTs (median, 2 VTs) inducible during the procedure.

**Transcoronary Ethanol Ablation**

Outcomes are summarized in the flow chart in Figure 1. Coronary angiography showed no coronary artery stenosis deemed to require revascularization. Of 27 patients undergoing catheterization for the procedure, administration of alcohol was not performed in 5 (19%) patients because of the absence of a sufficiently sized coronary artery perfusing the target region in 2 patients, extensive collaterals in 2, and no effect of saline injection on VT in 1. Of these 5 patients, RFCA was subsequently performed in 2, and the remaining 3 received only drug therapy. Patients for whom TCEA was judged to be not feasible had a larger number of stenosed major epicardial arteries (2.4 ± 1.3 versus 0.6 ± 1.0, \(P=0.007\)), and none had presumptive septal arteries as possible targets compared with those with targets (0% versus 68%, \(P=0.02\)) but were otherwise similar to those in whom TCEA could be performed (Table 2).

In 22 initial TCEA procedures, ethanol was injected into 28 coronary arteries (1.3 ± 0.6 arteries/person; range, 1–3 arteries/person). Coronary artery segments (according to the American Heart Association classification) targeted for TCEA are shown in Figure 2; 64% were branches of the left anterior descending coronary artery, 14% were branches of the left circumflex coronary artery, and 22% were branches to the left ventricle from the right coronary artery. In total, 57% of the targeted arteries supplied the septum (including collateral flow). Of the 15 patients with TCEA performed for presumed septal VT, 8 had prior RFCA performed at sites on both sides of the septum. Of the 7 patients with nonseptal coronary targets, 6 had failed endocardial ablation, and 4 had failed epicardial ablation. Of these, 3 had coronary artery disease, 2 nonischemic dilated cardiomyopathy, 1 hypertrophic cardiomyopathy, and 1 valvular disease. Of the 14
patients without prior epicardial ablation, in 10 (71%), VT was believed to originate from the septum, and a septal artery was targeted. All of the targeted sites were related to prior detected scar. In 1 patient, initial coronary angiography revealed a patent stent in the left anterior descending coronary artery located over the first septal branch that was the target vessel, but this septal branch was successfully engaged and cannulated. In 2 patients, the target vessel was accessed through a coronary artery bypass graft (posterior descending branch through saphenous vein graft to right coronary artery and distal septal branch through left internal mammary artery graft to distal left anterior descending coronary artery). Of the 13 patients with prior epicardial ablation, RF application was limited by proximity of a coronary artery to a target region in 1, with a circumflex marginal branch in proximity to a VT exit. This branch was successfully occluded in the following TCEA procedure.

The 22 patients who had initial TCEA received a total dose of ethanol of 3.2/2.4 mL (range, 1–9 mL; median, 3 mL) and received 165/79 mL (range, 75–275 mL; median, 200 mL) of angiographic contrast. The saline injection test was not performed in 3 patients because targeted VT terminated with balloon inflation in the target vessel and was not inducible. After TCEA, no VT was inducible in 10 (46%) patients (acute success). Eight (36%) patients had inducible monomorphic VT that was different from the VT targeted for TCEA (modified). In 3 patients, postablation attempts to reinduce VT were not performed to avoid aggravating their poor hemodynamic status. In 1 patient, the targeted VT remained inducible. No VT was inducible in 60% and 14% of the patients with septal and nonseptal targets, respectively.

Early Outcome and Complications

Of 13 patients with intact AV conduction who had undergone TCEA to arteries that supplied the septum, complete heart block occurred in 5 (38%). All 5 patients had an AV block of more than first degree and a bundle branch block present before ablation (left bundle branch block in 3, right bundle branch block in 2). Patients receiving septal alcohol embolization who developed heart block were similar to those who did not develop heart block (Table 3). AV conduction subsequently recovered in 1 patient 2 days after the procedure. Complete heart block persisted in the other 4 patients.
and 3 patients underwent additional left ventricular lead implantation for implantable cardioverter-defibrillator upgrade to cardiac resynchronization therapy. None of these 5 patients had heart block during balloon occlusion or saline injection in the vessel that received ethanol injection. Another patient had transient severe hypotension after ethanol injection that required intraaortic balloon counterpulsation. Temporary coronary spasm occurred in 1 patient. There were no coronary artery dissections or perforations or other complications related to catheter manipulation in the coronary circulation.

CK, CK-MB, and troponin I at their maximum values after TCEA were 597±380 U/L (normal reference value, 27–218 U/L), 68±40 ng/mL (0.0–5.0 ng/mL), and 14±10 ng/mL (0.0–0.04 ng/mL), respectively. There was no significant relationship between the cardiac enzyme increase and the number of arteries targeted for ablation.

Estimated glomerular filtration rate 24 and 72 hours after the procedure were not significantly different from prepreamo procedure (24 hours, 61±24 versus 66±23 mL/min per 1.73 m², P=0.29; 72 hours, 61±24 versus 66±23 mL/min per 1.73 m², P=0.37). However, 4 patients met criteria for contrast-induced nephropathy. In all 4 patients, renal function recovered without additional therapy or long-term consequences.

Overall echocardiographic assessment a median of 8 days after TCEA did not show a statistically significant change in left ventricular ejection fraction (P=0.35), although some individual changes were observed (online-only Data Supplement Figure). This comparison is limited, however, by variable time of assessment among different patients in relation to VT and cardiac arrest episodes. No unanticipated new wall motion abnormalities were noted.

Three patients died within 30 days of the procedure (online-only Data Supplement). All had a history of ischemic heart disease and heart failure. Deaths were attributed to continued VT and hemodynamic deterioration in 1 patient, cholesterol embolization syndrome with multiorgan failure in the second patient, and failure to recover from prior strokes associated with biventricular assist device placement before ablation in the third patient (online-only Data Supplement). Another patient had a heart transplant 110 days after the procedure. The median time from TCEA to discharge was 7 days.

### Recurrent VT

Fourteen (64%) patients experienced VT recurrence, which occurred at a median of 16 days after the first TCEA procedure. However, 8 of the 14 had a history of VT storm or incessant VT, and 6 remained free of VT storm or incessant VT and were therefore classified as improved. Thus, 14 (63.6%) patients were free of recurrent VT or had improved VT control.

Patients with recurrent VT had a larger number of previous ablation attempts (2.4±0.2 versus 1.0±0.5, P=0.07), had more ablled coronary arteries (1.4±0.6 versus 1.0±0.0, P=0.07), and received a greater amount of injected ethanol (4.1±2.7 versus 1.6±0.6, P=0.03) compared with patients who remained free of VT (Table 4). VT recurred in 9 of 15 (60%) patients with septal artery targets and 5 of 7 (71%) with nonseptal artery targets. Of 14 patients with VT recurrence after TCEA, 7 received additional RFCA (including RFCA after the second TCEA procedure in 1 patient). Three of these 7 patients remained free of VT, and VT was improved in another 2 patients. A second TCEA procedure...
was performed in 2 patients 32 and 42 days after the first TCEA, respectively. Coronary angiography revealed that total occlusion of the vessel initially ablated was still present in both patients. In 1 patient who had initial occlusion of a first septal branch, the second septal branch was targeted for ablation. He had prior complete heart block. VT recurred after the second procedure, and further RF ablation was performed in the left ventricular outflow region but also failed (Figure 1). In the second patient, TCEA was performed in a third obtuse marginal branch after prior ablation in the second obtuse marginal branch. He subsequently remained free of VT.

Of 8 patients who underwent repeated electrophysiological study (during TCEA, 2; RFCA, 6) for recurrent VT, VT originated from the same region as was targeted at the prior TCEA in 7 patients (Table 5). In all patients, however, the recurrent VT had a different QRS morphology than observed at the first TCEA, suggesting that the substrate had been modified.

After a mean follow-up of 20±11 months after the last ablation procedure, total mortality was 32%, early mortality (within 30 days) was 14%, and late mortality (beyond 30 days) was 18%. Peak serum cardiac enzymes after TCEA were not statistically different comparing the patients with early mortality and late mortality (CK-MB, 78±47 versus 69±29 ng/mL, \( P=0.80 \); troponin I, 23±10 versus 13±8 ng/mL, \( P=0.36 \)). In univariate analysis, mortality was associated with a history of cardiac surgery (\( P=0.02 \)) and worse New York Heart Association functional class score (\( P=0.006 \)). Peak CK-MB and troponin I levels after TCEA procedure were not different between the 2 groups. At the end of follow-up, 13 patients who had TCEA were alive and free of VT or had improved control of VT.

**Discussion**

Although RFCA for recurrent VT due to structural heart disease usually reduces VT recurrences, it continues to fail in a significant number of patients. The presence of intramural or epicardial arrhythmogenic substrates is an important cause of failure for which ablation options are limited. This report demonstrates that TCEA can prevent recurrent VT and VT storm or control incessant VT in more than one half of such patients. These results are more impressive considering that TCEA was largely a last-resort therapy after previous failed ablation and drug therapy. However, there are a number of concerns and problems that these data highlight.

Based on ours and other series of ablation,5–7 we routinely consider TCEA when RFCA fails. However, use of percutaneous epicardial mapping and ablation has continued to increase. TCEA was considered before epicardial ablation in cases where a deep intramural substrate was suspected, as when there is earliest activation at the interventricular septum. Patients with cardiac surgery that renders pericardial access more difficult also would likely be considered for TCEA rather than a surgical epicardial ablation.

We anticipated that TCEA may be most useful for those septal VTs that do not have an endocardial origin and cannot be approached from the epicardium. Although the majority of target vessels identified were septal, TCEA was also feasible for some nonseptal VTs.

Planned TCEA was aborted in 19% patients usually because of anatomic constraints. Technical limitations to TCEA are more likely to be encountered in patients with multivessel coronary artery disease and prior coronary artery bypass graft surgery. Paradoxically, TCEA is more likely to be of interest in this subgroup because percutaneous epicardial mapping often is not possible after cardiac surgery. In some patients with ischemic heart disease, a coronary vessel perfusing the target region identified from catheter mapping could not be identified or was not accessible because it was perfused only by a network of multiple collateral vessels. We also observed that extensive collateral vessels from a possible target vessel can be present, perfusing more distant myocardium, which would presumably create a risk of larger infarction beyond the VT substrate. In such cases, we deferred ablation.

Anatomic variations are important considerations. Because the septum is the most common target region, septal perforators from the left anterior descending coronary artery were

---

**Table 5. Patients With Repeat Procedures**

<table>
<thead>
<tr>
<th>Case</th>
<th>VT Origin</th>
<th>Coronary Artery Injected</th>
<th>VT Termination by Saline Injection Test</th>
<th>Induction Test Postprocedure</th>
<th>Repeated Procedure</th>
<th>VT Origin</th>
<th>Therapy</th>
<th>Outcome Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Septal</td>
<td>First septal</td>
<td>Yes</td>
<td>Nonclinical VT</td>
<td>Septal*</td>
<td>RFCA</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>Septal</td>
<td>First septal</td>
<td>Yes</td>
<td>No VT</td>
<td>Septal*</td>
<td>Second TCEA for second septal</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>Posterior</td>
<td>Second OM</td>
<td>Yes</td>
<td>Nonclinical VT</td>
<td>Posterior*</td>
<td>Second TCEA for third OM</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Posterior</td>
<td>OM+ diagonal</td>
<td>Yes</td>
<td>Nonclinical VT</td>
<td>Posterior*</td>
<td>RFCA</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>Septal</td>
<td>Distal septal+diagonal</td>
<td>Yes</td>
<td>No VT</td>
<td>Septal*</td>
<td>RFCA</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>Septal</td>
<td>First septal</td>
<td>Yes</td>
<td>No VT</td>
<td>Septal*</td>
<td>RFCA</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>Septal</td>
<td>First septal</td>
<td>Yes</td>
<td>No VT</td>
<td>Septal*</td>
<td>RFCA</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>8</td>
<td>Septal</td>
<td>First septal</td>
<td>Yes</td>
<td>No VT</td>
<td>Distal septal inferior</td>
<td>RFCA</td>
<td></td>
<td>Yes</td>
</tr>
</tbody>
</table>

OM indicates obtuse marginal branch; RFCA, radiofrequency catheter ablation. Other abbreviation as in Table 1.

*Same as first TCEA.
important targets. In 1 patient, a right superior septal perforator from a conus branch of the right coronary artery was found and successfully occluded. An ectopic origination of the septal branch is infrequent, occurring in 3% of patients.13

What is the mechanism of VT recurrence? At repeat electrophysiological studies in 8 patients, the recurrent VT seemed to originate from the same general region targeted at the first TCEA in 88% of patients. In all of these patients, saline injection or interruption of blood flow in the targeted vessel had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

The mortality of 32% during 20±11 months of follow-up remains concerning in this sample. Although these patients had depressed ventricular function and uncontrollable VT, which is a marker for increased mortality and heart failure, before ablation, damage to contracting myocardium is a potential contributing factor. Single-point measurement of serum troponin I in the early phase after myocardial infarction performs well for estimation of final infarct size and is a well-established prognostic marker after myocardial infarction.15,16 In the present study, CK-MB and troponin I levels had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

The mortality of 32% during 20±11 months of follow-up remains concerning in this sample. Although these patients had depressed ventricular function and uncontrollable VT, which is a marker for increased mortality and heart failure, before ablation, damage to contracting myocardium is a potential contributing factor. Single-point measurement of serum troponin I in the early phase after myocardial infarction performs well for estimation of final infarct size and is a well-established prognostic marker after myocardial infarction.15,16 In the present study, CK-MB and troponin I levels had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

The mortality of 32% during 20±11 months of follow-up remains concerning in this sample. Although these patients had depressed ventricular function and uncontrollable VT, which is a marker for increased mortality and heart failure, before ablation, damage to contracting myocardium is a potential contributing factor. Single-point measurement of serum troponin I in the early phase after myocardial infarction performs well for estimation of final infarct size and is a well-established prognostic marker after myocardial infarction.15,16 In the present study, CK-MB and troponin I levels had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

The mortality of 32% during 20±11 months of follow-up remains concerning in this sample. Although these patients had depressed ventricular function and uncontrollable VT, which is a marker for increased mortality and heart failure, before ablation, damage to contracting myocardium is a potential contributing factor. Single-point measurement of serum troponin I in the early phase after myocardial infarction performs well for estimation of final infarct size and is a well-established prognostic marker after myocardial infarction.15,16 In the present study, CK-MB and troponin I levels had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

The mortality of 32% during 20±11 months of follow-up remains concerning in this sample. Although these patients had depressed ventricular function and uncontrollable VT, which is a marker for increased mortality and heart failure, before ablation, damage to contracting myocardium is a potential contributing factor. Single-point measurement of serum troponin I in the early phase after myocardial infarction performs well for estimation of final infarct size and is a well-established prognostic marker after myocardial infarction.15,16 In the present study, CK-MB and troponin I levels had terminated VT, and TCEA was acutely successful. It is possible that small collateral vessels developed over time in some14; however, VT recurred earlier than 24 hours in some patients. Persistent coronary occlusion without clear collaterals was observed in 2 patients who had repeat angiography after VT recurrence. Interestingly, the VT substrate did seem to be modified because the VTs that recurred had a different QRS morphology than the initially targeted VT. Thus, it seems likely that the lesion created by TCEA was insufficient to damage all of the VT substrate and that there may have been some recovery in the border region.

Complications
AV block is an anticipated complication when the septum is targeted for TCEA. Furthermore, the location of the arrhythmia substrate in this region and prior catheter ablation may impair AV conduction. Thus, almost two thirds of patients had some degree of AV block, and one half had bundle branch block. Complete heart block occurred in 38% of patients who underwent TCEA of the septum. TCEA for hypertrophic cardiomyopathy causes complete heart block in ≈11% of patients and is more likely if preexisting heart failure is present and if >1 septal artery is injected.17,18 In a previous study, Kay et al4 reported on 4 (36%) patients with complete heart block after 11 TCEA procedures for VT. Interestingly, heart block could not be predicted by balloon occlusion or saline infusion into the target vessel, suggesting that the area of injury produced by ethanol injection is larger than the area of ischemia produced by saline injection.

Limitations
This study has several limitations. These patients are largely referred to our tertiary center and undoubtedly are a selected group. Because the study sample is small, statistical power is very low and the chance of type I error may be very high because of small sample sizes in subgroups combined with multiple testing at a fixed 5% significance level. Further studies are required to better identify patient groups most likely to benefit and that are at risk for complications. To our knowledge, however, these data comprise the largest series of patients treated with TCEA. VTs were often documented by implantable cardioverter-defibrillator interrogation, and 12-lead ECG morphologies of spontaneous VTs were often absent. This descriptive study of TCEA was from an experienced center. It is important to note that these findings may not be applicable to less-experienced operators or centers.

Conclusions
In patients with difficult-to-control VT who fail RFCA, TCEA prevents all VT recurrences in 36% and improves arrhythmia control in an additional 27%. With attempted broader use, we found that inadequate target vessels, collaterals, and recurrence of modified VTs limit efficacy but that TCEA continues to play an important role for difficult VTs in high-risk patients with failed RFCA.

Sources of Funding
Dr Kojodjojo was funded by a British Heart Foundation Travel Fellowship (FS/09/047).

Disclosures
Dr Koplan is a consultant of Boston Scientific, St Jude Medical, and Sanofi Aventis. Dr John is a consultant of St Jude Medical and Spectranetics Inc. Dr Epstein is a consultant of Boston Scientific, St Jude Medical, and Medtronic. Dr Stevenson is a coholder of a patent for needle ablation consigned to Brigham and Women’s Hospital. Dr Tedrow is a consultant of St Jude Medical.

References


**CLINICAL PERSPECTIVE**

Catheter ablation of scar-related ventricular tachycardias (VTs) still fails in some patients, usually because of an inability to reach the arrhythmia substrate. Transcoronary ethanol ablation (TCEA) offers an alternative for these patients. We began offering TCEA routinely at our institution when endocardial and epicardial ablation fail or when a deep intramural substrate, such as in the septum, is anticipated. To our knowledge, this update provides the largest reported experience of TCEA for VT and further clarifies its limitations with present techniques. In 19% of patients, TCEA was considered but not performed because of unsuitable coronary anatomy. Of the 22 patients who received TCEA, VT recurrences were completely prevented in 36%, and arrhythmia control was improved in an additional 27%. Complete heart block occurred in 5 patients, and 1 patient with advanced heart failure died 5 days after the procedure. Thus, TCEA is a useful treatment option when catheter ablation fails but has significant limitations.
Transcoronary Ethanol Ablation for Recurrent Ventricular Tachycardia After Failed Catheter Ablation: An Update

Michifumi Tokuda, Piotr Sobieszczyk, Andrew C. Eisenhauer, Pipin Kojodjojo, Keiichi Inada, Bruce A. Koplan, Gregory F. Michaud, Roy M. John, Laurence M. Epstein, Frédéric Sacher, William G. Stevenson and Usha B. Tedrow

_Circ Arrhythm Electrophysiol._ 2011;4:889-896; originally published online October 7, 2011;
doi: 10.1161/CIRCEP.111.966283

_Circulation: Arrhythmia and Electrophysiology_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/4/6/889

Data Supplement (unedited) at:
http://circep.ahajournals.org/content/suppl/2011/10/07/CIRCEP.111.966283.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation: Arrhythmia and Electrophysiology_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation: Arrhythmia and Electrophysiology_ is online at:
http://circep.ahajournals.org//subscriptions/
Individual changes of left ventricular (LV) ejection fraction before and after transcoronary ethanol ablation (TCEA) are shown for patients who survived ablation and follow-up (left panel) and those who died early (within 30 days) or late (right panel).
Details of Post-Procedure Mortality

The first patient was a 60-year-old male with ischemic cardiomyopathy with prior coronary artery bypass surgery and stenting, and left ventricular (LV) ejection fraction 10 – 15% that had improved after biventricular implantable cardiac defibrillator placement. He was admitted with frequent episodes of polymorphic and monomorphic ventricular tachycardia (VT) with syncope despite amiodarone that had been increased to 800mg daily. Echocardiography (15 days prior to ablation) showed LV ejection fraction of 30%, with a 4.8 cm apical LV thrombus; LV ejection fraction on positron emission tomography imaging was 22%. Episodes of VT increased despite therapy with lidocaine, amiodarone, mexiletine, quinidine and metoprolol. Angiography showed no targets for revascularization. He was evaluated, but felt not to be an option for cardiac transplantation or destination left ventricular assist device. Due to the LV thrombus transcoronary ethanol ablation (TCEA) procedure of the distal left anterior descending artery (LAD) branch was performed without LV endocardial mapping, targeting a right bundle branch block
configuration VT with a cycle length of 340 ms. TCEA of the distal LAD abolished VT1, but other VTs remained inducible. After TCEA procedure he remained on intraaortic balloon counter pulsation and inotropic support. Episodes of VT continued with further hemodynamic deterioration. Subsequently comfort measures were instituted and antiarrhythmic therapy withdrawn as the family wished and he died 3 days after the TCEA.

Patient #2 was a 73-year-old female with prior anterior wall infarction, hypertension, aortic aneurysm, and coronary artery bypass surgery. She was transferred to us for management of recurrent monomorphic VT despite amiodarone and mexiletine and prior endocardial radiofrequency catheter ablation attempt. LV ejection fraction the day of the procedure was 35%. RF ablation at the infero-septal LV slowed and terminated VT, but VT remained inducible. TCEA of a septal branch of the apical LAD was performed after balloon catheter placement in the vessel terminated VT. She initially tolerated the procedure well, and had no VT, but over the next 24 hours developed oligura, hypotension, progressive acidosis, and
multi-organ failure subsequently attributed to cholesterol embolization syndrome, followed by death. Echocardiogram 1 day after the procedure showed no change in estimated LV ejection fraction of 35%.

Patient #3 was a 62-year-old female, with prior infero-septal myocardial infarction and heart failure, resection and primary closure of a mid inferior wall left ventricular pseudoaneurysm and placement of a biventricular assist device (BiVAD), followed by embolic strokes. She had continued to have episodes of VT and VF with acceptable, but diminished VAD flows. Ablation was requested in the hope of achieving some improvement in hemodynamic stability after VT and VF increased despite medical therapy with quinidine. Due to the recent BiVAD placement, there was concern about potential risk of embolization of thrombus. TCEA of the 1st septal branch of the LAD was performed without LV endocardial mapping. TCEA abolished VT, but her neurologic status and general condition failed to improve. She was transitioned to comfort measures only and died 28 days after the TCEA procedure.