Ventricular Tachycardia Ablation
Evolution of Patients and Procedures Over 8 Years

Frédéric Sacher, MD; Usha B. Tedrow, MD; Michael E. Field, MD; Jean-Marc Raymond, MD; Bruce A. Koplan, MD; Laurence M. Epstein, MD; William G. Stevenson, MD

Background—Evolving management of coronary artery disease, heart failure, and the use of implantable cardioverter-defibrillators impacts the characteristics of patients with recurrent ventricular tachycardia (VT). We investigated the substrate, procedure, and outcome evolution of all patients referred for VT ablation during the past 8 years.

Methods and Results—From 1999 to 2006, 493 consecutive patients (358 male, 57±16 years) underwent 623 VT ablations: 131 had no structural heart disease (SHD), 213 had ischemic cardiomyopathies (ICMP), and 149 had nonischemic cardiomyopathies (NICMP). Although the main substrate is ICMP, the proportion of NICMP has increased from 27% to 35% (P=0.06) from 1999–2002 to the 2003–2006. The procedure abolished or modified inducible VTs in ≥75% of patients in all groups, but abolition of all monomorphic VTs was achieved in 125 (83%) patients without SHD, 180 (65%) with ICMP, and 99 (51%) with NICMP (P<0.0001). During a mean follow-up of 3.3±2.4 years, no deaths occurred in patients without SHD, but 75 patients (35%) with ICMP and 26 patients (17%) with NICMP died after a median of 13 months. Multivariate Cox regression analysis found that age, ejection fraction, and need for preprocedural mechanical hemodynamic support predicted mortality.

Conclusions—The substrate causing VT in patients requiring ablation is evolving and determines the long-term outcome. In the setting of a normal heart, VT ablation is associated with a low risk of subsequent mortality, with no deaths occurring during a mean follow-up of >3 years. In contrast, in patients with SHD and recurrent VT, VT ablation can be helpful to suppress drug refractory VT, but long-term mortality remains significant. (Circ Arrhythmia Electrophysiol. 2008;1:153-161.)

Key Words: tachycardia • catheter ablation • mortality • ventricles

Evolving management of coronary syndromes and heart failure therapies in nonischemic and ischemic diseases impact the substrate causing ventricular tachycardia (VT) with structural heart disease (SHD). The predominant strategy for treating VT remains palliative, using antiarrhythmic drugs (AADs) and implantable cardioverter-defibrillators (ICDs). Even though ICDs improve survival and reduce sudden death in high-risk patients with SHD,1–3 10% to 20% of patients with ICDs experience “electrical storm” with repeated device therapies.4,5 Although AADs reduce the frequency of VT episodes, efficacy has been disappointing6,7 and side effects are an important problem.8 Radiofrequency (RF) catheter ablation is an option to control recurrent VT. Often it is used as a sole therapy in patients without SHD or in combination with an ICD and antiarrhythmic therapy in scar-related VT associated with SHD. Several investigators reported ablation outcomes for series of patients selected for having one predominant morphology of VT. Other studies reporting RF catheter ablation for VT considered only patients with one underlying cause, relatively small numbers of patients, or short follow-up periods.

Editorial see p 147
Clinical Perspective see p 161

The objectives of this study were to evaluate the evolution of the substrate associated with VT, the procedural evolution, and the long-term mortality of all patients with VT ablation during the last 8 years.

Methods

Study Population
From January 1999 to December 2006, 493 consecutive patients (358 [73%] male, 57±16 years) underwent 623 VT ablations at our institution. One hundred thirty-one had no SHD, 213 had ischemic cardiomyopathy (ICMP) with coronary artery disease defined as history of prior myocardial infarction or documented obstructive coronary artery disease, and 149 had a nonischemic cardiomyopathy (NICMP) (Table 1). Approximately 53% of these patients have also been reported in smaller cohorts investigating specific mapping and ablation methods.9–19 In all patients, at least one episode of VT

Received January 28, 2008; accepted May 1, 2008.
From Brigham and Women’s Hospital, Harvard Medical School, Boston, Mass.
Guest Editor for this article was Douglas P. Zipes, MD.
Correspondence to William G. Stevenson, Cardiac Arrhythmia Department, Cardiovascular Division, Brigham and Women’s Hospital, 75 Francis Street, Boston, MA 02115. E-mail frederic.sacher@chu-bordeaux.fr
© 2008 American Heart Association, Inc.
Circ Arrhythmia Electrophysiol is available at http://circep.ahajournals.org DOI: 10.1161/CIRCEP.108.769471

153
VT was stable, mapping continued during VT. If the circuit could not be identified, ablation was performed through the presumptive exit based on voltage mapping combined with pace mapping. In patients without SHD, a focal source for the arrhythmia was sought from a combination of activation mapping and pace mapping.

Ablation lesions were created with RF current with a maximum power of 50 W (EP Technologies Inc or Stockert, Biosense Webster Inc) by using various types of catheter ablation over the study period (4-mm or 8-mm standard tip, internally or externally irrigated tip catheters).

The acute outcome of the ablation procedure was defined as follows: success—no monomorphic VT was inducible (including VTs that had not been observed to occur spontaneously, often referred to as nonclinical); indeterminant—monomorphic VT was inducible but was different and usually faster than VTs induced at the beginning of the procedure, suggesting that the arrhythmia substrate has been modified, or the acute outcome could not be reliably determined because of the inability to reliably induce the arrhythmia, but no arrhythmia was provokable after ablation (particularly in patients without SHD); and failure—VT inducible at the beginning of the procedure remained present or inducible.

After ablation, AADs were reduced or discontinued depending on the substrate and the acute success of the procedure. We report only the major complications (resulting in long-term disability, requiring intervention, or prolonging hospital stay) including the life-threatening complications (with immediate or short-term risk of death).

### Data Collection and Follow-Up

Data were collected from a centralized system that contained complete records of all patients treated and followed at the Brigham and Women’s Hospital. These records provide a detailed history and diagnosis for all patients, including ablation report, emergency department visits, and outpatient visits, as well as data recorded during inpatient care. Patients local to the hospital were followed up in Brigham and Women’s Hospital clinics. Referring cardiologists were contacted for clinical follow-up of their patients. Mortality was assessed from the social security death index queried in October 2007.
Baseline characteristics of the patients were compared with the $\chi^2$ test for the categorical variables and with a $t$ test for the quantitative variable depending on the substrate. The event-free survival was graphically displayed according to the method of Kaplan and Meier, with unadjusted comparisons of mortality by the log-rank test. Univariate and multivariate Cox proportional hazards regression analyses were used to evaluate the contribution of cardiomyopathy type (ischemic or nonischemic), age, left ventricular ejection fraction (LVEF), acute outcome, complications related to the procedure, hemodynamic support, AADs, and number of VTs induced to mortality. A 2-sided $P$ value $<0.05$ was considered statistically significant. The authors had full access to the data, and take full responsibility for the integrity of the data.

### Results

#### Substrate and Clinical Presentation

Our idiopathic VT (no SHD) population was composed of 78 patients with typical right ventricular (RV) outflow tract VT, 12 with left ventricular (LV) outflow tract VT, 10 fascicular VT, 8 RV VT outside the outflow tract, 14 LV other than outflow tract or fascicular VT, and 9 epicardial VTs. Although the main substrate for VT due to SHD is ICMP, there has been a trend for an increasing proportion of NICMP from 27% to 35% ($P=0.06$) from 1999–2002 to 2003–2006 period (Figure 1).

Patients with ICMP had a mean of $2.2\pm0.9$ coronary vessels diseased. Their last myocardial infarction occurred $10\pm8.3$ years (median 9 years) before ablation. One hundred eight (51%) patients with ICMP had prior coronary artery bypass surgery, and 70 (33%) had prior percutaneous coronary intervention before ablation. Only 61 (29%) patients had not had a revascularization procedure, 31% during 1999–2002 versus 26% during 2003–2006 ($P=NS$). In ICMP, the predominant scar region was the inferior wall, but the proportion of patients with septal scars increased over time (Table 2).

NICMP substrates during this 8-year period, were idiopathic dilated cardiomyopathy ($n=106; 54%$), valvular heart disease ($37; 19%$), congenital heart disease ($14; 7%$), arrhythmogenic RV dysplasia ($30; 15%$), and sarcoidosis ($8; 4%$). Their mean LVEF was $39\pm16%$. In NICMP, the septum, inferior and lateral wall as well as perivalvular areas, and epicardium were almost equally involved with sites of low-voltage scar giving rise to VT (Table 2). In the 106 patients with idiopathic dilated cardiomyopathy, 46 patients had a predominant basal scar, 10 patients had a predominant apical scar, 21 patients had scar involving the mid left ventricle, and 16 patients had no scar. Accurate data on base versus apex location were not available in the 13 remaining patients.

Most of the patients with SHD ($288; 80%$) had an ICD before referral VT ablation and were referred for recurrent ICD therapies. In the 74 remaining patients with SHD and no ICD at presentation, 19 were referred for frequent recurrent nonsustained VT, 43 because of stable sustained VT and 12 because of VT causing syncope. In the 131 patients without SHD, 51 patients were referred for frequent recurrent non-

| Table 2. Evolution of the Patients Undergoing Ventricular Tachycardia Ablation Between 1999–2002 and 2003–2006 According to Substrate |
|---------------------------------|---------------------|---------------------|---------------------|---------------------|
| **Demographic, n (%)**          | 136 (48)            | 142 (42)            | &nbsp;              | 36 (13)              | 70 (21)              | &nbsp;              |
| **Age, y**                      | 68±11               | 65±11               | 0.02               | 52±16               | 58±14               | NS                 |
| **Sex (male), n (%)**           | 116 (85)            | 132 (93)            | 0.03               | 30 (83)             | 55 (79)             | NS                 |
| **LVEF, %**                     | 29±12               | 28±13               | NS                 | 30±11               | 32±12               | NS                 |
| **Scar area**                   |                     |                     |                    |                     |                     |                    |
| Procedure with voltage map      | 132                 | 134                 | 31                 | 66                  |                     |                    |
| Inferior scar, n (%)            | 96 (73)             | 80 (60)             | 0.02               | 10 (32)             | 16 (24)             | NS                 |
| Anterior scar, n (%)            | 30 (23)             | 40 (30)             | NS                 | 2 (6)               | 11 (16)             | NS                 |
| Septal scar, n (%)              | 43 (33)             | 66 (49)             | 0.004              | 7 (23)              | 18 (27)             | NS                 |
| Lateral scar, n (%)             | 31 (23)             | 38 (28)             | NS                 | 2 (6)               | 12 (18)             | NS                 |
| Apex, n (%)                     | 35 (27)             | 45 (34)             | NS                 | 7 (23)              | 8 (12)              | NS                 |
| LV outflow tract, n (%)         | 4 (3)               | 1 (0.7)             | NS                 | 7 (23)              | 14 (21)             | NS                 |
| Epicardial ablation, n (%)      | 7 (5)               | 13 (9)              | NS                 | 8 (26)              | 18 (27)             | NS                 |

LVEF indicates left ventricular ejection fraction; LV, left ventricle.
sustained VT, 55 for stable sustained VT, and 25 because of VT leading to syncope.

VT Characteristics and Procedure
Of the 623 VT ablation procedures, we had detailed data on VT characteristics available in 587 (94%) (139 [93%] for no SHD, 268 [96%] for ICMP and 180 [92%] for NICMP). A mean of 2.2±1.7 monomorphic VTs per procedure was induced. Of the 587 procedures, unstable VTs (requiring termination for hemodynamic compromise) alone were present in 131 (22%), stable VTs (monomorphic hemodynamically well tolerated) alone were seen in 183 (31%), and both were present in 273 (47%) procedures.

There were several electrophysiological differences according to the underlying heart disease. At the time of the procedure, patients with no SHD often had only premature ventricular contractions or nonsustained inducible VT (80 patients; 58%), compared with 6 (2%) and 24 (13%) of ICMP and NICMP patients, respectively (P<0.001). Very slow (<150 bpm) and very fast (>200 bpm VTs) ventricular contractions were more common in patients with SHD.

In 2003–2006, 78% of the VT ablations in patients with SHD were performed using a combination of substrate mapping, pace mapping, entrainment mapping, and activation mapping. Of note, epicardial ablations increased from 7% to 12% of procedures (P=0.04) from 1999–2002 to 2003–2006 period. Mechanical hemodynamic support (intraaortic balloon pump or assist device) was used in 21 procedures.

Table 3. Complications During the 48 Hours After Ventricular Tachycardia Ablation

<table>
<thead>
<tr>
<th>complication</th>
<th>No SHD n=4 (2.7%)</th>
<th>ICMP n=32 (11.5%)</th>
<th>NICMP n=12 (6.2%)</th>
<th>No. (%) n=48 (7.7%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major life-threatening complications</td>
<td></td>
<td></td>
<td></td>
<td>23 (3.7)</td>
</tr>
<tr>
<td>Tamponade-cardiac perforation</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>8 (1.3)</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>7 (1.1)</td>
</tr>
<tr>
<td>Acute respiratory distress</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4 (0.6)</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2 (0.3)</td>
</tr>
<tr>
<td>Stroke</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2 (0.3)</td>
</tr>
<tr>
<td>Other major complications</td>
<td></td>
<td></td>
<td></td>
<td>25 (4)</td>
</tr>
<tr>
<td>Transient ischemic event</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>3 (0.5)</td>
</tr>
<tr>
<td>Other embolic event</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2 (0.3)</td>
</tr>
<tr>
<td>Atrioventricular block</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>5 (0.8)</td>
</tr>
<tr>
<td>Local vascular complication</td>
<td>0</td>
<td>7</td>
<td>2</td>
<td>9 (1.4)</td>
</tr>
<tr>
<td>Groin hematoma (requiring blood transfusion)</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>5 (0.8)</td>
</tr>
<tr>
<td>Phrenic nerve injury</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1 (0.2)</td>
</tr>
</tbody>
</table>

SHD indicates structural heart disease; ICMP, ischemic cardiomyopathies; NICMP, non-ICMP.

In univariable Cox proportional hazards analysis, age and LVEF were among the predictors of mortality (Table 4). ICMP had a 2-fold (95% CI, 1.3 to 3.2) increased risk of mortality compared with NICMP. Other univariable risk factors for mortality were complications related to the procedure, the number of failed AADs, and the number of VTs induced during the procedure. This increased risk was explained by older age and lower ejection fractions among these patients. Age, LVEF, and mechanical hemodynamic support were independent predictors of mortality in a multivariable model (Table 4). For every year of age, there was a 4.3% mortality increase, and for every percent of ejection fraction increase, there was a 3.3% mortality decrease. Those patients requiring mechanical support pre- or periprocedurally had a 4-fold greater independent risk of mortality, with worse prognosis than those suffering a major procedural complication. Arrhythmic storm and incessant VT were not predictors of mortality in this population.
VT recurred after a median of 1 month in 29% of ICMP and in 39% of NICMP patients, and arrhythmic outcome was not available for 30 of these procedures (6%). Data for recurrent VT during late follow-up were not reliably obtainable for the patients without SHD because of the referral nature of the population.

**Discussion**

**Main Findings**

During a mean follow-up of 3.3±2.4 years, no deaths were seen after VT ablation in patients without SHD and long-term mortality was 35% for ICMP and 17% for NICMP. Age, LVEF, and necessity for mechanical hemodynamic support during the procedure were independent risk factors for mortality. During VT ablation, patients without SHD more often had only inducible premature ventricular contractions, whereas patients with ICMP and NICMP had sustained VTs that could be very slow or very fast. A combination of substrate mapping, pace mapping, activation mapping, and entrainment was predominantly used especially in patients with SHD. Although the main substrate for VT ablation is ICMP (especially with inferior scar, late after myocardial infarction), the proportion of patients with NICMP is increasing. These patients have more variable scar locations (Table 1).

**Long-Term Outcome and Predictors of Mortality**

VT in normal hearts (so called idiopathic VT) is generally thought to be benign, based on small historical series. In our study, there was no death with a follow-up of 4.2±2.2 years in this sizable observational group. For patients with SHD, VT ablation is a palliative therapy that can reduce the number of ICD shocks.23 However, the presence of shocks may indicate more severe disease.24 This concern is consistent with the mortality rate of 35% at 3 years in the ICMP group. Mortality was somewhat better in the NICMP group (18% at

**Table 4. Predictive Factors of Mortality in Patients With Cardiomyopathy Undergoing Ventricular Tachycardia (VT) Ablation**

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted</th>
<th>Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hazard Ratio</td>
<td>95% CI</td>
</tr>
<tr>
<td>Age &gt;62 y</td>
<td>2.508</td>
<td>1.622–3.877</td>
</tr>
<tr>
<td>LVEF &lt;30%</td>
<td>2.212</td>
<td>1.477–3.314</td>
</tr>
<tr>
<td>ICMP</td>
<td>2.054</td>
<td>1.315–3.209</td>
</tr>
<tr>
<td>Complications</td>
<td>1.956</td>
<td>1.419–2.698</td>
</tr>
<tr>
<td>Mechanical hemodynamic support</td>
<td>3.352</td>
<td>1.687–6.660</td>
</tr>
<tr>
<td>No. of failed AAD</td>
<td>1.213</td>
<td>1.019–1.444</td>
</tr>
<tr>
<td>No. of VT induced</td>
<td>1.184</td>
<td>1.060–1.321</td>
</tr>
<tr>
<td>Prior failed ablation</td>
<td>1.240</td>
<td>0.834–1.843</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; ICMP, ischemic cardiomyopathies; AAD, antiarrhythmic drugs. Cutoff value for age and LVEF were the median.
3 years), likely related, at least in part, to younger age and generally better LV function in the NICMP patients.

Arrhythmic storm has been shown to predict mortality in NICMP and ICMP. In our study, patients with incessant VT or electrical storm had similar outcomes to those with less frequent VT. It is possible that ablation improved outcome for patients with electrical storm or incessant VT, such that their prognosis was then similar to those with intermittent VT. There are also, however, substantial selection biases that influence our results. The sickest patients may not have been thought appropriate for VT ablation and not been referred. The nature and provocative factors for electrical storm also varies among different studies. In our population, ablation was not performed in patients with a secondary cause of arrhythmic storm (such as ischemia and metabolic disorders), whereas in the study of Exner et al, 65.4% of arrhythmic storms were attributed to 1 or more of these secondary causes.

**Procedure**

In patients with SHD, a combined approach (substrate mapping, pace mapping, and activation mapping) was usually used (Figure 3). When VT is stable, substrate mapping during sinus rhythm can be used to identify regions for further evaluation during VT, minimizing the time spent in VT. Brief entrainment is often possible, even for unstable VTs, to confirm the location of a reentry circuit, potentially allowing ablation with a smaller number of RF lesions than when ablation is guided only by substrate mapping.

Of note, the characteristics of VT, specifically the VT cycle length, were different depending on the substrate. Patients without SHD more often have only premature ventricular contractions at that time of the procedure with more difficulty inducing VT, possibly consistent with the nonreentrant mechanisms thought to be common in this population. Patients with ICMP have slower VT (cycle length > 400 ms) than those with NICMP probably because of the substantial extent of scar with slow conduction after myocardial infarction.

In patients with SHD, acute success with abolition of all inducible VTs (59%) was similar to previous reports (averaging respectively 38% to 75%). Despite the evolution of therapy, the acute procedural outcome is failure in 13% of our patients at our referral center and modified substrate (still inducible VT but different from those initially inducible) in 28% of procedures.

In the multicenter study of Calkins et al (using an internally irrigated catheter for ablation of mappable VT ablation in ICMP), ablation of all mappable VTs was achieved acutely in 75% of patients with an 8% risk of major complication. We observed roughly similar acute success rate and complication rates in a more diverse population of patients with SHD, including patients with unstable VTs. Both studies demonstrate that major complications are not negligible in this particularly sick population.

Failure may be the result of inaccurate mapping, inadequate lesion creation, or to the presence of deep intramural or epicardial arrhythmia substrate. Concerning the first item, many studies have been published in the last 10 years that have improved our ability to interpret electrograms and identify components of reentry circuits using entrainment and characterize entrainment, and to better define the substrate based on low-voltage areas, unexcitable electric scar, and electrograms. Concerning inadequate lesion formation, progresses have been made by using larger ablation electrodes and cooled-tip electrodes that increase lesion size. However, even with irrigated tip catheters ventricular lesions may not be transmural. It is clear that epicardial approaches are required for some patients.

Deep intramural circuits or foci may still be difficult to eliminate with available approaches. New technologies, such as needle-irrigated RF ablation, that allows deliver energy directly inside the myocardium, that are under investigation.
Substrate
In ICMP, there was often a substantial latency between infarction and the VT ablation because of occurrence of multiple, refractory VTs, with a median delay between the last myocardial infarction and VT ablation of 9 years. This observation suggests a role for continued late remodeling occurring after myocardial infarction. The recent observation from the Multicenter Automatic Defibrillator Implantation Trial-II that appropriate ICD therapy for VT or VF predicts increased mortality also suggests that arrhythmias are a marker for disease severity and possibly remodeling. Thus, the occurrence of frequent VT, resulting in referral for VT ablation may be a marker of a more advanced or malignant disease consistent with mortality rates in our post MI patients of 16%, 24%, and 35% at 1, 2, and 3 years after ablation.

There has been an increase in the proportion of patients with NICMP and VT compared with ICMP over the last 8 years. It is possible that more aggressive reperfusion strategies for acute myocardial infarction result in smaller infarcts and reduce the number of infarct survivors that eventually need VT ablation. It is also possible that more NICMP patients with VT are surviving because of a better medical therapy and a greater use of ICDs in that population. Changes in referral patterns may also be responsible for this trend. The recognition that ablation of these VTs can be challenging may lead to earlier referral to a tertiary center.

Clinical Implication
VT ablation may be considered as a reasonable first- or second-line therapy in patients with idiopathic VT that is symptomatic or sufficiently frequent to raise concern about causing depressed ventricular function. In patients with SHD, ablation can be useful to prevent or reduce recurrent episodes of VT, usually as an adjunctive therapy to an ICD. The risks are greater, and mortality remains significant after ablation in these patients with recurrent, drug refractory VT. Attention to optimizing treatment of the underlying disease, as well as controlling VT recurrences, seems prudent. A recent study suggested that outcomes may be better in patients undergoing ablation after initial presentation with VT. Further studies are needed to help define the role of ablation as the patient population and technologies continue to evolve.

Limitations
(1) Our findings are based on a retrospective observational analysis. Although we adjusted for potentially confounding difference, we cannot exclude that other factors have contributed to our findings. (2) Being a referral center for VT ablation, our population is selected and may be skewed toward a sicker VT ablation population, consistent with the relatively high proportion of patients who had prior ablation attempts. It is possible that long-term mortality would be better for ablation performed before failure of multiple antiarrhythmic medications. (3) Although our findings of no mortality in patients without SHD are reassuring, it should be appreciated that some myopathic processes, such as sarcoidosis and arrhythmogenic RV dysplasia can be subtle in their clinical manifestations and may escape detection. A careful search for underlying disease is warranted in these patients. (4) Patients without SHD often stop seeing their referring cardiologist during late follow-up, such that clinical follow-up for VT recurrences was not available for 71% of our patients without SHD. Use of the social security death index allowed mortality to be obtained for all patients. In SHD patients, follow-up was based on reports of ICD interrogations, which may have been incomplete. (5) Most of our SHD patients had ICDs that undoubtedly extend survival in this patient population. The impact of an ablation strategy on mortality either with or without an ICD cannot be assessed from our data. (6) The ablation and mapping technology used in an individual patient was influenced by its availability and uncontrolled patient and physician factors. We did not, therefore, attempt to compare different ablation technologies overall in this population.

Conclusions
In this large observational series, the proportion of patients with NICMP compared with ICMP with VT requiring ablation is increasing. Procedures targeting these substrates have evolved through the last 8 years with availability of epicardial mapping and common use of a combination of substrate mapping, pace mapping, and activation mapping.

In the setting of a normal heart, prognosis is excellent, with no deaths occurring over a mean follow-up >3 years. For patients with recurrent VT due to SHD, ablation is a palliative option, which suppresses or decreases drug refractory VT episodes, whereas long-term mortality remains significant.

Disclosures
We report for Dr Frederic Sacher an educational grant from Biosense Webster (modest). When these patients were studied, Dr Tedrow received research funding from Boston Scientific and Medtronic (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (modest), and St Jude Medical Inc (modest). Dr Bruce Koplan received speaking honoraria from Boston Scientific (modest), Medtronic (modest), and St Jude Medical Inc (modest). Dr Laurence Epstein was a consultant to Biosense Webster (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (modest), St Jude Medical Inc (modest), and Biosense Webster (modest). Dr William Stevenson was a consultant to Biosense Webster (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (Modest), St Jude Medical Inc (modest), and Biosense Webster (modest).

Funding sources
F.S. was supported by a grant from the French Federation of Cardiology.

References


CLINICAL PERSPECTIVE

Ventricular tachycardia (VT) ablation may be considered as a reasonable first-line therapy in patients with idiopathic VT that is symptomatic or sufficiently frequent to raise concern about causing depressed ventricular function. In these patients, prognosis is excellent after ablation, with no deaths occurring over a mean follow-up of 3 years. In patients with SHD, ablation can be useful to prevent or reduce recurrent episodes of VT, usually as adjunctive therapy to an ICD. Complications rate and mortality (35% for ischemic cardiomyopathy and 17% for nonischemic cardiomyopathy, with a 3.3 ± 2.4-yr follow-up) remain higher in this population. Age, LVEF, and necessity for mechanical hemodynamic support during the procedure were independent risk factors for mortality in this large observational studies. Attention to optimizing treatment of the underlying disease, as well as controlling VT recurrences, seems prudent. A recent study suggested that outcomes may be better in patients undergoing ablation after initial presentation with VT (Reddy V et al. N Engl J Med. 2007;357:2657–2665). Further studies are needed to help define the role of ablation, as the patient population and technologies continue to evolve.
Ventricular Tachycardia Ablation: Evolution of Patients and Procedures Over 8 Years
Frédéric Sacher, Usha B. Tedrow, Michael E. Field, Jean-Marc Raymond, Bruce A. Koplan, Laurence M. Epstein and William G. Stevenson

Circ Arrhythm Electrophysiol. 2008;1:153-161; originally published online January 1, 2008; doi: 10.1161/CIRCEP.108.769471
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 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/1/3/153

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/