REVIEW

# Ablation of Ventricular Tachycardia in Patients with Structural Heart Disease

KATJA ZEPPENFELD, M.D.* and WILLIAM G. STEVENSON, M.D.†
From the *Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands and †The Cardiovascular Division, Brigham and Women's Hospital, Boston, Massachusetts

*Catheter ablation is an important therapeutic option for controlling recurrent ventricular arrhythmias in patients with heart disease. Although implantable defibrillators are generally first line therapy in this patient population, a substantial number of patients require additional therapy with either antiarrhythmic drugs, ablation, or both. Studies of mapping and ablation have produced further insights into pathophysiologic mechanisms of these arrhythmias, which are now well characterized. The majority is due to reentry through regions of ventricular scar. Methods for identifying scar based on electrogram characteristics now allow arrhythmogenic areas to be targeted for ablation during stable sinus rhythm, such that ablation is often an option even when multiple and unstable ventricular tachycardia are present. Ablation failure can also be due to anatomical obstacles; however, methods for accessing the pericardial space for mapping and ablation and technological progress can be expected to further improve its efficacy. (PACE 2008; 31:358–374)*

***ablation, electrophysiology-clinical, mapping, VT***

## Introduction

The superiority of implantable cardioverter defibrillators (ICDs) to medical therapy in preventing sudden death in patients resuscitated from a life-threatening arrhythmia and their effectiveness in terminating many ventricular tachycardias (VTs) with antitachycardia pacing have fostered greater use of ICDs for high-risk patients.[1–4] However, several considerations support an important role for therapies that prevent episodes of VT. ICD trials for secondary prevention of sudden death excluded patients with stable VT, and ICDs do not reduce mortality compared to drug therapy in patients with relatively preserved ventricular function.[5,6] ICDs do not prevent VT and 39–70% of patients require additional antiarrhythmic therapy to reduce the number of arrhythmia episodes.[7–9] A significant number of tachycardia terminations necessitate shocks which reduce quality of life.[10,11] Furthermore, episodes of VT or ventricular fibrillation (VF) predict increased mortality and heart failure even when they are effectively treated by an ICD.[12,13] Therapies that prevent VT can be expected to improve quality of life for patients with ICDs. Whether they would translate into improvements in survival will require further study. Antiarrhythmic drug therapy has disappointing efficacy and adverse drug effects that may outweigh benefits. Catheter ablation also offers the potential for preventing VT recurrences. Interestingly, in one series of patients referred for ablation, Della Bella et al. found that the elimination of all inducible VTs predicted reduced risk of death from heart failure.[14]

## Guidelines

The recent American College of Cardiology (ACC)/American Heart Association (AHA)/ European Society of Cardiology (ESC) guidelines state that radiofrequency catheter ablation (RFCA) is useful as a palliative and adjunctive therapy to ICD implantation in patients who receive multiple ICD shocks due to drug refractory sustained VT (Class I, level of evidence C), in patients with bundle branch reentry (BBR) (Class I, C), and for incessant monomorphic VT after failed drug therapy (Class IIa).[15]

In patients with otherwise low risk of sudden cardiac death (SCD) who present with sustained VT (Class I) or nonsustained VT (Class IIa) for which drug therapy is ineffective or not tolerated, ablation is also an accepted therapy. Most of these patients have idiopathic VT in the absence of structural heart disease, for which ablation has a success rate exceeding 85%.[16] Ablation strategies for this entity will not be discussed in this review.

For patients with prior myocardial infarction (MI), but relatively preserved left ventricular (LV) ejection fraction (>40%), curative catheter

Disclosures: William G. Stevenson is consultant to Biosense Webster, Inc., with speaking honoria from Medtronic, Boston Scientific, and St. Jude Medical.

Address for reprints: Katja Zeppenfeld, M.D., Leiden University Medical Center, PO Box 9600, 2300RC Leiden, The Netherlands. Fax: 31-715248144; e-mail: k.zeppenfeld@lumc.nl

Received March 31, 2007; revised July 14, 2007; accepted September 14, 2007.

ablation may be even considered in lieu of ICD therapy (Class IIb).

Ablation of ectopic foci that trigger recurrent polymorphic VT is an acceptable strategy for controlling electrical storm (Class IIb).

This article will review the present methods and outcomes of catheter ablation of VT for patients with heart disease.

## Monomorphic VTs

The strategy for catheter mapping and ablation is determined by the type of VT and the underlying substrate. Repetitive ventricular activation from the same circuit or focus generates monomorphic VT. Between 1993 and 2002, most VTs considered for ablation were drug refractory, monomorphic, hemodynamically tolerable, and reproducibly inducible. These preconditions render them mappable in the electrophysiology laboratory such that an activation sequence could be defined from point by point mapping.[17–22] The critical regions could then be targeted for focal ablation.

However, most patients with VT and structural heart disease have unstable VTs due to hemodynamic intolerance or poor reproducibility.[23–25] In addition, multiple morphologies of VT are typically inducible. These challenges have led to a shift away from targeting single, mappable VTs to a substrate-based approach that targets broader regions containing the likely substrate causing VT without the need for mapping during VT.

## Scar-Related Reentrant VT

Reentry involving areas of ventricular scar due to prior MI is the most common cause of VT in patients with structural heart disease; most studies have focused on this patient population.[26,27] However, scars also occur in nonischemic cardiomyopathies.

In patients with scar-related VTs, areas of dense fibrosis and the valve annulae form regions of conduction block that often define reentry circuit borders and create intervening isthmuses, also referred to as channels, of surviving myocardial bundles.[28–30] Fibrosis between the surviving myocyte bundles functionally prolongs the pathway for impulse propagation creating slow conduction. In addition, cell-to-cell coupling may be diminished due to decreased gap junction density and altered connexin expression, contributing to slowed conduction through the scar and may create regions of functional conduction block.[28,31–33]

Propagation of excitation wavefronts through regions of slowed conduction and reentry circuit isthmuses does not usually contribute to the surface electrocardiogram (ECG) QRS configuration.[30,34] The QRS of the VT begins when the excitation wavefront emerges from the exit of an isthmus often located at the border of the scarred myocardium and propagates rapidly away from the scar to depolarize the remainder of the ventricles (Figs. 2 and 5). The wavefront may then propagate along the border of the scar (outer loop) to return to the entrance of the isthmus, or through a path within the scar (inner loop). Areas within the scar may be activated with conduction delay but are not participating in the specific reentry circuit (bystander regions). However, these bystander regions may content a critical part of a different reentry circuit. Multiple potential loops create figure of eight types of circuits (Fig. 5).

### Targeting Isthmuses in Stable Scar-Related VTs

Identification of a critical isthmus allows ablation with a small set of radiofrequency (RF) lesions.[22,23,35] Studies of these isthmuses have generally included patients with relatively slow VTs. Protected isthmuses in tolerated postinfarct VTs have an average length of 31 ± 7 mm (range: 18–41 mm) and a width of 16 ± 8 mm.[30] The range of 6–26 mm width may explain that an important number of VTs can be ablated with a small number of RF lesions. However, broader isthmuses require linear lesions.

### Activation Mapping during VT

The QRS complex begins when the excitation wavefront emerges from the isthmus rapidly depolarizing the remainder of the ventricles producing systole. Depolarization of the isthmus is presystolic, prior to the QRS onset. Isolated low amplitude, diastolic potentials (IP) separated from adjacent larger potentials are observed at 50% of isthmus sites and are markers for isthmuses where RF ablation can terminate VT.[35,36] These potentials are also seen, however, in bystander areas that may be dead end pathways, or channels that are not participating in the VT under evaluation.[35,36] Bystanders with IPs can be recognized by entrainment mapping (Fig. 2) or pacing from a remote site to dissociate the IP from the VT.[36]

IPs can also be far-field activation from depolarization of tissue remote from the ablation site that can also be recognized from entrainment.[37] A significant number of VTs can be interrupted by ablation at sites that do not have IPs, often at a reentry circuit exit.

### Entrainment Mapping

During entrainment of a reentrant VT, pacing slightly faster than the VT continuously resets the reentry circuit (Figs. 1 and 2). At sites in the reentry circuit, the postpacing interval (PPI) is equal to the revolution time through the circuit, which is the tachycardia cycle length (CL). The PPI increases with increasing conduction time from the

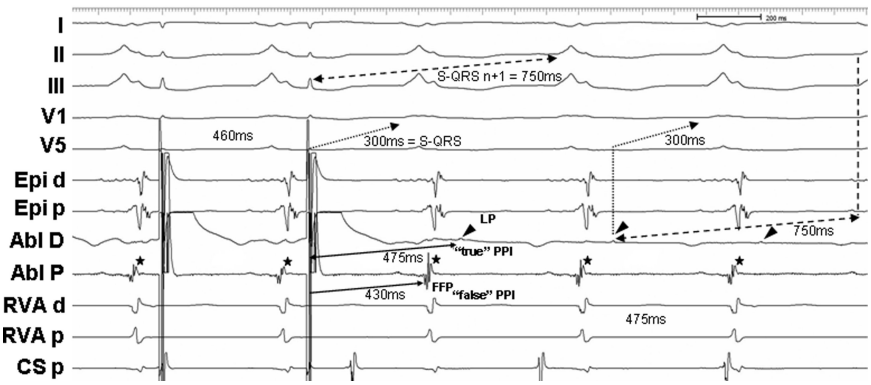


**Figure 1.** *Methods for assessment of potential errors during entrainment due to the presence of far-field potentials (FFP) are shown. From the top surface leads (I, II, III, $V_1$, $V_5$), bipolar intracardiac electrograms recorded from the distal (Epi d) and proximal (Epi p) electrode pairs of a mapping catheter in the pericardial space, from the distal (Abl D) and proximal (Abl P) electrode pairs of a mapping catheter on the endocardium, and from catheters placed in the right ventricle (RV) and coronary sinus (CS). During VT with a cycle length (CL) of 475 ms (QRS complex 4 and 5), two potentials are recorded from the distal (Abl D) and proximal (Abl P) electrode pair marked with an asterisk and an arrowhead, respectively. Unipolar pacing from the distal electrode of the ablation catheter at a CL of 460 ms entrains tachycardia with concealed fusion (QRS complex 1–3). The stimulus artifact obscures the smaller potential (arrowhead), indicating that this is likely the local potential (LP), whereas the larger potential (asterisk) remains visible and separate from the pacing stimulus, indicating that it is a FFP. The true postpacing interval (PPI) measured from the stimulus artifact to the LP equals the VT CL of 475 ms. Measuring the interval from the stimulus artifact to the FFP would result in a false short PPI of 430 ms (arrow). Comparing the S-QRS intervals to electrogram — QRS interval during entrainment can also be helpful in assessing whether the site is in the reentry circuit, particularly if the stimulus artifact obscures signals at the time of interest. The S-QRS (dotted arrow) is 300 ms, which equals the electrogram to QRS interval measured from the LP to the QRS onset of the fifth beat (dotted arrow). The S-QRS comparison is useful when fusion is concealed; however, small amounts of fusion can be difficult to detect. The second beat (QRS n + 1) is from the VT circuit and is not fused. Measurement of the S-QRS n + 1 is 750 ms. Assessing the relation of this interval to the LP signal (dashed arrow) reveals that the LP precedes the QRS onset of the n + 1 beat by that interval, consistent with a reentry circuit site. A remote intracardiac electrogram (e.g., from the right ventricle apex catheter) can also be used as a reference instead of the QRS.*

pacing site to the circuit. Interpretation is based on three fundamental assumptions. First, pacing does not alter the circuit path or initiate another VT. Second, conduction time through the circuit is the same during entrainment as during the VT. If the conduction slows during entrainment, the PPI prolongs. Third, and a common source of confusion, is the requirement that the electrogram selected for measuring the PPI indicates depolarization at the pacing site. Electrograms with multiple deflections are commonly recorded in scarred tissue. These potentials can be local signals from depolarization of tissue beneath the recording electrode, or far-field signals from depolarization of tissue adjacent or remote from the pacing site.[37] Far-field signals are common sources of a PPI that is shorter than the tachycardia CL (Fig. 1). They can often be recognized during pacing when they remain visible and separate from the stimulus artifact during entrainment. Local potentials from tissue directly depolarized by the pacing stimulus are not visible during pacing. The stimulus artifact obscures the potential produced in the tissue immediately after the stimulus and these potentials reappear after pacing.[37] With some recording systems, the recording amplifier does not recover for a second or more, obscuring all potentials on the distal electrodes, preventing measurement of the PPI from the distal electrode recording. Analyzing the signal from the proximal recording electrodes, when it is present on both proximal and distal electrodes, or measuring the n + 1 difference described by Soejima et al. can overcome this problem. The interval from the last stimulus that resets VT to the second beat after the stimulus is compared to the interval between the local electrogram during VT and the second beat after that electrogram. The difference between these two intervals, the n + 1 difference, correlates

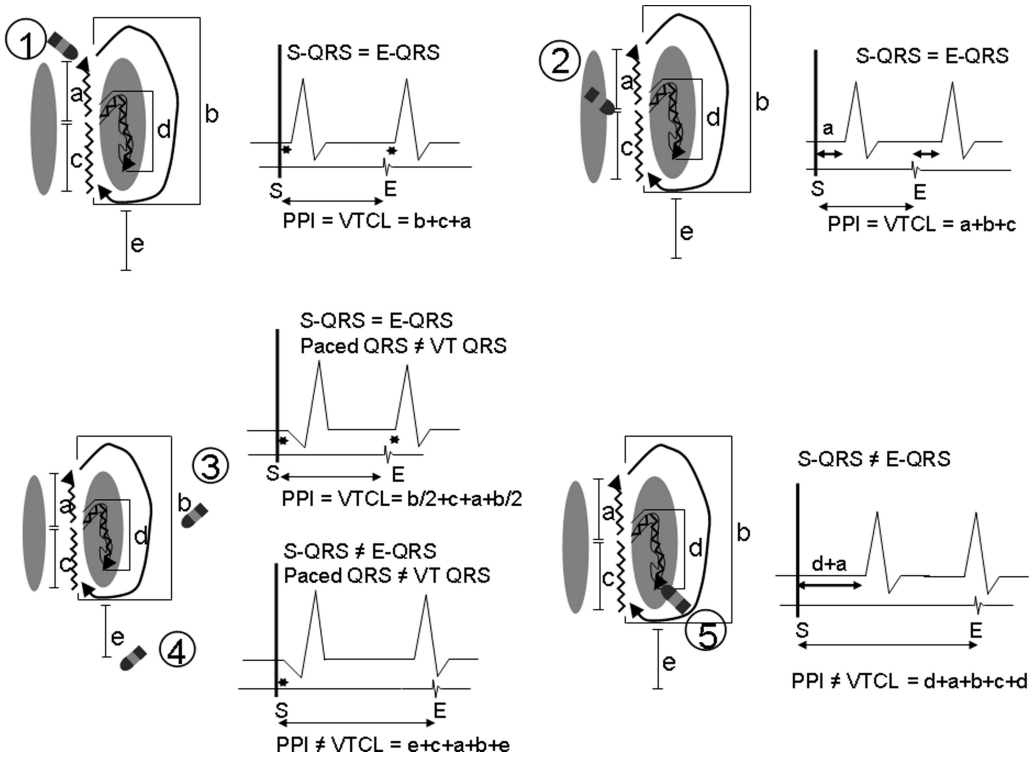


**Figure 2.** *Entrainment mapping — different responses at different sites. Schematic of a scar related reentry circuit. Grey areas represent scar, (a + c) represents the conduction time through the isthmus, (b) through the outer loop, (d) through a central bystander, and (e) to a remote bystander. Pacing is slightly faster as the VT cycle length resets (entrains) the VT reentry circuit. Pacing at an exit site (1) results in a short stimulus to QRS (S-QRS) interval identical to the local electrogram to QRS (E-QRS) interval. The paced QRS complex is identical to the VT QRS complex (concealed fusion) and the PPI approximates the revolution time through the circuit which is the conduction time through the outer loop (b) and the isthmus (a + c). At a central isthmus site (2), the S-QRS is prolonged and equals the E-QRS with identical QRS morphology, resembling the conduction time (a). At an outerloop site (3), the PPI is identical with the VTCL but the paced QRS complex differs from the VT QRS due to fusion. Pacing at a remote bystander (4) results in a different paced QRS complex. The PPI is prolonged by the propagation time (e) to and from the reentry circuit. Pacing at an adjacent bystander (5) results in concealed fusion with a S-QRS interval that is longer than the E-QRS interval, and a prolonged PPI interval due to the propagation time (d) to and from the circuit.*

well with the difference between PPI and ventricular tachycardia cycle length (VTCL) allowing entrainment mapping when the PPI cannot be measured.[38]

At reentry circuit isthmus sites, pacing entrains VT without changing the ventricular activation remote from the scar, producing entrainment with concealed fusion (a form of concealed entrainment) (Figs. 1 and 2). The stimulus to QRS interval (S-QRS) equals the conduction time from the pacing site to the reentry circuit exit, and is short (e.g., < 30% of the tachycardia CL) in the exit region, and longer at sites proximal to the exit (Fig. 2). At inner loop sites, the S-QRS may be very long, exceeding 70% of the VTCL. Outer loop sites are recognized from a PPI that approximates the VTCL, but QRS fusion is produced by propagation of stimulated antidromic wavefronts away from the scar border (Fig. 2). QRS fusion is often not detected when less than 22% of the QRS duration is from the antidromic wavefront.[39] Therefore, some outer loop sites are misidentified as exit or inner loop sites.

In one study, RF ablation with standard 4- or 5-mm electrodes terminated VT after an average of 10 seconds at 29% of 85 reentry circuit sites that met the combined criteria of entrainment with concealed fusion with a PPI–TCL difference <30 ms and S-QRS of less than 70% of the VTCL.[34] In 48 selected patients with slow VTs (CL491 ± 84 ms),

the combination of concealed entrainment with a S-QRS/E-QRS match ≤30 ms reached a sensitivity of 83% and a specificity of 84% for a successful ablation site.[22,40]

### Mechanical and Ablation Effects during VT

When catheter position is stable, RF current application heats ventricular tissue without producing propagated depolarizations. Termination of VT is then a sign that conduction has been interrupted in the reentry circuit, similar to cryomapping during cryoablation. At isthmus sites identified by entrainment, RF ablation typically terminates VT within 20 seconds.[34]

Mechanical trauma from the mapping catheter that terminates VT without eliciting propagated response is also an indication that the mapping catheter is at a reentry circuit isthmus site. When this occurs, VT may not be inducible for a variable period of time, preventing further mapping. Ablation at the site can be successful. Bogun et al. observed catheter-induced termination without premature beats, or abolishment of inducible VT during catheter placement at an endocardial site during mapping of 7 of 62 VTs. RF ablation at the site abolished targeted VT with no recurrence during 15 ± 11 months follow-up.[41]

Reproducible termination of VT by a pacing stimulus that does not produce a propagated response is also an indication that the pacing site is in a circuit isthmus. The stimulus likely captures locally, but the propagated impulse blocks before exiting the scar region and creates bidirectional conduction block in the reentry circuit.[41,42] Alternatively, the stimulus prolongs refractoriness at the site through an electrotonic effect.[43,44] This finding is specific for predicting a successful ablation site, but is observed infrequently, with a sensitivity of only 16%. Termination of VT by pacing stimuli without global ventricular capture was observed in 15 of 62 mapped VTs in one study and RF ablation at the site terminated all 15 VTs.[45]

### Outcomes of Ablation for Stable VT Due to Infarct Scars

Ablation targeting critical isthmuses for stable VTs is successful, abolishing the inducible "targeted" or "clinical" VT in 71–86% of selected patients.[14,17–21,36,46] During average follow-ups ranging from 9 to 41.5 months, 13–46% of patients experience a recurrence of nonfatal VT; the risk of SCD is low (0–6%), reflecting common use of ICDs for patients felt to be at risk. In patients with failure of ablation or another VT inducible, the 3- to 4-year risk of VT recurrence is much higher (60–64%) as compared to patients with no inducible monomorphic VT of any morphology (recurrence 14–20%).[14,46]

### Targeting Unstable Scar-Related VTs

Only 30% of patients referred for treatment of VT are suitable for ablation guided only by mapping during VT. Most have VT that is not tolerated hemodynamically to allow extensive mapping during the arrhythmia. VTs can also be unstable for mapping because of frequent change of morphology or inability to reproducibly induce the VT. Della Bella et al. found that unstable VTs were present in 57% of patients referred for catheter ablation.[14] In another series of 40 consecutive patients referred for VT ablation of postinfarct VT, 143 different VTs were inducible (3.6 ± 2.1 VTs per patient), but only 17.5% of patients had exclusively stable, tolerated VTs; a third of patients had only unstable VTs and the majority had both stable and unstable VTs.[23]

Unstable VTs can be approached using multielectrode mapping arrays that sample electrograms from multiple sites simultaneously during brief episodes of VT after deployment of the basket or balloon catheter in the ventricle.[47–49] An alternative approach attempts to identify the substrate causing the VT from mapping during stable sinus or paced rhythm (Fig. 4).

### Substrate Mapping—Lessons Learned from Surgery

Subendocardial resection of the arrhythmogenic substrate in the borderzone of the infarcted myocardium, encircling endocardial ventriculotomy, or encircling cryoablation with or without VT induction is highly effective in treating VT. Of 292 selected patients who underwent surgery for drug refractory VT, 56 (19.2%) were inducible after operation and only 6.2% experienced a spontaneous VT recurrence during a follow-up of 36 months. The operative mortality ranges from 3% to 14%.[50–55] In the operating room, the surgeon can identify the region of scar from visual inspection. Mapping during sinus rhythm (SR) in the operating room has shown that the areas of late activation and fractionated electrograms are often present in the infarct border and are likely involved in causing VT.[56,57] The application of these and other findings to catheter mapping has been facilitated by the development of mapping systems that enable electrophysiological information to be displayed on anatomic reconstructions of the ventricles.

### Voltage Maps to Identify Scars

The reduction of myocytes in regions of scar reduces the amplitude of recorded electrograms, allowing areas of scar to be recognized from the peak-to-peak electrogram voltage. In animal models and humans, infarct regions typically have bipolar electrogram amplitude <1.5 mV (recorded

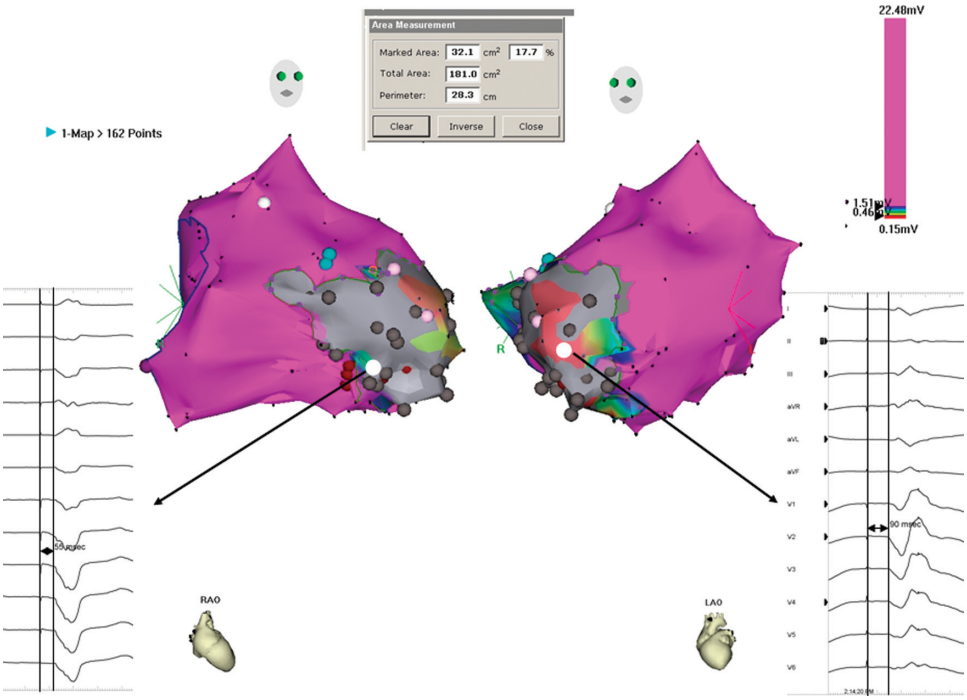


**Figure 3.** *Right anterior oblique (RAO) and left anterior oblique view (LAO) of an electroanatomical voltage map (CARTO, Biosense Webster, Inc.) of the left ventricle in a patient with a relatively small apico-septal myocardial infarction. Bipolar electrogram amplitudes are color coded according to the color bar. Normal voltage (amplitude: >1.5 mV) areas are displayed in purple. Although the surface of the infarcted area in this example is only 32 cm², the circumference is 28.3 cm². Successful ablation was achieved without completely encircling the scar by targeting sites (white tags) where pacing resulted in a stimulus to QRS delay of 55 and 99 ms, respectively, consistent with slow conduction and with a QRS morphology that matched that of VT. RF lesions (red tags) rendered the VT noninducible.*

with a 4-mm tip catheter with a 1-mm interelectrode spacing, filtered at 10–400 Hz).[24,25,58,59] Areas of scar or infarction can be shown graphically in three-dimensional anatomic maps by color coding the peak-to-peak electrogram amplitude, referred to as voltage maps. In patients with VT, these low voltage areas are generally large, ranging in size from 30 to 110 $cm^2$.[23–25] Ablation of the entire area or even its circumference is usually not practical. Additional criteria are used to subselect regions of the scar that contain reentry circuit exits or isthmuses for ablation (Figs. 3 and 4).

### Identifying Exit Regions with Pace-Mapping

Potential exit regions along the border of the low voltage region can be identified by pace-mapping. The 12-lead ECG of inducible VTs is recorded on the electrophysiology (EP) laboratory recording system. Pacing at sites along the border of the low voltage scar is performed to identify regions where the pace-map matches that of an induced VT (Fig. 3). Marchlinski et al. used pace-mapping to guide placement of lines of RF ablation lesions during SR in nine post-MI patients with a mean of 1.8 unstable VTs. After an average of 44 RF applications (range: 8–71) over a mean length of 16.2 cm, inducible VT was abolished in four of nine patients, one was not tested, and four had other, typically faster inducible VTs. Only one patient experienced a VT recurrence during a mean follow-up of 8.6 months.[24] Reddy et al. abolished all inducible VTs in 7/11 patients using a saline-irrigated tip catheter.[25] Pace-mapping is not a precise guide to the exit location. In some patients, a paced QRS complex resembles the VT QRS over a broad region.[60] Therefore, placement of a line of RF lesions, rather than a focal ablation is likely to be warranted. It is also important to recognize that a pace-map that does not resemble VT does not necessarily indicate that the site is remote from the reentry circuit.

If the reentry circuit is large, the pacing site may be distant from the exit. In particular, pacing at sites near the entrance to a channel may produce a completely different activation sequence

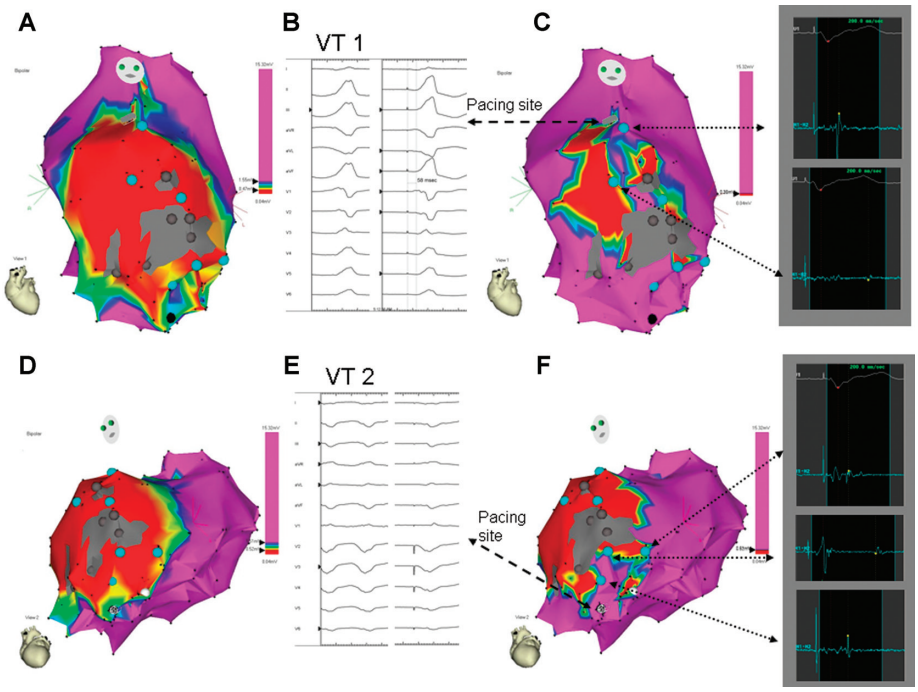


**Figure 4.** *Bipolar voltage maps and selected electrogram recordings showing a potential channel containing late potentials are shown. Bipolar voltage maps of the left ventricle constructed during RV pacing are shown in a modified AP view (A, C) and a modified LAO view (D, F). In panels A and D, voltage is color coded with the upper voltage threshold set at a commonly employed normal value of >1.5 mV, such that the areas exceeding this amplitude are displayed in purple. The lower amplitude threshold is set for <0.5 mV, such that these areas are red. In panel C, the upper voltage threshold has been reduced to 0.38 mV and the lower threshold to 0.28 mV. In panel F, the upper voltage threshold is 0.62 mV and the lower threshold is 0.52 mV. These changes in panels C and F expose potential channels of relatively greater amplitudes (which are now purple) between lower amplitude regions or electrically unexcitable scar (grey areas). Within these channels (dotted arrows) late potentials are recorded after the QRS complexes (right hand panels). Twelve-lead ECGs during pace-mapping near the exit from these channels (central panels) demonstrated a good match to VT 1 and VT 2, respectively, as shown.*

(and hence QRS morphology) compared to that of the tachycardia. During pacing at other isthmus sites, wavefronts also might emerge from the scar at different locations than the exit, producing a different QRS morphology than during VT.

### Recognizing Channels and Slow Conduction from Electrograms and Pace-mapping

During pace-mapping a delay of >40 ms between the stimulus and QRS onset is consistent with slow conduction away from the pacing site. A paced QRS that matches the VT with a long S-QRS delay is consistent with pacing in a potential reentry circuit isthmus.[23,61] If, however, an isthmus is defined by functional block, that is only present during VT, pace-mapping at these sites will likely produce a different activation sequence and QRS morphology than that of VT.

Pace-mapping can also detect some areas of electrically unexcitable scar, defined by a high pacing threshold that may form the border for a reentry circuit isthmus. Within low voltage regions, Soejima et al. defined electrical unexcitable scar (EUS) based on a unipolar pacing threshold >10 mA at 2-ms pulse width in 14 patients with prior infarction. EUS formed at least one border of 20 VTs reentry circuits identified by entrainment (n = 12) or pace-mapping. Ablation across the delineated isthmuses abolished all inducible VTs in 10 of 14 patients with marked reduction of spontaneous VT (from a mean of 142 ± 360 to 0.9 ± 2.0 episodes per month) during a follow-up of 168 ± 126 days.[62] Fibrotic areas that form boundaries of conducting channels are likely to have lower amplitude electrograms. Dense scar has been arbitrarily defined as sites with a peak-to-peak bipolar electrogram amplitude of <0.5 mV on voltage maps. However, with the variation in number and size of

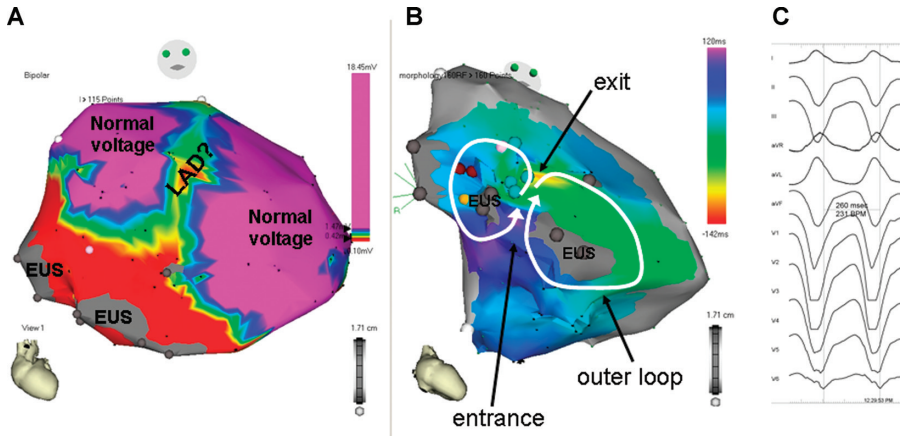


**Figure 5.** *(A) AP view of a epicardial electroanatomical voltage map (CARTO, Biosense Webster, Inc.) in a patient with no detectable structural heart disease based on available image modalities (such as Echo, MRI, LV and RV angiogram, and endocardial RV and LV voltage mapping) who presented with a monomorphic VT. Bipolar electrogram amplitudes are color coded according to the color bar. Normal voltage (amplitude: >1.5 mV) areas are displayed in purple. Epicardial mapping revealed a large low voltage area consistent with scar and findings consistent with scar-related macroreenty. The anterior small low voltage area that extends to the basis of the LV perhaps indicates the location of the left anterior descending artery (LAD). The grey EUS areas indicate electrical unexcitable scar (unipolar pacing with 10 mA and 2 ms impulse width). (B) Modified AP view (tilted and shifted to a more rightward and inferior projection) of the epicardial activation map during the clinical VT (CL 260 ms). Activation time is color-coded according to the corresponding color bar. The activation map demonstrates a figure-of-eight pattern of the VT with two wavefronts propagating around two areas of EUS. Linear RF lesion connecting these two areas of EUS rendered the VT noninducible. (C) Twelve-lead ECG of the VT.*

surviving bundles of myocytes that can form conducting channels, as well as the proximity of larger masses of surviving myocardium to channels, the electrogram amplitude in these regions can vary. Potential channels can be detected in some patients by individual adjustment of the voltage criterium for scar (Fig. 4). Arenal et al. systematically reduced the upper and lower voltage threshold in bipolar voltage (BV) maps starting at 0.51 and 0.5 mV, respectively, to expose channels of relatively greater amplitude between lower amplitude regions. A total of 23 channels were identified in 20 of 26 patients, 20 of them were related to at least one VT based on entrainment and pace-mapping. The majority of these channels could be defined by a scar definition of $\leq$0.2 mV. Detection of complete channels that connect normal voltage areas was more likely with a scar definition of $\leq$0.1 mV voltage and for patients with inferior MI. The channels were 23 $\pm$ 11 mm in length and 9 $\pm$ 3 mm in width.

Ablation in these channels abolished 88% of inducible VTs and 77% of patients remained free of VT during an average follow-up of 17 months.[63]

Using electrogram amplitude alone is not sufficient to identify some channels. Pacing at low amplitude sites to assess capture provides complimentary information to identify areas of dense fibrosis that are electrically unexcitable.[62] Soejima et al. observed that some isthmuses have very low amplitude electrograms (e.g., <0.5 mV) that would be classified as "dense scar" by some criteria. The average SR electrogram amplitude at isthmus sites was 0.32 $\pm$ 0.16 mV, but ranged from 0.08 to 0.91 mV; and 24% of excitable sites in infarct scars, where the pacing threshold was 10 mA or less, had very low amplitude (<0.25 mV) electrograms. Eight of 20 VT isthmuses would not have been evident using a definition of <0.25 mV for dense scar.

Potential channels and areas of slow conduction can also be detected from isolated late potentials (IP), inscribed after the end of the QRS during SR or right ventricular (RV) pacing (Fig. 4). Isolated delayed potentials, separated by $\geq$50 ms by an isoelectric interval or by very low amplitude signals, were targeted by Arenal et al. in 18 patients with unmappable VTs predominantly after MI. A mean of 13 $\pm$ 8 RF lesions abolished all inducible VTs in 13 of 18 patients (two patients were not tested and three remained inducible for fast VT). During a follow-up of 9 $\pm$ 4 months five patients experienced a VT recurrence.[64] Of interest, RV pacing increased the sensitivity for identifying late activated areas, demonstrating the effect of the direc-

tion of the activation wavefront on conduction in scar areas.

Studying patients with stable VTs, Bogun et al. found that the pace-maps at sites with isolated SR potentials (>20 ms separation) were more often good or perfect matches for the VT QRS, consistent with an exit region and that the S-QRS interval at these sites was longer than at sites with only abnormal/fragmented electrogram. The combination of these criteria was a good indicator of a critical isthmus site.[65]

### Integrating Substrate Mapping with VT Mapping

Substrate mapping can also be combined with limited mapping during VT, allowing regions of interest to be defined during stable SR. After inducing VT, evaluation of VT electrograms and entrainment, and potential ablation at the site during VT, can be restricted to the predefined regions and may be feasible even for poorly tolerated VT. Soeijima et al. used SR voltage mapping, pace-mapping, and limited entrainment mapping during VT to define a potential isthmus in 40 patients with stable and unstable VTs. Inducible VTs were abolished in 75% and modified in the remaining patients. Less ablation with shorter RF lines ($4.9 \pm 2.4$ vs $7.4 \pm 4.4$ cm) were required when an isthmus was identified compared to guiding placement of ablation lesions only by electrogram voltage and pace-mapping.[23] Kottkamp et al. achieved acute complete success (completely noninducible) in 79% of 28 patients using a similar strategy.[66] There were no major complications in either series.

These studies of substrate mapping are encouraging; although relatively small, patients are somewhat heterogeneous and different methods have not been directly compared. An approach that utilizes a variety of indicators of potential reentry circuit locations is evolving.

The potential for long fluoroscopy times and substantial radiation exposure to patients and operators is also a concern with extensive substrate modification approaches.[24] Remote navigation systems have been shown to allow substrate mapping with minimal fluoroscopy.[67] The enhanced maneuverability of the catheter might also facilitate mapping of difficult-to-reach areas. The mapping time is still significant (e.g., $84 \pm 44$ min; $48 \pm 18$ seconds per point for the LV) and the true usefulness of these systems will be better defined as irrigated, and large tip catheters are incorporated into so that ablation can be performed with the same system.

## Intramural and Epicardial Circuits

Scar-related reentry circuits commonly extend deep to the endocardium, although a portion is usually located on the endocardium. Absence of an adequate target on the endocardium, where catheter ablation can interrupt reentry, is a major cause of ablation failure. Circuits located deep within the septum accounted for 17% of VTs of patients who underwent endocardial and epicardial mapping during surgery.[68] Epicardial circuits are common in nonischemic dilated cardiomyopathy (DCM) and with inferior wall infarcts (see below).

### Increasing Ablation Lesion Size and Depth with Irrigation

Ablation of large reentry circuits that can extend deep to the endocardium is facilitated by technologies that increase ablation lesion size.[69] Active cooling of the ablation electrode with irrigation maintains a low electrode–tissue interface temperature during RF application which prevents coagulum formation and impedance rise, allowing greater energy delivery.[70] In animal models, irrigation increases RF lesion size by 30–50%.[71] Two different systems are available; a 4-mm tip internal saline irrigation (Chili, Boston Scientific, Inc., Natick, MA, USA) and a 3.5-mm tip externally irrigated catheter (ThermoCool, Biosense Webster, Inc., Diamond Bar, CA, USA). In animal models, the external irrigation system provides greater surface cooling with less risk of thrombus formation and charring than the internally irrigated system.[72] However, the external irrigation system administers a volume load to the patient which can precipitate pulmonary edema if not considered and managed.

Compared with standard RF, cooled RF is more effective for terminating VT, particularly at isthmus sites where an isolated potential was present (89% vs 54% termination sites).[69] Safety and efficacy of the internally irrigated catheter has been studied in a prospective multicenter trial of patients with VT due to structural heart disease.[73] Major complications occurred in 8%, including death in 2.7%, which is comparable with complication rates (5–12%) reported in VT ablation with standard catheters.[17–20,74] Thromboembolic complications and stroke occurred in 2.7% of patients.

### Intracoronary Ethanol Ablation

Selective intracoronary alcohol injection in a branch that likely supplies a critical part of the reentry circuit has been effective in treating uncontrollable VTs in a small number of patients. An adequate vessel cannot always be identified. The presumed extend of the MI has to be weighed up against the risk of surgery.[75–77]

### Epicardial Mapping and Ablation

Percutaneous transthoracic epicardial catheter mapping and ablation was introduced by Sosa et al. for treatment of epicardial VTs, initially in Chagas disease.[78] Ablation catheters can be introduced in the pericardial space by

a subxiphoid pericardial puncture. If access to the pericardial space is limited by adhesions from prior pericarditis or cardiac surgery, a direct surgical approach via a subxiphoid pericardial window can allow epicardial mapping and ablation.[79] Approximately 15% of patients, with recurrent VT late after MI, require epicardial ablation. Epicardial reentry circuits seem to be more common with inferior rather than anterior wall infarctions and in patients with nonischemic cardiomyopathy.[68,80,81]

Substrate and VT mapping approaches similar to those described for the endocardium have been employed, although some investigators have noted greater difficulty in achieving reliable epicardial capture for pace-mapping and entrainment mapping.[82–84] Limitations of pacing for pace-mapping and entrainment mapping in the epicardium may be due to freedom of catheter movement in the pericardial space, as well as the possible presence of epicardial fat, which is concentrated along the coronary sulcus and the interventricular grooves (Fig. 5). During surgery d'Avila et al. observed that more than 5 mm of epicardial fat produced a pacing threshold exceeding 10 mA.[85] Epicardial fat may also attenuate lesion formation. In an animal model standard, RF application over an epicardial fat layer with an average thickness of 3.1 mm resulted in no significant myocardial lesion, whereas ablation with a closed-loop irrigated tip catheter created lesions to a depth of 4.1 mm (45 ± 4.4 W).[86] Cooled tip ablation is likely to be more effective than standard RF ablation in the absence of convective electrode cooling in the pericardial space. Epicardial fat seemed to have less of an effect on electrogram amplitude; however, available data are inconsistent. In the animal model, BV electrograms exceeded 1.5 mV despite the presence of epicardial fat (mean thickness of the fat layer 2.6 ± 1.2 mm)[86]; therefore, voltage criteria alone might not be sufficient to delineate areas of epicardial fat.

Delineation of epicardial scar based on BV mapping seems to be feasible, but may have limitations due to the epicardial fat layer (Fig. 5). In a porcine model, 95% of normal, epicardial electrograms were >1.4 mV (4-mm tip catheter, Navistar, Biosense Webster, Inc.). In this study, sites with electrogram amplitude lower than 1.4 mV were concentrated near the base of the heart where epicardial fat is more abundant. Scars from healed MI corresponded well with bipolar electrogram amplitude <1.5 mV. Short electrogram durations of less than 50 ms were indicative of normal myocardium particularly at lower voltage sites near the atrioventricular (AV) groove.[87] However, low voltage regions due to fat rather than scar were demonstrated in a patient who underwent heart transplantation.[88]

In 14 patients with stable VTs after inferior MI, 39% (7/18) of all mappable VTs could be interrupted by epicardial ablation. Epicardial entrainment mapping was often difficult in this series due to a high stimulation threshold. Empiric thermal mapping (RF application for 10 sec to assess VT termination) was applied after localization of coronary arteries by coronary angiography. During follow-up all epicardial ablated patients remained asymptomatic.[82] Schweikert et al. ablated 8 of 10 VTs from the epicardium in seven post-MI patients and three DCM patients after unsuccessful endocardial irrigated RF ablation. Transient pericarditis occurred in three of 48 patients who underwent an epicardial approach; there were no major complications.[83] Brugada et al. found epicardial ablation successful in 80% of patients with incessant VT in whom endocardial ablation failed or was thwarted by LV thrombus or difficult vascular access.[84] During follow-up (18 ± 18 months), one patient experienced a VT recurrence.

Cesario et al. performed epicardial and endocardial mapping in eight patients (six MI, two DCM) with unstable VT, previous failed endocardial ablation, and a suspected infero-posterior scar or an ECG suggestive for an epicardial VT origin. Scar identified by voltage mapping was located inferior or posterior in all patients (six MI, two DCM). Voltage mapping (scar arbitrarily defined as <0.5 mV, borderzone defined as 0.5–1.5 mV) combined with pace-mapping in the borderzone was used to guide placement of extensive linear lesions (mean RF time: 118 min, range: 65–202 min) connecting scars or anatomical boundaries. After one year of follow-up, 75% of patients were free of VT.[88]

Epicardial ablation is often required for VT due to DCM. In 7 of 28 patients in whom endocardial ablation failed, low amplitude areas consistent with scar (<1.5 mV) were detected adjacent to a valve annulus in the basal LV (5), RV (2), RV outflow tract (3) and also at the lateral LV (2). Average epicardial low voltage scar area was larger than the endocardial scar area (37.5 ± 10.4 $cm^2$)[89] which is in contrast to 12 patients after MI who had an endocardial low voltage area that was three times greater than the epicardial low voltage area.[88] Using unipolar pacing isthmuses could be identified by entrainment or pace-mapping in six of seven patients. In three patients, slow conduction defined as S-QRS >40 ms was related to low voltage areas consistent with scar. Ablation was acutely successful in five of seven patients. In two patients, RF was limited due to the proximity of the phrenic nerve or coronary artery; three of the seven patients had recurrences during follow-up.

Complications of the epicardial approach include accidental right ventricular puncture with hemopericardium in 7% and transient pericarditis in 6% of patients.[78,82,83] Use of intrapericardial

glucocorticoids is under study for prevention of pericarditis. Rare cases of intraabdominal bleeding, likely due to inadvertent puncture of a subdiaphragmatic vessel, have occurred.[90] Phrenic nerve injury can probably be avoided by high output pacing to detect proximity to the nerve before RF delivery. A case of separating the nerve from the epicardium using a balloon catheter inflated in the pericardial space to overcome the limitation of ablation sites close to the phrenic nerve has been reported.[91]

Damaging epicardial coronary arteries during RF application is of concern. In animal models, standard RF application with a 4-mm tip electrode <1 cm from a coronary artery causes fibrosis of the media after 14 days, and can cause muscular hyperplasia and/or intravascular thrombosis. There was no arterial injury when RF were delivered more than 1 cm distant to the epicardial vessel. Larger vessels are less susceptible to injury than small vessels, likely related to greater cooling from blood flow.[85] Ablation in infarct-related areas is likely to involve territories of occluded infarct arteries. Of 215 patients in one series, one suffered MI from occlusion of a marginal artery.[90] However, the relevance of the possible media injury during very long-term follow-up is unclear and warrants continued investigation.

Cryoablation is an alternative to RF ablation that is not limited by absence of cooling blood flow in the pericardial space and is less prone to cause coronary artery injury in animal models.[92] Direct freezing on the epicardial coronary arteries in dogs can, however, cause neointima proliferation after 4–6 weeks, with a theoretical risk of late stenosis.[93] Further studies in humans seem warranted.

## Scar Related VTs in Specific Diseases

Most of the data discussed to this point is derived from patients with prior MI. The methods for mapping and ablation are also applicable to other disease associated with scar related VTs.

### Dilated Cardiomyopathy (DCM)

Only 14% of patients with DCM have grossly visible scar at necropsy, but interstitial or replacement fibrosis is present in the majority.[94] In explanted hearts, De Bakker et al. demonstrated that the discontinuous and circuitous conduction through zones of dense, patchy fibrosis produces slow conduction and fractionated electrograms and the substrate for reentrant VT.[95] Evidence of scar is often present in DCM patients undergoing ablation for VT. Hsia et al. found low voltage (<1.8 mV) areas (mean: $41 \pm 28$ cm$^2$) predominantly near the ventricular base and the perivalvular region in 19 patients. Of 57 VTs, 88% had exit sites near the basal regions. Ablation was acutely successful in 14 of 19 patients; however, only five patients were alive without VT recurrence after $22 \pm 12$ months.[96]

Kottkamp et al. reported eight patients with hemodynamically stable VTs, half with incessant VT. Ablation targeting fragmented, presystolic, or middiastolic activity in six patients and pace-map sites in two was acutely successful in six of nine targeted VTs. In six patients, VTs other than the targeted VTs were inducible after ablation and only two remained free of VT during $8 \pm 5$ month follow-up.[97]

Epicardial scar and reentry is often found when endocardial ablation fails. Using combined endocardial and epicardial approaches, Soejima et al. reported that 54% of patients with myocardial reentry due to DCM were free of VT during a follow-up of $334 \pm 280$ days in one series.[89]

The reasons for high recurrence rates despite good acute success are not completely clear; disease progression may contribute. Assessment of acute success may be hampered by unreliable reproducibility of VT induction in some patients.[97,98] Moreover, deep intramural or subepicardial circuits targeted from the endocardium may be more likely to recur after healing of ablation lesions.

### Arrhythmogenic Right Ventricular Dysplasia (ARVD)

ARVD is characterized by loss of right ventricular myocytes with replacement of fibro-fatty tissue beginning in the subepicardium and progressing to the subendocardium. Although predominantly located in the RV, the process can also involve the left ventricle.[99,100] These areas can be identified by low unipolar and bipolar electrogram amplitude using electroanatomical mapping techniques.[101–103] Low voltage areas are frequently localized adjacent to the lateral tricuspid annulus (TA), the right ventricular outflow tract (RVOT), and the apex which is in line with the anatomical findings of the disease.[102,104] Electrophysiolgic findings are usually consistent with reentry, although focal patterns of activation have been described.[103]

The macroreentrant reentrant circuits tend to be clustered around the TA and the RVOT,[103–107] whereas focal origin VT seemed to originating more often from the RV free wall,[103] perhaps representing endocardial breakthrough of an epicardial reentry circuit.[108] Catheter ablation based on entrainment mapping in stable VT was acutely successful for eight of 19 VTs in five patients in one series, failing in the VTs that originated from the body of the RV.[105]

Satomi et al. aimed to identify the entire reentry circuit using an electroanatomical mapping

system in 17 patients. In six of 13 mappable VTs, the critical slow conduction area was located between scars (defined by BV<1.5 mV) or between scar and TA. Abolishing these VTs sometimes requires linear lesions most likely due to a broad isthmus. Four VTs had a focal endocardial activation pattern, but could be entrained consistent with reentry.[104] Three VTs had multiple endocardial exits suggesting an epicardial circuit with multiple endocardial breakthrough sites.

Substrate mapping can be used to guide linear RF lesions in patients with otherwise unmappable VTs. Verma et al. encircled abnormal regions that showed good pace-maps or connected these areas to anatomical boundaries or adjacent scar areas (defined as BV <0.5 mV) and achieved acute success in 82% of 22 patients. There was one acute pericardial tamponade requiring drainage.[102] VT recurred in 47% of patients after 3 years follow-up.

In four reported series, overall recurrence during average follow-ups ranging from 6 to 50 months was 38% (range: 25–50%), perhaps due to the progressive disease.[102–104,109] Although the RV wall can be thin, ablation seems to require irrigated-tip or 8-mm tip catheters or epicardial ablation in some patients.[102] Perforation and tamponade does not appear to be markedly increased despite these anatomic considerations.

### Right Ventricular Cardiomyopathy

Marchlinski et al. reported on 21 patients with VT due to cardiomyopathy involving the RV, but with no family history of ARVD or SCD. Low voltage areas extending from the perivalvular regions but sparing the apex were observed. After VT ablation guided by activation and entrainment mapping in three patients with stable VTs and pace-mapping and application of linear lesions during SR in 16 patients with unmappable VTs, 17 of 19 patients were free of recurrent VT during 27 ± 22 months of follow-up.[110]

Although these patients would have largely met task force criteria for ARVD, the nonfamilial occurrence and the low recurrence rate suggest the possibility of a nongenetic origin (e.g., postinflammatory) or alternatively a spontaneous mutation with a different phenotype and perhaps less risk for disease progression than those with recognized familial ARVD. Long-term control of VT by ablation in this patient group seems to be more likely.

### Sarcoidosis

The anatomical feature of the disease is non-caseating granulomas, but areas of active granulomatous inflammation can be replaced by fibrosis, perhaps providing the substrate for reentrant VT. Cardiac involvement can occur without other detectable organ involvement.[111] Of 98 patients with nonischemic cardiomyopathy referred for catheter ablation of monomorphic VT, eight (8%) had histologically proven cardiac sarcoidosis. Low amplitude regions were present in the RV in all and in the LV in all six patients in whom LV mapping was performed and in the epicardium in one of two patients who had epicardial mapping. Ablation targeting 32 VTs abolished at least one inducible VT in six patients; two patients had no inducible VT, but six out of eight patients experienced a VT recurrence during follow-up.

### VT after Repair of Congenital Heart Disease

VT late after repair of congenital heart disease are usually related to reentry involving scars associated with ventricular incisions. Small series and anecdotal cases of catheter ablation have been reported for recurrent VT in patients with tetralogy of Fallot, double outlet right ventricle, ventricular septal defect (VSD) repair, transposition of the great arteries combined with VSD, and severe infundibular pulmonary stenosis.[112–114] Catheter ablation guided by activation and entrainment mapping can be successful, but reported results are variable in these small series with recurrence rates up to 40% during 3.8 years of follow-up.[114] Unstable VTs, noninducibility of VT, and complex anatomy contribute to failures. Three-dimensional electroanatomical mapping may facilitate ablation.[115–117]

### Bundle-Branch Reentry VT

Bundle-branch reentry VT is observed in 4.5–6% of patients with VT associated with coronary artery disease and in up to 16.7–41% of patients with monomorphic VT associated with various cardiomyopathies, myotonic dystrophy, and valvular heart disease.[118–120] It is possible that this VT is under-recognized with the increasing practice of ICD implantation without electrophysiologic study.

Most commonly the macroreentry occurs with propagation up the left bundle branch (LBB) and antegrade down the right bundle branch (RBB), giving rise to a left bundle branch block (LBBB) pattern VT. Evidence of His-Purkinje disease is usually present with interventricular conduction delay or LBBB, and His-ventricular (HV) prolongation (average of 75.3 ± 13.2 ms) is usually present but not required.[121–123] During VT a stable His-electrogram preceding each QRS and a HV-interval longer, equal to or less than 10 ms shorter than the HV-interval during SR, is typically seen. Oscillations of the interval between His or Right bundle potential anticipating ventricular CL changes confirm participation of the bundle branches in the tachycardia.[122] Entrainment of the VT from the RV

apex with a PPI-TCL of less than 30 ms is suggestive for BBR-VT.[124]

Catheter ablation of the RBB is curative; however, other VTs that are scar-related are frequently inducible and ICD implantation is often warranted. Marked prolongation of the HV interval >90 ms warrants pacemaker (or ICD) implantation in 20–30% of patients. LBB ablation might have a lower risk of AV block in some patients but may be followed by LV dyssynchrony requiring implementation of biventricular pacing.

### Beyond Myocardial Scar? Targeting Triggers of Polymorphic VT

In 1994, Leenhardt et al. reported a "short coupled variant of torsade de pointes (TdP)" in 14 patients with structural normal hearts.[125] Subsequently, Haissaguerre et al. demonstrated in a similar group of patients that the short coupled premature ventricular contractions (PVCs) could be targeted for ablation to abolish recurrent episodes of polymorphic VT and VF. In 23 of 27 patients, the initiating beat was identical to preceding premature ventricular beats with a short CI of 280 ± 26 ms and often originating from the distal Purkinje system. In four patients, the premature beats originated in the RVOT with a coupling interval of 355 ± 30 ms. Ablation of these initiating PVC foci eliminated recurrent VF during follow-up of 24 ± 28 months in 24 patients.[126] Focal PVC triggers can also play a role in Brugada Syndrome and long QT syndrome and successful control of arrhythmia storms by ablation has been reported.[127] VF episodes tended to cluster in time with long periods of quiescence, months or years later. Successful mapping and ablation requires prompt performance during a period of arrhythmia exacerbation, and all patients should receive an ICD until extensive long-term follow-up data are available.

The recognition that RV outflow tract PVCs can trigger VF in some susceptible patients has raised concern regarding the ability to distinguish benign from malignant RV outflow tract arrhythmias.[128]

Benign RVOT PVC/VT seem to have a longer CI (mean 0.44 ± 0.08; range: 0.32–0.6) than short coupled RVOT VT (mean: 0.35 ± 0.02; range: 0.32–0.40), which have been associated with malignant polymorphic VT and idiopathic VF (mean: 0.3 ± 0.04; range: 0.22–0.44).[129] In the largest reported series, however, including 16 patients with polymorphic VT or VF originating from the RV outflow tract, the frequency of isolated PVCs and their coupling intervals were not different to those observed in 85 patients with idiopathic monomorphic RV tachycardia. Patients with VF or polymorphic VT had more often a history of syncope (69% vs 18%) and the CL of nonsustained VT on Holter monitoring was significantly shorter (245 ± 28 ms vs 328 ± 65 ms). Ablation abolished all PVCs in 13 and the targeted PVC in three patients with no recurrence of VF during a follow-up of 54 ± 39 months.[128]

Acute MI complicated by recurrent polymorphic VT/VF despite betablocker, amiodarone and in the absence of ongoing ischemia is rare, occurring in only four out of 2340 acute infarctions.[130] These tachyarrhythmias may be triggered by monomorphic PVCs, perhaps arising from Purkinje fibers close to the infarct borderzone. Targeting PVC sites of presystolic sharp potentials, consistent with Purkinje origin abolished recurrent VT in one small series of four patients. Recurrent polymorphic VT after infarction may have a similar mechanism. Marrouche et al. reported on 29 patients with an ischemic cardiomyopathy after remote MI (>6 month) who experienced VF storm initiated by monomorphic PVCs (CI: 195 ± 45 ms); eight who were refractory to medical treatment underwent mapping and ablation. Purkinje-like potentials (PLP) preceding the PVCs located in the borderzone of the infarct were identified in five patients; in the remaining three ablations were guided by the presence of Purkinje-like potentials during SR. Following ablation, VF storm subsided in all patients; one had a recurrence of VF during a follow-up of 10 ± 6 months.[131] Ablation targeting triggering PVCs that appeared to originate from the Purkinje system has also been successful in controlling recurrent VT/VF in rare patients with cardiac amyloidosis[132] and fulminant myocarditis.[133]

### Summary

VT ablation is an important palliative and adjunctive therapy to ICD and medical therapy for control of recurrent ventricular tachyarrhythmias. Medical therapies that extend survival in patients with depressed ventricular function, including ICDs that reduce sudden death, are likely to increase the need for this therapy. Most of these patients have scar-related arrhythmias. Advances in knowledge and technology of mapping and ablation such as 3D-electroanatomical mapping, irrigated tip catheter, and nonsurgical catheter access to the epicardial space have improved acute success rates and have extended the indication to what were formerly labeled "unmappable" VTs, such as those that are hemodynamically unstable, as well as to VTs with a subepicardial substrate.

The recognition of triggers for polymorphic VT and VF that can be targeted for ablation has further advanced the catheter ablation frontier. Ablation failure is often due to an anatomic problem; such as an intramural or epicardial substrate, but methods are evolving that are increasing success with these challenges.

## References


1. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. N Engl J Med 1997; 337:1576–1583.
2. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, Mitchell LB, et al. Canadian implantable defibrillator study (CIDS): A randomized trial of the implantable cardioverter defibrillator against amiodarone. Circulation 2000; 101:1297–1302.
3. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: The Cardiac Arrest Study Hamburg (CASH). Circulation 2000; 102:748–754.
4. Higgins SL. Impact of the Multicenter Automatic Defibrillator Implantation Trial on implantable cardioverter defibrillator indication trends. Am J Cardiol 1999; 83:79D–82D.
5. Connolly SJ, Hallstrom AP, Cappato R, Schron EB, Kuck KH, Zipes DP, Greene HL, et al. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trials. AVID, CASH and CIDS studies. Antiarrhythmics vs Implantable Defibrillator study. Cardiac Arrest Study Hamburg . Canadian Implantable Defibrillator Study. Eur Heart J 2000; 21:2071–2078.
6. Domanski MJ, Sakseena S, Epstein AE, Hallstrom AP, Brodsky MA, Kim S, Lancaster S, et al. Relative effectiveness of the implantable cardioverter-defibrillator and antiarrhythmic drugs in patients with varying degrees of left ventricular dysfunction who have survived malignant ventricular arrhythmias. AVID Investigators. Antiarrhythmics Versus Implantable Defibrillators. J Am Coll Cardiol 1999; 34:1090–1095.
7. Fahy GJ, Sgarbossa EB, Tchou PJ, Pinski SL. Hospital readmission in patients treated with tiered-therapy implantable defibrillators. Circulation 1996; 94:1350–1356.
8. Pacifico A, Hohnloser SH, Williams JH, Tao B, Saksena S, Henry PD, Prystowsky EN. Prevention of implantable-defibrillator shocks by treatment with sotalol. d,l-Sotalol Implantable Cardioverter-Defibrillator Study Group. N Engl J Med 1999; 340:1855–1862.
9. Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES, Thorpe K, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: The OPTIC Study: A randomized trial. JAMA 2006; 295:165–171.
10. Miller JM, Hsia HH. Management of the patient with frequent discharges from implantable cardioverter defibrillator devices. J Cardiovasc Electrophysiol 1996; 7:278–285.
11. Schron EB, Exner DV, Yao Q, Jenkins LS, Steinberg JS, Cook JR, Kutalek SP, et al. Quality of life in the antiarrhythmics versus implantable defibrillators trial: Impact of therapy and influence of adverse symptoms and defibrillator shocks. Circulation 2002; 105:589–594.
12. Goldenberg I, Moss AJ, Hall WJ, McNitt S, Zareba W, Andrews ML, Cannom DS. Causes and consequences of heart failure after prophylactic implantation of a defibrillator in the multicenter automatic defibrillator implantation trial II. Circulation 2006; 113:2810–2817.
13. Moss AJ, Greenberg H, Case RB, Zareba W, Hall WJ, Brown MW, Daubert JP, et al. Long-term clinical course of patients after termination of ventricular tachyarrhythmia by an implanted defibrillator. Circulation 2004; 110:3760–3765.
14. Della BP, De Ponti R, Uriarte JA, Tondo C, Klersy C, Carbucicchio C, Storti C, et al. Catheter ablation and antiarrhythmic drugs for haemodynamically tolerated post-infarction ventricular tachycardia; long-term outcome in relation to acute electrophysiological findings. Eur Heart J 2002; 23:414–424.
15. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, Gregoratos G, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: A report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). J Am Coll Cardiol 2006; 48: e247–e346.
16. Joshi S, Wilber DJ. Ablation of idiopathic right ventricular outflow tract tachycardia: Current perspectives. J Cardiovasc Electrophysiol 2005; 16(Suppl. 1):S52–S58.
17. Stevenson WG, Friedman PL, Kocovic D, Sager PT, Saxon LA, Pavri B. Radiofrequency catheter ablation of ventricular tachycardia after myocardial infarction. Circulation 1998; 98:308–314.
18. Rothman SA, Hsia HH, Cossu SF, Chmielewski IL, Buxton AE, Miller JM. Radiofrequency catheter ablation of postinfarction ventricular tachycardia: Long-term success and the significance of inducible nonclinical arrhythmias. Circulation 1997; 96:3499–3508.
19. Gonska BD, Cao K, Schaumann A, Dorszewski A, von zur MF, Kreuzer H. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: Results and long-term follow-up. J Am Coll Cardiol 1994; 24:1506–1514.
20. Kim YH, Sosa-Suarez G, Trouton TG, O'Nunain SS, Osswald S, McGovern BA, Ruskin JN, et al. Treatment of ventricular tachycardia by transcatheter radiofrequency ablation in patients with ischemic heart disease. Circulation 1994; 89:1094–1102.
21. Morady F, Harvey M, Kalbfleisch SJ, el Atassi R, Calkins H, Langberg JJ. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. Circulation 1993; 87:363–372.
22. El Shalakany A, Hadjis T, Papageorgiou P, Monahan K, Epstein L, Josephson ME. Entrainment/mapping criteria for the prediction of termination of ventricular tachycardia by single radiofrequency lesion in patients with coronary artery disease. Circulation 1999; 99:2283–2289.
23. Soejima K, Suzuki M, Maisel WH, Brunckhorst CB, Delacretaz E, Blier L, Tung S, et al. Catheter ablation in patients with multiple and unstable ventricular tachycardias after myocardial infarction: Short ablation lines guided by reentry circuit isthmuses and sinus rhythm mapping. Circulation 2001; 104:664–669.
24. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. Circulation 2000; 101:1288–1296.
25. Reddy VY, Neuzil P, Taborsky M, Ruskin JN. Short-term results of substrate mapping and radiofrequency ablation of ischemic ventricular tachycardia using a saline-irrigated catheter. J Am Coll Cardiol 2003; 41:2228–2236.
26. de Bakker JM, Coronel R, Tasseron S, Wilde AA, Opthof T, Janse MJ, van Capelle FJ, et al. Ventricular tachycardia in the infarcted, Langendorff-perfused human heart: Role of the arrangement of surviving cardiac fibers. J Am Coll Cardiol 1990; 15:1594–1607.
27. de Bakker JM, van Capelle FJ, Janse MJ, van Hemel NM, Hauer RN, Defauw JJ, Vermeulen FE, et al. Macroreentry in the infarcted human heart: The mechanism of ventricular tachycardias with a "focal" activation pattern. J Am Coll Cardiol 1991; 18:1005–1014.
28. de Bakker JM, van Capelle FJ, Janse MJ, Tasseron S, Vermeulen JT, de Jonge N, Lahpor JR. Slow conduction in the infarcted human heart. 'Zigzag' course of activation. Circulation 1993; 88:915–926.
29. Wilber DJ, Kopp DE, Glascock DN, Kinder CA, Kall JG. Catheter ablation of the mitral isthmus for ventricular tachycardia associated with inferior infarction. Circulation 1995; 92:3481–3489.
30. de Chillou C, Lacroix D, Klug D, Magnin-Poull I, Marquie C, Messier M, Andronache M, et al. Isthmus characteristics of reentrant ventricular tachycardia after myocardial infarction. Circulation 2002; 105:726–731.
31. Kawara T, Derksen R, de Groot JR, Coronel R, Tasseron S, Linnenbank AC, Hauer RN, et al. Activation delay after premature stimulation in chronically diseased human myocardium relates to the architecture of interstitial fibrosis. Circulation 2001; 104:3069–3075.
32. Kostin S, Rieger M, Dammer S, Hein S, Richter M, Klovekorn WP, Bauer EP, et al. Gap junction remodeling and altered connexin43 expression in the failing human heart. Mol Cell Biochem 2003; 242:135–144.
33. Peters NS, Green CR, Poole-Wilson PA, Severs NJ. Reduced content of connexin43 gap junctions in ventricular myocardium from hypertrophied and ischemic human hearts. Circulation 1993; 88:864–875.
34. Stevenson WG, Friedman PL, Sager PT, Saxon LA, Kocovic D, Harada T, Wiener I, et al. Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. J Am Coll Cardiol 1997; 29:1180–1189.
35. Kocovic DZ, Harada T, Friedman PL, Stevenson WG. Characteristics of electrograms recorded at reentry circuit sites and bystanders during ventricular tachycardia after myocardial infarction. J Am Coll Cardiol 1999; 34:381–388.
36. Bogun F, Bahu M, Knight BP, Weiss R, Paladino W, Harvey M, Goyal R, et al. Comparison of effective and ineffective target sites that demonstrate concealed entrainment in patients with coronary artery disease undergoing radiofrequency ablation of ventricular tachycardia. Circulation 1997; 95:183–190.

37. Tung S, Soejima K, Maisel WH, Suzuki M, Epstein L, Stevenson WG. Recognition of far-field electrograms during entrainment mapping of ventricular tachycardia. J Am Coll Cardiol 2003; 42:110–115.
38. Soejima K, Stevenson WG, Maisel WH, Delacretaz E, Brunckhorst CB, Ellison KE, Friedman PL. The N + 1 difference: A new measure for entrainment mapping. J Am Coll Cardiol 2001; 37:1386–1394.
39. Ormaetxe JM, Almendral J, Martinez-Alday JD, Villacastin JP, Arenal A, Pastor A, Echeverria T, et al. Analysis of the degree of QRS fusion necessary for its visual detection: Importance for the recognition of transient entrainment. Circulation 1997; 96:3509–3516.
40. Bogun F, Kim HM, Han J, Tamirissa K, Tschopp D, Reich S, Elmouchi D, et al. Comparison of mapping criteria for hemodynamically tolerated, postinfarction ventricular tachycardia. Heart Rhythm 2006; 3:20–26.
41. Bogun F, Good E, Han J, Tamirisa K, Reich S, Elmouchi D, Igic P, et al. Mechanical interruption of postinfarction ventricular tachycardia as a guide for catheter ablation. Heart Rhythm 2005; 2:687–691.
42. Garan H, Ruskin JN. Localized reentry. Mechanism of induced sustained ventricular tachycardia in canine model of recent myocardial infarction. J Clin Invest 1984; 74:377–392.
43. Ruffy R, Friday KJ, Southworth WF. Termination of ventricular tachycardia by single extrastimulation during the ventricular effective refractory period. Circulation 1983; 67:457–459.
44. Shenasa M, Fromer M, Borggrefe M, Breithardt G. Subthreshold electrical stimulation for termination and prevention of reentrant tachycardias. J Electrocardiol 1992; 24(Suppl.):25–31.
45. Bogun F, Krishnan SC, Marine JE, Hohnloser SH, Schuger C, Oral H, Pelosi F, et al. Catheter ablation guided by termination of postinfarction ventricular tachycardia by pacing with nonglobal capture. Heart Rhythm 2004; 1:422–426.
46. Borger van der Burg AE, de Groot NM, van Erven L, Bootsma M, Van der Wall EE, Schalij MJ. Long-term follow-up after radiofrequency catheter ablation of ventricular tachycardia: A successful approach? J Cardiovasc Electrophysiol 2002; 13:417–423.
47. Della BP, Pappalardo A, Riva S, Tondo C, Fassini G, Trevisi N. Noncontact mapping to guide catheter ablation of untolerated ventricular tachycardia. Eur Heart J 2002; 23:742–752.
48. Schalij MJ, van Rugge FP, Siezenga M, van der Velde ET. Endocardial activation mapping of ventricular tachycardia in patients: First application of a 32-site bipolar mapping electrode catheter. Circulation 1998; 98:2168–2179.
49. Strickberger SA, Knight BP, Michaud GF, Pelosi F, Morady F. Mapping and ablation of ventricular tachycardia guided by virtual electrograms using a noncontact, computerized mapping system. J Am Coll Cardiol 2000; 35:414–421.
50. Miller JM, Kienzle MG, Harken AH, Josephson ME. Subendocardial resection for ventricular tachycardia: Predictors of surgical success. Circulation 1984; 70:624–631.
51. Ostermeyer J, Breithardt G, Borggrefe M, Godehardt E, Seipel L, Bircks W. Surgical treatment of ventricular tachycardias. Complete versus partial encircling endocardial ventriculotomy. J Thorac Cardiovasc Surg 1984; 87:517–525.
52. Garan H, Nguyen K, McGovern B, Buckley M, Ruskin JN. Perioperative and long-term results after electrophysiologically directed ventricular surgery for recurrent ventricular tachycardia. J Am Coll Cardiol 1986; 8:201–209.
53. Haines DE, Lerman BB, Kron IL, DiMarco JP. Surgical ablation of ventricular tachycardia with sequential map-guided subendocardial resection: Electrophysiologic assessment and long-term follow-up. Circulation 1988; 77:131–141.
54. Guiraudon GM, Thakur RK, Klein GJ, Yee R, Guiraudon CM, Sharma A. Encircling endocardial cryoablation for ventricular tachycardia after myocardial infarction: Experience with 33 patients. Am Heart J 1994; 128:982–989.
55. Frapier JM, Hubaut JJ, Pasquie JL, Chaptal PA. Large encircling cryoablation without mapping for ventricular tachycardia after anterior myocardial infarction: Long-term outcome. J Thorac Cardiovasc Surg 1998; 116:578–583.
56. Bourke JP, Campbell RW, Renzulli A, McComb JM, Cowan JC, Guzman F, Hilton CJ. Surgery for ventricular tachyarrhythmias based on fragmentation mapping in sinus rhythm alone. Eur J Cardiothorac Surg 1989; 3:401–406.
57. Miller JM, Tyson GS, Hargrove WC III, Vassallo JA, Rosenthal ME, Josephson ME. Effect of subendocardial resection on sinus rhythm endocardial electrogram abnormalities. Circulation 1995; 91:2385–2391.
58. Wrobleski D, Houghtaling C, Josephson ME, Ruskin JN, Reddy VY. Use of electrogram characteristics during sinus rhythm to delineate the endocardial scar in a porcine model of healed myocardial infarction. J Cardiovasc Electrophysiol 2003; 14:524–529.
59. Callans DJ, Ren JF, Michele J, Marchlinski FE, Dillon SM. Electroanatomic left ventricular mapping in the porcine model of healed anterior myocardial infarction. Correlation with intracardiac echocardiography and pathological analysis. Circulation 1999; 100:1744–1750.
60. Stevenson WG, Sager PT, Natterson PD, Saxon LA, Middlekauff HR, Wiener I. Relation of pace mapping QRS configuration and conduction delay to ventricular tachycardia reentry circuits in human infarct scars. J Am Coll Cardiol 1995; 26:481–488.
61. Brunckhorst CB, Delacretaz E, Soejima K, Maisel WH, Friedman PL, Stevenson WG. Identification of the ventricular tachycardia isthmus after infarction by pace mapping. Circulation 2004; 110:652–659.
62. Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM. Electrically unexcitable scar mapping based on pacing threshold for identification of the reentry circuit isthmus: Feasibility for guiding ventricular tachycardia ablation. Circulation 2002; 106:1678–1683.
63. Arenal A, del Castillo S, Gonzalez-Torrecilla E, Atienza F, Ortiz M, Jimenez J, Puchol A. Tachycardia-related channel in the scar tissue in patients with sustained monomorphic ventricular tachycardias: Influence of the voltage scar definition. Circulation 2004; 110:2568–2574.
64. Arenal A, Glez-Torrecilla E, Ortiz M, Villacastin J, Fdez-Portales J, Sousa E, del Castillo S, et al. Ablation of electrograms with an isolated, delayed component as treatment of unmappable monomorphic ventricular tachycardias in patients with structural heart disease. J Am Coll Cardiol 2003; 41:81–92.
65. Bogun F, Good E, Reich S, Elmouchi D, Igic P, Lemola K, Tschopp D, et al. Isolated potentials during sinus rhythm and pace-mapping within scars as guides for ablation of post-infarction ventricular tachycardia. J Am Coll Cardiol 2006; 47:2013–2019.
66. Kottkamp H, Wetzel U, Schirdewahn P, Dorszewski A, Gerds-Li JH, Carbucicchio C, Kobza R, et al. Catheter ablation of ventricular tachycardia in remote myocardial infarction: Substrate description guiding placement of individual linear lesions targeting noninducibility. J Cardiovasc Electrophysiol 2003; 14:675–681.
67. Aryana A, d'Avila A, Heist EK, Mela T, Singh JP, Ruskin JN, Reddy VY. Remote magnetic navigation to guide endocardial and epicardial catheter mapping of scar-related ventricular tachycardia. Circulation 2007; 115:1191–1200.
68. Kaltenbrunner W, Cardinal R, Dubuc M, Shenasa M, Nadeau R, Tremblay G, Vermeulen M, et al. Epicardial and endocardial mapping of ventricular tachycardia in patients with myocardial infarction. Is the origin of the tachycardia always subendocardially localized? Circulation 1991; 84:1058–1071.
69. Soejima K, Delacretaz E, Suzuki M, Brunckhorst CB, Maisel WH, Friedman PL, Stevenson WG. Saline-cooled versus standard radiofrequency catheter ablation for infarct-related ventricular tachycardias. Circulation 2001; 103:1858–1862.
70. Nakagawa H, Yamanashi WS, Pitha JV, Arruda M, Wang X, Ohtomo K, Beckman KJ, et al. Comparison of in vivo tissue temperature profile and lesion geometry for radiofrequency ablation with a saline-irrigated electrode versus temperature control in a canine thigh muscle preparation. Circulation 1995; 91:2264–2273.
71. Ruffy R, Imran MA, Santel DJ, Wharton JM. Radiofrequency delivery through a cooled catheter tip allows the creation of larger endomyocardial lesions in the ovine heart. J Cardiovasc Electrophysiol 1995; 6:1089–1096.
72. Yokoyama K, Nakagawa H, Wittkampf FH, Pitha JV, Lazzara R, Jackman WM. Comparison of electrode cooling between internal and open irrigation in radiofrequency ablation lesion depth and incidence of thrombus and steam pop. Circulation 2006; 113:11–19.
73. Calkins H, Epstein A, Packer D, Arria AM, Hummel J, Gilligan DM, Trusso J, et al. Catheter ablation of ventricular tachycardia in patients with structural heart disease using cooled radiofrequency energy: Results of a prospective multicenter study. Cooled RF Multi Center Investigators Group. J Am Coll Cardiol 2000; 35:1905–1914.
74. Strickberger SA, Man KC, Daoud EG, Goyal R, Brinkman K, Hasse C, Bogun F, et al. A prospective evaluation of catheter ablation of ventricular tachycardia as adjuvant therapy in patients with coronary artery disease and an implantable cardioverter-defibrillator. Circulation 1997; 96:1525–1531.
75. Brugada P, de Swart H, Smeets JL, Wellens HJ. Transcoronary chemical ablation of ventricular tachycardia. Circulation 1989; 79:475–482.
76. Kay GN, Epstein AE, Bubien RS, Anderson PG, Dailey SM, Plumb VJ. Intracoronary ethanol ablation for the treatment of recurrent

sustained ventricular tachycardia. J Am Coll Cardiol 1992; 19:159–168.
77. Delacretaz E, Seiler J, Tanner H, Hess OM. Ablation of ventricular tachycardia: Neither inside nor out, thus back to alcohol. Heart Rhythm 2006; 3:1230–1231.
78. Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. J Cardiovasc Electrophysiol 1996; 7:531–536.
79. Soejima K, Couper G, Cooper JM, Sapp JL, Epstein LM, Stevenson WG. Subxiphoid surgical approach for epicardial catheter-based mapping and ablation in patients with prior cardiac surgery or difficult pericardial access. Circulation 2004; 110:1197–1201.
80. Svenson RH, Littmann L, Gallagher JJ, Selle JG, Zimmern SH, Fedor JM, Colavita PG. Termination of ventricular tachycardia with epicardial laser photocoagulation: A clinical comparison with patients undergoing successful endocardial photocoagulation alone. J Am Coll Cardiol 1990; 15:163–170.
81. Littmann L, Svenson RH, Gallagher JJ, Selle JG, Zimmern SH, Fedor JM, Colavita PG. Functional role of the epicardium in postinfarction ventricular tachycardia. Observations derived from computerized epicardial activation mapping, entrainment, and epicardial laser photoablation. Circulation 1991; 83:1577–1591.
82. Sosa E, Scanavacca M, d'Avila A, Oliveira F, Ramires JA. Nonsurgical transthoracic epicardial catheter ablation to treat recurrent ventricular tachycardia occurring late after myocardial infarction. J Am Coll Cardiol 2000; 35:1442–1449.
83. Schweikert RA, Saliba WI, Tomassoni G, Marrouche NF, Cole CR, Dresing TJ, Tchou PJ, et al. Percutaneous pericardial instrumentation for endo-epicardial mapping of previously failed ablations. Circulation 2003; 108:1329–1335.
84. Brugada J, Berruezo A, Cuesta A, Osca J, Chueca E, Fosch X, Wayar L, et al. Nonsurgical transthoracic epicardial radiofrequency ablation: An alternative in incessant ventricular tachycardia. J Am Coll Cardiol 2003; 41:2036–2043.
85. d'Avila A, Gutierrez P, Scanavacca M, Reddy V, Lustgarten DL, Sosa E, Ramires JA. Effects of radiofrequency pulses delivered in the vicinity of the coronary arteries: Implications for nonsurgical transthoracic epicardial catheter ablation to treat ventricular tachycardia. Pacing Clin Electrophysiol 2002; 25:1488–1495.
86. d'Avila A, Houghtaling C, Gutierrez P, Vragovic O, Ruskin JN, Josephson ME, Reddy VY. Catheter ablation of ventricular epicardial tissue: A comparison of standard and cooled-tip radiofrequency energy. Circulation 2004; 109:2363–2369.
87. Reddy VY, Wrobleski D, Houghtaling C, Josephson ME, Ruskin JN. Combined epicardial and endocardial electroanatomic mapping in a porcine model of healed myocardial infarction. Circulation 2003; 107:3236–3242.
88. Cesario DA, Vaseghi M, Boyle NG, Fishbein MC, Valderrabano M, Narasimhan C, Wiener I, et al. Value of high-density endocardial and epicardial mapping for catheter ablation of hemodynamically unstable ventricular tachycardia. Heart Rhythm 2006; 3:1–10.
89. Soejima K, Stevenson WG, Sapp JL, Selwyn AP, Couper G, Epstein LM. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: The importance of low-voltage scars. J Am Coll Cardiol 2004; 43:1834–1842.
90. Sosa E, Scanavacca M. Epicardial mapping and ablation techniques to control ventricular tachycardia. J Cardiovasc Electrophysiol 2005; 16:449–452.
91. Buch E, Vaseghi M, Cesario DA, Shivkumar K. A novel method for preventing phrenic nerve injury during catheter ablation. Heart Rhythm 2007; 4:95–98.
92. Aoyama H, Nakagawa H, Pitha JV, Khammar GS, Chandrasekaran K, Matsudaira K, Yagi T, et al. Comparison of cryothermia and radiofrequency current in safety and efficacy of catheter ablation within the canine coronary sinus close to the left circumflex coronary artery. J Cardiovasc Electrophysiol 2005; 16:1218–1226.
93. Lustgarten DL, Bell S, Hardin N, Calame J, Spector PS. Safety and efficacy of epicardial cryoablation in a canine model. Heart Rhythm 2005; 2:82–90.
94. Roberts WC, Siegel RJ, McManus BM. Idiopathic dilated cardiomyopathy: Analysis of 152 necropsy patients. Am J Cardiol 1987; 60:1340–1355.
95. de Bakker JM, van Capelle FJ, Janse MJ, Tasseron S, Vermeulen JT, de Jonge N, Lahpor JR. Fractionated electrograms in dilated cardiomyopathy: Origin and relation to abnormal conduction. J Am Coll Cardiol 1996; 27:1071–1078.
96. Hsia HH, Callans DJ, Marchlinski FE. Characterization of endocardial electrophysiological substrate in patients with nonischemic cardiomyopathy and monomorphic ventricular tachycardia. Circulation 2003; 108:704–710.
97. Kottkamp H, Hindricks G, Chen X, Brunn J, Willems S, Haverkamp W, Block M, et al. Radiofrequency catheter ablation of sustained ventricular tachycardia in idiopathic dilated cardiomyopathy. Circulation 1995; 92:1159–1168.
98. Chen X, Shenasa M, Borggrefe M, Block M, Hindricks G, Martinez-Rubio A, Haverkamp W, et al. Role of programmed ventricular stimulation in patients with idiopathic dilated cardiomyopathy and documented sustained ventricular tachyarrhythmias: Inducibility and prognostic value in 102 patients. Eur Heart J 1994; 15:76–82.
99. Kies P, Bootsma M, Bax JJ, Zeppenfeld K, van Erven L, Wijffels MC, Van der Wall EE, et al. Serial reevaluation for ARVD/C is indicated in patients presenting with left bundle branch block ventricular tachycardia and minor ECG abnormalities. J Cardiovasc Electrophysiol 2006; 17:586–593.
100. Tada H, Shimizu W, Ohe T, Hamada S, Kurita T, Aihara N, Kamakura S, et al. Usefulness of electron-beam computed tomography in arrhythmogenic right ventricular dysplasia. Relationship to electrophysiological abnormalities and left ventricular involvement. Circulation 1996; 94:437–444.
101. Boulos M, Lashevsky I, Gepstein L. Usefulness of electroanatomical mapping to differentiate between right ventricular outflow tract tachycardia and arrhythmogenic right ventricular dysplasia. Am J Cardiol 2005; 95:935–940.
102. Verma A, Kilicaslan F, Schweikert RA, Tomassoni G, Rossillo A, Marrouche NF, Ozduran V, et al. Short- and long-term success of substrate-based mapping and ablation of ventricular tachycardia in arrhythmogenic right ventricular dysplasia. Circulation 2005; 111:3209–3216.
103. Miljoen H, State S, de Chillou C, Magnin-Poull I, Dotto P, Andronache M, Abdelaal A, et al. Electroanatomic mapping characteristics of ventricular tachycardia in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia. Europace 2005; 7:516–524.
104. Satomi K, Kurita T, Suyama K, Noda T, Okamura H, Otomo K, Shimizu W, et al. Catheter ablation of stable and unstable ventricular tachycardias in patients with arrhythmogenic right ventricular dysplasia. J Cardiovasc Electrophysiol 2006; 17:469–476.
105. Ellison KE, Friedman PL, Ganz LI, Stevenson WG. Entrainment mapping and radiofrequency catheter ablation of ventricular tachycardia in right ventricular dysplasia. J Am Coll Cardiol 1998; 32:724–728.
106. Ouyang F, Fotuhi P, Goya M, Volkmer M, Ernst S, Cappato R, Kuck K. Ventricular tachycardia around the tricuspid annulus in right ventricular dysplasia. Circulation 2001; 103:913–914.
107. Stark SI, Arthur A, Lesh MD. Radiofrequency catheter ablation of ventricular tachycardia in right ventricular cardiomyopathy: Use of concealed entrainment to identify the slow conduction isthmus bounded by an aneurysm and the tricuspid annulus. J Cardiovasc Electrophysiol 1996; 7:967–971.
108. Reithmann C, Hahnefeld A, Remp T, Dorwarth U, Dugas M, Steinbeck G, Hoffmann E. Electroanatomic mapping of endocardial right ventricular activation as a guide for catheter ablation in patients with arrhythmogenic right ventricular dysplasia. Pacing Clin Electrophysiol 2003; 26:1308–1316.
109. O'Donnell D, Cox D, Bourke J, Mitchell L, Furniss S. Clinical and electrophysiological differences between patients with arrhythmogenic right ventricular dysplasia and right ventricular outflow tract tachycardia. Eur Heart J 2003; 24:801–810.
110. Marchlinski FE, Zado E, Dixit S, Gerstenfeld E, Callans DJ, Hsia H, Lin D, et al. Electroanatomic substrate and outcome of catheter ablative therapy for ventricular tachycardia in setting of right ventricular cardiomyopathy. Circulation 2004; 110:2293–2298.
111. Koplan BA, Soejima K, Baughman K, Epstein LM, Stevenson WG. Refractory ventricular tachycardia secondary to cardiac sarcoid: Electrophysiologic characteristics, mapping, and ablation. Heart Rhythm 2006; 3:924–929.
112. Gonska BD, Cao K, Raab J, Eigster G, Kreuzer H. Radiofrequency catheter ablation of right ventricular tachycardia late after repair of congenital heart defects. Circulation 1996; 94:1902–1908.
113. Furushima H, Chinushi M, Sugiura H, Komura S, Tanabe Y, Watanabe H, Washizuka T, et al. Ventricular tachycardia late after repair of congenital heart disease: Efficacy of combination therapy with radiofrequency catheter ablation and class III antiarrhythmic agents and long-term outcome. J Electrocardiol 2006; 39:219–224.
114. Morwood JG, Triedman JK, Berul CI, Khairy P, Alexander ME, Cecchin F, Walsh EP. Radiofrequency catheter ablation of ventricular tachycardia in children and young adults

with congenital heart disease. Heart Rhythm 2004; 1:301–308.
115. Stevenson WG, Delacretaz E, Friedman PL, Ellison KE. Identification and ablation of macroreentrant ventricular tachycardia with the CARTO electroanatomical mapping system. Pacing Clin Electrophysiol 1998; 21:1448–1456.
116. Horton RP, Canby RC, Kessler DJ, Joglar JA, Hume A, Jessen ME, Scott WP, et al. Ablation of ventricular tachycardia associated with tetralogy of Fallot: Demonstration of bidirectional block. J Cardiovasc Electrophysiol 1997; 8:432–435.
117. Chinushi M, Aizawa Y, Kitazawa H, Kusano Y, Washizuka T, Shibata A. Successful radiofrequency catheter ablation for macroreentrant ventricular tachycardias in a patient with tetralogy of Fallot after corrective surgery. Pacing Clin Electrophysiol 1995; 18:1713–1716.
118. Cohen TJ, Chien WW, Lurie KG, Young C, Goldberg HR, Wang YS, Langberg JJ, et al. Radiofrequency catheter ablation for treatment of bundle branch reentrant ventricular tachycardia: Results and long-term follow-up. J Am Coll Cardiol 1991; 18:1767–1773.
119. Blanck Z, Dhala A, Deshpande S, Sra J, Jazayeri M, Akhtar M. Bundle branch reentrant ventricular tachycardia: Cumulative experience in 48 patients. J Cardiovasc Electrophysiol 1993; 4:253–262.
120. Tchou P, Mehdirad AA. Bundle branch reentry ventricular tachycardia. Pacing Clin Electrophysiol 1995; 18:1427–1437.
121. Lopera G, Stevenson WG, Soejima K, Maisel WH, Koplan B, Sapp JL, Satti SD, et al. Identification and ablation of three types of ventricular tachycardia involving the His-Purkinje system in patients with heart disease. J Cardiovasc Electrophysiol 2004; 15:52–58.
122. Li YG, Gronefeld G, Israel C, Bogun F, Hohnloser SH. Bundle branch reentrant tachycardia in patients with apparent normal His-Purkinje conduction: The role of functional conduction impairment. J Cardiovasc Electrophysiol 2002; 13:1233–1239.
123. Guo H, Hecker S, Levy S, Olshansky B. Ventricular tachycardia with QRS configuration similar to that in sinus rhythm and a myocardial origin: Differential diagnosis with bundle branch reentry. Europace 2001; 3:115–123.
124. Merino JL, Peinado R, Fernandez-Lozano I, Lopez-Gil M, Arribas F, Ramirez LJ, Echeverria IJ, et al. Bundle-branch reentry and the post-pacing interval after entrainment by right ventricular apex stimulation: A new approach to elucidate the mechanism of wide-QRS-complex tachycardia with atrioventricular dissociation. Circulation 2001; 103:1102–1108.
125. Leenhardt A, Glaser E, Burguera M, Nurnberg M, Maison-Blanche P, Coumel P. Short-coupled variant of torsade de pointes. A new electrocardiographic entity in the spectrum of idiopathic ventricular tachyarrhythmias. Circulation 1994; 89:206–215.
126. Haissaguerre M, Shoda M, Jais P, Nogami A, Shah DC, Kautzner J, Arentz T, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circulation 2002; 106:962–967.
127. Haissaguerre M, Extramiana F, Hocini M, Cauchemez B, Jais P, Cabrera JA, Farre J, et al. Mapping and ablation of ventricular fibrillation associated with long-QT and Brugada syndromes. Circulation 2003; 108:925–928.
128. Noda T, Shimizu W, Taguchi A, Aiba T, Satomi K, Suyama K, Kurita T, et al. Malignant entity of idiopathic ventricular fibrillation and polymorphic ventricular tachycardia initiated by premature extrasystoles originating from the right ventricular outflow tract. J Am Coll Cardiol 2005; 46:1288–1294.
129. Viskin S, Rosso R, Rogowski O, Belhassen B. The "short-coupled" variant of right ventricular outflow ventricular tachycardia: A not-so-benign form of benign ventricular tachycardia? J Cardiovasc Electrophysiol 2005; 16:912–916.
130. Bansch D, Oyang F, Antz M, Arentz T, Weber R, Val-Mejias JE, Ernst S, et al. Successful catheter ablation of electrical storm after myocardial infarction. Circulation 2003; 108:3011–3016.
131. Marrouche NF, Verma A, Wazni O, Schweikert R, Martin DO, Saliba W, Kilicaslan F, et al. Mode of initiation and ablation of ventricular fibrillation storms in patients with ischemic cardiomyopathy. J Am Coll Cardiol 2004; 43:1715–1720.
132. Mlcochova H, Saliba WI, Burkhardt DJ, Rodriguez RE, Cummings JE, Lakkireddy D, Patel D, et al. Catheter ablation of ventricular fibrillation storm in patients with infiltrative amyloidosis of the heart. J Cardiovasc Electrophysiol 2006; 17:426–430.
133. Zeppenfeld K, Blom NA, Bootsma M, Schalij MJ. Incessant ventricular tachycardia in fulminant lymphocytic myocarditis: Evidence for origin in the Purkinje system and successful treatment with ablation. Heart Rhythm 2007; 4:88–91.