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Elimination of Local Abnormal Ventricular Activities A New End Point for Substrate Modification in Patients With Scar-Related Ventricular Tachycardia

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Background—Catheter ablation of ventricular tachycardia (VT) is effective and particularly useful in patients with frequent defibrillator interventions. Various substrate modification techniques have been described for unmappable or hemodynamically intolerable VT. Noninducibility is the most frequently used end point but is associated with significant limitations, so the optimal end point remains unclear. We hypothesized that elimination of local abnormal ventricular activities (LAVAs) during sinus rhythm or ventricular pacing would be a useful and effective end point for substrate-based VT ablation. As an adjunct to this strategy, we used a new high-density mapping catheter and frequently used epicardial mapping.

Methods and Results—Seventy patients (age, 67 ± 11 years; 7 female) with VT and structurally abnormal ventricle(s) were prospectively enrolled. Conventional mapping was performed in sinus rhythm in all, and a high-density Pentaray mapping catheter was used in the endocardium ($n=35$) and epicardially. LAVAs were recorded in 67 patients (95.7%; 95% confidence interval, 89.2–98.9). Catheter ablation was performed targeting LAVA with an irrigated-tip catheter placed endocardially via a transseptal or retrograde aortic approach or epicardially via the subxiphoid approach. LAVAs were successfully abolished or dissociated in 47 of 67 patients (70.1%; 95% confidence interval, 58.7–80.1). In multivariate analysis, LAVA elimination was independently associated with a reduction in recurrent VT or death (hazard ratio, 0.49; 95% confidence interval, 0.26–0.95; $P=0.035$) during long-term follow-up (median, 22 months).

Conclusions—LAVAs can be identified in most patients with scar-related VT. Elimination of LAVAs is feasible and safe and is associated with superior survival free from recurrent VT. (*Circulation*. 2012;125:2184-2196.)

Key Words: catheter ablation ■ endpoint determination ■ epicardial mapping ■ tachycardia, ventricular

Catheter ablation of ventricular tachycardia (VT) in the setting of structural heart disease is challenging. Current ablation strategies often rely on the ability to identify a reentrant circuit and to target a critical isthmus through activation and entrainment mapping.^{1–5} A reliably inducible, well-tolerated, and single monomorphic VT is ideal but unfortunately not the rule. Factors such as noninducibility or poor hemodynamic tolerance may render the VT unmappable. In such situations, various ablation strategies such as 3-dimensionally guided substrate mapping and pace mapping have been described.^{6–12} Although late potentials were recently proposed as ablation targets, the impact of ablation and

correlations with clinical outcomes have not been reported.¹³ In addition, the proposed approach relied solely on noninducibility as an end point, which has important caveats: Reproducibility is limited; it does not consistently predict long-term outcomes; and the significance of inducing nonclinical VT(s) remains uncertain.

Editorial see p 2175 Clinical Perspective on p 2196

We hypothesized that elimination of local abnormal ventricular activities (LAVAs) recorded during sinus rhythm or ventricular pacing would be feasible as an end point for VT

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ablation and that complete elimination of LAVAs would lead to a reduction in arrhythmia-free survival.

Methods

Study Population

From January 2006 to January 2010, 70 consecutive patients undergoing VT ablation at 2 centers were prospectively enrolled after providing written informed consent. Inclusion criteria consisted of documented episodes of repetitive sustained VT resistant to antiarrhythmic drug therapy and requiring external cardioversion or implantable cardioverter-defibrillator (ICD) antitachycardia pacing or shocks and structural heart disease with ischemic or nonischemic dilated cardiomyopathy. Patients were excluded if ventricular arrhythmias were attributable to an acute or reversible cause. Patients with repetitive premature ventricular contractions or nonsustained VT in the absence of sustained VT were also excluded.

Preprocedural Preparation

All patients underwent an imaging study to assess ventricular function and to identify areas of ventricular scar. In patients without an ICD, magnetic resonance imaging was the preferred imaging modality. In others, an echocardiogram was performed. Barring extreme rhythm instability, all antiarrhythmic drugs but amiodarone were discontinued for a period of at least 5 half-lives.

Left Ventricular Access and Electrophysiological Study

The electrophysiological study and ablation were conducted with postabsorptive conscious sedation with midazolam 0.02 mg/kg before femoral puncture, morphine sulfate 0.1 to 0.2 mg/kg, and supplementary analgesia (sufentanil 0.05–0.15 μ g) under supervision of an anesthesiologist for the pericardial approach. Noninvasive or intra-arterial blood pressure monitoring and digital pulse oximetry were performed continuously. ICD therapies were inactivated.

Vascular sheaths were inserted into the right femoral vein (2–3), right femoral artery (1), and/or subxiphoid area (1 or 2) under local anesthesia (bupivacaine). A steerable quadripolar or decapolar catheter (2–5–2 mm, Xtrem, ELA Medical, Montrouge, France) was positioned in the right ventricular apex or coronary sinus. The left ventricle (LV) was accessed by transseptal (BRK Needle, St. Jude Medical) and/or retrograde routes with or without pericardial access for additional epicardial mapping. The mode of access to the LV was at the operator's discretion. However, in the absence of contraindications, dual or triple accesses were encouraged. After LV access was attained, a 50-U/kg heparin bolus was administered intravenously and repeated as necessary to target an activated clotting time >250 seconds.

Pericardial Approach

Pericardial access was obtained if a previous endocardial procedure had failed, if an epicardial substrate was suspected (based on VT morphology on a surface ECG and the nature of the underlying heart disease),¹⁴ and if endocardial mapping did not reveal LAVAs or if endocardial ablation did not result in LAVA elimination. The pericardial approach, as described by Sosa et al,¹⁵ was modified to access the pericardial space through an anterior puncture because it was considered safer than the conventional inferior puncture. The pericardial puncture was guided by a 90° left lateral fluoroscopic projection (Figure 1).¹⁶ To facilitate subsequent pericardial punctures, 50 to 100 mL air was instilled into the pericardial space and aspirated thereafter.¹⁷ A standard steerable sheath (Agilis, St. Jude Medical) was used to provide catheter stability and maneuverability.

Mapping

Surface ECGs and bipolar intracardiac electrograms were monitored continuously on a LabSystem Pro (Bard Electrophysiology, Lowell, MA). Signals were sampled at 1 kHz and filtered at 0.1 to 50 Hz for

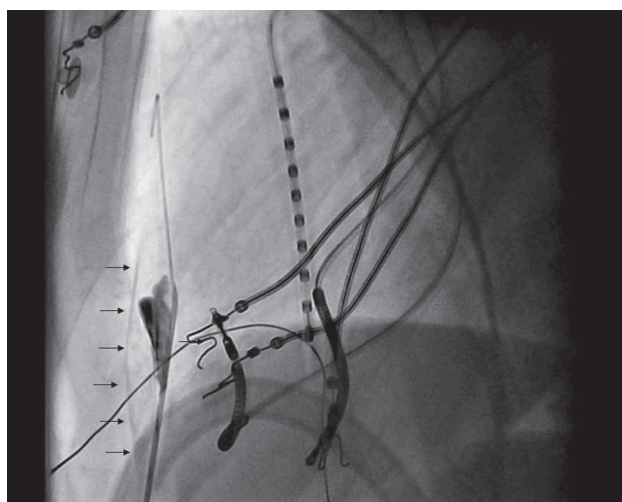


Figure 1. Lateral fluoroscopic projection showing the subxiphoid pericardial puncture with a Tuohy needle. The course of the needle in the substernal space is almost tangential to the posterior border of the sternum. It penetrates the parietal layer of the pericardium anteriorly as visualized from the contrast staining the anterior pericardium. Intrapericardial entry is confirmed with contrast trickling into the pericardial space (arrows). The guidewire is then inserted.

surface ECGs and 30 to 250 Hz for intracardiac signals, displayed at an amplification of 0.1 mV/cm.

VT induction was attempted at baseline with (1) programmed ventricular stimulation from 2 right ventricular sites (outflow tract and apex), at 2 drive trains (400 and 600 milliseconds), with up to 3 extrastimuli decremented to ventricular refractoriness or 250 milliseconds, whichever was higher, and (2) incremental burst pacing up to 200 milliseconds. If hemodynamically stable VT was induced, it was mapped to identify the site of earliest ventricular activation and abnormal presystolic ventricular potentials, if any. Tachycardia was then terminated by overdrive pacing. Detailed LV endocardial and epicardial mapping was performed with the roving ablation (Thermocool or Navistar, Biosense Webster) and/or a high-density mapping catheter (Pentaray, Biosense Webster) to identify and localize LAVAs. Concealed entrainment was used whenever possible and appropriate. Use of 3-dimensional electroanatomic mapping systems was at the operator's discretion.

Ventricular mapping was undertaken to identify and localize regions displaying LAVAs and to characterize healthy and scarred areas by conventional voltage criteria. Thereafter, substrate mapping focused on abnormal myocardium and bordering zones based on low-voltage areas with 3-dimensional mapping or identified scar from prior cardiac imaging, the VT morphology on a surface ECG, and scar information obtained from previous mapping procedures whenever available.

Definition of LAVAs

LAVAs were defined as sharp high-frequency ventricular potentials, possibly of low amplitude, distinct from the far-field ventricular electrogram occurring anytime during or after the far-field ventricular electrogram in sinus rhythm or before the far-field ventricular electrogram during VT that sometimes displayed fractionation or double or multiple components separated by very-low-amplitude signals or an isoelectric interval and were poorly coupled to the rest of the myocardium, as demonstrated by the maneuvers detailed below. These high-frequency sharp signals were considered indicative of local electric activity arising from pathological tissue.^{18–20} Examples are shown in Figure 2.

To confirm the nature of LAVAs and to distinguish them from far-field ventricular electrograms in the presence of ambiguity, different maneuvers were used. Programmed ventricular stimulation was performed from the right ventricular apex or sites in closer

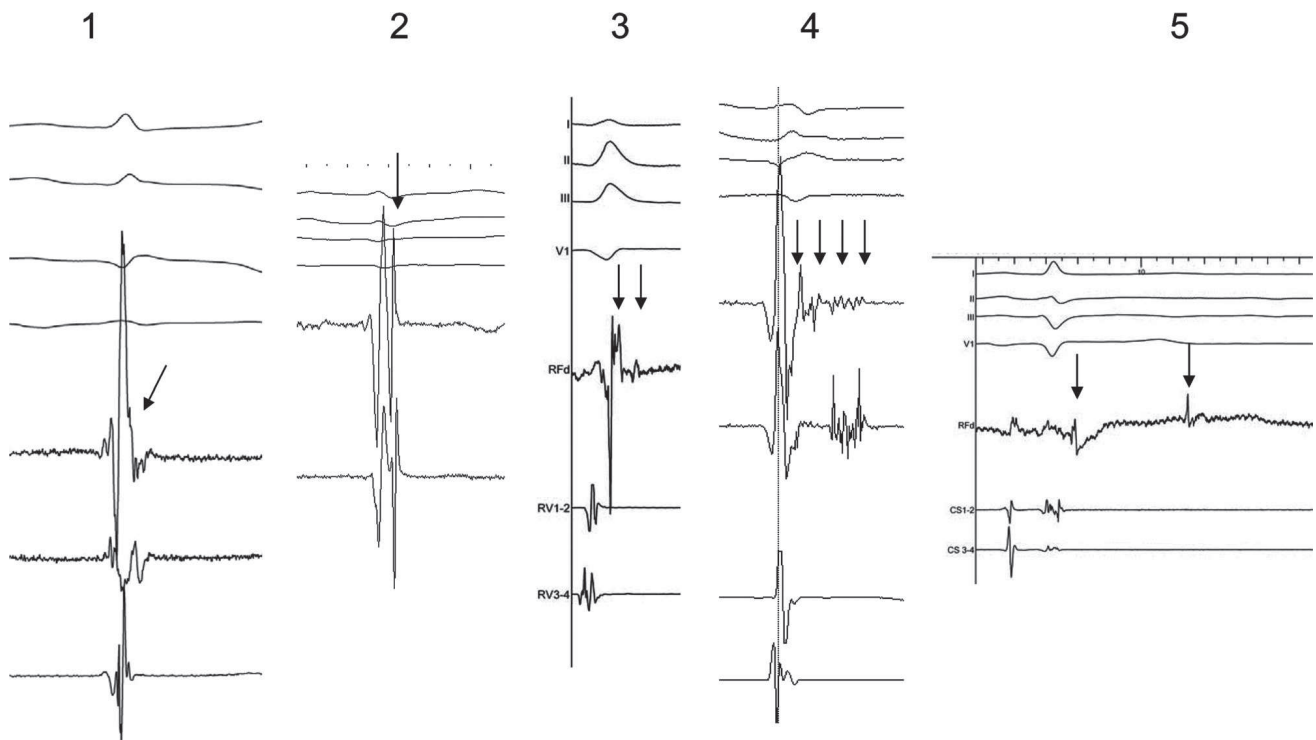


Figure 2. Electrogram recordings from different patients to show various characteristics of local abnormal ventricular activities (LAVAs; arrows). 1, The potential representing LAVA is fused with the terminal portion of the far-field ventricular signal, making it difficult to identify the LAVA as a separate signal. 2, LAVA potential occurs just after and with a slightly higher frequency than the far-field ventricular potential. LAVAs in 1 and 2 occur within the QRS complex. 3, LAVA is a double-component potential that closely follows the far-field ventricular signal. The early component is a high-frequency potential that is almost fused with the preceding far-field ventricular signal. It occurs within the terminal portion of the QRS complex. Another low-amplitude signal follows an isoelectric interval and represents the late component of LAVA, which occurs after the QRS complex. 4, LAVAs are represented by pluricomponent signals without isoelectric intervals. These signals can be visualized distinctly from the preceding far-field ventricular signal. 5, Double-component LAVA signal. Although the early component is recorded just after the QRS complex, the late component is recorded after the inscription of the T wave on the surface ECG.

proximity to LAVAs. LAVAs were observed to progressively split further away from the far-field ventricular electrogram after an earlier extrastimulus (S2), which sometimes resulted in conduction block (ie, absence of LAVAs at a site where a LAVA was previously recorded after ensuring adequate catheter stability and contact; Figure 3). Local ectopics (Figure 4A) or local pacing with decremental output was also used (Figure 4B). Loss of capture of the far-field component was marked by a distinct change in the stimulus to QRS delay and QRS morphology (Figure 4B). Pacing endocardially while simultaneously recording on the corresponding epicardial surface allowed documentation of intramyocardial conduction time. Substantial delay in intramyocardial conduction was caused by poor coupling between the LAVA-generating myocardial bundles and remaining myocardium (Figure 5).

LAVAs were characterized by measuring the signal amplitude and local delay with reference to the far-field ventricular electrogram and the end of the QRS complex on the surface ECG. The amplitude and duration of far-field ventricular electrograms were also recorded. Measurements were undertaken offline by 2 observers using electronic and manual calipers.

Catheter Ablation

As a general rule, for all patients in whom LAVAs were observed, ablation of LAVAs in sinus rhythm was encouraged, with complete LAVA elimination targeted as the procedural outcome. In patients in whom at least 1 VT was inducible and well tolerated (ie, 11 during the first intervention, 3 during subsequent interventions), ablation was guided by conventional entrainment mapping criteria to identify the critical isthmus.⁵ After restoration of sinus rhythm, further mapping and ablation were undertaken to completely eliminate LAVAs (Figure 6). When VT was not inducible or was poorly

tolerated, ablation was conducted in sinus rhythm with the same end point.

Ablation was performed with a 3.5-mm externally irrigated-tip catheter.³ Radiofrequency (RF) energy with a power of 25 to 50 W was delivered at all sites displaying LAVAs. If a definite sequence of activation of LAVA was clearly discerned on the Pentaray catheter, the earliest signals were targeted first (Figure 6B and 6C). If both epicardial and endocardial LAVAs were observed, ablation was performed initially on the endocardial side to abolish the potentials transmurally, with subsequent epicardial ablation if required (Figure 6B and 6C). Four types of responses to catheter ablation of LAVA were predefined: no effect, delay, abolition, or dissociation. Complete elimination was defined as abolition or dissociation at all LAVA sites, whereas partial elimination was defined as abolition or dissociation at some but not all LAVA sites. For epicardial ablation in the pericardial space, the procedure was terminated if 15 minutes of RF energy delivery failed to eliminate LAVAs in the region of interest. No time limit was imposed for RF ablation on the endocardial surface, which remained at the operator's discretion.

After ablation, the ventricle was remapped. Additional substrate ablation was performed if residual LAVAs were identified or if VT remained inducible. Remapping and inducibility testing were avoided in unstable patients to limit procedural duration in the interest of patient safety. In addition to complete elimination of LAVAs, noninducibility was considered a procedural end point in patients with previously inducible VT.

Postprocedural Care

Venous and arterial sheaths were withdrawn immediately. Pericardial sheaths were retracted after a dry pericardial aspirate was confirmed. ICD therapies were reprogrammed with active VT and

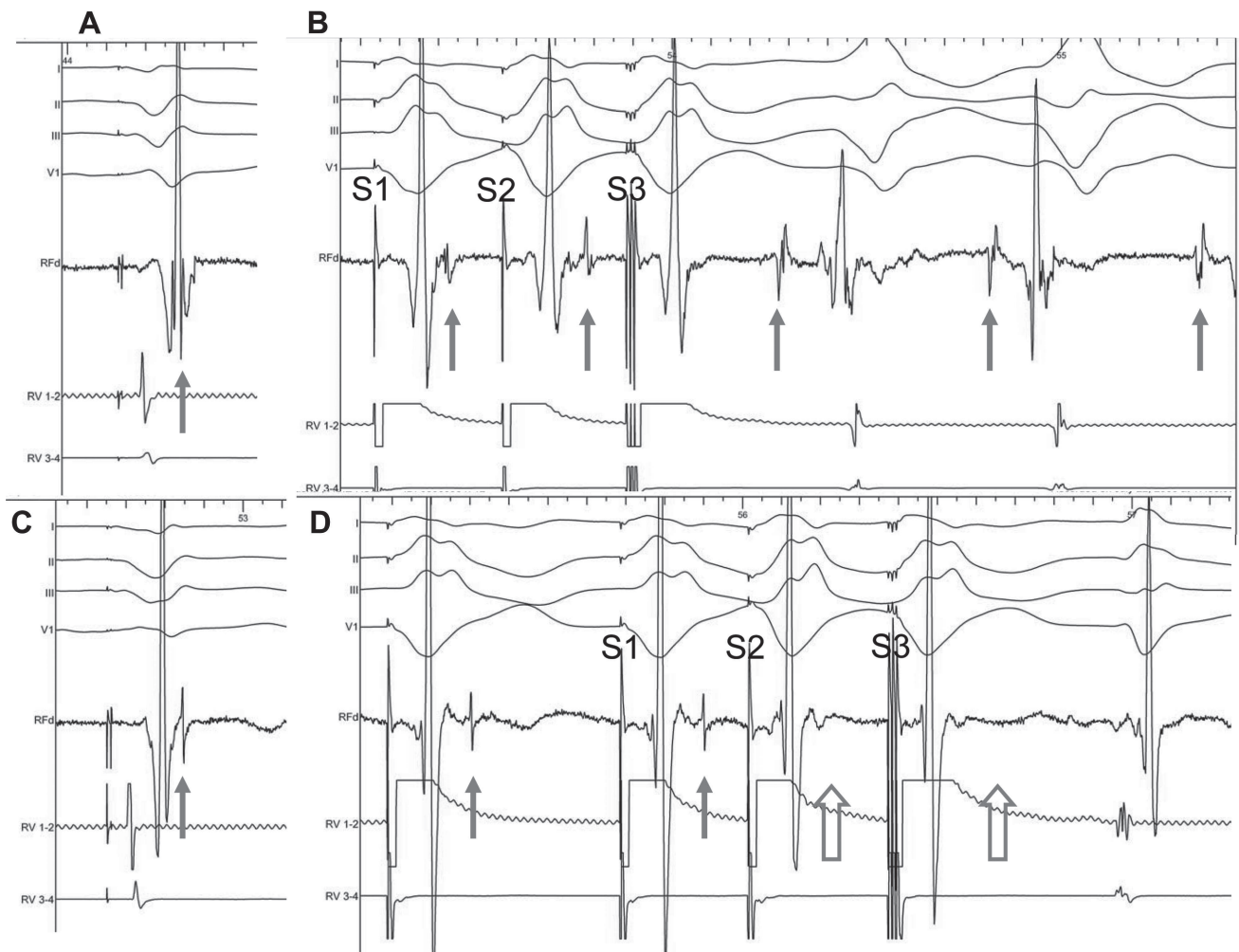


Figure 3. Role of local abnormal ventricular activities (LAVAs) in the induction of ventricular tachycardia (VT) and the influence of radiofrequency energy on LAVAs. Before radiofrequency energy delivery (RF): **A**, At first sight, the local ventricular electrogram during baseline paced rhythm looks simple. However, in the terminal portion of this simple-looking signal, a very high-frequency component (LAVA) can be identified. **B**, Programmed electric stimulation from the right ventricle (RV) unmasks the LAVA potential by increasing the delay from the far-field signal. The delay observed during RV pacing suggests poor coupling of the muscle bundle generating the LAVA signal. The delay is maximal with S3, which is associated not only with a change in the polarity of LAVA but also with the induction of VT. Post-RF energy delivery: **C**, After delivery of RF energy, there is a remarkable delay (see **A**) between the far-field ventricular signal and LAVAs during baseline paced rhythm. **D**, Repeat programmed electric stimulation from the RV results in the absence of LAVA signals after the far-field ventricular potential during S2 and S3 (open arrows). The absence of LAVAs is associated with an inability to induce the VT. Although ablation has rendered VT noninducible, further RF energy application is indicated to completely eliminate the LAVAs.

ventricular fibrillation zones. Patients were monitored at least 48 hours in the hospital. Preprocedural antiarrhythmic drugs were continued unless contraindicated or not tolerated.

Follow-Up

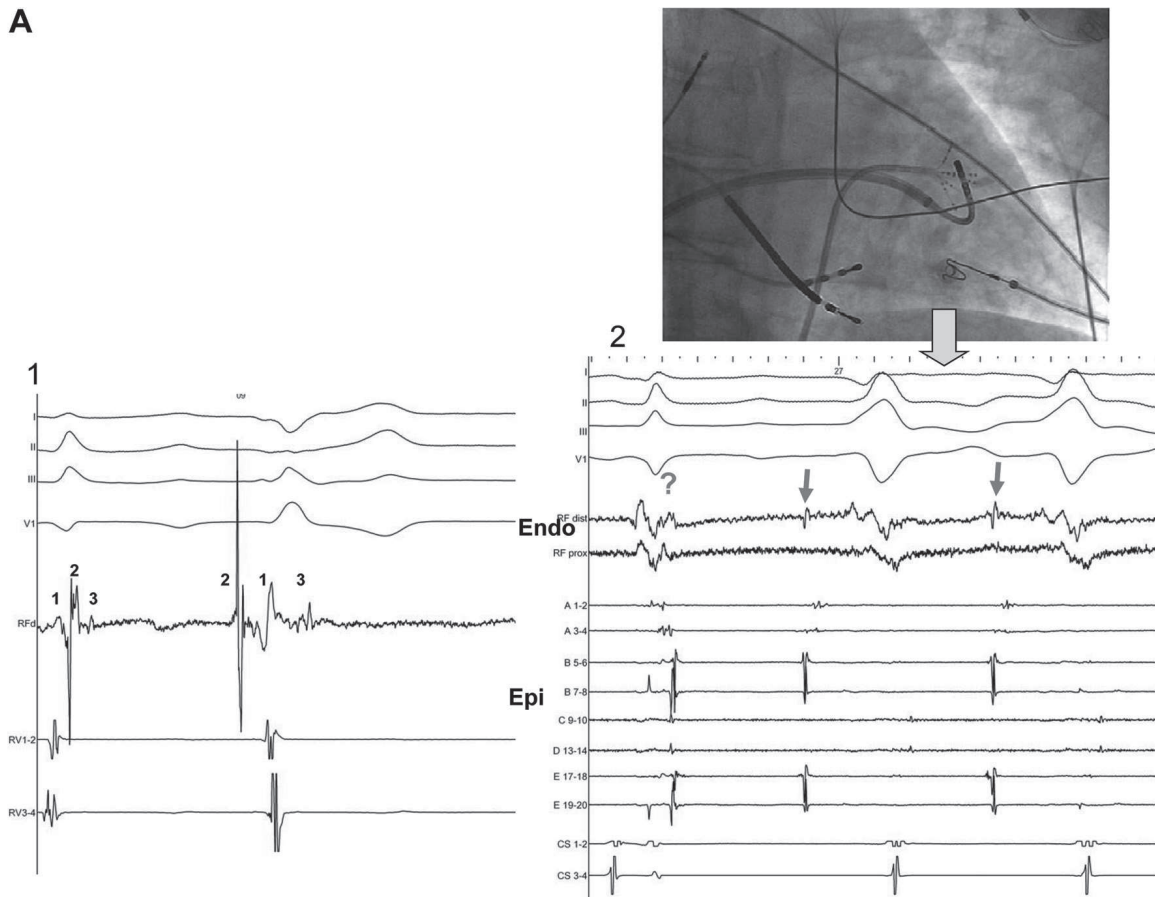
Patients were followed up at 1, 3, 6, and 12 months for the first year and every 6 months thereafter. ICDs were interrogated at each visit, and arrhythmia logs were retrieved. A detailed history, Holter monitoring, and ECG were performed in symptomatic patients without ICDs. An echocardiogram was conducted at 6 or 12 months to reassess ventricular function. Patients with relapse(s) were offered repeat ablation on a case-by-case basis. The primary end point was a composite of death or recurrent VT. Qualifying arrhythmias included any ECG-documented VT, whether detected by ICDs, 12-lead ECGs, Holter monitors, or rhythm strips, regardless of morphology or rate.

Statistical Analysis

Continuous variables are summarized by mean \pm SD or median and interquartile range (IQR; 25th–75th percentile), depending on the

normality of distribution, as assessed by normal probability and quartile plots. Categorical variables are represented by frequencies and percentages. Two-group comparisons between clinical (Table 1), LAVA (Table 2), and procedural (Table 3) characteristics in patients with and without LAVA elimination were performed by use of independent Student *t* tests, Wilcoxon rank-sum tests, or Fisher exact tests as appropriate. However, because multiple VTs may have been induced in a given patient, generalized estimating equations were used to compare VT cycle lengths by specifying an identity link and exchangeable correlation structure. The LV ejection fractions before ablation and at the last follow-up visit were compared by use of a paired Student *t* test. Factors associated with LAVA elimination were assessed in univariate logistic regression models. Because no such factor was identified, multivariate logistic regression was not performed. Freedom from recurrent VT or all-cause death was plotted with the Kaplan-Meier method in patients with and without LAVA elimination and compared by use of the log-rank statistic. Time 0 was defined as time of initial VT ablation. Cox proportional hazard models were used to assess predictors of recurrent VT or death in patients with LAVA identified during the index procedure. Proportional hazards assumptions were verified by assessing time-

A



B

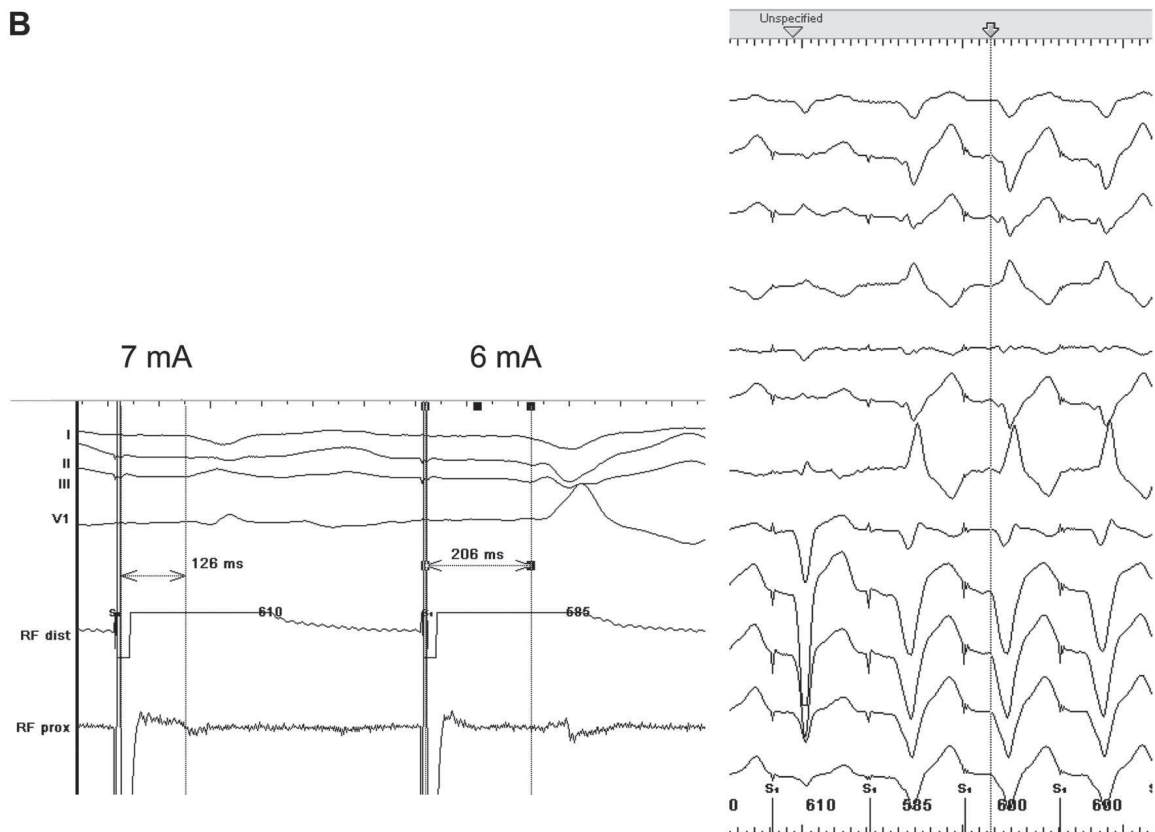


Figure 4. A, Local abnormal ventricular activities (LAVAs) during sinus and ectopic beats in a patient with anterior myocardial infarction. **A**, Surface ECG leads show 1 sinus beat followed by an ectopic beat. During sinus beat, a complex ventricular electrogram signal

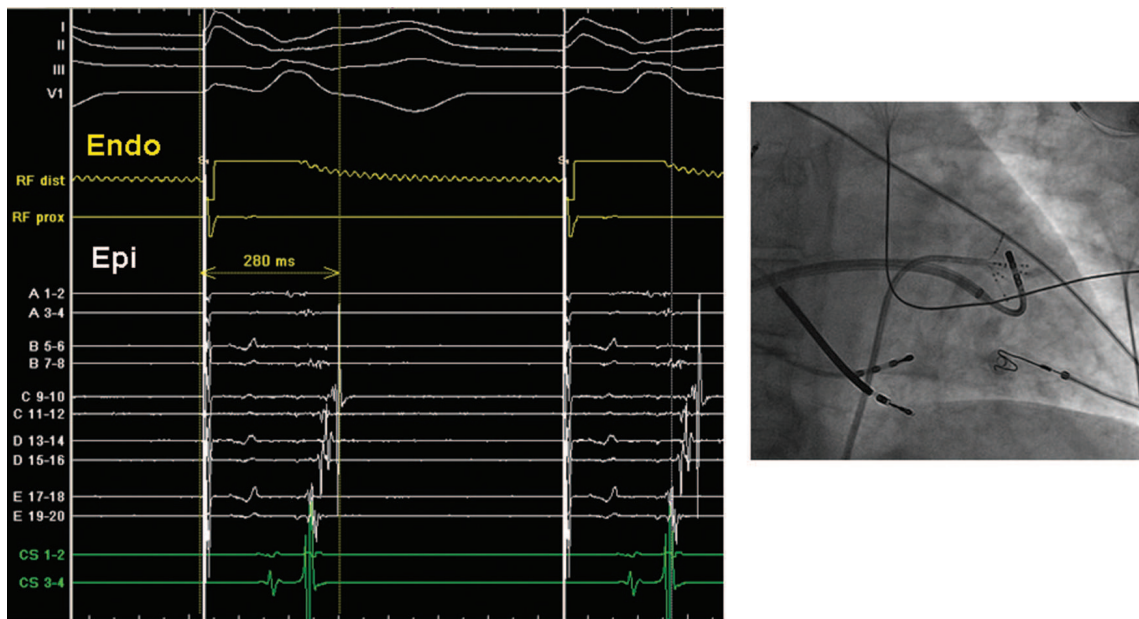


Figure 5. Intramyocardial conduction time in a patient with anterior myocardial infarction. **Left,** Pacing the endocardial site of local abnormal ventricular activities (LAVAs) with the ablation catheter (radiofrequency) and recording the signals from the corresponding epicardial sites with the Pentaray (splines A to E) catheter. The intramyocardial conduction time between the endocardially (Endo) delivered pacing stimulus and the signal recorded on the corresponding epicardial site is 280 milliseconds. Such an extreme delay for the impulse to reach the epicardial (Epi) aspect suggests the presence of poorly coupled muscle bundles surviving in the scar tissue. **Right,** Fluoroscopic image showing a transseptal sheath used to map the endocardial left ventricle with the ablation catheter and a Pentaray catheter lying epicardially facing the endocardial catheter.

dependent covariates (with time modeled linearly and logarithmically) and by plotting Schoenfeld residuals supplemented by testing for nonzero slopes. Baseline imbalances in patients with and without LAVA elimination and variables significant at the 0.2 level in univariate analyses were considered in an automated stepwise multivariate Cox regression model (entry, 0.05; removal, 0.10). Candidate variables included all clinical, LAVA, and procedural characteristics listed in Tables 1 through 3. Agreement between LAVA elimination and noninducibility as procedural outcomes was assessed by the κ statistic and summarized as percent overall, positive, and negative agreement. Values of $P < 0.05$ were considered statistically significant. Statistical testing was performed with SAS software version 9.2 (SAS Institute, Cary, NC).

Results

Study Population

During the study period, 133 patients underwent VT ablation. Fifty-seven patients were excluded because of the following nonqualifying diagnoses: arrhythmogenic right ventricular cardiomyopathy (n=20), idiopathic VT (n=22), congenital heart disease (n=8), valvular heart disease (n=3), hypertrophic cardiomyopathy (n=1), and catecholaminergic VT (n=1). An additional 6 patients declined consent. The study population consisted of the remaining 70 patients. Baseline characteristics are summarized in Table 1.

Figure 4 (Continued). shows 3 components labeled 1, 2, and 3 in the order of their occurrence with fusion between the first 2. Component 1 represents far-field ventricular signal; components 2 and 3 represent high- and low-frequency LAVA signals, respectively. The sequence of occurrence of these components is altered during an ectopic beat. High-frequency component 2, which precedes the QRS complex, is followed by the far-field ventricular signal (component 1) with a marked delay between them, suggestive of local conduction disturbance. Also note the change in the polarity of high-frequency LAVA signal (2) between the 2 beats. Component 3 represents low-frequency LAVAs originating from another poorly coupled local muscle bundle. **Right, Top,** Fluoroscopic image showing a transseptal sheath used to map the endocardial (Endo) left ventricle with an ablation catheter and a Pentaray catheter lying epicardially (Epi) facing the endocardial catheter. **Bottom,** During the first beat, in sinus rhythm, a far-field low-amplitude potential is recorded endocardially on the ablation catheter, suggestive of a scar tissue. The terminal deflection of this signal occurring at the end of the QRS complex is suspected to be LAVAs. The ambiguity is resolved when the suspected LAVA potential clearly precedes the far-field ventricular signal in the 2 local ectopic beats after the sinus beat. A low-amplitude but high-frequency LAVA potential precedes the onset of QRS (and far-field ventricular signal) by >100 milliseconds, demonstrating the poor coupling of the bundle generating LAVAs with the rest of the myocardium. Interestingly, the LAVAs are better seen on the corresponding epicardial aspect of the ischemic scar. **B,** Outcomes of pacing with a progressive reduction in the current strength from the site recording LAVA endocardially in a patient with ischemic cardiomyopathy. **Left,** The delay between the pacing stimulus and the QRS onset increased from 126 to 206 milliseconds, and the QRS complex became wider when the pacing current strength was reduced from 7 to 6 mA. When the output is reduced, the capture might be more limited to the muscle bundles (near field) lying amid the scar tissue and responsible for generating the LAVA signal. Because of their poor coupling to the rest of the myocardium, the stimulus to the QRS onset delay is prolonged with a reduction in current strength. Narrower QRS complex could be explained by a wider field of capture and a different route of exit of the impulse with higher current strength than otherwise. **Right,** Twelve-lead ECG during pacing at progressively reduced current strength (output energy) shows a change in the morphology of QRS as described above.

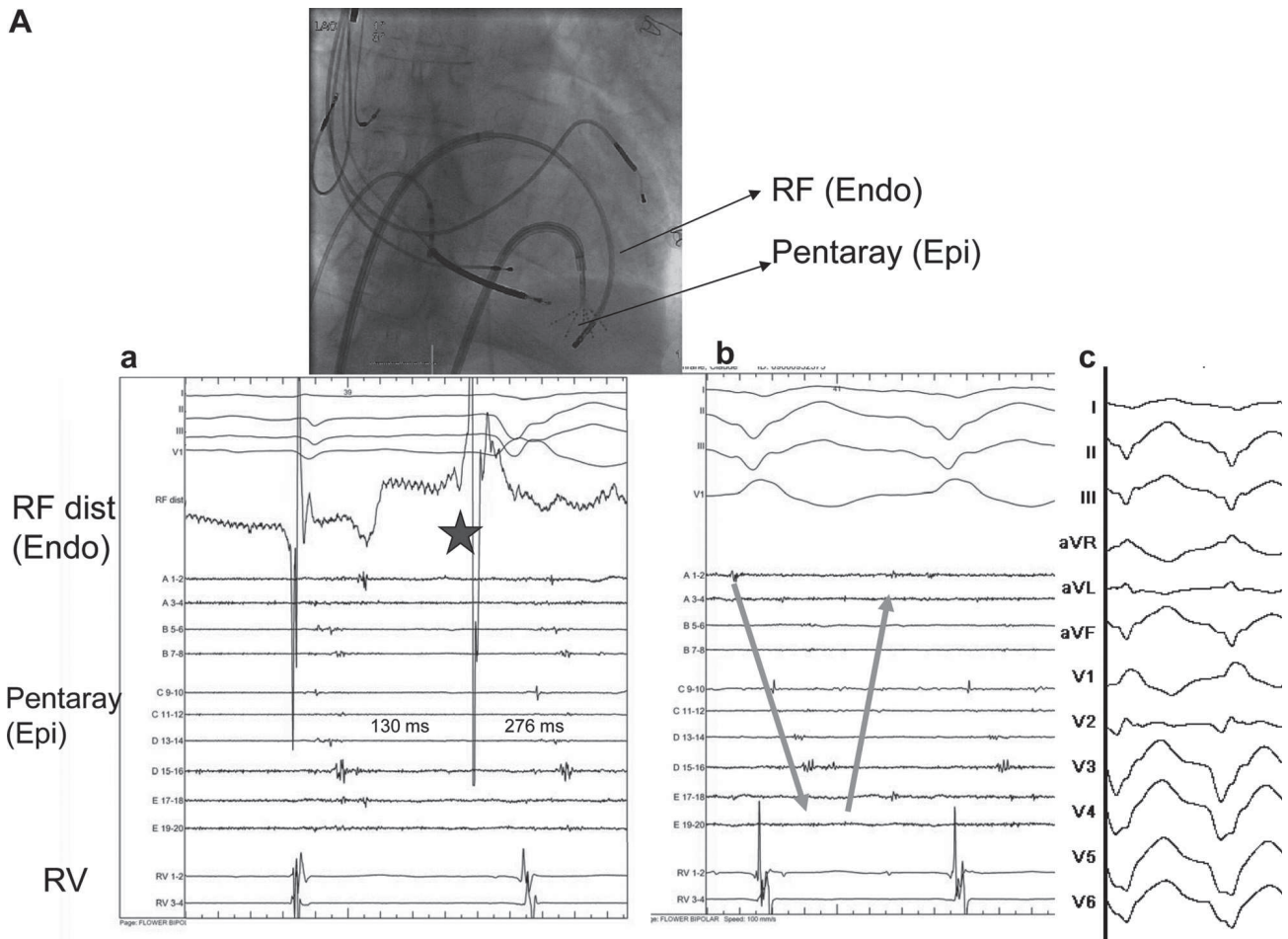


Figure 6. A, Endocardial and epicardial local abnormal ventricular activity (LAVA) recordings in a patient with severe ischemic left ventricular (LV) dysfunction (LV ejection fraction, 26%) and scar-related ventricular tachycardia (VT). **Top**, Fluoroscopic image showing a transseptal sheath used to map the endocardial LV with the ablation catheter and a Pentaray catheter lying epicardially (Epi) facing the endocardial (Endo) catheter. **Bottom**, LAVA is recorded on all the splines (A–E) of the Pentaray with a maximum delay of 130 milliseconds from the far-field endocardial ventricular potential in sinus rhythm. During a local endocardial ectopy, the delay on the corresponding epicardial site is remarkably increased to a maximum value of 276 milliseconds (a). During VT, the epicardial LAVA recordings cover almost the entire cycle length of the tachycardia (b). Twelve-lead ECG during VT (c). **B**, Effect of endocardial ablation on epicardially recorded LAVAs in the patient described in **A**. **Top**, Four fluoroscopic images in different projections (anteroposterior [AP], left anterior oblique [LAO]) showing transseptally accessed LV during endocardial ablation at different sites and a Pentaray catheter lying epicardially facing the endocardial catheter to monitor the effect of ablation on the epicardial LAVAs. The absence of direct contact between both catheters allows convenient monitoring of LAVAs in the absence of artifacts during radiofrequency (RF) delivery shown in the corresponding panels below each image. **Bottom (a)**, LAVA recorded on the endocardial site is later than the latest LAVA on the epicardial site. Because endocardial LAVA earlier than the epicardial LAVA is the target site for ablation, ablation is not undertaken at the suboptimal site shown here (a). The ablation catheter is moved to another site where continuous LAVAs (arrows) are recorded endocardially immediately after the end of the far-field ventricular potential (b). Such a site where endocardial LAVA precedes the epicardial recordings is considered appropriate for endocardial ablation. Ablation at the site shown in b is associated with changes in LAVA epicardially (c). Ablation is continued endocardially with an end point of complete abolition of LAVA. Ablation results in abolition of LAVA on most of the splines of the Pentaray (d). Dissociated LAVAs can be observed on some splines. The end point is adequately reached. **C**, This electroanatomic map obtained from another patient depicts the epicardial activation in sinus rhythm in a patient with dilated cardiomyopathy. The area with the latest activation appears in blue where some of the potentials are shown with LAVAs. LAVAs at the center show the latest activities, with predominantly superior input that would be targeted first. No LAVAs were recorded endocardially in this patient. Most of the ablation was carried out epicardially. RV indicates right ventricle.

LV Access

A retrograde approach was used in 61 patients (87%) and a transseptal approach in 32 patients (46%). Percutaneous epicardial access was obtained in 21 patients (31%), 15 of whom had a prior myocardial infarction and 6 of whom had nonischemic dilated cardiomyopathy. One patient underwent concomitant epicardial ablation (without endocardial ablation) during open-chest surgery.

LAVA Mapping and Characteristics

LAVAs, the primary target of ablation, were found in 67 of 70 patients (95.7%; 95% confidence interval [CI], 89.2–98.9) during the initial ablation procedure. In patients with 3-dimensional electroanatomic mapping, LAVAs occupied 39 ± 32 cm² of the 245 ± 174 cm² of the LV surface (16%). Endocardial and epicardial LAVAs were present in 63 of 70 patients (90%) and 17 of 21 patients (81%), respectively.

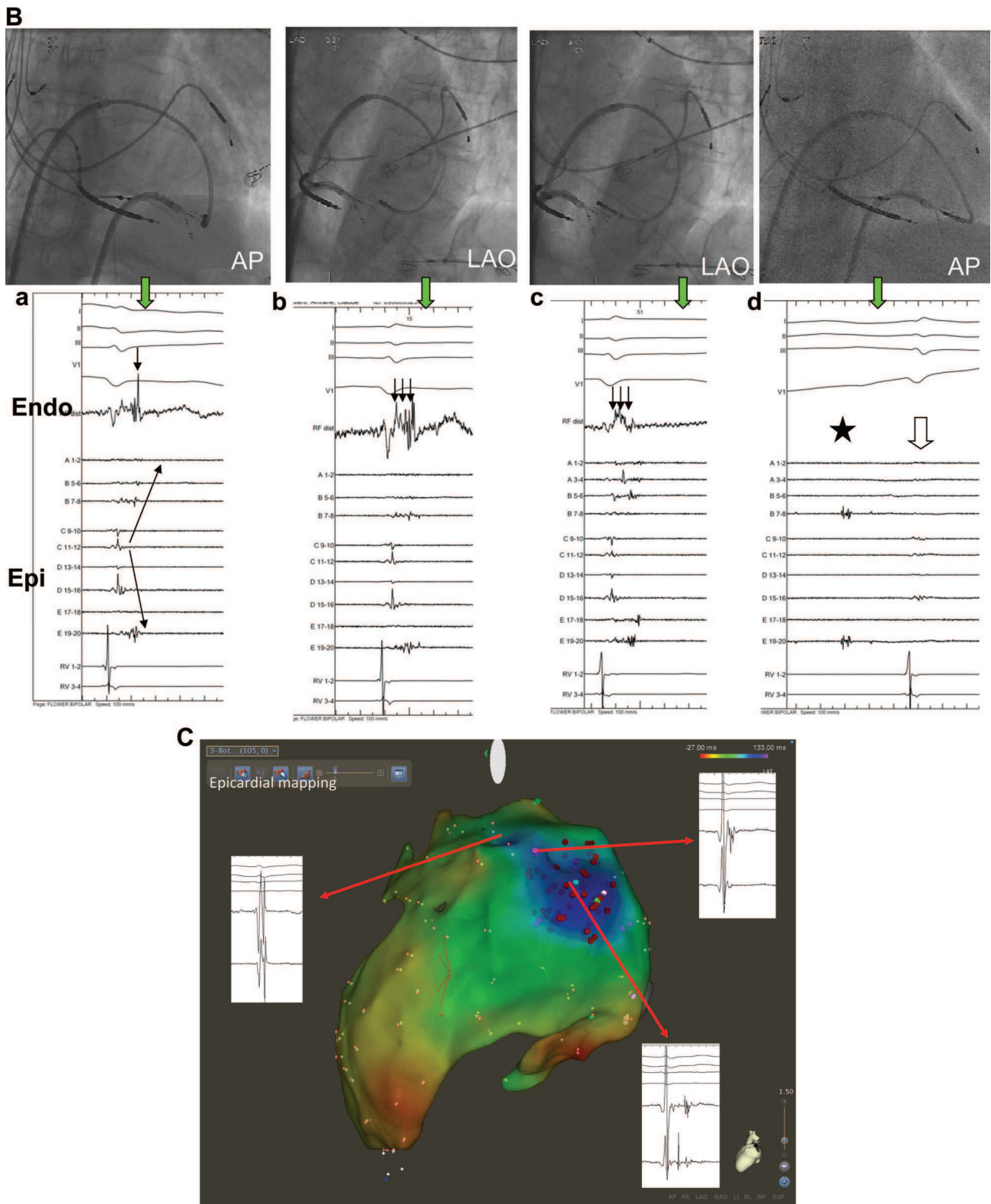


Figure 6 (Continued).

LAVAs were observed epicardially in 4 of the 7 patients with no endocardial LAVAs, whereas the remaining 3 patients had no identified LAVA.

Characteristics of the LAVA signals are summarized in Table 2. The LAVA amplitude was significantly higher when recorded from the epicardium (median, 0.37 mV; IQR,

0.20–0.60 mV) compared with the endocardium (median, 0.11 mV; IQR, 0.08–0.22 mV; $P=0.002$). At sites where LAVAs were recorded, the median amplitude and duration of the far-field endocardial ventricular electrogram were 0.20 mV (IQR, 0.10–0.50 mV) and 60 milliseconds (IQR, 50–83 milliseconds), respectively. Normal amplitude (>1.5 mV)

Table 1. Baseline Clinical Characteristics in All Patients and According to Whether Local Abnormal Ventricular Activities Were Completely Eliminated

	All Patients (n=70)	LAVAs Eliminated (n=47)	LAVAs Not Eliminated (n=20)	P*
Age, y	67±11	66±12	68±9	0.66
Male sex, n (%)	63 (90)	41 (87)	19 (95)	0.67
Hypertension, n (%)	31 (44)	22 (47)	7 (35)	0.41
Diabetes mellitus, n (%)	18 (26)	12 (26)	6 (30)	0.55
Hypercholesterolemia, n (%)	40 (57)	30 (64)	9 (45)	0.59
Active smoking, n (%)	32 (46)	24 (51)	8 (40)	0.77
Type of heart disease, n (%)				0.19
Ischemic	56 (80)	40 (85)	14 (70)	
Nonischemic	14 (20)	7 (15)	6 (30)	
Implantable cardioverter-defibrillator, n (%)	53 (76)	37 (79)	14 (70)	0.53
Cardiac resynchronization therapy, n (%)	13 (19)	8 (17)	4 (20)	0.74
Left ventricular ejection fraction, %	35±10	34±10	37±11	0.28
Amiodarone, n (%)	42 (60)	28 (60)	13 (65)	0.79
β-blockers, n (%)	56 (80)	40 (85)	14 (70)	0.16
Procedures per patient, n†	1 (1–1)	1 (1–1)	1 (1–2)	0.11

LAVA indicates local abnormal ventricular activity.

*Comparisons between patients with and without complete LAVA elimination.

†Nonnormally distributed variables are summarized as median and interquartile range (25th–75th percentile).

was found in only 2 patients. The median delay between the far-field ventricular electrogram and LAVAs was 80 milliseconds (IQR, 60–110 milliseconds), and the median delay between the end of QRS on the surface ECG and onset of LAVA was 0 milliseconds (IQR, 0–40 milliseconds). LAVAs occurred before the end of the QRS in 13 of 63 patients (21%; negative delay). No specific LAVA characteristic predicted the ability to achieve complete LAVA elimination.

Ablation

The Pentaray catheter was used to map the endocardium in 35 patients and in all epicardial procedures. In addition, standard entrainment and electroanatomic mapping were each performed in 11 patients; both mapping modalities were performed in 3 patients. LAVA was eliminated in 47 of 67 patients (70.1%; 95% CI, 58.7–80.1), with signals abolished in 40 (60%) and dissociated in 7 (10%). No baseline clinical characteristic, including age, ejection fraction, and type of

cardiomyopathy, was predictive of LAVA elimination. Interestingly, in 5 cases, endocardial ablation also could abolish potentials recorded from the epicardium (Figure 6 B). As noted in Table 3, procedural and fluoroscopy times were similar in patients with and without complete LAVA elimination. However, a significantly longer RF energy delivery time was observed in cases when all LAVAs were successfully ablated. In addition, LAVA elimination was achieved more frequently when a transseptal approach was used and when VT was inducible. There was little agreement between noninducibility of VT and LAVA elimination as procedural end points (κ statistic, 0.038 ± 0.097 ; $P=0.70$). Overall percent agreement was 54.0% (95% CI, 41.7–65.7), with a positive percent agreement of 30.0% (95% CI, 14.6–52.2) and negative percent agreement of 65.1% (95% CI, 50.1–77.6).

Adverse Events

Adverse events potentially related to the procedure occurred in 6 patients (8.6%; 95% CI, 3.5–16.6), 4 of 47 (9%) with and

Table 2. Characteristics of Local Abnormal Ventricular Activities During the Initial Intervention in All Patients and According to Whether Local Abnormal Ventricular Activities Were Completely Eliminated

	All Patients (n=70)	LAVAs Eliminated (n=47)	LAVAs Not Eliminated (n=20)	P*
LAVA endocardial amplitude, mV	0.11 (0.08–0.22)	0.12 (0.08–0.21)	0.14 (0.10–0.29)	0.41
LAVA epicardial amplitude, mV	0.37 (0.20–0.60)	0.39 (0.20–0.70)	0.22 (0.18–0.50)	0.38
Far-field ventricular endocardial amplitude, mV	0.20 (0.10–0.50)	0.18 (0.10–0.60)	0.25 (0.10–0.43)	0.80
Duration of far-field ventricular signal, ms	60 (50–83)	61 (50–80)	60 (50–108)	0.66
Endocardial far-field ventricular to LAVA delay, ms	80 (60–110)	90 (70–115)	70 (55–100)	0.13
Endocardial QRS to LAVA delay, ms	0 (0–40)	20 (0–40)	0 (0–40)	0.50

LAVA indicates local abnormal ventricular activity.

*Comparisons between patients with and without complete LAVA elimination.

Table 3. Procedural Characteristics in All Patients and According to Whether Local Abnormal Ventricular Activities Were Completely Eliminated

	All Patients (n=70)	LAVAs Eliminated (n=47)	LAVAs Not Eliminated (n=20)	<i>P</i> *
Transseptal access, n (%)	32 (46)	26 (55)	4 (20)	0.013
Epicardial access, n (%)	21 (30)	17 (36)	4 (20)	0.11
Inducibility of VT at baseline, n (%)	49 (70)	31 (66)	17 (85)	0.012
VT morphologies induced, n†	2 (1–3)	1 (1–3)	2 (1–3)	0.27
Cycle length of mapped VTs, ms	400 (322–450)‡	400 (328–460)‡	400 (318–440)‡	0.23§
Entrainment mapping, n (%)	11 (16)	7 (15)	4 (20)	0.72
Electroanatomic mapping, n (%)	11 (16)	8 (17)	3 (15)	1.00
LAVAs in sinus rhythm, n (%)	67 (96)	47 (100)	20 (100)	NA
LAVAs in VT, n (%)	36 (51)	25 (53)	10 (50)	1.00
Endocardial LAVAs, n (%)	63 (90)	42 (89)	20 (100)	0.55
Epicardial LAVAs, n (%)	17/21 (81)‡	13/17 (76)‡	4/4 (100)‡	1.00
VT inducibility after ablation, n (%)	21 (30)	15 (32)	6 (30)	0.78
Radiofrequency ablation time, min	23±11	26±11	18±9	0.007
Fluoroscopy time, min	42±20	44±19	36±21	0.14
Total procedure duration, min	148±73	186±78	155±57	0.092

LAVA indicates local abnormal ventricular activities; VT, ventricular tachycardia.

*Comparisons between patients with and without complete LAVA elimination.

†Nonnormally distributed variables are summarized by median and interquartile range (25th–75th percentile).

‡The denominator reflects the number of patients with epicardial access.

§*P* value derived from a generalized estimating equations analysis that adjusts for multiple observations in the same patient.

2 of 20 (10%) without LAVA elimination. Complications in patients with LAVA elimination included atrial fibrillation requiring anticoagulation with late development of tamponade, as well as prolonged pericardial bleeding after epicardial ablation. Both were managed conservatively. In addition, 2 patients died within 24 hours of the procedure, one as a result of a low-flow state and the other as a result of low flow and arrhythmia recurrence. Complications in patients without LAVA elimination consisted of right ventricular perforation requiring surgical repair and tamponade during endocardial ablation managed conservatively.

Follow-Up

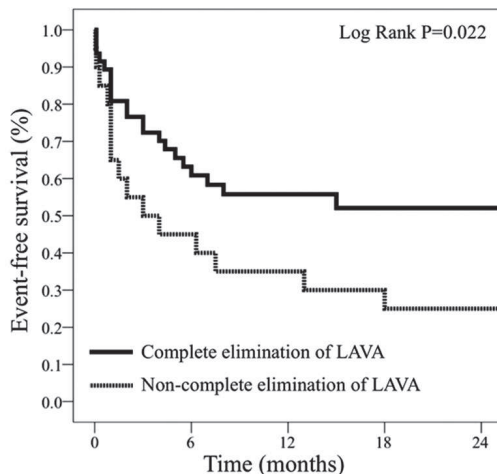
Patients were followed up for a median of 22 months (IQR, 14–27 months) from the initial ablation procedure. The combined end point of VT recurrence or death occurred in 39 patients (55.7%; 95% CI, 44.0–66.8), with recurrent VT in 32 (46%) and death in 13 (19%). The combined end point occurred in 21 of 47 patients (45%) with LAVA elimination, with recurrent VT in 15 (32%) and death in 9 (19%). In contrast, the combined end point occurred in 16 of 20 patients (80%) without LAVA elimination, with recurrent VT in 15 (75%) and death in 4 (20%). None of the 3 patients without LAVA identified at the index procedure died during follow-up. However, 2 experienced recurrent VT at 6 and 9.5 months. Overall, 6 patients who died experienced recurrent VT before their demise. Three patients died during the first 24 hours of an ablation procedure: 2 patients died after the initial intervention (as mentioned above), and a third with ischemic heart disease and an ejection fraction of 20% developed pulseless electric activity during reintervention. Two patients died of heart failure at 10 days and 6 months. One patient

without an ICD died suddenly at 5 weeks; another died at 30 months of VT storm. Six others died of noncardiac causes unrelated to the procedure from 20 days to 40 months after intervention. One patient with VT relapse underwent heart transplantation 5 months after the procedure.

In univariate Cox regression analyses, elimination of LAVA was the only factor significantly associated with a reduction in recurrent VT or death (hazard ratio, 0.48; 95% CI, 0.25–0.92; *P*=0.027). Noninducibility (*P*=0.11) and ischemic versus nonischemic heart disease (*P*=0.29) were not predictive of VT-free survival. Event-free survival curves are portrayed in Figure 7. Elimination of LAVA remained significantly associated with a reduction in VT or death in the multivariate regression analysis that controlled for baseline imbalances (eg, age, type of heart disease, and RF ablation time) between patients with and without complete LAVA elimination (hazard ratio, 0.49; 95% CI, 0.26–0.95; *P*=0.035).

Repeat Ablation

A redo procedure was performed in 14 patients, 6 of whom had LAVAs successfully eliminated during the first intervention and 8 of whom had persistent LAVAs. LAVAs had recovered in 5 of the 6 patients with initial LAVA elimination and were successfully re-eliminated. The sixth patient had reintervention for symptomatic premature ventricular beats from the left ventricular outflow tract, with no relationship to the index procedure. In the 8 patients with initially persisting LAVAs, repeat ablation eliminated LAVAs in 4 and failed in the other 4. At last follow-up, 62.7% of patients remained free of VT recurrence after the last ablation procedure. The LV



Number at risk					
Complete LAVA elimination	47	27	17	11	6
Non-complete LAVA elimination	20	9	6	6	4

Figure 7. Kaplan-Meier curves depict freedom from recurrent ventricular tachycardia or death in patients with and without complete elimination of local abnormal ventricular activities (LAVAs).

ejection fraction at the last follow-up visit was not significantly different from baseline ($P=0.34$).

Discussion

Main Findings

This study describes a new approach for substrate modification in scar-related VT, targeting LAVAs and aiming at either isolating or dissociating these electrically surviving myocardial fibers amid the scar areas. It also emphasizes the advantage of using a high-density mapping catheter and the benefit of a concomitant epicardial approach. The main findings are the following: (1) LAVAs are observed in the majority of patients with ischemic or dilated cardiomyopathy; (2) LAVAs can be eliminated or dissociated by catheter ablation in sinus rhythm; (3) ablation of LAVAs appears reasonably safe; (4) LAVA elimination is a clear procedural end point; and (5) complete elimination of LAVAs is associated with a superior clinical outcome.

Significance of LAVA

Residual electric activity persisting within scar areas has previously been reported.^{6,11,20–23} This phenomenon has been studied in animal preparations, particularly in myocardial infarction models.²⁴ Surviving cells surrounded by fibrosis were poorly coupled to the rest of the myocardium and were demonstrated to be responsible for the critically slow conduction needed for reentry. Inhomogeneous scars and/or heterogeneous necrosis, as seen in ischemic and dilated cardiomyopathy, therefore provide an ideal substrate for VT.²⁵ In humans, many investigators have reported such abnormal electric potentials, using such terms as delayed, isolated, or late.^{6,11,20,21,26,27} However, prior descriptions of electric characteristics were limited, with no data on amplitude and timing in relation to the QRS or to local or far-field ventricular signals. In addition, the precise distinction between the origin of the near versus far-field signal had not been described.

Involvement of this abnormal electric activity in VT circuits has previously been demonstrated by activation and entrainment mapping.⁶ One group of investigators who proposed targeting it during VT ablation did not report elimination or dissociation of this abnormal activity.¹³ Moreover, no data on the impact of catheter ablation targeting LAVA were reported, and LAVA elimination was never assessed as an end point to define procedural success. Instead, noninducibility was regarded as the procedural end point. The only report describing a similar strategy included a small number of patients with arrhythmogenic right ventricular cardiomyopathy.²⁸ The authors reported that LAVA elimination was associated with a significantly better outcome. Interestingly, a similar observation was noted in our study wherein the population consisted of patients with ischemic and nonischemic dilated cardiomyopathy.

LAVA Elimination Defining Acute Procedural Success

Most strategies for VT ablation aim at rendering VT noninducible, but this end point has several disadvantages. In some patients, VT cannot be induced despite clinical documentation. At the other end of the spectrum, several VTs of different morphologies can be induced in some patients. Whereas all clinical VTs should be eliminated, the strategy for VTs never observed clinically but induced after ablation is less clear. Ablation can modify VT circuits and exits without abolishing the ability to sustain ventricular arrhythmia, and nonclinical VTs may represent genuine circuits and therefore the substrate for relapse. In addition, a nonclinical VT that might never have been documented could become clinical after the procedure. Nevertheless, targeting nonclinical VTs may lead to excessive ablation.

The strategy of complete LAVA elimination overcomes some of these limitations of inducibility as the procedural end point. One advantage is that VT is required for neither mapping and ablation nor for the evaluation of efficacy. This approach is associated with substantially improved comfort for the patient and physician, does not require complex invasive techniques such as hemodynamic support, and allows online monitoring of the impact of ablation on the arrhythmogenic substrate. It does not necessarily rely on 3-dimensional mapping systems, entrainment mapping, or pace mapping.

Complete elimination of LAVAs was confirmed in 70.1% of patients in whom they had been documented. Interestingly, procedures associated with LAVA elimination had significantly longer RF ablation times. Although it may be possible that more extensive ablation could improve procedural outcomes independently of LAVAs, the superior survival free from recurrent VT in patients with complete LAVA elimination was independent of RF ablation time. Therefore, these results suggest that this strategy more thoroughly addresses the arrhythmogenic substrate and carries the potential to improve long-term results.

High-Density Mapping and Pericardial Approach

An additional novel feature of this study is its use of the Pentaray catheter in the pericardial space to provide high-

density maps of a large region. The splines of the catheter are soft and do not produce a substantial amount of mechanical ectopies. The catheter was stable within the pericardium and provided clean (noise-free) electric signals. During endocardial catheter ablation at sites facing the epicardial splines, very few or no artifactual interferences were observed, enabling careful monitoring of transmural response to ablation. Alternative multielectrode catheters such as basket or balloon arrays are of interest but cannot be positioned within the pericardial space and may restrict catheter manipulation within the ventricular cavity. Moreover, the signals may be influenced or altered by catheter-to-catheter contact during endocardial mapping.

Safety

The systematic use of an anterior (superficial) approach to access the pericardium instead of a conventional inferior approach likely contributed to the reasonable procedural safety in patients with an epicardial approach. In particular, it may explain the absence of abdominal complications during pericardial puncture.

Clinical Follow-Up

Elimination of LAVAs was associated with a significant reduction in VT recurrence or death. Our definition of long-term success was strict because it considered all-cause death or any arrhythmia relapse as a failure, whereas others have relied on outcomes limited to clinical VT or a reduction in arrhythmia burden.⁶ Moreover, although some have reported freedom from VT after ablation in the 75% to 100% range, direct comparisons are obscured by the shorter follow-up (7–8 months), fewer number of patients (≤ 20), and inclusion limited to patients with ischemic heart disease.⁷ Our observed event-free survival rate (Figure 7) is consistent with other large multicenter series reporting success rates of 51% to 53% in ischemic heart disease.^{3,29} A short-term 51% success rate has been reported in nonischemic substrates.³⁰

Study Limitations

We consider that targeting beyond the so-called clinical VT to attain complete LAVA elimination offers a more complete modification of the VT substrate compared with the usual end point of noninducibility. However, because the study was observational in nature, it remains possible that patients in whom LAVA could be eliminated may have had VT that was more amenable to ablation in general and may therefore have had superior outcomes regardless. Our promising results provide the grounds for pursuing a randomized clinical trial to definitely assess whether LAVA elimination strategy improves outcomes. It should also be noted that, in a minority of patients, entrainment and electroanatomic mapping were also performed. The study was not designed to determine the optimal density of maps nor the role of adjunctive mapping techniques in targeting and eliminating LAVA. We recognize that a potential disadvantage of this strategy could be greater tissue destruction than required to achieve clinical success. However, the postablation LV ejection fraction remained unchanged in our study, suggesting that the ablation of surviving cells in scar areas was not deleterious.

Conclusions

Local abnormal ventricular potentials are identifiable in most patients with scar-related VT. Pending detailed mapping, catheter ablation can be used to eliminate them in sinus rhythm with a reasonable safety profile and distinctly clear end point. This comprehensive substrate modification strategy is associated with superior survival free from recurrent VT.

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CLINICAL PERSPECTIVE

Catheter ablation of ventricular tachycardia (VT) is effective and particularly useful in patients with frequent defibrillator interventions. Various substrate modification techniques have been described for unmappable VT. Noninducibility is the most frequently used end point but is associated with significant limitations, and the optimal end point remains unclear. We hypothesized that elimination of local abnormal ventricular activities (LAVAs) during sinus rhythm or ventricular pacing would be a useful and effective end point for substrate-based VT ablation. LAVAs are usually assimilated to potentials occurring after the QRS. We demonstrate that some are buried in the QRS and give clues to identify them. As an adjunct to this strategy, we used a new high-density mapping catheter and frequently used epicardial mapping. A very superficial approach of the needle is described to prevent intra-abdominal route and potential associated complications. Seventy patients with VT and structurally abnormal ventricle(s) were prospectively enrolled. Conventional mapping was performed in sinus rhythm in all patients, and a high-density Pentaray mapping catheter was used in the endocardium (n=35) and epicardially. LAVAs were recorded in 67 patients (95.7%). Catheter ablation was performed targeting LAVA with an irrigated-tip catheter placed endocardially via a transseptal or retrograde aortic approach or epicardially via the subxiphoid approach. LAVAs were successfully abolished or dissociated in 47 of 67 patients (70.1%). In multivariate analysis, LAVA elimination was independently associated with a reduction in recurrent VT or death during long-term follow-up. In conclusion, elimination of LAVA is feasible and safe and associated with superior survival free from recurrent VT.