

The Value of Defibrillator Electrograms for Recognition of Clinical Ventricular Tachycardias and for Pace Mapping of Post-Infarction Ventricular Tachycardia

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- Objectives** The purpose of this study was to assess the value of implantable cardioverter-defibrillator (ICD) electrograms (EGMs) in identifying clinically documented ventricular tachycardias (VTs).
- Background** Twelve-lead electrocardiograms (ECG) of spontaneous VT often are not available in patients referred for catheter ablation of post-infarction VT. Many of these patients have ICDs, and the ability of ICD EGMs to identify a specific configuration of VT has not been described.
- Methods** In 21 consecutive patients referred for catheter ablation of post-infarction VT, 124 VTs (mean cycle length: 393 ± 103 ms) were induced, and ICD EGMs were recorded during VT. Clinical VT had been documented with 12-lead ECGs in 15 of 21 patients. The 12-lead ECGs of the clinical VTs were compared with 64 different inducible VTs (mean cycle length: 390 ± 91 ms) to assess how well the ICD EGMs differentiated the clinical VTs from the other induced VTs. The exit site of 62 VTs (mean cycle length: 408 ± 112 ms) was identified by pace mapping (10 to 12 of 12 matching leads). The spatial resolution of pace mapping to identify a VT exit site was determined for both the 12-lead ECGs and the ICD EGMs using a customized MATLAB program (version 7.5, The MathWorks, Inc., Natick, Massachusetts).
- Results** Analysis of stored EGMs by comparison of receiver-operating characteristic curve cutoff values accurately distinguished the clinical VTs from 98% of the other inducible VTs. The mean spatial resolution of a 12-lead ECG pace map for the VT exit site was 2.9 ± 4.0 cm² (range 0 to 17.5 cm²) compared with 8.9 ± 9.0 cm² (range 0 to 35 cm²) for ICD EGM pace maps. The spatial resolution of pace mapping varied greatly between patients and between VTs. The spatial resolution of ICD EGMs was <1.0 cm² for ≥ 1 of the target VTs in 12 of 21 patients and 19 of 62 VTs. By visual inspection of the ICD EGMs, 96% of the clinical VTs were accurately differentiated from previously undocumented VTs.
- Conclusions** Stored ICD EGMs usually are an accurate surrogate for 12-lead ECGs for differentiating clinical VTs from other VTs. Pace mapping based on ICD EGMs has variable resolution but may be useful for identifying a VT exit site. (J Am Coll Cardiol 2010;56:969–79) © 2010 by the American College of Cardiology Foundation

Patients who receive frequent appropriate shocks from implantable cardioverter-defibrillators (ICDs) often are referred for catheter ablation of ventricular tachycardia (VT). Stored ICD electrograms (EGMs) of spontaneously occurring VTs often are the only documentation of VT in patients undergoing VT ablation. Multiple configurations of VT typically are inducible in these patients. No studies

have assessed the value of ICD EGMs as a surrogate for 12-lead electrocardiograms (ECGs) to differentiate clinical VTs from previously undocumented VTs. The purpose of this study was to assess the value of ICD EGMs for differentiating clinical VT from other VT configurations that are as yet documented. In addition, the spatial resolution of pace mapping of post-infarction VT on the basis of the 12-lead ECG and an ICD EGM were compared.

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Methods

Patient characteristics. Twenty-one patients (2 women; mean age 70 ± 10 years; mean ejection fraction $0.25 \pm$

From the *Division of Cardiovascular Medicine, University of Michigan, Ann Arbor, Michigan; and the †Department of Electrical Engineering and Computer Science, University of Michigan, Ann Arbor, Michigan. Dr. Bogun has a patent pending for the technique described in this report. All other authors have reported that they have no relationships to disclose. Dr. Yoshida and Ms. Liu contributed equally to this work.

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**Abbreviations
and Acronyms**

CC = correlation coefficient
ECG = electrocardiogram
EGM = electrogram
ICD = implantable cardioverter-defibrillator
RMSd = root mean square difference
ROC = receiver-operating characteristic
VT = ventricular tachycardia

0.13) with post-infarction VT and implanted ICDs were referred for catheter ablation of VT. All patients had histories of myocardial infarction (anterior in 9 patients, inferior in 12 patients). Before ablation, all patients had failed to respond to antiarrhythmic drug treatment. Ten of the 21 patients were being treated with amiodarone at the time of ablation, 3 patients were being treated with mexiletine, 3 patients with metoprolol, 2 patients with dofetilide, 1 patient with sotalol, 1 patient with procainamide, and 1 patient with

lidocaine. Six of the patients were treated with a combination of amiodarone and mexiletine. All patients experienced frequent ICD discharges before ablation. At least 1 clinical VT was documented with a 12-lead ECG (GE Marquette MAC 5000 or 5500, GE Medical Systems, Milwaukee, Wisconsin) in 15 of 21 patients. In the remaining 6 patients, 7-lead telemetry recordings were available for at least 1 VT.

Electrophysiologic study, mapping, and ablation. The study protocol was approved by the institutional review board at the University of Michigan. After informed consent was obtained, a multipolar electrode catheter was inserted into a femoral vein and positioned in the right ventricular apex. A 7-F multipolar catheter was placed at the His bundle position. Programmed ventricular stimulation was performed from 2 right ventricular sites using up to 4 extrastimuli (1). After ablation, the entire stimulation protocol (with up to 4 extrastimuli) was repeated at the same 2 sites. Before ablation, a total of 124 distinct monomorphic VTs (mean cycle length 393 ± 103 ms; 47 left bundle branch block configuration and 77 right bundle branch block configuration) were induced in the 21 patients.

In all patients, a single spontaneous VT was recorded on a 12-lead ECG ($n = 15$) or on a 7-lead telemetry recording ($n = 6$) before the ablation procedure. Only the spontaneous VTs for which 12-lead ECGs were available were defined as clinical VTs. The configuration of the clinical VT was compared with that of the induced VT. A match was required in all 12 leads for the induced VT to be considered identical to the clinical VT. All VTs for which critical sites could be identified were targeted for radiofrequency ablation.

An electroanatomical mapping system (CARTO, Biosense Webster, Inc., Diamond Bar, California) was used in all patients, with an 8-F mapping and ablation catheter with a 3.5-mm irrigated-tip electrode and a 2-mm ring electrode separated by 1 mm (ThermoCool, Biosense Webster, Inc.). Intracardiac EGMs were filtered at 50 to 500 Hz. The intracardiac EGMs and leads V₁, I, II, and III were displayed on an oscilloscope and recorded at a speed of 100 mm/s. The recordings were stored on optical disc (EP Med

Systems, Inc., West Berlin, New Jersey). Systemic heparinization was maintained throughout the procedure.

Left ventricular access was obtained using a retrograde aortic approach. A left ventricular endocardial voltage map was constructed during sinus rhythm. Pace mapping was performed at low-voltage sites. Sites were considered distinct if they were separated by ≥ 5 mm. Low voltage was defined as a bipolar voltage < 1.5 mV. Dense scar was defined as a voltage < 0.5 mV. The low-voltage area was measured and correlated with the number of pace mapping sites. The sampling density was defined as the number of points per square centimeter of low-voltage tissue where pace mapping was performed. Sites surrounding dense scar with voltage of 0.5 to 1.5 mV were defined as border zones (2). Bipolar pace mapping was performed at 10 mA and a pulse width of 2 ms. If no capture occurred, the pacing output was increased progressively to 20 mA.

Radiofrequency energy was delivered as previously described (3). In brief, the power of radiofrequency energy was titrated to achieve an impedance drop of 10 Ω . The maximal temperature was 45°C. Radiofrequency energy was delivered at isthmus sites or at VT exit sites. At exit sites, radiofrequency energy was delivered at sites with matching pace maps for 60 to 120 seconds and/or until the capture threshold after ablation was > 10 to 20 mA. Additional lesions were created at adjacent sites with matching pace maps.

ICD manufacturers and ICD EGMs. The ICDs were from the following manufacturers: Medtronic, Inc. (Minneapolis, Minnesota) ($n = 10$); Boston Scientific Corporation (Natick, Massachusetts) ($n = 9$); and St. Jude Medical, Inc. (St. Paul, Minnesota) ($n = 2$). A can-to-coil configuration was chosen for the far-field EGM when possible (Medtronic and St. Jude Medical devices). The far-field and near-field EGMs both were analyzed for spatial resolution and for discriminatory value. The ICD EGM with the best spatial resolution was reported.

Determination of cutoff values on the basis of receiver-operating characteristic (ROC) curves. For comparison of clinical VTs (including the corresponding ICD EGMs) with other induced VTs and for comparison of pace maps (including the corresponding ICD EGMs) with the configuration of the targeted VTs, ROC curves were constructed. To do this, the real-time ICD EGM recordings during the induced VTs and during pace mapping were slaved from the ICD programmer into the electrophysiologic recording system (EP Med Systems, Inc.). The filter setting was adjusted between 0.05 and 300 Hz to obtain the closest match with the real-time analog ICD signals.

For data analysis, the ICD EGMs along with the corresponding 12-lead ECGs of the VTs and pace maps were exported and analyzed with a customized MATLAB version 7.5 (The MathWorks, Inc., Natick, Massachusetts) program in a manner described previously (4). In brief, the

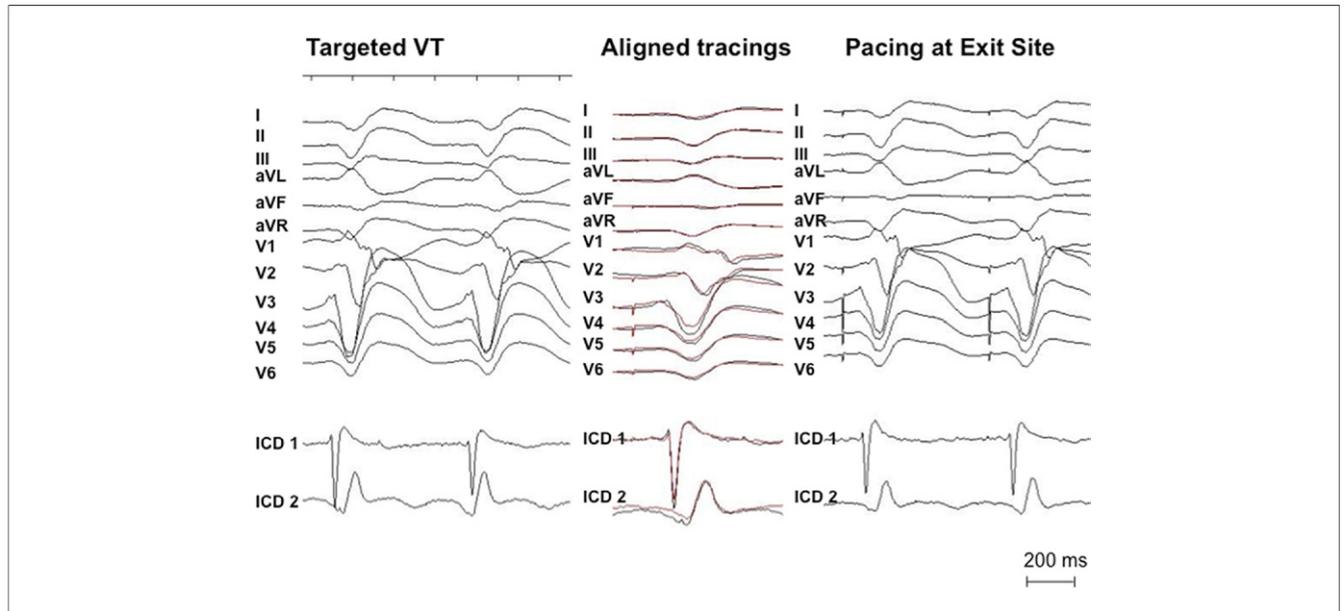


Figure 1 Targeted VT and Pace Mapping at Exit Site

(Left) The 12-lead electrocardiogram (ECG) of a clinical ventricular tachycardia (VT) that was targeted for ablation (template signals). **Below** are the near-field implantable cardioverter-defibrillator (ICD) electrograms (EGMs) (ICD 1) and the far-field ICD EGMs (ICD 2). **(Right)** A 12-lead pace map (test signals) at the VT exit site, with the ICD EGMs below. **(Middle)** The aligned test and template signals of the targeted VT and the pace map at the exit site for the 12-lead ECG tracings and the ICD EGMs. The correlation coefficient was 0.983, and the root mean square difference was 19.5.

12-lead ECG complexes of each VT (Fig. 1) and the corresponding far-field and near-field ICD EGMs were used as template signals against which pace maps were compared (test signals) (Figs. 1 and 2). The correlation coefficient (CC) and the root mean square difference

(RMSd) between the template and test signals were calculated. This was done for each ECG lead and for the far-field and near-field ICD EGMs. The CC and RMSd were then averaged for the 12 ECG leads to obtain a single value.

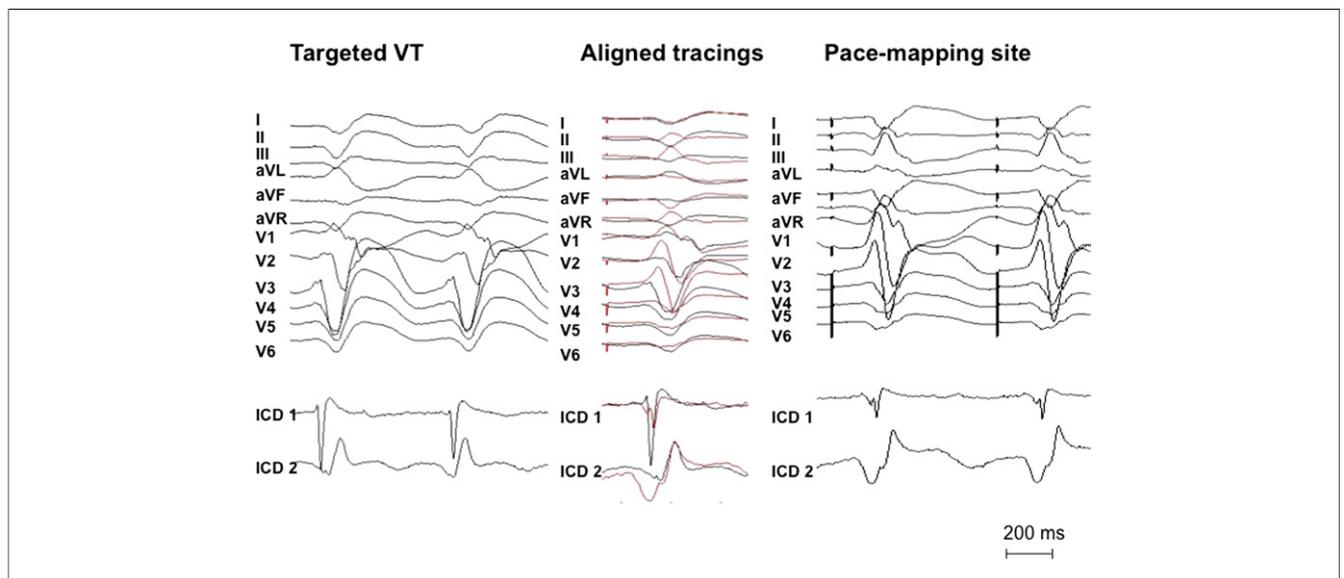


Figure 2 Targeted VT and Pace Mapping Distant From Exit Site

(Left) The 12-lead electrocardiogram (ECG) of a targeted ventricular tachycardia (VT), along with the implantable cardioverter-defibrillator (ICD) electrograms. **(Right)** A 12-lead pace map at a site that was at a distance of 30 mm from the VT exit site. **(Middle)** The aligned test and template signals of the targeted VT and the pace mapping site (shown in Fig. 3). The correlation coefficient was 0.442 and the root mean square difference was 56.3 for the 12-lead ECG, and these values were 0.864 and 50.6 for the ICD electrograms, respectively.

Two observers independently compared the 12-lead ECG pace maps post hoc and determined whether at least 10 of 12 leads matched with the targeted VT. Discrepancies were resolved by consensus. All pace mapping sites classified as matching during the procedure were confirmed to be matching in this post hoc analysis.

Pace maps were classified in a binary fashion as positive (≥ 10 of 12 matching leads) or negative (< 10 matching leads). With positive pace maps, the CC between the VT and the pace map is expected to be high, and the RMSd is expected to be low. Using the CC and RMSd statistics, we varied the discrimination thresholds and determined the ROC curve. The highest value of the sum of sensitivity and specificity was chosen as the cutoff value for classifying the pace maps in matching and nonmatching pace maps.

Spatial resolution. This cutoff value was subsequently used to determine the spatial resolution. The spatial resolution of pace mapping was defined as the endocardial area encompassing sites with matching pace maps. This area was measured on the electroanatomical map and encompassed the pacing sites with CCs that were equal to or higher than the cutoff CC value and represented the spatial resolution for pace mapping an exit site (Fig. 3). The spatial resolution was arbitrarily defined as 0 cm^2 if there was a single site with a matching pace map. An exit site was defined as a site where the pace map matched the targeted VT and where the stimulus-QRS interval was $\leq 30\%$ of the VT cycle length when pacing was performed during sinus rhythm. The distance of each pace mapping point from the VT exit site was measured on the electroanatomical map (Fig. 3), and this was correlated with the CC of the pace map from each site.

Far- and near-field ICD EGMs were analyzed in the same fashion as the 12-lead ECGs for assessment of the spatial resolution of the ICD EGMs.

To assess for beat-to-beat variability, between individual QRS configurations within a train of pace maps, we compared 3 paced QRS complexes with respect to the CC. The mean CCs between the comparisons were 0.98 ± 0.03 for 12-lead ECGs, 0.99 ± 0.02 for the far-field EGMs, and 0.97 ± 0.09 for the near-field EGMs. There were no significant differences in pairwise comparisons between the groups.

The discriminatory value of the ICD EGMs to differentiate clinical VT from other VTs. After the clinical VT was identified on the basis of comparison of ECGs, the ICD EGMs recorded during the clinical VT (template signal) were compared with the ICD EGMs (test signal) of all inducible VTs (Fig. 4).

To objectively compare the ICD EGMs of the clinical VT with ICD EGMs of other VTs, an ROC curve was constructed with the ICD EGM of the clinical VT as the template signal (Fig. 5), comparing it with other ICD EGMs on the basis of EGMs of nonmatching pace maps. A cutoff value was thereby determined. Then the CC of the ICD EGMs recorded during induced VTs that were pre-

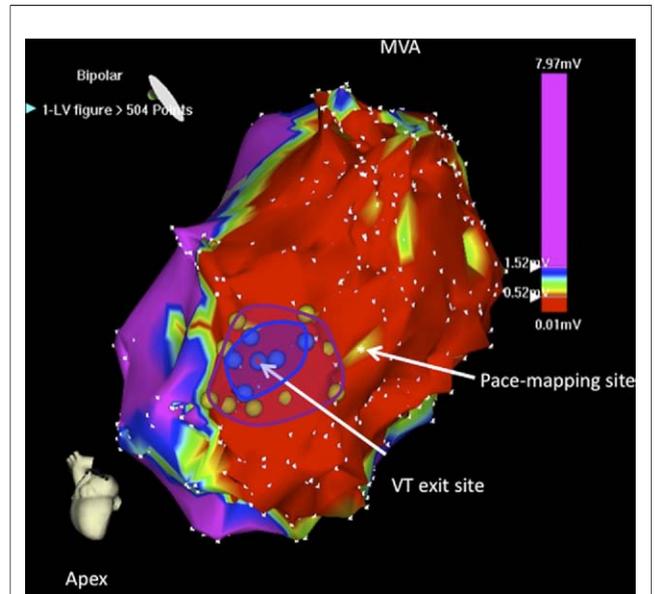


Figure 3 A Voltage Map in a Patient With Prior Inferolateral Myocardial Infarction

Shown is the inferolateral wall of the left ventricle (LV) with the apex and the mitral valve annulus (MVA). The low-voltage area covered 105 cm^2 , and pace mapping was performed from 62 sites, resulting in a sampling density of $0.61/\text{cm}^2$. The exit site (red asterisk) and the sites with matching 12-lead pace maps are indicated in blue. The correlation coefficient cut-off value on the basis of receiver-operating characteristic curve analysis was 0.965. The blue circle surrounds all the points with correlation coefficients equal to or higher than the cutoff value for this particular ventricular tachycardia (VT). The area contained within the blue line is 1.4 cm^2 , which was considered the spatial resolution of pace mapping for this particular VT. The spatial resolution of pace mapping based on implantable cardioverter-defibrillator electrograms is shown within the olive-colored tags and the olive line. The area contained in the olive line is 3.6 cm^2 . The pace mapping site illustrated in Figure 2 is indicated by an arrow and a white asterisk.

viously undocumented was compared with the template ICD EGMs of the clinical VT. If the CC was lower than the cutoff CC value of the template ROC curve, the ICD EGMs of the VT were considered different from the clinical VT (Fig. 4). The VT cycle lengths of the induced and clinical VTs also were compared.

To assess the accuracy of visual inspection, the real-time ICD EGMs of the induced VTs were compared with the stored ICD EGMs of the clinical VTs. Two observers reviewed the EGMs to distinguish clinical VTs from the previously undocumented VTs. Differences between the 2 observers were resolved by consensus.

Follow-up. All patients were seen every 3 to 6 months in an ICD device clinic. Treatment with amiodarone was discontinued in 2 patients, and mexiletine was discontinued in 5 patients. Treatment with other antiarrhythmic medications was continued after the procedure.

Statistical analysis. Continuous variables are expressed as mean \pm SD and were compared using Student *t* test. Discrete variables were compared using the Fisher exact test or by chi-square analysis as appropriate. Paired *t* tests were used to compare near- and far-field ICD recordings. One-

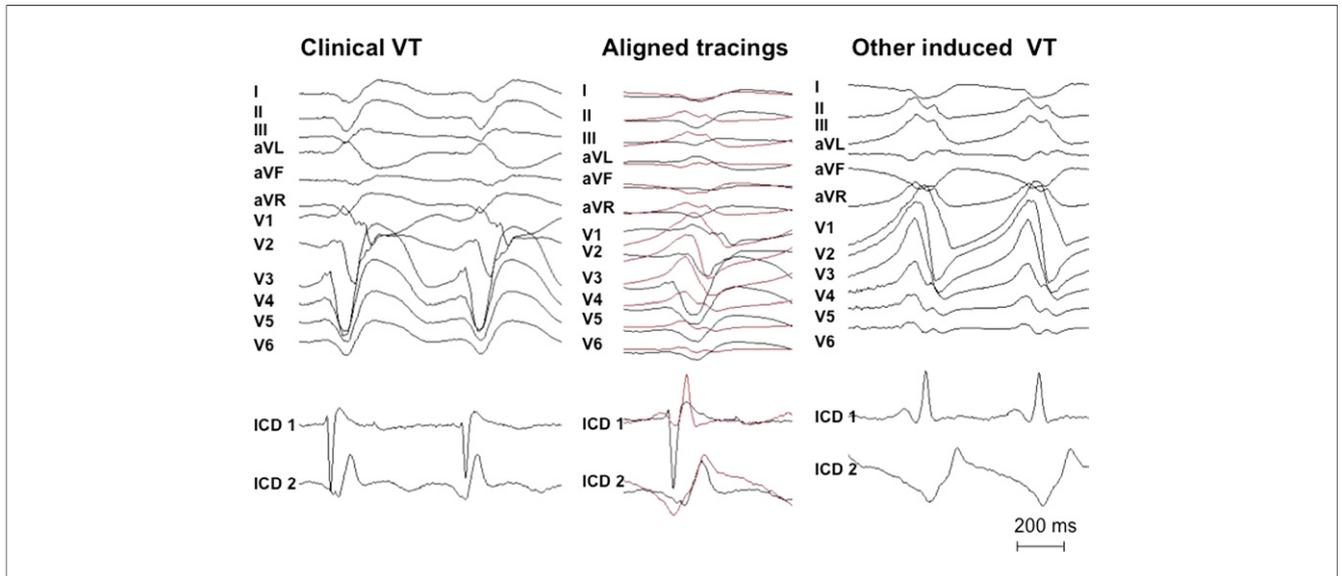


Figure 4 Clinical VT and Other Induced VT

(Left) The 12-lead electrocardiogram of a clinical ventricular tachycardia (VT) that was induced in the electrophysiology laboratory. Below are the near-field implantable cardioverter-defibrillator (ICD) electrograms (ICD 1) and the far-field ICD electrograms (ICD 2). (Right) A previously undocumented VT that was induced in the electrophysiology laboratory. The corresponding ICD recordings are shown below. (Middle) The aligned test and template signals of the clinical VT compared with an induced VT of uncertain clinical relevance. The correlation coefficient was 0.443 and the root mean square difference was 44.9 for ICD 1, and these values were 0.844 and 32.1 for ICD 2, respectively.

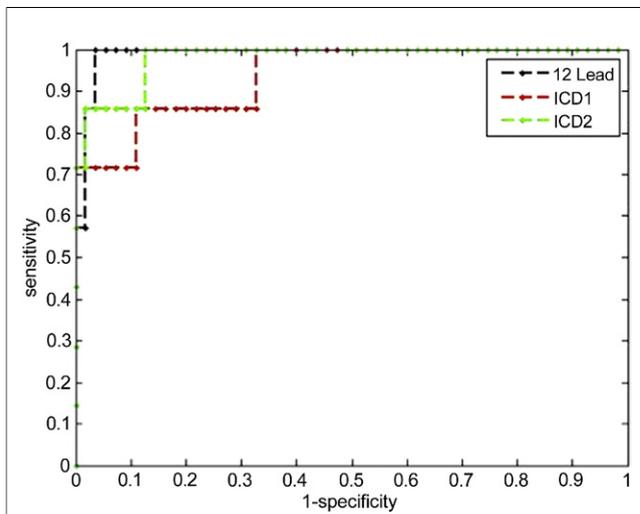


Figure 5 The ROC Curves for the Clinical VTs Based on the 12-Lead ECG Pace Maps (Test Signals) That Were Compared With the Clinical VTs (Template Signals)

The receiver-operating characteristic (ROC) curve of the 12-lead electrocardiogram (ECG) is shown as the **black dashed line**. The cutoff value for differentiating a matching pace map from a nonmatching pace map is 0.935. The ROC curves of the implantable cardioverter-defibrillator (ICD) electrograms (EGMs) are shown as **green** (far-field, ICD 2) and **red** (near-field, ICD 1) **dashed lines**. The cutoff values of these ROC curves are 0.965 and 0.977, respectively. When the ICD EGMs of the induced ventricular tachycardia (VT) of uncertain clinical relevance (Fig. 4) were compared with the ICD EGMs of the clinical VTs, the correlation coefficient was 0.443 for ICD 1 and 0.844 for ICD 2 EGMs. Because both correlation coefficients were lower than the cutoff value of the ROC curve of the template VT, this VT was considered different from the clinical VT on the basis of the ICD EGMs.

way analysis of variance was used to compare the means of multiple samples. Spearman's rank correlation was performed to correlate the spatial resolution of pace mapping with the distance to normal-voltage areas (Fig. 6). VT data are used throughout the report as the unit of analysis. The analytic method we chose assumed that the data from the VTs were independent from each other. ROC curves were constructed for the CC and the RMSd using the classification by observers of matching and nonmatching pace maps as the gold standard. Cohen's kappa value was determined to compare agreement between the observers comparing

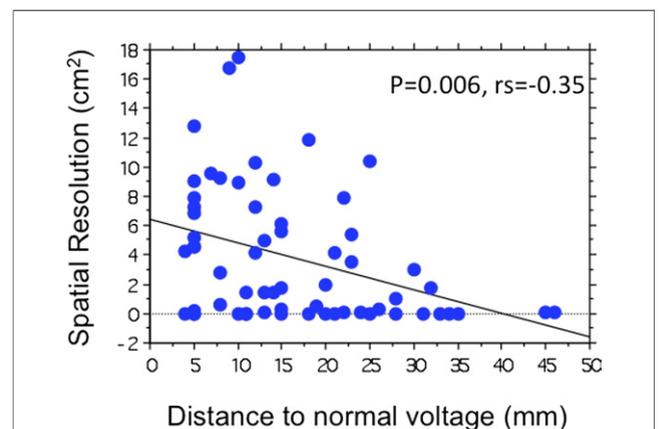


Figure 6 Correlation Between Spatial Resolution and Distance From Normal Tissue

The correlation between spatial resolution (y-axis in square centimeters) of the pace map on the basis of the 12-lead electrocardiogram and the distance from normal-voltage (>1.5 mV) tissue (x-axis in millimeters).

Table 1 Spatial Resolution of Pace Mapping for 12-Lead ECG and Far-Field ICD Signals

Patient # (ICD Manufacturer)	VT #	Cutoff Value for CC of 12-Lead ECG	Spatial Resolution of 12-Lead ECG (cm ²)	Cutoff Value for CC of ICD EGMs	Spatial Resolution of ICD EGMs (cm ²)
1 (Medtronic)	VT 1	0.894	9.1	0.915	31.3
	VT 3	0.944	1.4	0.918	1.4
2 (Boston Scientific)	VT 1	0.987	0	0.998	0
	VT 2	0.685	16.8	0.825	20.6
	VT 3	0.625	8.9	0.954	0
3 (Medtronic)	VT 1	0.836	1.4	0.987	1.4
	VT 2	0.932	0	0.998	0
	VT 3	0.921	9.6	0.963	19.4
4 (Medtronic)	VT 2	0.861	7.9	0.987	11.3
	VT 3	0.920	0	0.939	8.9
	VT 4	0.877	6.1	0.889	10.2
5 (Boston Scientific)	VT 1	0.835	9.2	0.972	15.9
	VT 4	0.867	7.3	0.923	7.4
	VT 7	0.893	5.4	0.929	8.3
6 (Medtronic)	VT 3	0.933	0	0.990	0
	VT 4	0.864	12.8	0.986	18.5
	VT 5	0.915	7.3	0.970	9.3
	VT 6	0.984	0	0.964	15.1
	VT 7	0.898	10.3	0.851	36.5
7 (Medtronic)	VT 1	0.765	0	0.988	0.1
	VT 2	0.867	0.2	0.953	0.7
	VT 3	0.920	0.6	0.971	12.6
8 (St. Jude Medical)	VT 3	0.954	11.9	0.989	11.9
	VT 4	0.962	0	0.987	10.2
	VT 5	0.933	0.3	0.968	4.6
	VT 7	0.905	9.0	0.831	15.6
	VT 8	0.977	0.1	0.920	20.1
9 (Boston Scientific)	VT 2	0.944	0	0.992	0
10 (Boston Scientific)	VT 3	0.902	4.1	0.971	4.1
	VT 4	0.890	4.2	0.803	33.2
11 (Medtronic)	VT 2	0.897	0	0.994	0
	VT 5	0.910	5.2	0.979	12.8
	VT 6	0.853	4.6	0.983	12.1
	VT 10	0.901	2.8	0.991	7.6
12 (St. Jude Medical)	VT 4	0.871	3.5	0.973	0.1
	VT 7	0.808	0	0.969	14.6
	VT 9	0.875	0.1	0.923	5.3
13 (Medtronic)	VT 1	0.908	1.8	0.945	1.8
	VT 4	0.935	1.4	0.965	3.6
	VT 5	0.961	0	0.996	0
14 (Boston Scientific)	VT 3	0.939	2.0	0.914	5.6
	VT 4	0.820	5.0	0.969	5.3
	VT 8	0.949	4.1	0.986	6.4
15 (Boston Scientific)	VT 1	0.931	6.9	0.994	6.9
	VT 4	0.829	0	0.979	0.1
	VT 6	0.663	7.9	0.952	10
16 (Boston Scientific)	VT 2	0.960	0.1	0.992	7.9
17 (Boston Scientific)	VT 1	0.832	10.4	0.978	17.5
	VT 3	0.960	0.1	0.991	8.5
	VT 5	0.833	17.5	0.945	22.7
	VT 7	0.942	1.8	0.984	6.8
	VT 8	0.894	1.0	0.968	33
18 (Medtronic)	VT 1	0.986	0.1	0.983	0.1
	VT 3	0.949	4.6	0.944	26.2
	VT 5	0.968	0	0.956	0.1

Continued on next page

Table 1 Continued

Patient # (ICD Manufacturer)	VT #	Cutoff Value for CC of 12-Lead ECG	Spatial Resolution of 12-Lead ECG (cm ²)	Cutoff Value for CC of ICD EGMs	Spatial Resolution of ICD EGMs (cm ²)
19 (Medtronic)	VT 1	0.868	3.0	0.970	10
	VT 2	0.868	0	0.829	35
20 (Boston Scientific)	VT 1	0.864	5.6	0.964	23.9
	VT 4	0.934	0	0.951	0.1
21 (Medtronic)	VT 2	0.905	0.5	0.728	12.6
	VT 5	0.986	0	0.997	0
	VT 7	0.933	0.3	0.979	0.1

Reported spatial resolution data for the 12-lead ECGs are based on the CC, and reported spatial resolution data for the ICD EGMs are based on the CC of the far-field EGMs. CC = correlation coefficient; ECG = electrocardiogram; EGM = electrogram; ICD = implantable cardioverter-defibrillator; VT = ventricular tachycardia.

ICD EGMs. Pairwise comparisons of the CC were performed when assessing the beat-to-beat variability of 3 paced beats. A p value <0.05 was considered significant.

Results

Mapping and ablation. A total of 124 VTs were induced by programmed stimulation. In 13 of 15 patients in whom the clinical VTs were documented on 12-lead ECGs, VTs with matching configurations were inducible by programmed stimulation. In 2 of 15 patients, the documented VTs could not be induced; however, there were frequent premature ventricular contractions during the procedure that matched the clinical VT on the basis of 12-lead ECGs.

Pace mapping was performed at 1,296 sites (a mean of 62 sites per patient, resulting in a sampling density of 0.75 points/cm² within low-voltage tissue) within low-voltage areas, and 62 distinct exit sites were identified. Matching pace maps were identified for an additional 15 VTs, but these were not considered exit sites because they had long stimulus-QRS intervals when pacing was performed during sinus rhythm. VT ablation sites were identified by entrainment mapping in 9 VTs and by pace mapping for 68 VTs.

The mean procedure time was 383 ± 97 min. The total amount of radiofrequency energy delivered was 72 ± 41 min.

After ablation, 10 VTs (8%) remained inducible. None of the clinical VTs remained inducible. Fourteen patients (67%) had no inducible VTs after ablation. Outcomes were no different if the exit site was identified on the basis of 10, 11, or 12 matching leads in the pace maps.

VT exit sites and scar. The VT exit sites were located within low-voltage areas and had a mean bipolar voltage of

0.44 ± 0.43 mV. They were located a mean of 16 ± 10 mm (range 4 to 46 mm) from sites with normal voltage (>1.5 mV). The total mean low-voltage area was 82 ± 35 cm² (bipolar voltage <1.5 mV) and 68 ± 31 cm² (bipolar voltage <1.0 mV).

Spatial resolution of pace mapping in scar using the 12-lead ECG. The spatial resolution of pace mapping for identifying the exit site of a VT was 3.8 ± 4.5 cm² (range 0 to 17.5 cm²) for the CC and 3.6 ± 4.5 cm² (range 0 to 18.3 cm²) for the RMSd (Table 1). When combined, the spatial resolution was 2.9 ± 4.0 cm². The spatial resolution for 10 or 11 of 12 matching pace maps was 2.6 ± 3.7 cm² and for 12 of 12 pace maps was 3.4 ± 4.3 cm²; this was not statistically different (p = 0.44). The distance between the exit site and the site where the pace map was obtained was inversely related to the CC (r_s = -0.51, p < 0.0001) and directly related to the RMSd (r_s = 0.51, p < 0.0001). The spatial resolution for the exit site was <1 cm² in 32 of 62 mapped VTs (52%). The spatial resolution correlated inversely with the distance from normal voltage tissue (r_s = -0.35, p = 0.006) (Fig. 6). The spatial resolution of pace mapping was better in dense scar than in the border zone (2.9 ± 3.5 cm² vs. 6.1 ± 5.3 cm², p = 0.03).

Table 2 Comparison of Far- and Near-Field ICD EGMs for Determination of Spatial Resolution of Pace Mapping

Variable	Far-Field EGM	Near-Field EGM	p Value
ROC cutoff value CC	0.949 ± 0.057	0.906 ± 0.093	0.006
ROC cutoff value RMSd	47.5 ± 41.8	51.8 ± 34.1	0.73
Spatial resolution (cm ²) for CC	10.1 ± 10.0	19.6 ± 16.4	<0.0001
Spatial resolution (cm ²) for RMSd	10.2 ± 10.5	19.0 ± 15.4	<0.0001

RMSd = root mean square difference; ROC = receiver-operating characteristic; other abbreviations as in Table 1.

Table 3 Comparison of ICD EGMs by Manufacturer

Variable	Medtronic	Boston Scientific	St. Jude Medical	p Value
Spatial resolution of far-field EGM for CC (cm ²)	9.7 ± 10.8	10.6 ± 10.1	10.3 ± 6.6	NS
Spatial resolution of far-field EGM for RMS (cm ²)	11.7 ± 11.6	7.6 ± 8.7	10.9 ± 10.7	NS
Spatial resolution of near-field EGM for CC (cm ²)	16.8 ± 16.0	22.2 ± 17.4	23.0 ± 14.7	NS
Spatial resolution of near-field EGM for RMS (cm ²)	17.2 ± 14.5	19.6 ± 16.7	25.5 ± 14.7	NS
Discrimination of clinical VT from other VTs	35/36 (97%)	12/12 (100%)	16/16 (100%)	NS

The p values compare data obtained from Medtronic versus Boston Scientific, Medtronic versus St. Jude Medical, and Boston Scientific versus St. Jude Medical devices. Abbreviations as in Tables 1 and 2.

Table 4 Discriminatory Value of ICD Signals

Patient #	Clinical VT (Spontaneous VT/Induced VT Cycle Length [ms])	ROC Cutoff Value for Clinical VT (ICD EGM)	Compared VTs (Cycle Length [ms])	CC of Clinical VT vs. Other VTs
1	VT 1 (430/500)	0.915	VT 2 (260) VT 3 (570)	0.235 0.219
2	NA	NA	NA	NA
3	VT 2 (540/660)	0.998	VT 1 (485) VT 3 (470)	0.536 0.855
4	VT 4 (430/350)	0.889	VT 1 (490) VT 2 (360) VT 3 (390) VT 5 (385)	0.686 0.544 0.943 0.784
5	VT 1 (470/640)	0.972	VT 2 (330) VT 3 (270) VT 4 (370) VT 5 (360) VT 6 (315) VT 7 (670)	0.873 0.424 0.463 0.178 0.835 0.826
6	VT 3 (350/350)	0.990	VT 1 (330) VT 2 (345) VT 4 (315) VT 5 (380) VT 6 (560) VT 7 (445) VT 8 (485)	0.398 0.803 0.782 0.857 0.939 0.561 0.409
7	Noninducible (330/NA)	NA	NA	NA
8	NA	NA	NA	NA
9	VT 2 (330/210)	0.992	VT 1 (280) VT 3 (280) VT 4 (340)	0.961 0.972 0.856
10	Noninducible (420/NA)	NA	NA	NA
11	VT 10 (400/485)	0.991	VT 1 (365) VT 2 (310) VT 3 (320) VT 4 (325) VT 5 (315) VT 6 (370) VT 7 (345) VT 8 (450) VT 9 (410)	0.984 0.887 0.104 0.935 0.841 0.973 0.867 0.896 0.176
12	VT 7 (310/300)	0.969	VT 1 (315) VT 2 (360) VT 3 (370) VT 4 (315) VT 5 (310) VT 6 (275) VT 8 (445) VT 9 (415) VT 10 (390)	0.861 0.259 0.280 0.822 0.937 0.111 0.923 0.286 0.067
13	NA	NA	NA	NA
14	NA	NA	NA	NA
15	VT 1 (500/530)	0.994	VT 2 (360) VT 3 (390) VT 4 (350) VT 5 (505) VT 6 (400)	0.975 0.971 0.952 0.989 0.960
16	NA	NA	NA	NA

Continued on next page

Table 4 Continued

Patient #	Clinical VT (Spontaneous VT/Induced VT Cycle Length [ms])	ROC Cutoff Value for Clinical VT (ICD EGM)	Compared VTs (Cycle Length [ms])	CC of Clinical VT vs. Other VTs
17	VT 1 (540/575)	0.978	VT 2 (360)	0.442
			VT 3 (390)	−0.156
			VT 4 (350)	0.664
			VT 5 (505)	0.892
			VT 6 (400)	0.749
			VT 7 (360)	0.798
			VT 8 (400)	0.908
			18	NA
19	VT 1 (520/570)	0.970	VT 2 (460)	0.968
20	VT 1 (295/395)	0.964	VT 2 (340)	0.843
			VT 3 (325)	−0.525
			VT 4 (220)	−0.336
21	VT 5 (620/510)	0.997	VT 1 (470)	0.974
			VT 2 (450)	−0.437
			VT 3 (340)	−0.362
			VT 4 (330)	0.987
			VT 6 (770)	−0.282
			VT 7 (420)	0.982

The VT cycle length is indicated in **bold** if the comparison VT has a cycle length that is within 10% of the clinical VT cycle length.
NA = not applicable; other abbreviations as in Tables 1 and 2.

Spatial resolution of ICD EGMs within scar. Using far-field ICD EGMs, the spatial resolution of pace mapping for identifying the exit site of a VT was $10.1 \pm 10.0 \text{ cm}^2$ (range 0 to 36.5 cm^2) for the CC and $10.2 \pm 10.5 \text{ cm}^2$ (range 0 to 35 cm^2) for the RMSd (Table 2). When near-field and far-field data were combined, the spatial resolution improved to $8.9 \pm 9.0 \text{ cm}^2$. The spatial resolution of pace mapping with ICD EGMs was $<1 \text{ cm}^2$ for 19 of 62 of the mapped VTs (31%). The spatial resolution of the 12-lead ECGs correlated with the spatial resolution of the ICD EGMs ($r_s = 0.54$, $p < 0.0001$). The far-field ICD EGMs at 81% of the exit sites displayed equal or better spatial resolution for pace mapping (indicating higher accuracy) than the near-field EGMs (Table 2). The ICD brand did not affect the spatial resolution of the ICD EGMs (Table 3).

To assess whether there were determinants of the spatial resolution of the ICD EGMs, they were grouped in 3 categories of spatial resolution $\leq 3.0 \text{ cm}^2$ ($n = 18$), >3.0 to $\leq 10 \text{ cm}^2$ ($n = 19$), and $>10.0 \text{ cm}^2$ ($n = 25$). None of the analyzed parameters, infarct location (anterior vs. inferior, basal vs. apical), pace mapping score (12 of 12 vs. 10 or 11 of 12 matches), or bundle branch block, was associated with any of the categories.

Discriminatory role of ICD EGMs. The clinical VTs were inducible in 13 of the 15 patients with 12-lead documentation of spontaneous VT (Table 4). In 2 of 15 patients, the clinical VTs were not inducible, but frequent premature ventricular contractions with identical configurations were present. The template signals of the clinically documented VTs were compared with a total of 64 test signals of VTs that were induced during programmed stimulation (Table 4). The ICD EGMs were almost as accurate as the 12-lead

ECGs for differentiating the clinical VT from previously undocumented VTs. All clinical VTs were accurately identified on the basis of the 12-lead ECG of the clinical VT, whereas 63 of 64 VTs (98%) were correctly distinguished from the clinical VT on the basis of computerized analysis of the ICD EGMs. In 8 of 13 patients (67%) in whom the clinical VT was induced, the cycle length of the clinical VT was within 10% of the cycle length of at least 1 other induced VT that was not previously documented. Furthermore, the induced clinical VT differed by $>50 \text{ ms}$ from the previously documented VT in 8 of 13 patients in whom the clinical VT could be induced. A mean of 1.4 ± 1.8 induced VTs per patient overlapped in cycle length with the clinical VTs. The cycle length of the clinically documented VTs was $433 \pm 98 \text{ ms}$, compared with the cycle length of the induced clinical VT, which was $459 \pm 138 \text{ ms}$ ($p = 0.3$). The mean of the absolute difference in cycle length between the documented VT and the induced clinical VT was $75 \pm 49 \text{ ms}$ (range 0 to 170 ms) (Table 4).

The ICD EGMs of the clinical VTs and the previously undocumented VTs were compared by visual inspection (including the ICD EGMs of the noninducible patients who had premature ventricular contractions with identical configurations to the clinical VTs). By visual inspection of the ICD EGMs, 96% of the clinical VTs were accurately differentiated from the 64 previously undocumented VTs. The kappa value of the 2 readers for agreement of the compared EGMs was 0.83.

Follow-up. During a mean follow-up time of 10 ± 5 months, 1 of the 21 patients had recurrent VT and required another ablation procedure. The other patients did not have VT on the basis of their ICD interrogations.

Discussion

When ECG documentation of clinical VTs is not available in patients with ICDs, the ICD EGMs can be used to discriminate the clinical VTs from other VTs that are induced in post-infarction patients. Furthermore, ICD EGMs may be helpful for pace mapping when a clinical VT is not inducible during a mapping procedure.

ICD EGMs and the clinical VT. Identification of clinically relevant VTs in post-infarction patients undergoing VT ablation is difficult unless a 12-lead ECG of the VT is available. The VT cycle lengths of induced VTs that have not been previously documented frequently overlap with the VT cycle length of the clinical VT, making the VT rate less reliable as a differentiating criterion. Identification of the clinical VT is important because this may be the only VT requiring therapy. Furthermore, a particular VT may respond to antitachycardia pacing, and the identification of a particular VT on the basis of ICD EGMs might allow antitachycardia pacing for that VT, thereby avoiding unnecessary ICD discharges. This study demonstrates that ICD EGMs are capable of identifying an induced VT as having occurred clinically. This can be accomplished in the electrophysiology laboratory by simple visual inspection without offline computerized analysis.

Spatial resolution of pace mapping for identifying a VT exit site. The spatial resolution of pace mapping using 12-lead ECGs in patients without structural heart disease is approximately 1.8 cm², or smaller than the size of a dime (about 2.5 cm²). In this study, the spatial resolution within post-infarction scar was approximately double that size. This region of interest constitutes <5% of the total endocardial scar area. In most patients, the spatial resolution of pace mapping was <1.0 cm² for at least 1 VT.

The area with matching pace maps varied greatly from patient to patient and within a given patient from 1 VT to another. More sites were ablated for VTs in which the spatial resolution was poor. The closer the exit site was to normal tissue, the lower the spatial resolution (i.e., the area of a matching pace map for an exit site within a border zone was larger than for exit sites within dense scar tissue). Therefore, an ablation strategy that targets the border zone is likely to require more ablation than a strategy targeting sites within scar.

The large variability in spatial resolution of pace mapping within scar is most likely due to tissue heterogeneity and the variable presence of surviving myocardial bundles.

ICD EGMs and the identification of the VT exit site. The spatial resolution of the 12-lead ECG to identify the exit site of a VT with pace mapping within scar tissue is approximately the size of a penny (about 2.83 cm²). The spatial resolution of pace mapping using the ICD EGM is not as good and is larger than a silver dollar (about 5.8 cm²). Of note is that ICD EGMs are generated by a single lead in

which the vector may be combined with that of the ICD generator, depending on the ICD manufacturer. When pacing was performed within the exit site of a VT, the ICD EGM was always identical to the ICD EGM recorded during the clinical VT. In most of the patients, the spatial resolution of the ICD EGM was excellent (<1.0 cm²) for at least 1 VT. Therefore, the ICD EGM can be used for pace mapping of a clinical VT that has been recorded by an ICD but not with an ECG. A major limitation of the ICD EGMs, however, is that the spatial resolution is lower than the spatial resolution of the 12-lead ECG for identifying a VT exit site. Despite this limitation, ICD EGMs can be useful for identifying a VT exit site. The value of ICD EGMs in targeting noninducible VTs remains to be determined.

Study limitations. ICD EGMs from different manufacturers were analyzed. This was not a homogeneous patient population, and the results need to be confirmed in a larger study. Although the ICD EGMs of 98% of previously undocumented VTs were accurately distinguished from the clinical VTs by computerized analysis, if multiple clinical VTs are present and their VT exit sites are in close proximity, ICD EGMs might not accurately differentiate between these VTs. Another limitation is that the influence of body position on ICD EGMs was not assessed in this study.

The pulse strength required for capture might affect the ICD EGMs. A retrograde P-wave also could affect the 12-lead configuration of the targeted VTs. Pace mapping was used to target most of the VTs, and there is a discrepancy between the total number of targeted VTs and the number of sites with matching pace maps. Yet the majority of VTs were no longer inducible after ablation. Similar results were found in a prior study (3). A possible explanation is shared VT circuits (5). A final limitation is that the sampling density for pace mapping was a mean of 0.75 points/cm². A higher sampling density might have improved accuracy data.

Clinical implications. In post-infarction patients with VT, ICD EGMs can be used to differentiate clinical VTs from inducible VTs of uncertain clinical relevance. Although the spatial resolution of pace mapping based on ICD EGMs is inferior to the spatial resolution of 12-lead ECGs, they may be useful for determining whether an ablation catheter is located at a VT exit site. In this study, the value of ICD EGMs as a surrogate for 12-lead ECGs was demonstrated in a quantitative fashion by offline computerized analysis. However, simple visual analysis of the ICD EGMs was found to be as accurate as computerized analysis for differentiating clinical VTs from previously undocumented VTs. Therefore, during mapping of VTs in the electrophysiology laboratory, visual comparisons of stored and real-time ICD EGMs provides a simple and practical technique for identifying clinical VTs.

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