Localization of the Site of Origin of Postinfarction Ventricular Tachycardia by Endocardial Pace Mapping
Body Surface Mapping Compared With the 12-Lead Electrocardiogram

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Background. The purpose of this study was to assess the value of body surface mapping and the standard 12-lead ECG in localizing the site of origin of postinfarction ventricular tachycardia (VT) during endocardial pace mapping of the left ventricle.

Methods and Results. Simultaneous recordings of 62-lead body surface QRS integral maps and scalar 12-lead ECG tracings were obtained in 16 patients with prior myocardial infarction during a total of 26 distinct VT configurations and during subsequent left ventricular catheter pace mapping at 9 to 24 different endocardial sites. Anatomic pacing site locations were computed by means of a biplane cineradiographic method and plotted on a polar projection of the left ventricle. The QRS integral map and the QRS complexes of the 12 standard leads of each VT morphology obtained in a particular patient were compared independently with the different paced QRS integral maps and paced QRS complexes of the 12-lead ECG generated in that same patient. The stimulus site locations of the best matching paced QRS integral map and paced QRS complexes of the 12-lead ECG were indicated on the polar projection and subsequently compared with the endocardial location of the corresponding site of VT origin identified during intraoperative (surgical ablation) or catheter activation sequence mapping (catheter ablation). The localization resolution of pace mapping was established separately for each electrocardiographic technique by computing the size of endocardial areas with similar morphological features of the QRS complex. Pace mapping advocated with body surface mapping or the 12-lead ECG enabled adequate reproduction of the VT QRS morphology in 24 of 26 VTs (92%) and 25 of 26 VTs (96%), respectively. Activation sequence mapping identified the site of origin in 12 of 26 previously observed VT configurations (46%). Ten and 11 VTs were localized by activation sequence mapping and pace mapping combined with body surface mapping or the 12-lead ECG, respectively. Pace mapping applied with body surface mapping identified the site of origin correctly (distance ≤2 cm) in 8 of 10 compared VTs (80%); an adjacent site (distance between 2 and 4 cm) or a disparate site (distance ≥4 cm) was identified in the remaining 2 of 10 VTs (20%). Pace mapping used with the 12-lead ECG localized the site of origin correctly in 2 of 11 VTs (18%); the site of origin was identified correctly next to an additional adjacent site in 5 of 11 VTs (55%); and an adjacent site or a disparate site was found in 1 of 11 VTs (9%) and 2 of 11 VTs (18%), respectively. The difference in localization accuracy of both electrocardiographic techniques was statistically significant (P=.02). The mean size of endocardial areas where a comparable QRS morphology was obtained during pace mapping was 6.0±4.5 cm² with the application of body surface mapping and 15.1±12.0 cm² with the use of the 12-lead ECG.

Conclusions. These results demonstrate that application of the 62-lead instead of the 12-lead ECG during endocardial pace mapping enhances the localization resolution of this mapping technique and enables more precise identification of the site of arrhythmogenesis in the majority of compared postinfarction VT episodes. (Circulation. 1993;88[part 1]:2290-2306.)

Key Words • mapping • electrocardiography • tachycardia • morphology
Surgical1-12 or catheter13-22 ablation of drug-refractory ventricular arrhythmias is aimed at selective elimination of the arrhythmogenic substrate. Careful prior localization of the site of origin of ventricular tachycardia (VT) is crucial to the successful clinical outcome of these ablative procedures. Mapping the endocardial sequence of electrical activation during VT is currently the accepted technique for accurate identification of the area of arrhythmogenesis and can be performed either by catheter during electrophysiological study23-26 or in the exposed heart during surgery.1,12-27,34 However, during sequential activation mapping VT may appear to be nonmappable because of hemodynamic compromise or transient appearance of the arrhythmia.3,5,7,12,35 During surgery, one may additionally be confronted with mapping time restrictions and the inability to obtain arrhythmia induction.1,3,5,7,10-12 Catheter pace mapping14,16,25,36-38 during sinus rhythm has been proposed as an alternative localization technique and is based on comparison of the surface ECG configuration produced during endocardial stimulation at multiple left ventricular sites, with the surface ECG morphology of spontaneous or induced VT. Application of the 12-lead ECG during pace mapping has demonstrated that although an approximation of the ventricular region where VT originates may be obtained, precise localization of the site of origin appears not to be possible.36

The present study was undertaken to determine whether the use of 62 torso leads instead of the standard 12 leads would improve the VT localization procedure during left ventricular endocardial pace mapping in comparison with VT localization obtained by intraoperative or catheter endocardial activation sequence mapping.

Methods

Patients

This study includes a population of 16 consecutive patients with previous myocardial infarction and drug-refractory ventricular arrhythmias who were selected to undergo mapping-guided antiarrhythmic surgery (15 subjects) or radiofrequency (RF) catheter ablation (1 subject) (Table 1). All patients had recurrent spontaneous episodes of symptomatic sustained monomorphic VT. VT was defined as sustained if it lasted over 30 seconds or if hemodynamic deterioration necessitated termination; VT was defined nonsustained if it ended spontaneously within 30 seconds and did not provoke hemodynamic compromise. Myocardial infarction occurred from 1 month to 26 years before the electrophysiological investigation. Location of myocardial infarction was based on standard Q wave analysis of the 12-lead ECG; anterior infarcts were present in 9 patients, inferior in 5 patients, anterior and inferior in 1 patient, and lateral in 1 patient. Antiarrhythmic drugs were administered in 5 surgical patients during the preoperative electrophysiological study and were discontinued before the operative procedure. Prior informed consent was obtained from each patient.

Electrophysiological Arrhythmia Induction and Left Ventricular Endocardial Pace Mapping

A 6F quadripolar catheter was introduced percutaneously into a femoral vein and positioned at the right ventricular apex or outflow tract to induce VT by programmed electrical stimulation. The arrhythmia terminated either spontaneously or was interrupted by programmed stimulation or electrical cardioversion (hemodynamic instable VT) after 12-lead and 62-lead ECG recordings had been obtained simultaneously. A second 6F or 7F quadripolar catheter was advanced into the left ventricle through a femoral or brachial artery and was used for left ventricular pace mapping. Bipolar pacing was performed with the distal electrode pair (interelectrode distance, 0.5 cm). Current intensities were just above the diastolic stimulation threshold and ranged from 0.5 to 16 mA. To prevent renewed arrhythmia initiation and hemodynamic compromise, pacing was conducted at stimulation rates varying between 100 and 130 beats per minute. The pace mapping sequence was not individually directed by immediate evaluation of the paced electrocardiographic sequences. Given the purpose of the study, a random pace mapping procedure with off-line electrocardiographic analysis was preferred. Care was taken, however, that all major areas of the left ventricle were explored by maneuvering the catheter to a minimum of 9 widely distributed endocardial locations. The time required for a pace mapping procedure ranged from 25 to 60 minutes (mean, 43±11 minutes). The endocardial stimulus site locations were computed with respect to anatomic reference points and displayed on a polar projection of the left ventricle using a previously described cineangiographic method.26,39,40 Briefly, 45° left anterior oblique (LAO) and 45° right anterior oblique (RAO) fluoroscopic images were simultaneously acquired at every pacing site. Biplane contrast cineventriculography was carried out to determine the position of the anatomic reference points: the center of the mitral and aortic valve ring (MVR and AVR) and the left ventricular apex. Location-specific cylindrical coordinates used in the polar representation of each pacing site were subsequently calculated by computer after digitizing end-diastolic fluoroscopic frames. LAO and RAO fluoroscopic images and the corresponding polar projection, including computed locations of 16 pacing sites obtained in a patient with anterior myocardial infarction, are illustrated in Fig 1.

Electrocardiographic Data Collection

The method of acquisition, processing, and display of 62-lead ECG recordings has been documented

<table>
<thead>
<tr>
<th>Table 1. Patient Characteristics</th>
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<tr>
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<tr>
<td>No. of narrowed coronary arteries†</td>
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<tr>
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<td>Clinical arrhythmia</td>
</tr>
<tr>
<td>VT</td>
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<td>VT+VF</td>
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LV indicates left ventricular; clinical arrhythmia, spontaneously occurring arrhythmia; VF, ventricular fibrillation; and VT, ventricular tachycardia.

*Mean±SD; †>50% angiographic lumen reduction; ‡severe segmental dyskinesis on biplane cineventriculograms.
In summary, a portable mapping system$^{43}$ and radiotransparent carbon electrode array$^{44}$ were used to sample unipolar ECG tracings simultaneously from 62 torso sites (panel A in Fig 2)$^{45-49}$ during monomorphic VT and during subsequent left ventricular pace mapping. Since the standard precordial leads were also incorporated in the 62-electrode array, the 12-lead ECG was acquired simultaneously with body surface mapping. Waveforms of single beats were converted to a digital format at a rate of 500 Hz and stored on floppy disk. An interpolation algorithm ensured correction of linear baseline drifting or offset differences after manual selection of an isoelectric time instant before the QRS complex and after the T wave. In the absence of an isoelectric interval (ie, with extremely rapid VT episodes), a waveform with a steep initial Q wave was first identified from the 62 individual waveforms. The time instant for baseline correction was subsequently chosen just before the QRS onset of this selected waveform. An average of one lead recording per map demonstrated unsatisfactory signal quality and was substituted by a computed value obtained from neighboring lead recordings. Thereafter, hard-copy body surface maps (isopotential maps) were produced every 2 milliseconds during the QRS complex and the early part of the ST-T interval. QRS onset was determined at the time instant at which one of the extreme amplitudes on the body surface maps reached $\pm 0.2$ mV, whereas QRS offset was visually defined at the J-point. Finally, data reduction was performed by computing an integral map (isointensity map)$^{50}$ of the total QRS complex (panel B in Fig 2) for each VT configuration and each pacing sequence. The rationale for the use of the integral of the total QRS instead of the integral of a fixed initial QRS interval (eg, first 40 milliseconds) has been explained recently.$^{40,51}$

Printouts of scalar 12-lead ECG tracings were generated with 10-mm vertical and horizontal spacing representing 0.5 mV and 200 milliseconds (equaling a paper speed of 50 mm/s), respectively.

Fig 1. Fluoroscopic diagrams of RAO (right anterior oblique) and LAO (left anterior oblique) views are shown along with the endocardial polar projection of the left ventricle. The end-diastolic contour of the ventricular cavity, the outline of the mitral and aortic valve ostium (dashed lines), and the positions of pacing sites and anatomic reference points—apex, center of the mitral valve ring (MVR), and aortic valve ring (AVR)—are indicated in the biplane images. In the polar projection, the anatomic reference points and pacing sites are demonstrated together with the locations of the endocardial quadrants, the anterior (APM) and posterior (PPM) papillary muscles; the estimated position of the postinfarction scar is depicted by the hatched area. The outline of the polar projection marks the position of the plane of the mitral valve ostium, the center defines the position of the apex, and the radius corresponds with the mitral valve apical axis. Compare the opposite locations of pacing sites 10 and 8 at the septal and lateral quadrants vs sites 15 and 3 at the junction of the anteroseptal quadrants and the posterior quadrant of the polar projection with the corresponding left and right vs top and bottom locations of these sites in the LAO image.
Fig 2. A, Schematic representation of the lead sites and their relation to the anatomic landmarks of the human thorax. Lead site selection was based on optimal limited lead array configurations designed by Lux et al., supplemented by the 6 standard precordial lead sites (open circles overlying the heart silhouette) and optimal lead sites developed by other investigators. The carbon electrodes are mounted in flexible straps with a minimal interelectrode distance of 3.3 cm. The straps are applied vertically on the chest using the location of the V₁ lead site as a reference. Double adhesive tape is used to secure their position throughout the mapping procedure. The anterior electrode straps are topped by a connecting wire to remove the upper part of the straps immediately from the chest when defibrillation after placement of external pads is performed. The suprasternal notch marks the upper side and the umbilicus marks the bottom side of the lead array. The three electrodes on the right margin are represented once again on the left margin (column of three open circles) for the purpose of map display. B, The format of body surface map display is demonstrated by an example of a QRS integral map obtained during sustained monomorphic ventricular tachycardia with a cycle length of 460 milliseconds in a patient with previous anterior myocardial infarction. The interval for integral computation is depicted by the hatched area in the V₁ waveform below the map. A schematic demarcation of the position of the sternum and spinal column is shown at the left and right top sides of the map, respectively. The spatial locations of the extremes are indicated by plus (maximum) and minus signs (minimum). The voltage amplitudes of the extremes are expressed in mVms at the bottom right margin of the map. Lead sites with equal time integral values are connected by isointegral lines; solid lines in the shaded area of the map delineate positive values, dashed lines represent negative values, and the dotted line indicates the zero level. The incremental steps between isointegral lines are linear; their number depends on the individual extreme amplitudes with a maximum of 20 contour lines per map. The incremental steps in this particular example are 2 mVms (positive) and 8 mVms (negative). Notice that the QRS integral map reflects a right bundle branch block pattern (positive polarity in V₁) with slightly superior and rightward-directed electrical forces.
Activation Sequence Mapping

The method of intraoperative endocardial mapping has already been documented in detail. In short, during normothermic cardiopulmonary bypass, an incision was made in the postinfarction scar of the left ventricle to gain access to the ventricular cavity. An inflatable balloon covered with 64 electrodes (approximately interelectrode distance, 1.2 cm) was subsequently inserted into the left ventricular cavity. A ridge over the long axis of the balloon placed between the papillary muscles ensured a stable recording position and served as an anatomic landmark. After VT induction by means of programmed electrical stimulation, 64 unipolar endocardial electrograms were sampled simultaneously with reference to a needle electrode in the left shoulder. Bipolar reference electrograms were additionally recorded from both the ventricles and the right atrium. The site of earliest endocardial activation (the VT origin) was subsequently identified and indicated on the left ventricular polar projection after computing local activation times for each of the 64 electrodes. To allow morphological comparison with the preoperatively documented clinical VT configurations, standard surface ECG leads I, II, and III were also acquired during the intraoperatively induced arrhythmia. The latter surface ECG measurements were obtained simultaneously with the intracardiac recordings.

One VT morphology was ablated by catheter using RF energy. For this particular VT, catheter endocardial activation sequence mapping of the left ventricle was performed immediately after the pace mapping procedure. Local electrograms were obtained at a total of 19 distinct endocardial sites. The earliest diastolic activation (ie, the site of VT origin) preceded the onset of the surface QRS complex with 155 milliseconds. At the site of origin, slow conduction and concealed entrainment were additionally observed. Digitized biplane end-diastolic fluoroscopic images were subsequently used to compute the spatial location of the VT origin on the polar projection of the left ventricle.

Data Analysis

The analysis in this study deals with spontaneously occurring monomorphic VT (clinical arrhythmia), which was previously documented by means of 12-lead ECG recordings and could be reproducibly induced during electrophysiological study. When two or three VT morphologies were found in a single patient, the arrhythmia was defined as distinct if one or more of the following criteria were met: (1) contralateral bundle branch block morphology, (2) frontal plane QRS axis demonstrating more than 90° divergence, and (3) clearly different QRS configuration in at least one of the 12 standard leads associated with a difference in the tachycardia cycle length. An intraoperatively mapped VT episode was considered to have the same morphology as the clinical arrhythmia when surface leads I, II, and III were identical.

Pace mapping and body surface mapping. In each patient, the VT QRS integral map was visually and mathematically compared with the QRS integral maps that were generated in that same patient during pace mapping. Visual comparison was performed by two observers who were unaware of the localization results obtained by pace mapping in combination with the 12-lead ECG. This comparison included a detailed evaluation of the resemblance in map pattern, thereby considering the location and mutual orientation of the extremes and the morphology of the zero line. Mathematical comparison was used to corroborate the visual analysis and included the calculation of a correlation coefficient between a pair of maps (ie, between the VT QRS integral map and every individual paced QRS integral map). This was achieved by advocating a normalization procedure for the compared pair of maps and subsequently computing a single correlation coefficient as the inner product of the 62 lead values of both maps. Replication of the VT integral pattern was considered adequate if the paced pattern was visually nearly identical or very similar and if the r value was ≥.80. The stimulus site location of the best matching paced QRS integral map was thereafter delineated on the endocardial polar projection of the left ventricle. The localization accuracy of pace mapping applied in conjunction with 62-lead ECG mapping was established by determining the distance between the selected stimulus site and the site of origin obtained during intraoperative (surgical ablation) or catheter activation sequence mapping (catheter ablation). Sites were described to be identified correctly with pace mapping when the distance between them was ≤2 cm, adjacent if they were between 2 and 4 cm apart, and disparate if the distance was ≥4 cm.

To establish the localization resolution of pace mapping combined with body surface mapping, we additionally selected paced QRS integral maps demonstrating a pattern that was visually similar to the pattern of the best matching paced QRS integral map with regard to the above described spatial map features; pattern resemblance was mathematically established through the calculation of correlation coefficients. The cineradiographic method used for the computation of the endocardial pacing site locations was subsequently applied to estimate the size of endocardial areas where similar QRS patterns were generated. This was achieved by calculating the circular area between two pacing sites with comparable electrocardiographic features (ie, between the best matching pacing site and the site with a comparable paced pattern); the distance between these two pacing sites determined the diameter of the circular area. The largest area was selected when more than two pacing sites produced comparable map features.

Pace mapping and 12-lead ECG. The QRS complexes of the VT configuration(s) and paced sequences obtained in each patient were visually compared by two observers who were unaware of the localization results obtained by pace mapping combined with body surface mapping. This comparison was conducted according to the grading system developed by Morady et al. A comparison was graded “excellent” if the VT and paced QRS complexes were identical or very similar in all 12 lead tracings, “good” if the VT and paced QRS complexes were similar in 9 of 12 lead tracings, and “poor” if the VT and paced QRS complexes were similar in only 8 of 12 lead tracings. The endocardial stimulus site location(s) at which the best VT match was obtained were marked on the polar projection of the left ventricle. The localization results were evaluated by determining the dis-
tance between a pacing site selected on the basis of the 12-lead ECG with the site of origin obtained during intraoperative (surgical ablation) or catheter activation sequence mapping (catheter ablation) as well as with the stimulus site selected when using the 62-lead ECG. A localization was defined to be correct when the distance between two compared sites was ≤2 cm, adjacent if the distance was between 2 and 4 cm, and disparate if the distance was ≥4 cm.

The localization resolution of pace mapping used in conjunction with the 12-lead ECG was assessed by determining for each VT morphology the paced configurations that were graded as “excellent” and “good” or, when no such level of replication was obtained, the paced morphologies that were graded as having a “poor” resemblance with the VT configuration. The circular area between two pacing sites with such equal grading characteristics was then computed according to the procedure that was used to determine the localization resolution of pace mapping with body surface mapping. The largest area size was chosen if equal grading was obtained for the QRS complexes produced at more than two pacing sites. Thus, an approximation of the dimensions of the endocardial area where comparable 12-lead ECG morphologies were generated during pacing was acquired.

Statistical testing. Statistical comparison of the localization accuracy of pace mapping combined with body surface mapping or the 12-lead ECG was performed with Fisher’s exact test. A two-tailed P value of <.05 was considered significant. All data are given as mean±SD.

Results

Ventricular Tachycardia Features

QRS integral maps of 26 distinct monomorphic VTs (1 to 3 per patient) and 9 to 24 endocardial pacing sequences (mean, 15.8±4.7) were obtained during electrophysiological study of 16 patients. Tachycardia characteristics are listed in Table 2. Right bundle branch block (RBBB) morphology (positive polarity in V1) was acquired in 19 VTs; left bundle branch block (LBBB) morphology (negative polarity in V1) was obtained in 7 VTs. The VT cycle length varied between 230 and 600 milliseconds (mean, 367±101 milliseconds). Fourteen of the 26 VTs (54%) observed during electrophysiological study were either nonsustained and not well reproducible or caused rapid hemodynamic deterioration requiring external DC shock cardioversion.

Endocardial Pace Mapping Results

Body surface mapping. Visual and mathematical comparison of VT QRS integral maps with paced QRS integral maps revealed a matching pattern in 24 of 26 tachycardias (92%). Table 2 contains the different sites of VT origin (ie, the endocardial location of the selected stimulus sites) and the correlation coefficient between matching VT and paced QRS integral patterns. Correlations of the matching VT and paced integral maps ranged from r=.80 to r=.98 (mean, .91±.06). A matching paced QRS configuration was not assessed in two VTs with a LBBB pattern (VT C in patient 11 and VT A in patient 12) due to the inability of obtaining a pacing site where adequate replication (r≥.80) of the VT QRS integral configuration could be obtained. For 13 VT morphologies obtained in 11 patients, paced QRS integral maps with similar patterns as the selected best matching paced QRS integral maps (mean, r=.96±.03) were found at 1 to 3 other sites per VT morphology (mean, 1.4±0.6 sites). The resolution of pace mapping used in combination with body surface mapping was subsequently determined by estimating the size of the endocardial area where similar patterns were produced; these area sizes ranged from 1.8 to 16.3 cm² (mean, 6.0±4.5 cm²).

Twelve-lead ECG. A total of 25 of 26 VTs (96%) were localized on the basis of a visual comparison of the QRS complexes of the 12-lead ECG recorded during VT and pace mapping according to the grading system of Morady et al14 (Table 2). VT replication was considered “excellent” in 10 of 25 VTs (40%), “good” in 11 of 25 VTs (44%), and “poor” in 4 of 25 VTs (16%). No satisfactory VT reproduction could be achieved in one VT with a LBBB morphology (VT A in patient 12). For 19 VTs acquired in 13 patients, an equal grading of VT replication was obtained at 2 to 9 pacing sites per VT configuration (mean, 3.7±1.6 sites). The resolution of pace mapping applied together with the 12-lead ECG was obtained by approximating the size of the endocardial areas at which an equal grading of VT reproduction was acquired. The area sizes appeared to range from 2.0 to 45.2 cm² (mean, 15.1±12.0 cm²).

Comparative Analysis of Ventricular Tachycardia Localization by Endocardial Pace Mapping Using Body Surface Mapping or the 12-Lead ECG and Activation Sequence Mapping

During surgery, 11 of 25 previously observed VT configurations (44%) were induced and mapped in 10 of 15 patients (Table 2). It was not possible to induce any clinical arrhythmia after ventriculotomy in 4 patients, while technical difficulties precluded the acquisition of satisfactory intraoperative mapping data in 1 patient. One VT morphology was mapped by catheter before RF ablation (VT A in patient 16) (Table 2).

The results of pace mapping combined with body surface mapping were compared with the activation mapping data of 10 VTs; the activation mapping data of 2 tachycardias (VT C in patient 11 and VT A in patient 12) were not compared because localization by pace mapping was not achieved. The site of origin was localized correctly by pace mapping and body surface mapping in 8 of 10 VTs (80%), and an adjacent site or a disparate site was identified in the remaining 2 of 10 VTs (20%) (Table 2).

The localization results obtained by pace mapping combined with the 12-lead ECG were compared with the activation mapping data of 11 VTs. Pace mapping used with the 12-lead ECG enabled correct identification of the site of origin in 2 of 11 VTs (18%). Localization of the site of origin and an additional adjacent site was obtained in 6 of 11 VTs (55%); an adjacent site or a disparate site was identified in 1 of 11 VTs (9%) and 2 of 11 VTs (18%), respectively (Table 2). It should be noted that 1 of the latter 2 disparate localizations (VT C in patient 11) could not be localized with body surface mapping because the quantitative matching criteria (r≥.80) were not met. This tachycardia was, however, graded as a “good” VT replication on the basis of the 12-lead ECG characteristics. Statistical comparison of the performance of both electrocardiographic techniques...
revealed that the difference in localization accuracy was significant \( P = .02 \).

An evaluation of the localization results obtained by applying the 12-lead ECG during pace mapping relative to the localization data acquired with the use of body surface mapping during pace mapping was performed in 24 of 26 VTs. Application of the 12-lead ECG during pace mapping localized the same site that was identified by body surface mapping during pace mapping (designated as the correct site) in 9 of 24 VTs (38%), the correct site together with an adjacent site in 9 of 24 VTs (38%), the correct site and in addition an adjacent and a disparate site in 2 of 24 VTs (8%), an adjacent site in one of 24 VTs (4%), an adjacent site together with a disparate site in 2 of 24 VTs (8%), and a disparate site in one of 24 VTs (4%) (Table 2).

Representative examples of body surface QRS integral maps and scalar 12-lead ECG recordings of matching VT and endocardially paced sequences along with the polar representation of the corresponding stimulus sites and the site of VT origin assessed with activation sequence mapping are represented in Figs 3 to 5. Individual pacing sites are referred to numerically for descriptive purposes. Fig 3 features a VT with a RBBB morphology obtained in a patient with a prior inferior myocardial infarction (patient 9) that was localized to the basal part of the lateral wall during intraoperative mapping. Endocardial pace mapping using body surface mapping identified the site of origin correctly (ie, within 2 cm of the intraoperatively determined site of VT origin); pacing at only one site at the basal lateral wall (site 2) produced a QRS integral map with an adequate

### TABLE 2. Mapping Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Infarct Location</th>
<th>Mode of Therapy</th>
<th>VT ID</th>
<th>Config</th>
<th>CL, ms</th>
<th>Appearance at EPS</th>
<th>BSM Site of Origin*</th>
<th>BSM Correlation VT-Pacing (r)</th>
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</table>

*Topographic description of the location of the site of origin was performed according to long-axis endocardial quadrants (septum, anterior, lateral, and posterior), each divided into three short-axis transverse sections (apical, mid, basal); locations at the apex were described as superior or inferior. tGrading system defined in "Methods."

Config indicates configuration; EPS, electrophysiological study; CL, cycle length; VT, ventricular tachycardia; BSM, body surface mapping; AM, activation mapping; AMI, anterior myocardial infarction; LMI, lateral myocardial infarction; IMI, inferior myocardial infarction; SA, surgical ablation; CA, catheter ablation; LBBB, left bundle branch block; RBBB, right bundle branch block; S, sustained; NS, nonsustained; RHD, rapid hemodynamic deterioration; NMMP, no matching mapping pattern; IIF, intraoperative induction failure; ITF, intraoperative technical failure; A, adjacent; C, correct; and D, disparate.
quantitative match with the VT QRS integral map (ie, \(r \geq .80\)). Use of the 12-lead ECG during pace mapping resulted in the identification of 2 different sites at which the paced QRS configuration was visually graded as a “good” replication of the VT QRS morphology. These sites included the above-mentioned site at the basal lateral wall (site 2) but also an adjacent site (ie, distance between 2 and 4 cm of the intraoperatively assessed site of VT origin) at the middle lateral wall (site 1). Figs 4 and 5 illustrate 2 tachycardia configurations with different RBBB morphology acquired in a patient with a previous anterior myocardial infarction (ie, VT A and VT B in patient 10). Intraoperative mapping data could only be obtained in the VT displayed in Fig 4 and revealed that this tachycardia arose from the middle section of the anterior wall. Endocardial pace mapping combined with body surface mapping correctly localized the origin of this tachycardia; it was demonstrated that the best visual match and highest correlation coefficient \((r = .98)\) with the VT QRS integral map was achieved during pacing at the midanterior wall (site 3). In contrast, pacing at 4 different sites located at the middle (site 1-3) and basal part of the anterior wall (site 4) produced QRS complexes in the 12 standard leads that were all graded as an “excellent” reproduction of the QRS complexes during VT. Thus, application of the QRS morphology of the 12-lead ECG during pace mapping in this particular tachycardia resulted in the identification of a somewhat larger endocardial area that included the site of VT origin but also an adjacent site. Localization of the second VT (Fig 5) using pace mapping in conjunction with body surface mapping showed that the QRS integral map generated at just 1 pacing site at the basal lateral wall (site 2) met the criteria of an adequate quantitative replication of the VT QRS integral map \((r = .81)\). With the 12-lead ECG applied during pace mapping, the same site was identified at the basal lateral wall (site 2), although an adjacent site at the basal anterior wall (site 1) was additionally selected. The standard lead QRS configurations obtained at both pacing sites had received an equal “poor” grading of VT replication. Fig 6 demonstrates an RBBB tachycardia with a
rightward-oriented electrical axis that was obtained in a patient with prior anterior myocardial infarction (patient 3). The origin of this tachycardia was localized to the middle anterolateral wall during intraoperative mapping and to a disparate endocardial location at the basal anteroseptal wall during pace mapping using both the 12-lead or 62-lead ECG. This result was obtained despite the fact that an adequate level of VT replication was achieved with both electrocardiographic techniques; the best matching paced QRS integral map expressed very similar spatial features and a high correlation coefficient (r=.94) with the VT QRS integral map, and the paced 12-lead ECG tracings produced at the same site (site 2) and a nearly identical site (site 1) were graded as a “good” VT reproduction. An additional pacing sequence is shown that appeared in retrospect to be generated at an endocardial location (site 3) corresponding with the intraoperatively localized site of VT origin. It is interesting to observe a clearly different RBBB morphology of both the QRS integral map and the QRS complexes of the 12-lead ECG characterized by a superior and rightward-oriented axis, a low correlation with the VT QRS integral pattern (r=.42), and configuration differences of the QRS complexes in 7 of the 12 standard leads compared with the QRS complexes during VT.

Fig 3. The QRS integral map and 12 scalar waveforms of a sustained monomorphic right bundle branch block ventricular tachycardia (VT) acquired in patient 9 with a prior inferior myocardial infarction are presented. The selected best matching paced QRS integral map and two pacing sequences at which a “good” grading of VT replication was obtained with the 12-lead ECG are also displayed. The computed correlation coefficient between the VT QRS integral map and the matching paced QRS integral map is indicated at the right bottom corner of the pair of maps. Corresponding pacing site locations are referred to numerically and are marked on the endocardial polar projection (for general description see the legend of Fig 1). Pace mapping in combination with body surface mapping localized a site at the basal lateral wall as the presumed VT origin (site 2); the 12-lead ECG applied during pace mapping detected the same site next to an adjacent site (site 1) at the middle lateral wall. It can be seen in the polar projection that intraoperative VT localization confirmed the result obtained by pace mapping combined with body surface mapping or the 12-lead ECG. It should be noted that use of the 12-lead ECG during pace mapping also resulted in the identification of a site adjacent to the site of VT origin.
Fig 4. QRS integral maps and 12-lead ECG recordings of a ventricular tachycardia (VT) configuration with a right bundle branch block morphology and their best matching paced sequences are shown. This nonsustained monomorphic tachycardia was induced in patient 10, who had undergone a remote anterior myocardial infarction. The correlation coefficient between the pair of maps is indicated at the right side of the bottom map. Of note is the very high qualitative and quantitative correspondence of the VT QRS integral map with the matching paced QRS integral map. One may observe that the QRS complexes of all 12 leads obtained at four pacing sites are very similar to the QRS complexes of the 12-lead ECG obtained during VT (ie, grading as “excellent” VT reproduction). The left ventricular polar projection (for general description see the legend of Fig 1) illustrating the location of the corresponding pacing sites (referred to numerically) and the site of origin assessed during intraoperative mapping is indicated on the right upper side. Compared with the VT origin mapped intraoperatively, pace mapping in combination with body surface mapping enabled correct identification of the arrhythmogenic focus at the middle anterior wall (site 3). Pace mapping in conjunction with the 12-lead ECG on the other hand identified a larger endocardial area that contained sites 1-4 (middle and basal anterior wall). The VT QRS integral map is obtained from the same tachycardia episode as the example shown in Fig 2.
Discussion

Localization of the Site of Origin of Ventricular Tachycardia by Endocardial Pace Mapping

Reentry is generally considered to be the arrhythmogenic mechanism responsible for VT occurring in the setting of remote myocardial infarction. Intracardiac activation sequence mapping of postinfarction VT has demonstrated that the earliest electrical activity may be recorded epicardially or even intramurally but usually appears to be confined to a small focal area on the endocardium of the left ventricle. This circumscribed area represents the exit site of the reentrant circuit and is commonly referred to as the site of origin of VT. Pace mapping is founded on the principle that stimulation at the site of origin will mimic the electrocardiographic pattern of VT. Although the use of pace mapping was first reported during open heart surgery, Josephson et al introduced the technique combined with 12-lead ECG recordings in the catheterization laboratory as a corroborative method to localize the arrhythmogenic origin in patients with rapid or noninducible VT. The procedure involves successive stimulation at several scattered locations in the left ventricle and comparison of the paced QRS patterns with the QRS pattern of the clinical arrhythmia until adequate replication is obtained.

Present study. Since prior observations have shown that VT localization with the use of pace mapping and the 12-lead ECG results in the identification of rather large endocardial regions, we have conducted the current investigation to systematically evaluate whether the use of 62 chest leads would allow more detailed
localization of the arrhythmogenic origin in postinfarct VT. In individual patients, an independent selection was made of the best matching paced QRS integral map to classify VT QRS integral maps and of the best matching paced QRS complex of the 12-lead ECG to classify the standard lead QRS complexes obtained during VT. After subsequently applying a quantitative biplane cineradiographic method to determine the accurate endocardial location of each pacing site, we were able to identify the site of origin in 24 of 26 VTs (92%) with body surface mapping and 25 of 26 VTs (96%) with the 12-lead ECG. Comparison with endocardial activation sequence mapping was performed in 10 VTs (body surface mapping) and 11 VTs (12-lead ECG) to evaluate the accuracy of the pace mapping results. With pace mapping used in combination with body surface mapping, the site of origin was obtained correctly (distance ≤2 cm) in 8 VTs (80%); an adjacent site (distance between 2 and 4 cm) or a disparate site (distance ≥4 cm) was found in the remaining 2 VTs (20%). Application of pace mapping together with the 12-lead ECG yielded less accurate results in that correct localization of the site of origin was achieved in only 2 of 11 VTs (18%), whereas the site of origin was localized correctly in combination with an additional adjacent site in 6 of 11 VTs (55%), and a sole adjacent or disparate site was localized in 3 of 11 VTs (27%). The difference in localization accuracy of body surface mapping and the 12-lead ECG was statistically significant.

Previous studies: Josephson et al applied endocardial pace mapping in combination with 12-lead ECG recordings to determine the value of this technique in localizing the site of VT origin. After prior identification of the tachycardia origin by catheter activation sequence mapping, these authors reported that pacing at the site of origin resulted in a QRS morphology similar to the QRS morphology observed during VT, although pacing at adjacent areas could demonstrate similar QRS configurations. They concluded that the scalar QRS morphology of the 12-lead ECG allows localization of the origin of VT to a relatively large area of 20 to 25 cm². The present results underline these findings and also show that a significantly higher resolution can be obtained when the spatial QRS integral configuration obtained in multiple surface leads is analyzed. We also observed similar QRS integral patterns and comparable QRS morphologies of the 12-lead ECG at closely spaced or adjacent pacing sites, but the size of the corresponding endocardial area was much smaller with body surface mapping (mean approximated size of 6.0±4.5 cm²) than with the 12-lead ECG (mean approximated size of 15.1±12.0 cm²). The lower resolving power of the standard ECG is furthermore illustrated by a comparison of the localization results of both electrocardiographic techniques. The 12-lead ECG enabled identification of the same site as determined by body surface mapping (referred to as correct site in Table 2) in 83% of the VTs. However, an adjacent site (Figs 3 to 5) and sometimes even a disparate site were additionally delineated as the presumed area of arrhythmogenesis in 50% of the latter subset of VTs. Kadish et al recently evaluated the resolution of pace mapping in combination with the 12-lead ECG by performing unipolar stimulation at 1 catheter site per patient and visually examining the QRS complexes produced by each pole of a quadripolar catheter (interelectrode distance, 0.5 cm). Visual examination concentrated on assessing minor configuration differences (notch, new small component, amplitude change of individual component, or change in QRS shape), major configuration differences (new large component, marked change in amplitude of existing component, or two minor changes), and peak to peak changes in amplitude. Their patient group included 29 patients, of which 11 had remote myocardial infarction. They concluded that the QRS complex obtained during pacing at sites that were 1.5 cm apart was mostly similar if major configuration differences were concerned; sites that were 0.5 cm apart could usually be differentiated if minor differences in configuration and amplitude were taken into consideration. It was suggested that application of the latter approach would allow identification of an area of 1 to 4 cm². We applied 12-lead ECG analysis criteria that were previously developed by Morady and coworkers and that seem to relate to the "major configuration differences" criteria used in their recent report. Despite application of these criteria, we obtained a considerably lower spatial resolution with the standard ECG. This might be understood by the fact that in the study by Kadish et al, only one catheter position in each patient was compared and the maximum distance between pacing sites was 1.5 cm. It should also be realized that although they performed pacing at areas with abnormal electrical activity, sites at which the stimulation threshold was more than 5 mA were not included. Thus, areas with dense infarct scarring requiring higher current amplitudes to obtain ventricular capture were not analyzed. A considerable number of pacing sites where a current amplitude of more than 5 mA was needed were found in 13 of our 16 patients (2 to 6 sites per patient). This might also partly explain why the presence of a prior infarct in the report by Kadish et al resulted in fewer QRS amplitude differences but did not influence QRS configuration differences among compared paced sequences. Lin et al demonstrated in a canine model of experimental myocardial infarction that tachycardia localization by ventricular pace mapping used in conjunction with orthogonal surface ECG leads can be performed with a resolution of 1 cm in 88% of the VTs. Since activation mapping and pace mapping were carried out with an array of 64 endocardial and epicardial electrodes, these authors did not consider it likely that a higher resolution could be achieved in humans with fluoroscopically directed single site pace mapping procedures.

In the above-mentioned study by Josephson et al, it was reported that next to similar QRS patterns, substantially different QRS configurations could also be obtained during pacing at adjacent sites; changes from RBBB to LBBB patterns were noted during pacing at adjacent sites at the left side of the ventricular septum. We have previously performed left ventricular pace mapping in patients without structural cardiac disease or with remote myocardial infarction and developed a reference data base of specific 62-lead QRS integral map patterns after pacing at 25 (no myocardial disease), 22 (inferior infarction), and 18 (anterior infarction) circumscribed left ventricular segments. During pacing at adjacent segments in patients with or without myocardial disease, we observed the largest differences in QRS pattern at the septum, in particular at the anteroseptal and posteroseptal regions. At the latter regions, the paced QRS complex demonstrated changes...
from a superior to an inferior axis and from an RBBB to an LBBB pattern. Since this finding (1) did not depend on the presence or absence of structural heart disease, (2) was very location dependent, and (3) could be reproduced in different patients, we feel that this is a normal phenomenon that can be explained in terms of the intrinsic regional differences in electrocardiographic sensitivity of the left ventricle.\textsuperscript{58-59} This means that subtle differences in the location of ectopic activation onset in an electrocardiographically sensitive region such as the septum will result in relatively large effects on the surface ECG.

In a study by Kuchar et al.,\textsuperscript{37} endocardial pace mapping was applied with the purpose of developing criteria for analysis of the QRS pattern observed during VT in patients with previous myocardial infarction. Pacing was performed at 24 endocardial regions that were identified on a biplane fluoroscopic coordinate grid. A localization algorithm was developed on the basis of the paced QRS polarity obtained in 8 selected leads from...
the standard 12-lead ECG. With the use of this algorithm, these authors were able to localize the site of tachycardia origin to the correct ventricular region in 39% and to an adjacent region in 36% of the studied VTs. Although the latter approach does not consider the interindividual variation in the electrocardiographic effects of coronary artery disease (different infarct locations), it holds the important practical advantage that once a general set of analysis criteria is designed, application of a complete pace mapping procedure in each individual patient is no longer required.

Pace mapping has also been used as a complementary tachycardia localization technique in the catheter ablative treatment of VT. Morady et al. used the method as an adjunct to activation sequence mapping and predominantly applied DC shocks to endocardial sites where local activity preceded the surface QRS complex and where pacing produced a 12-lead ECG morphology similar to the morphology observed during VT. Similarly, Klein et al. more recently targeted delivery of RF energy at sites where both the earliest endocardial activation as well as the best pace map with the 12-lead ECG were obtained. Garan et al. used pace mapping in combination with 12-lead ECG recordings as a directive procedure for the identification of a specific endocardial region of interest. Detailed activation mapping was thereafter performed at this particular area to determine the ultimate site of DC shock delivery. Apart from the application during sinus rhythm, pace mapping has also been performed during the tachycardia in selected patients with hemodynamically well tolerated VT to identify areas with properties of slow conduction and where entrainment or concealed entrainment could be obtained in addition to optimal electrocardiographic VT replication in the 12 standard lead recordings. 

Discordance Between Catheter Pace Mapping and Activation Sequence Mapping

In the present report we were not able to attain a high localization resolution with body surface mapping in all the studied tachycardias. Pace mapping identified a site of origin at an adjacent or a disparate endocardial site in 2 of 10 VTs (20%) that could be compared with activation mapping data; these two VTs were recorded in patients who underwent antiarrhythmic surgery. It appeared retrospectively that in both instances pacing had also been conducted during electrophysiological study at a site nearly identical to the intraoperatively determined site of origin. This resulted in paced QRS integral patterns and paced QRS complexes of the 12-lead ECG that were clearly different from the QRS morphology during VT. For instance, in patient 3 (Fig 6), pacing at the middle anterolateral wall (site of origin determined during surgery) produced a QRS integral map demonstrating superior and rightward-directed electrical forces, whereas the VT QRS integral map displayed rightward-oriented forces. Since the site where an ectopic ventricular impulse exits the infarcted zone and propagates into the normal myocardium as well as the subsequent location of epicardial breakthrough determine to a large extent the general morphology of the surface QRS complex, it is very likely that there was a different route of conduction during VT than during ventricular pacing. In the situation described above, this would imply that the tachycardia activation wave front showed preferential conduction from its endocardial origin at the middle anterolateral wall toward a disparate exit site at the anterosaposal base of the heart, where it initiated activation of the bulk of noninfarcted myocardium and then broke through onto the epicardium. On the other hand, during pacing at the site of origin, the activation wave front must have followed a more direct route toward the overlying epicardium. The different route of the impulse during VT might have been caused by the transient presence of rate-related areas of functional block and fixed anatomic boundaries resulting from inhomogeneous formations of electrically inactive fibrous tissue within the healed infarct. Thus, the best matching paced sequence was most probably produced during pacing at the exit site of the VT impulse. Interestingly, the two VTs for which an adjacent and a disparate site of origin was predicted by pace mapping combined with body surface mapping were obtained in patients with prior anterior myocardial infarction. A possible explanation can be found in the fact that in general, a larger amount of the left ventricle is destroyed with anterior as opposed to inferior infarction, which in turn may lead to a more disturbed activation pattern as was suggested by Miller et al. These authors designed an algorithm to...
localize the site of tachycardia origin on the basis of the 12-lead ECG obtained during VT. They found that the presence of anterior compared with inferior infarction was one of the factors associated with a lower predictive accuracy of their localization algorithm.

**Limitations**

Next to the inherent restrictions of fluoroscopy in assessing the precise anatomic location of pacing sites,37,40,64 application of this technique in an anatomically distorted left ventricle might introduce additional localization inaccuracies.64 We have attempted to overcome the majority of such limitations by applying a quantitative cineradiographic method.26,39 This method offers the advantage of a relatively high localization accuracy that is not seriously impeded by the presence of anatomic derangements such as postinfarction scar and ventricular aneurysm. Hauer et al26 localized VT in patients with prior myocardial infarction within an area of 3 cm² after comparing catheter with intraoperative activation sequence mapping.

A different analysis approach was adopted for the two compared electrocardiographic techniques. Analysis of the 12-lead ECG pace mapping data was based on qualitative criteria; the body surface mapping results were additionally subjected to quantitative methods of analysis. It would have been preferable to have adopted similar sophisticated quantitative analysis techniques with the 12-lead ECG data. However, at present, no such technique is available for analysis of scalar ECG data. In fact, the difficulties one may encounter with detailed morphological analysis of scalar waveforms can be noted in Fig 4. When using the visual analysis criteria of minor configuration differences developed by Kadish et al,36 it may be observed that pacing at site 2 gives rise to a notch in the second part of the QRS complex of four remote leads (ie, leads II, III, aVL, and aVF) that is also present in the VT QRS complexes of the same leads. Based on these visual criteria, one might deduce that the 12-lead ECG produced at site 2 demonstrates the best match with the VT pattern. This pacing site is, however, situated 2.8 cm from the pacing site that is actually closest to the VT origin (ie, site 3). Thus, the use of such subtle morphological criteria for 12-lead ECG waveform analysis does not result in improved localization performance. We believe that the failure of the 12-lead ECG to predict only the correct site (ie, within 2 cm of the VT origin) as opposed to predicting the correct site next to additional adjacent or disparate sites does not appear to be analysis dependent but is more likely the result of an intrinsically lower resolving power caused by the limited number of surface leads used.

The amount of time needed to perform pace mapping can be regarded as a limitation for the clinical application of this technique.36 It may be difficult to obtain reliable capture and to manipulate the catheter to all the different endocardial areas until satisfactory reproduction of the tachycardia morphology is obtained. Despite these technical limitations, we never required more than an hour (mean, 43 ± 11 minutes) to perform a complete pace mapping procedure. However, an important reduction in time can be obtained with the additional application of 12-lead37,63-65 or multiple-lead ECG localization algorithms40,51 to identify the endocardial region of interest before the pace mapping procedure. Finally, it should be realized that comparison of the VT morphologies preoperatively and intraoperatively is limited by the availability of only three surface ECG leads during surgery and the possibility of intraoperative ECG signal distortion due to altered thoracic (sternotomy) and ventricular geometry (ventriculotomy).

**Implications**

Although catheter activation sequence mapping is a reliable and useful method,24-26 its clinical application is hampered by the fact that induced VT sometimes appears nonmappable. VT may be poorly tolerated hemodynamically or present only as a nonsustained tachycardia that is not well reproducible.4,7,11,12,39 In both situations, localization results remain incomplete because there is not enough time to obtain a sufficient number of recordings from the various endocardial regions. Josephson et al35 reported a 40% incidence of nonmappable VT episodes in the catheterization laboratory. Similar disadvantages are met during intraoperative activation mapping when local electrograms are recorded sequentially.3,5,12 Moreover, mapping time during surgery is restricted, and among other factors, the inability to induce VT after left ventriculotomy is an important problem that is often encountered.1,3-5,7,10-12 The significance of mapping all observed distinct VT morphologies has been stressed in the literature.66-68 Hargrove and Miller66 showed that the success rate of antiarrhythmic surgery is closely related to the percentage of completely mapped VT configurations. In addition, it has been reported that multiple VT configurations occur frequently and originate often from widely separate endocardial sites,3,7,66 in which case an adapted surgical approach to obtain more adequate control over arrhythmia recurrence might be required.24,7 Lawrie et al68 demonstrated the importance of comprehensive VT localization before electrophysiologically guided surgical ablation since incomplete preoperative mapping was associated with an unfavorable prognosis. In the present study, we observed that 54% of the studied clinical VT morphologies showed nonmappable characteristics during electrophysiological study and that 56% of the clinical VT configurations could not be induced or adequately mapped during surgery. With the use of body surface mapping during endocardial pace mapping, we were able to accurately localize (ie, within 2 cm of the endocardial site of VT origin) 80% of the VT morphologies including multiple tachycardia configurations that were associated with widely separate sites of origin.

In addition to localization of VT foci before antiarrhythmic surgery, the present method could also be particularly useful in the treatment of VT by means of catheter-mediated RF or DC shock ablation.12-23 Because of the localized character of transcatheter electrical current delivery and the resultant compact lesion size,69 a great deal of attention must be given to the precise localization of the site of VT origin. Pace mapping is usually applied to complement the results of activation sequence mapping13,14,18,19,21,22 or is used as part of a procedure that involves localization of the zone of slow conduction.16,17,20-22,61 The use of 62 instead of 12 surface leads during pace mapping clearly offers more refined comparison of the VT and paced QRS
morphology and could contribute to the improvement of the clinical efficacy of catheter ablative techniques.

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References

Localization of the site of origin of postinfarction ventricular tachycardia by endocardial pace mapping. Body surface mapping compared with the 12-lead electrocardiogram.
A SippensGroenewegen, H Spekhorst, N M van Hemel, J H Kingma, R N Hauer, J M de Bakker, C A Grimbergen, M J Janse and A J Dunning

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