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ELECTROPHYSIOLOGIC STUDIES

Electrocardiographic Localization of the Site of Origin of Ventricular Tachycardia in Patients With Prior Myocardial Infarction

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The utility of the 12 lead electrocardiogram (ECG) in identifying the site of origin of sustained ventricular tachycardia in patients with previous myocardial infarction was studied. A new mapping grid, based on biplanar fluoroscopic imaging of the heart, was utilized for the definition of left ventricular endocardial sites. On the basis of QRS configurations resulting from left ventricular endocardial pacing at disparate sites in 22 patients (Group I), ECG features that were specific for particular sites were identified and used to construct an algorithm. Apical and basal sites were differentiated by the QRS configuration in leads V_4 and aVR, anterior and inferior sites by that in leads II, III and V_6 and septal and lateral sites were differentiated using leads I, aVL and V_1.

The algorithm was used to predict the site of earliest endocardial activation during 44 episodes of sustained ventricular tachycardia in a second group of 42 patients (Group II) in a blinded fashion. Anterior sites were correctly predicted in 83% of cases, inferior sites in 84%, septal sites in 90% and lateral sites in 82% of cases. Apical and basal sites were each correctly predicted in 70% of cases, whereas intermediate sites were less well predicted (29 to 55%) on the basis of QRS configuration. Precise localization of the site of origin of ventricular tachycardia (in all three planes) was achieved in 17 cases (39%), and in 16 cases (36%) the site of origin was immediately adjacent to the predicted site.

Prediction of the site of origin of ventricular tachycardia from the 12 lead ECG may serve as a useful, time-saving adjunct to, but not a substitute for, activation sequence mapping during ventricular tachycardia.

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Left ventricular activation mapping with an electrode catheter during ventricular tachycardia depicts the spread of electrical activity from an area of earliest endocardial breakthrough, which characteristically precedes the inscription of the QRS complex of the surface electrocardiogram (ECG) (1–3). This area, often designated the site of origin, has been used as the target for ablative procedures, including surgical resection, and application of various energy sources through catheter delivery systems in an attempt to eliminate the tachycardia (4–6). In addition to activation sequence mapping during ventricular tachycardia, endocardial pace-mapping has been suggested (7,8) as a method for corroborating the site of origin of ventricular tachycardia. By estimating the approximate location of the site of origin of the arrhythmia with techniques that do not require the patient to be in ventricular tachycardia, detailed mapping could be confined to a more precise location, effectively reducing the duration of the procedure and the period of potential hemodynamic instability.

Although the surface ECG offers much information on the electrical activation of the heart (9–11), only few data are available (12–15) on the value of the 12 lead ECG in localizing the origin of ventricular tachycardia. We, therefore, performed this study to further evaluate the utility of the 12 lead surface ECG in identifying the site of origin of ventricular tachycardia defined by endocardial activation mapping.

Methods

Study protocol. Initially, we analyzed the electrocardiographic (ECG) configurations resulting from left ventricular endocardial pace-mapping at multiple sites in a group of patients with prior myocardial infarction (Group I). It was hypothesized that electrical stimulation of the endocardium at or near the site of origin of ventricular tachycardia should

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affect a similar spread of activation to that seen during sustained ventricular tachycardia, resulting in similar surface ECG manifestations. The ECG features that allowed discrimination between distinct sites on a triaxial mapping grid (see later) were extracted and used to construct a flowchart that could then be used to perform a blinded analysis of the ECGs recorded during sustained ventricular tachycardia in a second distinct group of patients (Group II). The sites predicted by the flowchart were then compared with the sites identified as the earliest activation during left ventricular endocardial catheter mapping during ventricular tachycardia in Group II patients. A scoring system was applied to assess the predictive accuracy of this ECG technique. All patients gave informed consent for these studies.

Patients. *Group I (pace-mapping).* This group comprised 19 men and 3 women with prior myocardial infarction (anterior in 9, inferior in 10, anterior and inferior in 3); the mean age was 61 ± 8 years. Eight patients had angiographic evidence of left ventricular aneurysm. The mean left ventricular ejection fraction was 32 ± 12%. A 12 lead ECG was recorded for a total of 93 paced sites (4.2 sites/patient). Ten patients were taking antiarrhythmic drugs at the time of the pace-mapping study (including five patients receiving amiodarone).

Group II (sustained ventricular tachycardia). This group comprised 38 men and 4 women with a mean age of 61 ± 10 years. Infarct location was anterior in 26, inferior in 12 and anterior and inferior in 4 patients; 19 patients had a left ventricular aneurysm. The mean left ventricular ejection fraction was 28 ± 10%. Endocardial activation sequence mapping was performed during 44 different monomorphic ventricular tachycardias in this group (two patients had two morphologically distinct tachycardias that were each studied). Thirty patients were taking antiarrhythmic drugs at the time of the mapping procedure (including 12 patients receiving amiodarone).

Left ventricular pacemapping. A mapping grid was constructed by dividing the endocardial surface of the left ventricle into three planes defined by the fluoroscopic outline determined in two standard positions (Fig. 1). In the 30° right anterior oblique projection, the left ventricle was divided into three regions along the long axis from apex to base (numbered 1 to 3), and into three sections in the short axis identified as anterior (A), inferior (I) and middle (M). In the 60° left anterior oblique projection, the left ventricle was divided into septal (S), lateral (L) and central (C) sections. Each catheter position was studied first in the 30° right anterior oblique projection and immediately after in the 60° left anterior oblique projection; care was taken to avoid catheter movement during the change from one oblique projection to the other. By mutual agreement between at least two observers, each position of the catheter tip within the ventricular cavity was assigned a coordinate in each of the three axes, and each position was uniquely identified by its three coordinates. For instance, an apicoinferolateral location was assigned the coordinates 1, inferior (I) and lateral (L) (Fig. 2). This mapping scheme describes 24

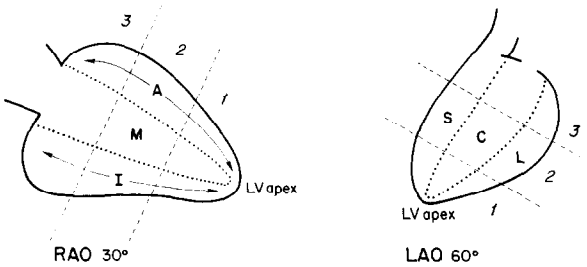
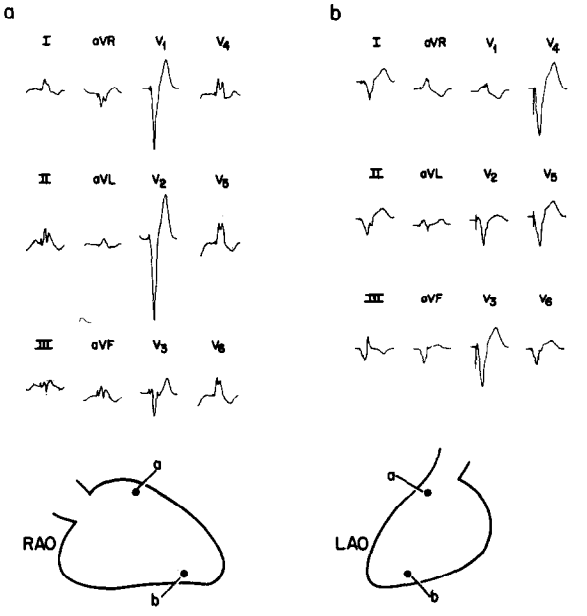


Figure 1. Left ventricular endocardial mapping grid. The grid is divided into three sections in each of three planes, determined from two fixed fluoroscopic planes. A = anterior; C = central; I = inferior; L = lateral; LAO = left anterior oblique projection; M = middle; RAO = right anterior oblique projection; S = septal; 1,2,3 = three regions along the long axis of the left ventricle (LV): apical, mid-ventricular and basal.

Figure 2. Assignment of QRS morphology during left ventricular pacemapping. Twelve lead electrocardiogram during left ventricular pacemapping from two endocardial sites marked a and b. These sites are depicted on the left ventricular outlines. The QRS morphology in each lead is described below as positive (+), negative (−) or equipotential (o). 3AS = site a (region 3, anteroseptal); 1IL = site b (region 1, inferolateral).



Designation of QRS morphology

	I	II	III	aVR	aVL	aVF	V_1	V_2	V_4	V_6	SITE
a	+	+	0	−	+	+	−	−	+	+	3AS
b	−	−	−	+	−	−	+	−	−	−	1IL

possible endocardial sites (sites where M and C coexist are, by definition, mid cavity sites and, therefore, do not specify endocardial catheter sites).

Pace-mapping was performed from multiple sites in the left ventricle at a rate of 150 beats/min (or less if not hemodynamically tolerated at this rate) at just above the diastolic pacing threshold to avoid stimulation of distant sites. A 7 or 8 French electrode catheter was inserted percutaneously through the femoral artery and passed under fluoroscopic guidance over a flexible guide wire to the left ventricular cavity. Two to 10 disparate sites per patient in the left ventricle and at least 2 right ventricular sites (apex and outflow tract) were paced while a 12 lead ECG was recorded. The number of sites studied in an individual was determined by the ease of catheter placement at multiple sites in the left ventricle, ability to achieve reliable capture at these sites and patient tolerance of the procedure.

Analysis of electrocardiogram. The ECG was analyzed lead by lead according to the direction of maximal voltage of the QRS complex. Each lead was categorized as positive, negative or equipotential (Fig. 2). This was a qualitative decision based on an estimate of the total area under the QRS deflection in each lead. For each of the three axes of the mapping grid, ECG configurations were separately analyzed to determine characteristic configurations that would allow discrimination of the separate sections within each axial plane. On the basis of these discriminators, a flowchart was constructed. This flowchart was then applied by two observers to predict, in blinded fashion, the site of origin of ventricular tachycardia from the ECGs of Group II patients recorded during ventricular tachycardia at the time of endocardial activation mapping in the electrophysiology laboratory. Only those induced tachycardias associated with documented presystolic endocardial activation were analyzed.

Mean ECG frontal and horizontal axes were calculated from the limb leads and precordial leads, respectively, by two observers who determined these data on two occasions, independently and without knowledge of the patients' names. To make quantitative comparisons easier, negative axis values were avoided and each axis was assigned a degree between 0 and 360, using the same reference frame for all ECGs (for example, +315° instead of −45°). Intraobserver and interobserver variability was determined, and the value for each of the ECG axes was derived from the average of four determinations (two determinations each by the two observers). Numerical axis measurements were not directly incorporated into the formulation of the algorithm.

Activation sequence mapping. For this study, a 6F quadripolar electrode catheter was inserted into the right ventricle for programmed ventricular stimulation, and a 7 or 8F bipolar catheter was inserted into the left ventricular cavity as already described. The details of activation sequence mapping in our laboratory have been previously reported (16). In most cases, the left ventricular catheter was positioned at multiple separate stable positions on the endocardium, after which ventricular tachycardia was induced from the right ventricular apex or outflow tract by programmed stimulation using one to three extrastimuli. In cases where ventricular tachycardia was hemodynamically tolerated, the left and right ventricular catheters were moved to multiple endocardial sites to record local electrograms during sustained ventricular tachycardia. Activation times were determined from the onset of the initial rapid deflection of the bipolar local endocardial electrogram, with the onset of the surface QRS complex serving as the 0 time reference. Each catheter position marking the site of earliest activation during ventricular tachycardia was assigned to a ventricular site from the triaxial mapping grid by mutual agreement between at least two observers. The spatial resolution was angiographically estimated to be 2 cm in both oblique projections; that is, two catheter positions assigned the same three coordinates of our mapping grid (Fig. 1) at two different times during mapping in the same patient did not differ by >2 cm along any of the three axes. Local electrograms were simultaneously recorded with three surface ECG leads on an Electronics for Medicine VR-16 recorder. A separate ECG recorder was also attached to the patient to allow a 12 lead tracing of all induced sustained ventricular tachycardias.

Scoring system. To quantify the accuracy of predicted sites against the actual sites of origin of ventricular tachycardia, a scoring system was devised in which a score of 0 to 2 was assigned for each coordinate: a score of 2 was assigned if the predicted site matched the actual site, 1 for an adjacent location and 0 for a remote position. For example, if the predicted site had coordinates 1, anterior (A) and septal (S), but the actual site determined from activation sequence data had coordinates 1, midventricular (M) and lateral (L), a score of 3 (from a possible 6 points) would be calculated as follows: a score of 2 (site 1 correct) + 1 (site M and A adjacent) + 0 (site S and L remote) = 3.

Statistical analysis. Data were expressed as mean values ± SD. Comparative analyses were performed with the use of Students *t* test or chi-square analysis wherever appropriate.

Results

Electrocardiographic Analysis of Left Ventricular Endocardial Pace-Mapping (Group I Patients)

Distinguishing characteristics of particular endocardial locations (Table 1). Examples of ECGs obtained during pacing from disparate left ventricular sites are shown in Figure 2. A total of 93 pacing sites were distributed evenly among anterior and inferior sites and among septal and lateral sites, but with a relative predominance of apical sites (46 sites from region 1, 32 from region 2 and 15 from region 3). Analysis of QRS configurations in each of the three axial planes revealed

Table 1. Electrocardiographic Configuration Observed During Left Ventricular Pace-Mapping*

Endocardial Site	I			II			III			aVR			aVL			aVF			V_1			V_2			V_4			V_6		
	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0	+	−	0
1	7	35	4	5	37	4	10	30	6	40	5	0	25	15	6	10	35	1	24	6	3	21	17	8	2	37	7	4	41	1
2	5	20	7	10	22	0	12	18	2	11	19	2	15	10	7	10	22	0	24	4	2	20	8	4	4	15	13	4	16	12
3	9	5	1	10	4	1	9	5	1	4	10	1	7	6	2	10	4	1	6	5	3	8	5	2	12	0	3	9	4	4
A	6	34	3	22	19	2	26	10	7	27	14	2	10	35	8	25	18	0	27	10	5	14	18	1	8	26	9	13	26	4
M	5	4	1	3	5	2	4	6	0	6	4	0	6	3	1	4	5	1	5	4	1	4	6	0	4	5	1	4	5	1
I	10	22	8	0	39	1	1	37	2	37	1	2	30	4	6	1	38	1	32	1	2	32	5	3	6	21	13	0	38	2
S	10	12	5	9	16	2	10	17	0	15	10	2	17	9	1	10	16	1	12	11	3	17	1	4	6	8	8	4	16	2
C	11	30	3	8	33	3	12	28	4	39	5	0	24	10	10	11	33	0	34	4	3	23	12	9	6	29	9	4	37	2
L	0	18	4	8	14	0	10	8	4	16	5	1	5	13	4	9	12	1	18	0	2	16	2	4	5	9	8	3	16	3

For each lead of the surface electrocardiogram (ECG), the number of ECGs with positive (+), negative (−) and equipotential (0) QRS complexes are shown for each site. Total number recorded is less for certain precordial ECG leads compared with limb leads, either because of missing leads or baseline artifact interfering with morphologic interpretation. A = anterior; C = central; I = inferior; L = lateral; M = middle; S = septal; 1, 2, 3 = three regions along the long axis of the left ventricle from apex to base.

a group of characteristics that appeared to distinguish particular endocardial locations: 1) In lead V_4, a negative QRS was never seen during pacing from basal or region 3 sites; these sites were also characterized by a higher incidence of negative QRS configuration in lead aVR, whereas the converse was true for apical or region 1 sites. 2) In lead II, a positive QRS configuration was never seen during pacing from inferior (I) sites; this was also the case in lead V_6. Anterior (A) sites were characterized by either positive or negative QRS configuration in the inferior leads (II, III and aVF), and midventricular (M) sites were characterized by an appearance intermediate to that seen at sites A and I. 3) In lead I, a positive QRS configuration was never seen during pacing from lateral (L) sites. In lead aVL, there was a predominance of negative QRS in lateral sites and a predominance of positive QRS configuration during pacing from septal sites. Lead V_1 provided further distinction between septal and lateral sites: a negative QRS configuration was characteristic of septal regions, occasionally seen at central regions (C in Fig. 1), but never seen during pacing from lateral sites.

These generalizations are summarized in Table 2. Figure 3 illustrates the algorithm used in predicting the site of origin of ventricular tachycardia, based on the data shown in Table 1.

Table 2. Electrocardiographic Characteristics Specific to Endocardial Regions

ECG Lead	QRS Configuration	Probable Site	Comment
I	Positive	S,C	C: aVL −
II	Positive	A,M	M: if III −
V_1	Negative	S,C	Usually S
V_4	Negative	1,2	2: if aVR −

Abbreviations as in Table 1.

Frontal and horizontal QRS axes during pace-mapping. The total (inter- and intraobserver) variability for axis determination was ±13° for the frontal axis and ±23° for the horizontal axis. The QRS frontal axis was useful in differentiating anterior from inferior sites and septal from lateral sites: for anterior sites, the mean QRS axis was 93 ± 13° for anteroseptal and 135 ± 42° for anterolateral sites; for inferior sites, the mean axis was 259 ± 4° for inferoseptal and 226 ± 24° for inferolateral sites ($p < 0.01$ comparing anterior and inferior sites and comparing septal and lateral sites). The QRS horizontal axis differentiated apical and basal sites: the mean QRS axis was 225 ± 46° for apical sites (region 1), 14 ± 47° for basal sites (region 3) and 170 ± 48° for region 2 ($p < 0.001$ comparing apical and basal sites).

Electrocardiographic Analysis During Left Ventricular Mapping of Ventricular Tachycardia (Group II Patients)

The mean cycle length of the 44 ventricular tachycardias during activation sequence mapping was 402 ± 81 ms (range 220 to 580). The earliest local endocardial activity preceded the onset of the surface QRS complex by 63 ± 31 ms during ventricular tachycardia. The distribution of ventricular sites at which earliest activity was recorded is shown in Table 3.

Localization of endocardial site of origin (Table 4). With the scoring system outlined under Methods, an overall score of 224 of a possible 264 (that is, 6 × 44 ventricular tachycardias) was attained (85% accuracy). Precise localization of the endocardial site of origin using the triaxial mapping grid (that is, a score of 6) was achieved in 17 cases, (39%) and in 16 (36%) other episodes of ventricular tachycardia, the site of origin was at the predicted site in two axes and at a site immediately adjacent in the remaining axis (that is, a score of 5). Table 4 summarizes the accuracy of the predictions

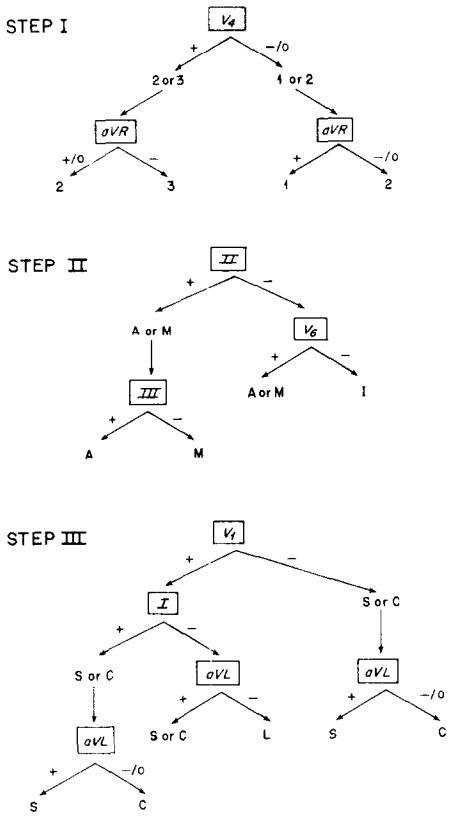


Figure 3. Flowchart used for predicting site of origin of ventricular tachycardia: a three step flowchart for predicting each of the three axis coordinates. To determine these sites, begin with the designated electrocardiographic lead and, depending on the QRS morphology in the lead, follow the **arrows** to the **bottom** of the diagram. The electrocardiographic leads are enclosed by **boxes**, whereas endocardial sites are indicated in **bold type**. Abbreviations as in Figure 1.

according to regions in each axial plane. The algorithm was most useful in discriminating anterior, inferior, septal and lateral sites (82 to 90% success). Central and midventricular sites (regions C and M) were poorly predicted although, in each case, an adjacent region was identified by the algorithm. Differentiation of sites in the long axis of the ventricle was of only moderate success (55 to 70%).

Influence of the site of myocardial infarction. In the long-axis plane (apical-basal), the site of origin of ventricular tachycardia was more often located in an apical region (site 1) in patients with anterior myocardial infarction (17 of 25), whereas in patients with inferior myocardial infarction, it was more often located in a midventricular or basal region (11 of 14). The distribution of site of origin of ventricular tachycardia was similar in anterior and inferior infarcts in each of the short-axis planes. The site of infarction did not significantly influence the precision of the algorithm for predicting the site of origin: anterior infarcts had a mean score of 5.1 ± 0.9 compared with 4.9 ± 0.9 for inferior infarcts (p = NS). The exact site of origin (all three coordinates correct) was identified correctly in 10 (40%) of 25 patients with anterior infarction, 4 (29%) of 14 patients with inferior infarction and 3 (60%) of 5 patients with both an anterior and an inferior infarction.

Table 3. Location of Site of Earliest Activation During Ventricular Tachycardia

Site	No.
Left ventricular long axis	
Apical (region 1)	23
Mid-ventricular (region 2)	11
Basal (region 3)	10
Left ventricular short axis	
Anterior	12
Mid	7
Inferior	25
Septal	21
Central	12
Lateral	11

Discussion

Usefulness of surface QRS configuration to localize site of origin of ventricular tachycardia. This study demonstrates the utility of the surface ECG in the approximate localization of the site of earliest endocardial activation during ventricular tachycardia in patients with prior myocardial infarction. An algorithm derived from analysis of the QRS complex configuration during ventricular endocardial pace-mapping served as the model for predicting the site of origin of ventricular tachycardia in a group of patients with previous myocardial infarction. This algorithm is based on prior observations (7,15) that pacing at the sites of earliest activation usually mimics the ECG configuration of ventricular tachycardia, presumably because the tachycardia originates in the subendocardium and "breaks through" to the surface at the endocardial level. The results show that our algorithm applied to the 12 lead ECG may be used as a practical guide to abbreviate the duration of ventricular mapping during ventricular tachycardia. The algorithm is particularly useful in classifying the site of ventricular tachycardia as anterior or inferior and septal or lateral. However, its precision is not high enough for this technique to be a substitute for activation sequence mapping, which is required for the exact localization of the site of origin of ventricular tachycardia.

Previous studies. Using multiple plane fluoroscopy, Waxman and Josephson (13) previously described an ECG analysis of left ventricular endocardial pacing based on a 12 site map. In patients without wall motion abnormalities, pacing produced predictable changes in QRS configuration and frontal axis. Holt et al. (8) constructed an ECG "atlas" by performing multiple site epicardial and limited endocardial pacing and noted that resultant ECGs were consistent with

Table 4. Accuracy of the QRS Flowchart in Predicting Site of Origin of Ventricular Tachycardia

	Site								
	1	2	3	A	M	I	S	C	L
Correct	16	6	7	10	2	21	19	6	9
Adjacent	6	5	3	2	5	2	2	6	1
Remote	1	0	0	0	0	2	0	0	1
% Correct	70	55	70	83	29	84	90	50	82

Abbreviations as in Table 1.

their expectations based on deductive reasoning; however, their patients were essentially free of significant left ventricular disease. The mapping grid proposed by Josephson et al. (7) and Waxman and Josephson (13), although very suitable for intraoperative mapping, is less easily adaptable in the catheterization laboratory to locate catheter positions using fluoroscopy. Our findings during pace-mapping in patients with prior myocardial infarction and ventricular tachycardia essentially confirm those of Waxman and Josephson (13), despite the differences in the patient groups of the two studies.

Josephson et al. (7) also showed that in patients with sustained monomorphic ventricular tachycardia, although pacing from the site of earliest activation reproduced the ECG recorded during ventricular tachycardia, pacing at sites in close proximity might result in quite different patterns. The examples they included (7) demonstrated that especially in tachycardias with a septal site of origin, relatively small alterations in catheter site along apical-basal or superior-inferior direction might result in gross mismatch between ECGs recorded during ventricular tachycardia and pace-mapping. To avoid this problem, our protocol involved careful assessment of catheter position by assigning to each position one of three possible coordinates along the apical-basal as well as the anterior-inferior direction. Even with care, however, minor alterations in catheter placement might result in large changes in ECG configuration, and this factor very likely contributed to lowering the predictive accuracy of our algorithm.

Electrocardiographic correlation with site of endocardial stimulation. On the basis of our pace-mapping data, there were particular ECG findings that characterized specific left ventricular sites: pacing from basal sites never produced a negative QRS configuration in lead V_4, so that this finding was specific for more apical locations. A differentiation of anterior and inferior sites was also possible based on the appearances of ECG leads II and V_6. However, caution should be exercised in drawing conclusions from the polarity and the configuration of precordial leads V_4 and V_6. As with other precordial leads, modest changes in the position of leads V_4 or V_6 may substantially alter the polarity of the QRS complex. Meticulous care should be taken in positioning the precordial leads. Even then, the coordinates for the site of origin of ventricular tachycardia should not be based on a single precordial lead; rather, corroborative information from other ECG leads should be sought.

Leads I and V_1 were useful in distinguishing septal and central sites. This latter finding was previously noted by Josephson et al. (17), who suggested that there was preferential right ventricular activation through transseptal conduction in the presence of an extensively diseased left ventricle. These investigators (17) also noted changes similar to those in our study in the ECG configuration and axis when moving the site of stimulation in the anterior-inferior and septal-lateral planes; however, they found little change resulting from pacing in the apical-basal plane. This difference between the two studies may be explained by patterns related to the horizontal axis, not systematically assessed by Josephson et al. (17), in which marked changes were noted in our patients by alterations in pacing site along apical to basal direction. These alterations were reflected by changes in QRS morphology in leads V_4 to V_6.

Prediction of ventricular tachycardia site of origin. The similarity between the pace-mapped ECG at the site of origin of ventricular tachycardia and the ECG recorded during ventricular tachycardia has previously been described (7,8). The algorithm derived from our pace-mapping data predicted the anterior, inferior, septal or lateral locations with a relatively high degree of accuracy (80 to 90%), despite the varied locations of myocardial infarction that could theoretically alter the activation sequence of these arrhythmias and give rise to inconsistent surface ECG configurations. In a recent study by Miller et al. (14), a different algorithm was derived from the 12 lead ECG to predict the site of origin of ventricular tachycardia. Those investigators (14) predicted the site of origin of ventricular tachycardia in 93% of their patients. However, in their study, the entire left ventricular endocardial surface was divided into 10 relatively large sections, in comparison with our coordinate grid with 24 distinct locations. This difference in density of the mapping grid may, in part, explain the higher predictive accuracy of their algorithm. Their study also found a relation between the predictive accuracy of their algorithm and infarct location, whereas in our study, a similar association was not

detected. This difference between the studies may be partly a result of the greater number of patients studied by Miller et al. (14) and the particularly small number of patients with inferior infarction in Group II in our study.

Limitations. This study was limited by the resolution of the catheter mapping technique. Limitations of this technique have been discussed previously (2,13,15). Using the site of earliest endocardial activation during ventricular tachycardia as our standard, we were able to accurately predict these sites with a resolution of approximately 2 cm along each axis. Although the criterion is universally accepted, reliance on the earliest evidence of activation recorded by a catheter as the sole criterion to determine the site of tachycardia origin introduces another limitation. Furthermore, during ablation procedures, it is of utmost importance to save as much functional tissue as possible, while eliminating the areas responsible for the genesis of the arrhythmia. The resolution of the ECG algorithm is clearly not high enough to accomplish this. As the algorithm is intended as an adjunctive technique to catheter or intraoperative mapping, rather than a substitute, it remains a sufficiently useful tool, even without finer resolution, to warrant its clinical application.

In obtaining our pace-mapping data, we included a relative paucity of basal sites (site 3) and mid-ventricular septal sites, partly because pace-mapping from these areas was difficult owing to the presence of the mitral valve apparatus or the instability of septal catheter positions beneath the left ventricular outflow tract. In contrast, apical sites were usually more accessible with the catheter. However, the differentiation of anterior from inferior and septal from lateral regions was often difficult at region 1 because of the clustering of these sites toward the left ventricular apex, and this difficulty constitutes another technical limitation. Ideally, a study of this kind should include patients in whom all sites in the mapping grid are paced and are also recorded from during ventricular tachycardia. Indeed, the demonstration of the value of lead V_6 in differentiating inferior from anterior sites may not have held true if more inferobasal sites had been interrogated by the pacemapping technique.

This study concentrated on the ECG characteristics of left ventricular pacing and arrhythmias. ECG configurations similar to those seen from pacing left ventricular septal sites can be stimulated by right ventricular pacing (7,15). The absence of right ventricular sites in the coordinate system used is a limitation of our algorithm. Although the vast majority of ventricular tachycardias are of left ventricular origin (2,7,16,17) in patients with known coronary artery disease and prior myocardial infarction, ultimately the exact localization of the site of origin of septal ventricular tachycardia requires simultaneous activation sequence mapping on both sides of the interventricular septum.

Finally, our flowchart is limited by the shortcomings inherent in the pacemapping technique. Even with precise localization of the site of earliest endocardial activation, pacing from this site only approximates the global spread of activation during ventricular tachycardia unless one paces from a site known to be within the reentrant circuit by other electrophysiologic criteria, which are not always available.

Implications. The 12 lead ECG obtained during ventricular tachycardia provides useful information to help localize the ventricular site of origin of the arrhythmia in the majority of the patients. This finding is important in directing the electrophysiologist to the approximate region of interest, thereby saving time during detailed arrhythmia mapping, especially when ventricular tachycardia is poorly tolerated hemodynamically.

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