**Diagnosis Methods**

**Arrhythmia**

Relationship between the 12-lead electrocardiogram during ventricular tachycardia and endocardial site of origin in patients with coronary artery disease

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**Abstract**

Previous studies in patients with sustained ventricular tachycardia (VT) have demonstrated the efficacy of surgical and catheter-mediated ablative procedures based on activation mapping during VT. Since extensive preoperative or intraoperative mapping may be impractical due to time constraints or patient intolerance, we sought to define characteristics of the 12-lead electrocardiogram (ECG) during VT that could suggest a particular endocardial region of origin and thus facilitate mapping studies. Endocardial mapping was performed during 182 VTs in 108 patients with prior myocardial infarction of either the anterior or inferior wall. Endocardial sites of origin (sites from which ≥ 40 msec of presystolic electrical activity was consistently recorded) were identified with use of catheter (154 VTs) or intraoperative (85 VTs) activation mapping (both methods used in 57 VTs). Twelve-lead ECGs obtained during these VTs were characterized by four features: location of infarction, bundle branch block type configuration, quadrant of QRS axis, and precordial R wave progression pattern. A specific combination of these four features was associated with a particular endocardial region containing the mapped site of origin in 87 VTs (48% of total). An association (≥ 70% positive predictive accuracy) was more likely to be found in the presence of left, as opposed to right, bundle branch block type patterns (53/73 [73%] vs 34/109 [31%]; p < .001) and in the presence of VT related to inferior, as opposed to anterior, infarction (40/54 [74%] vs 47/128 [37%]; p < .001). An algorithm developed with the above criteria was then applied prospectively to 110 VTs (all mapped) in an additional 63 patients. Each author, blinded to mapping data, used the algorithm to correctly predict endocardial region of origin for a mean of 60 of 65 (93%) VTs to which the algorithm could be applied. These data indicate that the 12-lead ECG during VT can be used to suggest an endocardial region of origin in approximately one-half of VTs in patients with a single site of myocardial infarction. Although this information should not be a substitute for careful mapping when such studies are possible, the findings of this study may be used to facilitate placement of recording electrodes in areas likely to contain sites of origin and thus expedite mapping.

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In the last decade, a variety of surgical and catheter-mediated means of ablating ventricular tissue have been developed to treat sustained ventricular tachycardia (VT) associated with prior myocardial infarction.1-5 Precise knowledge of the location of areas from which tachycardias arise is imperative to destroy the arrhythmogenic tissue without causing significant damage to more normally functioning ventricular myocardium. Currently, the most reliable method for acquiring information concerning the location(s) of arrhythmogenic areas is activation mapping of the heart during VT6; prior studies have shown that eradication of endocardial tissue from which presystolic electrical activity during VT is recorded can result in cure of VT.7-7 Detailed activation mapping during VT is not always possible due to hemodynamic intolerance of VT or changes in QRS morphology of VT.8 Alternative methods for obtaining information regarding "sites of origin" of VT have been applied, including pacemapping and noninvasive means of detecting areas of earliest wall motion (gated nuclear scans, echocardiography).10-13 A previous study from this...
laboratory correlated features of the 12-lead ECG during VT with sites of endocardial origin in a small number of patients. The current study expands on this experience with the use of the 12-lead electrocardiogram (ECG) during VT as a guide to the location of VT sites of origin. The study consisted of (1) correlation of features of the 12-lead ECG of VT with activation mapping data in one group of patients, (2) formulation of an algorithm comprising these correlative features, and (3) prospective application of this algorithm in a second group of patients. Our results show that the 12-lead ECG of VT can be used in a large number of cases to suggest the location of arrhythmogenic areas and thus aid in mapping and directing ablative procedures.

Methods

Study population. One hundred eighty-two 12-lead ECGs of sustained VT were analyzed in the first part of this study. These ECGs were obtained from 108 individuals with prior (2 weeks to 24 years) anterior wall (73) or inferior wall (35) acute myocardial infarction, diagnosed by characteristic isoenzyme and electrocardiographic changes and/or cineangiographic demonstration of an occluded coronary artery and associated wall motion abnormality. Only patients with single areas of infarction were considered to avoid possible alterations in electrocardiographic patterns during VT which may result from multiple areas of infarction. There were 94 men and 14 women with ages ranging from 37 to 78 years in this population. All patients had experienced at least one episode of spontaneous sustained (lasting >30 sec) VT or cardiac arrest, and all underwent electrophysiologic studies to guide treatment of their arrhythmias.

Electrophysiologic studies/mapping studies. Catheter endocardial mapping was performed during 154 VTs in the study group by use of techniques previously described. Briefly, one or more No. 6F quadripolar catheters were inserted percutaneously into femoral or brachial veins and advanced to the right ventricular apex and/or outflow tract for stimulation and a stable timing reference. A single No. 6F quadripolar catheter was advanced to the left ventricle via femoral arterial puncture. All catheters had 0.5 cm electrode spacing. VT was initiated by programmed electrical stimulation and left ventricular endocardial mapping was performed with confirmation of catheter location by multiplane fluoroscopy. VT was hemodynamically tolerated in all cases included in this study. Twelve-lead ECGs were obtained of each morphologically distinct type of VT (see definitions) during these studies with single or multichannel standard electrocardiographic machines; most patients demonstrated more than one morphologically distinct VT (up to six), and as many were mapped as possible. Mapping of each distinct VT morphology required 5 to 15 min, during which a minimum of six, and a maximum of 12 (mean nine) standard sites were sampled as previously described. Bipolar left ventricular endocardial electrograms were recorded via the distal and third electrodes (1 cm interelectrode distance), amplified and filtered from 30 to 400 Hz, stored on magnetic tape (Honeywell 5600 C), and written on paper at speeds of 200 to 250 mm/sec (Siemens Mingograf). In all VTs in this series, at least one endocardial site had electrical activity preceding the surface QRS onset by at least 40 msec, and the site from which the earliest electrical activity in the latter half of diastole was recorded was determined to be the site of origin of VT.

Intraoperative mapping was performed at the time of surgery for 85 VTs by techniques previously reported. Briefly, the heart was exposed through a median sternotomy, normothermic cardiopulmonary bypass was instituted, and VT was initiated with the use of pacing techniques as above. The infarct was incised and left ventricular endocardial mapping was performed with a roving bipolar or quadripolar probe electrode (2 mm interelectrode distance) by sampling sites on a clock face format. A minimum of 15 sites were sampled during each VT (mean 43); adjacent sites were separated by 1 cm or less. During mapping, surface electrocardiographic leads I, II, III, and V6R were recorded, as well as bipolar reference electrograms from the right and left ventricles; probe electrograms were amplified and filtered from 50 to 400 Hz, recorded on magnetic tape (Honeywell 5600-E), and written on paper at 200 mm/sec (Siemens Mingograf). The site of origin was determined in the same manner as for catheter mapping. Since 12-lead ECGs were not recorded during the surgical procedure, VTs observed in the operating room were considered the same as one from which a preoperative 12-lead ECG was obtained only if leads I, II, and III were identical and V6R had a similar configuration to V6 on the 12-lead ECG and the patient had never demonstrated VTs of other morphologies with which the intraoperative VT could be confused. Both catheter and intraoperative maps were obtained of 57 VTs, and the respective sites of origin were estimated to be within 1 to 2 cm in all cases. All VT sites of origin in this series were within the left ventricular endocardium. Although right ventricular reference electrograms (either preoperatively or intraoperatively) occasionally occurred before the surface QRS onset, in each case left ventricular endocardial sites with still earlier timing were always located.

Definitions. (1) Right bundle branch block (RBBB) type pattern during VT: dominant (and usually terminal) R wave in lead V1 (QR, monophasic R, Rs, RSR'). (2) Left bundle branch block (LBBB) type pattern: dominant (and usually terminal) S wave in lead V1 (QS, Rs). (3) Morphologically distinct VT: two VTs that on the 12-lead ECG differed by having either (A) contralateral bundle branch block patterns or (B) frontal plane QRS axes more than 90 degrees divergent. (4) VT site of origin: the endocardial site from which the earliest activity in the latter one-half of diastole was recorded (always ≥40 msec before the onset of the QRS). (5) Precedial R wave progression (RWP) patterns: Eight unique RWP patterns were observed in this study. Each VT could be characterized as displaying one of the following patterns based on the height of the R wave (figure 1): increasing — gradual increase in R amplitude from V1 to V6; none or late — either QS in V1-V6, or with Rs in V1 and/or QR in V6; regression/growth (not QS) — Rs, R, or qR in V6, followed by Rs in V5-V4 and Rs or Rs in V6; regression/growth (QS) — Rs, R, or qR in V6, followed by Rs in V5-V4 and Rs or Rs in V6; dominant R— qR, Rs, or R in V1-V6; abrupt loss — abrupt change from R>S in at least V1 and V2 to QS through V6; late reverse — gradual decrease in R amplitude with a translation from R>S to R<S in V5 or V6; early reverse — gradual decrease in R amplitude with a transition from R>S to R<S in V3 or V4. (6) Characteristic morphology of VT: the unique combination of infarct site, bundle branch block configuration, QRS axis, and RWP pattern. Theoretically a total of 126 possible combinations of these four characteristics exists. (7) Specific morphology of VT — a characteristic morphology that was associated with a particular region of the endocardial mapping schema in figure 2, with greater than 70% positive predictive accuracy.

Data analysis. For purposes of data analysis, the left ventricular endocardium was divided into 11 regions. The area within each region was from 10 to 20 cm² depending on the particular region and individual heart size, as shown in figure
2. Each 12-lead ECG of VT was characterized according to location of associated infarction (anterior or inferior), bundle branch block type configuration (right or left, see definitions), quadrant of frontal plane axis (all four combinations of right, left, inferior, and superior), and RWP pattern (see definitions and figure 1). The mapped site of origin of each VT was then plotted on a schema of the left ventricular endocardium according to these 4 characteristics (figure 3). Among the group of VTs having the same combination of these 4 features ("characteristic morphology"; see definitions), a determination was made of which characteristic morphologies were associated with a particular mapping region (A through J on figure 2) with at least an arbitrary 70% positive predictive accuracy (that is, 70% of ECGs having a characteristic morphology had to originate from a specific region). A minimum of five examples of a given characteristic morphology was arbitrarily required before such an association was made. Characteristic morphologies fulfilling these criteria were said to be "specific" for a region of endocardial origin. This criterion was waived if a given combination of infarct location, bundle branch block configuration, and QRS axis in VT correlated with a specific endocardial region regardless of RWP.

Prospective analysis. The second part of the study consisted of a prospective application of the results of the retrospective analysis in an additional 110 VTs from 63 patients (36 with anterior and 27 with inferior infarcts). All 110 VTs were mapped (93 by catheter mapping, 40 intraoperatively, 23 with both methods). Each author was then given a copy of the 12-lead ECG of VT coded only by number (1 to 110) and infarct site (anterior or inferior), and an algorithm for determining endocardial regions of origin based on the retrospective analysis (figure 4). Each reader was required to assign a region of origin for each VT whether or not the algorithm applied to each VT, and the results were analyzed according to (1) percent of VT correctly regionalized and (2) interobserver agreement.

Results

Retrospective analysis. The mapped site of origin of each of the 182 VTs was plotted on a schema of the left ventricular endocardium based on each unique combination of infarct site, bundle branch block type configuration, and QRS axis by quadrant (figure 3). The requirement of at least five examples of a characteristic morphology occurring within each cell of figure 3 in order to suggest an association between characteristic morphology and region of origin excluded 33 VTs (18%). This left 149 VTs (82%) that could be assessed for an association between characteristic morphology and region. Based on this analysis, such an association existed in 87 VTs (48% of the total), and these constituted specific VT morphologies (see definitions). Specific morphologies were more common in cases of inferior infarction than in the presence of anterior infarction (40/54 [74%], vs 47/128 [37%]; p < .001) and in the presence of VTs with LBBB vs RBBB configurations (53/73 [73%], vs 34/109 [31%]; p < .001). Specific morphologies are discussed below.

Inferior infarction

LBBB-VT. Of the 23 VTs with a LBBB morphology configuration, one specific morphology was evident: that of a left superior axis and "increasing" RWP. Sixteen VTs had this morphology, 14 of which (88%) had mapped origins in region F of figure 2, the inferobasal septum. Among the remaining seven VTs in this group, fewer than five examples of a given characteristic morphology occurred.

RBBB-VT. Specific morphologies in this group of VTs included those with either right superior or left superior axes and either early or late reverse RWP and they tended to have mapped origins in the inferobasal...
free wall (areas G and H; 14 VTs or 88%). VT with a left superior axis appeared to arise from the medial aspect of this region (region G). VT having a right inferior axis had late reverse RWP and this specific morphology arose from the inferolateral free wall (region H) in six of eight cases (75%). Too few characteristic morphologies were present among the remaining VTs to allow any conclusions as to specific regions of origin.

**Anterior infarction**

LBBB-VT. Specific morphologies accounted for 37 of the 50 VTs in this group (74%). VT having a left superior axis and no or late RWP had mapped origins in the inferoapical septum (region A) in 19 of 21 (90%) of cases with these features. Thirteen of 16 VTs (81%) with a left inferior or right inferior axis, regardless of RWP, were mapped to the anteroapical septum (region B). Thirteen additional VTs could not be associated with a particular region; five had the same characteristic morphology (regression-growth [QS]), but the mapped origins of these VTs were scattered across the septum. Inadequate numbers of characteristic morphologies were noted among the eight remaining VTs to allow analysis of specific areas of origin.

RBBB-VT. This group of VTs comprised the largest sample (78 VTs) but the lowest proportion of specific morphologies. Only 10 VTs (13%), with two RWP patterns (abrupt loss of R waves and dominant R waves), among right inferior axis VTs met the criterion of 70% positive predictive accuracy; these VTs had mapped origins in the anteroapical septum (region B). All other characteristic morphologies displayed wide variability in the location of mapping origins.

On the basis of these results, an algorithm was developed to enable prediction of regions of VT origin with use of the 12-lead ECG in VT (figure 4).

**Prospective analysis.** The 110 VTs analyzed in this portion of the study included 64 in patients with anterior infarction and 46 in patients with inferior infarction. The algorithm could be applied to 65 (59%) of these VTs, among which the mean correctly regionalized by the four readers was 60 (93% correct), with a range of 58 to 63. This included a mean of 33 of 36 (91%) of VTs in patients with inferior infarctions, and 27 of 29 (94%) of VTs in patients with anterior infarctions.

**Inferior infarction**

LBBB-VT. Fourteen of the 19 VTs in this category had the specific morphology of left superior axis/increasing RWP; all were regionalized by each reader to the inferoapical septum (region F). This area contained the actual site of origin of 13 of 14 (93%) of these VTs.

RBBB-VT. Twenty-two of the 27 VTs in this group had specific morphologies. Fourteen had a right superior or left superior axis morphology with early or late reverse RWP and all had mapped origins in the inferoapical free wall (regions G, H). These were correctly regionalized by the readers to this region in a mean of 12.5 (89%) VTs (range 11 to 13). Of the eight VTs with right inferior axis and late reverse RWP, a mean of 7.25 (91%, range 6 to 8) were correctly regionalized by the readers to the inferoapical free wall (region H), where all mapped origins occurred.

**Anterior infarction**

LBBB-VT. The algorithm could be applied to 23 of the 29 VTs in this category; 15 had the specific morphology of left superior axis/no or late RWP; all 15 were mapped to the inferoapical septum (region A) and all were correctly identified as such by the four readers. Eight other VTs had either right inferior or left inferior axis morphologies, and according to the algorithm should have had mapped origins in the anteroapical septum (region B); all eight were mapped to this area,
and the readers were correct in applying the algorithm in a mean of seven (88%) of these cases.

RBBB-VT. Of the 35 VTs in this group, the algorithm could be applied in only six (right inferior axis, either abrupt precordial R wave loss or dominant R pattern). All six had mapped origins in the anteropapical septum (region B), as predicted, and the readers correctly identified this area in a mean 5.25 (88%) cases.

Among the 65 VTs in the prospective series to which the algorithm could be applied, all four readers agreed on the correct mapping region in 56 (86%), three of four agreed in four (6%), two of four agreed in three (5%), and in no case was only one correct. The algorithm failed entirely (all four readers incorrect) in two (3%) VTs. In the 45 VTs to which the algorithm could not be applied, the readers correctly identified the mapped origin in a mean of 32 VTs (71%; range 31 to 34). This differs significantly from the 93% correctly identified with the algorithm (p < .01).

FIGURE 3. Mapped sites of origin for the 182 VTs in the retrospective analysis. LBBB VTs are shown on the left and RBBB VTs on the right; among each bundle branch block type, anterior infarcts are shown on the left and inferior on the right. QRS axis is segregated by horizontal rows, as shown in the far left column. The left ventricular endocardium is displayed as in figure 2, and each individual characteristic morphology is plotted according to the key at bottom.
Discussion

The use of ablative forms of therapy for sustained VT, such as various forms of transcatheter energy delivery\(^4,5\) and surgery,\(^1\) has increased in recent years. The success of these interventions depends on destroying the arrhythmogenic tissue while minimizing damage to more normal areas of myocardium, which in turn is critically dependent on correctly identifying the location of the arrhythmogenic tissues.

Methods of localizing arrhythmogenic tissues. The most frequently used method of localizing arrhythmogenic tissue is endocardial activation mapping (catheter or intraoperative) during VT, in which the operator samples local electrical activity from several areas, seeking electrograms that precede the onset of the surface QRS complex.\(^6\) Although this method is reliable, there are significant disadvantages, among which are: the time required to randomly sample a number of endocardial sites during one of potentially several morphologically distinct VTs, the inability to initiate (and thus map) all morphologic variants of VT in a given patient, and the hemodynamic intolerance of VT. Thus, other methods have been sought that would yield the same information but lack these drawbacks.

Endocardial mapping during sinus rhythm has been advocated as a potential means of circumventing some of the problems associated with activation mapping during VT; studies have shown, however, that this method lacks both specificity and sensitivity for identifying sites of origin of VT.\(^15\) Endocardial catheter pacemapping, in which pacing is performed at multiple endocardial sites with the aim of producing a 12-lead ECG duplicating that of the spontaneous arrhythmia, is useful to corroborate the findings of activation mapping.\(^9\) This method has the disadvantage of yielding a significant number of false-positive and false-negative results. Also, the random pacing at several sites in the left ventricle in the hope of chancing upon a site that produces the correct 12-lead electrocardiographic morphology is a time-consuming and inefficient process.

Noninvasive methods have also been proposed to help localize arrhythmogenic tissue, including Fourier-analyzed phase images of a gated blood pool scan,\(^10\)
and two-dimensional echocardiography.\textsuperscript{13} Both methods rely on determination of areas of earliest ventricular wall motion, presumably occurring at the periphery of an infarcted area near arrhythmogenic tissue. These studies have yielded variable results thus far; problems include poor spatial resolution of data, time required for acquisition of data, and imprecise correlation with results of endocardial activation mapping data.

**Current study.** The current study suggests an additional noninvasive means of localizing arrhythmogenic areas based on the standard 12-lead ECG in VT. Analysis of 12-lead electrocardiographic patterns to predict the site of arrhythmogenic tissue is not new; several algorithms have been proposed for localizing the site of atrioventricular bypass tracts in patients with the Wolff-Parkinson-White syndrome based on the delta wave vector in the first 40 msec of a maximally preexcited QRS complex.\textsuperscript{17-19} A previous study from this laboratory reported correlations between certain features of the 12-lead ECG of VT and endocardial site of origin in 41 VTs, six of which were not due to prior infarction.\textsuperscript{14} The current study extends the same principles of use of the information from the 12-lead ECG to localize arrhythmogenic tissues in a large series of infarct-associated VT. The retrospective analysis showed that the electrocardiographic patterns in almost half (48\%) of all VTs in this series were associated with at least a 70\% positive predictive accuracy for a particular endocardial region of 10 cm\(^2\) or less. An additional 110 unselected VTs were then prospectively evaluated with use of an algorithm based on the retrospective findings, and four independent readers were able to correctly regionalize mapped VT origins in 93\% of the 65 VTs (59\% of the total number) to which the algorithm could be applied. The localization thus provided is not precise but is sufficiently specific for most ablative surgical procedures currently in use, and can serve as a guide for catheter placement when mapping VT before catheter-mediated ablation procedures. Even in cases in which VT is poorly tolerated, a catheter could be positioned in the region of the left ventricular endocardium suggested by the morphologic characteristics of the 12-lead ECG during VT. VT could then be initiated and recordings made for several seconds to confirm whether presystolic electrical activity was present at the site, after which VT could be terminated before hemodynamic embarrassment ensued. Thus, the application of the results of this study may conserve time during endocardial mapping studies as well as allow greater patient comfort.

In this study, VTs with a LBBB configuration were significantly more likely than RBBB VTs to be associated with a specific endocardial region of origin. This may have been due to the fact that practically all LBBB VTs originate in the left ventricular septal endocardium (figure 3), and thus there is a small potential area from which these VTs could arise compared with RBBB VTs (which may have either septal or free wall origins). Also, VTs associated with inferior infarction were more likely to be related to a specific endocardial region than those associated with anterior infarction. This again may be due to the presence of a smaller area of damage with inferior infarction and thus a smaller potential area from which VT may originate in or at the border of the infarction. Eighty-seven percent of inferior infarction–related VTs in this study originated from regions F, G, and H (figure 2). It may also be that the smaller area of damage and resultant anatomic distortion with inferior, as opposed to anterior, infarction leads to less disturbance in the pattern of ventricular activation once impulses “break out” into ventricular muscle to generate QRS complexes.

The results of the prospective analysis suggest that the algorithm derived from the retrospective study can be used successfully to regionalize VT origins, and is superior to a “best guess” concerning origins of VTs for which the algorithm cannot be applied. Since 71\% of these latter VTs were still correctly regionalized by the four readers, some undefinable “experiential” interpretive skills may also have been operative. There was excellent overall agreement between independent readers in the categorization of electrocardiographic patterns as well as the use of the algorithm. The greater relative number of VTs having specific morphologies in the applied, as opposed to retrospective, analysis was likely due to a slightly greater proportion of VTs associated with inferior infarction (42\% vs 30\%), as well as LBBB VTs (44\% vs 40\%), since both of these categorizations had higher prevalences of specific morphologies.

**Limitations.** There are several potential limitations to the current study. First, the algorithm derived was neither perfect nor universally applicable to all VTs in this series; future refinements of the analysis may make such applications more useful. Second, the population of VTs studied was somewhat select: only those associated with a single site of infarction (anterior versus inferior) were included. Additional areas of infarction may alter QRS expression from any given arrhythmogenic area, and these criteria may not apply in such a situation; the same might be said of VTs associated with nonischemic cardiomyopathy. Third, we arbitrarily determined that a minimum of five examples of any characteristic morphology be present before an analysis.
of an association with regions of VT origin could be made. This was to reduce the likelihood of chance associations, but probably resulted in exclusion of some VTs that appeared otherwise to have rather specific morphologies. Among these are VTs with LBBB/right superior axis and no or late RWP morphology associated with anterior infarction (all three examples in the retrospective analysis and the one example in the applied analysis originated in the midapical septum), as well as those with LBBB/left inferior axis and increasing RWP morphology associated with inferior infarctions (one of two examples in the retrospective and all four in the prospective analysis originated in the inferobasal septum). Finally, we cannot be absolutely certain that the 28 VTs that were mapped only intraoperatively were the same as those of which 12-lead ECGs were recorded earlier; the close correlation between results of catheter and intraoperative mapping suggests that the VTs were likely the same as those of which 12-lead ECGs had been obtained.

In conclusion, the results of this study indicate that the 12-lead ECG during VT contains adequate information to specify a region of the left ventricular endocardium that is likely to contain the VT site of origin in approximately half of all VTs in patients with a single prior infarction. Further work is necessary to improve on these results, but the present study has produced a simple algorithm that may be applied in appropriate circumstances to facilitate electrode placement at the time of preoperative or intraoperative mapping or catheter or surgical ablation attempts. Prediction of the VT site or origin with this algorithm should not serve as a substitute for endocardial mapping except in cases in which this procedure is technically not feasible or sustained VT cannot be readily initiated. As the use of localized ablative procedures continues to increase, methods that can reliably identify the responsible arrhythmogenic areas, such as prediction of site of origin from the 12-lead ECG of VT, will also find increasing application.

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