Body Surface Mapping of Ectopic Left and Right Ventricular Activation

QRS Spectrum in Patients Without Structural Heart Disease

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The value of simultaneous 62-lead electrocardiographic recordings in localizing the site of origin of ectopic ventricular activation in a structurally normal heart was assessed by examining body surface QRS integral maps in 12 patients during left and right ventricular (LV and RV) pacing at 182 distinct endocardial sites. A data base of 38 characteristic mean integral maps was composed after visually selecting subgroups with nearly identical total QRS integral morphology and numerically evaluating intrasubgroup pattern uniformity and intersubgroup pattern variability. Corresponding endocardial pacing site locations were computed by a biplane cineradiographic method and outlined as segments on a standardized LV and RV polar projection. LV pacing resulted in 25 markedly different mean total QRS integral patterns, showing higher electrocardiographic sensitivity for anteroseptal (18 patterns) compared with posterolateral regions (seven patterns). RV pacing demonstrated 13 mean total QRS integral patterns, exhibiting less intersubgroup variation and comparatively low electrocardiographic sensitivity for the basal anterior and outflow regions. Comparison of LV with RV pacing revealed that QRS configurations produced at LV apical and LV midseptal sites closely resembled QRS configurations generated at RV apical, RV septal, and RV anterior sites, respectively. Total QRS time integral amplitudes showed considerable intrasubgroup variation but permitted global differentiation of spatially similar QRS patterns obtained during pacing at LV and RV sites. This study demonstrates that the QRS pattern of the total body surface electrocardiogram allows discrimination among 38 different LV and RV segments of ectopic endocardial impulse formation in patients with normal cardiac anatomy. (Circulation 1990;82:879–896)

The morphology of the electrocardiogram (ECG) during ectopic ventricular activation has been the subject of study throughout this century. Based on the hypothesis that the configuration of the QRS complex relates to the site of origin of ventricular activation, remote field bipolar lead recordings were used in early animal experiments to discriminate between left- and right-sided ventricular stimulation. After the advent of the precordial unipolar leads, invasive data obtained in humans predominantly included information on the electrocardiographic morphology produced by implanted cardiac pacemakers. It became feasible to obtain a detailed electrocardiographic description of the site of origin of ectopic activation after catheter and intraoperative pace mapping procedures were introduced into the management of cardiac arrhythmias. Analysis of the 12-lead ECG generated during ventricular pacing at multiple endocardial and epicardial sites led to the identification of ventricular regions with a characteristic QRS configuration but did not permit precise localization of the site of activation onset. Multiple-lead electrocardiography has been applied in a similar setting and seems a promising alternative because of its documented value in detecting local-
ized electrophysiological phenomena. In animal experiments, Spach et al demonstrated that ectopic sequences, originating from electrical stimulation of epicardial sites lying 2–3 cm apart around the ventricular circumference, can be readily discriminated from one another by their corresponding typical QRS and ST-T potential distributions on the thorax. An extension of this approach to the human situation by determining the total body surface QRS patterns during endocardial pacing at multiple accurately localized sites in the normal left and right ventricle has not yet been reported.

The purpose of the present study was to establish the resolution of body surface mapping in localizing the site of ectopic stimulation in the normal heart and to create a reference data base containing the spectrum of electrocardiographic map patterns.

**Methods**

**Patients**

After a routine diagnostic programmed electrical stimulation protocol, left (LV) or right ventricular (RV) pacing was performed in a group of 12 patients with cardiac arrhythmias and no evidence of structural heart disease (Table 1). Normal LV and RV function was confirmed by two-dimensional echocardiography and biplane cineventriculography. Informed consent was obtained from each patient before the study.

**Endocardial Pacing**

After local anesthesia and heparinization, a 6F quadripolar USCI catheter was introduced percutaneously into a femoral artery or vein for LV or RV pacing. Bipolar stimulation was conducted with the distal pair of electrodes (interelectrode distance, 0.5 cm); the tip electrode was used as the cathode. Stimulus amplitudes were slightly above diastolic threshold with a duration of 1 msec. The pacing rate was chosen to be either 100 or 120 beats/min, depending on the heart rate during sinus rhythm. In patient 9, the rate was adjusted to 150 beats/min to cope with a temporary increase in the sinus rhythm frequency (≤125 beats/min).

**Anatomical Localization of Pacing Sites With Biplane Fluoroscopy**

The catheter was positioned at a variety of randomly chosen sites to get an overall coverage of the LV or RV endocardial surface. During each pacing sequence, biplane cineradiograms were obtained synchronously in 45° left anterior oblique (LAO) and 45° right anterior oblique (RAO) projections, at a stationary level of end-tidal volume inspiration. Two video monitors, which continuously displayed LAO and RAO views during fluoroscopy, were used to check the position of the diaphragm to ensure similar respiratory phases during the course of the study. These monitors were also used to verify catheter stability and to provide additional on-line control marking of the catheter position. Biplane contrast cineventriculography was performed immediately after pace mapping; end-diastolic frames were digitized to delineate positions of anatomical reference points—the LV or RV apex and the center of the mitral and aortic valve ring, or tricuspid and pulmonary valve ring. Also, the position of a steel reference frame applied on the radiographic tubes and the optical center of the equipment were marked to correct for fluoroscopic magnification and distortion. Subsequently, the end-diastolic biplane cineradiograms acquired during every pacing sequence were selected, digitized, and superimposed on the end-diastolic cineventriculograms. The three-dimensional location of each pacing site was then computed with respect to the position of the anatomical reference points and represented by cylinder coordinates according to the method developed by Hauer et al. These authors found a fluoroscopic localization error of ≤0.7 cm.

### Table 1. Patient Summary

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex/age (yr)</th>
<th>Cardiac arrhythmia</th>
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<th>RV pacing sites (n)</th>
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LV, left ventricular; RV, right ventricular; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia; V, verapamil; F, flecainide; P, propafenon.
Display of Pacing Sites on Endocardial Polar Projection

A schematic LV or RV endocardial representation was used to compare the computed anatomical location of pacing sites obtained from different patients in a standardized format. The polar projection of the left ventricle in conjunction with the corresponding endocardial anatomy is portrayed in Figure 1. The radius in this polar projection equals the mitral valve–LV apical axis, the center marks the position of the LV apex, and the outline defines the plane of the mitral valve ostium. Similarly, in the RV endocardial projection, the radius equals the tricuspid valve–RV apical axis, the center marks the position of the RV apex, and the outline defines the plane of the tricuspid valve ostium. An individual polar projection was first determined for each patient; cineradiographically computed cylinder coordinates were used to define the actual pacing site positions on this projection. Subsequently, standardization of the polar projections from different patients was obtained by normalizing the radius of each projection. Figure 2 gives an example of a LV polar projection and a depiction of the corresponding LAO and RAO end-diastolic LV endocardial contours, containing the digitized location of six pacing sites and anatomical reference points obtained in patient 1.

Body Surface Mapping

Electrode array. The lead set consists of 62 irregularly positioned radiotransparent carbon electrodes\(^2^1\) fitted in a set of flexible straps\(^2^2\) that cover both the anterior and posterior sides of the chest (Figure 3A). The format of the lead set, as well as 42 of its electrode positions, were based on two versions of a clinically practical 32-lead array. These two limited lead arrays were designed by Lux et al.\(^2^4\) who used a uniform transformation matrix to extrapolate the thoracic lead sites with the highest signal information content from a regular 192-electrode grid. In the present study, linear interpolation was used as an alternative technique to determine potentials at unmeasured lead sites.\(^2^5\) This method made it necessary to extend the number of recording sites. Meanwhile, the clinical practicality in terms of the number of applied electrodes had to be considered. Therefore, a maximum of 62 recording sites were chosen by adding optimal lead sites determined in other studies\(^2^6-^2^9\) and by integrating the six standard precordial leads.

Recording and processing. A portable microcomputer-based mapping system\(^3^0\) was used to acquire a set of 62 ECGs during LV and RV pacing. Unipolar lead tracings were simultaneously recorded using Wilson’s central terminal as reference. Shielded lead wiring and a driven right leg circuit\(^3^1\) ensured a noise level and MAINS frequency interference at less than 27 \(\mu\)V. The waveforms of a single cardiac cycle were amplified,\(^3^2\) sampled, converted to a digital format at a frequency of 500 Hz, and stored on floppy disk; for each sampling sequence performed every 2 msec, the total time difference involved from the onset of
sampling of the first electrode through the 62nd electrode was 15 µsec. A linear interpolation algorithm was used to adjust for baseline drift or offset after manual selection of an isoelectric time instant before the stimulus artifact and after the T wave. Inferior-quality waveforms were rejected (mean, 1±1.7 per map) and replaced by values computed from surrounding lead recordings. After data transfer to a PDP 11/73 minicomputer, hard-copy body surface maps (isopotential maps) were produced at 2-msec intervals during the QRS complex and at 20-msec intervals during the ST-T interval. QRS onset was specified as the earliest time instant at which an extreme voltage of ±0.2 mV was observed, whereas QRS offset was visually determined at the J-point. Finally, the data were transferred to a VAX 11/750 minicomputer to perform data reduction by generating integral maps of the total QRS complex and the first 40 msec of the QRS complex; an integral map (isoregion map) was constructed from 62 lead time integral values that were each determined by multiplying the sampling interval with the sum of the potentials sampled within a certain time interval.33

Display. Figure 3 illustrates a total QRS integral map (B) in relation to the thoracic anatomy and lead positions (A). The left side of the map corresponds to the front and the right side to the back of the chest. Linear interpolation25 was used to connect points with equal time integral values to form isointegral lines. Positive and negative isointegrals are represented by the solid and dashed lines in the shaded area of the map, respectively, whereas the zero isointegral is marked by the dotted line. The isointegral lines are separated by an automatically determined linear incremental step that depends on the magnitude of each extreme and is, for display clarity, restricted to an upper limit of 20 contour lines per map. The locations of the centers of the positive (maximum) and negative (minimum) extremes are indicated by the plus and minus signs, and their amplitudes are expressed in mVms below the map. The map format and construction are identical for integral maps and body surface maps. In the latter representation, however, contour lines specify positive and negative isopotential values in millivolts at a certain time point in the cardiac cycle.

Analysis Protocol

Maps generated by LV or RV pacing were analyzed as separate groups. Body surface maps were inspected to document sequential morphological changes during ectopic ventricular activation and repolarization. Subgroups of integral maps with nearly identical total QRS morphology were selected by careful comparison of the distribution of positive and negative values on the map, while the observer was blinded to the corresponding stimulus site locations. Three spatial parameters were visually judged: 1) location of the extremes, 2) mutual distance of the extremes, and 3) zero line configuration. Mean total QRS integral maps were computed for each subgroup, and endocardial locations of pacing sites, belonging to the corresponding subgroups, were delineated on the polar projection of the endocardium. Thus, a set of characteristic mean total QRS integral maps with their respective endocardial seg-
ments of activation onset was obtained for each ventricle. Finally, the selection of subgroups was quantitatively validated, and the size of the endocardial segments was approximated.

**Quantitative Analysis**

Values are given as mean±SD. Instead of acquiring quantitative data on each of the three spatial parameters that were considered during visual analysis of the integral maps, quantitative pattern evaluation was based on a more comprehensive comparison of the QRS integral morphologies by computing the correlation coefficient between the time integral values of the 62 recording sites of pairs of integral maps. Application of this technique offers simultaneous evaluation of the three visual spatial parameters and the generation of a single value to express the quantitative relation between two integral map patterns. The correlation coefficient was used to conduct the following three-step statistical analysis. First, pattern uniformity within each subgroup of integral maps was determined by performing a jackknife procedure; individual maps were correlated, one by one, with the mean map of the membering maps within the subgroup. The mean values and SDs of the generated correlation coefficients were used to quantify the similarity in pattern within each subgroup. Second, pattern variability within each set of mean integrals was established individually for LV and RV pacing by applying an intraset cross correlation (i.e., all individual mean integrals within a set were correlated with each other to quantify the differences in pattern between the various mean integral maps). Third, pattern variability between the set of mean integrals produced by LV pacing and the set of mean integrals generated by RV pacing was evaluated by performing an interset cross correlation (i.e., every mean LV integral was correlated with each individual mean RV integral).

The mathematical method used to compute the endocardial location of pacing sites was also applied to calculate the distance between different pacing sites in individual patients. This enabled us to estimate the area size of endocardial segments with nearly identical QRS integral patterns and to validate the use of the pace-mapping technique by examining its reproducibility; pairs of disparate pacing sites with nearly identical map morphologies,
obtained in individual patients, were selected. The size of the corresponding endocardial segment was approximated by calculating the circular area between the two sites. The diameter of the circle equaled the distance between the two sites. The largest area was chosen to represent the size of a segment in case multiple pairs of disparate sites were obtained in that segment. In individual patients, pacing was performed twice at a similar endocardial location at different time instants during the procedure. The pattern uniformity of the two generated integral maps was assessed, and the distance between the pacing sites was computed.

Results

Body Surface Maps

Body surface maps were generated during ventricular pacing at 182 endocardial sites in 12 patients. Pacing was performed at 99 LV sites in eight patients and 83 RV sites in six patients (Table 1). Ectopic ventricular activation was characterized by dipolar potential distributions in all pacing sequences. In two thirds, the dipolar pattern remained stable throughout the QRS complex. A typical example of a temporally uniform distribution of potentials is displayed in Figure 4. In this case, the inferior apex of the right ventricle was paced in patient 7. Both extremes and the zero line remain stationary at their initial position and show only a subtle movement toward the end of the QRS complex. Voltage amplitudes increase gradually, demonstrating high-negative and low-positive potentials, and subsequently decrease at the end of ventricular activation.

In the remaining third of the pacing sequences, there was a clear shift of the potential extremes during the onset of ventricular excitation. With RV pacing, this shift was observed during the first 22±14 msec (range, 8–64 msec) in 20% of the body surface maps. The majority of the maps generated by pacing of the lateral right ventricle (69%) showed this phenomenon. With LV pacing, a shift was noted during the initial 31±15 msec (range, 10–62 msec) in 42% of the body surface maps; the corresponding pacing sites were equally distributed over the different LV regions. Figure 5 gives a representative activation sequence obtained in patient 8 during pacing at the middle section of the LV anterolateral wall. During the first 40 msec, the minimum shows a major shift from the upper right anterior thorax toward the left back without changes in amplitude, whereas the maximum shows a gradual voltage increase and moves from the lower left anterior side toward the right side of the chest. In contrast, stable dipolar potential distributions with decreasing amplitudes and oppositely oriented electrical forces are present during the remaining part of the QRS complex.

Ventricular recovery potentials displayed the overall mirror image of the QRS potential distribution, although slight pattern variations in the orientation of the extremes or the contour of the zero line did occur in the ST-T maps.

Total QRS Integral Maps

The integral of the total QRS was considered superior to represent each pacing sequence compared with the 40-msec QRS integral due to the frequently observed variation in the potential pattern during the initial part of ventricular activation. From the 99 total QRS integral maps produced by LV pacing, 25 subgroups of maps with nearly identical patterns were composed; from the 83 maps generated by RV pacing, 13 subgroups were formed. To facilitate description of the following two representative subgroups, individual pacing sites belonging to a subgroup are referred to by letter. Total and
40-msec QRS integral maps of a subgroup in which three of five body surface maps showed initial pattern variation during LV pacing at the midanterior wall (segment LV22) are given in Figure 6. All total QRS integral maps show uniform patterns with negative integral values covering the left axilla and back and positive values over the right anterior thorax. In contrast, the integrals over the first 40 msec of the QRS complex of sites B, C, and D express a considerable variation in pattern. Extreme amplitudes demonstrate some degree of variation, with both total and 40-msec QRS integral maps. The integral maps of site B were computed from the same ectopic sequence as the body surface maps displayed in Figure 6; note that the 40-msec integral map pattern matches the morphology of the body surface map at 30 msec, whereas the total QRS integral shows a pattern compatible with the potential distribution during the larger part of the R wave (40–90 msec). An example of a subgroup of integral maps obtained during RV pacing at the inferior septum (segment RV12) is given in Figure 7. All patterns display a minimum on the lower right anterior chest and a left axillary maximum; both extremes are closely spaced and interlined by a characteristic zero line morphology.

**Endocardial Location of Pacing Sites With Nearly Identical Total QRS Integral Map**

The anatomical locations of the pacing sites belonging to each subgroup of nearly identical total QRS integral maps are indicated as segments on the endocardial polar projection of the left and right ventricle. On the LV polar projection (Figure 8), 18 segments can be identified in the anterosetal compared with only seven segments in the posterolateral region. The overlap of adjacent segments (e.g., LV3, LV4, LV5, and LV23) is probably due to the error introduced by the fluoroscopic localization of the pacing site. It should also be noted that the polar representation causes some distortion in the size of apical versus basal segments; the latter is displayed as relatively large compared with the former. The area size of seven LV segments, located in the apical septal and anterior regions, could be approximated in individual patients and ranged from 1.9 cm² (segment LV11) to 6.0 cm² (segment LV23) (mean, 3.3±1.4 cm²). There were no differences in area size distribution when comparing LV apical with LV basal locations. The segments on the RV polar display (Figure 9) are fewer compared with LV segments and, except for the anterior region, do not discriminate between middle or basal locations. Overlap is present at the outflow tract (segments RV4, RV5, and RV7). The area sizes of 11 RV segments could be estimated in individual patients and varied between 2.2 cm² (segment RV9) and 12.8 cm² (segment RV4) (mean, 6.7±2.9 cm²). Apart from the basal anterior and outflow region, which exhibits a large segment size and consequent low electrocar-
diographic sensitivity, the other RV regions expressed a comparable area size distribution.

**Mean Total QRS Integral Maps**

*Left ventricular pacing.* The mean total QRS integral maps of the 25 subgroups produced during LV pacing are displayed in Figure 10. Discrimination of adjacent pacing sites at the base of the heart can be performed by regarding the rotation in the position of the maximum and minimum when advancing horizontally through the upper row of mean integral maps, from map 21/23 (basal lateral/anterior pacing) to map 18/20 (basal posterior/lateral pacing). The same phenomenon can be observed in the second and third rows of maps, which correspond with pacing at the middle transverse section of the heart. The maps produced by apical pacing (bottom two rows) are dominated by superiorly directed forces—the minimum lies at a quite similar location on the lower anterior thorax, whereas the maximum moves from the upper front to the upper back with pacing performed at apical and apical posterior sites (segments LV1 and LV15) or apical septal sites (segments LV2 and LV11), respectively. Discrimination at the longitudinal level (from middle to basal) can be clear-cut with large differences in extreme location and zero line contour (e.g., segments LV9 and LV10) or subtle with a comparable zero line morphology and similar extreme orientation but a significantly different distance between the extremes (segments LV5 and LV25). The ultimate right column of three mean integral maps have been ranked jointly due to their correspondence in pattern, even though they were produced during pacing at the anterior (segments LV3 and LV22) and lateral (segment LV20) quadrants of the heart. It is interesting to observe the corresponding three endocardial pacing areas on the LV scheme with an oblique orientation from midanterior to basal lateral. The same accounts for the ultimate left column of mean integral maps (segments LV24 and LV21) and a posterior column (segments LV19 and LV18). This phenomenon might
be explained by the natural oblique leftward orientation of the heart in the chest. A left bundle branch block (LBBB) morphology (negative QRS polarity in V1) was noted in eight mean integral maps, corresponding with pacing at the apex and the apical, middle, and basal septum (segments LV1, LV2, LV4, LV7–LV9, LV11, and LV13).

Positive and negative mean QRS time integral amplitudes (Table 2) display high voltages when the maximum (e.g., segment LV20) or minimum (e.g., segment LV7) is situated near the precordial area versus low voltages when the maximum (e.g., segment LV2) or minimum (e.g., segment LV17) is located on the back of the chest. The mean QRS time integral amplitudes demonstrate fairly high standard deviations, reflecting a considerable voltage variability within the subgroups. The mean QRS duration for each subgroup is listed in Table 2. Five larger regions with characteristic mean QRS durations can be identified: basal septum and basal anteroseptal junction (segments LV6, LV10, LV13, and LV14), 95±8 msec; middle septum and midanteroseptal junction (segments LV2, LV4, LV5, LV9, and LV12), 105±10 msec; apex, apical septum, and apical posterior wall (segments LV1, LV7, LV11, and LV15), 114±13 msec; anterior and midlateral wall (segments LV3, LV19, and LV22–LV25), 122±14 msec; and basal lateral and basal posterior wall (segments LV16–LV18, LV20, and LV21), 129±21 msec. It should be noted that three patients received antiarrhythmic agents (Table 1), which might have influenced the QRS width during pacing.

**Right ventricular pacing.** Figure 11 represents the 13 mean total QRS integral maps computed from each subgroup generated during RV pacing. The mean QRS integrals display less pattern variation compared with the QRS morphologies produced by LV pacing. In all 13 maps, the minimum is located on the anterior thorax (LBBB pattern in V1), and pattern discrimination relies predominantly on the zero line morphology and the position of the maximum. Separation of mutually distant locations can be well defined (e.g., pacing at the outflow tract and posterior wall [segments RV6 and RV11]) with the mean QRS integral maps displaying inferior and superior forces, respectively. However, it is difficult to distinguish among maps showing a leftward-oriented electrical axis with comparable zero line contour and extreme positions (e.g., pacing at the lateral and septal wall [segments RV9 and RV13]). Discrimination among adjacent sites can be straightforward (e.g., with QRS integrals generated by pacing at the anterior wall [segments RV2–RV4]), showing marked differences in the location of the maximum and the zero line contour, or complex (e.g., with maps produced by pacing at the posterior wall and septum [segments RV10–RV12]), where a subtle
shift of the maximum is the only discriminating parameter.

Most mean QRS time integral amplitudes (Table 3) are dominated by a steep voltage gradient with high negative voltages on the anterior chest (e.g., segment RV13) due to the proximity of that torso area to the current sources within the heart, whereas positive voltages have a low amplitude (e.g., segment RV2) due to a location on the body surface that is considerably more distant from the cardiac current sources. It should be noted that the mean voltage standard deviations exhibit the presence of within-segment amplitude variability. Nevertheless, when large mean voltage differences exist between two segments, negative time integral amplitudes might be used as an adjunct to discriminate among maps with comparable spatial QRS characteristics and produced by pacing at mutually distant sites (e.g., segments RV9 and RV13). Four regions with comparable QRS durations can be deduced from the listings in Table 3: anterior outflow tract (segment RV5), 104±11 msec; anterior wall, posterior outflow tract, and midlateral wall (segments RV2–RV4, RV6, and RV8), 115±12 msec; posterolateral wall, posterior wall, and septum (segments RV9–RV13), 123±18 msec; and apex and anterolateral wall (segments RV1 and RV7), 132±18 msec.

Quantitative Evaluation of Mean Total QRS Integral Map Patterns

First, a numerical assessment of the uniformity of the integral map patterns within each subgroup, in relation to the number of sites and number of patients, is expressed in Tables 2 and 3. A high pattern uniformity corresponds with a high mean correlation coefficient and a low standard deviation. For LV pacing, r values ranged from 0.98±0.01 (segment LV17) to 0.89±0.07 (segment LV22). It is worth mentioning that even though the lowest quantitative spatial uniformity was found in the subgroup of segment LV22, the actual qualitative pattern differences appear to be marginal (left column of integral maps in Figure 6). For RV pacing, the mean r values were higher and varied between 0.99±0.01 (segments RV9, RV11, and RV13) and 0.95 (segment RV6). Second, quantitative pattern comparison within the set of 25 LV mean QRS integrals was obtained by performing a total of 300 different cross correlations. The results ranged from high negative correlations (major pattern differences) between
mean integral maps obtained during pacing at mutually distant sites (i.e., septal and lateral regions, \( r = -0.98 \) [segments LV8 and LV20]) to high positive correlations (minor pattern differences) at some adjacent sites (i.e., posterobasal zone, \( r = 0.96 \) [segments LV17 and LV18]). The coefficient generated in 295 of 300 LV mean integral cross correlations (98%) was lower than the mean correlation coefficient obtained with the jack-knife procedure in the two subgroups (intrasubgroup map correlation) belonging to each compared pair of LV mean integral maps. This means that the pattern differences between the LV mean integrals were nearly always greater than the pattern differences within the corresponding individual subgroups. The remaining five cross correlations showed values that were equal (i.e., \( r = 0.94, 0.96, 0.94, \) and 0.95 [segments LV7 and LV8, LV17 and LV18, LV21 and LV23, and LV23 and LV25, respectively]) with one of the two corresponding mean intrasubgroup map correlation coefficients listed in Table 2, or slightly higher (i.e., \( r = 0.94 \) [segments LV12 and LV16]) than the two corresponding mean intrasubgroup map correlation coefficients shown in Table 2. It should be noted that the latter five cross correlations represented mean integral maps acquired during pacing at adjacent segments. Seventy-eight different cross correlations were carried out to assess the quantitative variability among the 13 RV mean integral maps. The results ranged from negative pattern correlations among distant sites, outflow tract, and inferior septum (\( r = -0.40 \) between segments RV5 and RV12) to high positive correlations among adjacent sites at the lateral wall (\( r = 0.96 \) between segments RV7 and}

**FIGURE 9.** Polar projection of right ventricular endocardium, including a 13-segment subdivision. Segments represent areas in which pacing produced a nearly identical total QRS integral morphology. Positions of anatomical reference points (i.e., apex and center of pulmonary valve ring [PVR] are marked in common with orientation of endocardial quadrants. Note that segment 5 is situated at anterior side of outflow tract, whereas segment 6 is located at posterior side. A high density of segments can be observed in apical and middle anterior wall (segments RV2 and RV3), whereas other regions, specifically basal anterior and outflow zone (segment RV4), exhibit a lower electrocardiographic resolution.

- = pacing site

○ = segment with nearly identical total QRS integral

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RV8). Considerable pattern compatibility was also found among distant RV regions (i.e., septal and lateral/posterior pacing sites \( r = 0.97, 0.94, \) and 0.92 between segments RV13 and RV8, RV9, and RV10, respectively). Despite this observation, it was found that the coefficient obtained in 77 of 78 RV mean integral cross correlations (99%) was lower than the corresponding two mean intrasubgroup map correlation coefficients. These findings demonstrate that there were virtually always greater pattern differences between RV mean integral maps than within the corresponding individual subgroups. Only one cross correlation revealed a coefficient of 0.96 (segments RV3 and RV8) that was equal with one of the two corresponding mean intrasubgroup map correlation coefficients displayed in Table 3. Third, intergroup pattern comparison of the LV set with the RV set of mean integrals was obtained by performing a total of 325 different cross correlations; negative correlations were found in five LV mean integrals, low positive correlations \( (r = 0.43 \pm 0.23) \) in 13 LV mean integrals, and high positive correlations \( (r = 0.97 \pm 0.02) \) in seven LV mean integral maps. The latter group of seven LV mean integrals demonstrated LBBB morphology in \( V_1 \) and originated from pacing at the apex or the apical and middle septum (segments LV1, LV2, LV4, LV7–LV9, and LV11), whereas the matching RV mean integrals were generated by apical, anterior, lateral, and septal pacing (segments RV1–RV4, RV8, and RV13) (Table 4). The negative QRS time integral amplitudes were additionally listed in Table 4 because they might perform as a potential discriminator between these two subsets of matching LV and RV mean integral maps, high negative voltages being characteristic for RV pacing (mean, 237±63 mV−sec) and low negative voltages for LV pacing (mean, 164±71 mV−sec).

**Validation of Pace-Mapping Technique**

The pace-mapping technique was tested for its reproducibility by pacing twice at a similar endocardial location in 10 patients. Total QRS integral maps were generated at five different pairs of LV sites and
five different pairs of RV sites. A representative pair of integral maps, obtained in patient 4 during pacing at the apical septum of the left ventricle (segment LV11), is displayed in Figure 12; nearly identical extrema locations, zero line morphologies, and voltage distributions can be observed. For the 10 pairs of integral maps, a high mean $r$ value of 0.98±0.02 and mean distance between the pacing sites of less than 0.7 cm was found. There were no significant differences in the results obtained from LV or RV pacing.

**Discussion**

This is the first report on an extensive total body surface electrocardiographic data base for high-resolution localization of the site of origin of ectopic ventricular activity in the structurally normal human heart. Analysis of the QRS integral morphology produced during LV and RV pacing at 182 distinct endocardial sites resulted in a total of 58 characteristic body surface map patterns. Manifest qualitative and quantitative spatial differences were observed in the 25 mean QRS integrals generated by LV pacing at endocardial segments with an approximated area size of 3.3±1.4 cm$^2$. The 13 specific ectopic RV activation sequences exhibited less obvious qualitative and quantitative morphological variation and larger estimated segment sizes of 6.7±2.9 cm$^2$. Although mean QRS time integral amplitudes showed intrasubgroup variability, they might prove advantageous in discriminating some of the RV regions with less obvious pattern differences (septum and lateral wall). Similarly, amplitude information could appear important to distinguish maps with LV activation onset (apex and midseptum) from spatially similar maps with RV activation onset (apex, septum, and anterior wall).

The experimental design in the present study was directed at investigating a large number of pacing sites (mean, 15.2 sites per patient) in a limited number of patients instead of studying a small number of pacing sites in a much larger series of patients. This particular approach provides enhanced power of the analysis and allows unambiguous interpretation of the results because all factors within one patient remain constant, whereas the only variable of change is the location of the pacing site. The pace-mapping technique, using anatomical fluoroscopic localization of stimulus sites, proved to be reproducible and offered a unique method of representing all 182 endocardial sites in a simple polar projection of the left and right ventricles. The composition of endocardial segments was guided by the selection of

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Pacing sites (n)</th>
<th>Patients (n)</th>
<th>Intrasubgroup map correlation (r)</th>
<th>Positive QRS integral amplitude (mVsec)</th>
<th>Negative QRS integral amplitude (mVsec)</th>
<th>QRS duration (msec)</th>
<th>Location of endocardial segment</th>
</tr>
</thead>
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<td>4</td>
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<tr>
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<td>0.96 ± 0.02</td>
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<td>0.95 ± 0.02</td>
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<td>128 ± 10</td>
<td>100 ± 10</td>
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<td>5</td>
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<td>76 ± 18</td>
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<td>40 ± 4</td>
<td>121 ± 5</td>
<td>Basal anterior</td>
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TABLE 3. Characteristics of Total QRS Integral Map Subgroups—Right Ventricular Pacing

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<tr>
<th>Subgroup</th>
<th>Pacing sites (n)</th>
<th>Patients (n)</th>
<th>Intrasubgroup map correlation (r)</th>
<th>Positive QRS integral amplitude (mVmsec) Mean</th>
<th>SD</th>
<th>Negative QRS integral amplitude (mVmsec) Mean</th>
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<td></td>
<td>344 23</td>
<td></td>
<td>126 2</td>
<td></td>
<td>Middle septum</td>
</tr>
</tbody>
</table>

Figure 11. Mean total QRS integral maps generated during right ventricular pacing in combination with a diagram of right ventricular endocardium that contains anatomical locations of 13 corresponding segments. Encircled numbers (1–13), indicated above each map and on anatomical diagram, associate a specific mean integral map morphology with its endocardial pacing origin. Diagram features endocardium after cutting free wall open at its septal and basal insertions; it was first used by Josephson et al13 as a six-site catheter mapping scheme (thin-lined numbers 13–18). Mean integral map display is simplified by deleting isointegral lines; maps are arranged according to a global subdivision of right ventricle into longitudinal quadrants (septum, anterior, lateral, and posterior), next to an apical and superior section (outflow). Mean integral map 1 seems to demonstrate two maxima that are in fact representative of equal positive time integral values obtained with spatially opposed electrodes on upper side of anterior and posterior torso. Note that endocardial segment 6 is located at posterior side of outflow tract. Right ventricular endocardial diagram is reproduced with permission of American Heart Association.

quantitatively validated uniform QRS integral map patterns and not performed according to a predetermined mapping scheme. The currently used schemes are based on an anatomical5–9 or geometric9 subdivision of the ventricles and are limited by the fact that they take no account of the intrinsic variation in electrocardiographic sensitivity for the different cardiac areas. Application of a predetermined subdivision of the heart will result in an underestimation of the resolution in areas with a high electrocardiographic sensitivity (e.g., anteroseptal left ventricle) and an overestimation in areas with a low electrocardiographic sensitivity (e.g., posterolateral left ventricle). It is our opinion that the actual resolution of body surface maps in localizing the site of ectopic activity is better defined by a regional ventricular subdivision that emanates from the observed variation in ECG patterns. Using this approach, we were able to obtain a complete assessment of the major regional differences in electrocardiographic sensitivity.
Previous Studies

**Twelve-lead electrocardiogram.** Josephson et al.\(^5\) used the frontal plane QRS axis to differentiate among endocardial pacing at six different RV regions. In another report, Waxman and Josephson\(^7\) used the scalar QRS morphology and the frontal plane QRS axis to distinguish among endocardial pacing at 12 different LV regions. By applying multiplane fluoroscopy to direct catheter positioning, these authors were able to detect endocardial regions of 5–10 cm\(^2\) in patients with and without wall motion abnormalities.\(^{35}\) A similar study in patients with previous myocardial infarction was recently conducted by Kuchar et al.\(^8\) They defined an algorithm for localization of the origin of ventricular tachycardia, based on the QRS polarity obtained during endocardial pacing at 24 LV sites. Holt et al.\(^10\) used epicardial pacing at 27 LV and RV sites next to endocardial pacing at the mitral valve papillary muscles to describe the QRS pattern in a heterogeneous patient group. By using the frontal and horizontal plane QRS axis, these authors were able to assess regionally characteristic parameters but did not reach the full resolution of their epicardial mapping scheme. Essentially, the 12-lead electrocardiographic criteria defined in the abovementioned studies\(^5,7,9,10\) are in agreement with our results. It should be noted, however, that much of the discriminative information in the body surface QRS integral maps was located in thoracic areas not covered by the standard precordial leads. Moreover, we have shown that even with multiple-lead recordings, differentiation of QRS configurations produced during pacing at adjacent sites in certain ventricular regions can be difficult to achieve. For example, separating QRS morphologies on the basis of the 12 standard leads, recorded during pacing at segments LV16–LV19 (posterolateral), is hardly possible when one considers the comparable superior frontal axis and precordial distribution of positive QRS time integrals (R waves) in all four of the corresponding mean integral maps (Figure 10).

**Body surface mapping.** Ushijima et al.\(^11\) reported on LV and RV pacing at seven epicardial sites in patients with nonischemic ventricular tachycardia. Body surface map characterization was performed by means of the locations of the maximum and minimum 40 msec after QRS onset. In a patient population without myocardial infarction, Kamakura et al.\(^12\) suggested the use of the location of the minimum, with an amplitude of −0.5 mV during early QRS, as a pattern determinant for endocardial pacing at 18 LV and RV sites. It appeared from our results that the location of one or two extremes, as the sole criterion for analysis, was not sufficient for a detailed localization of ectopic activity. Especially with RV pacing, we obtained fairly equal locations of the minimum in most of the endocardial regions. Moreover, we considered body surface maps of initial ventricular activation unreliable because of the frequently observed pattern instability during the first 8–64 msec of QRS. In patients without myocardial infarction, Hayashi et al.\(^13\) performed LV and RV pacing at seven endocardial and epicardial regions with the objective of classifying ventricular extrasystoles. Apart from the extreme positions at 30 msec in the QRS complex, body surface map discrimination additionally included analysis of sequential changes in later phases of ventricular excitation. Our data compared well with five of the seven QRS patterns which they described. In the latter two studies,\(^12,13\) there was no mention of the applied fluoroscopic techniques or the consequent accuracy with which locations of pacing sites were assessed. Other groups\(^7,38\) determined the endocardial position of

### Table 4. Comparable Mean Total QRS Integral Maps—Left and Right Ventricular Pacing

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Location of LV endocardial segment</th>
<th>Negative QRS integral amplitude—LV pacing (mean mV·msec)</th>
<th>Subgroup</th>
<th>Location of RV endocardial segment</th>
<th>Negative QRS integral amplitude—RV pacing (mean mV·msec)</th>
<th>LV-RV mean map correlation (r)</th>
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<td>141</td>
<td>RV pacing</td>
<td>Apex</td>
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<td>Middle septum</td>
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<td>128</td>
<td></td>
<td>Basal anterior</td>
<td>221</td>
<td>0.92</td>
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</table>

LV, left ventricular; RV, right ventricular.

**Figure 12.** Pair of highly correlated total QRS integral maps obtained after pacing twice at similar endocardial locations. Both patterns demonstrate nearly equal spatial features. Number of positive isointegral lines is larger in total QRS integral map of site B due to a different linear incremental step: 4 mV·msec compared with 8 mV·msec in map of site A.
RV pacemaker leads quantitatively with the use of anteroposterior and lateral fluoroscopic projections. Instead of using descriptive map pattern analysis, modeling techniques were evaluated on their ability to locate the origin of RV activation. Pacing lead positions were computed from the body surface map with a mean error of 2.5 cm or 4.18 cm.

Left Bundle Branch Block Morphology During Left Ventricular Pacing

In normal subjects as well as in patients with structural heart disease, it has been reported that LBBB QRS configurations can be obtained with LV pacing.8,7 Waxman and Josephson7 demonstrated this phenomenon by pacing at the apical and middle parts of the septum and attributed it to preferential RV excitation after transeptal propagation of the activation wave front. We obtained similar results with an additional site at the basal septum. While the voltage distributions were significantly different, the spatial comparability of LBBB QRS integrals produced during LV pacing with certain LBBB QRS integrals obtained during RV pacing was remarkable. In fact, quantitative comparison revealed that nearly all of the QRS patterns produced at these septal LV segments demonstrated the highest correlation coefficient with a pattern generated at an exactly opposite septal or anterior RV segment.

Experimental Perspectives

Ectopic ventricular activation by epicardial stimulation is characterized by stable dipolar body surface potential distributions18,39,40 and is caused by uniform muscle-to-muscle propagation of a single wave front that advances centrifugally from its site of origin in one direction across the heart toward the opposing ventricle.31 Our data underline these experimental findings despite the fact that endocardial stimuli were applied in the present study. However, we did observe a clear shift of the extremes at the onset of QRS in some of our pacing sequences. This might be understood by intramural and remote field body surface recordings performed by Scher and Young42 during endocardial pacing in the dog heart. They observed an initial 30-msec negative deflection in lead II coinciding with the spread of the activation wave front from its endocardial stimulus site at the LV apex toward the overlying epicardium (compare with V1 in Figure 5). It remains unclear why this phenomenon is only noted in one third of our data. Other factors might be involved (e.g., local Purkinje invasion41,43) or the effect of the distance of the exploring surface lead area to the region of early activity.39,40

The importance of low-level body surface potentials in distinguishing localized cardiac events has been emphasized in the literature.18,23,39 It also appeared from our data that subtle QRS integral pattern discrimination of cardiac activity in electrocardiographic less sensitive regions (e.g., posterolateral left ventricle) can be achieved only with a detailed evaluation of the zero line contour.

Separation of map morphologies produced by epicardial stimulation of adjacent basal sites with a distance of 2–3 cm has been reported in animals; in both large and small chimpanzees,18 seven equal regional map patterns were found. Similar results were obtained in adults44 as well as in infants45 with Wolff-Parkinson-White syndrome by discriminating ventricular potential distributions into one of seven underlying ventricular preexcitation sites. Accordingly, we found comparatively little effect of interpatient differences in sex and body size on body surface map morphology; the segmental subgroups, which predominantly contained total QRS integral maps from different patients, expressed a high qualitative and quantitative spatial uniformity.

Study Limitations

The accuracy of catheter pace mapping is limited by the use of fluoroscopy to determine the endocardial location of the stimulus site.35 A biplane fluoroscopic coordinate grid, with a 2-cm resolution in each image, has been proposed9 as an alternative for conventional visual translation of two-dimensional radiographic views into a diagramatic representation of the heart's anatomy.35,36 To improve the fluoroscopic localization accuracy, we used a quantitative cineradiographic method with an anatomical resolution of ≤0.7 cm.19 Nevertheless, we found slight segmental overlap on the polar projections to most likely be caused by the catheter localization error. Other limitations of catheter pace mapping include the technical difficulties encountered when trying to obtain stable catheter positions with reliable capture at all endocardial regions of a nondilated normally contracting ventricle. Therefore, pacing at the anterolateral LV apex was not achieved, and at three of the LV segments pacing appeared possible at only one site per segment. In this respect, it is also important to realize that the electrocardiographic resolution that we obtained depends to a certain extent on the total number and distribution of pacing sites. Clearly, adding more pacing sites would have resulted in an improved delineation of the segmental sizes and their endocardial locations. However, considering the amount and scattered distribution of sites in the present study, it is unlikely that this would have resulted in an important increase of the number of endocardial segments with a characteristics body surface electrocardiographic morphology during ectopic ventricular activation.

Clinical Implications

The purpose of the present study was to establish a detailed reference data base of mean body surface QRS integral maps characteristic of the onset of ectopic ventricular activation in localized endocardial segments. The clinical value of this data base lies in its potential to provide a localization of the site of origin of ventricular arrhythmias that is more precise
than a mere regionalization, which is currently acquired with the 12-lead ECG. It should be noted, however, that the present high localization resolution was obtained in patients with normal ventricular anatomy. Because it has been reported that organic heart disease may cause abnormal activation patterns during ventricular pacing,\textsuperscript{7,8,10,14} it is important to establish the nature and extent of these changes on the total body surface ECG; the current data base can be used as a qualitative and quantitative reference frame. In addition to an improved electrocardiographic performance to localize ectopic events, use of a single integral map instead of 12 waveforms to specify a certain QRS morphology not only offers data reduction but also simplifies visual pattern recognition. Moreover, the use of a data base of maps holds the possibility of performing independent mathematical pattern comparison. Ultimately, the data base could be implemented in an intelligent system that not only records but also performs fast on-line interactive analysis in the coronary care unit and the electrophysiologic laboratory. The use of this system may be time saving during endocardial catheter mapping of ventricular tachycardia by directing the localization procedure to a precise endocardial area of interest.

Acknowledgments

The authors wish to thank Dr. A. Linnenbank, Dr. F.J.L. van Capelle, Dr. R. Coronel, and Dr. J.G.P. Tijssen for their valuable statistical advice and/or editorial suggestions and Mrs. S. Teengs for preparing the illustrations.

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Key Words • body surface mapping • QRS morphology • ventricular arrhythmias
Body surface mapping of ectopic left and right ventricular activation. QRS spectrum in patients without structural heart disease.
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