Noninvasive Identification of Initial Site of Abnormal Ventricular Activation by Least-square Phase Analysis of Radionuclide Cineangiograms

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SUMMARY  Least-square phase analysis (LSPA) of radionuclide cineangiograms demonstrates the sequence of onset of inward ventricular movement noninvasively. To validate the method and explore its ability to identify abnormal initial sites of ventricular activation, LSPA was applied to 14 patients with pacemakers (one with electrodes in two locations) (group 1) and three patients with recurrent ventricular tachycardia (VT) (group 2) who had undergone electrophysiologic endocardial mapping. The segment in which the site of initial ventricular activation was located was correctly identified in 13 of 15 paced studies and in two of three group 2 patients during VT. Pacing increased the duration of spread of onset of inward ventricular movement, and the duration of spread of onset correlated well with the duration of the QRS (r = 0.80). The sequence of onset of inward ventricular movement during VT was similar to the sequence of depolarization in all three group 2 patients. These preliminary results suggest that the sequence of onset of ventricular contraction as depicted by LSPA is a valid representation of the actual contraction sequence and that LSPA of radionuclide cineangiography correctly identifies abnormal sites of initial ventricular activation.

ELECTROCARDIOGRAPHICALLY synchronized radionuclide cineangiograms (RNCAs) of the heart are usually displayed in a cinematic presentation or as sets of end-diastolic (ED) and end-systolic (ES) images. However, various methods of generating functional images from RNCAs have been described. The production of such images by means of Fourier analysis of time-activity curves (TACs) of individual pixels was first described by Geffers et al.1 Each TAC was represented as the sum of sinusoidal functions of several frequencies. The amplitude and phase of the sinusoid representing the base frequency, i.e., the frequency corresponding to the average RR interval, were then used to generate two functional images that were taken to represent, respectively, the magnitude and sequence of the onset of regional wall motion.

Since the initial work of Geffers et al., other investigators have described methods of producing functional images representing the phase and amplitude of cardiac contraction. Some have been variations of the method of Geffers et al. and others have been original.2-4 We have described a method that fits the ejection and rapid filling portions of the TAC of each pixel with a sinusoidal function, a technique we refer to as least-square phase analysis (LSPA).2 To validate LSPA and explore its ability to identify abnormal sites of onset of ventricular activation, we applied it to RNCAs of patients with transvenous or epicardial pacemakers, as well as patients with recurrent ventricular tachycardia (VT).

Materials and Methods

Patients

Fourteen patients (group 1), 11 with programmable transvenous and three with epicardial pacemakers, were studied. Ten of these patients had undergone pacemaker implantation because of sick sinus syndrome and four for treatment of second- or third-degree atrioventricular block. All patients with an underlying normal sinus rhythm (NSR) had normal QRS complexes during that rhythm.

Three other patients were studied who had coronary artery disease and recurrent, hemodynamically stable VT (group 2). All three had undergone electrophysiologic mapping. All patients were studied after giving signed, informed consent in accordance with a protocol approved by the Human Investigation Committee of Rush-Presbyterian-St. Luke's Medical Center.

Radionuclide Cineangiography

RNCAs of patients in group 1 were obtained in a manner similar to that described by Green et al. Red blood cells were labeled in vivo with 25-35 mCi of technetium-99m. Equilibrium RNCAs were collected in the 30° right anterior oblique (RAO) and 45° modified (30° caudal tilt) left anterior oblique (LAO) projections with an Anger scintillation camera (LEM, Siemens Gammasonics, Inc.) interfaced to a dedicated minicomputer (Gamma-11, Digital Equipment Corporation). The camera was fitted with a high-
sensitivity, parallel-hole collimator with holes slanted at 30°. Using the acquisition software supplied by the manufacturer of the minicomputer, data were collected in 21–37 20-msec 64 × 64 frames at 3.4–5.7 × 10⁶ counts/frame. Patients in group 1 were studied during ventricular pacing. One patient was restudied with the pacemaker tip in a second location. The paced heart rate was selected for each patient so that it was just high enough to suppress capture by sinus or spontaneous ventricular complexes. (The range of paced rates thus selected was 72–115 beats/min.) Eight patients in group 1 were also studied during NSR. Thus, 23 studies (15 during pacing and eight during NSR) were performed on the 14 patients in group 1.

All three patients in group 2 were studied during NSR using the method described above. Two of the three also underwent RAO and LAO RNCAs by the same method while in hemodynamically stable VT. The third patient was studied in the LAO projection only while in bigeminy and trigeminy with couplets and short runs of VT. In this case, data were collected in the list mode, and cycles shorter than 500 msec (the coupling interval of the premature ventricular complexes) were reformatted into 20-msec frames.

**Ventricular Function**

Statistical errors in LSPA or Fourier phase analysis are relatively great when ventricular wall motion is poor. Furthermore, when correlating the duration of ventricular depolarization with the duration of onset of inward ventricular movement, we wished to exclude subjects who had severe regional contraction abnormalities. Therefore, a preliminary evaluation was made of the RNCAs to determine the state of regional and global ventricular contraction.

Left ventricular ejection fractions (LVEFs) were determined from the LAO views of the RNCAs by a modification of the method described by Parker et al.¹⁰ This modification has been validated in our laboratory against LVEFs determined by radiographic ventriculography. Regions of interest (ROIs) were drawn around the left ventricle at ED and ES with a manually controlled cursor. The operator was aided by guide marks drawn around the stroke volume (ED–ES) image with the cursor. A background ROI was drawn adjacent to the inferolateral quadrant of the LV ED ROI. The LVEF was then computed from the counts within the LV ROI at ED and the counts within the LV ROI at ES in the customary manner after subtraction of background counts normalized to the area of each ROI. The LVEF for each study was the mean of three determinations by the same operator.

LVEFs during pacing in the 14 group 1 patients ranged from 0.33 to 0.73. LVEFs were normal (≥ 0.53) in eight cases and ≥ 0.40 in 12. Among patients studied during NSR as well as pacing, LVEFs were not significantly different during the two rhythms (mean 0.54, both with and without pacing; greatest intrapatient difference was 0.04). The LVEFs of the patients in group 2 were 0.34, 0.53 and 0.38 during NSR and 0.23, 0.38 and 0.29, respectively, during VT (mean 0.42 in NSR, 0.30 in VT; p < 0.025).

The ventricular myocardium was divided into 10 segments (fig. 1). Since the diaphragmatic and posterobasal portions of the LV myocardium (not indicated in figure 1) are not visualized on the views in this experiment because of superimposition of the right ventricular (RV) and LV blood pools in the RAO projection, they were not considered. Cinematic displays of the RNCAs were inspected by an experienced observer who subjectively determined the degree of movement of each segment. Among the 14 patients in group 1, 10 had normal or nearly normal segmental ventricular contraction and four had one or more severely hypokinetic, akinetic or dyskinetic ventricular segments. In the patients in group 1 who were studied during NSR, no changes in the degree of regional wall motion were induced by pacing. In group 2, one patient had normal ventricular contraction during NSR, one had three hypokinetic ventricular segments and one had one hypokinetic ventricular segment.

**Determination of the Actual Pacemaker Sites and Lengths of the QRS**

The actual sites of the pacemaker electrodes and the length of the QRS complexes of patients in group 1 were determined by two independent observers who had no knowledge of the results of the phase analysis. The pacemaker sites were determined from posteroanterior and lateral radiographs of the chest. There was no interobserver variation. The lengths of QRS complexes were determined from standard electrocardiographic limb leads. When the two estimates of a given QRS length differed, the estimates were averaged. The durations of the QRS during both NSR and pacing were determined in the cases of patients studied during both rhythms.

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**Figure 1. Nomenclature of ventricular segments.** AB = anterobasal; AL = antero-lateral; AP = left ventricular apex; RV = right ventricle; BS = basal-septal; AS = apical-septal; IL = inferolateral; PL = posterolateral.
Endocardial Mapping

Endocardial mapping was performed as described by Josephson et al.\(^1\) Two quadriporal catheters were introduced percutaneously, one from the femoral vein and another from the femoral artery. The inter-electrode distance was 10 mm, and the two distal electrodes were used for mapping of the endocardial activation sequence. Intracardiac electrograms were simultaneously displayed with three surface ECG leads, and recordings were done at filter settings of 40–500 Hz on an Electronics for Medicine VR-12 oscillographic recorder. The paper speed was 100 mm/sec. All activation times were measured from the earliest onset of the QRS in any of the recorded ECG leads.

Phase Analysis

LSPA has been described.\(^7\) In brief, a cosine function (CF) was fitted to the ejection and rapid filling portions of the TAC of each pixel by a least-square technique. The parameters of the fit were the amplitude, the phase and the average value of the CF. The period was taken as 500 msec if the heart rate during collection of data was 120 beats/min or less, and was set equal to the period of the average cardiac cycle for faster heart rates. The phase of each fitted CF was taken to represent time of onset of inward movement of the portion of the myocardium that was located within the associated pixel at ED. The phase information was displayed as a series of isophase frames in a manner similar to that described by Verba et al.\(^8\) Each CF was divided into 16 or 25 equal segments, corresponding to the intervals represented by the frames of the LSPA display. Each frame was displayed as the ED image of the heart, with pixels shown in black if the CF corresponding to that pixel reached its maximal value during the interval represented by that frame. The result was a series of functional images in which onset of mechanical systole was represented as a wave of onset of inward movement that swept over the atria and ventricles (fig. 2).

We have shown that the statistical error (standard deviation) inherent in the determination of the phase of a TAC by means of LSPA is proportional to \(\sqrt{A_0/A_1}\), where \(A_0\) is the average value (i.e., the number of counts in a pixel averaged over the CF cycle) and \(A_1\) is the amplitude (Von Behren PL, Turner DA: unpublished data). Thus, calculated phases are subject to large statistical errors in regions of the image in which average values or relative amplitudes are low (e.g., extracardiac structures and akinetic myocardial segments). Display of the erroneous phase data in these regions is distracting and misleading. Thus, we used several methods to suppress such data in the final display. In the software used in the first part of this study, the phase of a pixel was not displayed unless it fell in the same frame as four or more adjacent pixels. Later, the TACs of four adjacent pixels were combined (i.e., the matrix was reduced to 32 \(\times\) 32) before processing, and the display of phase data was suppressed if the standard deviation of the computed phase was greater than 10–25% of the length of the isophase frame. In some cases, phase data were

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Portions of 25-frame least-square phase analysis displays (20 msec/frame) of a patient studied in normal sinus rhythm (UNPACED) and while paced from right ventricular (RV) apex and RV inflow segment (lower right panel). Arrows indicate sites of pacemaker electrodes. RAO and LAO = right and left anterior oblique projections.
not displayed outside an ROI drawn around the atria and ventricles.

Interpretation of the Phase Displays

Group 1

We hypothesized that during ventricular pacing, the onset of myocardial contraction, like electrical activation, begins at the tip of the pacing electrode and spreads over the myocardium slowly, whereas during NSR, the onset of contraction spreads throughout the myocardium more rapidly. We reasoned that if LSPA gives a valid representation of the sequence of onset of myocardial contraction, one should be able to distinguish between artificially paced and unpaced rhythms on the basis of the pattern of onset of contraction as represented by LSPA. Furthermore, one should be able to identify the ventricular segment in which the pacing electrode is located.

To test these hypotheses, the phase displays of the RNCAs of patients in group 1 were randomly ordered and then inspected by three independent observers who had no knowledge of the rhythm under which specific studies were collected or the sites of the pacemaker electrodes, except the knowledge that transvenous pacing electrodes are frequently located in the apex of the right ventricle. Each observer determined which studies were collected during pacing and which during NSR on the basis of the pattern of onset of contraction. They also indicated, in the case of studies that appeared to be paced, the segment in which contraction first began, using the schema shown in figure 1. The observers determined, for each study, the first and last frames in which onset of inward movement of either ventricle occurred, i.e., the first and last frames in which any pixel in either ventricular region was displayed in black. The interval between these two frames was taken as an estimate of the duration of spread of onset of inward movement of the ventricles. The duration of spread of onset of inward ventricular movement for a given study was then taken as the average of the estimates by the three observers.* The sites of initial inward ventricular movement during pacing were compared with the sites of the pacemaker electrodes as determined by radiography. The duration of spread of onset of inward ventricular movement was compared with the duration of the QRS complex in patients with normal or nearly normal ventricular contraction by means of linear regression analysis. Patients with severe segmental wall motion abnormalities were excluded from this comparison because it was deemed likely that the time of onset of inward movement of severely abnormal segments frequently would not correspond to the time of electrical activation.

Group 2

The studies of patients with recurrent VT were interpreted by one observer, who inspected the phase displays of the RNCAs collected during ventricular arrhythmia and determined the sequence of onset of inward ventricular movement by segment, using the schema shown in figure 1. The sequence so determined was then compared, in each case, with the sequence of depolarization as determined by endocardial mapping.

Statistical Methods

To determine the relationship between the length of the QRS and the duration of onset of ventricular contraction, linear regression analysis was performed and Pearson's correlation coefficient was computed. The significance of the difference between the mean LVEFs during NSR and VT was determined with the paired t test.

Results

Figure 2 shows representative images of the LSPA displays of three studies from a patient in group 1 who was examined while in NSR and during pacing from two locations in the right ventricle. Each frame represents 20 msec. The images in the two upper panels represent the phase analysis of RAO and LAT RNCAs during NSR. They are typical of studies of normal hearts performed during NSR. Regions beginning to contract in a given frame are represented in black in that frame. During NSR, contraction begins in several regions of both ventricles in the first 40 msec of movement (blackened areas, LAO frame 24, RAO frame 25) and rapidly spreads to all of both ventricles. Onset of contraction is completed within 80–100 msec. The lower panels are displays of the LSPA of RAO RNCAs of the same patient collected during pacing from two sites in the right ventricle. During the study shown in the left panel, the electrode was at the apex of the right ventricle, as indicated by the arrow in frame 23. Contraction begins at the RV apex and spreads proximally and to the left in a wave, over an interval of approximately 140 msec. During the study shown in the right panel, the electrode was in the RV inflow segment, as indicated by the arrow in frame 23. Contraction begins in that segment and spreads distally, proximally and to the left.

The rhythms during which the 23 RNCAs of group 1 patients were performed were correctly identified in every case by inspection of the LSPAs, with no interobserver variation. The segment in which the pacing electrode was located was correctly identified by inspection of the LSPA displays in 13 of 15 paced studies (87%), again without interobserver variation. In both of the cases in which errors were made, the radiographic location of the pacemaker electrode was the right ventricular apex, whereas the LSPA indicated earliest inward movement in the apical septal segment of the left ventricle. Although the reason for these errors is unknown, they might have occurred because of improper positioning of the LAO views with superimposition of the LV and RV apexes. Minor degrees of superimposition would have been difficult to detect by inspection of the images because the interventricular septum is a curved structure. An alternative explanation is that the pacemaker ele-

*Interobserver variability was small, 10.88 msec.
trodes, both of which were transvenous, may have perforated the interventricular septum and become seated in the apical septal segment.

The duration of spread of onset of inward ventricular movement correlated well with the duration of the QRS complex in the 10 patients who exhibited normal ventricular contraction or slight-to-moderate diffuse hypokinesis ($r = 0.80$) (fig. 3). Furthermore, pacing produced a marked increase in the duration of onset of inward movement in all patients who were also examined during NSR (fig. 4).

The sequence of onset of contraction during VT as depicted by LSPA was similar to the sequence of depolarization during VT determined by endocardial mapping in all three patients of group 2. The segment in which the arrhythmia originated could be identified by LSPA in two of these three patients. Figure 5 shows a 16-frame (30 msec/frame) display of the entire LSPA of the LAO view of a patient in group 2 (case 1) studied during hemodynamically stable VT. After a "quiet interval" during which no sustained onset of inward ventricular movement is seen (frames 6 and 7), contraction begins in the boundary between the basal septal and apical septal segments (LV midseptum), indicating that this is the site of origin of the arrhythmia. This was also the initial site of depolarization as determined by endocardial mapping.

Contraction then spreads to the LV apical septum, the RV apex, the RV inflow and outflow segments, and back to the left ventricle. Figure 6 is a comparison of the sequence of onset of inward movement as depicted by a 25-frame (18 msec/frame) display of the same LSPA with the sequence of depolarization as depicted by endocardial mapping. The sequences are the same, except for the two segments marked with asterisks.

The images shown in figure 7 are from the group 2 patient (case 2) in whom the segment of origin of VT could not be identified by LSPA. The study was performed during hemodynamically stable VT. The figure shows dyskinesis of the RV and LV apexes during VT; these segments contracted normally during NSR. Figure 7B shows a 16-frame (32 msec/frame) representation of the entire LSPA of the same RNCA. Contraction of the base of the left ventricle is
ONSET OF INWARD VENTRICULAR MOVEMENT
VERSUS
REGIONAL DEPOLARIZATION

FIGURE 6. Case 1, group 2 — sequence of onset of inward movement (from right and left anterior oblique 25-frame least-square phase analysis displays) is same as sequence of depolarization as determined by electrophysiologic study, except for segments marked with asterisk. Depolarization data were not available for all 10 segments. LV = left ventricular; RV = right ventricular.

well under way by frame 2, and onset of contraction has spread to the right ventricle by frame 3. The dyskinesis is manifested as a wave of late onset of inward movement that sweeps down the inferior lateral LV segment (frames 4–7), reaches the LV and RV apexes in midcycle (frames 8–12), moves up the interventricular septum during the last part of the cycle (arrow, frame 16; lower arrow, frame 1), and temporally overlaps the onset of contraction of the posterolateral segment (upper arrow, frame 1), which was the site of origin of the arrhythmia. Because there is no “quiet interval,” the segment in which the arrhythmia originates cannot be identified from the LSAP. Even the higher time resolution afforded by the 25-frame (20 msec/frame) display (fig. 7C) does not result in temporal separation of the dyskinetic wave (arrow, frame 25; lower arrow, frame 1) from the onset of contraction of the posterolateral segment (upper arrow, frame 1). Nonetheless, the sequence of onset of inward ventricular movement depicted by the LSAP was the same as the sequence of depolarization, except for the dyskinetic regions.

Discussion

The observations made in this and other experiments suggest that the sequence of contraction of normal myocardial segments parallels the sequence of myocardial depolarization.19-24 Thus, examination of the sequence of onset of inward movement of the ventricles by means of RNCA indirectly provides, invasively, information regarding the sequence of depolarization. Although this information is available from cinematic displays of RNCA, it is difficult to appreciate. Our data suggest that LSAP of RNCA provides a valid representation of the sequence of onset of inward ventricular movement in a format that makes it relatively easy for an observer to perceive. In this experiment, the segments in which sites of origin of abnormal ventricular activation were located were readily identified in 15 of 18 cases (83%). The sequence of the onset of contraction as demonstrated by LSAP paralleled the sequence of depolarization as demonstrated by endocardial mapping in all three patients in group 2. The validity of LSAP is further supported by the observations that pacing increased the duration of onset of inward movement and that the latter correlated well with the duration of the QRS in patients with relatively normal segmental wall motion.

Although these preliminary results suggest that LSAP of RNCA may provide a noninvasive method of determining the sites of origin of ventricular arrhythmias, certain limitations must be considered. Arrhythmia-induced wall motion abnormalities may obscure the source of VT, as in case 2 of group 2. Furthermore, sources near or within akinetic or severely hypokinetic segments may be difficult to detect. The statistical error inherent in the determination of the phase of a TAC is proportional to √A/A. Because A is small when contraction is poor, statistical errors in the determination of the phase of segments that move poorly will tend to be large. This might be a substantial limitation of phase analysis in the detection of sources of ventricular tachycardia, because such sources are frequently located in the vicinity of poorly contracting myocardium. Furthermore, VT itself seems to result in depressed global and regional myocardial contraction: The LVEFs of the patients in group 2 were significantly less during VT than during NSR, and there were concomitant regional decreases in A.
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(LVEF did not change with pacing, even at heart rates as great as 115 beats/min). Statistical errors resulting from poor segmental contraction can be reduced by increasing the number of counts collected. However, the number of counts must be increased by a factor of 4 in order to decrease the error by a factor of 2. Thus, count requirements become very high as A1/Ao falls below 0.1.

To ensure good statistical quality in this preliminary investigation, we collected as many as 2.1 × 10⁷ counts per RNCA, but this required collection periods of 10–15 minutes. Thus, we studied patients during VT only if they were hemodynamically stable while in VT or if they had intermittent, short runs of VT and unifocal premature complexes from the same focus of origin as the VT. However, it might be possible to apply LSPA of RNCA to patients who can tolerate only brief periods of VT, particularly if contraction is relatively normal. Friedman et al.18 studied patients who had severe ventricular asynergy and epicardial pacemakers. They used Fourier phase analysis of RNCA and detected the location of the pacemaker electrodes in two patients despite relatively short collection periods and collection of relatively few counts (2 × 10⁶ per RNCA). Data collection periods might be shortened further by adding data collected during serial first transit studies performed with radioisotopes that have ultrashort half lives.

As noted, statistical errors in the determination of phase are usually quite large over extracardiac structures and akinetic segments, because A1/Ao is relatively small in these regions. We suppressed the display of phase data from such areas because these data are distracting and misleading. Although suppression of statistically "noisy" phase data facilitates interpretation of the phase display, it must be done with caution. Excessive suppression will eliminate the display of valid information regarding ventricular contraction. We obtained satisfactory results by suppressing phase data if the standard deviation was greater than 10–25% of the frame length of the display. However, different thresholds for suppression might be appropriate in other applications. In the detection of tricuspid insufficiency, for example, Pavel et al.19 suppressed the phase display in regions where A1 is less than 7% of maximal amplitude.

We obtained both RAO and LAO views in all but one case. The preliminary data of Friedman et al.18 suggest that abnormal initial sites of ventricular activation are more readily detected when several views are obtained than when only one view is available. We are in agreement with this conclusion, although we have not systematically studied the problem. The onset of inward movement of a given myocardial segment is timed most reliably when that segment is viewed tangentially and without overlap of other regions of the cardiac blood pool. For example, the site of initial activation in patients with anterolateral epicardial pacemakers was most readily apparent on the RAO view in our series, whereas the posterolateral segment could be seen only on the LAO view. The addition of a left posterior oblique view would have facilitated evaluation of the diaphragmatic and posterobasal regions of the myocardium.

In conclusion, preliminary experience with LSPA of RNCA suggests that it provides a valid representation of the sequence of onset of inward ventricular movement. One can detect sites of origin of ventricular arrhythmias with this method. More experience is necessary to determine the frequency with which identification of sites of origin of VT will be frustrated by preexisting or arrhythmia-induced wall motion abnormalities.

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Should a Fixed External Reference System Be Used to Analyze Left Ventricular Wall Motion?

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SUMMARY To investigate whether a fixed external reference system should be used to most accurately describe regional left ventricular wall motion, we used vectors to analyze a simple model of ventricular contraction. If measured in a fixed external reference system, motion of implanted radiopaque midwall markers may contain translational as well as contractile components. Therefore, comparisons of different reference systems that use marker motion measured in a fixed reference system as a standard will be biased unless proper corrections are included. We conclude that evidence to date does not indicate that the use of a fixed reference system is superior to other methods for analyzing regional ventricular wall motion.

THE USE of contrast ventriculography to quantify left ventricular segmental wall motion has resulted in the development of methods to measure regional endocardial movement. As the methods have proliferated, the accuracy of these alternative approaches in separating abnormal from normal wall motion has been compared. These comparisons are difficult to evaluate for the same reasons that have led to the development of multiple methods of analysis: (1) By examining a two-dimensional silhouette of the three-dimensional ventricle, one cannot determine with certainty which segments exhibit truly abnormal motion and to what extent the motion is abnormal. (2) There is a choice between external and internal reference systems. (3) The ventricle has multiple landmarks that may be used to superimpose a reference grid system. (4) There are choices between measurements (segmental area, hemisaxial lengths, and radial lengths), the number of radii, chords or regions to be measured, and the frequency of the measurement (end-diastole and end-systole or frame-by-frame).

Ingels and co-workers reported that left ventricular midwall dynamics were measured most accurately in the 30° right anterior oblique plane by a fixed external reference system using polar co-

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