Three-Dimensional Electrophysiological Imaging of the Intact Canine Left Ventricle Using a Noncontact Multielectrode Cavitary Probe: Study of Sinus, Paced, and Spontaneous Premature Beats

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Background—The feasibility of measuring cavitary electrograms using a noncontact probe and reconstructing endocardial surface electrograms and activation sequences during paced beats was previously demonstrated in the isolated canine left ventricle (LV). The objective of the present study was to develop and test a high-resolution, three-dimensional, endocardial electrophysiological imaging technique that simultaneously reconstructs endocardial surface electrograms and their corresponding activation sequences during normal and abnormal beats with the use of cavitary electrograms measured with a noncontact multielectrode probe in the intact canine LV.

Methods and Results—A 128-electrode probe was inserted into the intact canine LV. Probe unipolar electrograms were simultaneously acquired during sinus, artificially paced, and spontaneous premature beats. Representative endocardial electrograms were measured directly using eight needle electrodes (the “gold standard”). A probe-cavity realistic, three-dimensional geometric model was constructed using two-dimensional epicardial echocardiography. Boundary element methods and numeric regularization were used to compute electrograms at 194 sites on the endocardium. In eight pacing protocols, computed endocardial electrograms correlated well with directly measured electrograms ($r = 0.88$). Corresponding activation times were also in agreement with those determined from measured endocardial electrograms (activation error, 4.7 ms). The earliest region of activation was invariably in the vicinity of the pacing needle (spatial error, 9.2 mm). Subsequently, the site of origin of ischemia-induced spontaneous ventricular premature beats and the ensuing sequence of depolarization was identified.

Conclusions—Noncontact mapping provides realistic, three-dimensional electrophysiological images of the endocardium, on a beat-by-beat basis, that localize the sites of origin of premature beats and reconstruct their activation sequences. (Circulation. 1998;97:399-409.)

Key Words: electrophysiology endocardium mapping

In the United States, ≈300 000 patients die annually from sudden cardiac death, primarily due to cardiac arrhythmias (ventricular tachycardia and ventricular fibrillation). Current antiarrhythmic pharmacological therapy often is ineffective and can be proarrhythmic.1,2 With an increased emphasis on nonpharmacological therapy, catheter ablation has been routinely used as a safe and effective first-line therapy for managing supraventricular arrhythmias.3 However, for ablation to be clinically successful in managing ventricular arrhythmias, catheter mapping of brief, hemodynamically unstable, or polymorphic ventricular arrhythmias on a beat-by-beat basis and at multiple simultaneous sites is required.4 Therefore, the selection of appropriate pharmacological therapies and the advancement of catheter ablation techniques for managing ventricular arrhythmias are contingent on the development of advanced mapping techniques that enable identification of the mechanisms of these arrhythmias and localization of their sites of origin.

In patients with ischemic heart disease, the earliest site of activation during monomorphic ventricular tachycardia has often been located on the endocardial surface of the left ventricle (LV).5 Furthermore, because the endocardium is more safely accessible than the epicardium, most electrophysiological mapping techniques have focused on endocardial approaches. However, current techniques of mapping the endocardium have certain limitations. Traditional electrode-catheter mapping performed during electrophysiology procedures is confined to a limited number of recording sites, is time consuming, and is carried out over several heartbeats without accounting for possible beat-to-beat variability in activation.6 On the other hand, although recently introduced multielectrode basket-catheters7,8 measure endocardial electrograms at multiple sites simultaneously by expanding the basket inside the heart so the electrodes are in direct contact with the endocardium, the system is limited to a fixed number of recording sites, may require multiple deployments for record-
ing additional sites, and may result in complications such as irritation of the myocardium and difficulty in collapsing and withdrawing the basket.

Earlier, an alternative mapping approach was introduced that used a cavitary noncontact multielectrode catheter-probe. The probe measures cavitary electrical potentials (electrograms) from multiple directions simultaneously and can be easily inserted into the blood-filled cavity without occluding it. Unlike the basket, however, the probe is not necessarily in direct contact with the surface of the endocardium. Theoretical and experimental models have shown that noncontact sensing by the probe results in low-amplitude and smoothed-out electrical potential patterns. Recently, endocardial surface potentials and activation sequences have been mathematically computed, during selected intervals, from measured cavitary probe electrograms (the “inverse problem”) in the isolated, Langendorff-perfused canine LV. The computed electrograms successfully identified the sites of origin of artificially paced ectopic foci and further demonstrated the potential clinical applicability of the technique for routine electrophysiology procedures without the use of a basket-catheter or the need for surgery. However, this initial study was limited to controlled paced rhythms, the probe-cavity geometric model was approximated manually after the completion of the experiment, and the number of sampling points on the endocardium were limited to the same number of probe electrodes.

The objectives of the present study were (1) to develop and test mathematical and experimental methods to compute simultaneous endocardial surface electrograms and activation sequences at multiple sites, on a beat-by-beat basis, from cavitary electrograms measured with a noncontact multielectrode probe in the intact canine LV, and (2) to use epicardial echocardiography in situ to construct high-resolution, realistic, three-dimensional electrophysiological images of the LV during sinus as well as artificially paced control rhythms and, for the first time, during ischemia-induced spontaneous premature depolarization.

Methods

Mathematical Formulation

The electrical potential within the blood cavity, bounded by the probe surface on one side and the endocardial surface on the other, is described mathematically by Laplace’s equation. To solve this equation in a realistic probe-cavity geometry, a standard boundary element method was used. The resulting equation is $V_{P} = A^{-1}V_{E}$, where $V_{P}$ and $V_{E}$ are values of potentials at $N_{P}$ and $N_{E}$ sampling points (nodes) on the probe and endocardial surfaces, respectively, and $A$ is a matrix (of order $N_{P} \times N_{E}$) that describes the geometric relation between the endocardial surface and the probe surface (including position and orientation within the cavity). The problem of computing the endocardial potentials in the above equation, for a given set of measured probe potentials, is ill posed; that is, small variations in the data $V_{P}$ caused by measurement noise or errors in determining the geometry result in an unphysical solution $V_{E}$. Similar to previous methods, a physical solution was obtained by using the Tikhonov zero-order numeric regularization technique in conjunction with the composite residual and smoothing operator for selecting the regularization parameter.

Experimental Model

A 25-kg mongrel dog was anesthetized with sodium pentobarbital (30 mg/kg). The dog was intubated and ventilated with room air with the use of an external respirator. The heart was exposed through a median
sternotomy and suspended in a pericardial cradle. The study was conducted in accordance with institutional guidelines.

Electrogram Potential Measurement

A custom-made cylindrical probe, containing 128 silver electrodes on its surface arranged in 16 circumferential rings (8 electrodes per ring), was used to measure LV cavitary electrograms. The probe was 9 mm in diameter, and the distance between the proximal and distal rings was 60 mm (Fig 1A). To minimize motion artifacts, a 5-mm-long needle was fixed at the tapered tip of the probe. Guided by epicardial echocardiography, the probe was inserted through a purse-string suture in the LV apex and positioned along the center of the cavity, with the tip anchored between the mitral and aortic valves (Fig 1B). The 4 proximal rings of electrodes were not completely inside the cavity and were not used. This resulted in an effective probe of 96 electrodes. To prevent rotation artifacts, the extracardiac portion of the probe shaft was affixed to the rib spreader.

Endocardial electrograms were directly measured from eight representative sites through the use of needle electrodes (Fig 1B). The needles were 15 mm long and contained 5 electrodes (2-mm interelectrode separation starting from the tip). The tip electrode protruded slightly into the cavity and was used to measure endocardial electrograms. Four electrodes along the needle shaft were used for pacing. Measured endocardial electrograms were used to evaluate the accuracy of computed endocardial electrograms.

Geometry Measurement

Probe-cavity geometry was determined using two-dimensional epicardial echocardiography. Similar to previous work, a 5-MHz echocardiography transducer (model SONOS 1000; Hewlett-Packard) was hand-held perpendicular against an external marker that was in turn attached parallel to the shaft of the probe (Fig 2). The needle electrodes were 15 mm long and contained 5 electrodes (2-mm interelectrode separation starting from the tip). The tip electrode protruded slightly into the cavity and was used to measure endocardial electrograms. Four electrodes along the needle shaft were used for pacing. Measured endocardial electrograms were used to evaluate the accuracy of computed endocardial electrograms.

Electrophysiology Protocols

Cavitary and endocardial electrograms were initially recorded during baseline sinus rhythm. Electrograms were then recorded during LV pacing at 8 subendocardial sites using needle electrodes (base, 2 sites; midway, 2 sites; and apex, 4 sites). Bipolar pacing was applied using 4-ms pulses at twice-diastolic threshold at a cycle length of 300 ms with an external stimulator (model S8800; Astro-Med).

Once all pacing protocols had been completed, the left anterior descending coronary artery was ligated at a point just above the first diagonal branch near the base of the heart. Approximately 15 minutes after occlusion, spontaneous ventricular premature depolarizations were observed in the surface ECG, and for the first time, representative cavitary and endocardial electrograms were simultaneously recorded. The dog subsequently developed ventricular fibrillation.
Results

Artificially Paced Ventricular Rhythms

Endocardial electrograms were computed at all 194 nodes using cavitary electrograms obtained while pacing at 8 different needle sites. Representative computed electrograms are shown in Fig 5 (thick tracings) while pacing at site 7. Electrograms measured simultaneously through the 8 needle electrodes while pacing at the same apical site are also shown in Fig 5 (thin tracings). As noted from these representative tracings, there was an excellent agreement between computed and measured endocardial electrograms, with a correlation coefficient of .86 to .98. There also was a high correlation between activation times determined from computed electrograms and activation times determined from measured endocardial electrograms. Note that site 7 reflected the earliest activation time compared with all other sites. Errors in activation time ranged from 0 to 8 ms in Fig 5.

Results of all 8 pacing protocols are summarized in Table 1. Compared with 8 measured endocardial electrograms, computed electrograms resulted in an overall correlation coefficient of .88 and error in activation time of 4.7 ms.

Isopotential contour maps of endocardial potentials computed early after pacing are shown in Fig 6A through 6D. The maps are shown for the 4 apical pacing sites, needles 5 through 8 (Fig 5). The separation between the needles is 8.8, 10.2, and 20.1 mm, respectively. Primary potential minima appeared at 5 ms in Fig 6A, 10 ms in Fig 6B, 12 ms in Fig 6C, and 19 ms in Fig 6D. Note that a primary potential minimum (depolarization) consistently initiated in the vicinity of the pacing needle, so four unique sites of origin could be identified from the isopotential maps.

Histological sections of the LV in the apical pacing regions showing fiber direction are provided in Fig 6E through 6H for the 4 apical pacing regions. The alignment of the region of peak positive potential (maximum) with respect to the primary potential minimum in the isopotential maps in Fig 6A through Fig 6C agreed qualitatively with the endocardial fiber direction in Fig 6E through Fig 6G, respectively. The appearance of the primary potential minimum in Fig 6D at a later time and the alignment of the maximum and minimum potentials compared with the direction of underlying endocardial fibers in Fig 6H suggest that pacing might have been applied at a deeper myocardial site.

Maps of isochrones (activation maps) determined from computed electrograms are shown in Fig 7. The maps correspond to the same four apical pacing protocols of Fig 6 (needles 5 through 8). The pacing needle site was always the region of earliest activation. Earliest activation appeared at 6 ms in Fig 7A, 13 ms in Fig 7B, 14 ms in Fig 7C, and 22 ms in Fig 7D.

In all pacing protocols, earliest activation appeared at an average of 16.5 ms from pacing stimulus. The distance between the calculated site of earliest activation and the needle site (ie, spatial error) was 9.8 mm in Fig 7A, 8.2 mm in Fig 7B, 0 mm in Fig 7C, and 6.6 mm in Fig 7D. Average error, for all pacing protocols, in determining the site of origin of the pacing stimulus was 9.2 mm, as summarized in Table 1.

Spontaneous Ventricular Premature Depolarization Versus Sinus Rhythm

For the first time, cavitary probe electrograms were recorded simultaneously during ischemia-induced spontaneous ventricular premature depolarizations (5 nonconsecutive beats). Sur-
face ECGs (leads I, II, and III) during sinus rhythm interrupted by a ventricular premature depolarization are shown in Fig 8.

Endocardial electrograms were initially computed during 5 sinus rhythm beats preceding 5 ventricular premature depolarizations. Compared with endocardial electrograms measured at the same 8 sites in Fig 5, electrograms computed during sinus rhythm resulted in an average correlation coefficient of .86 and an average error in activation time of 4.9 ms (Fig 9A). Electrograms computed during ventricular premature depolarization resulted in an average correlation coefficient of .81 and an average error in activation time of 4.5 ms (Fig 9B). Earliest activation was clearly at site 1 in Fig 9B. Furthermore, electrogram fractionation and change in morphology were observed at sites 1, 2, and 3, which is characteristic of ischemia-related conduction.

Isopotential contour maps of endocardial potentials computed during sinus rhythm are shown in Fig 10. A primary potential minimum (potential $\leq -5$ mV) appeared initially in an area on the posterior paraseptal wall in the lower half of the LV (Fig 10A). Within 4 ms, another primary potential minimum (potential $< -5$ mV) appeared in the anterior apex extending toward the septum in the anterior paraseptal area (Fig 10B). Depolarization wave fronts spread rapidly from these two areas, as they merged after 6 ms from the onset of the initial primary minimum. Most of the LV endocardium was depolarized within 11 ms, except for a posterobasal area, an anterolateral area, and an anteropapical area (Fig 10C). The last part to depolarize on the endocardium was the posterobasal area, 20 ms from the initial depolarization.

Unlike sinus rhythm, the initial primary potential minimum (potential $< -5$ mV) appeared during spontaneous ventricular premature depolarization in high anteroseptal region just below the mitral valve (Fig 11A). The depolarization wave front propagated downward along the septum and throughout

<table>
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<td>Mean</td>
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TABLE 1. Computed Endocardial Electrograms With Realistic Geometry

Error indicates spatial separation between earliest activation site determined from computed electrograms and actual site of pacing needle; r, average linear correlation coefficient between computed and measured endocardial electrograms at eight sites during one cardiac cycle (100 ms); Tc–Tm, average absolute error in activation times determined from computed and from measured endocardial electrograms at eight sites.
Figure 6. A through D, Three-dimensional isopotential contour maps constructed from computed electrograms at 194 left ventricular nodes during apical pacing at sites 5 through 8, respectively, and plotted at 5, 10, 12, and 19 ms, respectively, from the beginning of the stimulus. The pacing site is indicated by ×. Locations of the potential minimum and maximum are indicated by − and +, respectively. Dashed line is a relative indication of orientation between both extrema. All maps are plotted at the same scale. Anterior left ventricle is displayed as viewed from the epicardial side. E through H, Histological sections from epicardium to endocardium obtained from myocardial blocks in the 4 pacing regions are shown below each map as viewed from the epicardium.

Figure 7. Three-dimensional isochrone maps constructed from computed electrograms at 194 nodes. The maps correspond to the same apical pacing protocols in Fig 6. The pacing site is indicated by ×. The maps are plotted at the same scale.
the anterior and posterior LV as shown in Fig 11B after 25 ms. Most of the LV endocardium was depolarized 40 ms after the initial depolarization and completed after 55 ms. The alignment of the initial maximum and minimum potentials was similar to the direction of fibers between epicardial and midwall layers depicted in Fig 11C.

Role of Geometry

Using the same cavity electrograms, endocardial electrograms were computed assuming an idealized probe-cavity geometric model (Fig 4). Fig 12 shows representative computed electrograms that correspond to the same pacing protocol in Fig 5 (pacing at site 7). Computed electrograms were degraded, and site 5 incorrectly reflected an earlier activation time compared with the actual pacing site. Results of pacing are summarized in Table 2. Compared with measured electrograms, computed endocardial electrograms resulted in an overall correlation coefficient of .75 and an error in activation time of 9.9 ms. Compared with the realistic geometry, the average error for all pacing protocols in determining the site of origin of the pacing stimulus was 17.0 mm.

Discussion

New methods were developed and tested in the present study, and new findings were achieved beyond that accomplished in previous reports. In the present study, a noncontact multielectrode probe was used to measure cavity electrograms from multiple directions in the blood-filled canine LV. A three-dimensional model of the probe-cavity geometry was determined in situ in the beating heart using two-dimensional epicardial echocardiography. These cavity electrogram and geometry data were then used to mathematically compute the corresponding electrograms on a high-resolution, three-dimensional endocardial surface. Similar to previous studies, the methods were tested during artificially paced control rhythms and further extended, for the first time, to construct electrophysiological images during sinus rhythm and spontaneous ventricular premature depolarization.

The present study confirmed that LV wall pacing consistently gave rise to a potential minimum during early activation that was invariably in the vicinity of the pacing electrode. Paced rhythms served as controls for subsequent analysis of intrinsic sinus and spontaneous premature beats. During pacing, electrograms computed on the endocardial surface were in excellent agreement with electrograms measured directly at selected sites (8 sites) on the endocardium ($r=.88$). Furthermore, activation times determined from computed electrograms correlated well with those determined from measured electrograms (average error, 4.7 ms). Earliest region of activation in isochrone maps, determined from computed electrograms, envelope the pacing site, with the earliest site of activation always in close proximity to the pacing site (average spatial error, 9.2 mm). It should be noted that these discrepancies were determined assuming that recordings obtained with the endocardial needle electrodes represented the gold standard, ignoring possible measurement or spatial errors in the needles.

The spatial distribution of reconstructed endocardial potentials (position and orientation of initial maxima and minima) reflected underlying cardiac fiber direction. This result was consistent with previous studies identifying the effect of myocardial anisotropy (fiber direction) on propagation of excitation wave fronts and on the ensuing potential distribution. Due to anisotropy, ectopic excitation spread faster in a direction parallel to cardiac fibers than perpendicular to them. As a result, this generated potential maxima in the areas toward which excitation propagated along fibers, thereby reflecting fiber direction at the level of the ectopic event within the myocardium.

For the first time, endocardial electrograms were computed in the present study during sinus rhythm. Similar to artificially paced ventricular rhythms, electrograms computed during intrinsic sinus rhythm correlated well with endocardial electrograms measured at 8 selected sites. More importantly, the reconstructed sequence of depolarization during sinus rhythm was consistent with published reports on the overall pattern of LV activation. Detailed mapping studies by Durrer et al on isolated human hearts showed that endocardial areas that activate early include the posterior paraseptal area at about one third the distance from apex to base and the anterior paraseptal area toward the apex. Furthermore, areas of early activation become confluent at 15 to 20 ms, and the latest part to be activated is the posterobasal area. This apparent initial scattering of the wave fronts at different sites, before merging with one another, was similarly described by Arisi et al by recording potential fields on the ventricular epicardial surface of the exposed canine heart during sinus rhythm. The depolarization at different sites was perhaps a reflection of the underlying bifascicular nature of the left bundle branch of the Purkinje system.

Recently, analysis of cavity electrograms was conducted before and after experimental myocardial infarction produced by occlusion of the left anterior descending coronary artery. More recently, the use of an electrocardiographic inverse solution, coupled with body surface potential mapping, was demonstrated in localizing acute ischemia in patients undergoing percutaneous transluminal coronary angioplasty. For the first time, endocardial surface electrograms were computed in the present study during ischemia-induced spontaneous ventricular premature depolarization. The objective of the study was not to fully describe the mechanism of arrhythmogenesis or underlying tissue electrophysiology but rather to substantiate the quality of the computed potential patterns and sequence of depolarization during spontaneous premature depolarization. Computed endocardial electrograms were in excellent agreement with measured electrograms. Furthermore, the site
of origin of the initial primary potential minimum and the subsequent sequence of depolarization were distinctly different from those computed in the preceding sinus rhythm cycle. Consistent with previous reports,29,30 this site of origin was in the vicinity of the area of occlusion of the coronary artery. The LV endocardium required a longer time to completely depolarize compared with sinus rhythm. Unlike sinus rhythm,
which is initiated by the rapid conduction system, activation initiated by spontaneous ventricular premature depolarization may have originated from an epicardial site. This was corroborated by the similarity between the alignment of the initial maximum and minimum potentials and the direction of fibers in the epicardial/midwall region. The effect of myocardial anisotropy on spontaneous premature depolarization was also consistent with results of artificial pacing.

As described in this as well as previous reports, computation of endocardial surface potentials requires a knowledge of both cavity probe potentials and probe-cavity geometry. Geometry is a requisite to obtain the transfer relation between the probe and endocardial surfaces (matrix A). In the present study, the volume conductor geometric model, including probe position and orientation, were determined in situ, for the first time, using epicardial echocardiography. Unlike previous work, the endocardial surface was sampled (discretized) at twice the number of probe nodes (electrodes). This approach therefore enabled us to construct electrophysiological images at higher spatial resolution. Epicardial echocardiography used in the present study provided an improved and more accurate approach in determining the geometry compared with previous work. Yet, to be applicable for use in routine catheterization procedures, other less invasive real-time methods for determining the geometry will have to be implemented.

Endocardial electrograms were in excellent agreement with directly measured electrograms while using a realistic probe-cavity geometry. Simplifying the geometry to an ellipsoidal endocardium and a concentric probe reduced the correlation between computed and measured electrograms and doubled the discrepancy in activation time. These results confirm the importance of obtaining detailed representation of the probe-cavity geometry when computing endocardial electrograms. Furthermore, idealization of the geometry almost doubled spatial error in localizing the site of origin of paced beats. This can lead to misinterpreting features of underlying activation pattern. Therefore, in situ determination of the geometry is paramount to successful application of the noncontact mapping technique in routine clinical catheterization procedures.

**Study Limitations**

Our study has limitations. First, the probe used in the present study was not yet suited for percutaneous insertion. However, this prototype design provided us with new data that permitted us to further test and advance our imaging methods. Second, global validation of activation patterns with measurements obtained from an extensive array of contact endocardial electrograms was not performed. This was limited in the present study to qualitative correlation with underlying fiber direction. Third, the ability to resolve two simultaneous adjacent ectopic events was not tested in the present study. Fourth, endocardial electrograms were computed assuming a single probe-cavity geometry (ie, end diastole), which was determined at the beginning of the experiment, without
accounting for possible variability throughout the experiment. This was feasible as long as the electrical event (i.e., depolarization) preceded the mechanical event (i.e., contraction). Fifth, endocardial electrograms were computed using a fundamental numeric regularization method, namely, Tikhonov zero-order, and the electrical potentials at each time sample were treated independently (quasistatic) using a single regularization parameter throughout the entire image. However, the use of temporal and spatial numeric methods has recently been demonstrated to be advantageous.33–35

Study Significance
The study used a multielectrode cavitary probe that can be easily miniaturized into a catheter-based probe, allowing for percutaneous insertion into the blood cavity in a way that is similar to electrode-catheters used in routine electrophysiology studies. This noncontact mapping approach reconstructs surface electrograms and activation sequences from measured cavitary probe electrograms, providing high-resolution, three-dimensional isopotential and isochrone maps. This mapping technique is carried out over a single cardiac cycle. Therefore, mapping can be conducted on a beat-by-beat basis, allowing for the study of brief, rare, or even hemodynamically compromising rhythm disorders that are difficult to evaluate with existing techniques. Moreover, with the advent of catheter ablation, the noncontact mapping approach can be preeminent in advancing ablation for managing heart rhythm disorders by localizing arrhythmogenic sites and directing ablation without the need for surgery.

Conclusions
We further extended our mathematical and experimental methods to compute endocardial surface electrograms and activation sequences at multiple simultaneous sites using electrograms measured with a custom-made, cavitary, noncontact, multielectrode probe. While measuring the probe-cavity geometric model in situ, we were able to successfully construct high-resolution three-dimensional electrophysiologic images of the intact canine LV endocardium during sinus, artificially paced, and spontaneous premature beats. Further testing of the methods using a miniaturized, catheter-based system is warranted.
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