Activation Sequence of Ventricular Tachycardia: Endocardial and Epicardial Mapping Studies in the Human Ventricle

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Thirty-five patients with ischemic heart disease and ventricular arrhythmias underwent intraoperative activation mapping at the time of coronary artery bypass surgery. During ventricular tachycardia, the sequence of activation in the intact ventricle was recorded simultaneously from 110 endocardial or 110 epicardial sites, or both. A balloon array of electrodes, inserted across the mitral valve, was used to obtain endocardial recordings in the left ventricle, and this appeared to facilitate the induction of ventricular tachycardia. Of 61 episodes of tachycardia, 16 (15 patients) were recorded with the epicardial sock and 45 (20 patients) with the additional use of the endocardial balloon. The sequence of activation during tachycardia was observed to conform to one of four configurations: monoregional spread was the most common activation sequence recorded on both the endocardium and epicardium, while biregional activation and figure eight sequences were recorded exclusively on the epicardium and endocardium, respectively. The fourth sequence was a circular spread of activation observed on both surfaces. Continuous activation throughout the tachycardia cycle length was an infrequent finding.

Simultaneous recordings of endocardial and epicardial activation were obtained in 45% of episodes. The sequence of activation recorded on one surface was matched by a similar sequence on the remaining surface in less than half of these. The onset of endocardial activation preceded that of the epicardium in >90% of tachycardia episodes, and the duration of left ventricular endocardial excitation often exceeded that recorded epicardially over both ventricles. The epicardium, however, did appear to be an important determinant of surface electrocardiographic configuration.

Methods

Study patients. Thirty-five patients (33 men, 2 women; mean age 60 years) with ischemic heart disease and ventricular arrhythmias consented to intraoperative mapping. Most of the patients had severe triple vessel disease, and all but three required coronary artery bypass surgery for control of ischemic symptoms. Thirty-three had had one or more previous myocardial infarcts (anterior in 25, inferior in 11), and a left ventricular aneurysm was present in 24. The mean ejection fraction and left ventricular end-diastolic pressure for the group were 29% (range 11 to 51) and 20 mm Hg (range 7 to 36), respectively. In addition, all had experienced repeated episodes of ventricular tachycardia with an average of two syncopal episodes per patient. All had required long-term antiarrhythmic therapy.
Preoperative electrophysiologic studies were performed to confirm inducibility of stable, monomorphic ventricular tachycardia. These studies were conducted in a standardized manner, using programmed stimulation from the right or left ventricular apex, or both. After a basic train of eight stimuli (cycle lengths 600 and 400 ms), up to four programmed premature stimuli were used to induce ventricular tachycardia. The stimuli used were 2 ms in duration and twice the diastolic threshold strength.

Intraoperative mapping technique. The mapping system (1) enables endocardial and epicardial activation to be recorded simultaneously in a unipolar mode, using an array of electrodes attached to a balloon and sock, respectively. To record endocardial activation, 110 silver bead electrodes 2 mm in diameter are fixed at 1 cm intervals in a standardized manner onto a mesh-covered balloon (Fig. 1B). Similarly, epicardial activation is recorded using 110 button electrodes consisting of a 1.5 mm diameter silver pin set in a Teflon disk 8 mm wide and positioned over both ventricles in the same manner on a nylon mesh sock (Fig. 1A). More recently, the electrode array has been simplified to consist of 112 electrodes arranged in 14 equal rows. Multistranded, insulated, stainless steel wire connects the two sets of electrodes to a multiplexer unit that supplies the input signals to the recording system.

Recording system and signal analysis. The recording system provides an on-line analysis and video display of activation in the following manner: the local electrograms are differentiated, and the maximal negative rate of voltage change (dV/dt) used to generate an intensity signal that brightens at the time of local activation. All the intensity signals are then assembled into a single video format signal that can be displayed on a raster scan monitor in the operating room. The resulting image represents the electrode array and consists of a matrix of dots that brighten at the time of local activation, thereby allowing the sequence of activation to be determined at the time of operation.

A second component of the recording system stores the analog signals in the form of unipolar electrograms that can be replayed postoperatively on a Mingograf ink writer to provide an off-line record of the electrograms and the time code. Local activation time, defined as the steepest part of the negative deflection from the isoelectric line, is measured from a common time reference provided by the time code. These local activation times are then used to generate isochrone maps of endocardial and epicardial activation at 12 ms intervals. The earliest endocardial or epicardial activation time of the 220 sites recorded during ventricular tachycardia is considered the “site of onset” of tachycardia. At a paper speed of 250 mm/s, the measurements are accurate to 1 mm or an activation time of 4 ms. Seven surface electrocardiographic (ECG) leads—three limb leads, three augmented unipolar leads and one chest lead—are displayed and recorded at the time of operation. The surface ECG is stored with the accompanying time code and can be replayed.

Mapping procedure. At the time of surgery, the patient is placed on normothermic cardiopulmonary bypass. An electrode for ventricular stimulation is sutured over the right or left ventricle. The sock array of electrodes is then pulled over the heart and positioned such that the first row of electrodes is aligned along the left anterior descending coronary artery. The electrode array has been simplified to consist of 112 electrodes arranged in 14 equal rows. Multistranded, insulated, stainless steel wire connects the two sets of electrodes to a multiplexer unit that supplies the input signals to the recording system.

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approximates the course of the left anterior descending artery. Stable electrograms are obtained by inflating the balloon with dextrose solution. Approximately 60 ml of solution is required, and the pressure within the balloon is monitored throughout to prevent subendocardial ischemia. Intrinsic balloon pressure is determined for incremental volumes of solution before insertion. When the balloon is positioned and inflated in the left ventricle, pressure is again recorded. Left ventricular end-diastolic pressure equals recorded pressure in the ventricle minus intrinsic balloon pressure at the same volume. This is maintained at 15 to 20 mm Hg to reduce the likelihood of compromising subendocardial blood flow. Using this transmural approach, it is possible not only to record endocardial activation with the ventricle intact, but also to record endocardial and epicardial activation simultaneously. With the balloon and sock in position, programmed stimulation is then performed, and the protocol follows that found to be successful in inducing ventricular tachycardia at the preoperative electrophysiological study.

Results

Initiation of ventricular tachycardia. At the time of operation, it was possible to initiate and map ventricular tachycardia in 100% of the patients. Because the mapping system was originally developed for recording epicardial activation and the endocardial balloon has been a more recent development, the first 15 patients underwent epicardial mapping only. In most of these patients, this was followed by a ventriculotomy and sequential endocardial mapping using a roving electrode. Only the epicardial data on these patients will be discussed. Before the ventriculotomy, a total of 16 episodes of monoform ventricular tachycardia was initiated in the 15 patients, with a single configuration being induced in all but 1 patient. On two occasions we were unable to determine the sequence of activation during tachycardia because of technical difficulties, and obtained only partial isochronal maps. After the ventriculotomy, it was possible to reinstitute tachycardia in only 50% of the patients. By contrast, we were able to induce and record 45 episodes of stable monoform ventricular tachycardia in the 20 patients in whom the endocardial balloon was used, with as many as five configurations being recorded in 1 patient. Epicardial activation was recorded in 12 of these patients. In four patients it was not possible to record epicardial activation, either because of dense epicardial scarring and adhesions or because of the large size of the ventricular aneurysm.

Patterns of Activation

Four predominant patterns of activation were observed (Table 1):

**Monoregional activation (Fig. 2A and B).** This term was used to describe tachycardia that had a focal site of earliest activation from which there was sequential, radial spread over the epicardial and endocardial surfaces. The site of onset of tachycardia was localized to a discrete cluster of electrodes over a limited interelectrode distance. During all of these monoregional sequences of tachycardia, there were isoelectric periods of variable duration, and we were unable to demonstrate continuous activity throughout the tachycardia cycle length. Monoregional spread was the most frequently observed sequence of activation on both the endocardium and epicardium.

**Biregional activation (Fig. 3 and 4A and B).** This term described a tachycardia with two discrete, but not necessarily simultaneous, sites of breakthrough of activation during ventricular tachycardia. This pattern was observed

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>Endocardium</th>
<th>Epicardium</th>
<th>Both</th>
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<td>18</td>
<td>16*</td>
<td>27</td>
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*In two patients, technical difficulties precluded detailed analysis of the activation sequence.*

<table>
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<th>Number of monoform tachycardias induced</th>
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<th>Epicardium</th>
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<td>15</td>
<td>12</td>
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<table>
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<th>Configuration of tachycardia</th>
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<tbody>
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<td>5</td>
</tr>
<tr>
<td>Biregional</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Complete</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Incomplete</td>
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<td>5</td>
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<td>Complete</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Incomplete</td>
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Table 1. Distribution of the Activation Sequences During Tachycardia in 35 Patients
Figure 2. During ventricular tachycardia, a spontaneous shift in surface ECG configuration (bottom tracing) from a leftward to a rightward axis was observed. Panels A and B, Epicardial isochronic maps (12 ms) of the two respective surface patterns. Both are consistent with a monoregional sequence of activation with a localized site of onset and rapid sequential spread. The shift in ECG configuration corresponds to a shift in the site of earliest epicardial breakthrough and the direction of spread of the subsequent activation wavefront. Panels A' and B', Corresponding endocardial maps for A and B. Both are complete figure eight configurations with similar activation sequences and only a minor shift in the site of earliest activation. Two activation fronts proceed around a region of block then unite on the distal side of the block to be followed by reactivation on the proximal side at the onset of the next tachycardia cycle.

Figure 8 (Fig. 2A' and B' and 4A' and B'). This term defined tachycardia whose sequence of activation followed the figure eight configuration described by El-Sherif et al. (2) in canine chronic myocardial infarction. At the site of earliest activation during tachycardia, two wavefronts—one
Figure 3. Isochronic maps of biregional epicardial activation recorded during ventricular tachycardia in two patients. Endocardial recordings were not obtained in these patients, and thus earliest epicardial breakthrough is assigned time 0. In panels A and B there is simultaneous onset of epicardial activation (0) at two sites 6 and 7 cm apart, respectively.

clockwise, the other counterclockwise—spread out and circled two arcs of conduction block to form a common return limb back to the site of onset. The sequence of activation was considered complete when the return pathway was spatially and temporally well defined, and there was no, or only a minimal, isoelectric gap between tachycardia cycles. The return pathway was characterized by low amplitude signals and slowed conduction over relatively short distances. When the opposing wavefronts could be easily identified but the return pathway was spatially or temporally poorly defined, the sequence of activation was considered incomplete. Complete figure eight activation was recorded only on the endocardium and was, in most instances, accompanied on the epicardium by monoregional and biregional patterns.

Simultaneous recordings of endocardial and epicardial activation. These were obtained in 27 episodes of tachycardia (12 patients). Endocardial activation during tachycardia preceded that on the epicardium by 12 to 96 ms in 25 of the 27 recordings. In the majority of episodes, the duration of endocardial activation recorded from the left ventricle exceeded that recorded over the epicardial surface of both ventricles, with endocardial activation sometimes continuing after that over the epicardium had ceased.

The sequence of activation recorded on one surface was matched by a similar sequence of activation on the remaining surface in less than half of the 27 episodes. Thus, there was no correlation between the sequence of activation recorded simultaneously on the endocardium and epicardium, and many combinations of the four described configurations were observed. In 10 of the 12 patients, multiple configurations of ventricular tachycardia were induced. For any given patient, different surface electrocardiographic configurations were usually associated with different sequences of activation. However, in a small number of tachycardias, different surface configurations were observed to correspond to the same or similar endocardial activation sequence as illustrated by the following examples.

In one patient, during ventricular tachycardia, there was a spontaneous transition in surface limb lead configuration from a leftward to a rightward axis (Fig. 2). Both patterns were characterized endocardially by complete figure eight activation sequences with only a minor shift (1 cm) in the site of earliest activation. Epicardially, both were associated with monoregional activation, beginning 64 and 12 ms, respectively, after the onset of endocardial activation. However, the transition in surface configuration corresponded to a marked shift in the site of earliest epicardial breakthrough, as well as the direction of spread of the subsequent activation wavefront (Fig. 2). In a second patient, a similar spontaneous transition in surface electrocardiographic configuration (Fig. 4) during tachycardia was again associated with a complete figure eight activation sequence on the endocardium. Biregional activation was observed on the epicardium. The differing contributions of the two epicardial sites of breakthrough to the epicardial activation sequence appeared to determine the resulting surface configuration.

**Discussion**

**Mapping technique.** The use of an endocardial mapping balloon is not new (3,4), but has until now required a ventriculotomy for introduction into the left ventricle. The advantages of the transmitral approach are severalfold and are dependent on the ventricle being left intact. Thus, there is no possibility of rendering the tachycardia noninducible by interrupting the circuit with the ventriculotomy. The fall in intracavity temperature associated with a ventriculotomy is not observed. The wall tension in the ventricle may be increased by the dextrose-filled balloon and facilitate induction of tachycardia. That these factors are important is evidenced by our uniform success rate in inducing ventric-
Figure 4. A spontaneous shift in surface configuration (bottom tracing) was observed during tachycardia. Panels A and B, Epicardial maps of the two respective configurations demonstrate biregional activation. Panels A’ and B’, Corresponding endocardial maps of A and B show figure eight activation sequences with similar sites of onset and location of the return limb. The shift in ECG configuration (lead 3) corresponds to the differing contribution of the two epicardial sites to the activation sequence. This is exemplified in A and B by the shaded areas which represent the extent of epicardial activation at 96 ms after the onset of endocardial activation.

Endocardial and epicardial activation. By utilizing the transmural approach, it was possible to record endocardial and epicardial activation simultaneously from a total of 220 sites. Despite this, continuous sequential electrical activation throughout the tachycardia cycle length was observed in <20% of the tachycardia mapped. This is consistent with the results of other mapping studies (5). In the remaining episodes there were varying gaps in excitation, which were of fixed duration for each tachycardia. Furthermore, the isoelectric periods on the endocardium were not bridged by epicardial activation. It is possible that intramural activation, not accessible to our mapping system, bridged these gaps. However, similar breaks in excitation (3 to 64% of the tachycardia cycle) were found experimentally by Cardinal et al. (6), when they recorded from intramural as well as subendocardial and subepicardial sites during tachycardia in canine myocardial infarction. They attributed this to their small number of recording sites. Another possibility is that signals with low amplitude or frequency characteristics, or both, that could not be detected by our mapping system accounted for the gaps in activation. In this study, intramural plunge electrodes may have provided additional information, but this was not technically feasible at the time of endocardial mapping. In humans, the ventricular arrhyth-
mias of ischemic heart disease are, however, generally believed to originate within the subendocardium (7-9).

In general, when the earliest site of activation was located endocardially, the duration of activation recorded on the endocardium exceeded that on the epicardium. Thus, epicardial activation often began and ended within the period of endocardial activation, suggesting that the former was not integral to the tachycardia mechanism. However, epicardial activation did appear to be an important determinant of surface electrocardiographic configuration. In a small number of patients with multiple configurations of ventricular tachycardia, spontaneous alterations in surface ECG configuration were found to correspond to alterations in epicardial activation sequence with little or no change in the pattern of endocardial activation. That the endocardium and not the epicardium was critical to the tachycardia mechanism in these instances is suggested by the following: complete figure eight sequences were recorded endocardially in two of the patients during tachycardia, with epicardial activation detectable for 40 to 45% and 35 to 40% of the tachycardia cycle length, respectively. In a third patient, epicardial activation began 48 ms after the earliest endocardial site of activation. The role of epicardial activation in determining surface electrocardiographic configuration has been previously emphasized by Horowitz et al. (7,10). In their model of acute myocardial infarction, ventricular arrhythmias were found to originate from surviving subendocardial Purkinje fibers, but it was the site of earliest epicardial breakthrough that determined QRS configuration.

Patterns of activation. Although isolated cases of macroreentry had been demonstrated clinically (11), it was not until the recent report by Miller et al. (5) that an attempt was made to categorize the hitherto unknown patterns of activation during human ventricular tachycardia. In this study, examining 110 endocardial and 110 epicardial sites and, more recently, examining both simultaneously, have enabled us to further characterize the patterns and sequence of activation during ventricular tachycardia. On both the endocardium and epicardium, monoregional activation characterized by a localized site of onset and radial spread of activation was the most frequently observed configuration. Complete figure eight patterns were observed on the endocardium exclusively and correspond to that described by El-Sherif et al. (2) in the epicardium of canine chronic myocardial infarction (that is, a site of earliest activation from which two wavefronts of opposing direction circulate and then return through a narrow common limb, back to the site of earliest activation). Using cryothermal techniques to terminate the tachycardia, El-Sherif et al. (12) were able to prove reentry in this model. Unfortunately, because of technical limitations at the time of operation, we were unable to attempt termination in this manner. It is possible that some of the tachycardias described by Miller et al. (5) as having a centrifugal sequence of activation may indeed have corresponded to figure eight sequences. The common return limb previously described may not have been identifiable in their study for two reasons. As stated earlier, this return pathway was characterized by slowed conduction over relatively short distances (1 to 2 cm), whereas the resolution power of the isochronal maps described by Miller et al. was 3 to 5 cm. Furthermore, the return limb may have been distorted and rendered less readily identifiable by the aneurysmotomy.

Circular sequences of activation were the remaining configuration observed on the endocardium. On the epicardium, continuous activation was noted in one instance to circle an aneurysm from which no activation could be recorded. However, the presence of a discrete aneurysm was not a prerequisite for this pattern of activation: on both the endocardium and epicardium, continuously recirculating fronts of excitation from one cycle to the next were recorded where no aneurysm was observed. This resembled the leading cir-

Figure 5. Isochronic maps of circular activation during tachycardia. Panel A, A broad front of activation proceeds rapidly in a clockwise direction on the epicardium but an excitation gap of 100 ms remains between tachycardia cycles. Panel B, A continuous recirculating excitation front is maintained from one cycle to the next on the endocardium.
cle of reentry described by Allessie et al. (13) in the experimental model of atrial flutter and suggested that this sequence of activation may be consistent with ventricular flutter.

Conclusions. Using a mapping system that enables simultaneous recordings from multiple endocardial and epicardial sites, we have been able to further characterize the activation sequence during ventricular tachycardia in the human ventricle. Although some of the results are consistent with experimental models of reentry, proof of this as the primary mechanism remains to be determined. The observed gaps in excitation during tachycardia may possibly be accounted for by intramural activation or by signals of an amplitude or frequency not detected. It is hoped that these issues will be addressed by future refinements of current technology.

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References