Epicardial Mapping of the Onset of Ventricular Tachycardia Initiated by Programmed Stimulation in the Canine Heart with Chronic Infarction

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SUMMARY The initial beats of ventricular tachycardia (VT) induced by programmed stimulation (PS) of the heart have frequently been observed to differ in QRS configuration from the subsequent uniform QRS complexes of tachycardia. The transient nature of these initial beats has made their study difficult during epicardial mapping with conventional, hand-held recording electrodes. Twenty-four dogs were studied with PS 1–10 months after coronary ligation. Twenty-six epicardial electrograms were recorded simultaneously during PS. The data were digitized for computer generation of isochronic maps for any desired beat. Three patterns of initiation were observed in episodes of tachycardia in which the initial beats differed from the subsequent beats of VT (11 of 18 runs of VT). Most frequently, the initial beats of VT originated near the pacing electrode before moving to a stable infarction zone location. Less frequently, the initial beats were due to transient reentry in the bundle branches or a transient shifting of early breakthrough sites in the infarction zone.

Electrophysiologic studies in patients with recurrent ventricular tachycardia (VT) have shown that the arrhythmia can frequently be reproduced in the laboratory with programmed electrical stimulation of the heart. When VT is initiated in this fashion, the initial beats of tachycardia after the last test stimulus often have a QRS configuration different from that of the subsequent stable tachycardia. These differing initial beats generally have been attributed to self-limited reentry involving the main bundle branches or to reentry in local circuits around the site of the pacing catheter. Intracavitary and epicardial mapping with a hand-held probe for epicardial mapping in patients have successfully determined epicardial activation sequence for the stable phase of VT. Such studies use data from the recording of many epicardial points in sequence and require long runs of identical complexes. The transient nature of the changing initial beats precludes their study by this technique.

Reports from this laboratory have described a system capable of recording 26 evenly spaced epicardial electrograms simultaneously. The data are digitized for computer generation of isochronic maps of the epicardial activation sequence for each desired beat. We used this system to analyze the initial beats of VT induced by programmed stimulation of the ventricles, and each run of tachycardia could be analyzed after only a single induction of the arrhythmia.

Methods

Twenty-four mongrel dogs of either sex that weighed 18–25 kg were used in this study. Under general anesthesia (sodium pentobarbital 30 mg/kg), the heart was exposed through a lateral thoracotomy in the fifth interspace and suspended in a pericardial cradle. A parietal wall infarction of the left ventricle was produced by ligation of one or more diagonal or obtuse marginal arteries. The chest was closed and the dog allowed to recover. Thirteen of the 24 dogs were fed a protein-deficient diet during the peri-infarction period in an attempt to increase the yield of subsequent ventricular arrhythmias by encouraging ventricular aneurysm formation.

The dogs were restudied 1–10 months after coronary ligation. They were anesthetized with a cloralose (100 mg/kg) and ventilated with room air through an endotracheal tube attached to a Harvard pump respirator. The heart was exposed through a median sternotomy and suspended in a pericardial cradle. A reference electrode was sewn on the anterior right ventricle. Two fine insulated wires were inserted by means of a 21-gauge needle into 1) the right ventricular myocardium near the pulmonary outflow tract, 2) the left ventricle near the base of the heart anteriorly, and 3) the left ventricle near the border of the infarction. Their exposed tips served as close bipolar pacing pairs.

A nylon mesh “stocking,” with 26 uniformly spaced bipolar electrode pairs (separation 1 mm) entwined in the mesh, was pulled over the heart. Unipolar and bipolar inputs from each electrode were connected to amplifiers with an input impedance of $10^{11}$ Ω and a frequency response of 0.1–1.5 kHz (unipolar data) and 5–1.5 kHz (bipolar data). Unipolar data were used exclusively in this study. All data were recorded on a 32-channel FM analog tape recorder. In addition to the data leads, three limb leads, a ventricular electrogram, a time code, and a voice log completed the 32 channels of input. A 32-channel analog-to-
Table 1. Description of Ventricular Tachycardia (VT)

<table>
<thead>
<tr>
<th>Dog</th>
<th>VT no.</th>
<th>Length of VT (cycles)</th>
<th>Initiation</th>
<th>Mean cycle length in VT (msec)</th>
<th>Pacing site</th>
<th>Site of electrode showing earliest epicardial activation for initial beats of VT</th>
<th>Site of electrode showing earliest epicardial activation during stable VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>205</td>
<td>LV</td>
<td>Same as stable VT</td>
<td>Infarct</td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>5</td>
<td>S1S2S3</td>
<td>370</td>
<td>LV (base)</td>
<td>Same as stable VT</td>
<td>Pacer</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>180</td>
<td>LV (base)</td>
<td>RBB (beats 1,2,3)</td>
<td>Infarct</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>8</td>
<td>S1S2S3</td>
<td>140</td>
<td>LV (anterior wall)</td>
<td>Infarction zone (beats 1,2,3,4)</td>
<td>Infarct</td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>130</td>
<td>LV</td>
<td>Same as stable VT</td>
<td>Infarct</td>
</tr>
<tr>
<td>D</td>
<td>1</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>140</td>
<td>RV</td>
<td>RBB (beat 1)</td>
<td>Infarct</td>
</tr>
<tr>
<td>E</td>
<td>1</td>
<td>34</td>
<td>S1S2S3</td>
<td>260</td>
<td>RV</td>
<td>Pacer (beat 1)</td>
<td>RBB</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>S1S2S3</td>
<td>286</td>
<td>RV</td>
<td>Same as stable VT</td>
<td>Infarct</td>
</tr>
<tr>
<td>F</td>
<td>1</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>135</td>
<td>LV</td>
<td>Pacer (beat 1)</td>
<td>Infarct</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>18</td>
<td>S1S2S3</td>
<td>160</td>
<td>LV</td>
<td>Pacer (beat 1)</td>
<td>Infarct</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>14</td>
<td>S1S2S3</td>
<td>160</td>
<td>LV</td>
<td>Pacer (beats 1,2); infarction zone (beats 3,4,5)</td>
<td>Infarct</td>
</tr>
<tr>
<td>G</td>
<td>1</td>
<td>&gt;50</td>
<td>RVP</td>
<td>170</td>
<td>RV</td>
<td>Infarction zone (beats 1,2,3)</td>
<td>Infarct</td>
</tr>
<tr>
<td>H</td>
<td>1</td>
<td>5</td>
<td>S1S2S3</td>
<td>165</td>
<td>RV</td>
<td>Pacer (beats 1,2)</td>
<td>RBB</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>6</td>
<td>S1S2S3</td>
<td>160</td>
<td>RV</td>
<td>Same as stable VT</td>
<td>RBB</td>
</tr>
<tr>
<td>I</td>
<td>1</td>
<td>6</td>
<td>S1S2S3</td>
<td>180</td>
<td>LV</td>
<td>Pacer (beats 1,2); infarction zone (beats 3)</td>
<td>Infarct</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>&gt;50</td>
<td>S1S2S3</td>
<td>188</td>
<td>LV</td>
<td>Pacer (beats 1,2); infarction zone (beats 3)</td>
<td>Infarct</td>
</tr>
<tr>
<td>J</td>
<td>1</td>
<td>6</td>
<td>S1S2S3</td>
<td>105</td>
<td>LV</td>
<td>Same as stable VT</td>
<td>Pacer</td>
</tr>
<tr>
<td>K</td>
<td>1</td>
<td>5</td>
<td>S1S2S3</td>
<td>130</td>
<td>RV</td>
<td>Same as stable VT</td>
<td>Pacer</td>
</tr>
</tbody>
</table>

Abbreviations: S1S2 = VT initiated by one premature stimulus (S1) after eight driven ventricular beats (S1); S1S2S3 = VT initiated by two consecutive premature stimuli (S1S2); RVP = VT initiated by a burst of rapid ventricular pacing; RBB = earliest recorded epicardial activation over right ventricular paraseptal area (similar to normal sinus rhythm); Pacer = earliest recorded epicardial activation near site of pacing electrode; infarction zone = earliest recorded epicardial activation initially shows beat-to-beat variability in the infarction zone before a stable pattern is achieved; infarct = earliest recorded epicardial activation during stable VT is above or adjacent to infarct; LV = left ventricle; RV = right ventricle.

digital converter digitized the data, which were then fed to a DEC PDP 11/34 computer. The data were displayed on a Tektronix 4014 graphics terminal. The computer can automatically select activation times (rapid intrinsic deflection 3 or 4 mV/10 msec), or these can be selected manually. Another program generated isochronic maps for each beat of selected data.

To initiate VT, the dogs underwent 1) bursts of rapid atrial pacing at various cycle lengths, 2) bursts of ventricular pacing at various cycle lengths, and 3) programmed premature stimulation of the ventricles. For the latter, a drive of eight beats (cycle length 300–400 msec) was followed by a premature stimulus (S1) of increasing prematurity until the effective refractory period (ERP) of the ventricle was reached. If VT was not initiated, this sequence was repeated using a premature stimulus (S2) 50 msec longer than the ERP of the ventricle and progressive prematurity of a second consecutive premature stimulus (S3) until the ERP of the ventricle was reached. Stimulation was initially carried out from the right ventricle. If this was unsuccessful, the stimulation site was moved to the left ventricle. Stimulus duration was 1–1.5 msec and intensity was approximately 0.5 mA above diastolic threshold.

All hearts were examined pathologically to determine the extent of infarction and to correlate infarct location with electrode position.

Results

VT (at least five beats) was observed in 11 of 24 dogs. The mode of initiation and pacing site are summarized in table 1. Although most tachycardias were reproduced two or three times at a given cycle length and degree of prematurity of the extrastimuli, the episodes of tachycardia were not usually repeatedly produced and easily terminated. Some tachycardias could only be terminated by cardioversion, while others degenerated into ventricular fibrillation. Data after one or two cardioversions were confused by the
appearance of ectopic rhythms and were no longer considered to be interpretable. In one dog, VT could only be produced by bursts of rapid right ventricular pacing.

Five of 11 dogs had more than one type of VT or had variations in the tachycardia at the onset (table 1). Eighteen unique runs of VT were observed in the 11 dogs.

In seven of 18 runs of VT, all beats of tachycardia in a given run had identical QRS morphology. The QRS morphology of initial beats (beats 1–5) of tachycardia differed from that of subsequent tachycardia in the remaining 11 runs (table 1). Three patterns of initiation were observed in these 11 runs.

**Tachycardia Originating Near Pacing Site**

**Initial Beats of Tachycardia Originating at the Pacing Site**

Most often (seven of 11 runs), the initial beats of tachycardia showed epicardial breakthrough* at the pacing site; this occurred with both right and left ventricular pacing sites. After one or two such beats, the site of early breakthrough would move to a constant location near the infarction zone† (two runs), move to several infarction zone locations before a constant area of breakthrough persisted (three runs) or move to the right ventricular paraseptal area at the site where epicardial breakthrough occurred in sinus rhythm (two runs) (fig. 1). This experiment shows the clumping or closer spacing of the isochrones in the area around the pacing electrode associated with the premature stimuli (S2 and S3). If one assumes a similar direction of conduction of the paced impulses from the stimulus site, such clumping can be considered to represent progressive slowing of conduction with each premature stimulus. The first nondriver beat of this tachycardia (V1) shows earliest recorded epicardial activity at the electrode adjacent to the pacing site. The second beat of tachycardia (not shown) is similar to the first (V1). The earliest recorded epicardial activity for the third and fourth beats of tachycardia (V3, V4) occurs between the infarction and the anterior interventricular groove. By the fifth beat (V5), the QRS morphology has stabilized; thereafter, the earliest recorded epicardial activity occurs at the superior border of the infarction. The appearance of one or more beats originating at the pacemaker site after premature ventricular stimulation was not uncommon in this series of dogs (10 of 24) or in others studied in our laboratory (unpublished observations). These beats may or may not lead to sustained VT. In some dogs, they consistently preceded sustained VT; in others, they did not precede sustained VT when it occurred.

**All Beats of Tachycardia Originating Near the Pacing Site**

Brief runs of tachycardia (four to five nondriver beats) were seen in three of 24 dogs in which all beats of the tachycardia showed epicardial breakthrough near the pacing electrode. Although these episodes did not lead to sustained VT, they allow some interesting observations on pacemaker-induced "repetitive firing" (fig. 2). These nonsustained runs of VT shared several characteristics:

1) There was progressive crowding of isochrones around the pacing site associated with the two consecutive premature stimuli delivered after the basic drive. 2) There was a tendency toward progressive lengthening of the RR interval before the tachycardia terminated. 3) As the tachycardia progressed, more and more of the electrodes surrounding the pacing site showed early epicardial activation. By the last beat of tachycardia, a large area surrounding the pacing site showed early, nearly simultaneous epicardial activation.

**Initial Beats of Tachycardia Breaking Through over the Right Ventricle, with Early Right Ventricular Activation Similar to Sinus Rhythm**

In three dogs, premature stimulation of the ventricles (right ventricle in two dogs, left ventricle in one dog) was able to elicit one to six beats showing epicardial breakthrough over the right ventricular paraseptal area, with early activation identical or very similar to that during sinus rhythm. After early activation of the right ventricular paraseptal area, epicardial activation spread roughly concentrically from the electrode showing earliest activation, and the total duration of ventricular activation was at least 80 msec. This is in contrast to activation in normal sinus rhythm, in which there is generally a second area of relatively early epicardial breakthrough in the apical region of the left ventricle and ventricular activation lasts at most 50 msec. In two dogs, these early beats immediately preceded long runs of tachycardia, with early breakthrough occurring in the infarction zone during stable VT (fig. 3). In this experiment, the first three beats of tachycardia (V1–V3) have a different QRS configuration than the subsequent beats. The earliest recorded epicardial activity for beats V1–V3 is present over the anterior right ventricle, in the paraseptal zone. The early activation of the right ventricle is very similar to that during sinus rhythm (fig. 3H). After initial activation of the right ventricle, epicardial activation spreads radially. There is a progressive clumping of isochrones at the lower edge of the infarct from V1–V3. This intense clumping is seen when a late activated electrode is in close proximity to an early activated one. Beat V4 then shows earliest recorded epicardial activity adjacent to the

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*The zone of epicardial breakthrough is defined in this study as the point underlying the electrode showing earliest epicardial activation. It is clear that increasing the number of epicardial data points would give a better estimate of epicardial breakthrough.

†Epicardial breakthrough in the infarction zone is defined as earliest recorded epicardial activation at an electrode within the infarction or adjacent to the infarction. Because the extent of the infarction was usually not clear before pathologic examination, the electrode density over the infarction was often not optimal. The nonspecific term "infarction zone" reflects this limitation.
area of intense clumping of isochrones in V₃. The subsequent beats of tachycardia are similar to beat V₄.

**Initial Beats of Tachycardia Breaking Through in the Infarction Zone but Moving to a Different Infarction Zone after Several Beats**

In two dogs, the initial beats of VT showed variability of earliest recorded activity in the infarction zone but settled after several beats to an area of consistent early breakthrough in the infarction zone.

**Discussion**

The initial beats of VT induced by programmed electrical stimulation of the heart have frequently been observed to differ in QRS morphology from the ensuing uniform beats of tachycardia.¹⁻⁴ The origin of these beats and their possible role in the initiation of VT has not received much attention. In a recent study, 13 of 21 patients with recurrent VT, had initial beats differing from stable ventricular tachycardia.¹ The investigators felt that these initial beats were due to macroreentry involving the main bundle branches. This conclusion was based on the findings that 1) a critical delay of retrograde His bundle activation during premature stimulation appeared necessary to produce these beats, and 2) the QRS of these beats was usually similar to the paced complex and preceded by a His potential with an HV interval equal to or greater than that of a normally conducted antegrade impulse.
Another group was only able to identify retrograde His delay in six of eight patients with differing initial beats. These investigators felt that a delayed retrograde His deflection could frequently occur secondary to premature stimuli applied to the ventricles and did not necessarily implicate the bundle branches as the source of the arrhythmia.

Other investigators studied the mechanism of the appearance of only one or two reciprocating beats after premature stimulation of the right ventricle in man. They observed that a critically prolonged retrograde His deflection was required to produce the reciprocating beat. However, they showed that the main source of the retrograde conduction delay was, in fact, in the Purkinje network near the stimulated site. They concluded that the reciprocating beat was usually due to local reentry in the Purkinje network near the pacing electrode. The similarity of the reciprocating beats to the paced complexes, regardless of the pacing site, further supported this observation.

In our experimental model, epicardial mapping revealed that at least three patterns of initiation were possible when initial beats differed in QRS morphology from subsequent stable VT.

**Initial Beats of Tachycardia Originating at the Pacing Site**

This was the most frequent pattern of initiation when initial beats differed from stable tachycardia. The presence of spontaneous ectopic ventricular beats after paced beats, and similar in QRS morphology to the paced beats, has been attributed by some investigators to reentry in local circuits around the pacing electrode site. These beats may also be reasonably attributed to mechanical stimulation by the pacing electrode, effects of local injury caused by the pacing electrode or triggered automaticity. The following observations suggest that local reentry at the pacing site is the more plausible mechanism.

The occurrence of multiple undriven beats originating near the pacing site after premature stimulation of the ventricles was seen in three dogs (fig. 2). In these examples, the first beat or two show a slight beat-to-beat change in early breakthrough, although all are in the vicinity of the pacing electrode and distant from the area of infarction. In fact, the area of early activation enlarges from beat to beat as the RR interval between beats tends to lengthen. This observation may be explained by postulating a local circus movement with no anatomic obstacle but rather a functional obstacle created by premature stimulation. This could occur if portions of the ventricular myocardium were nonexcitable in response to the arrival of the premature stimulus, while adjacent segments were able to conduct slowly and reexcite the former segment. This functional obstacle may be expected to change from beat to beat, causing a beat-to-beat change in the reentrant circuit.

Enlarging of the reentry circuit is suggested by the progressive increase in RR intervals during these short runs of VT. Allessie and co-workers have shown the occurrence of local reentry induced by premature
stimulation in isolated normal rabbit atrium without an anatomic obstacle,\textsuperscript{19, 21} and observed a gradual increase in cycle length in the initial phase of tachycardia in all of their experiments. The findings in figure 2 would be more difficult to explain in terms of mechanical stimulation or triggered automaticity.\textsuperscript{22, 23} One would expect an automatic focus to show a uniform pattern of epicardial activation around the pacing site for each beat.

Reentry induced by programmed stimulation in normal canine ventricular tissue has also been demonstrated by Sasyniuk and Mendez.\textsuperscript{24}

The absence of latency between the stimulus artifact and the electrogram adjacent to the pacing site has been interpreted in clinical electrophysiologic studies to represent absence of "local" conduction delay in the vicinity of the pacing electrode.\textsuperscript{1, 20} Our data show that local conduction delay with premature stimuli may extend for several centimeters around the pacing site. This is not necessarily appreciated by examining only the latency between the stimulus artifact and a single electrogram very close to the pacing site. The reference electrogram in figure 2A is adjacent to the pacing electrode in the right ventricle. Little latency is
noted between the premature stimuli and R, in spite of impressive local slowing of conduction around the pacing electrode (figs. 2B, 2C and 2D).

Initial Beats of Tachycardia Breaking Through over the Right Ventricle, with Early Right Ventricular Activation Identical to Sinus Rhythm

This phenomenon was observed in three of the 24 dogs studied. Activation of the anterior wall of the right ventricle in normal sinus rhythm is initiated by the impulse reaching the anterior papillary muscle of the right ventricle via the right bundle branch. Epicardial breakthrough in man and dogs occurs over the anterior right ventricular paraseptal area, the area pretrabecularis. Early breakthrough of an ectopic ventricular beat over the same area activated early in normal sinus rhythm, with an activation sequence over the anterior right ventricle very similar to that of sinus rhythm, strongly suggests that the impulse arrived via the right bundle branch. Areas of early epicardial activation in normal sinus rhythm have been used by others to identify impulses emanating from the termini of the bundle branches.

We believe that these initial beats breaking out over the anterior right ventricle, as in sinus rhythm, are propagated via the right bundle branch. The most probable mechanism for this occurrence is reentry in the main bundle branches. The premature ventricular stimulus finds the right bundle branch refractory, but conducts slowly in a retrograde fashion up the left bundle branch system to the common His bundle and then antegrade down the recovered right bundle branch to reexcite the right ventricle. Bundle branch reentry has been postulated to occur in both experimental animals and man. In some cases has been thought to be the mechanism of prolonged recurrent episodes of VT in man. The nonuniform recovery of excitability in the main bundle branches would facilitate this form of reentry.

There are, of course, other mechanisms that would lead to activation of the heart via the right bundle branch. First, the presence of random supraventricular or His "capture" beats could occur at the beginning of VT. This is unlikely because of the reproducibility in some animals of one or more of these beats after extrastimuli with a given degree of prematurity. Atrioventricular node reentry is not entirely ruled out, but one would have to postulate that left bundle branch block occurred during atrioventricular nodal tachycardia in each of these cases.

Second, one could postulate the presence of a focus of excitation (either reentrant or triggered) associated with the infarction, which activated the left bundle branch by retrograde penetration of the distal Purkinje system to enter and emerge ultimately from the right bundle branch. If this were the case, one would reasonably expect at least a second area of
relatively early breakthrough in the infarction zone. This was not observed.

Since the principal goal of our experiments was to study epicardial activation during tachycardia, we did not attempt to record activity from the His bundle and main bundle branches. Although the absence of these data is a limitation in this study, the recording of His bundle activity during the induction of VT by pacing has not provided a clear distinction between the mechanisms of local pacemaker reentry with a concomitant retrograde His deflection and bundle branch reentry.4, 5, 6, 35

Bundle branch reentry was usually limited to one to six beats, although in one dog, a sustained tachycardia was elicited (up to 34 beats). In the same dog, another type of VT with consistent early breakthrough in the infarction zone also was elicited.

Initial Beats of Tachycardia Breaking Through in the Infarction Zone but Moving to a Different Infarction Zone after Several Beats

This pattern of initiation was only observed in two of 24 experiments. These initial unstable beats may be due to an early instability in infarction zone reentrant pathways or its routes of exit to normal myocardial tissue. In some of these initial unstable beats, the earliest recorded activity occurred several centimeters from the border of the infarction. The original impulse may have been related to the infarction zone, but could penetrate the distal conducting system and show epicardial breakthrough at sites removed from the original impulse.6, 10 It is also possible that greater electrode density over the infarction would have revealed earlier epicardial activation in this area.
Although this pattern of initiation occurred infrequently in this study using chronic infarction, it occurred in four of five dogs studied by a similar protocol 4 days after a 2-hour occlusion of a large coronary artery (unpublished observations). More instability might be expected in the reentrant pathways and its routes of exit in this more acute model of myocardial infarction.

Limitations of the Study

Determining the global epicardial activation sequence is only the initial step in elucidating the phenomena described. VT associated with previous myocardial infarction in man\(^1,3,4\) and experimental animals\(^5\) has been shown to be related to reentry and continuous electrical activity in the infarction zone. We did not observe these phenomena with our limited array of electrodes on the epicardium. The mechanisms that we have proposed based on epicardial data cannot be considered definitive. Simultaneous acquisition of endocardial, transmyocardial and epicardial data is required to define further the mechanism of these rapidly changing arrhythmias.

Several obstacles have previously hampered the intraoperative study of VT in man and experimental animals. The arrhythmia may be difficult to elicit consistently or be very short lived when it occurs. There may be beat-to-beat changes in the QRS complex or rapid deterioration of the arrhythmia to ventricular fibrillation. The recording of multiple electrograms simultaneously with on-line computer generation of activation data has made possible the beat-by-beat analysis of these complex, fleeting arrhythmias. Using such a system, we focused on the onset of VT induced by programmed stimulation of the ventricles. Ventricular tachycardia so induced may begin with variable QRS morphology before a stable pattern is established. These initial variable beats may be due to 1) reentry in the region of the pacing electrode, 2) reentry in the main bundle branches or 3) a beat-to-beat change in epicardial breakthrough in the infarction zone. When a stable ventricular tachycardia follows these initial beats, the earliest recorded epicardial activation for the beats of the stable phase is usually in the infarction zone.

The complexities of the initial beats of VT produced by programmed stimulation warrant caution in extrapolating the activation sequence of one or two induced beats to the mechanism of sustained VT.\(^36\)

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