The Incidence and Clinical Significance of Epicardial Late Potentials in Patients with Recurrent Sustained Ventricular Tachycardia and Coronary Artery Disease

MARK E. JOSPEHSON, M.D., MICHAEL B. SIMSON, M.D., ALDEN H. HARKEN, M.D., LEONARD N. HOROWITZ, M.D., AND RITA A. FALCONE

SUMMARY Seventy-eight patients with ventricular tachycardia associated with coronary artery disease underwent intraoperative mapping while in sinus rhythm to evaluate the frequency and significance of late potentials. In 30 of these patients, the surface ECG was subjected to signal averaging to correlate the incidence and duration of low-amplitude, delayed electrograms with the presence of late potentials recorded during epicardial mapping.

One to four epicardial late potentials were observed in nine patients (11.5%). These nine patients did not differ hemodynamically from patients without late potentials. In four patients, the site of epicardial breakthrough during ventricular tachycardia bore no relationship (i.e., >3 cm away) to the late potential or the site of origin of the tachycardia. In the five other patients with late potentials, epicardial breakthrough and site of origin of ventricular tachycardia were closely related to the free wall of an apical aneurysm. However, three of these patients had additional tachycardias from disparate sites. Twenty-seven of 30 patients in whom signal averaging was used had a low-amplitude signal in the terminal 40 msec of the amplified QRS complex. In 24 of these 27 patients (89%), the low-amplitude tail was demonstrated in the absence of epicardial late potentials. We conclude that epicardial late potentials are found infrequently in patients with ventricular tachycardia associated with coronary artery disease; epicardial late potentials cannot be used to localize ventricular tachycardia; and the specific low-amplitude tail on the signal-averaged electrogram is unrelated to epicardial events.

A RECURRENT, sustained ventricular tachycardia associated with coronary artery disease is most likely a reentrant arrhythmia. Slow conduction is considered a prerequisite for reentry, and it is believed that such slow conduction occurs in the areas of prior myocardial infarction in patients with this arrhythmia. Fontaine et al.1-4 used epicardial mapping during sinus rhythm to localize ventricular tachycardia and guide surgery in patients with ventricular tachycardia unassociated with coronary artery disease. Late potentials were recorded during sinus rhythm and were suggested as markers of slow conduction denoting an area in which reentry could take place. The almost uniform finding of epicardial late potentials in patients with ventricular tachycardia associated with arrhythmogenic right ventricular dysplasia led to the suggestion that late potentials can be used to guide surgery for all reentrant ventricular tachycardias, for they defined the electro-physiologic substrate of slow conduction required for the genesis of the tachycardia.

Signal averaging has been used to evaluate the propensity to ventricular tachycardia. Simson,3 Breithardt et al.,6 Rozanski et al.7 and Hombach et al.8 demonstrated that patients with recurrent sustained ventricular tachycardia and coronary artery disease have delayed, low-amplitude signals after the end of the QRS on the surface ECG. Low amplitude and terminal delay were relatively specific for patients with ventricular tachycardia.

The purpose of the present study was to evaluate the frequency and significance of epicardial “late” potentials recorded during sinus rhythm by epicardial mapping in patients with recurrent ventricular tachycardia due to coronary disease, and to correlate the presence of such epicardial delayed potentials with the incidence and duration of the low-amplitude, delayed signal on the surface ECG in patients with ventricular tachycardia.

Materials and Methods

Patients Seventy-eight patients with medically refractory ventricular tachycardia and coronary disease underwent intraoperative sinus mapping before surgery for their arrhythmia. There were 66 men and 12 women, ages 32–74 years. All patients had had at least one myocardial infarction; 70 patients had a left ventricular aneurysm. All eight patients without a left ventricular aneurysm had inferior infarctions and inferior akinesis.

The details of our intraoperative mapping procedure have been reported.9,10 Briefly, the heart is exposed...
through a median sternotomy and cannulated for cardiopulmonary bypass. The arterial cannula is placed in the ascending aorta and a venous cannula is placed through a pursestring suture in the left atrium. A Teflon-coated, stainless steel plunge electrode (0.005 inch in diameter) was inserted by a 23- or 25-gauge needle in the left ventricular free wall to record a reference electrogram. Initially, a similar plunge electrode was used to record a right ventricular reference electrogram. In the last 71 patients, the right ventricular reference electrogram was obtained from a perveneously introduced quadripolar electrode catheter inserted in the right ventricular apex. The proximal pair of electrodes was used for the right ventricular reference electrogram and a distal pair was used for programmed stimulation. Close bipolar (1–1.5 mm) electrograms were recorded at 54 predetermined epicardial sites with a hand-held probe or finger electrode. Electrograms were obtained from each of these sites during sinus rhythm and were simultaneously recorded with three surface electrograms (usually leads 1, 2 or aVF, V5, or V8) and the reference electrograms. The electrograms were filtered at 40–500 Hz and analyzed for timing and duration. Late potentials were defined as discrete activity existing at least 10 msec after inscription of the surface QRS. Discrete potentials were usually preceded by either low-amplitude, fractionated activity or one component of a split electrogram.

After completion of the epicardial sinus rhythm map, ventricular tachycardia was induced by programmed stimulation. The tachycardia was mapped both epicardially and endocardially, as previously described.9,10 The area of origin was defined by the site from which the earliest activity could be recorded.9,10

**Signal Averaging Technique**

Signal averaging of the surface ECG was accomplished in 30 patients by a signal averaging system developed by Simson.5 Bipolar X, Y and Z leads were used. A custom-built three-channel amplifier was used at a gain of 1000 and a band width of 0.05–300 Hz. The signal from each lead was amplified two to five times, passed through a four-pole, 250-Hz, low-pass filter, and then AD-converted to 12-bit accuracy (Analog Devices AD572) at 1000 samples/sec. The digital information was stored on tape by a Hewlett-Packard 9825 desktop computer. Each lead was recorded sequentially for 133 seconds. The electrocardiographic signals were averaged after passing through a template recognition program to reject ectopic beats and grossly noisy signals. An eight-point template began at the reference time and extended for 128 msec, spanning the distal QRS complex and early ST segment. A 512-msec segment of complexes matching the template was averaged beginning 100 msec before the QRS complex. Each averaged lead was filtered to eliminate low frequencies in the QRS complex and ST segments. A bidirectional digital filter eliminated impulse ringing of the filter. The filter processed forward in time until 40 msec into the QRS complex. The filter was then reset and the signal was processed backwards in time up to the same point in the QRS complex. The details of the analog signal processing, signal averaging, digital filtering and data analysis have been reported.5

**Results**

**Intraoperative Mapping**

Both right and left ventricular epicardial maps were obtained from all 54 sites in each of the 78 patients operated upon for ventricular tachycardia. One to four late potentials (i.e., discrete potentials occurring ≥10 msec after the QRS) were observed in nine patients (11.5%) (fig. 1). Eight of the patients had anteropapical aneurysms and one had an inferior infarction without a ventricular aneurysm. This patient population did not differ hemodynamically from the patients without late potentials, nor were the tachycardias different from those in the remainder of the group (table 1).

**Figure 1.** Epicardial late potential. (top) The epicardial grid of 54 predetermined sites. An apical aneurysm is denoted by the stippled area. The solid black circle near site 33 represents the site of a late potential. The cardiac silhouettes shown are, from left to right, anterior, left lateral, and inferior views. (bottom) Surface ECG leads 1, aVF, and V5, along with a right ventricular reference electrogram (RV ref) from an indwelling electrode catheter and a left ventricular reference electrogram (LV ref) from a plunge electrode near site 31, and a recording from left ventricular epicardial site 33. At LV site 33, a discrete late potential occurred 45 msec after the inscription of the QRS (dotted line). T = time lines.
TABLE 1. Clinical Data

<table>
<thead>
<tr>
<th></th>
<th>Late potential</th>
<th>No late potential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>9</td>
<td>69</td>
</tr>
<tr>
<td>Site of infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>8</td>
<td>54</td>
</tr>
<tr>
<td>Inferior</td>
<td>1</td>
<td>16 (8 with aneurysm)</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>8/9</td>
<td>62/69</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>14–45% (26%)</td>
<td>5–53% (30%)</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>10–35</td>
<td>5–32</td>
</tr>
</tbody>
</table>

In four patients, the site of epicardial breakthrough bore no relationship to the site of origin of the ventricular tachycardia (i.e., was more than 3 cm away from the earliest recorded activity). The site of earliest activity in each of these four patients was on the left side of the interventricular septum, whereas the late potentials were on the aneurysm on the free wall of the left ventricle. Moreover, epicardial breakthrough during the tachycardia in each of these patients differed from the site of the late potential and from the earliest recorded activity, which was always on the septal endocardium. An example of such a patient is shown in figure 2, in which the epicardial late potential was recorded on the free wall adjacent to the interventricular septum, the earliest epicardial breakthrough during ventricular tachycardia occurred at the right ventricular apex, and the earliest site of activation during the tachycardia occurred at the endocardium of the middle left ventricular septum. Thus, there was no relationship of the site of the late potential, epicardial breakthrough, and origin of the tachycardia.

In the remaining five patients, the late potential occurred near the earliest site of activation of the tachycardia: the free wall of the left ventricle at the endocardial border of the aneurysm (fig. 3). In these patients, the epicardial breakthrough, the late potential and the site of origin occurred within 3 cm of each other. Three of these five patients had additional tachycardias in which the origins differed from the late potential and occurred on the interventricular septum. Thus, even in patients in whom at least one tachycardia morphology was associated with the late potential, the site of the late potential could not be used as the sole guide for surgical intervention.

Signal Averaging

High-quality, signal-averaged electrograms were obtained in 30 patients before surgery. In 27 of these patients (90%), a low-amplitude tail occurred at the end of the amplified QRS and after apparent inscription of the QRS on the standard ECG. That is, the low-amplitude tail, which was less than 25 μV, occurred in the early part of the ST segment on the standard surface ECG. Of these 27 patients, three (11%) had epicardial late potentials. When the signal-averaged electrogram was compared with the epicardial activation times, none of the epicardial electrograms occurred simultaneously with the low-amplitude electrogram on the signal-averaged ECG, except in the three patients who had epicardial late potentials, in each of whom the late potentials occurred during inscription of the low-amplitude signal. In contrast, 24 of 27 patients (89%) without epicardial late potentials had delayed, low-
FIGURE 3. Close relationship between epicardial late potential, epicardial breakthrough during ventricular tachycardia, and endocardial site of origin. (A) The epicardial sinus rhythm map, as described in figures 1 and 2. An epicardial late potential is seen at the edge of an apical aneurysm at site 39. The analog record demonstrating this finding is shown in panel B. Surface leads 1, 2 and V5 are shown with electrograms from the right ventricular apex (RVA) and from epicardial left ventricular (LV) electrograms from sites 35, 39, 33, 34 and 28. A discrete late potential occurred 60 msec after inscription of the surface QRS at site 39. (C) An epicardial isochronic map during ventricular tachycardia. X marks the endocardial site of origin, which can be seen at the site of the epicardial late potential in panel A. Moreover, the epicardial breakthrough during ventricular tachycardia was at the adjacent border of the aneurysm in the normal tissue.

amplitude electrograms during signal averaging. Examples of signal-averaged QRS complexes in the presence or absence of late potentials are shown in figures 4 and 5. Thus, the presence of epicardial potentials was not a prerequisite for the presence of the low-amplitude, late, signal-averaged electrogram.

Discussion

One of the prerequisites for reentry is slow conduction. In the presence of coronary artery disease, this slow conduction is believed to result from the impulse traveling through diseased myocardium. Early studies in animals by Boineau and Cox and Waldo and Kaiser suggested that ischemia can cause fractionation of conduction manifested by fragmented and delayed electrograms. When these fragmented electrograms extended beyond the T wave, and hence the recovery period of the adjacent excitable tissue, ventricular tachyarrhythmias ensued. A similar set of circumstances probably exists in patients with chronic ischemic coronary disease who have ventricular tachycardia. Recently, signal averaging devices have been
used to detect this substrate of slow conduction. Simpson,5 Breithardt et al.,6 Rozanski et al.7 and Hombach8 demonstrated late, low-amplitude potentials in patients with recurrent ventricular tachycardia and coronary artery disease. These low-amplitude delayed signals were recorded in 92% of patients with ventricular tachycardia and were observed in less than 10% of patients without ventricular tachycardia.5 They are thought to represent the substrate of slow conduction required for the genesis of these arrhythmias.

Using epicardial mapping, Fontaine et al.1-4 delayed potentials during sinus rhythm in patients with ventricular tachycardia in the absence of coronary artery disease. These workers have demonstrated in patients with arrhythmogenic right ventricular dysplasia in whom ventricular tachycardia can be reproducibly initiated by programmed stimulation that late potentials can be recorded during sinus rhythm. When ventricular tachycardia was induced, these areas of late potentials often coincided with the epicardial breakthrough site, although the epicardial breakthrough frequently occurred after the onset of the QRS. Although detailed endocardial mapping was not performed, these workers believed that the late potentials were markers of slow conduction arising in abnormal tissue that identified the slow conducting component of the reentrant circuit of the arrhythmias. Similar late potentials have been described by Gallagher et al.9 in a patient with scleroderma and ventricular tachycardia. Thus, it appears that late potentials are a common finding in patients with ventricular tachycardia unassociated with coronary artery disease, particularly those with right ventricular dysplasia. Although late potentials appear to be markers of slow conduction, their exact role in the genesis of ventricular tachycardia in these cases was not established, for they may also represent areas of abnormal tissue from a blind loop of slow conduction unrelated to the tachycardia circuit.

Our data suggest that epicardial late potentials are neither sensitive nor specific markers of ventricular tachycardia associated with coronary artery disease and bear no relationship to the low-amplitude potential on the signal-averaged ECG. Although delayed potential may be a marker of slow conduction from an area that may be involved in a reentrant circuit, in four of nine cases there was no relationship between the finding of late potentials and the area of earliest activity recorded during the tachycardia. Even when the area of late potential was adjacent to an area from which the earliest activity during the tachycardia was recorded, the earliest activity was always recorded from the endocardium. Furthermore, in this latter group of patients, additional separate and distinct tachycardias occurred at sites distant from the area of the late potential. Thus, late potentials appear to be uncommon in the setting of coronary artery disease and cannot be used to localize tachycardias, even when they are demonstrated. The morphology of the ventricular tachycardia, the site of infarction or aneurysm formation and the hemodynamic pattern did not predict which patients would have epicardial late potentials.

Relationship of Other Studies

The early experience of Fontaine et al.,1-4 Wittig and Boineau,5 and Moran et al.,6 as well as experimental data,17 emphasized that epicardial recording may not adequately localize the site of origin of ventricular tachycardia associated with coronary artery disease. Epicardial mapping studies during the tachycardia frequently revealed one or more epicardial breakthrough sites that always followed the onset of the surface QRS during the tachycardia,3,9,10,16,18,19 which suggests that the epicardium was not the source of this arrhythmia. Support of the subendocardial origin of the arrhythmia is suggested by catheter mapping techniques,20-22 intraoperative endocardial mapping,4,10,19 and pathologic data.23 These data have shown that ventricular tachycardia associated with coronary artery disease appears to arise at or near the endocardium.

Late potentials have also not been a consistent finding in other studies from patients with myocardial infarction and ventricular tachycardia. Fontaine et al.1,2
found late potentials in six of 19 patients with ventricular tachycardia and coronary artery disease; in two of the late potentials correlated with the earliest epicardial breakthrough. In the remaining patients, the data were described as uninterpretable. Waldo et al. 24 and Arciniegas et al. 25 reported fractionated epicardial and/or endocardial electrograms in 20 of 21 patients with ventricular arrhythmias (17 of whom had sustained ventricular tachycardia), but did not specify which electrograms were epicardial and which were endocardial. They recorded delayed potentials in 12 of the 21 patients. However, without clarification of which of these 12 patients had epicardial late potentials, the exact frequency of epicardial late potentials during sinus rhythm in their studies is unknown. Our data are compatible, in that epicardial mapping during sinus rhythm infrequently demonstrates late potentials and there may be no relationship with these late potentials and the tachycardia.

The data of Waldo et al. 24 and Arciniegas et al. 25 who recorded both fractionated and late potentials from endocardial sites at surgery, and catheter endocardial mapping data from our laboratory. 21, 26, 27 suggest that delayed activity is frequently recorded in the endocardium. We presented preliminary data that the fractionated electrograms recorded on the endocardium with catheters correlated with the area of earliest activity during the tachycardia. More work, however, is necessary to evaluate the significance of fractionated endocardial electrograms and relate them to the site of origin of the tachycardia and to the low-amplitude signals recorded by signal averaging techniques.

References


The incidence and clinical significance of epicardial late potentials in patients with recurrent sustained ventricular tachycardia and coronary artery disease.
M E Josephson, M B Simson, A H Harken, L N Horowitz and R A Falcone

_Circulation_. 1982;66:1199-1204
doi: 10.1161/01.CIR.66.6.1199

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/66/6/1199

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/