Determinants of Ventricular Tachycardia in Patients with Ventricular Aneurysms: Results of Intraoperative Epicardial and Endocardial Mapping

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SUMMARY  We performed epicardial and endocardial mapping in 11 patients with ventricular aneurysms; six had chronic, recurrent ventricular tachycardia and five had no ventricular arrhythmias more severe than isolated ventricular premature complexes. Forty to 66 epicardial and 16–40 endocardial points were recorded during stable sinus rhythm in each patient. Local electrograms were evaluated as to timing and presence of fragmentation (duration > 50 msec, amplitude < 1 mV, absence of discrete intrinsicoid deflection). Activation of the epicardial surface of the aneurysm was abnormal in all patients, and extended beyond completion of the QRS in three patients in the arrhythmia group and two in the nonarrhythmia group (NS). Activation of the epicardial border zone was normal in all patients. Electrograms from the endocardial surface of the aneurysm were abnormally fragmented in all patients and the mean duration of activation was not different between patients with and without arrhythmias (85.5 ± 14.1 vs 96.2 ± 13.8 msec, NS). However, in patients with ventricular tachycardia, electrograms from 33–58.3% (mean 45.5 ± 8.8%) of the endocardial border zone showed fragmentation, compared with 0–16.7% (mean 4.9 ± 7.4%) of the endocardial border zone in patients without arrhythmias (p < 0.05).

Fragmentation was always along the septal border of the aneurysm. The mean duration of the most prolonged endocardial border zone electrogram was 97.5 ± 17.0 msec in ventricular tachycardia patients and 67.0 ± 27.1 msec in patients without arrhythmia (p < 0.05). Five of six ventricular tachycardia patients had electrical activity in the endocardial border zone extending beyond the end of the QRS, compared with one of five patients without ventricular tachycardia (p < 0.05).

We conclude that fragmented electrical activity is present in all patients with ventricular aneurysms, but the extent and severity of fragmentation in the endocardial border zone is greatest in patients with recurrent ventricular tachycardia.

VENTRICULAR ARRHYTHMIAS are a frequent, potentially lethal complication of chronic coronary artery disease and are related to the presence and severity of areas of abnormal contraction, especially ventricular aneurysms. The electrophysiologic correlates of these areas are not known. We performed detailed epicardial and endocardial mapping on 11 patients with ventricular aneurysms and compared the findings in patients with documented chronic, recurrent ventricular tachycardia with those in patients with no arrhythmias more severe than isolated ventricular premature complexes.

Methods

Patients

The clinical and angiographic data on our patients are summarized in Table 1. Each patient had a ventricular aneurysm clearly defined by cardiac catheterization and confirmed by visualization at surgery. Six of 11 patients had documented chronic, recurrent ventricular tachycardia: two had recurrent sustained ventricular tachycardia requiring multiple cardioversions, two had recurrent ventricular tachycardia/fibrillation requiring multiple defibrillations, one patient had recurrent nonsustained ventricular tachycardia and one episode of ventricular fibrillation with no acute cause and one had recurrent nonsustained ventricular tachycardia. Five patients had no history of significant arrhythmias and had no arrhythmias more severe than isolated ventricular premature complexes during at least 24 hours of monitoring. There was no significant difference in the number of coronary vessels with stenoses, ejection fraction, or length of the abnormally contracting segment (as a percentage of total diastolic circumference) between patients with and without arrhythmia. All patients gave informed consent for the protocol.

Mapping

The heart was approached through a median sternotomy and the conventional cannulations for cardiopulmonary bypass were carried out. A pair of Teflon-coated, stainless-steel reference electrodes with a 2-mm interelectrode distance was attached to the anterior right ventricle to serve as a reference electrode.

Epicardial mapping in normal sinus rhythm was performed before cardiopulmonary bypass using a finger-tip electrode with a 2-mm interelectrode distance. Bipolar electrograms (40–500 Hz) from 40–66 epicardial sites, the reference electrogram and three surface ECG leads were recorded simultaneously. In
After incision of the aneurysm, epicardial mapping was performed at 37°C and before the administration of cardioplegic solution. After incision of the aneurysm, electrograms from two to four sites on the endocardial surface of the aneurysm were recorded. The aneurysm was then excised, and 12–25 sites in the border zone of the aneurysm were mapped circumferentially. In each patient, recordings were made from equally spaced sites around the circumference of the aneurysm within 1 cm of the border. If fragmentation was noted, recordings were repeated at 2 and 3 cm from the border. Mapping was then performed at two to 10 remote sites. Five to 10 beats in normal sinus rhythm were recorded from each site. The entire mapping procedure took 15 minutes in each patient. After mapping, hypothermia (25–28°C) was instituted and cold potassium cardioplegia was administered. Coronary artery bypass grafting, in addition to closure of the aneurysm, was performed in seven patients. Four patients underwent isolated ventricular aneurysm resection.

Table 1. Clinical and Electrocardiographic Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>ECG</th>
<th>Arrhythmia</th>
<th>Location of aneurysm</th>
<th>CAD</th>
<th>EF</th>
<th>%ACS</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>F</td>
<td>Q V1-3</td>
<td>None</td>
<td>Anteroapical</td>
<td>LAD 100%</td>
<td>30%</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>F</td>
<td>Q III, F</td>
<td>None</td>
<td>Infroapical</td>
<td>LM 99%</td>
<td>15%</td>
<td>34</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
<td>M</td>
<td>QS V1-3</td>
<td>None</td>
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<td>LAD 100%</td>
<td>45%</td>
<td>46</td>
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<tr>
<td>4</td>
<td>48</td>
<td>M</td>
<td>QS V1-3</td>
<td>None</td>
<td>Anteroapical</td>
<td>LAD 100%</td>
<td>30%</td>
<td>46</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>F</td>
<td>Q V2-4</td>
<td>None</td>
<td>Anterior</td>
<td>LAD 100%</td>
<td>50%</td>
<td>31</td>
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<tr>
<td>6</td>
<td>52</td>
<td>M</td>
<td>QS V1-4</td>
<td>Recurrent sustained VT</td>
<td>Anteroapical</td>
<td>LAD 99%</td>
<td>26%</td>
<td>27</td>
</tr>
<tr>
<td>7</td>
<td>66</td>
<td>M</td>
<td>Q II, III, F</td>
<td>Recurrent VT/VF</td>
<td>Infroapical</td>
<td>LAD 96%</td>
<td>15%</td>
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<tr>
<td>8</td>
<td>35</td>
<td>F</td>
<td>Q II, III, F</td>
<td>Recurrent non-sustained VT</td>
<td>Infroapical</td>
<td>RCA 100%</td>
<td>45%</td>
<td>36</td>
</tr>
<tr>
<td>9</td>
<td>68</td>
<td>M</td>
<td>Q V1-3</td>
<td>Recurrent non-sustained VT, VF</td>
<td>Apical</td>
<td>LAD 100%</td>
<td>49%</td>
<td>33</td>
</tr>
<tr>
<td>10</td>
<td>57</td>
<td>M</td>
<td>QS V1-6</td>
<td>Recurrent sustained VT</td>
<td>Anteroapical</td>
<td>LAD 100%</td>
<td>30%</td>
<td>44</td>
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<tr>
<td>11</td>
<td>58</td>
<td>M</td>
<td>QS V1-4</td>
<td>Recurrent VT/VF</td>
<td>Anteroapical</td>
<td>LAD 100%</td>
<td>15%</td>
<td>45</td>
</tr>
</tbody>
</table>

Abbreviations: %ACS = abnormally contracting segment as a percentage of the end-diastolic ventricular circumference; EF = ejection fraction; LAD = left anterior descending coronary artery; LCF = left circumflex coronary artery; LM = left main coronary artery; RCA = right coronary artery; VF = ventricular fibrillation; VT = ventricular tachycardia.

Each patient, four to eight sites were on the surface of the aneurysm and 12–15 were within 1–2 cm of the visual border of the aneurysm. Signals were isolated, amplified and recorded on a physiologic recorder (Irex Medical Systems) at a paper speed of 200 mm/sec.

Epicardial activation time was determined as previously described. Measurements were made from the peak of the major deflection of the reference electrogram to the peak of the major deflection of the epicardial electrogram. The interval from the earliest onset of the QRS in the body surface leads to the reference electrogram was added to the interval from the reference electrogram to the epicardial electrogram to determine local activation time. For endocardial electrograms, measurements were made from the reference electrogram to the point where the endocardial electrogram deviated from baseline. The interval from the earliest onset of the QRS in the body surface leads to the reference electrogram was added to the interval from the reference electrogram to the point where the endocardial electrogram deviated from baseline to determine the time of onset of the electrogram. The duration of an electrogram was determined as the time from when the electrogram first deviated from baseline to the time when the electrogram returned to baseline. The duration of the QRS was from the earliest onset of the QRS in the body surface leads to the latest activation of the QRS in the body surface leads. An electrogram was considered to extend beyond the QRS when it extended beyond the latest activation in any surface lead. All measurements were calculated as the mean of five to 10 beats during stable sinus rhythm. Measurements
were considered accurate to ± 5 msec, and measurements of individual beats at a given site were always within this range.

Electrograms were considered abnormally fragmented if they had no discrete, intrinsicoid deflection, a duration greater than 50 msec and an amplitude less than 1 mV. The percentage of the endocardial border zone that had fragmented electrograms was determined by dividing the number of sites showing fragmentation by the total number of equally spaced sites on the circumference and within 1 cm of the aneurysm.

Statistical analysis was done by t test and chi-square analysis.

Results

Epicardial Mapping

Activation of the epicardial surface of the aneurysm was abnormal in all patients (table 2). One patient in the arrhythmia group and one in the nonarrhythmia group had no recordable electrical activity over the surface of the aneurysm. In all the other patients, a discrete electrogram was recorded from the epicardial surface of the aneurysm. Figure 1 shows epicardial electrograms from a patient with and a patient without ventricular tachycardia. In three patients with and two without arrhythmias, epicardial activation over the aneurysm was completed after the surface QRS. In the remaining four patients, activation over the aneurysm occurred during the QRS, but later than would be expected for the given anatomic segment. Two patients with and one without arrhythmias had low-voltage activity preceding the discrete deflection, but there were no fragmented epicardial electrograms. There was no difference in epicardial electrograms between patients with and without arrhythmias.

Electrograms from the entire epicardial border zone of the aneurysm were recorded. No electrograms from the epicardial border zone were fragmented and no electrograms extended beyond the QRS. Because the epicardial border zone points were in a variety of myocardial segments, we could not comment on smaller amounts of delay. All electrograms from remote sites were within the QRS and not fragmented.

Endocardial Mapping

Electrograms from the endocardial surface of the aneurysm were abnormal in all patients (table 2). One patient in the arrhythmia group and one patient in the nonarrhythmia group had no recordable activity from the endocardial surface of the aneurysm. In the remaining nine patients, endocardial activity within the aneurysm was fragmented. Figure 2 shows fragmented electrograms from the endocardial surface of the aneurysm in a patient without arrhythmias and a patient with ventricular tachycardia. In two patients with and two patients without ventricular tachycardia, endocardial activity within the aneurysm extended beyond the end of the surface QRS (NS). The average duration of the longest electrogram from the endocardial surface of the aneurysm was 85.8 ± 14.1 msec in the arrhythmia group and 96.2 ± 13.8 msec in the nonarrhythmia group (NS). Thus, electrograms from the endocardial surface of the aneurysm did not

<table>
<thead>
<tr>
<th>TABLE 2. Electrophysiologic Data</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aneurysm</strong></td>
</tr>
<tr>
<td>Pt</td>
</tr>
<tr>
<td>Nonarrhythmia</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>Mean</td>
</tr>
</tbody>
</table>

**Arrhythmia**

| Pt | Duration of QRS (msec) | Epicardial activation time (msec) | Onset (msec) | Duration (msec) | Amplitude (mV) | Fragmented (%) | Onset (msec) | Duration (msec) | Amplitude (mV) |
| 6 | 75 | 90 | NA | 58.3 | 0 | 110 | 0.3 |
| 7 | 95 | 85 | 10 | 74 | 0.1 | 50.0 | 0 | 125 | 0.2 |
| 8 | 90 | NA | 0 | 80 | 0.5 | 41.7 | 0 | 95 | 0.7 |
| 9 | 80 | 75 | 0 | 110 | 0.9 | 41.7 | 10 | 90 | 0.5 |
| 10 | 105 | 125 | 0 | 85 | 0.9 | 33.0 | 10 | 85 | 0.9 |
| 11 | 75 | 80 | 15 | 80 | 0.8 | 50.0 | 0 | 80 | 0.8 |
| Mean | 85.8 ± 14.1 | 45.8 ± 8.8 | 97.5 ± 17 |

Epicardial activation time refers to the activation time of the latest point on the epicardial surface of the aneurysm. Onset, duration and amplitude refer to the most prolonged electrogram from the endocardial surface or the border zone of the aneurysm.

Abbreviation: NA = no activity.
Differ between patients with and patients without ventricular tachycardia.

Mapping from the endocardial border zone is illustrated in figures 3 and 4. Fragmented electrograms were recorded from 33.0–58.3% of the border zone in patients with arrhythmias (mean 45.8 ± 8.8%), but only 0–16.7% of the border zone of patients without arrhythmias (mean 4.9 ± 7.4%, p < 0.05) (table 2). In the ventricular tachycardia patients, fragmented electrograms always occurred along the septal border of the aneurysm and extended 1–2 cm in from the edge of the aneurysm. The mean duration of the longest endocardial border electrogram was 97.5 ± 17.0 msec in patients with ventricular tachycardia and 67.0 ± 27.1 msec in those without (p < 0.05). Endocardial activity in the border zone that extended beyond the QRS was noted in five patients with ventricular tachycardia and one patient without arrhythmias (p < 0.05). Electrograms from remote endocardial areas were not fragmented and fell within the QRS.

Discussion

The association of ventricular aneurysms with malignant ventricular arrhythmias is well established, but the electrophysiologic factors that determine whether a patient with a ventricular aneurysm will have malignant ventricular arrhythmias are unknown. Although epicardial delay over ventricular aneurysms has been described, the correlation of this delay with the presence of ventricular arrhythmias is controversial. Gallagher et al. reported epicardial activation over a ventricular aneurysm after the QRS in a patient with ventricular tachycardia. Klein et al. found such delay in nine of nine patients with abnormally contracting segments and ventricular tachycardia, but in none of eight patients without ventric-

Figure 1. Electrograms from the epicardial surface of the ventricular aneurysm from a patient without ventricular tachycardia (A) and from a patient with recurrent ventricular tachycardia/fibrillation (B). The top two lines are surface leads, the third line is the reference electrogram and the fourth line is the epicardial electrogram. Numbers represent QRS duration and epicardial activation in milliseconds. The epicardial electrogram is after the completion of the QRS in both patients.

Figure 2. Electrograms from the endocardial surface of the ventricular aneurysm from a patient without ventricular tachycardia (A) and from a patient with recurrent ventricular tachycardia/fibrillation (B). Format as in figure 1. Numbers with arrows under the electrograms represent duration of the electrogram in milliseconds. Note the standard on the endocardial electrogram in the second panel. The endocardial electrogram is fragmented in both patients.
ular tachycardia, and suggested that epicardial delay was a determinant of ventricular tachycardia. However, Fontaine et al. recorded delayed potentials after the QRS in only one of 10 patients with coronary artery disease and ventricular tachycardia; Horowitz et al. found delayed potentials in only three of 31 such patients. In patients with sustained ventricular tachycardia, intraoperative mapping has shown that the earliest activation occurs in the endocardial border of the aneurysm, indicating that the arrhythmia originates in this area. The electrophysiologic properties of this tissue that allow the initiation of these arrhythmias are not known.

Our results indicate that ventricular aneurysms markedly changed the electrical properties of ventricular myocardium. Epicardial and endocardial mapping within the aneurysm showed no difference between patients with and patients without ventricular tachycardia. However, patients with ventricular tachycardia had fragmented electrical activity from a larger portion of the endocardial border zone and had more prolonged electrograms in this zone than did patients without ventricular tachycardia. This correlation suggests that fragmented, prolonged electrical activity in the septal endocardial border zone may be important in the pathogenesis of ventricular tachycardia.

Our findings are consistent with studies by Spelman et al. Using endocardial catheter mapping, these workers found more extensive areas of endocardial fragmentation in patients with myocardial infarction and chronic sustained ventricular tachycardia than in patients with myocardial infarction and no arrhythmia. Our intraoperative technique permits more precisely placed recordings under direct visualization than the catheter technique.

Areas from which the electrograms are fragmented and prolonged are a likely setting for the development of reentrant arrhythmias. The concentration of such activity along the septal border of the aneurysm may be related to the high density of Purkinje fibers in the area. Alternatively, the septum may exhibit prolonged, fragmented activity because of the coexistence of viable and nonviable tissue in this region of good collateral circulation.

The persistence of areas of markedly abnormal electrical activity after aneurysmectomy may explain the poor record of this operation in curing malignant ventricular arrhythmias. Our findings may explain the success of encircling endocardial ventriculotomy and endocardial resection operations directed at the endocardial border zone.

References

DETERMINANTS OF VENTRICULAR TACHYCARDIA


ship to cycle length and site of origin. (abstr) Am J Cardiol 47: 497, 1981


Determinants of ventricular tachycardia in patients with ventricular aneurysms: results of intraoperative epicardial and endocardial mapping.

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Circulation. 1982;65:856-861
doi: 10.1161/01.CIR.65.5.856

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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