Body Surface Detection of Delayed Depolarizations in Patients with Recurrent Ventricular Tachycardia and Left Ventricular Aneurysm

JOHN J. ROZANSKI, M.D., DAVID MORTARA, PH.D., ROBERT J. MYERBURG, M.D., AND AGUSTIN CASTELLANOS, M.D.

SUMMARY In eight patients with chronic ventricular tachycardia and left ventricular aneurysms, we detected delayed ECG wave forms after the QRS complex from the body surface using a high-resolution ECG recorder, amplification and signal averaging. Delayed wave-form activity (D wave) extended a mean of 70 msec beyond the termination of the QRS complex. This delayed activity frequently extended to the limit of the recording window, and may thus continue throughout much of diastole. Antiarrhythmic agents never abolished the delayed activity; however, it was abolished by aneurysmectomy in four patients. Ventricular tachycardia did not recur after surgery in the four patients during a mean follow-up of 1 year. The D wave was not found in eight control patients who had chronic recurrent ventricular tachycardia nor in 11 of 12 who had aneurysms alone. The surface D wave can be readily and reproducibly detected by high-resolution electrocardiography and appears to be specific for patients with left ventricular aneurysms who also have chronic recurrent ventricular tachycardia. This delayed wave-form activity has been noted during catheter and surgical endocardial and epicardial mapping. It may represent persistence of the cardiac impulse in islands of myocardium and may be a manifestation of the delayed and fractionated activity, noted by previous investigators.

HIGH-RESOLUTION electrocardiography has been in existence for over 8 years as pioneered by three research groups: Berbari et al.,1,4 Flowers et al.,4,9 and Stopczyk et al.5,12 However, technical limitations prevented widespread clinical application of signal averaging and amplification. Recent advances in electronics have made high-resolution recordings clinically feasible.5,13

In 1978, Berbari et al.14 detected discrete multiphasic wave forms appearing during the ST segment of a surface-averaged lead in experimental canine myocardial infarction. They postulated that these waves represented areas of myocardium that showed marked delay in activation. This activity derived from a very small total mass compared with the entire ventricular mass, thus explaining why such delayed activity had been undetectable by routine standard-gain surface electrocardiography.14 By appropriate noise filtration, amplification and signal averaging, these low-level signals, which were frequent in the microvolt range, could be recorded. These techniques were applied to the same canine model in the same laboratory in which El-Sherif et al.15,16 found that delayed and fractionated activity extended beyond the T wave and was associated with the onset of ventricular arrhythmias. Although similar slow, fragmented and delayed activity has been observed in experimental animals,17,18 not until the endocardial mapping studies of Josephson et al.19-22 and Horowitz et al.23 in patients with recurrent ventricular tachycardia (VT)
and left ventricular aneurysms was delayed fractionated activity documented in man in the subendocardial layers along the margins of the aneurysm. These investigators determined that endocardial mapping was superior to epicardial mapping because epicardial breakthrough of activation is a late event that may occur at some distance from the site of origin of the arrhythmias. By careful pre- and intraoperative endocardial mapping, they convincingly showed that areas of delayed depolarization could be detected during sinus rhythm and localized along the endocardial border zone of the aneurysm. They noted continuous diastolic electrical activity preceding the onset of sustained VT. Recently, Fontaine et al., Klein et al., and Waldo and Kaiser pioneered the epicardial detection and mapping of delayed activity in animals and in humans prone to VT. The limitations of epicardial mapping make the meaning of their data controversial, but markedly delayed activity was nevertheless consistently found. The preliminary report of Klein et al. confirms the presence of both endocardial and epicardial delayed activity in man. In theory, such delayed activity should be detectable non-invasively at the body surface.

In the present study we used a special high-resolution ECG to record delayed wave-form activity in patients with recurrent VT and left ventricular aneurysms.

**Methods**

The study population consisted of eight patients who had left ventricular aneurysms and chronic recurrent VT, eight patients who had chronic VT and no evidence of left ventricular aneurysm, and 12 patients who had left ventricular aneurysms and no VT (table 1). The diagnosis of left ventricular aneurysm was defined as the presence of wall segment dyskinesia during left ventricular angiography. Four of the eight patients who had left ventricular aneurysms and chronic recurrent VT underwent surgical aneurysmectomy. These four patients had no further recurrence of chronic VT during a mean follow-up of 1 year.

All patients had high-resolution ECGs recorded in the control state in normal sinus rhythm without antiarrhythmic medication for at least 72 hours. Recording during VT was not feasible in the context of this study. Subsequent recordings were performed after administration of the following antiarrhythmic medications in patients with D waves: lidocaine (200-mg i.v. bolus), procainamide, quinidine sulfate, and encainide (one patient) administered orally after therapeutic serum levels were achieved. The four patients who underwent aneurysmectomy were recorded 1–4 hours, 1 day and 1 week after operation, and then at intervals of 1 to several months.

The ECG recording technique included placement of four silver/silver chloride ECG electrodes (Marquette Electronics) in the following locations: the routine V$_4$ position, low V$_4$ (6 cm below V$_4$) and high V$_4$ (6 cm above V$_4$). A fourth indifferent electrode was positioned at the left subclavicular region. Preparation of the skin and contact placement of the electrodes were as described by the manufacturer. Signals were

<table>
<thead>
<tr>
<th>Patients with aneurysms and VT</th>
<th>Age (years)</th>
<th>Sex</th>
<th>VT rate (beats/min)</th>
<th>Delayed depolarization (D wave)</th>
<th>Effect of antiarrhythmic agents on D wave</th>
<th>After aneurysmectomy</th>
<th>VT recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>M</td>
<td>150</td>
<td>Yes</td>
<td>None</td>
<td>D wave abolished</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>M</td>
<td>145</td>
<td>Yes</td>
<td>None</td>
<td>D wave abolished</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>M</td>
<td>140</td>
<td>Yes</td>
<td>None</td>
<td>D wave abolished</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>59</td>
<td>M</td>
<td>152</td>
<td>Yes</td>
<td>Slight decrease of D-wave amplitude (3 μV) but not duration</td>
<td>D wave abolished</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>M</td>
<td>160</td>
<td>Yes</td>
<td>None</td>
<td>N/A</td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>M</td>
<td>180</td>
<td>Yes</td>
<td>No recording Off antiarrhythmics</td>
<td>N/A</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>55</td>
<td>M</td>
<td>140</td>
<td>Yes</td>
<td>No recording Off antiarrhythmics</td>
<td>N/A</td>
<td>Yes</td>
</tr>
<tr>
<td>8</td>
<td>59</td>
<td>M</td>
<td>150</td>
<td>Yes</td>
<td>No recording Off antiarrhythmics</td>
<td>N/A</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Controls</th>
<th>VT without aneurysm (n = 8)</th>
<th>Aneurysm without VT (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>53</td>
<td>59</td>
</tr>
<tr>
<td></td>
<td>(mean)</td>
<td>(mean)</td>
</tr>
<tr>
<td>VT rate (beats/min)</td>
<td>180</td>
<td>1</td>
</tr>
<tr>
<td>Delayed depolarization (D wave)</td>
<td>No recording</td>
<td>None</td>
</tr>
<tr>
<td>Effect of antiarrhythmic agents on D wave</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>After aneurysmectomy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT recurrence</td>
<td>No VT</td>
<td>No</td>
</tr>
</tbody>
</table>

Abbreviation: VT = ventricular tachycardia.
recorded and processed using a commercially available microprocessor-augmented ECG cart (Marquette Electronics) suitable for portable bedside recording.

The sampling rate was 2000 samples/sec. Output filters consisted of three-pole Butterworth low-pass filter at 300 Hz and high-pass filters at 0, 20, 40, and 80 Hz. For illustrative purposes all recordings in this paper were selected with the 80-Hz cutoff. Memory was provided by a Motorola 6800 microprocessor. The input signal range was ±5 mV and the gain was 1 × 10^5. Temporal stability of the trigger point within the cardiac cycle is less than 500 μsec. The trigger or fiducial point is based on the earliest onset of QRS activity.13 Paper speeds were 100, 200 or 400 mm/sec.

Data output consisted of six simultaneous leads (three bipolar and three augmented). These leads represent vectors at 30° increments in the plane defined by the three active electrodes. The position of the leads on the thorax is determined by the anticipated vector of the signal of interest. For example, the locations V1, V2, and V3 are used for His bundle recordings.13 The lead array described earlier was arbitrarily chosen to encompass the aneurysm and to avoid areas of chest wall movement. Most of the illustrations shown have a vector orientation similar to a standard V4 chest lead. Alternatively, one might approximate the orientation with bipolar electrodes placed at the V2 and 6 cm vertically below V6. The normal gain tracings included in the illustrations can be used to aid understanding of the orientation of that particular lead.

The hard copy data format consisted of ink printout of six leads, each having the averaged signal represented at three different gains and four different frequency bands. In each patient, 512–1024 cycles were averaged.

Conventional invasive electrophysiologic studies were performed in all patients with VT.25 In addition, limited left ventricular mapping was performed in the four patients who subsequently underwent aneurysmectomy. This consisted of attempts to localize the site of the tachycardia by recording the site of earliest activation using the methods of Josephson et al.19–21 and Horowitz et al.22 Recordings were displayed on a multichannel oscilloscopic photographic recorder (Electronics for Medicine VR 16) and recorded at paper speeds of 100–250 mm/sec using band-filtration settings ranging from 20–500 Hz to 400–500 Hz. A Medtronic 5325 pulse generator was used for programmed stimulation.

One patient underwent epicardial and endocardial mapping during open heart surgery using routine techniques.28 Standard tripolar hand-held probes were used (Electrode Catheter Corp.). Bipolar signals were displayed as above using a variety of filtration band settings.

Results

Table 1 is a summary of the clinical data and results of the recording studies. In all eight patients with recurrent VT and no evidence of left ventricular aneurysm by left ventricular angiography, no depolarizations after the QRS complex were recorded in any leads. In contrast, all eight patients with both left ventricular aneurysms and chronic recurrent VT had well-defined delayed wave forms (D waves) similar to that shown in figure 1, measuring 4 ± 3 μV (± SD) and extending a mean of 70 msec after termination of ventricular activity in the simultaneous normal-gain QRS. In two patients, delayed activity extended into diastole up to the limit of the recording window, which was 250 msec after QRS onset.

The presence of delayed wave forms was independent of frequency filtration, because in a given patient they were present on all recordings made at 20–300, 40–300, and 80–300 Hz. Although the precise amplitude and morphology of the D-wave activity varied slightly from lead to lead, and from day to day in the same lead, in cases where it occurred, D-wave activity was present in all leads and was reproducible. Conversely, when the D wave was eliminated, for example, by aneurysmectomy, it was absent in all leads at all gains and at all filtrations. Of the 12 patients with isolated left ventricular aneurysms and no chronic recurrent VT, only one patient had a delayed depolarization, which measured less than 3 μV. Of 98 other patients (not included in this study) who underwent determination of the noninvasive His bundle electrogram, delayed depolarization beyond 30 msec was found in only one additional patient, a 16-year-old girl with transposition of the great vessels. She had un-

Figure 1. Simultaneous high-resolution recording (A) and normal-gain ECG (B) during sinus rhythm in patient 7, who had left ventricular hypertrophy. Paper speed is 200 mm/sec. The tracing in panel A was filtered at 80–300 Hz. Corresponding atrial activity (A and P) and ventricular activity (V and QRS) are labeled. A delayed potential (D) occurred 50 msec after termination of QRS activity. D-wave amplitude is 2.5 μV. No corresponding wave-form activity is present in the elevated ST segment of the normal-gain tracing in panel B. Five hundred twelve cycles were averaged for both A and B. Lead orientation was V5, and a D wave was present in all six leads.
dergone three cardiac surgical procedures and had chronic ventricular arrhythmias but no known episodes of chronic recurrent VT.

In all instances, the D wave and its duration were reproducible. However, the exact morphology of the high-gain P wave, PR segment, QRS and D wave varied from day to day, secondary to even very slight variations in electrode placement. Unless all electrodes are positioned exactly in the same locations within 1 or 2 mm, the precise high-gain morphology or “fingerprint” of the tracing will vary slightly. The slight inevitable variability of the trigger or fiducial point mentioned above also contributed to the differences noted on different days. The effect of anti-arrhythmic medications on the morphology of the various waves is not known. Figure 2 shows how slight changes in D-wave morphology occurred when the electrodes were left in place and the only intervention was the i.v. administration of 200 mg of lidocaine 15 minutes previously. Very slight but definite differences in the morphology of all of the complexes and segments are present (compare figs. 2A and C). In our experience, if simple repeat recording is done without any intervention, the complexes and segments are identical. The PR segment is known to vary spontaneously due to differences in autonomic tone, and therefore the morphology of the P and PR segment can vary (fig. 2E). The most remarkable feature in figure 2 is that while lidocaine had very little, if any, effect on the D wave (figs. 2A and C), aneurysmectomy completely abolished the wave (arrow, fig. 2E).

The differences in the P wave and PR segment are due to the above factors as well as to inevitable unintentional slight differences in electrode position post-operatively. Differences in the QRS of both the high-gain (fig. 2E) and standard-gain (fig. 2F) tracings are usually secondary to the ventriculotomy. Nevertheless, delayed activity was essentially eliminated in all leads. No intervention other than aneurysmectomy appreciably suppressed or eliminated the delayed activity as compared with control recordings (arrows, fig. 2E and 3C), although the precise morphology may be affected (figs. 3A and B).

Electrophysiologic study in the four surgical patients with left ventricular aneurysms and chronic recurrent VT was performed primarily for induction testing of VT and evaluation of antiarrhythmic drug effects. Only very limited left ventricular endocardial mapping was performed. No delayed wave forms were recorded in any of the four patients. However, this lack of finding delayed potentials by limited catheter endocardial mapping of the left ventricle during normal sinus rhythm was to be expected. In one patient, however, epicardial and endocardial mapping were performed during surgery. Delayed wave-form activity was recorded in the endocardial border zone area superior and adjacent to the aneurysm (fig. 4D). The timing of the delayed activity compares favorably with that of the surface recording of the D wave (figs. 3 and 4). Whether the surface D wave represents activity from this endocardial site or from some far distant or epicardial site cannot be unequivocally stated.

**Figure 2.** Three sets of high-resolution ECGs during sinus rhythm from patient 1. Paper speed 200 mm/sec. Panels A, C, E were filtered at 80-300 Hz; 1024 cycles were averaged in all panels. The paired high-resolution and standard-gain ECGs (A and B, C and D, E and F) are simultaneous. Atrial (A), ventricular (V) and delayed (D) activity are indicated. Delayed activity was present in all six lead orientations. The electrode pair was placed at V2 and 6 cm below V2. Panels A and B are control tracings. Panels C and D were recorded 15 minutes after a 200-mg i.v. bolus of lidocaine HCl. The electrodes were not moved. The normal-gain ECG in panels B and D are identical, but the high gain ECG in panels A and C show subtle differences in the exact morphology of the ventricular and delayed wave forms; these differences are due solely to lidocaine. Panels E and F show a recording performed 1 week later, after aneurysmectomy. Delayed activity was absent (arrow) in all six leads at all filtrations. Differences in morphology of the P wave, PR segment and QRS are discussed in the text. The change in morphology of the QRS in the normal-gain tracing (panel F) is due to the ventriculotomy and aneurysmectomy.
A modification of the encircling endocardial ventriculotomy of Fontaine et al.23-25 was performed and included transection beyond this area of delayed activation. This modification28 was the addition of four transverse cuts perpendicular to the axis of the encircling incision to better interrupt any possible reentry pathways. In this patient, the endocardial and epicardial surface of the aneurysm proper were devoid of electrical activity. Subsequent epicardial mapping after ventricular closure and rewarming failed to show delayed wave-form activity in the new border zone area. Thus, endocardial, epicardial and surface delayed wave-form activity were all documented in one patient immediately before resection and were abolished immediately after resection.

**Discussion**

The recurrent sustained VT associated with ventricular aneurysms has many consistent features that make this syndrome in certain respects a "model" for study, and much information has been accumulated. Our study confirms that delayed depolarizations as found by others during endocardial and epicardial mapping can be reproducibly detected from the body surface using a high-resolution ECG. Such delay may occur due to delayed epicardial breakthrough and may not represent activity from actual endocardial sites, but it nevertheless appears to be specific for patients who have both recurrent VT and aneurysms and may be a marker that can be readily detected during normal sinus rhythm. Such delayed potentials probably reflect a major intramuscular conduction defect, in which the cardiac impulse remains active long after the QRS terminates on the routine surface ECG. As such we may be witnessing during sinus rhythm a **forme fruste** of ventricular reentry; that is, slow conduction persisting into diastole. Although one might postulate that some of the higher-amplitude activity of

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**Figure 3.** Recordings of lead V5 in patient 4 at a paper speed of 200 mm/sec. (A) Control. (B) Two days later after quinidine sulfate, 400 mg every 6 hours (blood level 4.2 μg/ml). Differences in precise morphology of the complexes are explained in the text. (C) After aneurysmectomy 5 days later, the PR interval is shortened, which results in different morphology of P and PR. Delayed activity (D), present in all leads during control and after lidocaine, procaniamide (Pronestyl), disopyramide phosphate (Norpace) and quinidine, is now absent (arrow). It was absent in all six leads and at all filtrations. A = atrial activity; V = ventricular activity.

**Figure 4.** Endocardial mapping of the aneurysmal border zone in patient 4. (A) Modified ECG lead V5, (B) An intramyocardial reference electrogram recorded from the left ventricular apex filtered at 300-500 Hz. (C) An endocardial reference wave form from a site 2 cm from that in panel D (filtration 20-300 Hz). (D) Recording from the superior aspect of the aneurysmal border zone endocardial surface, showing marked delay of depolarization activity. The timing of the delayed activity is comparable to that in figure 3.
the D wave is due to late depolarization of myocardium far from the so-called arrhythmogenic site, such as the epicardium, this delay is nevertheless abnormal. It is not generally seen, in our experience with over 100 patients, in any other conditions such as right or left bundle branch block, fascicular block, or any routine intraventricular conduction delay patterns not associated with left ventricular aneurysms. Several patients who had aneurysms but no D waves or VT had severe intraventricular conduction delays indicative of significant scarring and interruption of the normal patterns of intraventricular conduction, yet had no significant D-wave activity. Normal-gain ECG patterns from this group cannot be distinguished from those in the group with VT.

Further evidence that suggests that D-wave activity is related to the tendency to develop recurrent sustained VT is the disappearance of both VT and D-wave activity after aneurysmectomy. We caution that our series consists of only four patients and must await further confirmation. Alternative hypotheses abound to explain the disappearance of D waves after surgery. It may be simply an effect coincidental to surgery, rather than the actual removal of the delay-causing mass of tissue.

The possibility that the D wave represents mechanically induced artifact can be excluded for two reasons: Patients with similarly severe left ventricular aneurysms and no recurrent ventricular tachycardia did not have the D wave, and the delayed depolarization could be recorded even when electrodes were positioned in the right precordium and V6 position, so that precordial mechanical impulses could be completely excluded. The reason for the electrode position described in the methods section was precisely to avoid mechanical artifacts.

The comparability of serial tracings done on different days must be interpreted with caution. With high-gain averaged signals, factors such as antiarrhythmic drugs, surgical resection, autonomic tone, variability of the trigger point, and even very slight differences in electrodes positioning will cause appreciable differences in the morphology of the complexes. Because of the many variables involved and the complexity of data processing and filtration, slight differences in morphology of the complexes is inevitable on serial tracings done days apart. This does not invalidate the methods, because it is the timing of the wave forms rather than their precise morphology that is important. An analogy can be made with invasive electrograms, in which timing is more important than morphology, which is frequently not reproducible.

The remarkable reproducibility of the D wave morphology on serial recordings (figs. 2A and C) when the above variables are minimized argues strongly that this wave represents organized delayed activity that is reproducible and that therefore follows a definite anatomic, electrophysiologic substrate.

In conclusion, by the techniques of amplification and signal averaging, electrical signals with a fixed relationship to the QRS complex can be enhanced with respect to background random electrical noise. Delayed depolarizations so detected in patients with the combination of VT and ventricular aneurysms are similar to those detected by previous investigators and represent a new ECG surface wave form readily detectable at the bedside. Our observations support the hypothesis that D waves are related to the presence of intraventricular delayed activity. Further, endocardial mapping in patient 4 of our study (fig. 4) seems to substantiate both the D wave detected on the body surface and its anatomic relationship to the aneurysmal border zone.

Acknowledgment

We acknowledge the wise counsel of Eliseo Perez-Stable, M.D., and the administrative assistance of Gus Godoy. Thanks also to Blanche Garrett and Betty Gold for superior secretarial assistance.

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Body surface detection of delayed depolarizations in patients with recurrent ventricular tachycardia and left ventricular aneurysm.
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Circulation. 1981;63:1172-1178
doi: 10.1161/01.CIR.63.5.1172

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