Electrocardiographic Recognition of the Epicardial Origin of Ventricular Tachycardias

Antonio Berruezo, MD; Lluis Mont, MD; Santiago Nava, MD; Enrique Chueca, MD; Eduardo Bartholomay, MD; Josep Brugada, MD, PhD

Background—Some ventricular tachycardias (VTs) originating from the epicardium are not suitable for endocardial radiofrequency ablation and require an epicardial approach. The aim of this study was to define the ECG characteristics that may identify an epicardial origin of VTs.

Methods and Results—We analyzed the 12-lead ECG recordings during epicardial and endocardial left ventricular pacing in 9 patients to verify the hypothesis that the epicardial origin of the ventricular activation widens the initial part of the QRS complex. Then, we analyzed the ECG pattern in 14 VTs successfully ablated from the epicardium after a failed endocardial approach (group A), in 27 VTs successfully ablated from the endocardium (group B), and in 28 additional VTs that could not be ablated from the endocardium (group C). Four distinct intervals of ventricular activation were defined and measured: (1) the pseudodelta wave, (2) the intrinsicoid deflection time in V2, (3) the shortest RS complex, and (4) the QRS complex. VTs from groups A and C showed a significantly longer pseudodelta wave, intrinsicoid deflection time, and RS complex duration compared with VTs of group B. There was no difference between groups A and C. A pseudodelta wave of ≥34 ms has a sensitivity of 83% and a specificity of 95%, an intrinsicoid deflection time of ≥85 ms has a sensitivity of 87% and a specificity of 90%, and an RS complex duration of ≥121 ms has a sensitivity of 76% and a specificity of 85% in identifying an epicardial origin of the VTs.

Conclusions—ECG suggests VTs originating from the epicardium and those with an unsuccessful radiofrequency ablation from the endocardium. (Circulation. 2004;109:1842-1847.)

Key Words: electrocardiography ■ pericardium ■ tachycardia, ventricular

Endocardial radiofrequency (RF) ablation is the common approach for ablation of monomorphic sustained ventricular tachycardias (VTs). However, the success rate of the procedure depends on the underlying heart disease and the location of the circuit. Efficacy of RF ablation of VTs ranges from 45% to 76% in patients with ischemic heart disease1-3 to ~100% in patients with fascicular or bundle-branch block reentry tachycardias4,5 in which a crucial part of the reentrant circuit of the tachycardia is the conduction system, located in the subendocardium.

The reasons for failure of endocardial ablation are often difficult to ascertain. One proposed explanation is that the reentry circuits may be located deep in the subendocardium or in the epicardium, making them inaccessible to current ablation techniques.7,8 Epicardial mapping of arrhythmia foci or circuits has been attempted in open chest surgery9,10 and with RF ablation catheters.11,12 These studies suggest that the slow conduction zone may be located subepicardially in up to 33% of patients.

We recently described the feasibility and safety of nonsurgical transthoracic epicardial RF ablation of VTs in patients with ischemic heart disease, incessant VT, and depressed ventricular ejection fraction in whom classic endocardial ablation had failed. The observed success rate was 85%.13 These results suggest that the use of an epicardial approach may improve the success rate of VT ablation in case of an epicardial origin of the arrhythmia.

In 1990, Burgess et al14 and later Oster et al15 demonstrated the ability of ECG imaging to estimate the depth of intramyocardial electrophysiological events in experimental studies in dogs and their relation to the velocity of propagation of activation (more rapid along the endocardium, with the aid of the Purkinje system). However, no ECG criteria to predict an epicardial origin of VTs have been described to date. On the other hand, no reliable criteria have been proposed to identify these tachycardias from mapping data restricted to the endocardial surface.16

The aim of the present study was to analyze the ECG pattern of VTs ablated from the epicardium and to define the ECG characteristics that may predict an epicardial origin.

Previously, we tested the hypothesis that the epicardial origin of the ventricular activation may increase the duration of the initial part of the QRS complex on the surface ECG with respect to an endocardial origin.
Methods

Paced Patients
Nine consecutive patients in NYHA functional class III/IV (mean age, 55 ± 19 years; left ventricular ejection fraction, 24 ± 7%) in whom a cardiac resynchronization therapy device with possible right and left ventricular independent pacing (Guidant Contak Renewal 2) was implanted underwent electrophysiological study perioperatively. No patient was on antiarrhythmic drugs during the study. The pacing protocol was performed after written informed consent was obtained. The epicardium of the left ventricular free wall was paced at a cycle length of 600 ms through the epicardial electrode of the device placed at the distal venous system. Afterward, the endocardial aspect of the left ventricular free wall was paced at a similar site and at the same cycle length with a diagnostic deflectable 6F catheter. Multiple fluoroscopic positions were used to obtain optimal position of the endocardial catheter, ideally <10 mm away from the tip of the epicardial catheter (Figure 1). The stimuli were delivered in a bipolar configuration at twice the diastolic threshold and 2-ms duration. Data from the 12 surface ECG leads and endocardial signals were recorded concurrently.

VT Patients
We analyzed the 12-lead surface ECGs of 14 clinical VTs with right bundle-branch block (RBBB) morphology (group A) that were unsuccessfully ablated from the endocardium despite the use of standard and special catheters (8-mm tip in 6 patients, endocardial irrigated tip in 3 patients) and effectively ablated from the epicardium with a nonsurgical transthoracic epicardial approach. In addition, we analyzed a second group of 25 consecutive patients with 27 clinical VTs with an RBBB morphology successfully ablated from the endocardium (group B). VTs with left bundle-branch block pattern in V1 (dominant S wave) were excluded because these tachycardias commonly originate from the interventricular septum and are probably not suitable for epicardial access. Finally, another group of 24 patients with 28 VTs with an RBBB morphology that could not be ablated from the endocardium in whom no epicardial approach was used was retrospectively included for analysis (group C).

Figure 1 shows the epicardial and endocardial locations of successful ablation sites for each patient in groups A and B. The target sites were identified initially by activation mapping. Pacing maneuvers to identify the mechanism were done, including reset, entrainment, concealed entrainment, and pace mapping. In all cases, the nature of the VT was suggested to be reentrant. Point applications were used for ablation. Most cases required a few close applications to terminate the VT.

Electrophysiological Study
All patients gave written informed consent for the procedures. The electrophysiological study was performed in patients in the fasting state who were mildly sedated with 10 mg diazepam. Before the study, 9, 17, and 16 patients were receiving oral amiodarone in groups A, B, and C, respectively. All other antiarrhythmic drugs were stopped at least 5 half-lives before the procedure. The technique used for epicardial mapping and ablation has been already reported. The equipment used for recording was an EP-TRACER (Cardio Tek bv). The 12 surface ECG leads, distal and proximal bipolar electrograms of the epicardial ablation catheter, and endocardial signals from a diagnostic catheter positioned at the right ventricular apex were displayed.

Analysis of the ECG
For the purposes of this study, 4 distinct intervals of ventricular activation were defined. The intervals were measured using digitized tracings with calipers allowing 1-ms resolution at a screen velocity of 100 mm/s and with the signal amplified at 10 mm/mV. Two observers blinded to the pacing site or successful ablation location made all measurements. In case of discordance of >5 ms in a given measure between observers, a third observer was used.

Pseudodelta Wave
The pseudodelta (pseudo Δ) was measured from the earliest ventricular activation (from the stimulation artifact in paced patients) to the earliest fast deflection in any precordial lead. In paced patients, we measured also the transmural activation time, from the stimulation artifact in the epicardium to the largest rapid deflection recorded from the distal bipolar electrogram of the endocardial surface catheter (Figure 3).

Intrinsicoid Deflection Time
The intrinsicoid deflection time was defined as the interval measured from the earliest ventricular activation (from the stimulation artifact in paced patients) to the peak of the R wave in V2.

Shortest RS Complex
The shortest RS complex was defined as the interval measured from the earliest ventricular activation (from the stimulation artifact in paced patients) to the nadir of the first S wave in any precordial lead.

QRS Complex Duration
The QRS complex duration was defined as the interval measured from the earliest ventricular activation (from the stimulation artifact in paced patients) to the offset of QRS in the precordial leads.
Statistical Analysis
Continuous data are expressed as mean±SD. Means were compared by use of Student’s *t* test for paired data and 1-way ANOVA when appropriate. Receiver-operating characteristic curves were made to obtain the best values of sensitivity and specificity. Statistical analysis was performed with the SPSS 10.0 statistical package. A value of *P*≤0.05 was considered significant.

Results

Epicardial Pacing–Dependent Widening of QRS
In this group of patients, the baseline QRS complex duration averaged 194±19 ms, attributable to left bundle-branch block. The sites of endocardial and epicardial stimulation during the pacing protocol were as follows: lateral (basal or medial) in 6 patients, lateroapical in 2 patients, and anterior in 1 patient. The mean threshold value at 2-ms duration of stimuli was 1.4±1.2 V for epicardial pacing and 0.5±0.1 V for endocardial pacing. Epicardial pacing showed longest activation times (Table 1) compared with endocardial pacing despite a very similar morphology of the QRS complex (Figure 3). There was a small but significant increase in epicardially paced QRS complex duration (241±39 versus 266±25 ms; 12±13% increment; *P*=0.012). However, epicardial pacing led to a marked prolongation of the shortest RS interval (138±37 versus 183±37 ms; *P*<0.001), intrinsicoid deflection time in V2 (98±33 versus 138±38 ms; *P*<0.001), and pseudo Δ wave (20±14 versus 65±13 ms; *P*<0.001).

Of note, the absolute increase in milliseconds was similar (45±14 ms for the shortest RS interval, 40±24 ms for the intrinsicoid deflection in V2, 45±6 ms for the pseudo Δ wave).

| TABLE 1. Characteristics and Results of the Measured Intervals in Paced Patients |
|---------------------------------|-----------------|-----------------|
| Pacing                          | P               | Widening, ms    | Widening, %   |
| Endocardial                     | Epicardial      |                 |               |
| n                               | 9               | 9               |
| Age, y                          | 55±19           | 55±19           |
| Ischemic, n                     | 6               | 6               |
| LVEF, %                         | 24±7            | 24±7            |
| LVEDD, mm                       | 80±23           | 80±23           |
| Wall thickness, mm              | 10.4±1.5        | 10.4±1.5        |
| Pseudo Δ wave, ms               | 20±14           | 20±14           |
| ID time in V2, ms               | 98±33           | 138±38          |
| Shortest RS interval, ms        | 138±37          | 183±37          |
| QRS complex duration, ms        | 241±39          | 266±25          |

LVEF indicates left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; and ID, intrinsicoid deflection.
TABLE 2. Main Characteristics of Patients in the 3 Groups of VT

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>69±7</td>
<td>65±16</td>
<td>69±8</td>
</tr>
<tr>
<td>Cardiopathy (ischemic/dilated), %</td>
<td>90/10</td>
<td>64/36</td>
<td>65/35</td>
</tr>
<tr>
<td>Infarct location (anterior/inferior), %</td>
<td>33/66</td>
<td>48/52</td>
<td>42/58</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>30±8</td>
<td>36±9</td>
<td>36±10</td>
</tr>
<tr>
<td>VT cycle length, ms</td>
<td>430±76</td>
<td>416±88</td>
<td>409±80</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction.

ANOVA: group A vs B and group C vs B, P<0.05; group A vs C, P=NS. \(\chi^2\) test: \(P=NS\).

The duration of the pseudo \(\Delta\) wave when the ventricle was paced from the epicardium matched the transmural activation time measured from the stimulation artifact to the local activation time on the distal bipolar electrogram from the endocardial diagnostic catheter (Figure 3).

Intervals in VTs

Table 2 gives the main characteristics of patients in the 3 VT groups. Data on intervals measured on the 3 groups (a total of 69 VTs) are given in Table 3. Epicardially ablated VTs with RBBB morphology (group A) showed a mean duration of pseudo \(\Delta\) wave of 43±9 ms. However, the mean duration of pseudo \(\Delta\) wave in the 27 VTs with RBBB morphology successfully ablated from the endocardial aspect (group B) was 16±9 ms (P<0.05). In the 28 VTs with RBBB morphology that could not be ablated from the endocardium (group C), the mean duration of pseudo \(\Delta\) wave (42±9 ms) was similar to that of group A, and both groups differed from group B (group A versus B and C versus B, P<0.05; group A versus C, P=NS).

The same differences between groups were observed when the intrinsicoid deflection time in V2 and the shortest RS interval were compared. The mean duration was 97±21 and 149±39 ms in group A, 65±15 and 91±20 ms in group B, and 98±14 ms and 132±30 ms in group C, respectively (group A versus B and C versus B, P<0.05; group A versus C; P=NS).

The differences between all 3 groups with respect to the QRS interval duration were small but statistically significant: 217±24 ms from group A, 174±37 ms from group B, and 195±27 ms from group C (group A versus B and C versus B, P<0.05; group A versus C, P=NS). Thus, epicardially ablated VTs (group A) showed longer intervals compared with endocardially ablated VTs (group B), (P<0.05), and there were no differences between groups A and C. An example of intervals in VTs from each group is shown in Figure 4.

Receiver-operating characteristic curves showed that a mean duration of the pseudo \(\Delta\) wave of ≥34 ms has a sensitivity of 83% and a specificity of 95%, the intrinsicoid deflection time of ≥85 ms has a sensitivity of 87% and a specificity of 90%, and an RS complex duration of ≥121 ms has a sensitivity of 76% and a specificity of 85% to predict the failure of endocardial RF ablation of VTs.

Discussion

Paced Patients

This study demonstrates that epicardial pacing produces a widening of the QRS complex as a result of the initial part (pseudo \(\Delta\) wave) compared with endocardial pacing at a similar location of the left ventricular free wall.

Interestingly, the transmural activation time matches the duration of the visible pseudo \(\Delta\) wave in the 12-lead surface ECG. Its interval was ≈45 ms in these patients. In fact, the visible early fast deflection on the simultaneous 12-lead surface ECG corresponds with the local activation time measured on the distal bipolar electrogram of the diagnostic catheter positioned at the endocardial surface. It is possible that the duration of the pseudo \(\Delta\) wave in another cohort of patients (absence of cardiomyopathy, distinct wall thickness) may be different.

The differences between endocardially and epicardially stimulated QRS are probably due to fast depolarization of the ventricles along the specialized conducting system when the stimulus is delivered in the endocardium, resulting in a narrow QRS on the surface ECG and the absence of a pseudo \(\Delta\) wave. When initial activation occurs in the epicardium, the intramyocardial delay of conduction produces a slurred initial part of the QRS complex (pseudo \(\Delta\) wave).

Some discordance exists between the absolute value of the measured intervals in paced patients with respect to the VT patients. Some factors may play a role in this discordance. First, measurements have been performed from the spike, so the latency interval between the stimulus artifact and the onset of the QRS complex is included. Second, the paced patients are not comparable with the VT patients because the former showed an extremely enlarged cavity size and a very damaged conduction system. These factors may modify the intervals. Third, the onset of activation came indubitably from the epicardium in paced patients, whereas in group A patients (and inversely in group B), it came from near the epicardium, far from the endocardium, or deep into the left ventricular wall. For all these reasons, the values obtained in the paced patients are used only to demonstrate the hypothesis that epicardial pacing enlarges the initial duration of the QRS in each individual patient but cannot be used in absolute terms to validate the data to identify an epicardial origin of a VT.

VT Patients

Several factors affect the QRS width of VTs, ie, muscle mass or nonuniform anisotropy. Also, the His-Purkinje system plays an important role in impulse propagation in the VTs, and in occasional patients, investigators have been able to document morphologically similar tachycardias with differ-
ent QRS widths. In such cases, the QRS width appears to be related to His-Purkinje activation and its participation in global left ventricular depolarization.18

As far as we know, the extent to which intramural conduction delay contributes to QRS widening in the 12-lead ECG recording in VTs has never been assessed. Thus, this study also demonstrated how it is possible to recognize an epicardial origin of left ventricular free wall VTs by the ECG analysis of QRS activation times.

In VTs successfully ablated from the endocardium, there is not a visible pseudo Δ wave on the ECG or the pseudo Δ wave has a short duration (16±9 ms). In the same way, both the intrinsicoid deflection in V2 and the shortest RS complex have a shortest duration compared with epicardially ablated VTs. On the other hand, VTs that could not be ablated from the endocardium (group A and C) had the longest activation times. In these VTs, a duration of pseudo Δ wave of ≥34 ms and duration of intrinsicoid deflection in V2 of >85 ms provided a high sensitivity (83% and 87%, respectively) and specificity (95% and 90%, respectively).

Nonsurgical transthoracic epicardial ablation of VTs is a safe and feasible procedure, and only a small percentage of complications have been encountered. The high success rate of this approach (85% of attempted VTs in a recent series) is probably due to the selection of cases that could not be ablated previously from the endocardium.14 The proposed criteria have a high sensitivity and specificity in the analyzed group and provide a diagnostic tool that may help to decide the use of this approach as a first intention (aortic stenosis or prosthetic valves, recent intraventricular thrombus, high risk of bleeding, or severe artery disease). Furthermore, a VT selection based on the proposed criteria may increase the success of the epicardial ablation.

**Study Limitations**

It is possible that in VTs with a very short cycle length, the repolarization of the previous beat may cover the onset of the next QRS. However, such tachycardias are often not suitable for ablation because of poor hemodynamic tolerance. To minimize subjectivity in the measurements, simultaneous 12-lead ECG recordings were used to facilitate recognition of onset of intervals. However, sometimes it is difficult to measure the duration of the pseudo Δ wave; in these cases, measurement of the intrinsicoid deflection in V2 and of the shortest RS interval is much easier and more reliable.

The proposed criteria are valid only for VTs with an RBBB morphology. Prediction of the epicardial origin of VTs with left bundle-branch block morphology is of minor interest because a high percentage of these tachycardias originate from the interventricular septum and are probably not suitable for ablation from the epicardium.

One final limitation is the fact that irrigated tip catheters were not available in most patients with endocardial ablation failure retrospectively analyzed. Theoretically, some of these VTs could have been ablated from the endocardium with deeper lesions. However, the intervals measured in this group (group C) are the same as those of VTs ablated from the epicardium and unsuccessfully ablated from the endocardium.
and provide the gold standard to characterize and compare the epicardial origin.

Conclusions

The epicardial origin of activation produces a widening of the initial part of the QRS, visible in the conventional surface ECG as a pseudo $\Delta$ wave. Analysis of the conventional ECG suggests VTs originating from the epicardium and those with an unsuccessful ablation from the endocardium with a high sensitivity and specificity.

References

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