Idiopathic Left Bundle-Branch Block–Shaped Ventricular Tachycardia May Originate Above the Pulmonary Valve

Carl Timmermans, MD; Luz-Maria Rodriguez, MD; Harry J.G.M. Crijns, MD; Antoon F.M. Moorman, PhD; Hein J.J. Wellens, MD

Background—Idiopathic left bundle-branch block (LBBB)–like ventricular tachycardia (VT) is considered to originate in the right ventricular outflow tract (RVOT) or from the aortic root. Both regions are derived from the embryonic outflow tract. We now report that also the pulmonary trunk can give rise to VT, suggesting a common etiology of these tachycardias.

Methods and Results—We studied 6 patients with symptomatic idiopathic LBBB-VT using electrophysiological mapping techniques. The VT origin was determined by analyzing the electrograms and the angiographic location of the catheter tip at the successful ablation site or the earliest activation site. Eight VTs were induced. Two VTs, with a mean earliest endocardial activation time of −5 and −20 ms and optimal pace mapping, were successfully ablated in the RVOT. In the remaining 6 VTs, the earliest activation site was found in the pulmonary artery, and, at this site, a sharp potential was present −38±12 ms before the QRS complex in 5 VTs. The mean earliest endocardial activation time in the RVOT was −1±2 ms. Ablation was attempted in 5 of 6 VTs and resulted in an acutely successful procedure. After a mean follow-up of 10±4 months, 1 of 5 patients had a recurrence.

Conclusions—The site of origin of idiopathic LBBB-VT can be in the root of the pulmonary artery, suggesting a myocardial connection from this site to the RVOT. If no good criteria for ablation in the RVOT are found, detailed mapping of the pulmonary artery should be performed. (Circulation. 2003;108:1960-1967.)

Key Words: ablation • arrhythmia • tachycardia

In recent years, much attention has been given to cardiac arrhythmias originating in vessels connected to the heart. The pulmonary veins, superior caval vein, coronary sinus, and ligament of Marshall may contain arrhythmogenic myocardial tissue. When connected to the heart, atrial fibrillation or atrial tachycardia may ensue. Radio-frequency (RF) catheter disconnection of these structures has proven to be a successful therapy for these arrhythmias.1–4 It has also been shown that idiopathic ventricular tachycardia (VT) may originate from the aortic root.5,6 These findings made us decide to carefully map activation in the root of the pulmonary artery in patients presenting with an ECG labeled as idiopathic right ventricular outflow tract (RVOT) VT.

Methods

Study Population

Between March 1992 and May 2001, 72 patients (40 men, mean age 42±12 years) with idiopathic left bundle-branch block (LBBB)-shaped VT were referred to our institution for electrophysiological evaluation and RF ablation. The study population consisted of 6 patients with idiopathic monomorphic LBBB-shaped VT admitted for electrophysiological evaluation and RF ablation since May 2001. The diagnosis of idiopathic VT was made after an extensive workup (Table 1). There were 3 men and 3 women aged 19 to 56 years (mean, 38±13). One of these patients (patient A) has been described previously.7 Physical examination, 12-lead ECG during sinus rhythm, and chest radiograph were normal in all patients. Three patients presented with sustained monomorphic VT and 3 with recurrent runs of nonsustained monomorphic VT. All but 1 patient had been undergoing sotalol treatment. Patient B was treated with amiodarone. The abnormalities detected by MRI in 3 patients (Table 1) were similar to those reported by other authors.8 Three of the patients had a previous acutely failed RF ablation in the RVOT. Two of these previously failed procedures were performed in our institution, and the earliest endocardial activation during VT started −10 ms (patient D) and 0 ms (patient E) before the QRS complex. One patient (patient D) with a fast VT and an unsuccessful previous RF ablation had received an implantable defibrillator. The hospital ethics committee approved the study.

Electrophysiological Study

After obtaining informed consent, the patients were studied in the fasting state without sedation. Antiarrhythmic drugs were discontinued for at least 5 half-lives before the electrophysiological study. The patient treated with amiodarone (patient B) discontinued this medication 9 weeks before the study. Under local anesthesia, quadripolar catheters were positioned via the femoral...
RF catheter ablation was performed during VT for 60 to 90 seconds with a preset temperature of 70°C and a power limit of 50 W. A successful ablation was defined as the noninducibility of VT, with and without isoproterenol administration, during at least 30 minutes after ablation. Right ventricular angiographies in the right and left anterior oblique projections were made with the ablation catheter positioned at the successful application site. All patients had 24-hour Holter monitoring after the procedure. An exercise test was performed the second day after ablation. After ablation, all patients received oral anticoagulants for 3 months but no antiarrhythmic drugs. The follow-up consisted of visits to the outpatient clinic, the first visit at 6 weeks and thereafter every 3 months.

**Results**

Eight different monomorphic VTs were induced in the 6 patients. Table 2 summarizes the electrocardiographic and electrophysiological characteristics and ablation parameters of the VTs.

**Electrocardiographic Characteristics**

All 8 induced VTs had an LBBB-shaped morphology and were clinically documented, except for the VT with a very late R/S transition in the precordial leads in patient A. In 2 patients (patients A and D), 4 different VT morphologies were induced, of which 2 were successfully ablated from the RVOT. The remaining 6 VTs, shown in Figure 1, originated above the pulmonary valve. The frontal QRS axis was intermediate in 2 VTs and vertical in 3 VTs. There was 1 late R/S transition in precordial lead V6.

### Table 1. Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td>F</td>
<td>F</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>F</td>
</tr>
<tr>
<td><strong>Age, y</strong></td>
<td>39</td>
<td>44</td>
<td>19</td>
<td>30</td>
<td>38</td>
<td>56</td>
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<tr>
<td><strong>Symptoms</strong></td>
<td>Palpitations</td>
<td>Palpitations</td>
<td>Palpitations, chest discomfort</td>
<td>Palpitations, syncope</td>
<td>Palpitations, chest discomfort</td>
<td>Palpitations, dizziness</td>
</tr>
<tr>
<td><strong>Symptom duration</strong></td>
<td>1 month</td>
<td>1 year 8 months</td>
<td>3 years 4 months</td>
<td>8 years</td>
<td>12 years 3 months</td>
<td>5 years 5 months</td>
</tr>
<tr>
<td><strong>Clinical arrhythmia</strong></td>
<td>SMVT</td>
<td>RMVT</td>
<td>SMVT</td>
<td>SMVT</td>
<td>RMVT</td>
<td>RMVT</td>
</tr>
<tr>
<td><strong>No. of SMVT episodes</strong></td>
<td>1</td>
<td>...</td>
<td>21</td>
<td>11*</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td><strong>Previous AAD</strong></td>
<td>Sotalol</td>
<td>Sotalol, bisoprolol, amiodarone</td>
<td>Sotalol</td>
<td>Propranolol, flecainide, amiodarone, sotalol</td>
<td>Flecainide, amiodarone, sotalol</td>
<td>Sotalol</td>
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<tr>
<td><strong>SAECG</strong></td>
<td>NL</td>
<td>NL</td>
<td>NL</td>
<td>NL</td>
<td>NL</td>
<td>NL</td>
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<tr>
<td><strong>Exercise testing</strong></td>
<td>SMVT</td>
<td>SMVT</td>
<td>No VT</td>
<td>RMVT</td>
<td>SMVT</td>
<td>RMVT</td>
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<tr>
<td><strong>Echo (LVEF %)</strong></td>
<td>NL (66)</td>
<td>NL (61)</td>
<td>NL (53)</td>
<td>NL (60)</td>
<td>NL (60)</td>
<td>NL (60)</td>
</tr>
<tr>
<td><strong>Coronary, right, left ventricular angiography</strong></td>
<td>NL</td>
<td>NL</td>
<td>ND</td>
<td>NL</td>
<td>ND</td>
<td>NL</td>
</tr>
<tr>
<td><strong>MRT</strong></td>
<td>NL</td>
<td>Focal thinning</td>
<td>Dyskinesis mid</td>
<td>NL</td>
<td>Focal thinning</td>
<td>NL</td>
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<tr>
<td><strong>Previous ablation attempt</strong></td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

AAD indicates antiarrhythmic drugs; Echo, echocardiography; LVEF, left ventricular ejection fraction; ND, not done; NL, normal; RMVT, repetitive monomorphic VT; RVFW, right ventricular free wall; SAECG, signal averaged electrocardiography; and SMVT, sustained monomorphic VT.

*Including 4 VT episodes for which 5 shocks were delivered with the implantable defibrillator.
Electrophysiological Characteristics
Four of the 6 VTs originating above the pulmonary valve were induced during isoproterenol administration, 3 of them in combination with programmed electrical stimulation. Three VTs were induced by ventricular extrastimuli and 2 VTs by rapid ventricular pacing. The induced VT was sustained in 5 patients and nonsustained in patient E.

Mapping and Ablation Procedure
Two of the 8 VTs (patients A and D) were successfully ablated in the RVOT. Their earliest endocardial activation time, −5 and −20 ms, was found on the septal and midseptal side of the RVOT. Pacing at these sites showed an optimal pace map. The 2 VTs terminated and were no longer inducible after 1 (90 seconds, 65°C, 50 W) and 10 (36 ± 21 seconds [10 to 62], 58 ± 9°C [48 to 70], 29 ± 7 W [29 to 45]) applications, respectively.

The remaining 6 VTs originated above the pulmonary valve. Electrical activation started before the VT QRS complex, with a mean earliest activation time of −38 ± 12 ms (−24 to −60) (Figure 2). In these patients, the mean earliest endocardial activation time in the RVOT was

Table 2. Electrocardiographic and Electrophysiological Characteristics and Ablation Parameters

<table>
<thead>
<tr>
<th>Patient</th>
<th>VT Morphology</th>
<th>QRS Width, ms</th>
<th>R/S &gt;1</th>
<th>VT-CL, ms</th>
<th>VT Induction</th>
<th>Pace Map, QRS_p/QRS_VT</th>
<th>EAT, ms*</th>
<th>RF, n</th>
<th>RF (or EAT) site</th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>LBBB, RA</td>
<td>160</td>
<td>V4</td>
<td>310</td>
<td>VES</td>
<td>12/12</td>
<td>−5</td>
<td>1</td>
<td>RVOT-S</td>
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<tr>
<td></td>
<td>LBBB, VA</td>
<td>120</td>
<td>V6</td>
<td>290</td>
<td>Iso + VES</td>
<td>NC</td>
<td>−35 [−3]</td>
<td>6</td>
<td>PA</td>
</tr>
<tr>
<td>B</td>
<td>LBBB, RA</td>
<td>160</td>
<td>V4</td>
<td>300</td>
<td>VES</td>
<td>NC</td>
<td>−40 [0]</td>
<td>1</td>
<td>PA</td>
</tr>
<tr>
<td>C</td>
<td>LBBB, RA</td>
<td>120</td>
<td>V4</td>
<td>220</td>
<td>Iso + RVP</td>
<td>12/12</td>
<td>−35 [0]</td>
<td>4</td>
<td>PV</td>
</tr>
<tr>
<td>D</td>
<td>LBBB, VA</td>
<td>120</td>
<td>V4</td>
<td>320</td>
<td>VES</td>
<td>12/12</td>
<td>−20</td>
<td>10</td>
<td>PV</td>
</tr>
<tr>
<td>E</td>
<td>LBBB, IA</td>
<td>130</td>
<td>V5</td>
<td>250</td>
<td>VES</td>
<td>12/12</td>
<td>−60 [0]</td>
<td>5</td>
<td>PV</td>
</tr>
<tr>
<td>F</td>
<td>LBBB, VA</td>
<td>120</td>
<td>V4</td>
<td>310</td>
<td>Iso</td>
<td>12/12</td>
<td>−24 [0]</td>
<td>12</td>
<td>PV</td>
</tr>
</tbody>
</table>

*Value in brackets denotes EAT obtained in RVOT.
CL indicates cycle length; EAT, earliest (endocardial) activation time; IA, intermediate axis; Iso, isoproterenol infusion; n, No. of radiofrequency applications; NC, no capture; PA, main stem of pulmonary artery; PV, ≤1 cm above the pulmonary valve; QRS_p/QRS_VT, match between QRS morphology during pace mapping and clinical VT, with the number representing the number of ECG leads with identical QRS morphology; RA, right axis; RF, radiofrequency ablation; R/S >1, R/S transition >1 in precordial ECG leads; RF site, site of successful RF; RVOT-MS, midseptal side of right ventricular outflow tract; RVOT-S, septal side of RVOT; RVP, rapid ventricular pacing; VA, vertical axis; and VES, ventricular extrastimuli.

Figure 1. Twelve-lead electrocardiograms of 6 patients (A through F) with a LBBB-like VT originating in the pulmonary artery.
Figure 2. The 12-lead ECG and endocardial recordings during LBBB-like VT originating in the pulmonary artery of patients A through C (panel I) and patients D through E (panel II). Note a sharp potential (patients A, B, and D through E) or a fragmented electrogram (patient C) preceding the QRS complex in the endocardial recording from the RF ablation catheter at the site of the earliest activation time. RV denotes right ventricle.
In 5 of the 6 VTs, a sharp potential, preceding the QRS complex, was observed at the site of the earliest activation time (patients A, B, and D through F), whereas the electrogram at the successful ablation site of patient C was fragmented during tachycardia (Figure 2). A sharp potential or fractionated electrogram was not present in the 2 VTs ablated from the RVOT. Interestingly, the sharp potential was also recorded late after the ventricular electrogram during sinus rhythm in all 5 patients before ablation and disappeared after ablation (Figure 3). An optimal pace map was obtained in 3 of 6 VTs, and 11 of 12 leads between the clinical VT and the pace map were similar in patient E (Figure 4). Despite pacing with high output and long pulse width, no capture was obtained in the 2 patients in whom the VT originated more distal in the pulmonary artery (patients A and B). RF ablation was only attempted in the patients with sustained VT. All 5 VTs could be terminated and were no longer inducible after a median number of 5 (range, 1 to 12) applications. The mean application duration was 54±20 seconds (20 to 90), with a mean temperature of 53±10°C (30 to 70) and a mean power of 40±12 W (6 to 56). The site of successful ablation was several centimeters in the root of the pulmonary artery in 2 patients (patients A and B) (Figure 5) and within 1 cm above the pulmonary valve in the remaining 3 patients (patients C, D, and F) (Figure 6). In patient E, in whom no ablation was attempted, the earliest activation during nonsustained VT was recorded within 1 cm above the pulmonary valve. There were no procedural complications.

**Follow-Up**

In the 5 ablated patients, no VT occurred during the exercise test performed 2 days after ablation. All patients had postprocedural echocardiography without evidence of pulmonary valve dysfunction or thrombus formation above the valve. After a mean follow-up of 10±4 months (4 to 16), 1 of the 5 patients had a VT recurrence. The VT recurrence occurred in
the patient with an implantable defibrillator (patient D) and was treated with device therapy. The VT of the patient in whom no ablation was performed did not recur under treatment with sotalol.

**Discussion**

For the treatment of patients with idiopathic, LBBB-like, inferior-axis VT, RF energy is usually applied to the RVOT. Pace mapping and, to a lesser extent, activation mapping are used to select the appropriate endocardial site for catheter ablation.\(^9\),\(^10\) In patients who cannot be ablated from the right ventricle, specific characteristics of the QRS complex during VT may point to an origin in the left ventricular outflow tract or aortic root.\(^5\),\(^6\),\(^11\) Although, until now, no anatomically well-defined substrate for idiopathic RVOT-VT has been found, this study indicates that in some patients with a structurally normal heart and a LBBB-like VT, the site of origin is in myocardial tissue in or around the pulmonary artery. In all but 1 patient, at the site of successful ablation a sharp potential was recorded, preceding the onset of the QRS during VT by 24 to 60 ms. This sharp potential was also recorded late after a tiny ventricular electrogram during sinus rhythm before ablation and disappeared after ablation. Similar to the spikes found in the pulmonary veins in patients with atrial fibrillation, these sharp potentials indicate the presence of muscular tissue around the pulmonary artery.\(^1\) In 5

![Figure 4. An example of an optimal match between the clinically recorded 12-lead ECG of an idiopathic LBBB-shaped VT and the ECG recorded during pacing above the pulmonary valve with a slightly faster rate than the clinical VT.](image-url)
patients, a VT was successfully ablated in the pulmonary artery, indicating that this ectopic muscular tissue is connected with the right ventricle. In 3 patients, the successful ablation site was located closely above the pulmonary valve, but in the other 2 patients RF ablation several centimeters above the valve terminated the VT.

How to explain the presence of myocardial tissue in or around the pulmonary artery? Both the embryonic avian and mammalian outflow tract, as well as the outflow tract in adult primitive fish (called conus) and amphibians (called bulbus cordis), is surrounded by myocardium. It has been demonstrated that this myocardium supports semilunar valve function in adult fish and frogs and substitutes this function in embryonic chicken hearts. In contrast to the myocardium of the atrial and ventricular chambers, this myocardium retains its embryonic features, ie, slow propagation of the depolarizing impulse owing to the poor intercellular coupling of the cardiac muscle cells. This characteristic also is a prerequisite for nodal function. In normal

Figure 5. Right (left panel) and left (right panel) oblique view of a right ventriculogram showing the position of the RF catheter at the site of successful ablation in the root of the pulmonary artery of patient B. The pulmonary valve is indicated by arrows. RV indicates a catheter in the right ventricle.

Figure 6. Right (left panel) and left (right panel) oblique view of a right ventriculogram showing the position of the RF catheter at the site of successful ablation above the pulmonary valve, indicated by arrows, of patient C. A second catheter is positioned in RVOT. PA indicates pulmonary artery.
mammalian development, the proximal outflow tract myocardium becomes ventricularized by incorporation into the right ventricle, whereas the myocardium of the distal outflow tract disappears. It can be envisioned that if this retraction of myocardium does not disappear completely, remnants persist that may provide the substrate for these tachycardias.

Previous studies using catheter ablation in the RVOT to treat idiopathic LBBB-VT reported excellent outcomes.\textsuperscript{9,11,13} Our findings suggest that in more LBBB-VT patients, ectopic impulse formation starts in the pulmonary artery followed by conduction over a myocardial sleeve to the right ventricle. Interruption of conduction by catheter ablation either at its beginning (in the pulmonary artery) or at its insertion into the RVOT may cure these patients. This is similar to interruption of conduction in an accessory atrioventricular pathway, either at the atrial or the ventricular end. Although specific electrocardiographic patterns during VT may indicate a left ventricular outflow tract or aortic root site of origin in some patients with idiopathic LBBB-like VT, the electrocardiographic characteristics of the VT of these 6 patients did not allow to distinguish between an RVOT and a pulmonary artery origin of the VT.\textsuperscript{5,6,11} Although the mechanism of the VT originating from the pulmonary artery was not systematically studied, all VTs were induced during exercise testing or isoproterenol administration, indicating catecholamine sensitivity.

Echography in the RVOT and the pulmonary artery could be of help in the precise localization of the site of origin of the VT. We used right ventricular angiography that allowed us to visualize in all patients the exact site in relation to the pulmonary artery and pulmonary valve leaflets, where a successful catheter ablation was performed.

A better understanding of the site of origin of LBBB-like VT will improve our ability to successfully treat these patients with catheter ablation. The findings of our study suggest that in every patient with a structurally normal heart and a LBBB-like VT morphology, mapping of the RVOT should be first performed. At this moment in time, we suggest that when no early activation time during VT and no optimal pace mapping are found in the RVOT, detailed mapping of the pulmonary artery including the pulmonary valve should be performed. In patients with a previously unsuccessful RVOT ablation, a repeat procedure with careful mapping of the pulmonary artery is advised.

References

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