Transient Entrainment of Bundle-Branch Reentry by Atrial and Ventricular Stimulation

Elucidation of the Tachycardia Mechanism Through Analysis of the Surface ECG

Jose L. Merino, MD; Rafael Peinado, MD; Ignacio Fernández-Lozano, MD; Nicolas Sobrino, MD; Jose A. Sobrino, MD

**Background**—Different responses to entrainment have been reported in relation to the pacing site of a variety of tachycardias. However, transient entrainment of bundle-branch reentrant tachycardia (BBRT) has not been investigated systematically.

**Methods and Results**—We attempted entrainment of 13 BBRTs in 9 patients by pacing first the right ventricle and then the right atrium. The initial pacing cycle length (CL) was 10 ms faster than the tachycardia CL. Subsequent pacing sequences were performed with 5- to 10-ms CL decrements until tachycardia termination or loss of postatropine 1:1 AV conduction. Both full ventricular-paced and AV-conducted QRS complex references were obtained during sinus rhythm pacing from the same sites and with similar CL as during entrainment. Transient entrainment was achieved by ventricular and atrial stimulation in 11 and 8 tachycardias, respectively. Constant fusion was always present during entrainment by ventricular stimulation. There was no change in the QRS complex (orthodromically concealed fusion) during entrainment by atrial stimulation in 6 of 6 tachycardias with left bundle-branch block morphology and in 1 of 2 tachycardias with right bundle-branch block morphology.

**Conclusions**—BBRT, especially if it has a left bundle-branch block morphology, can be differentiated from other wide-QRS-complex tachycardia mechanisms through analysis of the ECGs recorded during tachycardia entrainment by atrial and ventricular stimulation. This diagnostic approach may be especially useful when it is difficult to record a stable or sufficiently sized His bundle electrogram or when spontaneous changes in the ventricular CL precede similar changes in the His bundle CL. (**Circulation. 1999;100:1784-1790.**)

Key Words: ablation ■ bundle-branch block ■ electrophysiology ■ entrainment ■ tachycardia

Bundle-branch reentry (BBR) is an uncommon but clinically relevant ventricular tachycardia (VT) mechanism, because catheter ablation can abolish the condition. It was previously thought to occur typically in patients with dilated cardiomyopathy but rarely in other clinical settings.1 However, more recently, BBR has commonly been found to be responsible for VT among patients with myotonic dystrophy2-3 or those who have undergone valve replacement.4 In addition, several reports have shown that BBR may be responsible for VT despite the observation of ventricular cycle length (CL) oscillations preceding rather than following His bundle CL oscillations,2,5 which is considered the most important diagnostic criterion. These findings suggest that BBR is often unrecognized and underline the need for additional diagnostic criteria.

Different responses have been described during transient entrainment of a variety of supraventricular6-8 and ventricular9-14 tachycardias. However, no systematic studies have been made of the responses to entrainment of BBR tachycardia (BBRT). The purpose of the present work was to study the responses to transient entrainment of BBRT by atrial and ventricular stimulation in an attempt to find new criteria to differentiate BBR from other wide-QRS-complex tachycardia mechanisms. The following hypotheses were tested (Figure 1): first, entrainment of BBRT by atrial pacing may result in the concealment of the antidromic wave front within the His-Purkinje system and in a QRS complex that displays neither fusion nor change; and second, entrainment of BBRT by ventricular pacing may result in the collision of the antidromic and orthodromic wave fronts within unprotected ventricular myocardium and in a QRS complex that displays manifest fusion.

**Methods**

**Patients**

Nine consecutive patients with spontaneous wide-QRS-complex tachycardia were included in the study. All of them had at least 1 inducible wide-QRS-complex tachycardia that met the diagnostic criteria of BBRT (see below). The patients’ characteristics are shown...
in Table 1. A more detailed description of 5 of these patients has been given previously.\(^2\)

**Electrophysiological Study**

Cardiac invasive electrophysiological studies were performed in accordance with the institutional guidelines concerning informed consent and after all antiarrhythmic drugs had been withdrawn. Three quadripolar catheters were introduced percutaneously through the right femoral vein and placed in the high right atrium (HRA), His bundle area, and right ventricle. Three or 4 surface ECG traces and 3 or 4 bipolar intracardiac recordings, filtered between 30 and 500 Hz, were displayed simultaneously on a digital multichannel oscilloscope (LabSystem, Bard Electrophysiology or Midas, PPG Biomedical Systems). All 12 ECG traces and intracardiac electrograms were stored on an optical disk for later reproduction at 25, 50, 100, or 200 mm/s. Programmed ventricular extrastimulation was performed at not less than 2 constant basic CLs from the right ventricular apex (RVA) and the right ventricular outflow tract (RVOT) with \(\leq 3\) extrastimuli. The stimulation protocol was repeated under isoproterenol infusion when no tachycardia was induced at baseline.

BBRT diagnosis was established according to previously published criteria (criteria A):\(^1\)  
(1) QRS complex morphology with typical bundle-branch–block pattern consistent with ventricular depolarization through the appropriate bundle branch; (2) AV dissociation during tachycardia; (3) exclusion of a tachycardia from supraventricular origin by established criteria; (4) prolonged HV interval during sinus rhythm; (5) a stable His or bundle-branch electrogram preceding each ventricular activation during tachycardia with an HV interval longer than, equal to, or <10 ms shorter than that recorded during sinus rhythm; (6) spontaneous changes in the bundle potential CL preceding similar changes in the ventricular CL; and (7) suppression of inducibility after right or left bundle-branch ablation, both at baseline and during isoproterenol infusion.

Because BBR has been found to be the tachycardia mechanism despite criterion 6 not being demonstrated,\(^2,3\) BBRT diagnosis was also established when all the following criteria (criteria B) were fulfilled: (1) all criteria outlined in criteria A were fulfilled except for criterion 6, that is, spontaneous changes in the bundle potential CL followed rather than preceded similar changes in the ventricular CL; (2) \(\pm 1\) additional BBRT morphology was also inducible and fulfilled all criteria given in criteria A; (3) the difference in tachycardia CL was \(\leq 30\) ms compared with those of the other induced BBRTs; (4) no myocardial VT, either sustained or nonsustained, was inducible; (5) the patient had no structural heart disease; and (6) the inducibility of all tachycardias was suppressed after bundle-branch ablation.

**Entrainment Pacing Protocol**

We attempted tachycardia transient entrainment by pacing first from the RVA and later from the HRA. Pacing was performed continuously during tachycardia for \(\geq 5\) seconds with a CL 10 ms shorter than that of the tachycardia. Subsequent entrainment sequences were performed with 5- to 10-ms decreases in the pacing CL until tachycardia termination or loss of 1:1 AV conduction. Entrainment was also attempted by pacing from the RVOT when there were similar QRS-complex configurations during tachycardia and during

### Table 1. Main Clinical and Electrophysiological Findings in 9 Consecutive Patients With BBRT Successfully Treated by Radiofrequency Catheter Ablation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>SHD</th>
<th>PR, ms</th>
<th>QRS</th>
<th>HV, ms</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>38</td>
<td>M</td>
<td>None*</td>
<td>210</td>
<td>IVCD</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>M</td>
<td>None</td>
<td>215</td>
<td>AFB</td>
<td>85</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>M</td>
<td>Ao Proth</td>
<td>200</td>
<td>RBBB</td>
<td>120</td>
</tr>
<tr>
<td>4</td>
<td>77</td>
<td>M</td>
<td>DCM</td>
<td>180</td>
<td>RBBB</td>
<td>80</td>
</tr>
<tr>
<td>5</td>
<td>37</td>
<td>F</td>
<td>None*</td>
<td>220</td>
<td>IVCD</td>
<td>65</td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>M</td>
<td>None*</td>
<td>240</td>
<td>LBBB</td>
<td>80</td>
</tr>
<tr>
<td>7</td>
<td>43</td>
<td>M</td>
<td>LVD* (MD)</td>
<td>290</td>
<td>IVCD</td>
<td>110</td>
</tr>
<tr>
<td>8</td>
<td>49</td>
<td>M</td>
<td>None*</td>
<td>210</td>
<td>Normal</td>
<td>80</td>
</tr>
<tr>
<td>9</td>
<td>40</td>
<td>M</td>
<td>None</td>
<td>180</td>
<td>RBBB</td>
<td>65</td>
</tr>
</tbody>
</table>

SHD indicates type of structural heart disease; QRS, QRS-complex morphology; HV, His-to-ventricle interval during sinus rhythm; IVCD, infraventricular conduction defect; AFB, left anterior fascicular block; Ao Proth, aortic valve prosthesis; RBBB, right bundle-branch block; DCM, idiopathic dilated cardiomyopathy; LBBB, left bundle-branch block; LVD, left ventricular systolic dysfunction; and *, myotonic dystrophy.


pacing from the RVA during sinus rhythm. Two milligrams of atropine, ≤2 μg/min isoproterenol, or both were infused when there was no 1:1 AV conduction at the tachycardia CL.

After tachycardia termination, full ventricular-paced and full AV-conducted QRS complex references were obtained during sinus rhythm, by ventricular and atrial pacing, respectively, from the same sites and with similar CLs (±25 ms) as during entrainment.

Twelve-lead ECG traces were printed at 50 mm/s and 10 mm/mV, and QRS-complex differences were analyzed by 2 independent evaluators.

Definitions

**Transient entrainment** was defined as transient and constant acceleration to the pacing of all components necessary for the tachycardia continuation (ventricular and His-Purkinje activations in the case of BBRT) and with resumption of the tachycardia at its intrinsic rate once pacing is stopped.6,9,13,17 There is a fixed relationship between the pacing CL and the first return cycle.

**Transient entrainment with fusion** was defined as transient entrainment in which there is evidence of ECG interpreted as during entrainment by atrial pacing; ∆CL, difference between tachycardia and shortest entrainment CL; Vent Ent, ECG response to entrainment by atrial pacing; ∆CL, difference between tachycardia and shortest entrainment CL; Vent Ent, ECG response to entrainment by ventricular pacing; L, left; R, right; S, superior; I, inferior; OCF, orthodromically concealed fusion; MF, manifest fusion; and IFR, interfascicular reentry.

*Infusion requirement to obtain 1:1 AV conduction and tachycardia entrainment by atrial stimulation.
†Clinically documented.
‡Entrainment was not achieved because the tachycardia was nonsustained.
§Entrainment was not achieved because no 1:1 AV conduction was obtained at the tachycardia CL.

**Results**

Inducible Tachycardias

Eleven sustained tachycardias were inducible (Table 2). Except for tachycardia 3 of patient 8 and tachycardia 2 of patient 9, these tachycardias fulfilled all criteria A of BBRT. The former tachycardias displayed oscillations in the VV interval preceding those of the HH interval and fulfilled all criteria B of BBRT.

Achievement of Transient Entrainment

Entrainment of all 11 sustained tachycardias was achieved by ventricular stimulation and, except for the tachycardias of patients 6 and 7 and tachycardia 3 of patient 8, also by atrial stimulation (Figures 2 through 5). In patients 6 and 7, tachycardia was not entrained by atrial stimulation because...
no 1:1 AV conduction was obtained at the tachycardia CL. No attempts were made to entrain tachycardia 3 of patient 8 by atrial stimulation because the tachycardia was not reinduced after termination by ventricular stimulation. The return cycle after entrainment by ventricular stimulation showed a flat pattern in all entrained tachycardias with an entrainment zone of $30 \text{ ms}$ except for the tachycardia of patient 7, tachycardias 1 and 3 of patient 8, and tachycardia 1 of patient 9, which showed a mixed flat/increasing pattern ($31.0 \pm 5.5 \text{ ms}$ return-cycle difference).

The infusion of atropine (1 to 2 mg) was required to obtain 1:1 AV conduction and tachycardia entrainment by atrial stimulation in all tachycardias except tachycardia 1 of patient 8 and the tachycardia of patient 5, who was undergoing isoproterenol infusion when entrainment was attempted (Table 2). There were no complications related to the administration of atropine or isoproterenol.

### Surface ECG Analysis

The QRS complex exhibited manifest fusion during entrainment by ventricular stimulation of all sustained tachycardias (Figures 2 and 3). Differences between spontaneous tachycardia, entrained, and fully paced QRS complexes were readily visible in all cases when pacing was performed from the RVA. However, in patient 2, these differences were much better appreciated when pacing was performed from the RVOT (Figure 3).

Orthodromically concealed QRS complex fusion was observed during entrainment by atrial stimulation (Figures 2 through 4) of all entrained tachycardias except for tachycardia 2 of patient 8 (Figure 5). In this latter tachycardia, the QRS complex displayed manifest fusion during entrainment by atrial pacing. All entrained tachycardias, except for tachycardia 1 of patient 9, showed differences between the QRS complex recorded during tachycardia and that recorded during pacing from the HRA atrium with the same CL as during entrainment and while the patient was in sinus rhythm.

### Catheter Ablation

BBRT was successfully treated by bundle-branch ablation guided by the recording of a bundle-branch potential in all patients except for patients 3 and 9, in whom no right bundle-branch potential recording was achieved but successful ablation was accomplished in the right bundle-branch area.

### Discussion

Diagnosis of BBRT is most commonly established by the observation of a His bundle electrogram preceding each QRS complex by an interval equal to or longer than that observed during sinus rhythm and that shows spontaneous changes in its CL preceding those of the ventricular activation. Other criteria, such as tachycardia initiation or termination with a critical conduction delay or block within the His-Purkinje system, are also dependent on the recording of a His or bundle-branch electrogram. However, there are limitations to this approach that could lead to the failure to recognize BBRT. First, and as in our patients 6 and 7, catheter displacement during tachycardia often makes the recording of a quality, stable His bundle electrogram difficult. Second, and as in our patients 3 and 4, BBRT typically occurs in patients with a severely diseased conducting system, which is associated with the recording of a low-amplitude or smooth His bundle electrogram, particularly at short CLs (Figure 4). Third, spontaneous changes in the His electrogram CL preceding those of the ventricular activation are typically seen in AV nodal or hisian tachycardias and therefore are not useful for the differentiation of these tachycardias from BBRT. Finally, spontaneous changes in the His electrogram CL

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Figure 2. From left to right, surface ECGs of patients 1 (top) and 4 (bottom): (1) during pacing (hRA Pac) from HRA while the patient was in sinus rhythm; (2) during tachycardia entrainment (hRA Ent) by pacing from HRA; (3) during tachycardia (VT) after pacing cessation; (4) during tachycardia entrainment (RVA Ent) by pacing from RVA; and (5) during pacing (RVA Pac) from RVA while patient was in sinus rhythm. All ECGs were recorded at 50 mm/s and 10 mm/mV. Tachycardia and pacing CLs are shown in milliseconds. Note differences among all QRS complexes except for those recorded during tachycardia and during hRA Ent, which show an identical configuration (most apparent on leads V3 through V6).
Figure 3. Top. Pacing from RVOT both during sinus rhythm (top left) and during entrainment (top right) of BBRT of patient 2. Bottom. Entrainment of same tachycardia by HRA pacing. Note acceleration of all tachycardia components to pacing CL, preservation of QRS-complex morphology, and resumption of tachycardia on pacing cessation. Simultaneous 100-mm/s tracings are surface ECG leads and bipolar intracardiac recordings.

Figure 4. ECG of patient 4 recorded at 50 mm/s during tachycardia (VT) (top left), during tachycardia entrainment (Atrial Ent) by pacing from the atrium (top center), during atrial pacing (Atrial Pac) after tachycardia termination (top right), and during sinus rhythm (SR). Note sudden shortening (>100 ms) of stimulus to QRS interval and change of QRS complex from Atrial Ent to Atrial Pac after a nonconducted atrial stimulus that terminates the tachycardia. This patient showed a broad, low-amplitude His bundle electrogram during sinus rhythm (bottom right, 100 mm/s) that was lost during tachycardia (bottom left, 100 mm/s). However, a high clinical suspicion, together with the response to transient entrainment, led to the recording of a distal right bundle-branch electrogram. This electrogram showed CL oscillations preceding those of the ventricular activation.
following rather than preceding those of the ventricular activation do not exclude BBR, 2,5 because changes in the HV interval (due to conduction variations in the antegrade conducting bundle branch) result in VV-interval oscillations driving those of the HH interval, as in tachycardia 3 of our patient 8 and tachycardia 2 of our patient 9. The present study introduces new criteria for the diagnosis of BBRT without the need for a His bundle or bundle-branch electrogram.

**Differential Diagnosis**

Several mechanisms of wide-QRS-complex tachycardia have to be ruled out before a BBRT diagnosis can be established. Myocardial VT and AV nodal tachycardias should be particularly considered, because VA dissociation is almost always present in BBRT. 1

**Myocardial VT With Secondary His-Purkinje Activation**

In the present study, BBRT entrainment by ventricular stimulation exhibited manifest QRS-complex fusion in all. Therefore, the observation of this ECG response does not distinguish between BBRT and myocardial reentrant VT, because manifest fusion during entrainment by RVA stimulation is demonstrated in ≈67% of postinfarction VTs. 13 On the other hand, concealed fusion, either antidromic or orthodromic, during tachycardia entrainment by RVA stimulation makes BBR unlikely and favors myocardial reentry. In this case, failure to recognize fusion, due to a similar QRS complex during tachycardia and during pacing from the RVA while the patient is in sinus rhythm, should be excluded, and, if this is the case, entrainment by RVOT stimulation is warranted.

Manifest fusion during entrainment by atrial stimulation has been reported in up to 54% of myocardial VT. 10,12 Conversion to a supraventricular QRS-complex morphology or no 1:1 AV conduction at the tachycardia CL were seen in the remainder. In those studies, neither atropine or isoproterenol infusions were made to achieve 1:1 AV conduction at the shortest atrial pacing CL. In the present study, BBRT entrainment by atrial stimulation exhibited orthodromically concealed fusion in all tachycardias except for tachycardia 2 of patient 8, 1 of the only 2 tachycardias with a right bundle-branch block morphology. Therefore, the observation of orthodromically concealed fusion during entrainment of a wide-QRS-complex tachycardia by atrial stimulation rules out a pure myocardial reentry mechanism and favors BBR.

**AV Nodal and Hisian Tachycardias**

The presentation of AV nodal tachycardias, either reentrant or automatic, with VA dissociation has been reported. 20,21 These tachycardias, although uncommon, should be differentiated from BBRT, particularly if the QRS complex recorded during tachycardia is similar to that recorded during sinus rhythm, as in tachycardia 1 of our patient 3, the tachycardia of our patient 6, and tachycardia 1 of our patient 9.

Entrainment of AV nodal reentrant tachycardia by atrial7 and ventricular7,8 stimulation has been studied, and in theory, the
same responses should be observed during entrainment of hisian tachycardias, either of microreentrant or focal mechanism. Orthodromically and antidromically concealed fused waves of the QRS complex are typically seen during entrainment by atrial and ventricular stimulation, respectively. Therefore, entrainment by atrial stimulation is not useful to distinguish between BBRT and AV nodal or hisian tachycardias. On the other hand, the observation of manifest fusion during entrainment of a wide-QRS-complex tachycardia by ventricular stimulation rules out a pure AV nodal or hisian mechanism in favor of BBRT.

**Interfascicular Reentrant Tachycardia**

There are few reports of interfascicular reentrant tachycardia.15,19 None of them studied entrainment in this particular setting, and therefore, the responses to entrainment are speculative. However, from a theoretical point of view and similar to that shown in Figure 5, the impulse should initiate conduction in the right bundle branch earlier than in either of the 2 left hemifascicles during entrainment by atrial stimulation, and therefore, in the absence of right bundle-branch block at the tachycardia CL, a change of the tachycardia QRS complex should be expected. Nevertheless, this tachycardia may be distinguished from BBRT because it usually displays a markedly shorter HV interval than that recorded during sinus rhythm.

**Limitations**

There are several limitations with regard to the use of entrainment techniques to differentiate BBRT from other mechanisms of wide-QRS-complex tachycardia. First, the tachycardia should be sustained and tolerated to allow pacing maneuvers. Second, the coexistence of chronic atrial fibrillation prevents entrainment by atrial pacing. Finally, a poor 1:1 AV conduction, as in our patients 6 and 7, may preclude the achievement of tachycardia entrainment by atrial stimulation even after atropine infusion. Nevertheless, entrainment was achieved by ventricular and atrial stimulation in the majority of BBRTs in the present study despite these potential limitations.

**Conclusions**

Analysis of the ECG during entrainment of BBRT by nonsimultaneous atrial and ventricular stimulation can be used to differentiate this mechanism from other types of wide-QRS-complex tachycardia. Observation of manifest fusion during entrainment by ventricular pacing rules out an AV nodal or hisian mechanism and should be expected in BBRT. Observation of orthodromically concealed fusion during entrainment by atrial pacing rules out VT due to pure myocardial reentry and should be expected in BBRT. However, observation of manifest fusion during entrainment by atrial stimulation does not rule out BBRT when it has a right bundle-branch morphology, and an alternative diagnostic approach is warranted. Finally, this diagnostic approach should be considered especially useful when it is difficult to record a stable or sufficiently sized His bundle or bundle-branch electrogram during tachycardia, when AV nodal or hisian tachycardias are considered, or when spontaneous changes in the ventricular CL precede rather than follow similar changes in the His bundle or bundle-branch CL.
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