Bundle-Branch Reentry and the Postpacing Interval After Entrainment by Right Ventricular Apex Stimulation
A New Approach to Elucidate the Mechanism of Wide-QRS-Complex Tachycardia With Atrioventricular Dissociation

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Background—Diagnosis of bundle-branch reentry ventricular tachycardia (BBR-VT) by the standard approach is challenging, and this may lead to nonrecognition of this tachycardia mechanism. Because the postpacing interval (PPI) after entrainment has been correlated with the distance from the pacing site to the reentrant circuit, BBR-VT entrainment by pacing from the right ventricular apex (RVA) should result in a PPI similar to the tachycardia cycle length (TCL). This factor may differentiate BBR-VT from other mechanisms of wide-QRS-complex tachycardia with AV dissociation, such as myocardial reentrant VT (MR-VT) or AV nodal reentrant tachycardia (AVNRT), in which the circuit is usually located away from the RVA.

Methods and Results—Transient entrainment by RVA pacing was attempted in 18 consecutive BBR-VTs and finally achieved in 13. Results were compared with those found in 59 consecutive MR-VTs and 50 consecutive AVNRTs. The mean PPI2-TCL difference was significantly (P<0.0001) shorter in the BBR-VT group (9±6 ms) than in the MR-VT (109±64 ms) and the AVNRT (150±29 ms) groups. No BBR-VT showed a PPI2-TCL>30 ms (range 2 to 24 ms). Except for 2 MR-VTs, no MR-VT (range 21 to 211 ms) or AVNRT (range 100 to 215 ms) showed a PPI2-TCL>30 ms.

Conclusions—A PPI2-TCL>30 ms, after entrainment by RVA stimulation, makes BBR-VT unlikely. Conversely, a PPI2-TCL<30 ms is suggestive of BBR-VT but should lead to further investigation by use of conventional criteria. (Circulation. 2001;103:1102-1108.)

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

In contrast to other ventricular tachycardia (VT) mechanisms, bundle-branch reentry (BBR) is easily and effectively treated by radiofrequency ablation.1,2 Recognition or exclusion of BBR, however, can be challenging and time-consuming.3–6 Criteria based on the recording of the His bundle electrogram have several limitations, such as failure to record the His bundle electrogram during tachycardia because of catheter displacement or severe His-Purkinje disease,3–7 and BBR nonrecognition when oscillations in the VV interval precede those of the HH interval because of conduction variations in the antegrade, rather than the retrograde, conducting bundle branch.6,8 More recently, demonstration of both orthodromic concealed fusion (concealed fusion with tachycardia QRS-complex morphology preservation) and manifest fusion during entrainment by atrial and ventricular pacing, respectively, has been proposed as an alternative approach to BBR diagnosis.6 This approach, however, demands that the patient is not in atrial fibrillation and usually requires the infusion of atropine, isoproterenol, or both to avoid AV block at the atrial pacing cycle length (CL). Hence, suspicion or exclusion of BBR by a simple and time-saving method without requiring His bundle electrogram recording or drug infusion during tachycardia is warranted.

The first postpacing interval (PPI) after tachycardia entrainment has been correlated with the distance from the pacing site to the reentrant circuit.9,10 We postulated (Figure 1) that BBR-VT entrainment pacing from the right ventricular apex (RVA) should result in a PPI similar to the tachycardia CL (TCL) because the right bundle-branch distal insertion is in this anatomic area. This approach may differentiate BBR from other mechanisms of wide-QRS-complex tachycardia with AV dissociation,3,11 such as ventricular myocardial reentry (MR) or AV nodal reentry (AVNR), where the circuit is usually away from the RVA.

Methods

Patients
Fourteen consecutive patients with 18 inducible BBR-VTs were included in the study. Results in this group of patients were compared with those found in a group of 48 consecutive patients (60±14 years old, 41 male) with 59 inducible and sustained
or more tetrapolar catheters with 5-mm interelectrode spacing were introduced percutaneously through the right, left, or both femoral veins and placed in the His bundle area and right ventricle. Additional electrode catheters were placed in the high right atrium in some patients with no atrial fibrillation and into the coronary sinus in some patients with AVNRT. Three or 4 surface ECG traces and 2 to 7 bipolar intracardiac recordings, filtered between 30 and 500 Hz, were simultaneously displayed on a digital multichannel oscilloscope (LabSystem, Bard Electrophysiology or Midas, PPG Biomedical Systems). All 12 ECG traces and intracardiac electrograms were stored on optical disks for later reproduction. Bipolar pacing was performed from the distal electrodes while recording from the proximal pair. All measurements were made at 200 mm/s with electronic calipers with a discrimination of 1 ms.

Particular attention was paid to recording the His bundle electrogram during tachycardia or to entrain the tachycardia by atrial pacing to confirm or exclude a BBR mechanism. When necessary, additional catheters, such as a decapolar catheter with short interelectrode distance or a steerable catheter, were introduced to record the His bundle electrogram, a bundle-branch electrogram, or both.

**Tachycardia Mechanism Definitions**

VT was considered to be reentrant when it was induced and terminated by electrical stimulation. The observation of manifest fusion during transient entrainment by pacing from the RVA in all entrained BBR-VTs and in 29 entrained MR-VTs also supported reentry. MR-VT diagnosis was established when BBR was ruled out as the reentrant VT mechanism.3,4

BBR-VT diagnosis was established according to previously published criteria (criteria A): 1) QRS-complex morphology with typical BBB pattern consistent with ventricular depolarization through the appropriate bundle branch; 2) AV dissociation during tachycardia; 3) exclusion of a tachycardia from supraventricular origin; 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; and (6) spontaneous changes in the bundle potential CL preceding similar changes in the ventricular CL. Because BBR has been found to be the tachycardia mechanism despite criterion 6 not being demonstrated,3,4 BBR-VT diagnosis was also established when all the following criteria (criteria B) were fulfilled: (1) all 6 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) ≤1 additional BBR-VT morphologies were also inducible and fulfilled all criteria A; (3) the difference in tachycardia CL was ≥30 ms compared with those of the other induced BBR-VTs; (4) no MR-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. Finally, observation of orthodromic concealed fusion (concealed fusion with tachycardia QRS-complex morphology preservation) during entrainment by pacing from the atrium was an additional criterion sufficient but not mandatory to distinguish BBR-VT from MR-VT.6

Suppression of inducibility after right or, in patient 6, left bundle-branch ablation, both at baseline and during isoproterenol infusion, was achieved in all except 2 entrained BBR-VTs. Bundle-branch ablation was not performed in these 2 BBR-VTs or in 2 nonentrained BBR-VTs because BBR-VT had never been clinically documented, it was well tolerated and difficult to induce, and defibrillator implantation (patients 10, 11, and 14) or successful catheter ablation (patient 13) of the clinical tachycardia was performed.

Interfascicular tachycardia was distinguished from the 4 BBR-VTs with an RBBB configuration by fascicular electrograms and because the former typically displays a markedly shorter HV interval (>10 ms) than that recorded during sinus rhythm.

AVNRT diagnosis was established according to previously published standard criteria.7

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**Electrophysiological Study**

Cardiac invasive electrophysiological studies were performed in accordance with institutional guidelines after informed consent. Two

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**Figure 1.** Schematic of main components of His-Purkinje system and depolarizing wave fronts (arrows) during postpacing cycle after BBR-VT, MR-VT, and AVNRT entrainment by RVA stimulation. Last paced impulse (asterisk) is conducted from RVA pacing site to circuit, then impulse is conducted orthodromically, completes a revolution, and finally is conducted back from circuit to RVA. Therefore, if pacing site is close to circuit, as RVA is in BBR, PPI is similar to TCL. Conversely, when circuit is remote from pacing site, as RVA is in most MR-VTs and AVNRTs, PPI is significantly longer than TCL.
Main Clinical and Electrophysiological Findings in 14 Consecutive Patients With 18 BBR-VTs in Which Entrainment Was Attempted by Pacing From the RVA

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SHD indicates structural heart disease type; QRS Str., intraventricular conduction defect during sinus rhythm; HV in., sinus rhythm His-to-ventricle interval; HV ex., tachycardia His-to-ventricle interval; Clinical, clinically documented tachycardia; QRS VT, tachycardia BBB configuration; aQRS VT, tachycardia QRS-complex axis; TCL, TCL after postspacing stabilization; ECL, entrainment CL; PPI, first PPI; MD, myotonic dystrophy; IVCD, idiopathic dilated cardiomyopathy; LVD, left ventricular systolic dysfunction; CAD, coronary artery disease.

*RVA catheter inadvertent displacement to a superior position close to the His bundle.
†Entrainment was not achieved because tachycardia was nonsustained.
‡His bundle electrogram was not recordable during tachycardia.
§Entrainment was not achieved because tachycardia was repeatedly terminated by pacing 10 ms faster than the TCL.

Entrainment Pacing Protocol

Transient entrainment was attempted in all tachycardias by pacing from the RVA. Pacing was performed continuously during tachycardia for >5 seconds (7±7 seconds) with a CL 10 to 30 ms shorter than the TCL. Subsequent entrainment sequences were performed with 5- to 20-ms decreases in the pacing CL until tachycardia termination or until the patient's condition deteriorated. Standard definitions were used to define successful tachycardia entrainment.6,9,10 The PPI was measured from the last stimulus artifact to the beginning of the first rapid deflection of the RVA electrogram.

Statistical Analysis

Parametric data are presented as mean±SD. ANOVA was used to compare parametric data differences between tachycardia groups. After demonstration of significant differences between the groups, post-hoc tachycardia group pair comparisons were made by use of Student-Newman-Keuls multiple comparison procedures. After demonstration of normality by Shapiro-Wilk's W test, nonpaired Student's t test was used to compare parametric data differences when tachycardias were divided into only 2 groups. A value of

Results

There were no significant differences (P=0.62) in TCL between the BBR-VT (297±41 ms) and MR-VT (304±55 ms) groups. The AVNRT group (347±56 ms) had a significantly (P<0.01) longer TCL than the other 2 tachycardia groups. Differences between TCL and entrainment CL were nonsignificant (P=0.35) between the BBR-VT (16±6 ms), MR-VT (15±6 ms), and AVNRT (14±8 ms) groups. No BBR-VT had 1:1 AV conduction.

PPI–TCL Differences According to the Tachycardia Mechanism

Tachycardia entrainment pacing from the RVA was not achieved in 3 BBR-VTs because they were nonsustained and in a fourth because it was repeatedly terminated by pacing 10 ms faster than the TCL. The difference between the PPI and the TCL (PPI–TCL) was significantly (P<0.0001) shorter in the BBR-VT group (9±11 ms, 95% CI 2 to 16 ms) than in the MR-VT (109±48 ms, 95% CI 96 to 122 ms) and the AVNRT (150±29 ms, 95% CI 142 to 158 ms) groups (Figures 2 through 4). The PPI–TCL was also significantly (P<0.001) shorter in the MR-VT group than in the AVNRT group. No BBR-VT showed a PPI–TCL (Figure 3) >30 ms (range −12 to 24 ms) except for the tachycardia of BBR-VT patient 1, which showed a 47-ms difference (Table). Retrospective review of the filmed RVA catheter fluoroscopic position revealed inadvertent displacement to a superior position close to the His bundle, and pacing from this site produced a QRS complex with an inferior axis. In addition, local activation time of this site during tachycardia was 15 ms after the onset of the QRS complex, in contrast to other BBR-VTs with LBBD configuration and short PPI–TCL, in which RVA local activation was never >5 ms after the
QRS-complex onset (−8.8±11 ms). This PPI was not included in the statistical analysis because of incorrect RVA catheter positioning. No MR-VT or AVNRT (Figure 4) showed a PPI−TCL <30 ms (range 21 to 211 and 100 to 215 ms, respectively) except for 2 MR-VTs (21 and 29 ms) in 2 patients with prior myocardial infarction. There was a high overlap of PPI−TCL values between the MR-VT and AVNRT groups (Figure 2).

**PPI−TCL Differences According to the QRS-Complex Configuration and the Underlying Heart Disease**

No significant differences (P=0.52) were found in the PPI−TCL between the 18 MR-VTs with an LBBB (103±55 ms, range 21 to 195 ms) and the 41 MR-VTs with an RBBB (112±46 ms, range 29 to 211 ms) QRS-complex configuration (Figure 4). No significant differences (P=0.46) were found in the PPI−TCLs among MR-VTs when grouped according to the underlying structural heart disease (101±50 ms in the prior myocardial infarction group, 138±30 ms in the idiopathic dilated cardiomyopathy group, 124±57 ms in the right ventricular dysplasia group, 101±56 ms in the valvular heart disease group, and 113±47 ms in the non-apparent structural heart disease group).

The PPI−TCL was significantly (P<0.0001) longer in AVNRTs with RBBB (181±20 ms, 95% CI 167 to 195 ms) than in AVNRTs without BBB (142±25 ms, 95% CI 134 to 150 ms). The PPI−TCL was 133 ms in the single AVNRT with LBBB.

**Discussion**

**PPI−TCL Differences According to Tachycardia Mechanisms**

The results of this study demonstrate entrainment of BBR-VT pacing from the RVA results in a PPI similar (<30 ms difference)⁹,¹⁰ to the TCL. Therefore, the lack of this response, provided that the RVA catheter is correctly positioned, should be considered highly suggestive of tachycardia mechanisms other than BBR. Conversely, PPI−TCL differences <30 ms should be considered indicative but not diagnostic of BBR, because this was also found in 2 of the 59 MR-VTs. In such cases, other methods should be used to establish the definitive tachycardia mechanism. A PPI−TCL <30 ms after entrainment pacing from the RVA in some MR-VTs is not surprising, because in this setting, part of the reentrant circuit may be located in the right ventricle¹² in close proximity to the pacing area at the RVA.

Results of the present study also demonstrated that differentiation of BBR-VT from the rare cases of nodal tachycardias with AV dissociation¹¹,¹³ can be readily and safely achieved by the PPI−TCL because no AVNRT showed this difference to be <100 ms. In theory, a similar response should be observed in microreentrant tachycardias confined to the His or proximal bundle branches.¹⁴ Other mechanisms of wide-QRS-complex tachycardia that theoretically may exhibit a short PPI−TCL, such as atrioventricular reentry through an accessory pathway, consistently have 1:1 AV conduction, and therefore, differentiation from BBR-VT can be easily achieved without the PPI.

No sustained interfascicular reentrant tachycardias were included in the study, and therefore, the PPI−TCL difference after entrainment by pacing from the RVA is speculative. From a theoretical point of view, however, a difference >30 ms should be expected in this setting, because the reentrant circuit is located in the left ventricle away from the RVA.

**PPI−TCL Differences According to the QRS-Complex Configuration and the Underlying Heart Disease**

Interestingly, there were no significant differences in the PPI−TCL found in BBR-VTs and MR-VTs with an LBBB QRS-complex configuration compared with that found in those with an RBBB QRS-complex configuration. This underlines the fact that VT QRS-complex configuration, of either a BBR or an MR mechanism, is simply dependent on the location of the exit from the slow conducting portion of the reentrant circuit rather than on the circuit location itself, close to or away from the RVA.

More remarkable was the lack of significant differences between the PPI−TCL in the MR-VT postinfarction and arrhythmogenic dysplasia groups. Right ventricular diffuse slow conduction and a basal or outflow tract location of the reentrant circuit far from the RVA in the latter group may explain this finding.

Finally, the right bundle-branch distal insertion is in the RVA, and this explains the QRS-complex superior axis in most BBR-VTs with LBBB configuration.⁴ Sometimes BBR-VT with LBBB QRS-complex configuration presents with an inferior axis, however, suggesting an arborizing or a
more superior insertion of the right bundle branch. Thus, theoretically the PPI – TCL could be >30 ms in this setting, although this was not the case in the single entrained BBR-VT with LBBB QRS-complex configuration and inferior axis.

Study Limitations

Because oscillations in the VV interval preceding those of the HH interval have been reported in BBR-VT,6,8 BBR cannot be completely ruled out as the tachycardia mechanism unless a His or bundle-branch electrogram is recorded that displays no 1:1 tachycardia association or unless manifest or antidromic concealed fusion (concealed fusion with the same QRS complex as that recorded during sinus rhythm pacing from the same site and with the same CL as during entrainment) is observed during entrainment by atrial pacing of a tachycardia with LBBB QRS-complex configuration. Therefore, some VTs failing to show these responses in the present and other1–4 studies could have been included in the MR-VT group despite having a BBR mechanism. BBR is highly unlikely in the absence of significant His-Purkinje disease,3–5 however, and except for 10 MR-VT patients, none showed prolonged QRS complex (>120 ms) or HV interval (>60 ms) during sinus rhythm in the present study. In 9 of these 10 patients, BBR was considered highly unlikely because the His bundle electrogram showed no 1:1 tachycardia association, because there were supraventricular captures with a different QRS-complex morphology, or because the His bundle electrogram followed the onset of the QRS complex and preceded the perihisian ventricular activation.

Four BBR-VTs could not be entrained by ventricular pacing because they were nonsustained or terminated at the longest pacing CL. These factors, together with TCL oscillations, limit the use of the PPI analysis for the elucidation of a wide-QRS-complex tachycardia mechanism. Despite these potential limitations, however, the PPI approach could be performed in the majority of BBR-VTs.

Clinical Implications

PPI analysis after entrainment pacing from the RVA offers several advantages to the conventional diagnostic approach of
Figure 4. Entrainment of representative examples of MR-VT with LBBB QRS-complex configuration (top), MR-VT with RBBB QRS-complex configuration (middle), and AVNRT with RBBB QRS-complex configuration (bottom). Note that (wide arrow) PPI is longer than TCL in all panels. RB indicates right bundle branch.
wide-QRS-complex tachycardia with AV dissociation. First, a PPI–TCL >30 ms rules out a BBR mechanism, provided that the RVA catheter is correctly positioned. The greater this value, the more reliable the certainty of exclusion. Therefore, this obviates the need to record a His bundle or bundle-branch electrogram or to achieve tachycardia entrainment by atrial pacing. Second, this approach does not require the introduction of catheters or pacing maneuvers different from those regularly used in standard electrophysiological studies of wide-QRS-complex tachycardia. Finally, this approach is time-saving and can be attempted even in poorly tolerated VTs, because only a brief ventricular pacing train is required.

Conclusions

The analysis of the PPI after tachycardia entrainment by pacing from the RVA is useful to differentiate BBR from other mechanisms of wide-QRS-complex tachycardia with AV dissociation. A PPI–TCL >30 ms suggests mechanisms other than BBR, provided that the RVA catheter is correctly positioned. Conversely, a PPI–TCL <30 ms is indicative but not diagnostic of a BBR mechanism, and further investigation is warranted to exclude or confirm pure ventricular MR by conventional approaches using His-Purkinje recordings or surface ECG analysis during atrial and ventricular entrainment. The simplicity of the PPI approach supports its inclusion early in the diagnostic workup of wide-QRS-complex tachycardia with AV dissociation.

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References

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