Paradoxical Ventricular Activation Sequence and ParaHisian Entrainment Response
Do They Challenge the Diagnosis?

Sachin Nayyar, MD, CCDS, CEPS; Glenn D. Young, MBBS; Prashanthan Sanders, MBBS, PhD;
Kurt C. Roberts-Thomson, MBBS, PhD

Introduction
Ventricular activation at right ventricular apex should precede activation elsewhere in right ventricle in clockwise bundle-branch re-entry. Additionally, entrainment pacing from His catheter should have long postspacing interval (PPI) in bundle-branch re-entry. We demonstrate a case where paradoxical findings challenged the diagnosis of clockwise bundle-branch re-entry.

Case Presentation
A 68-year-old man with nonischemic dilated cardiomyopathy, first-degree heart block (PR 320ms), left bundle-branch block (LBBB; QRS 200 ms; Figure 1, left panel), and severe left ventricular dysfunction had a biventricular defibrillator implanted 3 years ago. His heart failure symptoms were well controlled. However, during the past 1 year, he has had more than 1000 device therapies for a recurring symptomatic arrhythmia. This was poorly controlled with β-blocker and amiodarone. The tachycardia cycle length (TCL) was 360 to 390 ms and had never failed termination with antitachycardia pacing.

He presented for catheter ablation of his arrhythmia. Catheters were placed into the right ventricular (RV) apex and at the His position. The AH and HV interval in sinus rhythm (SR) were 130 and 140 ms, respectively. Atrial pacing did not change the HV interval or the QRS morphology. The clinical tachycardia was easily inducible with a single extrastimulus from the RV apex (Figure 1, right panel). The TCL was 410 ms with right bundle-branch block (QRS 180ms) morphology, left superior axis, and V3–V4 transition. There was VA dissociation, but 1:1 VH association. The tachycardia VH and HV intervals were 150 and 260 ms, respectively (Figure 1, right panel), and with His catheter advanced slightly into the ventricle, 100 and 310 ms, respectively (Figure 2). Ventricular activation at His catheter preceded RV apex by 10 ms and was 16 ms later to surface QRS onset in V1 (Figure 2). There was minor TCL variation, and HH interval changes preceded VV interval changes (Figure 3). A late extrastimulus from RV apex during tachycardia advanced the His and reset the tachycardia with a preserved HV relationship (Figure 4A), whereas a shorter coupled extrastimulus easily terminated the tachycardia (Figure 4B). Pacing from RV apex 10 ms shorter than the TCL entrained the tachycardia with manifest fusion and a PPI minus TCL of 26 ms. Entrainment pacing was then performed from the His catheter as shown in Figure 5. The 12 lead ECG is shown in Figure 6. Is the diagnosis of bundle-branch re-entrant ventricular tachycardia (BBRVT) justified?

Discussion
The presence of a drug refractory arrhythmia in dilated cardiomyopathy with conduction system disease and responding to RV apical antitachycardia pacing raises the possibility of BBRVT. This was also suggested by the presence of 1:1 VH relationship. However, a typical (counterclockwise) BBRVT has LBBB morphology, and it is rare to have clockwise BBRVT with underlying LBBB. The activation at RV apex should have preceded activation elsewhere in RV. Also, pacing from the His catheter resulted in a short PPI, which is against the diagnosis of BBRVT. Therefore, the possibilities for this tachycardia are (1) myocardial re-entrant ventricular tachycardia, (2) interfascicular re-entrant tachycardia, (3) anterograde left nodofascicular pathway, and (4) clockwise BBRVT.

(1) Myocardial re-entrant ventricular tachycardia. A septal myocardial re-entrant ventricular tachycardia can have 1:1 VH relation during tachycardia with passive retrograde activation of the His. However, tachycardia VH interval shorter than sinus HV interval is unlikely, but could be possible if the re-entry exit is close to the fascicles, and the retrograde conduction from the fascicles is faster than the anterograde conduction during SR. Also, if His–Purkinje system is engaged during the diastolic activation before the exit, the tachycardia VH interval can be shorter than sinus HV. Advancement of the His signal by a RV apical late extrastimulus is also possible in a myocardial re-entrant ventricular tachycardia and H–H change may deceivingly appear preceding the V–V change, with a preserved HV relationship. Also, the appearance of split His potentials (H1 and H2) coinciding with tachycardia termination could be possible, when the RV extrastimulus penetrates the tachycardia and simultaneously both right and left bundles with H1 resulting via left bundle activation. The PPI–TCL from RV apex and His position were short.

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From the Centre for Heart Rhythm Disorders, Royal Adelaide Hospital, Adelaide, Australia (S.N., G.D.Y., P.S., K.C.R.-T.).
Correspondence to Kurt C. Roberts-Thomson, MBBS, PhD, Centre for Heart Rhythm Disorders, Royal Adelaide Hospital, Level 5, McEwin Building, North Terrace, Adelaide, SA 5000, Australia. E-mail kurt.roberts-thomson@adelaide.edu.au
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and entrainment morphology resembled the tachycardia in the latter. As tachycardia is exiting on the left ventricular side of the septum, these entrainment responses from the RV are possible only if there is a large septal scar and the re-entry circuit is deep within the septum. However, regional endocardial voltages in the septal region were normal, making a large septal scar, and hence myocardial re-entrant ventricular tachycardia, unlikely.

(2) Interfascicular ventricular tachycardia. An interfascicular tachycardia is easily dismissed as it is unlikely to have tachycardia HV interval longer than sinus HV interval, and entrainment from RV (both apex and His region) should have long PPI–TCL.

(3) Nodofascicular re-entrant tachycardia. The presence of a nodofascicular pathway with anterograde conduction down the pathway to the posterior fascicle and retrograde conduction back up the conduction system to the AV node could produce a similar tachycardia with VH linking and VA dissociation. However, during atrial pacing, no evidence of pre-excitation was seen suggestive of presence of a pathway.

(4) Bundle-branch re-entrant ventricular tachycardia. Clockwise BBRVT with underlying LBBB is rare, but not impossible. In the present case, the left bundle is more diseased than the right bundle, and thus LBBB was observed during SR. However, conduction delay, rather than complete block, allowed conduction down the left bundle during clockwise BBRVT as well as during SR, as evident by r>1 mm in V1 in SR suggesting preserved left to right septal activation, and thus preserved left bundle conduction. The HV interval during BBRVT can be longer than during SR, and it can be significantly longer if the anterograde conduction is down the more diseased bundle, as was seen in this case. Also, His catheter position closer to the tachycardia turnaround point will record a longer HV interval than in a proximal position, as was observed in this case. The tachycardia induction during programmed extrastimulation from RV was dependent on critical delays in both limbs of the His–Purkinje system. The earliest surface QRS onset during tachycardia was in precordial lead V3. Relative to lead V3, onset occurred 28 ms later in V1, and 44 ms later in distal His. The numbers represent the annotated intervals in milliseconds.
intracardiac ventricular activation during tachycardia was earliest at distal His followed closely by 10 ms at the proximal RV apex. On closer observation, RV apex was activated nearly on time at proximal and distal bipoles, with proximal preceding distal RV apex by 6 ms. These observations were in congruence with the midseptal activation from an exit at the left bundle septal fibers, and subsequent left to right transeptal conduction with wavefront propagation both toward right basal septum and right apical septum (Figure 7). The septal activation toward right basal septum is bystander and does not form part of the BBRVT circuit, as the right bundle is engaged only by activation toward RV apex. On reviewing previously published tracings of clockwise BBRVT, the V signal at the His catheter was recorded before the RV apex and either on time or soon after the surface QRS onset, as it was in our case. The exact portion of the septum that participates in the BBRVT is unknown, however, it would be logical to infer from this relation between QRS onset and earliest intracardiac activation at His that the activation of at least some part of the septum is diastolic or bystander activation, and remains concealed on the surface ECG during BBRVT.

**Response to pacing maneuvers in BBRVT.** A late RV apical extrastimulus will advance the His and subsequent V, as was seen in this case. An earlier extrastimulus terminated the tachycardia, likely by rapid retrograde penetration into the right bundle provoking delay within the His bundle and appearance

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**Figure 3.** Cycle length variation during tachycardia. HH interval changes precede changes in the VV interval. Channels from top are surface ECG (I, II, V1, V5), His, and RVA. A indicates atrial signal; d, distal; H, His potential; p, proximal; RVA, right ventricular apex; S1 & S2, extrastimulus; and V, ventricular signal. The numbers represent the annotated intervals in milliseconds.

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**Figure 4.** Top panel, Resetting of the tachycardia by a late extrastimulus from right ventricular apex (RVA). His potential is advanced (H1) followed by slight HV delay and continuation of the tachycardia. **Bottom panel,** Termination of the tachycardia by an early extrastimulus from RVA. There is greater fusion in this paced beat compared with the top panel. H1 potential is further advanced and is followed by a delayed second His potential (H2). Channels from top are surface ECG (V1, V5), His, and RVA. A indicates atrial signal; d, distal; H, His potential; p, proximal; RVA, right ventricular apex; S1 & S2, extrastimulus; and V, ventricular signal. The numbers represent the annotated intervals in milliseconds.
of split His potentials (H1 and H2), further suggesting His bundle was possibly participating in the tachycardia.4 As RV apical septum is in the circuit, the PPI–TCL is expected to be <30 ms after entrainment pacing from RV apex,10 as was in this case. However, it will not be concealed unless it captures the right bundle only.

Response to entrainment pacing from His catheter. Entrainment pacing from the His catheter should have had a long PPI–TCL, and manifest fusion as the pacing stimulus has to engage the right bundle apically before it entrains the circuit. In this case, the PPI–TCL was short and the QRS morphology during entrainment resembled the tachycardia. These findings were most likely because of regional myocardial capture in the basal septum that, as conceived from the septal activation during tachycardia, was activated parallel with the diastolic pathway. As a result, the return activation at the end of pacing was shorted out arriving in advance than the full cycle length resulting in a quicker PPI. As pacing was done at rate close to the TCL, the fused QRS morphology resembled the tachycardia.3 The stimulus–H interval was longer than the tachycardia VH interval in congruence with sequential activation from stimulus to His during pacing, whereas during tachycardia, the local V activation at basal septum was initiated before the activation returned to His bundle. The stimulus–QRS was long, as the nth stimulus from the His catheter was driving the n+1 QRS. His capture would have produced shorter stimulus–H interval than tachycardia VH interval, whereas a pure His capture would have produced a concealed entrainment response, none of which were the case. Hence, the ventricular activation sequence and the entrainment response from His catheter were compatible with the diagnosis of clockwise BBRVT.

Right bundle-branch ablation was performed during tachycardia that terminated and became noninducible. There was transient complete AV block, which recovered within a few minutes to first-degree AV block (PR 440 ms, HV 300 ms) and complete right bundle-branch block. Interestingly, postright-bundle ablation, the QRS morphology, and HV interval during SR resembled that of the tachycardia, supporting anterograde activation was down the left bundle during tachycardia. No other tachycardias were inducible, and the patient was
Figure 7. Schematic for activation in clockwise bundle-branch re-entrant ventricular tachycardia. The anatomy of bundle-branches is drawn, as previously described by Massing and James. The left bundle-branch divides in the basal septum at a variable length below aortic valve to anterior and posterior fascicles (LAF, LPF). The additional midseptal fiber groups from left bundle distribute into the septum (yellow asterisks) within the septum. The LAF distributes in basal and anterior left ventricle, whereas LPF distributes more apically into posterior left ventricle (large black asterisks). The right bundle-branch (RBB) has a longer septal course toward apex before it exits at the base of anterior papillary muscle, where it divides into 2 divisions, entering moderator band and right ventricular (RV) endocardium (large asterisks). The course of activation during tachycardia in right and left bundle-branches is shown by the dotted line, and bold arrows represent points of entry and exit to and from the respective bundles. The crescentic shaded area represents the portion of the muscular septum participating in the circuit. There is no septal scarring and the numbers represent orthodromically activated time zones of septal myocardium after exit of the tachycardia impulse from the mid-septal fibers. Catheters are shown at His and RV apex (RVA) locations (black squares). When the exit is in the middle septum, ventricular activation at basal septum near His can precede or is simultaneous to activation at the RV apex (A). When the exit is in the high septum, ventricular activation at basal septum near His will precede activation at the RV apex (B). When the exit is in the low septum, ventricular activation at RV apex will precede activation at the basal septum near His (C).

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Disclosures
None.

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EDITOR’S PERSPECTIVE

Nayyar et al describe a patient with atypical bundle-branch re-entry that provide the student of electrophysiology an opportunity to appreciate the limitations of our knowledge regarding interventricular connections for conduction and the need for deduction when a complete circuit cannot be mapped. The case shows us that the RV apex is not the site of initial RV actionation when transseptal conduction occurs in this arrhythmia.

TRANSVENTRICULAR SEPTAL CONDUCTION IS THE RIGHT VENTRICULAR APEX ON PACING IN THE PARAHISIAN REGION

The ParaHisian Region

The penetrating bundle of His almost invariably is located on the membranous portion of the interventricular septum. This site is at the junction of the commissure between the right and noncoronary cusps of the aortic valve, and the septal and anterior leaflets of the tricuspid valve. A sleeve of annular insulance coats the His bundle (as well as the proximal right and left bundles) such that the surrounding ventricular myocardium in this region is not activated from the His bundle, but later, after the propagating wavefront emerges from the bundle-branches more distally. The ventricular myocardium at sites that record a His-like potential is relatively apical to the annulus, distal to the membranous septum, in the region of the right bundle. Appreciating this anatomy is essential for understanding the nuances of pacing at this location. High-output pacing with narrow interelectrode spacing at the membranous septum may capture the His bundle. More commonly, ventricular capture without His bundle activation is seen. As there is no ventricular myocardium around the true His bundle, ventricular capture indicates that the myocardium at the junction of the His and the right bundle is being captured. Chronic pacing of the His bundle is intuitively attractive, for maintaining synchronous electromechanical activation, but has been difficult to achieve. Similarly, for paraHisian pacing maneuvers to distinguish retrograde AV node versus accessory pathway activation, high-output pacing is often required to capture the His bundle, beneath the paraHisian fibrous tissue, but also often captures the neighboring ventricular myocardium, and as the output is reduced, His bundle capture is lost.

ParaHisian Entrainment

The effect of entrainment during paraHisian pacing depends on the tissue that is actually captured, specifically the right bundle and adjacent ventricular myocardium, which will vary with precise pacing location and stimulus output. Ventricular-only capture would result in short postspacing interval tachycardia cycle length difference, when the mid-ventricular myocardium is part of a re-entrant circuit. Usually, right-bundle capture is easily excluded when a distinct His/right bundle signal is seen well after the local ventricular electrogram (as in this case). However, when a split His or delay producing a distinct His and right-bundle signal occurs, one of the potentials may be captured and the other delayed.

Pseudointervals

As demonstrated in this case, much can be deduced from measurement of interelectrogram intervals. The interval between 2 points may indicate the conduction time from point A to point B. However, it can also be a pseudointerval, where a wavefront does not go directly from A to B, but rather conduction to each point occurs by a wavefront from some point elsewhere that is not mapped. For example, the authors note that during tachycardia, the paraHisian ventricular myocardium was activated 16 ms before the RV apical myocardium. As they demonstrate well, the true breakthrough site was somewhere in between relatively closer to the paraHisian region, and from that point conduction spread toward the RV apex and toward the annulus. However, the V–H and H–V intervals that were fundamental to their analysis represented true conduction intervals. When the His bundle recording catheter was advanced more apically, the V–H interval shortened and the H–V interval lengthened such that the sum of these intervals remained the same. Pseudointervals, rather than true conduction intervals, are the source of many enduring mysteries in electrophysiology, including AV node re-entry (the H–A and V–A intervals are pseudointervals), bundle-branch re-entry; the H–V is a pseudointerval (but the right bundle to V is not), and the A–V interval recorded along the coronary sinus in right atrial rhythms.

Gaps in Activation Maps

Part of the reason inferences from pacing maneuvers are important, as demonstrated by Nayyar, relates to limitations in identifying a re-entrant circuit when there are large gaps in what has been defined. In this case, recording the left bundle and multiple recordings along the left bundle and along the right bundle could potentially have been of value. Similarly, with re-entrant atypical atrial flutters and scar-related ventricular tachycardias, it is often difficult to determine activation times at fragmented electrograms producing significant gaps in the mapped circuit, adding to the difficulty in defining these arrhythmias. AV node re-entry is another example, where inability to record signals along the slowly conducting AV nodal region impedes understanding of this arrhythmia, the mechanism of which is still poorly understood.

Summary

Perhaps what the student learns the most from Nayyar et al’s contribution to the Teaching Points section in this issue of Circulation: Arrhythmias and Electrophysiology is the importance for critical analysis, attention to detail, and evaluation of all differential diagnostic possibilities, in a systematic manner, that allows them to not only appreciate the limitations of their reasoning, but also plan a simple, safe, and effective treatment strategy.
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