

Bundle branch reentry: A novel mechanism for sustained ventricular tachycardia in Chagas heart disease



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Introduction

Bundle branch reentrant ventricular tachycardia (BBR-VT) is a unique type of VT that involves the right and left bundle branches and the ventricular septum as components of a macroreentrant circuit, and classically has been described in individuals with cardiomyopathy and some degree of His-Purkinje system disease.^{1,2}

Among patients with Chagas heart disease (ChHD), some form of His-Purkinje system disease is commonly seen, clinically manifesting as intraventricular conduction delay,^{3–5} which can create the appropriate milieu for reentry within the bundles. However, BBR has not been described thus far as a potential mechanism underlying sustained VT in this clinical setting.

We herein report the first case of proven BBR-VT in a patient with ChHD. The patient presented with a rapid, hemodynamically unstable wide complex tachycardia, subsequently experienced multiple shocks from the implantable cardioverter-defibrillator, and ultimately was treated with ablation of the right bundle branch (RBB).

Case report

A 42-year-old man without any medical history had a sudden-onset episode of palpitations and collapsed. Upon presentation to the emergency room the patient was unresponsive, with hemodynamic instability owing to a wide complex tachycardia with heart rate of approximately 200 beats per minute (bpm), requiring prompt termination by a direct current shock. During subsequent hospital work-up, the patient was diagnosed with Chagas disease by 2 positive serologic tests (complement fixation / immunofluorescence tests). The baseline 12-lead electrocardiogram (ECG) during sinus rhythm showed a prolonged PR interval (240 ms) and

intraventricular conduction delay resembling left bundle branch block (LBBB) with left-axis deviation and QRS duration of 130 ms. Transthoracic echocardiogram revealed severe left ventricular enlargement (diastolic diameter, 73 mm), and left ventricular ejection fraction of 25% with an inferolateral akinesia and left ventricular apical dyskinesia. Coronary angiography was normal. The patient underwent a biventricular implantable cardioverter-defibrillator (ICD) placement and was discharged on optimized treatment for heart failure and on amiodarone. Within 2 months, he began experiencing frequent shocks from the ICD caused by episodes of sustained VT at cycle lengths of 300–320 ms (rates of 187–200 bpm), and he was referred to our institution for an electrophysiologic study (EPS) and for attempting radiofrequency catheter ablation of a presumptive myocardial scar-related VT. The EPS was carried out with the patient sedated and biventricular ICD was reprogrammed to a backup pacing mode. During EPS, baseline intervals in sinus rhythm showed a normal atrial-His interval of 110 ms, a prolonged His-ventricular (HV) interval of 80 ms, and QRS duration of 130 ms (Figure 1). Programmed ventricular stimulation with up to 3 extrastimuli delivered from right ventricular (RV) apex induced a sustained monomorphic LBBB VT with a rate of 190 bpm (Figure 2A). The typical LBBB morphology of VT resembled the nonpaced sinus rhythm QRS morphology. During VT, atrioventricular (AV) dissociation was observed (Figure 2A), and early activation of the RV apex was documented; onset of VT was preceded by His-bundle activation, and the HV interval during VT was longer (95 ms) than the HV interval in sinus rhythm; and spontaneous variations in V-V intervals were preceded by similar changes in H-H intervals, indicating that His-Purkinje activation drives the VT (Figure 2B). Further maneuvers, such as entrainment from RV, were not attempted because of hemodynamic instability during VT. The aforementioned findings strongly suggested that the mechanism of the induced VT was BBR, and the RBB was targeted for ablation. Radiofrequency energy was delivered at the anterior septum of the RV, where the proximal RBB potential was recorded in sinus rhythm (RBB-V interval of 65 ms; Figure 3A), and resulted in development of right

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KEY TEACHING POINTS

- Bundle branch reentrant ventricular tachycardia (BBR-VT), a unique form of reentrant VT involving the His-Purkinje system, generally occurs in patients with structural heart disease and associated His-Purkinje conduction disease
- Patients with Chagas heart disease (ChHD) usually display evidence of His-Purkinje system disease, clinically manifesting some degree of intraventricular conduction delay, most commonly right bundle branch block, left anterior fascicular block, and, less commonly, left bundle branch block, ultimately creating the appropriate milieu for development of BBR. However, BBR-VT has never been described in patients with ChHD.
- Our case highlights BBR as a potential mechanism underlying sustained VT in ChHD, which can be cured with catheter ablation and should be considered in the differential diagnosis of ChHD patients presenting with arrhythmic events.

pattern with absent or minimal S waves in leads I and aVL, also referred to as bilateral bundle-branch delay/block, was noted after RBB ablation (Figure 3B).⁶ After ablation, repeat ventricular stimulation showed no inducible ventricular arrhythmias and the patient's ICD was reprogrammed to atrial sensed biventricular pacing. After over 4 years of follow-up no arrhythmias were detected by the ICD, and ultimately the patient underwent heart transplantation owing to worsening heart failure.

Discussion

We describe a unique case of BBR-VT in a patient with ChHD, who experienced multiple shocks from a recently implanted ICD and was ultimately treated with radiofrequency ablation of the RBB. To the best of our knowledge, BBR-VT has never been described in the setting of ChHD.

Myocardial scar-reentrant VT is the most common type of sustained VT within the population of ChHD,^{7,8} and the reported patient was referred to our institution for attempting catheter ablation of a presumptive scar-related VT. Unexpectedly, during programmed ventricular stimulation a sustained LBBB-type wide QRS tachycardia resembling the nonpaced sinus rhythm QRS morphology was induced and diagnosis of BBR-VT was established with usual criteria^{1,2}: (1) the 12-lead ECG morphology of the VT exhibited a typical LBBB pattern, and AV dissociation was observed; (2) onset of VT was preceded by His-bundle activation, and the H-V interval during VT was longer than the H-V interval in sinus rhythm; (3) spontaneous variations in V-V intervals were preceded by similar change in H-H

bundle branch block (RBBB) on surface ECG, prolongation of QRS from 130 to 150 ms (Figure 3B), increase of HV interval from 80 to 95 ms, and disappearance of the RBB potential at the ablation site (Figure 3C). An atypical RBBB ECG

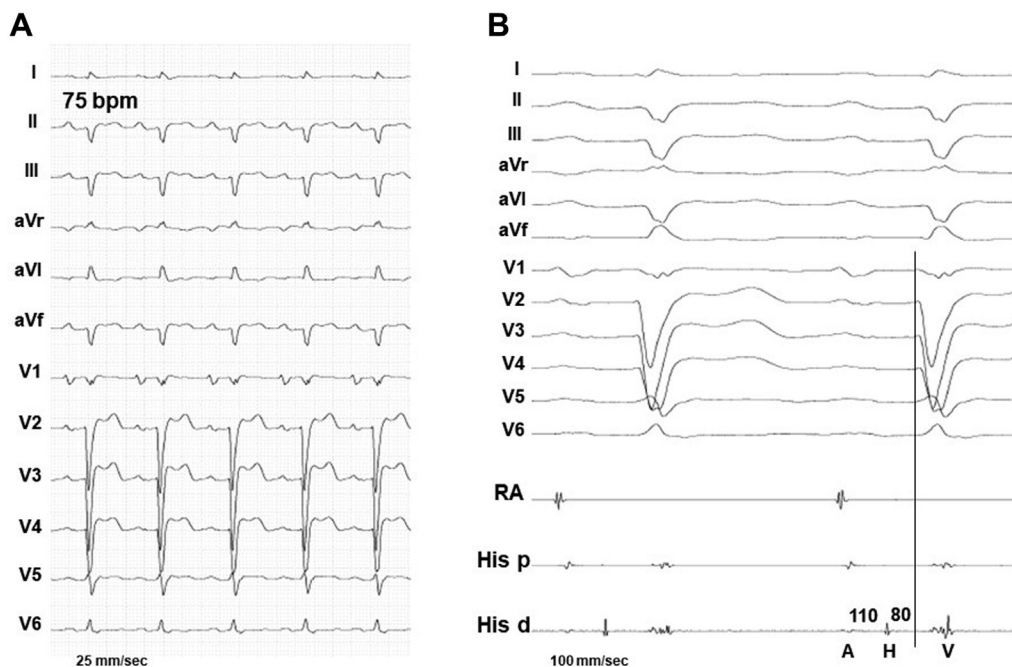


Figure 1 A: Twelve-lead electrocardiogram (ECG) showing sinus rhythm with prolonged PR interval (240 ms) and intraventricular conduction delay resembling left bundle branch block with left axis deviation. B: Baseline intracardiac recordings showing a normal atrial-His (AH) interval of 110 ms, a prolonged His-ventricular (HV) interval of 80 ms, and QRS duration of 130 ms. Displayed from top to bottom are ECG leads and intracardiac recordings from the right atrium (RA) and His-bundle proximal (His p) and distal (His d).

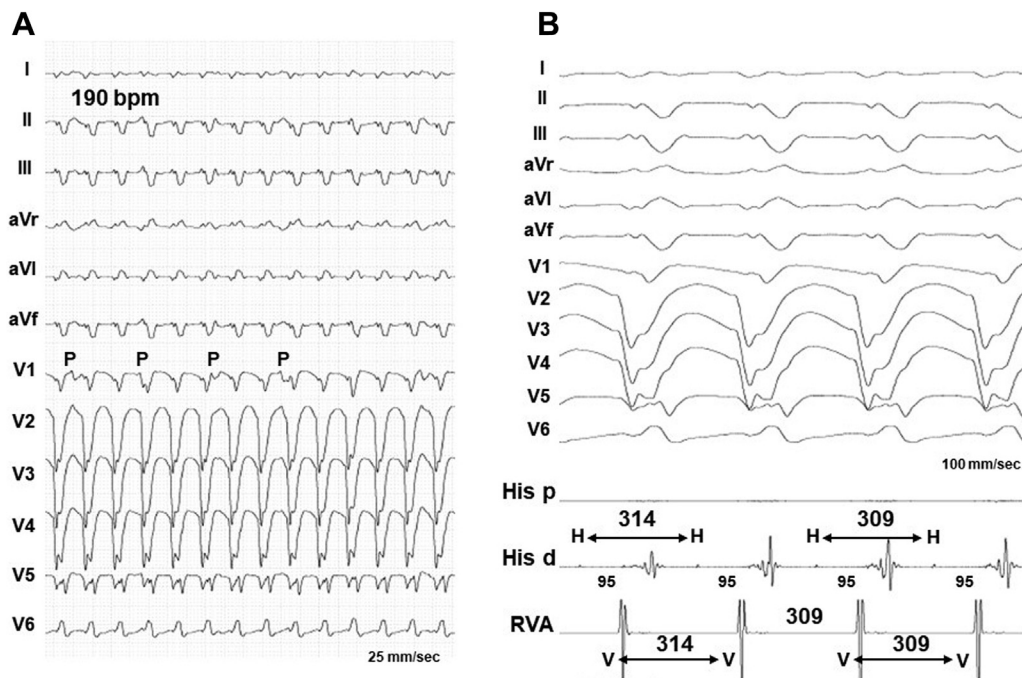


Figure 2 A: Twelve-lead electrocardiogram of the induced sustained ventricular tachycardia (VT) with a rate of 190 beats/min (bpm) and left bundle branch block morphology, similar to QRS configuration as in sinus rhythm. Dissociated P waves are also seen during VT, especially in the precordial lead V₁. B: Intra-cardiac recordings during induced sustained bundle branch reentrant VT. His bundle potential precedes each QRS complex with HV interval of 95 ms and early activation of the right ventricular apex (RVA) during VT; some irregularity of VT cycle length is seen, and changes in H-H intervals precede changes in V-V intervals, indicating that His-Purkinje activation drives the VT. Abbreviations as in Figure 1.

intervals; and finally (4) BBR-VT was noninducible after successful RBB ablation.

BBR-VT generally occurs in the background of dilated cardiomyopathy, ischemic cardiomyopathy, valvular heart disease, myotonic dystrophy, or even no heart disease with associated His-Purkinje system disease.^{1,2} ChHD is a chronic myocarditis that manifests as heart failure, segmental wall motion abnormalities (aneurysms), thromboembolic events, conduction system disturbances, and ventricular arrhythmias.^{3-5,7,8} Patients with ChHD usually display evidence of His-Purkinje system disease, clinically manifesting some degree of intraventricular conduction delay, most commonly RBBB, left anterior fascicular block, and the combination of RBBB and left anterior fascicular block, and less commonly LBBB and incomplete intraventricular conduction block,³⁻⁵ ultimately creating the appropriate milieu for development of BBR. However, BBR-VT has never been described in patients with ChHD.

Wide QRS tachycardia with LBBB morphology is the most common form of BBR-VT observed in clinical practice, in which the RBB serves as the antegrade limb, the LBB serves as the retrograde limb, and the ventricular septum provides the connecting link.^{1,2} This may be related to the fact that LBB is the preferred retrograde route of impulse propagation, as seen during both left and right ventricular extrastimulation.^{1,2} Alternatively, activation can proceed antegrade via the LBB, and retrograde via the RBB, creating a BBR-VT with RBB morphology.^{1,2}

One possible reason why BBR-VT has never been described in ChHD may be that sustained BBR is relatively

uncommon, even after electrophysiologic evaluation in patients with other cardiomyopathies, and probably has been unrecognized in patients with ChHD. Another reason may be that BBR-VT with LBBB morphology would be unexpected to occur in the presence of baseline RBBB ECG morphology,⁹ the most common form of intraventricular conduction delay in patients with ChHD,³⁻⁵ even though it should be pointed out that BBR-VT with a RBBB pattern may occur if the RBBB is capable of retrograde conduction.² Consistent with these observations, our patient presented with a baseline intraventricular conduction delay in the form of an LBBB, which may justify his susceptibility for developing an LBBB type of BBR-VT.

It is important to recognize BBR as a mechanism of sustained VT in ChHD because it can be cured effectively by catheter ablation of a bundle branch (typically the RBB),^{1,2} probably one of the easiest techniques for VT ablation, whereas scar-related VT caused by complex reentrant circuits in ChHD may be less susceptible to catheter ablation in centers without great expertise. In addition, BBR-VT usually results in marked hemodynamic compromise and often presents as serious arrhythmic events, such as unstable sustained VT, syncope, or sudden cardiac arrest.^{1,2} Elimination of BBR-VT reduces associated episodic hemodynamic collapse, and spares patients from the unnecessary distress of less effective or less desirable therapies such as antiarrhythmic drug therapy or ICD shocks. In the reported patient, BBR-VT was hemodynamically unstable secondary to very rapid ventricular rate (190 bpm) and poor underlying ventricular function, which

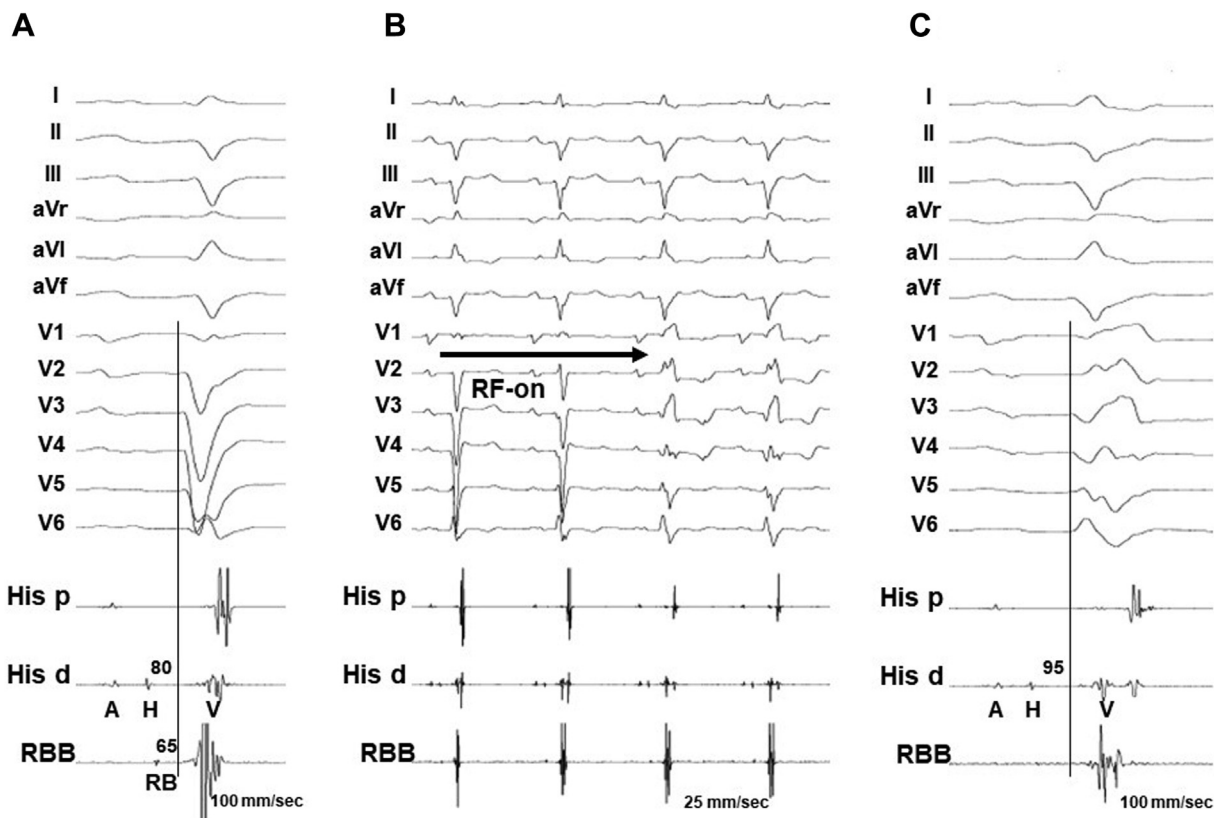


Figure 3 A: Baseline intracardiac recordings before right bundle branch (RBB) ablation showed HV interval of 80 ms and radiofrequency (RF) ablation catheter positioned at proximal RBB showed RBB-V interval of 65 ms. B, C: RF delivery at proximal RBB resulted in development of right bundle branch block pattern on surface electrocardiogram with prolongation of QRS to 150 ms, increase of basal HV interval to 95 ms, and disappearance of the RBB potential at the ablation site. Abbreviations as in Figure 1.

highlights the concept that this arrhythmia may be considered as a potential culprit in cases of unexplained sudden cardiac death in ChHD, a view that has not been yet incorporated in the diagnostic algorithms for this patient population.³⁻⁵

Catheter ablation of the RBB was curative for our patient, but led to further worsening of AV conduction, as expected.^{1,2,10} Importantly, even though our patient showed baseline LBBB, the fact that during RBB ablation the AV conduction was uninterrupted supports the concept that conduction within the right and left bundles was relatively delayed to each other rather than completely interrupted.¹⁰ This concept has been previously discussed by Schmidt and colleagues,¹¹ who showed absent conduction via the left anterior fascicle and slowed conduction via the posterior fascicle using electroanatomic mapping of the left-sided His-Purkinje system in patients with LBBB presenting with BBR-VT.¹¹ Thus, as has been pointed out in similar cases, development of RBBB in the presence of preexisting LBBB may not result in complete heart block, revealing very slow anterograde conduction over the left bundle branch.^{6,10} Of note, the atypical RBBB ECG pattern, with absent or minimal S waves in leads I and aVL that appeared during RBB ablation in our patient, has been reported in patients with preexisting LBBB that developed traumatic

transient RBBB during right heart catheterization, and has been previously designated as bilateral bundle-branch delay/block.⁶

Finally, the clinical course of our patient illustrates the progressive nature of Chagas cardiomyopathy.³⁻⁵ While long-term success in eliminating BBR-VT was achieved with catheter ablation of RBB, progression of the cardiomyopathy ensued, and 4 years later he underwent heart transplantation owing to worsening heart failure.

Conclusion

The reported case highlights BBR as a potential mechanism underlying sustained VT in ChHD, which can be cured with catheter ablation and should be considered in the differential diagnosis of patients presenting with arrhythmic events, such as sustained VT, syncope, or sudden cardiac arrest.

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