

# Incessant bundle branch reentrant ventricular tachycardia in a patient with corrected transposition of the great arteries



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## Introduction

Bundle branch reentrant tachycardia (BBRT) is an uncommon but clinically relevant macroreentrant ventricular tachycardia (VT) involving an impaired His–Purkinje system and the ventricle as a reentrant circuit.<sup>1,2</sup> BBRT is commonly associated with structural heart diseases, including cardiomyopathy, old myocardial infarction, valvular disease, and myotonic dystrophy.<sup>3,4</sup> Patients with congenitally corrected transposition of the great arteries (CCTGA) have unusual atrioventricular (AV) nodes and His bundles anatomy, which are vulnerable to fibrosis with advancing age. This leads to conduction disturbances, resulting in complete AV block as a natural manifestation.<sup>5</sup> CCTGA patients have a possible risk of BBRT because of conduction disturbances. This is the first report on catheter ablation for BBRT in a patient with CCTGA.

## Case report

A 69-year-old woman with CCTGA and atrial septal defect presented to our hospital. She had a history of recurrent palpitations and 2 failed attempts at radiofrequency (RF) ablation of a wide QRS tachycardia with left bundle branch (LBB) block morphology and left axis deviation in previous hospitals (Figure 1A). A 12-lead electrocardiogram (ECG) in sinus rhythm showed 2 discrete non-preexcited morphologies with an inferior axis and a superior axis (Figure 1B and C). Preprocedural contrast-enhanced computed tomography of the heart is shown in Figure 1E. Atrial situs was solitus,

**KEYWORDS** Atrial tachycardia; Bundle branch reentrant ventricular tachycardia; Catheter ablation; Congenitally corrected transposition of the great arteries; Electrophysiology

**ABBREVIATIONS** AV = atrioventricular; BBRT = bundle branch reentrant tachycardia; CCTGA = congenitally corrected transposition of the great arteries; CL = cycle length; ECG = electrocardiogram; LBB = left bundle branch; LV = left ventricle; RBB = right bundle branch; RF = radiofrequency; RV = right ventricle; VT = ventricular tachycardia (Heart Rhythm Case Reports 2015;1:434–438)

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AV discordance was present, and the great arteries were transposed across the ventricular septum. We performed a third ablation procedure because  $\beta$ -blockers, antiarrhythmic drugs, and verapamil were ineffective in suppressing the wide QRS tachycardia.

Three quadripolar catheters were positioned in the high right atrium, the posteroseptal mitral annulus, and the left ventricle (LV) as a subpulmonary chamber through a femoral vein. One duodecapolar catheter was positioned in the tricuspid annulus of the left side through the atrial septal defect using an internal jugular vein approach (Figure 1F). This was performed because coronary angiography revealed that a coronary sinus had returned to the left atrium. The posteroseptal mitral annulus electrode did not demonstrate a posterior His bundle electrogram, despite precise mapping, and showed only split atrial electrograms. Atrial pacing from the high right atrium, low lateral right atrium, and mid-septal mitral annulus did not change the QRS morphology with a superior axis. The earliest atrial activation was recorded at the mid-septal mitral annulus during left ventricular pacing. Antegrade and retrograde conduction of a posterior AV node was observed. However, an electrophysiological study did not show an anterior AV node. The wide QRS tachycardia was incessant and involved single morphology, despite the changes in atrial activation sequence and tachycardia cycle length (CL) (Figure 2A). Ventriculoatrial dissociation occurred during tachycardia and entrainment pacing from the left ventricular apex (Figure 2B and C). Rapid pacing, which was faster than the spontaneous tachycardia CL from the left ventricular apex, showed progressive fusion (Figure 2D). Therefore, the wide QRS tachycardia was diagnosed as reentrant VT. VT was inducible and terminated by atrial stimulation with a single extrastimulus and atrial premature contractions, and did not change by atrial entrainment pacing from the high right atrium (Figure 3A). The earliest site of ventricular activation was the LBB (Figure 3B). Therefore, the VT was diagnosed as BBRT with antegrade conduction through the LBB of the right-sided anatomic LV (Rt-LBBB) and retrograde conduction through the RBB of the left-sided anatomic right ventricle

## KEY TEACHING POINTS

- Patients with congenitally corrected transposition of the great arteries have a possible risk of bundle branch reentrant ventricular tachycardia. This is because these patients have unusual atrioventricular nodes and His bundles results in conduction disturbances with advancing age.
- His bundle electrogram recording is important for diagnosis of bundle branch reentrant tachycardia, but it is difficult or impossible in some cases. In that case, ventriculoatrial dissociation and concealed entrainment by atrial stimulation is useful for definitive diagnosis of bundle branch reentrant tachycardia.
- Ablation of the left bundle branch may be more appropriate than ablation of the right bundle branch, but is challenging for treatment of bundle branch reentrant ventricular tachycardia. Right bundle-branch block might cause mechanical dyssynchrony of the systemic ventricle in patients with congenitally corrected transposition of the great arteries.

(RV) (Lt-RBB) (Figure 1G). RF energy was delivered at the anteroseptal and inferoseptal region of the LV where Rt-LBB potential was recorded during tachycardia. Applications of RF were administered by a 4 mm nonirrigated tip ablation catheter at 55°C–60°C and for up to 60 seconds. Using RF ablation, we were able to create complete antegrade block in the Rt-LBB and then terminate the wide QRS tachycardia (Figures 1D, 3C). Multiple atrial tachycardias persisted after ablation, but they could not be treated because they were unmappable. The patient did not show recurrence of BBRT during 6 months of follow-up.

## Discussion

We performed successful catheter ablation of BBRT in a patient with CCTGA with RF application at the anterior and posterior fascicle of the Rt-LBB. BBRT was terminated when we created complete antegrade block in the Rt-LBB of the right-sided anatomic LV immediately after ablation.

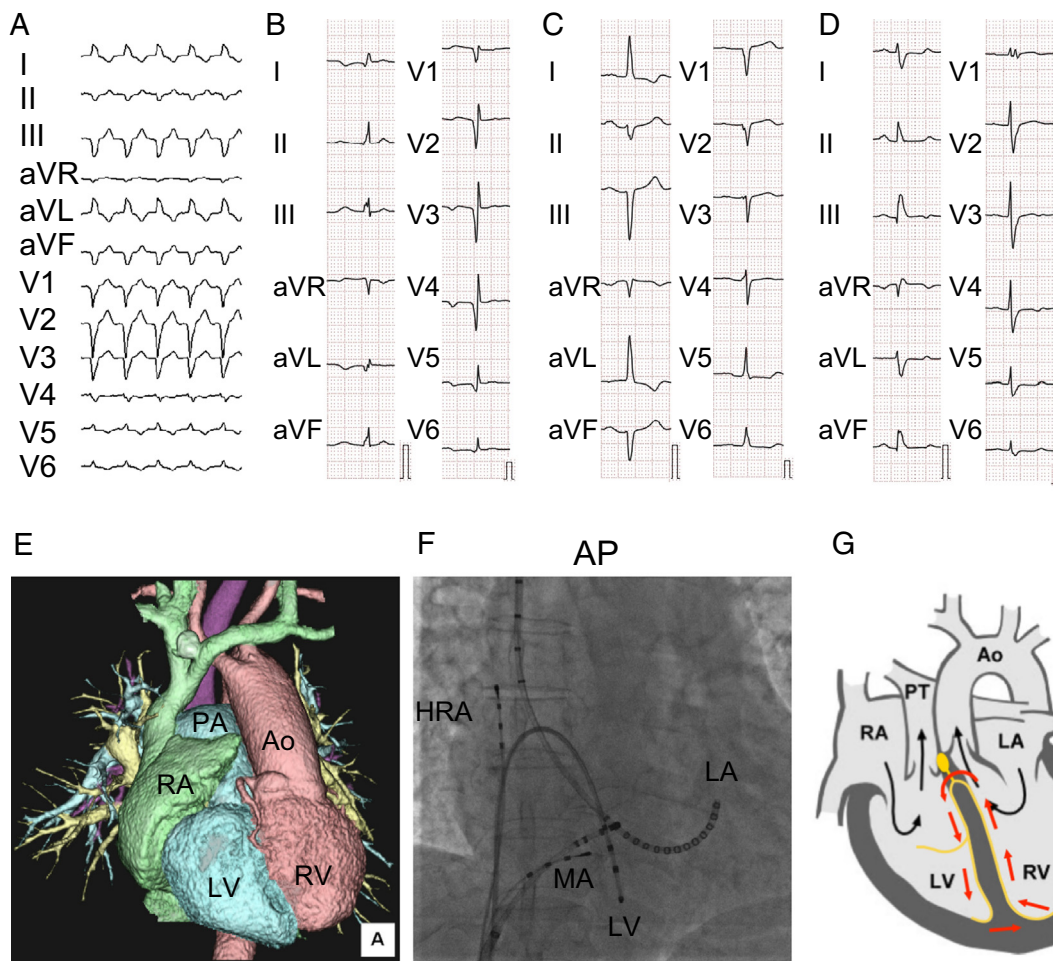
This patient had 2 discrete morphologies in preprocedural surface ECG, but an electrophysiological study showed a posterior AV node. The differential diagnoses of a wide QRS tachycardia in this patient were VT and supraventricular tachycardia with aberrant conduction. Supraventricular tachycardia was excluded by the finding of ventriculoatrial

dissociation. Intrahisian tachycardia, fascicular VT, and interfascicular VT were excluded by concealed entrainment from the high right atrium. His bundle recording is important for diagnosis of BBRT because spontaneous variation in His-His intervals precedes similar changes in ventricular-ventricular intervals. In patients with CCTGA, His bundle electrogram recording is difficult or impossible because of abnormal AV nodal locations. Therefore, ventriculoatrial dissociation and concealed entrainment by atrial stimulation are useful for definitive diagnosis of BBRT.<sup>6</sup> Our patient had both BBRT and multiple atrial tachycardias. Consequently, the CL of BBRT varied according to the CL of atrial tachycardias as an entrainment phenomenon.

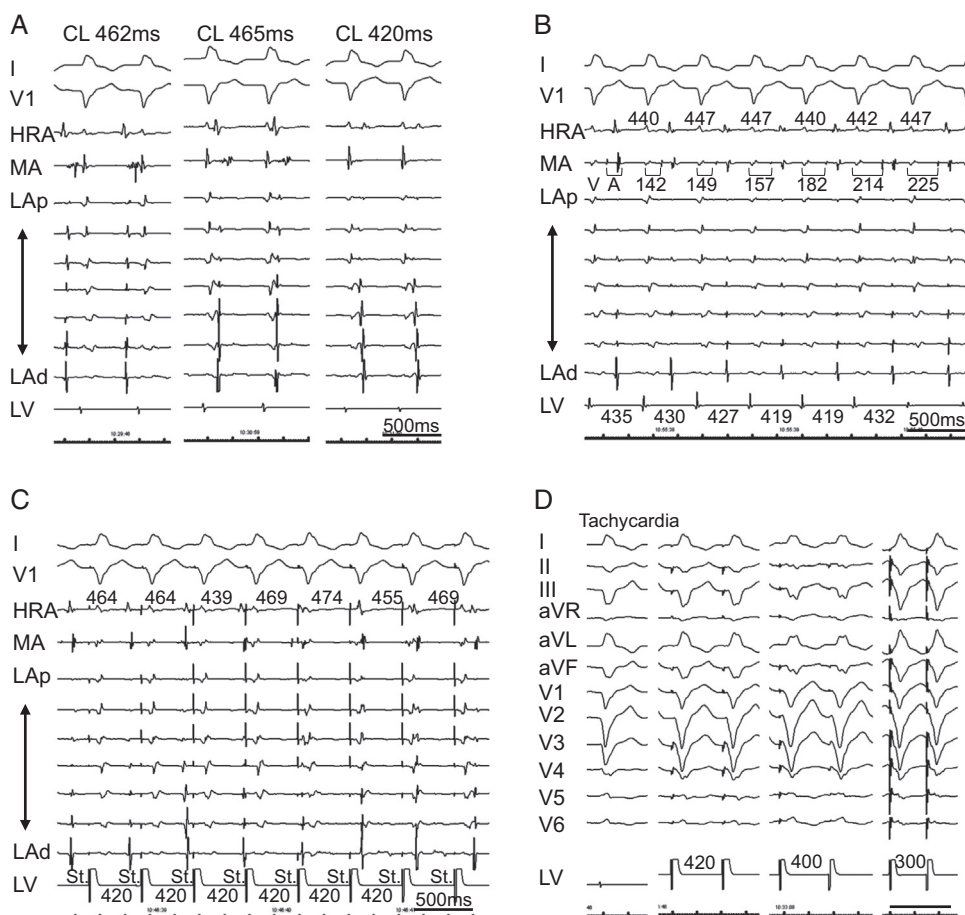
CCTGA is a rare anomaly and comprises <1% of congenital heart diseases. The right atrium enters the right-sided anatomic LV, which connects with the pulmonary artery. The left atrium enters the left-sided anatomic RV, which connects with the aorta. AV and ventriculoarterial discordance is present, but the direction of blood flow is normal. Most patients have 1 or more associated cardiac anomalies, such as ventricular septal defect, pulmonary stenosis, abnormalities of the systemic AV valve, and abnormalities of the conduction system. Many patients have an unusual AV nodal anatomy.<sup>7</sup> In patients with CCTGA, 1 (anterior or posterior) or both (twin) AV nodes develop according to malalignment between the interatrial and posterior interventricular septum.<sup>8</sup> Bundle branches are inverted in CCTGA. LBB in the right-sided anatomic LV shows a fan-like structure, and RBB in the left-sided anatomic RV similarly shows a cord-like structure.

RBB is typically the target of ablation for BBRT in the structurally normal heart.<sup>9,10</sup> Ablation of the LBB can be used as an alternative therapeutic approach for BBRT with normal LV function and LBB block on preprocedural surface ECG.<sup>11,12</sup> In patients with CCTGA, we may create Lt-RBB block more easily than Rt-LBB block by RF. However, Lt-RBB block in left-sided anatomic RV may cause mechanical dyssynchrony of the impaired systemic ventricle in patients with CCTGA. Ablation of the Rt-LBB may be more appropriate for treatment of BBRT in patients with CCTGA. However, ablation of the Rt-LBB is more challenging and requires special expertise because Rt-LBB is not a discrete structure like Lt-RBB.<sup>11</sup>

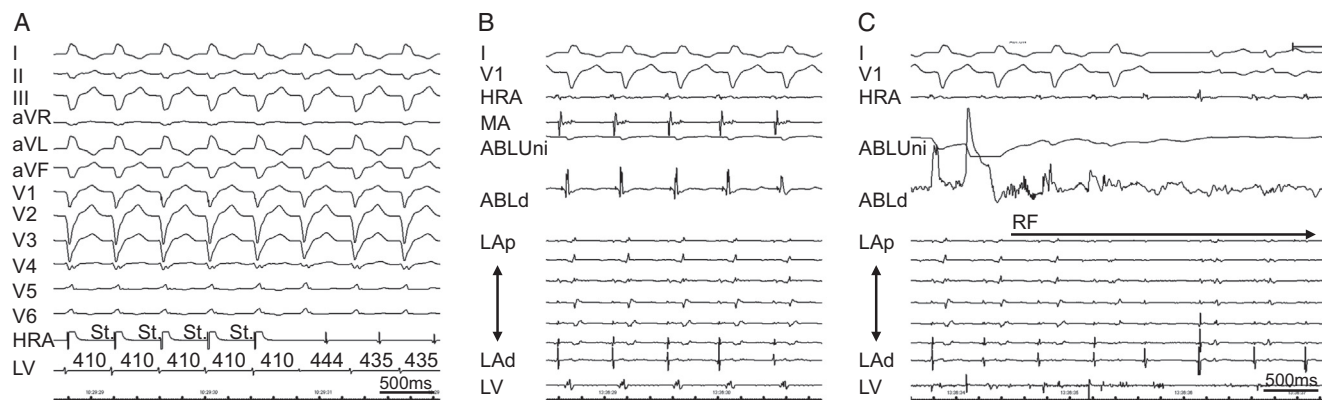
There are some limitations to this study. First, a His bundle electrogram could not be recorded because of the abnormal location of the AV nodes. Second, electrophysiological study in sinus rhythm was not systematically performed because of incessant BBRT before RF ablation. Finally, we did not investigate dyssynchrony of the systemic ventricle after the ablation procedure. However, our patient did not have exacerbation of congestive heart failure during the 6 months of follow-up.



**Figure 1** A 69-year-old patient with congenitally corrected transposition of the great arteries presenting a wide QRS regular tachycardia. **A:** Wide QRS tachycardia accompanied by left bundle branch block morphology with a leftward axis, QRS duration of 139 ms, and cycle length of 462 ms. **B, C:** Sinus rhythm shows 2 discrete non-preexcited morphologies with an inferior axis and a superior axis. The QRS duration was 102 ms. **D:** A surface electrocardiogram after ablation shows a right bundle branch block pattern with a superior axis in sinus rhythm. **E:** Anatomy of the heart in a patient with congenitally corrected transposition of the great arteries obtained by contrast-enhanced computed tomography in the AP projection. The green chamber is the right atrium. The blue chambers are the right-sided anatomic left ventricle and pulmonary artery. The yellow chamber is the left atrium behind the other chambers. The pink chambers are the left-sided anatomic right ventricle and ascending aorta. **F:** Position of catheters at an electrophysiological study in the AP projection. **G:** Tachycardia circuit estimated by electrophysiological study. Ao = aorta; AP = anterior-posterior projection; HRA = high right atrium; LA = left atrium; LV = left ventricle; MA = posteroseptal mitral annulus; PA = pulmonary artery; PT = pulmonary trunk; RA = right atrium; RV = right ventricle.



**Figure 2** Atrial activation patterns during the wide QRS tachycardia and entrainment from the left ventricular apex. **A:** Wide QRS tachycardia with single morphology was observed, but there were 3 patterns of atrial activation sequence and cycle length. **B:** Ventriculoatrial dissociation occurred during wide QRS tachycardia on an intracardiac electrocardiogram. **C:** Ventriculoatrial dissociation was present during entrainment pacing from the left ventricular apex. **D:** Entrainment pacing at a cycle length of 420, 400, and 300 ms from the left ventricular apex shows progressive fusion. CL = cycle length; HRA = high right atrium; LAp = proximal left atrium; LAd = distal left atrium; LV = left ventricle; MA = posteroseptal mitral annulus; St = stimulation.



**Figure 3** Entrainment from the high right atrium and termination of the wide QRS tachycardia by ablating the left bundle branch. **A:** QRS morphology in the surface electrocardiogram did not change during entrainment pacing at a cycle length of 410 ms from the high right atrium. **B:** Left bundle branch potential was recorded at the earliest site of ventricular activation on ABLd electrodes, which preceded the onset of QRS by 40 ms. **C:** Radiofrequency ablation terminated the tachycardia with complete antegrade block in the left bundle branch. ABLd = bipolar electrogram of an ablation catheter; ABLUni = unipolar electrogram of an ablation catheter; HRA = high right atrium; LAd = distal left atrium; LAp = proximal left atrium; LV = left ventricle; MA = posteroseptal mitral annulus; RF = radiofrequency; St = stimulation.

## References

1. Caceres J, Jazayeri M, McKinnie J, Avital B, Denker ST, Tchou P, Akhtar M. Sustained bundle branch reentry as a mechanism of clinical tachycardia. *Circulation* 1989;79:256–270.
2. Li YG, Grönefeld G, Israel C, Bogun F, Hohnloser SH. Bundle branch reentrant tachycardia in patients with apparent normal His-Purkinje conduction: the role of functional conduction impairment. *J Cardiovasc Electrophysiol* 2002;13:1233–1239.
3. Cohen TJ, Chien WW, Lurie KG, Young C, Goldberg HR, Wang YS, Langberg JJ, Lesh MD, Lee MA, Griffin JC. Radiofrequency catheter ablation for treatment of bundle branch reentrant ventricular tachycardia: results and long-term follow-up. *J Am Coll Cardiol* 1991;18:1767–1773.
4. Merino JL, Carmona JR, Fernández-Lozano I, Peinado R, Basterra N, Sobrino JA. Mechanisms of sustained ventricular tachycardia in myotonic dystrophy: implications for catheter ablation. *Circulation* 1998;98:541–546.
5. Daliento L, Corrado D, Buja G, John N, Nava A, Thiene G. Rhythm and conduction disturbances in isolated, congenitally corrected transposition of the great arteries. *Am J Cardiol* 1986;58:314–318.
6. Merino JL, Peinado R, Fernández-Lozano I, Sobrino N, Sobrino JA. Transient entrainment of bundle-branch reentry by atrial and ventricular stimulation: elucidation of the tachycardia mechanism through analysis of the surface ECG. *Circulation* 1999;100:1784–1790.
7. Anderson RH, Becker AE, Arnold R, Wilkinson JL. The conducting tissues in congenitally corrected transposition. *Circulation* 1974;50:911–923.
8. Kurosawa H, Becher AE. Atrioventricular Conduction in Congenital Heart Disease. In: *Surgical Anatomy*. Tokyo, Berlin, New York: Springer-Verlag; 1987.
9. Tchou P, Jazayeri M, Denker S, Dongas J, Caceres J, Akhtar M. Transcatheter electrical ablation of right bundle branch. A method of treating macroreentrant ventricular tachycardia attributed to bundle branch reentry. *Circulation* 1988;78:246–257.
10. Langberg JJ, Desai J, Dullet N, Scheinman MM. Treatment of macroreentrant ventricular tachycardia with radiofrequency ablation of the right bundle branch. *Am J Cardiol* 1989;63:1010–1013.
11. Blanck Z, Deshpande S, Jazayeri MR, Akhtar M. Catheter ablation of the left bundle branch for the treatment of sustained bundle branch reentrant ventricular tachycardia. *J Cardiovasc Electrophysiol* 1995;6:40–43.
12. Schmidt B, Tang M, Chun KR, Antz M, Tilz RR, Metzner A, Koektuerk B, Xie P, Kuck KH, Ouyang F. Left bundle branch-Purkinje system in patients with bundle branch reentrant tachycardia: lessons from catheter ablation and electroanatomic mapping. *Heart Rhythm* 2009;6:51–58.