

---

## Bundle Branch Re-Entry Tachycardia in a Patient with a Corrected Atrioventricular Septal Defect

Reinder Evertz<sup>1\*</sup>, Mohamed Boulaksil<sup>1</sup>, Rypko Beukema<sup>1</sup>, Sjoerd Westra<sup>1</sup> and Kevin Vernooy<sup>1,2</sup>

<sup>1</sup>Department of Cardiology, Radboud University Medical Center, Geert Grooteplein Zuid, Nijmegen, The Netherlands

<sup>2</sup>Department of Cardiology, Cardiovascular Research Institute Maastricht 2 (CARIM), Maastricht University Medical Center, Maastricht, The Netherlands

\***Corresponding author:** Reinder Evertz, Radboud University Medical Center, Geert Grooteplein Zuid 10, 6525 GA Nijmegen, The Netherlands.

**Received:** September 08, 2023; **Accepted:** October 15, 2023; **Published:** November 05, 2023

### Abstract

A rare case of bundle branch re-entry tachycardia (BBRT) and scar related VT in a patient after surgical correction of a partial atrioventricular septal defect (AVSD) is presented in this case report. Electrophysiological diagnosis and therapy are shown.

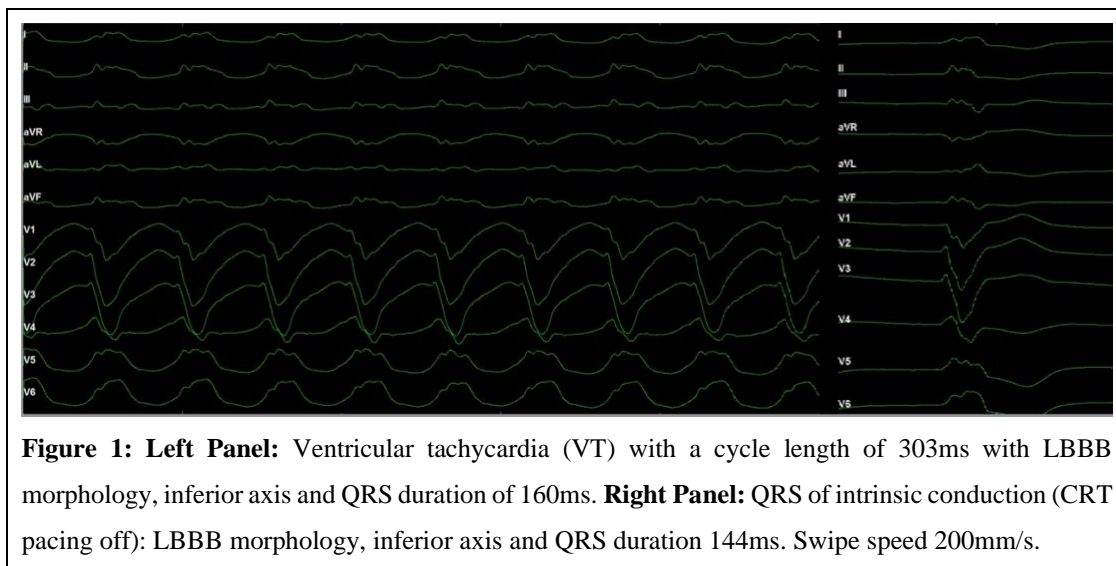
**Keywords:** Bundle branch re-entry; Ventricular tachycardia; Atrioventricular septal defect; Ablation; Evoked delayed potentials

### Case Report

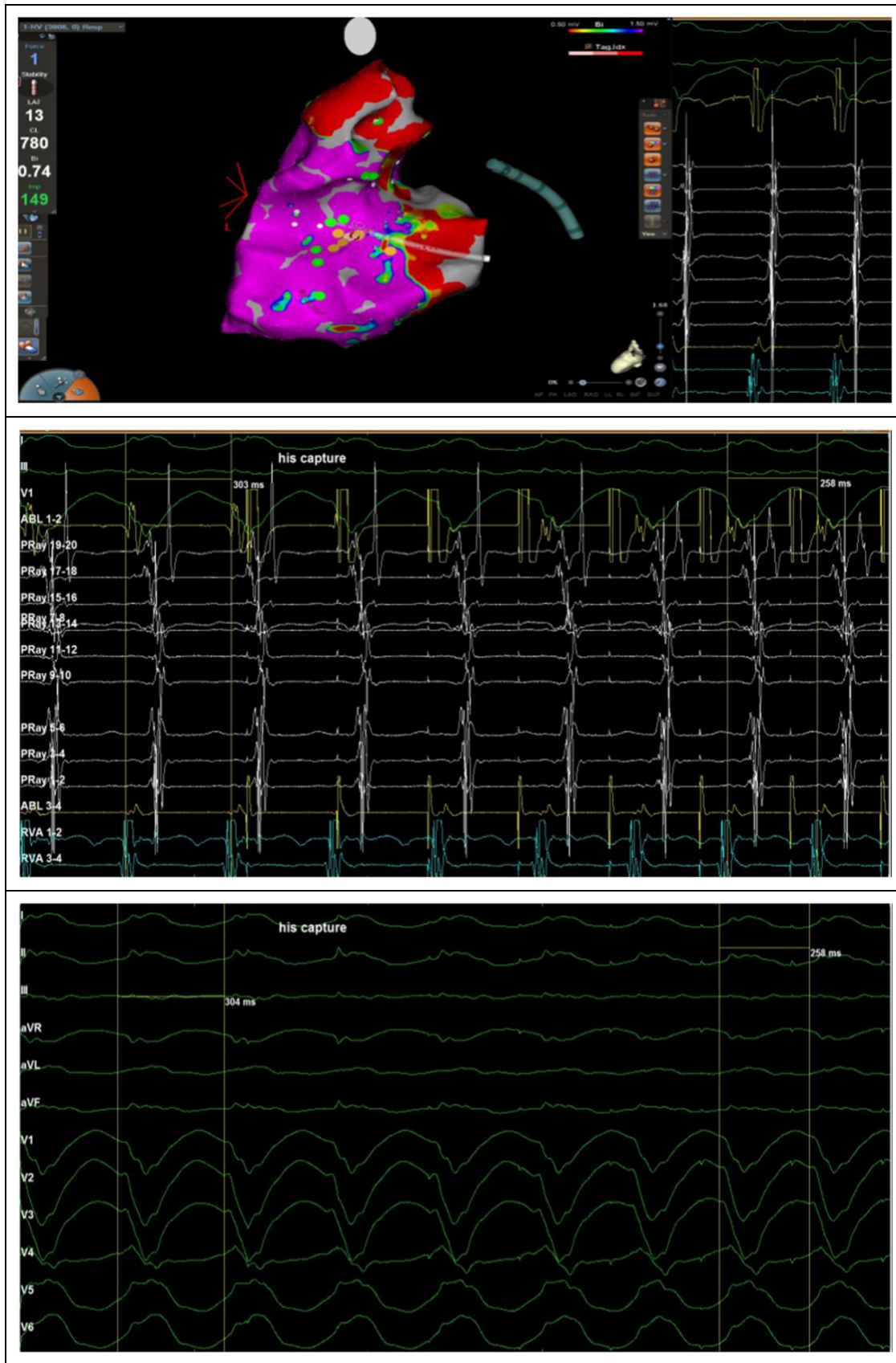
A 55-year-old male patient was referred to our center for ablation of a ventricular tachycardia (VT). By then, he has had several ICD therapies: ATP and shock because of VTs. The patient had a history of surgical correction of a partial AVSD at young age and had undergone ablation for atrial fibrillation and atypical flutter, but the atrial fibrillation became chronic afterwards. Furthermore, two years ago, a cardiac resynchronization device and defibrillator (CRTD) was implanted because of VTs and an impaired systolic left ventricular function. Moreover, a left sided atrioventricular valve plasty was performed because of severe valvular regurgitation. Prior to surgery, coronary angiography showed no coronary artery disease. His systolic left ventricular function improved afterwards, with an estimated ejection fraction of 50%, and mild residual valvular regurgitation. Nevertheless, he still suffered from several episodes of ICD therapy for VT (both ATP and shock). No twelve lead ECG of any VT was documented, device interrogation showed VTs with a cycle length of around 280-300ms. His medical treatment consisted of oral anticoagulation, a beta blocker at maximum tolerated dosage and an ACE-inhibitor. In line with the preference of both the patient and his treating physician, no antiarrhythmic drug was started. After informed consent the electrophysiological study and ablation was planned.

The electrophysiological study was performed under general anesthesia using the CARTO 3D mapping system (Biosense Webster, V7, J&J) and Workmate Claris electrophysiological registration system (Abbott). At baseline two distinct and presumed clinical VTs could be induced. The first with a right bundle branch block morphology (RBBB), superior axis and a cycle length of 290ms and the second with a left bundle branch block morphology (LBBB), inferior axis and a cycle length of 300ms. Because of hemodynamic instability during VT, activation mapping was not possible. The latter VT could easily be terminated with overpacing, the first VT, however, had to be terminated with external defibrillation. Next, we performed substrate mapping during intrinsic conduction (CRT pacing off) making a voltage map and tagging evoked delayed potentials (EDP) for ablation [1-3]. The left ventricle showed a small, patchy area of low voltage at the septum with a few EDPs at the anterior border zone which were subsequently ablated. Purkinje potentials presenting the left bundle in the septum were also tagged. At the basal to mid portion of the inferolateral wall, a larger area of low voltage was found with several EDPs at the border zone. After successful ablation of all EDPs at this inferolateral scar area, the first VT with RBBB morphology could no longer be induced.

The second VT with LBBB morphology was still inducible. Further mapping of the left ventricle did not show any additional targets for ablation. Furthermore, a voltage map of the right ventricle did not show any areas of low voltage either. Purkinje potentials presenting the right bundle were tagged. Pace mapping, both in the right and the left ventricle could not elucidate a clear area of interest. After reinduction of the VT, it was noticed that the VT had a similar morphology as the intrinsic conducted QRS, albeit the VT QRS being slightly broader (Figure 1).

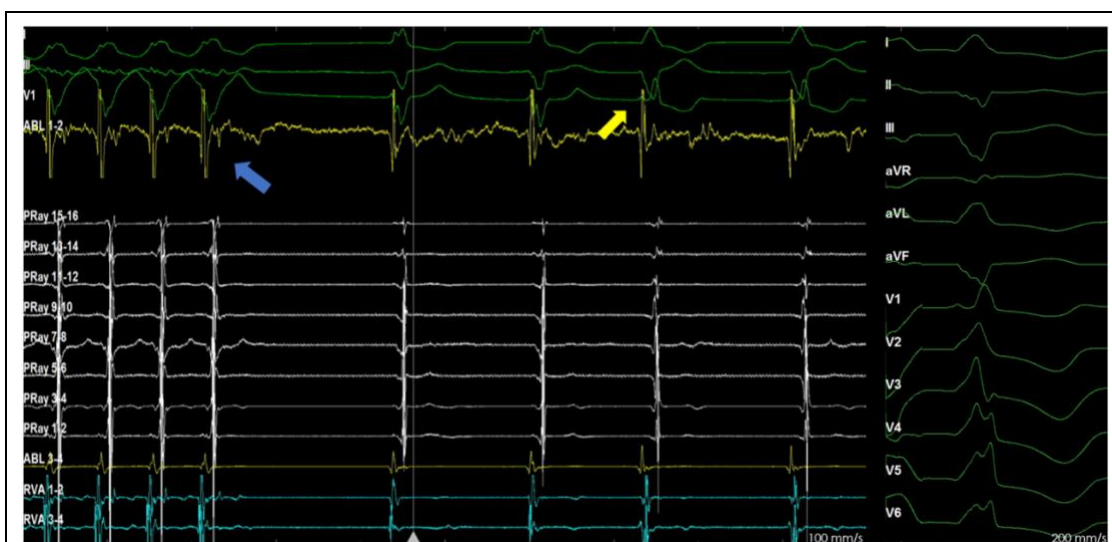


Thereafter, we positioned the ablation catheter at the right bundle (RB) and reinduced the tachycardia (Figure 2, Top panel, left side). During tachycardia, a clear right bundle potential preceding every ventricular electrogram (V) was appreciated (Figure 2, Top panel, right side). During entrainment pacing capturing the right bundle, showed concealed fusion on the 12 lead ECG (Figure 2, Mid and Bottom panel). RB-V interval during VT was similar to RB-V interval during intrinsic conduction (56ms).



**Figure 2: Top Panel:** On the left side CARTO voltage map of the right ventricle (posterior or septal view) with the ablation catheter positioned at the right bundle (RB) marked with yellow dot, showing, on the right side, a RB potential preceding every ventricular electrogram during VT. **Mid Panel:** Intracardiac electrograms showing entrainment pacing at 260ms capturing the RB (marked as 'his capture', from the 6<sup>th</sup> QRS complex onwards). **Bottom Panel:** 12 lead ECG showing concealed fusion during entrainment pacing at the RB. Swipe speed 200mm/s. Surface ECG in green. Ablation catheter (ABL) marked in yellow. Multi-electrode catheter marked in white (PRay), and positioned in the left ventricle. Quadripolar catheter (RVA), marked in blue in the right ventricular apex.

Hence, the diagnosis of a bundle branch reentry tachycardia (BBRT) was made. Radiofrequency ablation at the RB terminated the VT within 8 seconds (Figure 3, blue arrow), after which a clear right bundle branch block appeared (Figure 3, yellow arrow). Thereafter, no VT could be reinduced anymore. After a waiting period of around 20 minutes, still with non-inducibility of any VT and persistent RBBB, the procedure was designated successful. Next day, the patient was discharged home after an uneventful and successful ablation procedure.



**Figure 3: Left Panel:** Radiofrequency ablation at the right bundle terminated the VT within 8 seconds (blue arrow) after which a right bundle branch block (with left axis deviation) appeared (yellow arrow). **Right Panel:** 12 lead ECG showing RBBB at the end of the procedure. Swipe speed 100 and 200mm/s as indicated.

## Discussion

Both BBRT and AVSD rare, and to our knowledge this is the first case of a patient with BBRT after surgical correction of a partial AVSD. Moreover, the patient also had a scar-based VT and was ablated using different strategies. AVSD has an incidence of 4-5.3 per 10.000 live births and are mostly corrected surgically at young age. Reoperation, like in our patient, is reported in up to 28,9% of all patients. Overall survival is good, but patients are at increased risk for arrhythmias, mainly atrial [4].

BBRT has previously been described and is usually associated with underlying (structural) cardiac disease. Dilated cardiomyopathy and prior valve surgery are among the most common predisposing factors, though it has also been described in ischemic heart disease. Patients with BBRT commonly have conduction disturbances with a prolonged PR interval, QRS duration and HV interval [5]. Our patient was in atrial fibrillation but did have a wide QRS at baseline with a LBBB morphology. HV interval was not measured at baseline, but the RB to V interval was rather long (56ms). Three types of BBRT have been described in literature of which type A with an LBBB morphology is the most common. Our patient had a type A BBRT of which the anterograde activation proceeds through the RBB, goes leftward through the septum, retrogradely conducted via the LBB up to the septal summit and His bundle, responsible for the LBBB QRS morphology [5]. We did find a single case report of a BBRT in a patient with an atrial septal defect, and in whom the ablation of the right bundle also was curative for the VT [6]. Ablation of the right bundle or left bundle in patients with a BBRT has good acute and long term success rates. Pathak et al. reported targeting the right bundle in almost 60% of patients with a BBRT and with a mean follow-up duration of  $95 \pm 36$  months no recurrence of VT was documented. They preferably targeted the RB for ablation, resulting in only one patient with complete heart block. Nevertheless, the majority of patients already were protected with a pacing device. In patients without pacing device (25%) the LB was targeted [7]. Our patient did not develop complete heart block, but the RBBB was still present when CRT pacing was activated upon clinical discharge.

Because the patient had CRT pacing at baseline, it wasn't until halfway through the procedure that we realized the QRS morphology of the VT was similar to the intrinsically conducted QRS morphology. This emphasizes the importance of being aware of all the data of a patient and the fact that in patients referred for VT ablation it can be very useful, besides an ECG of the clinical VT if available, to have an ECG without pacing at baseline.

We incorporated different ablation strategies with successful substrate ablation based on EDP elimination of the inferolateral scar, treating one of the two induced VTs. Pace-mapping was used but in the end appeared not very helpful, and only after the recognition of the resemblance of the VT with intrinsic conduction we were able to diagnose the BBRT with a typical type A morphology correctly. Finally, mapping and ablation of the right bundle was successful rendering the patient to be non-inducible for any VT at the end of the procedure.

## Conclusions

We presented a rare case of both BBRT and scar-based VT in a patient with a congenital heart defect of AVSD. The ablation procedure was successful using different ablation strategies of substrate ablation, pace mapping and entrainment mapping and finally ablation of the RB.

## REFERENCES

1. De Riva M, Naruse Y, Ebert M, et al. Targeting the Hidden Substrate Unmasked by Right Ventricular Extrastimulation Improves Ventricular Tachycardia Ablation Outcome After Myocardial Infarction. *JACC Clin Electrophysiol.* 2018; 4: 316-327.
2. Jackson N, Gizurarson S, Viswanathan K, et al. Decrement Evoked Potential Mapping Basis of a Mechanistic Strategy for Ventricular Tachycardia Ablation. *Circulation-Arrhythmia and Electrophysiology.* 2015; 8: 1433-1442.



3. Porta-Sánchez A, Jackson N, Lukac P, et al. Multicenter Study of Ischemic Ventricular Tachycardia Ablation with Decrement-Evoked Potential (DEEP) Mapping with Extra Stimulus. *JACC Clin Electrophysiol.* 2018; 4: 307-315.
4. Calkoen EE, Hazekamp MG, Blom NA, et al. Atrioventricular septal defect: From embryonic development to long-term follow-up. *Int J Cardiol.* 2016; 202: 784-795.
5. Romero J, Santangeli P, Pathak RK, et al. Bundle branch reentrant ventricular tachycardia: review and case presentation. *J Interv Card Electrophysiol.* 2018; 52: 385-393.
6. De Lima GG, Dubuc M, Roy D, et al. Radiofrequency ablation of bundle branch reentrant tachycardia in a patient with atrial septal defect. *Can J Cardiol.* 1997; 13: 403-405.
7. Pathak RK, Fahed J, Santangeli P, et al. Long-Term Outcome of Catheter Ablation for Treatment of Bundle Branch Re-Entrant Tachycardia. *JACC Clin Electrophysiol.* 2018; 4: 331-338.