Idiopathic Accelerated Idioventricular Rhythm or Ventricular Tachycardia Originating from the Right Bundle Branch: An Unusual Type of Ventricular Arrhythmia

Running title: Chen et al.; Accelerated Rhythm or VT from the RBB

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Abstract:

**Background** - Accelerated idioventricular rhythm (AIVR) or ventricular tachycardia (VT) originating from the right bundle branch (RBB) is rare and published clinical data regarding such arrhythmia is scarce. In this study, we will describe the clinical manifestations, diagnosis and management of a cohort of patients with this novel arrhythmia.

**Methods and Results** - Eight patients (5 males; median age 25 years) with RBB-AIVR/VT were consecutively enrolled in the study. Pharmacologic testing, exercise treadmill testing, electrophysiological study and catheter ablation were performed in the study patients and ECG features were characterized. All RBB-AIVR/VTs were of typical LBBB morphology with atrio-ventricular dissociation. The arrhythmias demonstrated chronotropic variability, were often isorhythmic with sinus rhythm, and were accelerated by physical exercise, stress, and intravenous isoprenaline infusion. The rate of RBB-AIVR/VT varied from 45 to 200 bpm. Two patients experienced syncope and three had impaired LV function. Metoprolol was proven to be the most effective drug to decelerate the arrhythmia rate and relieve symptoms.

Electrophysiology study was performed in 5 patients and the earliest activation with a sharp RBB potential was localized in the mid or distal RBB area. Catheter ablation terminated the arrhythmia with subsequent RBBB morphology during sinus rhythm. During follow-up, patients’ symptoms were controlled with normalization of LV function either on metoprolol or by catheter ablation.

**Conclusions** - RBB-AIVR/VT is an unusual type of ventricular arrhythmia. It can result in significant symptoms and depressed ventricular function and can be successfully treated with catheter ablation.

**Key words:** ventricular tachycardia, electrophysiology, catheter ablation, accelerated idioventricular rhythm, right bundle branch
Introduction

Accelerated idioventricular rhythm (AIVR) is traditionally defined as an ectopic rhythm with more than three consecutive premature beats, with gradual onset and gradual termination and usually competitive with the sinus rhythm. The rate of AIVR, usually below 120 bpm, is normally faster than the usual ventricular intrinsic escape rate of 30 to 40 beats/min (bpm), but slower than the rate of most ventricular tachycardias (VT). AIVR, which is an uncommon ectopic rhythm, can sometimes be seen in infants or children and is regarded as a benign arrhythmia. In adults, however, it is often transiently encountered in patients with acute myocardial infarction during the reperfusion period following thrombolysis, or in the setting of post-resuscitation, digitalis intoxication or under anesthesia. In rare circumstances, AIVR can occur in patients with structurally normal hearts without any identifiable reasons and is regarded as truly “idiopathic”. Although the most common tachyarrhythmias from the left His-Purkinje system are intra or inter fascicular reentries or focal Purkinje ventricular tachycardias, idiopathic AIVR in adult patients originating from the left His-Purkinje system and with benign clinical course has also been described. On the right side, however, the most commonly reported AIVR is automatic activity originating from Mahaim fibers. Only recently have several case reports described focal VT or AIVR of right His-Purkinje system origin. There is, however, very limited data regarding drug treatment and catheter ablation of AIVR in a large cohort of patients. In this study, we describe 8 adult patients with idiopathic and persistent AIVR or VT originating from the right bundle branch or the moderator band area (RBB-AIVR/VT). The clinical course, ECG characteristics, drug therapy and catheter ablation in such patients are also described.
Methods

Study Population

From September 2009 to January 2013, 8 consecutive patients with AIVR/VT of identical left bundle branch block (LBBB) QRS morphology were included in the study. Their referral for clinical and electrophysiological assessment was due to persistent or intermittent AIVR/VT with or without symptoms recorded from surface ECG or Holter monitoring. Patient histories, routine testing such as echocardiography and 24 hour Holter monitoring were acquired during hospitalization. After written informed consent was signed, all patients agreed to undergo antiarrhythmic drug testing, provocative drug testing, exercise stress testing, and electrophysiology study.

Provocative Drug Tests and Treadmill Stress Tests

Anti-arrhythmic drugs (AAD) were discontinued for at least 5 half-lives before any testing was performed. The isoprenaline (Iso) provocation test was performed in a fasting state and supine position with defibrillator and intravenous AAD standby. The Iso infusion was stopped in the following situations: blood pressure (BP) increase over 180/120 mmHg or sudden drop below 90/60 mmHg, and/or development of sustained ventricular arrhythmia. During Iso provocation test, ECGs were recorded at baseline, Iso 1 μg/min, 2 μg/min, 3 μg/min, 4 μg/min, and every 2 minutes during the first 10 minutes of recovery and every 5 minutes during the following 20 minutes of recovery.

To confirm the influence of exercise on RBB-AIVR/VT, exercise stress testing was performed using the modified Bruce protocol using a treadmill system (General Electric, USA), with defibrillator and intravenous AAD standby. The stress test was stopped in the case of reaching target heart rate or the occurrence of sustained ventricular arrhythmias. During the
stress test, ECGs were recorded at baseline, each minute during exercise and each minute within the first 6 minutes of recovery.

The efficacy of AADs was evaluated by either reviewing the effect of previously used oral and/or intravenous AAD, or direct assessment of the sensitivity of intravenous or oral AAD during hospitalization.

**Electrophysiological Study**

For patients with persistent AIVR/VT, an electrophysiological study and catheter ablation was performed under conscious sedation and local anesthesia. Venous access was obtained from the femoral veins. A decapolar catheter (Synaptic Ltd, Beijing, China) was placed at the HIS-RBB position in the right ventricle (RV). A deflectable quadripolar open irrigated catheter (Navi-star, Biosense Webster) was inserted into the right ventricle for mapping and ablation. Endocardial activation maps were acquired during sinus rhythm, AIVR/VT and atrial incremental pacing to identify the His-right bundle activation sequence and timing. Intracardiac electrograms were recorded using a digital electrophysiological recording system (Prucka CardioLab, General Electric Health Care System Inc., Milwaukee, WI, USA or Bard Electrophysiology system, MA, USA) and were filtered from 30-300 Hz.

**Three Dimensional Electroanatomical Mapping and Catheter Ablation**

Detailed electroanatomic mapping of the RV was performed by obtaining contact bipolar electrograms (100-150 points, the timing of the sampled electrogram was set at the onset of the ventricular electrogram or the RBB potential) during AIVR/VT guided by three-dimensional (3-D) electroanatomic mapping using a CARTO system (Biosense Webster Inc.). Radiofrequency energy was delivered to the earliest activation point using an irrigated tip catheter (Navi-star, Biosense Webster Inc.) with a maximal temperature of 45 °C at a maximal power of 35 W and
infusion rate of 17mL/min. The ablation endpoint was the termination of AIVR/VT during RFCA with non-inducibility with and without drug provocation afterwards.

**Post Discharge Follow-up**

Antiarrhythmic agents were discontinued following a successful ablation procedure. In patients without ablation, AAD drug therapy was initiated. Regular out-patient follow-up visits were performed in all patients. A surface ECG and 24-hour Holter recording were performed after the procedure at one month and every six months thereafter. Furthermore, ECG or Holter recording was also performed at the time of any reported symptoms. Transthoracic echocardiography was also performed after the procedure at every 3 months during the follow-up period in patients with impaired LV function. Patients who had a documented recurrence of persistent RBB-AIVR/VT after ablation underwent a repeat procedure.

**Statistical Analysis**

Due to the small sample size of this study, the continuous variables were likely to be non-normally distributed. Therefore, these data were reported as median along with interquartile range (IQR).

**Results**

**Patients Characteristics**

Eight patients (5 males; median age 25 years, IQR, 18 to 28), who were diagnosed with AIVR/VT originating from the RBB, were enrolled in the study (Table-1). Palpitation was the most common symptom reported and was experienced by all patients. Two patients had a history of presyncope and/or syncope. The clinical arrhythmia was intermittent in 4 patients and persistent in 4 patients, respectively. Three of the 4 patients with persistent arrhythmias developed exertional dyspnea and were noted to have impaired left ventricular ejection fraction (LVEF) and
an enlarged left ventricle (LV). The estimated median (IQR) LVEF and LV end diastolic dimension (LVED) was 64.1% (48.8 to 67.8) and 48 mm (46 to 57), respectively. The AIVR/VT burden was defined as the proportion of ectopic ventricular beats over the total number of QRS complexes during 24-hour Holter recording. Membranous ventricular septal aneurysm and right coronary Valsalva sinus aneurysm with mirror-image dextrocardia was detected by echocardiography and CT angiography in patient 8. None of the patients had a family history of heart disease or sudden cardiac death, and no patient had cardiac risk factors, such as hypertension or diabetes.

**Features of the 12-lead ECG**

RBB-AIVR/VT in all patients demonstrated an identical LBBB QRS morphology in individual patient (Figure-1, Table-1). The axis in the inferior leads was not fixed, but varied from patient to patient. The median (IQR) RS interval (between the initial deflection of r wave and the nadir of the S wave) was 50 ms (38 to 57) and the median (IQR) QRS width was 129 ms (112 to 144) in lead V1, with the precordial transition lead at V4 or V5.

Based on Holter recordings, three types of RBB-AIVR/VT behavior were noted (Table-1): 1) persistent and dominant (AIVR/VT was the dominant rhythm with occasional sinus capture beats); 2) persistent and competitive (AIVR/VT is frequent and alternating or isorhythmic with sinus rhythm); 3) intermittent and competitive (sinus rhythm is the dominant rhythm, competing with short runs of AIVR). Five of the patients were found to have an AIVR/VT burden of greater than 20%.

**Provocative Drug Tests and Stress Tests**

Persistent AIVR/VT was noted to easily accelerate rate with great extent during exercise, stress and fever (Figure-2). Intermittent AIVR patients received Iso provocative testing and treadmill
stress testing. Acceleration of the arrhythmia rate was noticed during provocative testing, but competing with the sinus rhythm at a higher rate (Figure-3). The rate variability of AIVR competing with sinus rhythm differed from patient to patient, but was normally suppressed by the latter when the sinus rate was over 130 bpm.

**Sensitivity of RBB-AIVR/VT to Antiarrhythmic Drugs and Cardioversion**

The sensitivity of RBB-AIVR/VT to AAD in each patient is shown in Table-2. Briefly, persistent RBB-AIVR/VT could be slowed but not terminated by intravenous amiodarone, propafenone and metoprolol, and patients’ symptoms were alleviated. However, these rhythms did not respond to lidocaine and verapamil administration. In patient 1, who was hemodynamically unstable due to extremely rapid AIVR/VT, external direct current cardioversion failed to terminate and, in fact, accelerated the tachycardia with resulting in worsened clinical state. In patients with intermittent RBB-AIVR/VT, only propafenone, verapamil and metoprolol were attempted. The burden of AIVR was reduced after oral administration of metoprolol, but seemed resistant to propafenone and verapamil. No complete suppression of AIVR was observed after drug therapy in the study patients. In patient 8, during recovering period after treadmill test, AIVR was transiently suppressed by 5mg of intravenous metoprolol.

**Electrophysiological Findings and Catheter Ablation**

Five patients whose RBB-AIVR/VT was persistent or the burden was over 20% underwent an electrophysiology study. Among them, four patients had simultaneous catheter ablation during the index procedure. Spontaneous AIVR/VT was observed at baseline with a median (IQR) cycle length of 620 ms (570 to 814) in all five patients. Bolus injection of adenosine triphosphate (20 mg) was administered in three patients with fast AIVR/VT, and although termination did not occur, AIVR/VT was overdriven by subsequent accelerated sinus rhythm transiently. Multipolar
endocardial recordings during AIVR/VT showed a shortened HV interval and RBB activation dramatically preceded the local ventricular electrogram, with right bundle activation in a retrograde pattern (Figure-4 and Figure-5). During sinus capture, however, the His-RBB activation proceeded in an antegrade pattern from proximal to distal activation. Atrial overdrive pacing could suppress AIVR/VT with narrow QRS and normal His-RBB conduction. A detailed electroanatomic 3-D mapping was obtained during AIVR/VT in four patients. The earliest Purkinje potential was found in the mid or distal RBB area. Pacing mapping match scores at these points were poor. However, the delivery of radiofrequency energy at the earliest activation point immediately terminated AIVR/VT, and sinus rhythm was restored with RBBB morphology after termination of the arrhythmia. The local Purkinje activation at the target area preceded the onset of surface QRS by a median (IQR) of 22 ms (18 to 32). Patient 8 had unsuccessful ablation due to challenging anatomy from dextrocardia with a membranous septal aneurysm and a right coronary Valsalva sinus aneurysm (Figure-6). The large aneurysms prevented adequate catheter contact around the sites of earliest activation point within the RBB.

In patient 4 and patient 5, RBB-AIVR/VT recurred the second day after ablation, but with RBBB morphology (Figure-4C). A repeat procedure was performed and ablation was successful at a more proximal site within the RBB region. No complications occurred during and after the ablation procedures.

Follow-up

All patients were treated with metoprolol after discharge except patient 2 and those three patients who had successful ablation. Patient 1, who declined a catheter ablation attempt for fear of conduction block, had a better rate control of AIVR/VT after oral metoprolol administration, however, two months post discharge, she died from complications of an unexplained and
uncontrolled infection. In patient 2, whose AIVR burden was very low, metoprolol was not used and she remained asymptomatic. In patient 6, the reduction of AIVR burden was very minor, but the symptoms were dramatically relieved by metoprolol medication. In patient 7 and patient 8, the AIVR burden was reduced by 8% and 22% on metoprolol, respectively. The impairment in LV function was completely reversed in three patients six months after successful ablation or treatment with metoprolol (LVEF: 60% in patient 3, 64% in patient 4 and 56% in patient 8).

Discussion

We describe an unusual type of ventricular arrhythmia (RBB-AIVR/VT) which should not be overlooked by cardiologists or electrophysiologists due to potentially insidious clinical results.

Clinical Manifestations and Potential Risks

As described in our study, RBB-AIVR/VT is generally idiopathic, and not associated with structural heart disease, except in one patient with dextrocardia and membranous septal and right coronary Valsalva sinus aneurysms. And, as in previous reports, there do not appear to be any clear predisposing factors to development of such arrhythmias.4-11

RBB-AIVR/VT has chronotropic properties and is often isorhythmic with sinus rhythm. The behavior of this ectopic rhythm can be classified into three types: persistent and dominant; persistent and competitive; intermittent and competitive. The prominent feature of this rhythm is that it is easily provoked by stress, physical exercise or other clinical states such as fever, anemia or hypotension. The inappropriate acceleration of this ectopic rhythm can lead to very rapid ventricular rates and resultant hemodynamic compromise. The most common symptom of this ventricular arrhythmia is palpitations, but more severe symptoms of pre-syncope, Adams-Stokes attacks and dyspnea were also observed. Another adverse impact of this arrhythmia is tachycardia induced cardiomyopathy in the setting of incessant arrhythmia or the higher burden
of AIVR. The mechanism of this is similar to other ventricular tachyarrhythmia related cardiomyopathy.\textsuperscript{22,23} In addition, due to its RBB origin, the ventricular activation mode is similar to that of LBBB and the inter or intra-ventricular dissynchrony might occur and could probably contribute to depressed ventricular function,\textsuperscript{24} especially in those patients with high AIVR burden. Because of inappropriate acceleration and the potential possibility of developing arrhythmia induced cardiomyopathy, it is important to emphasize that RBB-AIVR/VT should be treated vigorously either with drug or ablative therapy.

The ECG characteristics of RBB-AIVR/VT are very distinct: typical LBBB morphology with sharp downstroke of the S wave and short rS duration (\(<\ 70\) ms) in precordial leads V1 and V2. The precordial transition is very late, normally at V4 or V5. These features are consistent with initial ventricular activation via the His-Purkinje system.\textsuperscript{25-27} The ventricular activation is faster than and dissociated with atrial activation. Although cardiac orientation has impact on the ECG morphology, the other important reason contributing to the slight morphological difference of the RBB-AIVR/VT among the study patients is its proximal or distal origin. The proximal RBB origin captures the left fascicle earlier by retrograde conduction while the distal RBB origin propagates its activation more quickly over the myocardium of the interventricular septum to the left side.

**Diagnosis and the Possible Mechanism**

The ECG manifestations can strongly suggest that AIVR/VT is of RBB origin, but other diagnoses can not be completely excluded. Junctional rhythm with LBBB, bundle branch reentry (BBRT) using the RBB in an antegrade fashion, or Mahaim fiber automaticity\textsuperscript{14-17} are all possibilities. However, endocardial recordings and pacing maneuvers can distinguish these types of arrhythmias. Multipolar endocardial recordings along the His-RBB axis clearly demonstrated
that the HV was short and the RBB potential was markedly earlier than local ventricular activation, and activated in a retrograde direction. During sinus or atrial capture beats, the RBB activation direction was reversed. Similar findings were also presented in previously reported case studies.\textsuperscript{18-21} This activation sequence of the RBB ruled out the possibility of BBRT or junctional rhythm with LBBB because these rhythms should have an antegrade RBB activation pattern. Also, a sharp, low-amplitude, high frequency (mahaim-like potentials) was not found to precede the local ventricular activation around the tricuspid annulus. Therefore, automaticity from the atriofascicular fiber can be excluded in the 5 patients with invasive procedure. Furthermore, junctional focus should have an identical HV interval to that of the sinus rhythm, whilst BBRT usually has a longer HV interval. Successful ablation at the site of earliest RBB potential activation eliminated the arrhythmia with subsequent sinus rhythm and RBBB morphology, supporting that these rhythms are of RBB origin, either from the proximal or distal area (moderator band). Incremental atrial pacing could also overdrive the ventricle with subsequent narrow QRS complex and HV normalization which excluded the presence of Mahaim fibers.

The likely mechanism of RBB-AIVR/VT appears to be increased automaticity because of its focal origin and its chronotropic response, yet the underlying reason why it occurs remains unknown and needs further study.

\textbf{Management}

As shown in our study, RBB-AIVR/VT was not truly benign, therefore, this arrhythmia should be appropriately treated. Based on antiarrhythmic drug use in our patients, \(\beta\)-blocker should be the first line drug therapy for treatment of RBB-AIVR/VT. It can adequately slow RBB-AIVR/VT, subsequently relieving symptoms and improving the hemodynamic state.
Furthermore, β-blocker therapy can also reduce the AIVR burden or even terminate AIVR in some patients. For persistent or incessant RBB-AIVR/VT, amiodarone or propafenone could also be used intravenously to slow the rate. In emergencies, however, electrical cardioversion was ineffective, whilst intravenous sedation or even general anesthesia may be necessary with simultaneous proper antiarrhythmic drug therapy. Lidocaine and verapamil were of no use for such arrhythmias and therefore should be avoided.

Catheter ablation is a good option for arrhythmia treatment in such patients. In this study, patients who underwent ablation were those who experienced presyncope or syncope, had an arrhythmia burden of over 20% on Holter monitoring and those with concomitant cardiomyopathy.

**Study Limitations**

Entrainment pacing was not performed in all patients undergoing EP study, therefore, a potential reentrant mechanism cannot be entirely excluded. This is similar to the case of fascicular VTs encountered on the left side where entrainment pacing may not always be possible. Also, atrial and ventricular programmed stimulation to construct a resetting curve was not done to depict the responses of this focal activation to the extra stimuli. Such stimulation should be done in the future cases.

Intracardiac echo was not used to confirm the anatomic location of the origin of RBB-AIVR/VT. However, the target electrogram, 3-D map and the fluoroscopy were the strong references for the location. Only one case (patient 3) may have been from distal RBB or the moderator band origin (see the supplemental file).

Exercise testing and provocative drug testing were not performed in all patients.

Finally, the anatomical location and mechanism was inferred from the ECG findings in 3
patients without EP study.

Conclusions

RBB-AIVR/VT is an unusual form of ventricular arrhythmia. Although the clinical presentation may be relatively benign, a tendency towards more rapid ventricular rates and/or higher arrhythmia burden can worsen the clinical course. β-blockers are an effective drug therapy to alleviate clinical symptoms, and catheter ablation is a good option for an ultimate cure for such arrhythmias.

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Conflict of Interest Disclosures: None.

References:


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<th>Activation Behavior</th>
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AIVR = Accelerated Idioventricular Rhythm; VT = Ventricular Tachycardia; Pt No. = Patient Number; F = Female; M = Male; y = years; Syn/Presyn = Syncope/Presyncope; LV Dd = Left ventricular diastolic dimension; LVEF = Left ventricular ejection fraction; HR<sub>max</sub> = Maximum rate of AIVR; HR<sub>min</sub> = Minimum rate of AIVR; QRS width = Width of QRS complex; RS-V1 = RS interval in lead V1
Table 2: Sensitivity of RBB-AIVR/VT to antiarrhythmic drugs

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Meto = Metoprolol; Verap = Verapamil; Prop = Propafenone; Lido = Lidocaine; Amio = Amiodarone; ATP = Adenosine triphosphate; D = Decelerated, HR of AIVR was decelerated but not terminated; PI = partially inhibited; NR = non-response; NA = not available; BR = AIVR burden reduced

Figure Legends

Figure 1: Twelve lead ECG of RBB-AIVR/VT at baseline in all the subjects. Notice the ECG characteristics: typical LBBB morphology with short rS interval, sharp downstroke of S wave, relatively narrow QRS width in the precordial leads V1 and V2. Electrodes of limb lead I were connected in reverse due to dextrocardia in patient 8.

Figure 2: Baseline 12-lead ECG of persistent AIVR/VT (left panel) and its acceleration during
fever (right panel) in Pt-1. Note that the heart rate was exactly doubled during this febrile state.

**Figure 3:** A Intermittent AIVR (black arrow) during baseline and isorhythmic competition with sinus rhythm. B Acceleration of intermittent AIVR during intravenous Isoprenaline infusion in the same patient as in Figure-3A. Its competition with sinus rhythm was still evident, but at higher rate.

**Figure 4:** A Surface 12-lead ECG of RBB-AIVR/VT and endocardial recordings in Pt-4. Tracings are all the surface leads and His-RBB recordings from proximal to distal. The RAO fluoroscopy image demonstrates a decapolar catheter placed along the His-RBB axis. The activation sequence of AIVR/VT and sinus capture was reversed. A=atrium, H=HIS; RBB=right bundle branch; RAO=right anterior objection. B Target ablation electrograms (left column) and the termination of AIVR/VT (right column) during radiofrequency (RF) energy delivery. A sharp deflection preceded the earliest surface QRS by 32 msec. RF delivery quickly terminated the ectopic rhythm and the sinus beats are subsequently conducted with RBBB block morphology. The fluoroscopic anterior-posterior view and the electroanatomic 3-D map demonstrated the earliest activation was in the mid septal area of the right ventricle. C AIVR/VT recurred with a RBBB pattern the second day after ablation. Minor change of QRS morphology of sinus capture was observed (circled in black), suggesting the site origin was slightly proximal to the previous ablation site (see schematic representation). The slight different activation sequence of sinus capture contributes to the minor morphologic changes of QRS complex. Please notice the axis of the inferior leads was slightly shifted down, suggesting that sinus conducted beats captured the left fascicles a little bit earlier and activated the distal septum slightly faster compared with the
RBB beats. AVN=atrioventricular node; HIS=HIS bundle; LAF=left anterior fascicle; LPF=left posterior fascicle; RB=right bundle

**Figure 5:** Endocardial recordings of Pt-5 during sinus rhythm, AIVR, atrial pacing, RV mapping and ablation. Tracings are surface lead I, aVF, V1, V2 and endocardial recordings from high right atrium (HRAd and HRAp), coronary sinus from proximal to distal, right bundle branch from proximal to distal and mapping catheter (ABLd and ABLp). The activation sequence of HIS-RBB axis in sinus rhythm and atrial pacing was the same, whilst a reversed sequence was noticed during AIVR/VT. Note that the septal ventricular electrogram during sinus rhythm recorded from the high density mapping catheter showed two components. The solid arrow indicates the initial low frequency electrogram, probably suggesting the far field signal from the left septum; the dashed arrow points to the latter high frequency electrogram, suggesting the near field RV septum activation. The interesting finding was that the proximal RBB activation sequence remained unchanged after ablation with RBBB morphology, but the two components were slightly apart, suggesting the right septal myocardium was activated from the left side due to RBB block.

**Figure 6:** Chest X-ray and 3-D computer tomography (right) showing dextrocardia and electroanatomic mapping (left) in Pt-8. Notice the large membranous septal aneurysm (MSA) and right coronary sinus aneurysm (RCSA) protruding into the right ventricle (RV) cavity. HIS bundle recording from the mapping catheter showed a short HV during AIVR and normal HV during sinus rhythm. PA=pulmonary artery; Ao=aorta; LV=left ventricle; A=atrium; H=HIS bundle; V=ventricle
Baseline; 90 bpm

During fever; 180 bpm
Idiopathic Accelerated Idioventricular Rhythm or Ventricular Tachycardia Originating from the Right Bundle Branch: An Unusual Type of Ventricular Arrhythmia

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SUPPLEMENTAL MATERIAL
Supplemental History of Pt-3

- 26 year-old male who had a history of palpitation and chest tightness with presyncope
- No family history of hereditary heart disease
- Diagnosed as slow VT with well chronotropic function (HR 76~129bpm)
- Metoprolol and Propafenone could slow down but not terminate the VT, Verapamil and Lidocaine were proved to be no use
The target electrogram showed a small, sharp potential preceding the ventricular electrogram at the distal end of RBB, which is close to the moderator band area.
Fluoroscopy of VT Target

The ablation target was at the mid-distal septum, which is close to or in the moderator band area.
Post-procedure ECG

Incomplete RBBB after successful ablation
Summary

Origin:
Distal RBB or moderator band area

3-D map
Target electrogram with a sharp potential preceding the ventricular electrogram
Incomplete RBBB QRS after ablation
Fluroscopy