Catheter ablation of complex arrhythmic anomalies: Bayes syndrome, Wolff-Parkinson-White syndrome, atrial and dilated cardiomyopathy

Piotr J. Stec, MD,* Michał Chrabąszcz, BS,† Marek Ujda, MD,*
Radosław Krawczykiewicz, MD,* Janusz Śledź, MD,† Sebastian Stec, MD, PhD†‡

*From the *Specialist Hospital in Stalowa Wola, Stalowa Wola, Poland, †ELMedica EP-Network, Kielce, Poland, and ‡Medinice Research and Development Centre, Aeropolis-Jasionka, Rzeszow, Poland.*

Introduction

Bayes syndrome is a newly recognized phenomenon representing interatrial block (IAB) and risk of supraventricular arrhythmias. Advanced IAB is defined as a P-wave duration more than 120 ms with a biphasic P wave and negative final component in leads II, III, and aVF.[1,2] It shows as prolongation of an interatrial conduction period that usually manifests in the 12-lead electrocardiogram (ECG) as prolongation of the P wave (140 ms), splitting of P wave (into right atrial and left atrial activation), and high prevalence of supraventricular arrhythmias.[3]

On the contrary, electrocardiographic criteria of pre-excitation consist of sinus rhythm with short PR interval (<120 ms) with combination of delta wave, prolongation of QRS complex (>120 ms), and repolarization abnormalities.[4]

We present a case of complex arrhythmic substrate arrhythmias including atypical left atrial flutters (AFL) with wide QRS complexes with aberrancy, and occasionally with 1:1 conduction and recurrences of narrow QRS tachycardia—orthodromic atrioventricular reentry tachycardia (OAVRT). Coincidence of several substrates and entities led to the development of atrial and dilated cardiomyopathy[5,6] that resolved very quickly after a successful zero-fluoroscopy ablation of left-sided atypical AFL and accessory pathway (AP) via the patent foramen ovale.

KEYWORDS Atrial cardiomyopathy; Bayes syndrome; Catheter ablation; Dilated cardiomyopathy; Wolff-Parkinson-White syndrome
(Heart Rhythm Case Reports 2019;5:476–479)

Dr Stec is a co-founder/stockholder of Medinice S.A. and co-author of patents (Pace-Press, Cryoapplicator, Cathaio, Ep-Bioptom catheter, Mini-Max for nonfluoroscopic navigation and procedures); Dr Śledź is co-author of Ep-Bioptom for nonfluoroscopic navigation and procedures. No product of the company was used during procedures. The other authors declare no conflicts of interest. **Address reprint requests and correspondence:** Dr Piotr J. Stec, Specialist Hospital in StalowaWola, 37–450 Stalowa Wola, Aleje Jana Pawła II 13A/10, Poland. E-mail address: piotrstec@wp.eu.

KEY TEACHING POINTS

- Advanced interatrial conduction block (Bayes syndrome) may facilitate supraventricular arrhythmias and has impact on standard PQ interval and recognition of pre-excitation.
- Coincidence of pre-excitation, orthodromic tachycardia, and Bayes syndrome may rapidly progress to atypical atrial flutter, atriopathy, tachycardia-induced cardiomyopathy, and life-threatening conditions.
- Zero-fluoroscopy catheter ablation with complex substrates is feasible and may lead to complete resolution of several electrophysiologic abnormalities associated with Bayes and Wolff-Parkinson-White syndromes.

Case report

A 57-year-old white man was admitted to the tertiary intensive care unit owing to hemodynamically stable wide QRS tachycardia with periodically variable frequency from 140 to 280 beats per minute (Supplemental Figure 1).

The first diagnosis was ventricular tachycardia. Glucose, insulin, potassium, and magnesium infusion; metoprolol (5 mg intravenous [iv]); and then amiodarone (150 mg iv in bolus and 300 mg iv within 3 hours) were given. The patient was diagnosed as having severe dilated cardiomyopathy (left ventricular ejection fraction 25%, left ventricular end-diastolic dimension: 59 mm, left atrium: 45 mm).[5]

Within 10 minutes of amiodarone infusion, the signs and symptoms of cardiogenic shock were revealed and urgent direct external cardioversion was performed under general anesthesia. Urgent coronary angiography showed normal coronary arteries and flow. After coronary angiography, acute contrast-induced nephropathy was diagnosed.[7] One week after improvement in renal function, the patient was transferred for urgent electrophysiological study and ablation.

https://doi.org/10.1016/j.hrcr.2019.07.006

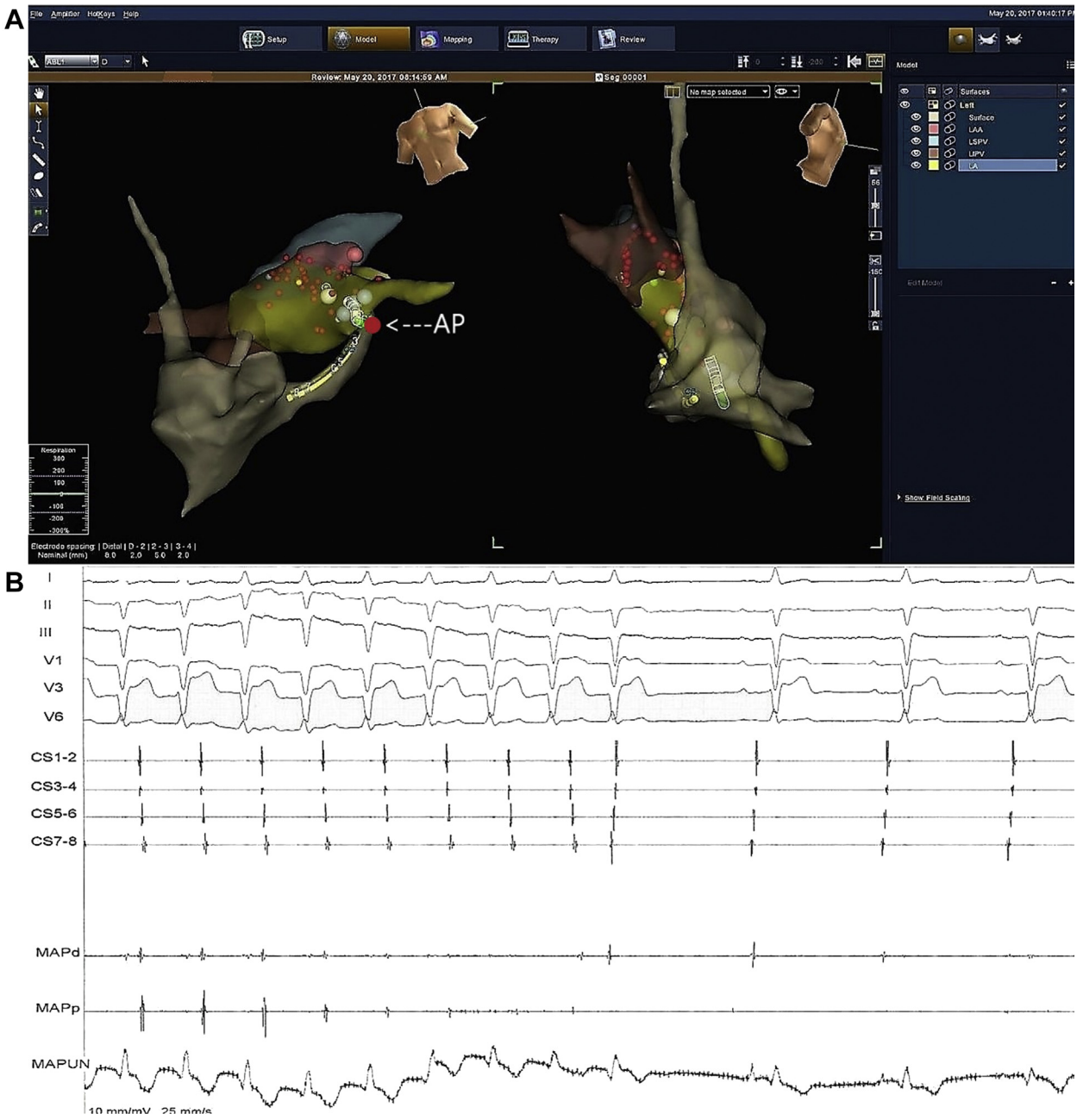

Figure 1 **A:** Simplified 3-dimensional electroanatomic mapping view. *Red dots* represent ablation point in ridge area at left superior pulmonary vein. After application in this area, tachycardia cycle length changed to 360 ms. Further applications within the isolation of the left pulmonary veins (*red dots*) jointly modulated the cycle into an orthodontic tachycardia (of cycle length 510 ms) with overt pre-excitation delayed owing to atrial conduction disturbances. Reverse tract with slight decay was observed by accessory pathway (AP) at hour 4:00 annulus mitral, resulting in disappearance of arrhythmia after application in this area (*gray dots*). *Yellow catheter* represents decapolar diagnostic catheter located in coronary sinus, *white with green tip* represents ablation catheter. **B:** Intracardiac electrocardiogram showing atrial tachycardia occurring during radiofrequency application.

Electrophysiological study and radiofrequency ablation

A simplified 2-catheter approach with femoral access was used. One quadripolar steerable mapping/ablation catheter (dynamic mapping) and 1 nonsteerable decapolar catheter (coronary sinus) (Biotronik, Berlin, Germany) were used (Figure 1).

At baseline, persistent nontypical AFL with 260 ms tachycardia cycle length (TCL) with 2:1 conduction and narrow QRS complexes was recorded. Several entrainments excluded AFL reentry circuit in the right atrium, superior vena cava, and proximal, medial, and distal coronary sinus. By using access via patent foramen ovale, left atrial mapping was continued with a small area of continuous fragmented signals with perfect

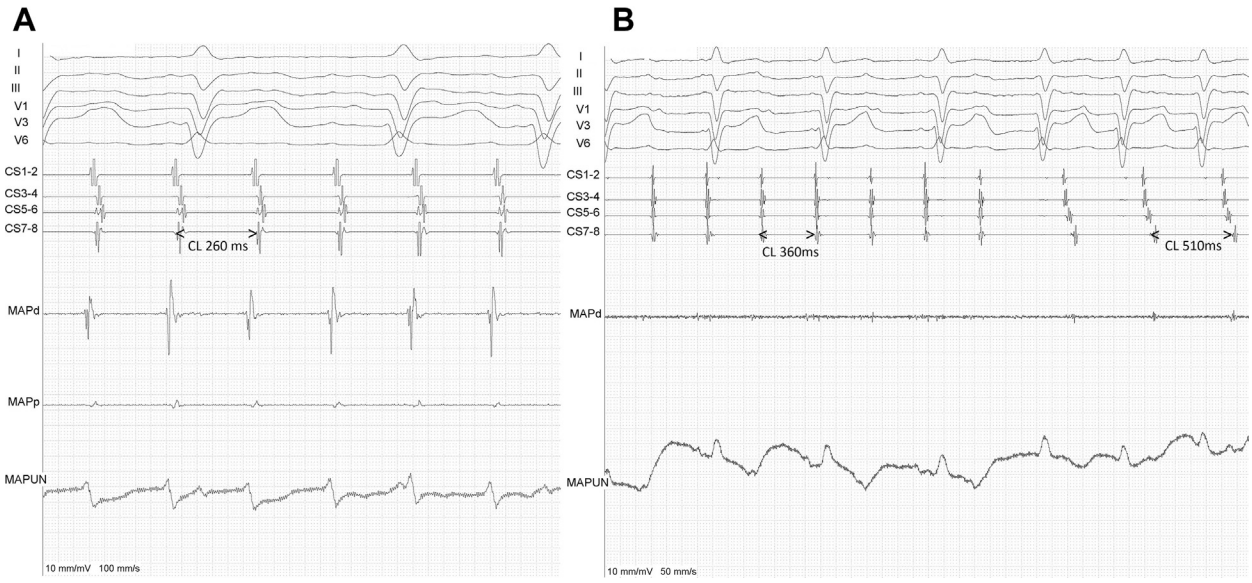

Figure 2 **A:** Intracardiac measurements of atypical atrial flutters (AFL) with cycle length (CL) 260 ms with microreentry in ridge area at left superior pulmonary vein. Radiofrequency (RF) energy delivered in this area changed cycle length to 360 ms. **B:** RF applications within the isolation of the left pulmonary veins jointly modulated the cycle length 360 ms atypical AFL into an orthodontic tachycardia of CL 510 ms.

entrainment parameters on the anterior ridge of the left superior pulmonary vein. After the first application, TCL changed into 310 ms and the second AFL was dependent on the left superior pulmonary vein (LSPV). After LSPV isolation, AFL stopped and immediately left-sided accessory pathway–dependent OAVRT (TCL 510 ms) appeared and was confirmed by activation mapping and right ventricular overdrive pacing. After OAVRT termination overt pre-excitation was presented with an effective refractory period of 330 ms and permanent induction of OAVRT (Figure 2).

During sinus rhythm, split P wave was noticed. Measurement of right atrial activation to delta wave and separate right atrial activation to J point interval was 220 ms and 340 ms, respectively. On the contrary, left atrial activation to delta wave and left atrial activation to J point interval was 110 ms and 240 ms, respectively. Left posterior–inferior accessory pathway was confirmed with retrograde conduction and OAVRT. Complete ablation of this pathway and disappearance of pre-excitation and arrhythmia induction was completed after an additional 5 applications. Therefore, LSPV isolation with bidirectional block as well as bidirectional block in the left posterior accessory pathway was confirmed during a 15-minute observational period.

After the procedure, split P wave in sinus rhythm wave was recorded with first-degree atrioventricular block. Within the next 6 months, the patient had uneventful follow-up and no pre-excitation was reported on several ECGs and Holter monitorings. Moreover, additional electrophysiology study confirmed the efficacy of previous applications with LSPV isolation, no anterograde and retrograde conduction through left-sided accessory pathway, and noninducibility of any kind of arrhythmia. At 6 months follow-up, the patient's dilated cardiomyopathy completely resolved, and P wave showed reduced IAB block.[5,6] Therefore, final recovery from electrophysiological sign of atrial cardiomyopathy, Bayes syndrome, and arrhythmia-induced cardiomyopathy, as well as Wolff-Parkinson-White syndrome, were confirmed (Figure 3).

Discussion

Conduction disturbances between atria are most commonly caused by the disturbance of conductivity within the Bachmann bundle. In this case, the atrial activation time is prolonged with PP splitting of 180 ms. The direction of atrial activation is delayed owing to Bachmann bundle block and left atrial activation through the coronary sinus musculature.[1,2]

The definition of the pre-excitation syndrome, in this patient's case, may lead to misdiagnosis of pre-excitation.[6,8] The distance from the onset of P wave to onset of delta wave was 220 ms. However, taking into account the PQ corrected by P-wave conduction disturbances, the left atrial activation to delta wave was 110 ms; therefore, the criterion for pre-excitation syndrome was met.

Advanced IAB is associated with biphasic P waves in the inferior leads and a duration >120 ms. Biphasic P waves in the inferior leads are present in ECG and the duration of the P wave is prolonged (180 ms). An advanced form of IAB is often associated with supraventricular tachyarrhythmias.[3,9] Additional atrial arrhythmias in the atrial conduction disorder may trigger atypical atrial flutter and atrial fibrillation.[10]

During the procedure, radiofrequency ablation of 3 different arrhythmias was performed at the same time: atypical AFL with a microreentry loop within the ridge at the LSPV, atypical AFL associated with LSPV isolation, and an accessory pathway ablation responsible for OAVRT and

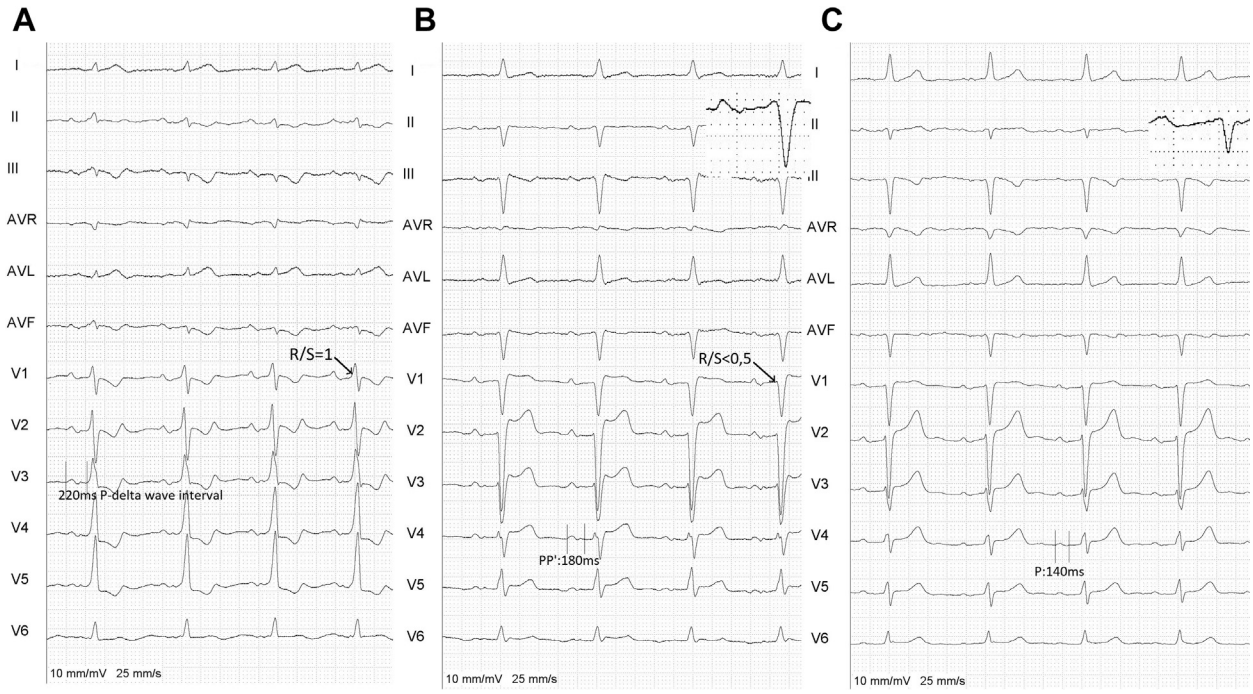

Figure 3 **A:** Pre-excitation during the sinus rhythm (distinguish delta wave by used to R/S >0.5 ratio in V_1).[8] **B:** Conduction disturbances between atria immediately after radiofrequency ablation with PP′ (right atrial and left atrial activation) splitting of 180 ms. **C:** A 12-lead electrocardiogram showing reduction of intraatrial block after 6 months of catheter ablation with P of 140 ms.

pre-excitation. Using 3-dimensional electroanatomic mapping with an access to the left atrium through the patent foramen ovale, a zero-fluoroscopy approach was achieved, without the need for contrast dye for venography and transseptal access, which decreases the risk of renal dysfunction in patients with contrast-induced nephropathy.

The functional conduction block associated with atrial cardiomyopathy was reduced after successful ablation of persistent atypical AFL and AP. After 6 months we observed both a significant reduction in atrial cardiomyopathy in the echocardiogram and improved intraatrial conduction revealed in the ECG by the shortening of the P wave.

Complete remodeling of several atrial and ventricular abnormalities showed that they might have developed very quickly; however, effective treatment may provide successful remission and positive remodeling of atrial and ventricular dysfunction.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2019.07.006.

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