

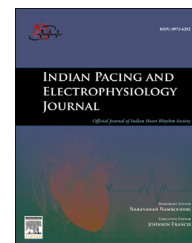
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Successful ablation of frequent atrial premature beats from non-coronary aortic cusp with remote magnetic navigation

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ABSTRACT

A 59-year-old female with structurally normal heart was admitted to our hospital for treatment of highly symptomatic, drug refractory atrial premature beats (APB). ECG revealed atrial parasystolic trigeminy. The arrhythmogenic focus was mapped and ablated using magnetic remote navigation and 3D electroanatomical mapping system. To our knowledge, this is the first report on successful ablation of frequent APBs in the non-coronary aortic cusp.

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Introduction

Atrial premature beats (APB) are commonly encountered in clinical practice. They have traditionally been considered benign, but recent evidence indicates that frequent APBs are a strong predictor of atrial fibrillation development and may be associated with increased risk of cardiovascular death in general population [1]. In addition, frequent APBs may cause cardiomyopathy [2]. We present a 59 year-old female with frequent highly symptomatic APBs. Her heart was structurally normal heart and there was no evidence of any sustained tachyarrhythmias. To our knowledge, this is the first report on

successful ablation of frequent APBs in the non-coronary aortic cusp.

Case report

A 59-year-old female with a long history of highly symptomatic frequent APBs presented to our hospital for catheter ablation. She had been treated ineffectively with several beta blockers and calcium antagonists. Flecainide was stopped because of intolerable adverse effects. Twelve-lead ECG showed normal sinus rhythm with frequent APBs. P wave was negative in inferior leads II, III, avF, positive in avR and V1–V3,

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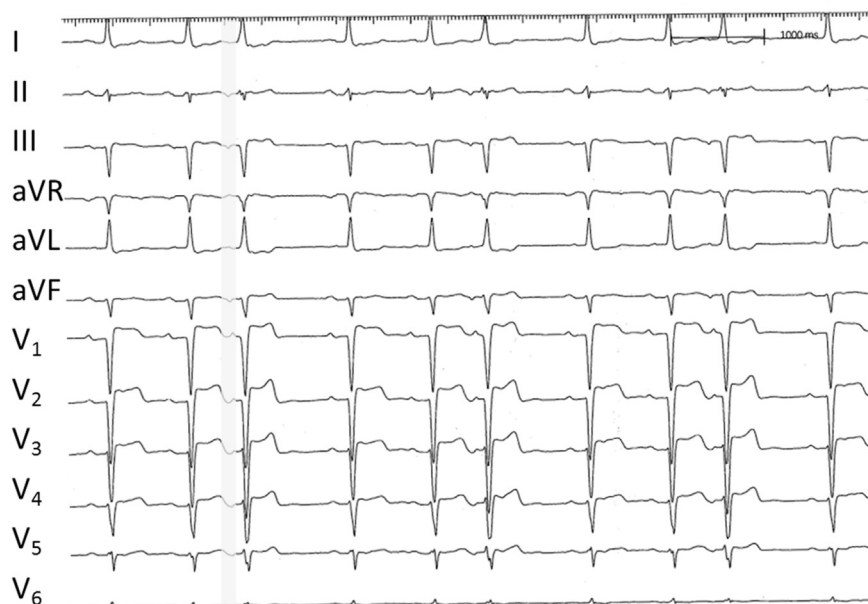


Fig. 1 – Twelve lead ECG showing frequent atrial premature beats. P wave was negative in inferior leads II, III, avF, positive in avR and V₁–V₃, indifferent in lead I, avL and V₄–V₆ (shaded area).

indifferent in lead I, avL and V₄–V₆ (Fig. 1). APB burden in repeated Holter recordings was 20–40% (29000 APBs in the last Holter recording). Neither atrial fibrillation nor any other sustained supraventricular tachyarrhythmia was detected. Clinical examination and thyroid function were normal. Transthoracic echocardiography revealed no structural abnormalities. Left ventricular ejection fraction (LVEF) was 76%.

Antiarrhythmic medication was discontinued two days before the electrophysiological examination. Diagnostic catheters were placed in the coronary sinus and right ventricular apex via right femoral vein. Mapping and ablation was

performed retrogradely via right femoral artery with a 3.5 mm tip open irrigated ablation catheter (Biosense Webster Navistar RMT, Diamond Bar, CA, USA) using remote magnetic navigation (Epoch, Stereotaxis Inc., St Louis, MO, USA) and 3D electroanatomical mapping system (Carto3, Biosense Webster). Mapping was started from the right atrium because of the P wave morphology and coronary sinus activation sequence. Fast anatomical activation mapping demonstrated that the earliest activation in the right atrium was in the vicinity of the His bundle (Fig. 2). No ablation attempt was made although local activation at this site preceded the P wave

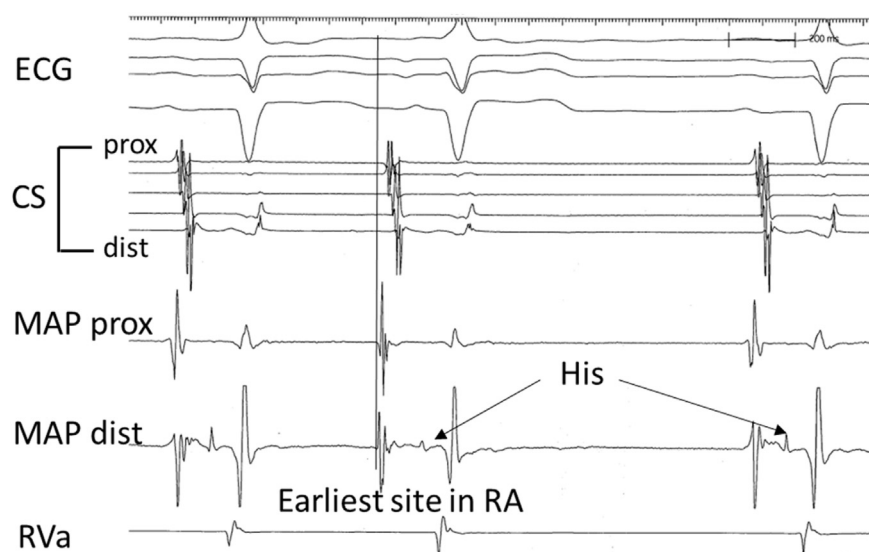


Fig. 2 – During the atrial premature beat coronary sinus activated from proximal to distal. The earliest local atrial activation in the right atrium was detected close to the His bundle. At this site no ablation was performed as the His deflection was clearly visible. Shown are surface ECG leads I, III, avF and V₁. CS = coronary sinus, His = His deflection, MAP = mapping catheter, RVa = right ventricular apex.

because of high risk of damage to the atrioventricular conduction system. Instead, we decided to map the aortic root. Meticulous mapping at the aortic root demonstrated earliest local activation of the clinical APB in the non-coronary sinus of Valsalva anterior and superior to the His bundle. At this site no His potential was detected, and the local electrogram was earlier than in the para-Hisian area in right atrium and preceded the P wave by about 50 ms (Fig. 3). Radiofrequency energy delivery at the non-coronary aortic cusp resulted in immediate cessation of the APBs. No junctional beats or PR

prolongation were observed during ablation (120 s with maximum power of 35 W). The ectopic activity did not recover during the 30-min postablation waiting period. Total duration of the procedure was 110 min and fluoroscopy exposure 2 min 35 s (3.0 Gy/cm²), respectively. At discharge a routine Holter recording was scheduled at 3 months and the patient was advised to contact her physician for ECG monitoring if any arrhythmia symptoms recurred. During the follow-up of 12 months the patient reported no recurrence of symptoms and there were no clinically relevant APBs in the Holter recording.

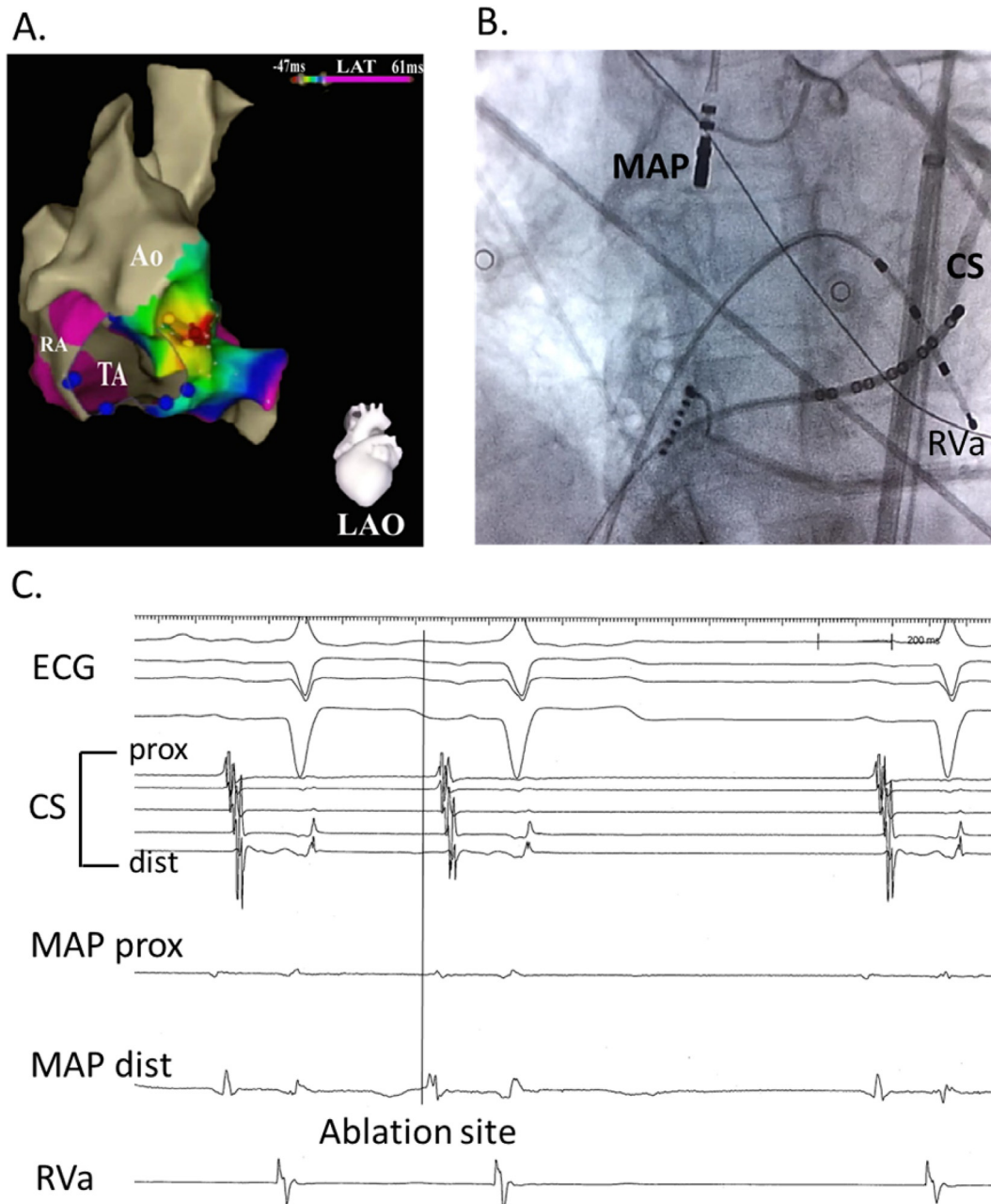


Fig. 3 – Three dimensional electroanatomic map (A) and fluoroscopic picture (B) in left anterior oblique (LAO) view showing the magnetic ablation catheter in the successful ablation site at the non-coronary aortic cusp. Intracardiac signals at time and site of the ablation are shown in panel C. At the ablation site the local activation was about 50 ms before the P wave and 20 ms earlier than in the right atrium. (C) Shown are surface ECG leads I, III, avF and V₁. Ao = aortic root, CS = coronary sinus, MAP = mapping catheter, RA = right atrium, RVa = right ventricular apex, TA = tricuspid annulus.

Discussion

Atrial premature beats are present in 10–20% of the general population. In most cases they are benign, but frequent APBs have been associated with development of atrial fibrillation and cardiomyopathy as well as with increased risk of cardiovascular mortality and stroke [1–3]. In our case no sustained atrial tachyarrhythmias were documented, and left ventricular function was normal despite long history of frequent APBs. Nevertheless, it is possible that the high APB burden could have caused atrial fibrillation and/or APB-induced cardiomyopathy if not treated [2].

The majority of APBs arise from within and around the pulmonary veins (PV). Other sites of origin include left atrial posterior wall, ligament of Marshall, coronary sinus, superior and inferior vena cava, crista terminals and tricuspid annulus. Catheter ablation has become the treatment of choice in patients with various paroxysmal supraventricular tachycardias and ventricular premature beats, but it is used quite rarely to eliminate isolated APBs. The frequently multiple sites of origin and capricious manifestation have made it difficult to ablate extrapulmonary APBs. However, in patients with frequent monomorphic APBs the focus can usually be identified by careful activation mapping and treated by ablation. To the best of our knowledge this is the first report on successful ablation of frequent APBs in the non-coronary aortic cusp. We used remote magnetic navigation (Stereotaxis, Epoch, St. Louis, MO, USA) for mapping and ablation. Based on our experience the main benefit of using magnetic navigation in cases like this is that mapping with a softer and more flexible magnetic catheter is less likely to cause perforation and to provoke catheter-induced extrasystoles than mapping with a manually steered ablation catheter. In addition, the magnetic technology has proven to reduce personnel and patient radiation exposure. The major limitations of the magnetic navigation technique include high initial and procedural cost. Lower contact force is a double-edged sword. It offers important safety benefit by reducing the risk of perforation but also makes creation of transmural lesions difficult in some areas (e.g., cavo-tricuspid isthmus).

The P wave morphology and right atrial mapping demonstrated that the APBs originated from a site close to the His bundle. We and others have previously shown that sustained

atrial tachycardia in this area can often be effectively and safely ablated within the non-coronary aortic cusp which is anatomically in close proximity to the interatrial septum and His bundle [4,5]. In the current case, the local atrial activation at the non-coronary aortic cusp was about 20 ms earlier than that recorded at the His region in the right atrium. At the ablation site no His potential was visible, and the catheter was stable and far from the coronary artery ostia.

This case demonstrated that in addition to focal atrial tachyarrhythmias and ventricular premature beats aortic root can be a source for frequent APBs. Due to the close anatomic relationship between the atrioventricular node and the non-coronary aortic cusp mapping of the aortic root and non-coronary cusp is recommended before ablating in the right atrial septum close to the His bundle.

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