Clinical Case Reports

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CASE REPORT

Catheter ablation of ventricular ectopy with para-hisian origin: importance of mapping both sides of the interventricular septum and understanding when to stop ablating

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Funding Information
No sources of funding were declared for this study.

Received: 31 March 2016; Revised: 28 June 2016; Accepted: 9 October 2016

Clinical Case Reports **2016; 4(12): 1195–1200**

doi: 10.1002/ccr3.739

Key Clinical Message

Catheter ablation of para-Hisian premature ventricular contractions (PVCs) still represents a challenge and is a compromise between success and inadvertent AV block. We describe a possible strategy to address PVCs from this location with high-amplitude His-bundle potentials at the site of earliest activation.

Keywords

Catheter ablation, His-bundle, para-Hisian premature ventricular contraction, premature ventricular contractions.

Introduction

Radiofrequency catheter ablation (RFCA) of idiopathic premature ventricular contractions (PVCs) is widely accepted as a safe and reliable therapy [1, 2]. Idiopathic PVCs are commonly originating from the right and left ventricular outflow tract (RVOT and LVOT, respectively), although a para-Hisian origin has been described [3–6]. In the 12-lead electrocardiogram (ECG), para-Hisian PVCs can mimic an origin from the posterior RVOT [3]. In such cases, careful analysis of the intracardiac electrograms and mapping of both sides of the interventricular septum and of the aortic root is required to determine the optimal ablation site.

Case Report

A 71-year-old man with ischemic cardiomyopathy who previously underwent secondary prevention defibrillator (ICD) implantation was admitted with repeated episodes of sustained ventricular tachycardia (VT) resulting in antitachycardia pacing and shocks. On admission, the ECG showed sinus rhythm (SR) with normal QRS complexes (duration 87 msec) and repolarization, and bigeminal monomorphic PVCs (QRS duration 148 msec, inferior axis, left bundle branch block morphology with transition in V_4, Fig. 1A). Echocardiography demonstrated reduced left ventricular (LV) ejection fraction (0.45) with akinetic basal and medium segments of the inferior wall and an additional septal dyskinesia. During telemetric ECG monitoring, frequent monomorphic PVCs, often bigeminal, were observed, and in the ICD's recordings, the PVC burden was >20%. Of note, the intracardiac electrograms stored during the VTs and the PVCs had different morphology. After obtaining informed consent, an electrophysiology study was performed under fasting conditions and conscious sedation. After substrate

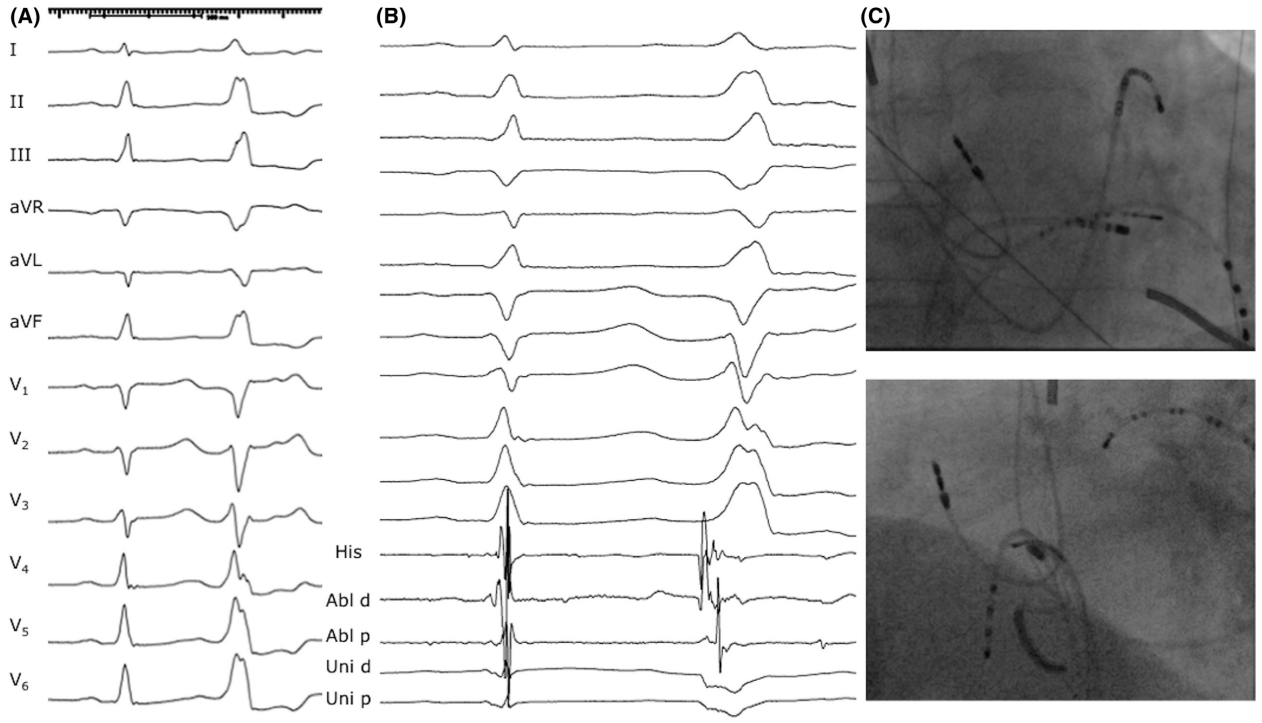

Figure 1. (A) Morphology of the first PVC (QRS duration 148 msec, inferior axis with humps on the inferior leads). (B) Earliest activation is seen at the RV septum above the His-bundle (preceding the QRS onset by 27 msec). In the position where a His-bundle potential is registered, the bipolar signal precedes the QRS onset by 25 msec with a slow downstroke slope in the unipolar signal; close above, an earlier bipolar signal with rapid QS on the unipolar and no His-bundle potential is found. (C) Two multipolar catheters are placed via the femoral vein at the right ventricular apex and at the His-bundle; a third one is inserted via the internal jugular vein into the coronary sinus. Right (30°, left panel) and left (50°, right panel) anterior oblique radiographic views show the ablation site.

modification ablation, confined to the LV inferior wall, we concentrated on mapping the bigeminal PVCs using conventional and 3D mapping (EnSite NavX, St Jude Medical, St. Paul, MN, USA). Ablation was performed using an irrigated tip catheter (Thermo-Cool, Biosense Webster, Diamond Bar, CA, USA) in a temperature-controlled fashion 10–35 Watt (W), 42°C. The PVCs were mapped starting initially in the RVOT, but the earliest activation was observed in the para-Hisian region (preceding the QRS onset by 27 msec, Fig. 1B). Pacemapping here was attempted but without good results because of direct capture of the specific conduction system even using low output. Application of radiofrequency (RF) energy here, increasing the power from 20 to 30 W, quickly eliminated the PVCs, without signs of AV nodal conduction impairment. A few minutes later, a slightly different morphology of PVCs (Fig. 2A) appeared, again in a bigeminal fashion. Mapping of the right para-Hisian region did not show near-field early activation or unipolar signals with steep downslope of the QS anymore, and additional RF applications here did not affect the PVCs. Therefore, mapping was performed in the LVOT retrogradely via the aorta. Earliest activation (−46 msec to the onset of the QRS) was seen under the right coronary aortic cusp (RCC) close to the membranous septum, where a His-bundle potential >0.5 mV was recorded (Fig. 2B). Careful RF application at 10 W was attempted, but immediately stopped because of fast junctional beats. RF application was then tried in a slightly more apical position with up to 25 W without affecting the PVCs (Fig. 3A). An intermediate position between the two application sites, with local activation (both in the bipolar and the unipolar recording) preceding the QRS onset by 44 msec, was attempted despite a His-bundle potential (0.15 mV) on the distal ablation bipolar recordings (Fig. 3B). As shown in Figure 3C, during initial RF energy application at 10 W, PVCs immediately disappeared. However, after carefully increasing the power to 25 W, after 60 sec, the AH time increased from 60 to 97 msec, and as soon as RF application was stopped, the PVCs reappeared, and, gradually, the AH interval returned to the baseline value. Another careful map of the right septum was performed, and a good combination of bipolar and unipolar signals was observed only on the midseptal tricuspid annulus (Fig. 4A) close to the fast pathway area, where, despite low-energy application, RF

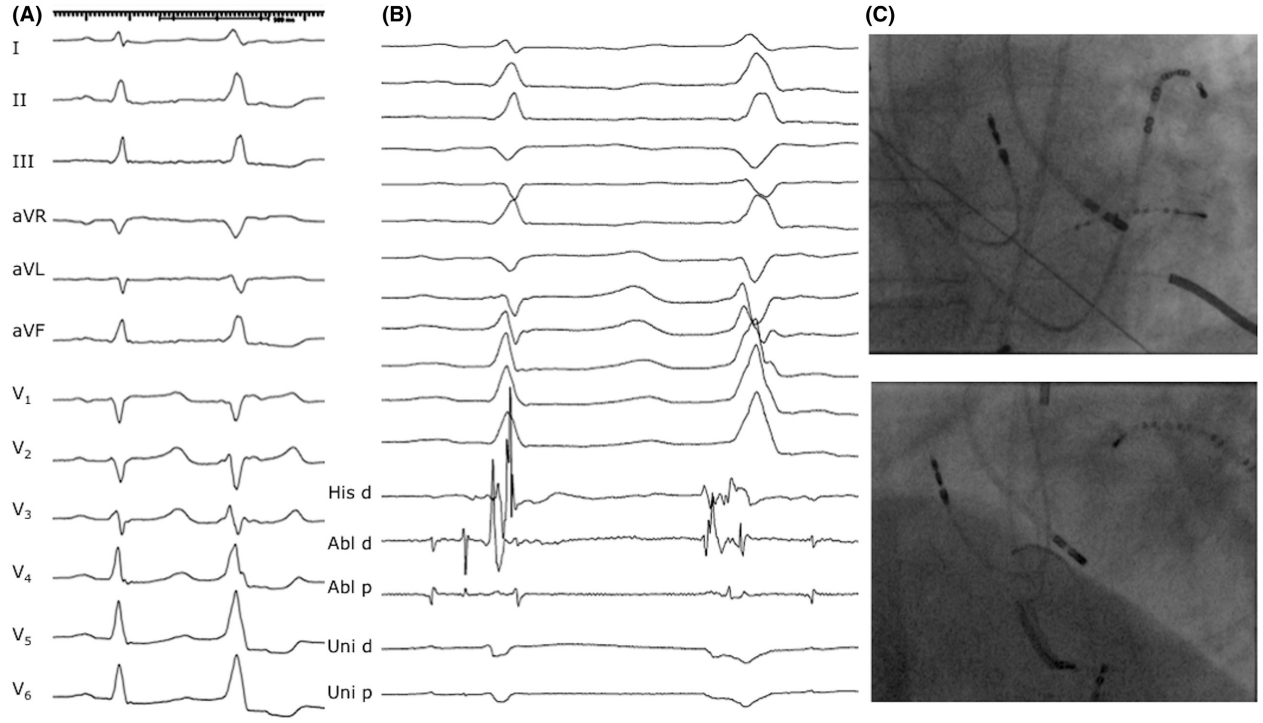

Figure 2. (A) Morphology of the second PVC. (B) Earliest activation is seen under the RCC (−46 msec). The activation time is the same on both sides of the interventricular septum, but the unipolar signal has a more rapid downstroke slope of the QS on the left compared to the right side. (C) Radiographic position of the catheters shown in B. Of note, abnormal LV substrate was confined to the inferior wall, and no low amplitude and/or fractionated potentials were seen on the septum.

resulted in fast junctional beats. The last RF energy application was attempted from the RCC (Fig. 4B). Despite relatively late local activation (−20 msec to the onset of the QRS), temporary elimination of the PVCs was achieved relatively late (40 sec) after the initiation of RF delivery at 35 W, with immediate reappearance of the PVCs after termination of energy application. Although the patient already had dual chamber pacing capabilities, further RF applications were not given for the presumed high risk of AV block.

Discussion

To the best of our knowledge, this is one of the very few cases of PVC RFCA with a high-amplitude His-bundle potential at the site of earliest activation. ECG findings suggesting a para-Hisian origin of PVCs are (1) a relatively narrow QRS with an inferior axis, (2) a monophasic R-wave in lead I, (3) a QS pattern in V_1, and (4) a relatively low R-wave amplitude in the inferior leads, with lower R-wave amplitude in lead III than in lead II [3, 4]. In some cases, a superior axis with left or right bundle branch block pattern has also been described [5, 6]. Furthermore, there are no reported criteria to differentiate a right- from a left-sided para-Hisian focus. Despite these ECG criteria, as the present case shows, it can be challenging to identify a para-Hisian origin, especially if the PVC morphology is relatively broad and there is late transition. In these cases, the diagnosis of para-Hisian PVCs is mostly realized during the electrophysiologic procedure. Careful and extensive mapping of both sides of the septum and of the aortic root is required, and initial ablation should ideally be attempted as far away as possible from the His-bundle in order to minimize inadvertent AV node injury. The distance between the ectopic focus and the His-bundle and the AV node, respectively, is of critical importance. In the largest published series on para-Hisian PVCs, a His-bundle potential was only registered on the proximal ablation electrode pair at the successful ablation site, whereas RFCA was avoided when a His-bundle electrogram >0.1 mV was recorded at the site of earliest ventricular activation. Therefore, the reported rates of damage to AV conduction [3–6] may underestimate the true risk. Recently, the Bordeaux group described a very didactical case of successful elimination of para-Hisian PVCs [7]. In that case, the initial RF application from the LVOT and the aortic root did not affect the PVCs; the ectopic focus was located on the right interventricular septum toward the distal His-bundle, where early local activation with QS unipolar electrogram morphology was

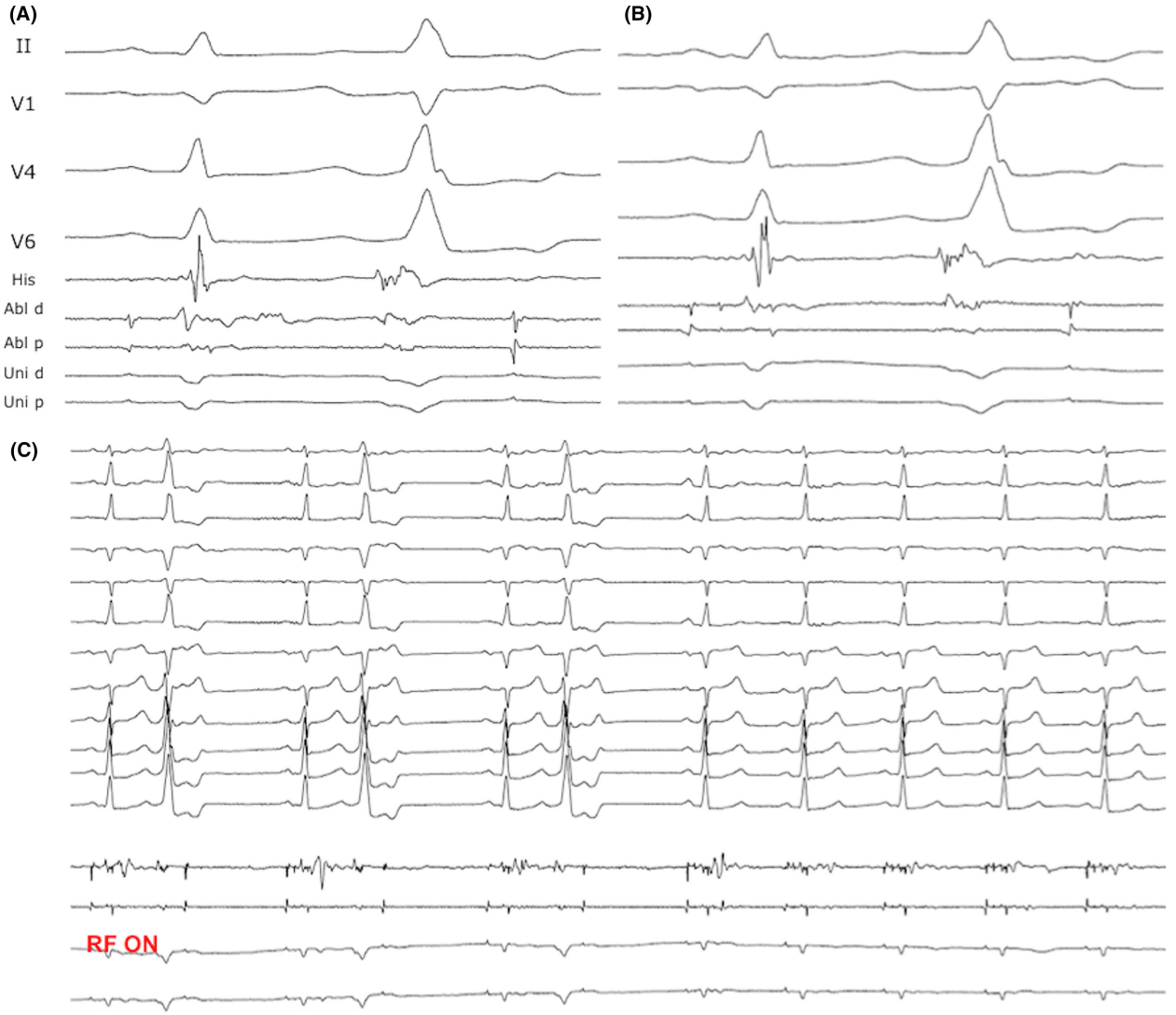

Figure 3. (A) Unsuccessful attempt slightly more apical in the LV (far field His-bundle on the ablation proximal) despite early local activation time (−42 msec) in the bipolar signal (but late beginning of the QS complex in the unipolar). (B) successful ablation site (−44 msec, low-voltage His-bundle on the ablation distal). (C) Immediate disappearing of the PVCs at this position B with inadvertent AH prolongation despite low-energy application after 60 sec.

seen, and the PVCs could be eliminated without damaging the AV node. As discussed by the authors, the AV block risk is high but the His-bundle is isolated from the adjacent myocardium by sheaths of fibrous tissue that presumably contribute to protection during RF energy application. In contrast, the AV node is not surrounded by fibrous tissue [7, 8]. Furthermore, the left His-bundle emerges into the LVOT immediately adjacent to the membranous septum, whereas the right bundle runs through deeper layers of the septum before emerging more apically on the surface [8]. Most likely, the ectopic focus in our case had a deeper location in the septum rather than being located endocardially, as it was possible to affect the PCVs (i.e., change of the morphology and temporary elimination) from both the right and left side of the septum as well as from the aorta but it was not possible to achieve permanent elimination from any of these locations. In contrast, in the presence of a right subendocardial focus as well as in cases of para-Hisian accessory pathways [9], an incremental RF energy strategy will be safe and effective. Furthermore, close proximity of the focus to the His-bundle is suggested both by the intracardiac signals and by the ability to affect the PVCs only in combination with AV node injury (AH prolongation and/or fast junctional beats) despite low RF energy. Cryo-energy may be an option to reduce the rate of AV

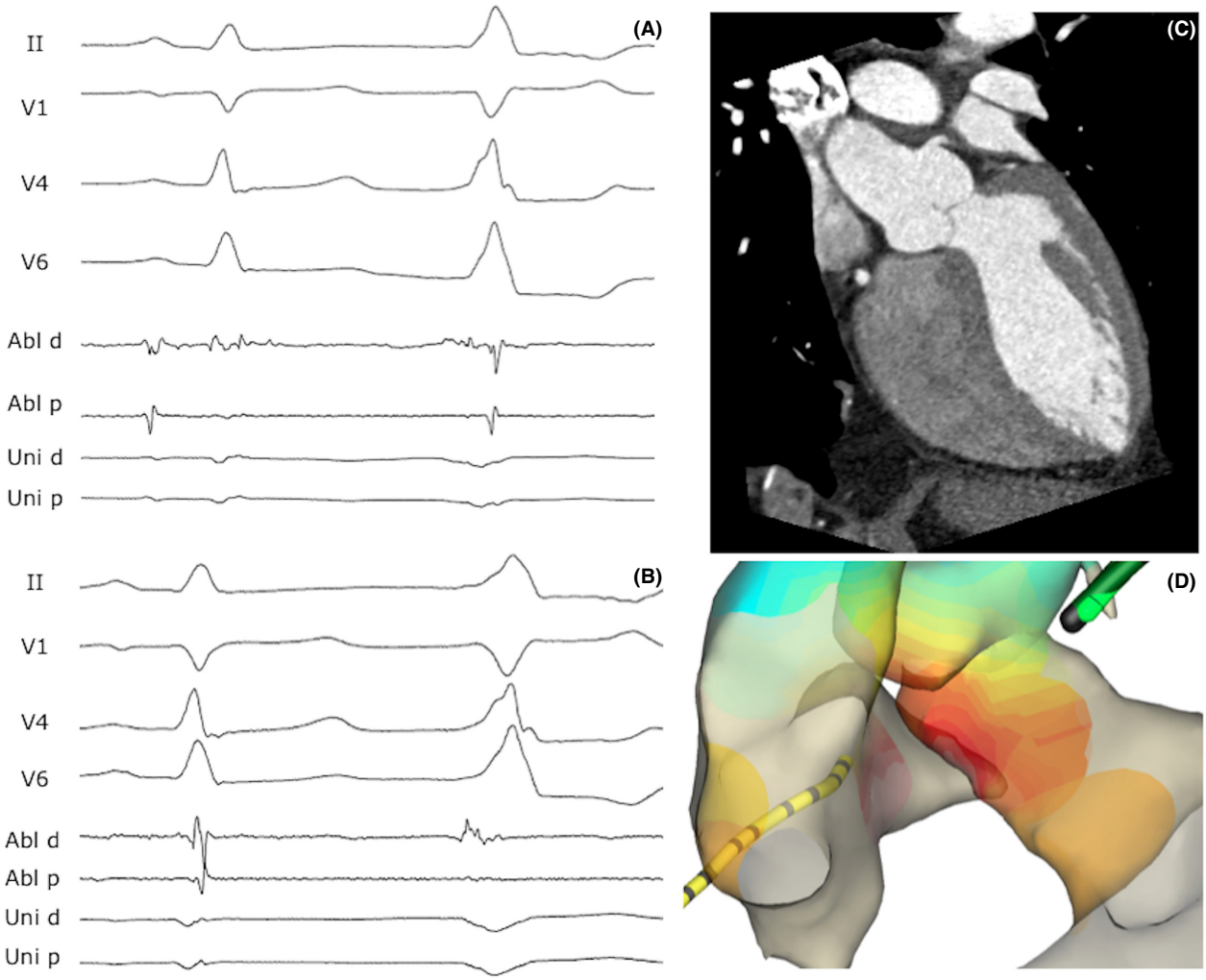

Figure 4. (A) Last attempt on the RV septum (−46 msec). In SR, on the distal ablation are recorded both atrial and ventricular signals with an His-bundle potential, while the proximal bipolar shows only an atrial signal without ventricle [11]. Despite low energy, fast junctional beats immediately occur. (B) Local activation in the RCC (−20 msec to the onset of the QRS). Disappearance of the PVCs is observed relatively late during application of 35 Watt. (C) Computed tomography scan showing the relationship between the right, the left septum, and the aortic root. (D) 3D Mapping of PVC 2 showing similar activation times on both sides of the interventricular septum.

block although the acute and long-term efficacy under these circumstances remains to be determined [10]. On the other hand, a more detailed mapping approach using different electrodes sizes and interspacing might help in precisely defining the location of the focus, as well as intracardiac echo would allow direct visualization of the region of interest.

Conclusions

Radiofrequency catheter ablation of para-Hisian PVCs is challenging and may be associated with a high complication rate and usually requires a compromise between success and inadvertent AV node injury. A careful approach with extensive mapping on both sides of the interventricular septum as well as in the aortic root and an incremental energy strategy starting away from the His-bundle may be prudent. Finally, in some cases, complete abolition of the PVCs may only be achievable at the price of AV block.

Conflict of Interest

A.G. has received speaker fees from St. Jude Medical, S.R. is a consultant for Medtronic and St. Jude Medical and has received speaker fees from St. Jude Medical, J.C.G. is a

consultant for St. Jude Medical, Biosense Webster, Boston Scientific, Medtronic, AstraZeneca, and Pfizer, and has received speaker fees from St. Jude Medical, Boston Scientific, Medtronic, Biotronik, AstraZeneca, Bayer, Berlin Chemie, Boehringer Ingelheim, Novartis, Meda, Pfizer, and Sanofi Aventis. There are no other conflicts of interest to report.

Authorship

All persons and only persons who meet authorship criteria are listed as authors. All authors certify that they have participated sufficiently in the work to take public responsibility for the content, including participation in the concept, design, analysis, writing, and/or revision of the manuscript.

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