# Long-term freedom from ventricular fibrillation despite persistent Purkinje ectopy after catheter ablation

Elodie Surget, MD,*† Josselin Duchateau, MD, PhD,*† Thomas Lavergne, MD,‡ F. Daniel Ramirez, MD,§ Ghassen Cheniti, MD,*† Michel Haissaguerre, MD, PhD*†

*From the *IHU Liryc, Electrophysiology and Heart Modeling Institute, Foundation Bordeaux Université, Bordeaux, France, †Electrophysiology and Ablation Unit, Bordeaux University Hospital (CHU), Pessac, France, ‡Cardiology Department, European Georges Pompidou Hospital, Paris, France, and §Division of Cardiology, University of Ottawa Heart Institute, Ottawa, Canada.*

## Introduction

The Purkinje system is the main source for premature ventricular complexes (PVCs) triggering idiopathic ventricular fibrillation (IVF).[1–3] Elimination of these PVCs efficiently prevents ventricular fibrillation (VF) recurrence.[2] However, little is known about long-term outcomes of patients with persistent PVCs after catheter ablation. Here, we describe 3 patients with IVF who remained free of VF recurrence despite the persistence of PVCs with more than 15 years follow-up after catheter ablation.

**KEY TEACHING POINTS**

- Idiopathic ventricular fibrillation (IVF) is defined by ventricular fibrillation (VF) occurring in young adults with no discernible structural or electrocardiographic abnormalities.
- A subset of IVF is induced by Purkinje ectopic beats (PurkE), and their ablation prevents VF recurrence.
- Ablation of the surrounding Purkinje system without elimination of culprit PurkE may be sufficient to prevent VF recurrence.

## Case report

### Case 1

A 31-year-old woman with a history of hypertension and obesity was admitted to hospital for syncope in 2005. Her baseline electrocardiogram (ECG) showed sinus rhythm and frequent PVCs with a coupling interval of 280 ms (Figure 1). The PVCs exhibited a sharp and rapid initial deflection and left bundle branch block morphology, suggestive of right Purkinje ectopy (PurkE). A transthoracic echocardiogram did not identify structural abnormalities. Pharmacological testing with isoproterenol, adrenaline, and ajmaline infusions was negative. Soon after admission, the patient had multiple runs of nonsustained VF and 1 sustained VF episode, prompting the decision to proceed to catheter ablation. The PVCs were confirmed to be right-sided and of Purkinje origin. The PVCs showed discrete morphological variations with Purkinje signals recorded on repetitive beats, suggesting repetitive focal activity or reentry in the Purkinje system.[2] Seventeen radiofrequency applications (total duration = 12.5 minutes) were delivered in the region of the moderator band: at the earliest activation site and at surrounding sites in an attempt to "prune" the Purkinje ramifications and minimize the risk of recurrences. Ablation acutely eliminated the culprit PurkE; however, it recurred several days later in rare and isolated forms. The patient refused an implantable cardioverter-defibrillator (ICD). Holter recordings were performed every year and showed persistent isolated (no couplets or triplets) PVCs of the same short-coupled morphology over 16 years of follow-up. A total of 2314 isolated PVCs were noted on her most recent 24-hour Holter monitor. Despite not being on any antiarrhythmic agents, the patient had any VF recurrence.

### Case 2

A 38-year-old man was hospitalized following several appropriate ICD shocks for VF. ICD was implanted 4 years ago for cardiac arrest. Transthoracic echocardiogram, cardiac magnetic resonance imaging, and coronary angiography were unremarkable. Pharmacological tests were not suggestive of Brugada syndrome, catecholaminergic polymorphic

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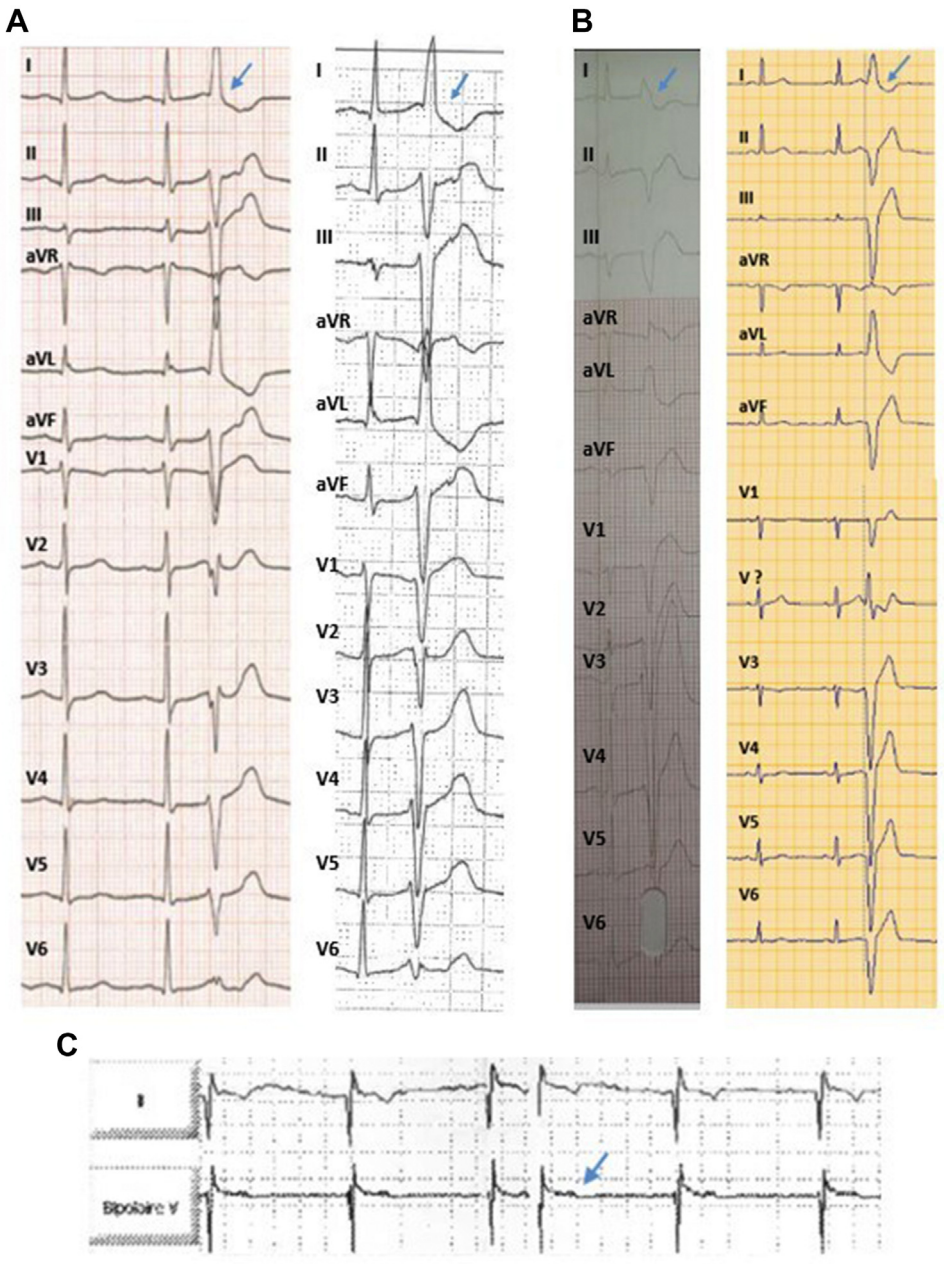


**Figure 1** **A:** (Left) Electrocardiogram (ECG) on admission of patient in case 1 shows a premature ventricular complex (PVC) with a short coupling interval (CI) at 280 ms. The sharp and rapid onset of the PVC and its left bundle branch block morphology are in favor of right Purkinje ectopy. (Right) Twelve-lead ECG during the follow-up shows the same PVC with a coupling interval at 300 ms. **B:** (Left) ECG before the ablation of patient in case 2 shows a right Purkinje ectopy with a coupling interval at 300 ms. (Right) During the follow-up, the 12-lead Holter ECG in case 2 shows right Purkinje ectopy with a short CI at 355 ms and a variation of morphology in $V_2$, probably because of a $V_2$ lead misplacement (inconsistent R-wave progression in $V_2$ in sinus rhythm). **C:** Implantable cardioverter-defibrillator interrogation of patient in case 3 shows PVC with a short coupling interval at 300 ms.

ventricular tachycardia, or long QT syndrome. His ECG showed narrow QRS complexes (83 ms) and a normal QTc interval (406 ms) with frequent PVCs—triggered by sitting position and adenosine injection—at a coupling interval of 240 ms with features suggestive of right-sided PurkE. During electrophysiological study, he presented scarce spontaneous PVCs. Pace mapping confirmed the right Purkinje origin and radiofrequency applications (total duration = 5 minutes) were delivered to the anterior right ventricle in its lower third over a region of 2 $cm^2$. At the end of the procedure, Purkinje ectopies were no longer inducible by adenosine injection and the patient was discharged from hospital on beta blockers. Over the subsequent 19 years, annual 24-hour Holters have shown recurrence of isolated short-coupled PurkE (mean 149 ± 306/24 hours), without repetitive forms (couplets, triplets…) (Figure 2). However, he has remained free from recurrent sustained ventricular arrhythmia. Comprehensive genetic testing (98 genes) was negative.

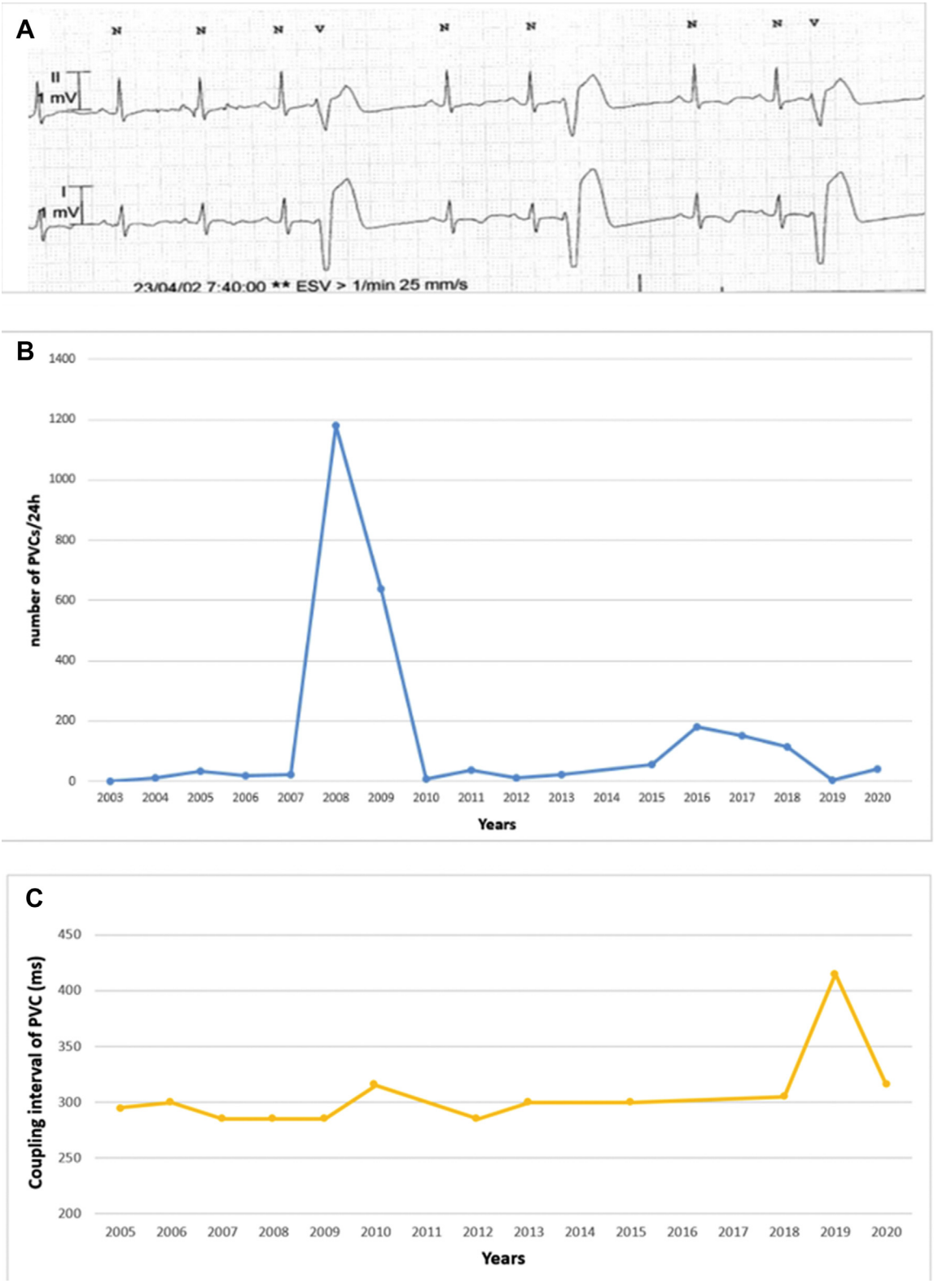


**Figure 2** Annual 24-hour Holters after ablation of patient in case 2. **A:** Premature ventricular contractions (PVCs) recorded on Holter electrocardiogram. **B:** Persistence of isolated PVCs after ablation. **C:** Mean coupling interval of the postablation PVCs.

## Case 3

A 25-year-old man was hospitalized for several VF episodes within 48 hours requiring multiple shocks. ECGs showed frequent short-coupled PVCs suggestive of right-sided PurkE. A secondary prevention ICD was ultimately implanted and calcium channel blockade (verapamil) was initiated, but he continued to suffer from recurrent VF. An electrophysiologic study was performed and localized the

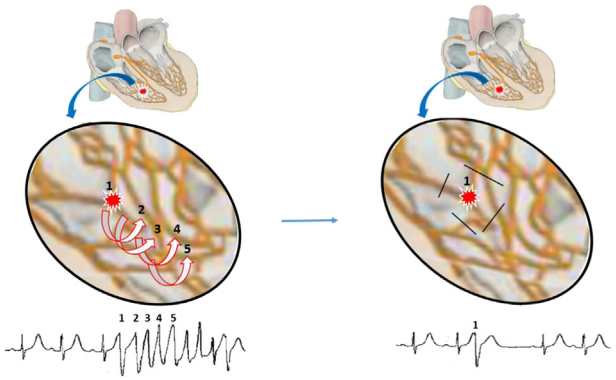


**Figure 3** Left: Purkinje ectopy is induced by triggered activity and spreads in the Purkinje network thanks to a mechanism of reentry essential for ventricular fibrillation (VF) initiation. Right: Ablation of the Purkinje network and the surrounding tissue prevents reentry in the Purkinje network, leading to isolated Purkinje ectopy without VF episode.

earliest Purkinje potential to the anterior aspect of the right ventricle adjacent to the tricuspid annulus. Ablation (total duration = 9.50 minutes) was performed at this site with acute PurkE elimination. The patient was then observed with continuous ECG monitoring for 4 days, during which the PurkE were seen to recur without repetitive forms. Over the subsequent 17 years, the patient has remained free from recurrent VF despite the persistence of the targeted PurkE.

## Discussion

The current case series suggests that total elimination of short-coupled PVCs may not always be required to suppress VF recurrences in cases of IVF treated with catheter ablation. To our knowledge, no previous study has reported this finding with such a long follow-up period.

A subset of IVF is usually triggered by PVCs arising from the Purkinje system,[2,4] right ventricular outflow tract,[5] or moderator band/papillary muscles,[6] with the Purkinje system accounting for approximately 90% of cases. The elimination of culprit PurkE can effectively prevent VF recurrence[2] and remains the gold standard for IVF ablation. However, in the 3 cases presented above, long-term freedom from recurrent VF was achieved by ablating the Purkinje network and surrounding Purkinje potentials without elimination of the culprit PurkE. One potential explanation for this relates to the growing evidence that reentry is essential in VF initiation (Figure 3). Herein, the observation of frequent PVCs with Purkinje signals recorded on repetitive beats suggested a mechanism of reentry or repetitive focal activity. By ablating more parts of the arborization of the Purkinje system, reentry leading to repetitive beats can be particularly minimized.

Reentry using the Purkinje system has been well described in monomorphic ventricular tachycardias such as bundle-branch reentry and intrafascicular or interfascicular reentry.[7] However, reentry can also occur at Purkinje-muscle junctions (PMJs), resulting in VF initiation.[8] Distinct electrophysiologic properties of the Purkinje system and of the surrounding myocardium usually prevent reentry at this interface. Among the most important of such characteristics, the action potential duration of peripheral Purkinje fibers gradually increases from proximal to distal fibers, preventing retrograde conduction.[9] This protective mechanism can be overcome in certain states, however, and ectopic beats within the distal Purkinje system are sometimes able to activate the myocardium at PMJs with short action potential durations.[10] Inhomogeneous and slowed anterograde conduction at the PMJ can result in functional anterograde block, allowing for retrograde conduction and reentry.[11] Ablation of the Purkinje system can disconnect the Purkinje system from the myocardium, or sever the ramifications, effectively preventing such reentry. Hypothetically, ablation of the Purkinje tissue could also prevent VF occurrence by reducing the propagation velocity within the Purkinje system and modifying the delicate balance between the elements involved.[12]

To our knowledge, only 2 other reports have been reported in which freedom from recurrent VF was achieved by catheter ablation without eliminating the trigger. In a multicenter study evaluating patients ablated for IVF, Knecht and colleagues[13] observed a recurrence of clinical PurkE in 2 of 38 patients, but they no longer triggered malignant ventricular arrhythmias over a follow-up of 63 months. Nogami[14] also described the case of a patient with left-sided PurkE in whom VF suppression was achieved with catheter ablation of the Purkinje network and not at the earliest site of Purkinje activation. In our study, the 3 patients exhibited right-sided PurkE and freedom from recurrent VF was achieved with ablation of the right Purkinje system, possibly in part because of the less complex and extensive arborization of the right Purkinje system, compared to the left.

## Conclusion

We described 3 patients with IVF in whom freedom from recurrent VF was observed with catheter ablation of the Purkinje system and of the surrounding myocardium, but without sustained elimination of the culprit PurkE trigger. Our findings demonstrated that regional ablation of the distal Purkinje system can avoid repetitive beats and be sufficient to prevent VF recurrence.

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