Ventricular fibrillation initiated by reentry involving the Purkinje network in a patient with after myocardial infarction

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Introduction

Ventricular fibrillation (VF) can occur shortly after myocardial infarction.[1–4] Surviving bundles from the Purkinje system have been shown to play a role in VF occurrence in this setting. Premature ventricular contraction (PVC) from the Purkinje network initiates VF. The commonly presented mechanism of the initial PVC is triggered activity or abnormal automaticity from the injured Purkinje network owing to ischemia. Herein, we report a case where a VF-triggering PVC originated from reentry involving the Purkinje network. Furthermore, ablation targeting the Purkinje–muscle junction to modify the reentrant circuit, not the earliest Purkinje potential, successfully suppressed the PVC and VF.

Case report

A 70-year-old man with a history of coronary artery bypass graft surgery presented with acute coronary syndrome. The patient underwent percutaneous coronary intervention of the left circumflex artery without complications. Twenty days after percutaneous coronary intervention, he suddenly developed multiple episodes of VF refractory to medical therapy, including amiodarone, lidocaine, and sedation. Only atrial pacing at 100 beats per minute suppressed VF; however, VF repeatedly occurred after ceasing atrial pacing. The same PVC with right bundle branch block morphology always initiated VF, which was compatible with a Purkinje origin (Figure 1A). Thus, we decided to perform catheter ablation targeting this PVC.

KEY TEACHING POINTS

- The Purkinje network is involved in ventricular fibrillation (VF) occurring shortly after myocardial infarction. Triggered activity or abnormal automaticity from surviving Purkinje fibers initiates VF.
- This case demonstrated that premature ventricular contraction (PVC) and VF could be initiated with reentry involving the Purkinje network. A retrograde conduction delay into the Purkinje network facilitated antegrade conduction to the local myocardium, initiating reentrant activity.
- Ablation targeting the distal Purkinje–muscle junction, not the earliest activation site during PVC, suppressed PVC and VF, presumably by modifying the reentrant circuit in the Purkinje network.

At the time of catheter ablation, endocardial left ventricle mapping was performed during sinus rhythm using Octaray (Biosense Webster, Diamond Bar, CA). A low-voltage area was observed in the basal anteroseptal region of the left ventricle (Figure 1B, left). The markedly delayed potential was confirmed in the same area where the Purkinje potential was recorded (Figure 1C). The distribution of this potential was relatively wide but was found locally in the proximal left fascicle, as illustrated by the purple dots in Figure 1B (left). Activation mapping, which annotated the latest potentials during sinus rhythm, further revealed that the area with the delayed potentials was very localized (shown as blue or purple dots; Figure 1B, right). Because the late potentials were not constantly observed, areas with late potentials could have been wider than observed. However,

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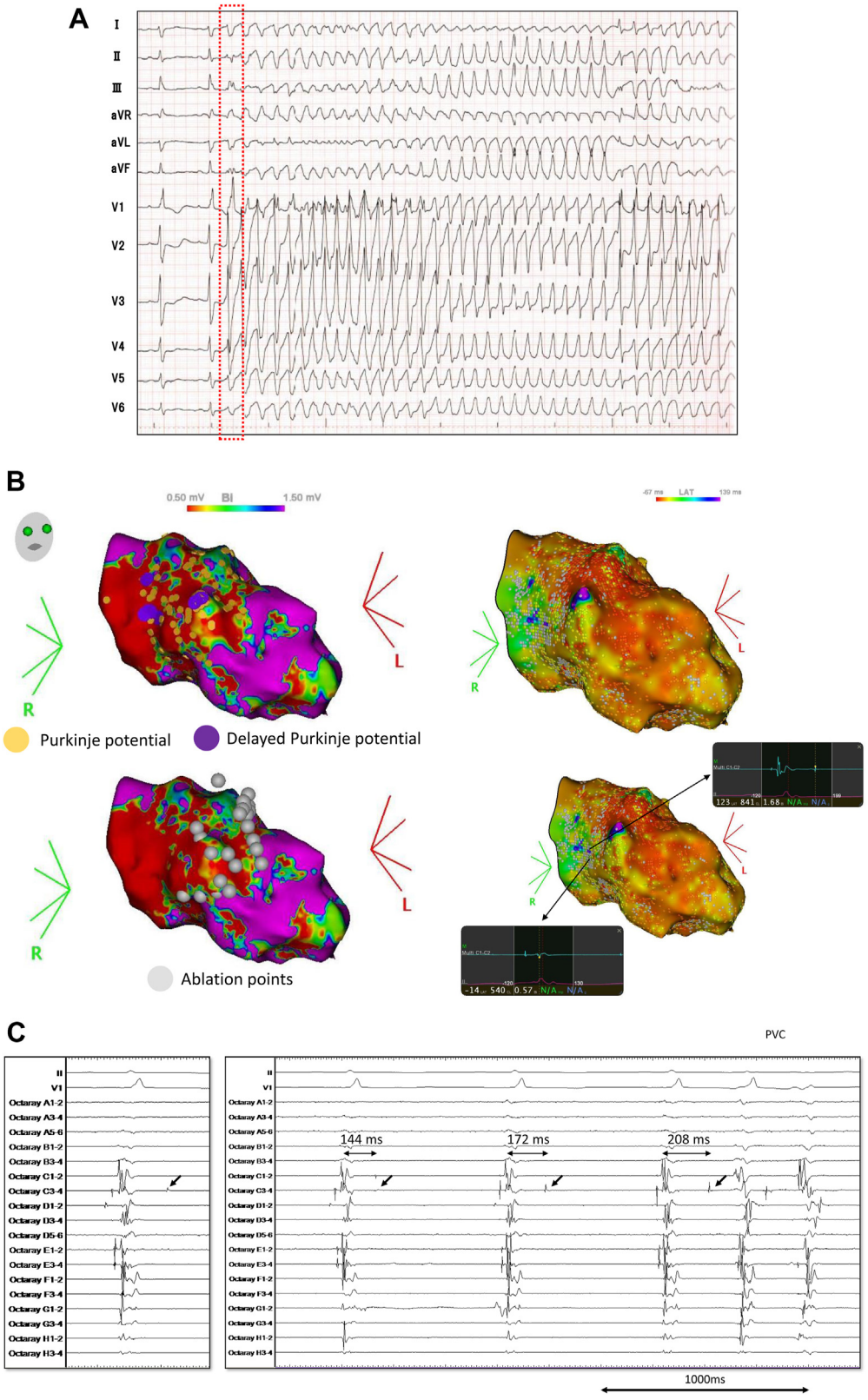

Figure 1 **A:** A 12-lead electrocardiogram of a 70-year-old man showing a right bundle branch block pattern during sinus rhythm. Premature ventricular contraction (PVC) with a coupling interval of 320 ms reproducibly initiated ventricular fibrillation (VF) (*dotted rectangle*). **B:** (Left) Voltage mapping of the left ventricle showed a low-voltage area at the basal to the mid ventricular septum. Yellow dots show the Purkinje potential recording area along the left bundle branch area during sinus rhythm, and purple dots show the area where delayed Purkinje potentials were recorded. Ablation was performed mainly at the distal Purkinje potential recording site during sinus rhythm (*white dots*), corresponding to the border zone of the ischemic scar at the left ventricular septum. (Right) Slow conduction or line of block was not evident in the activation mapping, which annotated the latest potentials during sinus rhythm. There were no delayed potentials around the areas with delayed Purkinje potentials. **C:** (Left) Recordings during sinus rhythm with a multielectrode catheter along the left bundle branch

slow conduction or line of block was not evident around these delayed potentials (Figure 1B, right). Although sustained VF did not occur during the procedure, PVC and nonsustained VF were observed spontaneously. This delayed potential always preceded PVC and nonsustained VF and was considered to represent Purkinje activity (Figure 1C). Interestingly, gradual prolongation of the conduction time from the QRS to the delayed Purkinje potential reproducibly resulted in PVC and nonsustained VF (Figure 1C), suggesting that a retrograde conduction delay to the Purkinje network facilitated reentrant antegrade conduction from the Purkinje network to the local myocardium. The Purkinje potentials preceded the local myocardial potential during the initial phase of nonsustained VF (Figure 2A). During atrial burst pacing, there was no delay in the conduction time from the QRS to the delayed Purkinje potential at a rate of more than 750 ms. Prolongation of the conduction time was associated with PVC and nonsustained VF at a higher atrial pacing rate (Figure 2B). Rapid pacing at less than 630 ms blocked the conduction (Figure 2B). The extrastimulus from the atrium also showed decremental conduction from the QRS to the delayed Purkinje potential, resulting in a conduction block with extrastimuli at 670 ms (Figure 2B).

This patient's baseline electrocardiogram showed a right bundle branch block morphology, and the delayed Purkinje potential was distributed at the proximal left fascicle (Figure 1B, left, purple dots); therefore, the ablation strategy targeting this earliest potential during PVC was considered to have a high risk of atrioventricular block. Since the VF mechanism in this patient was thought to be reentry involving the Purkinje network rather than triggered activity or abnormal automaticity, we decided to ablate the distal Purkinje network targeting the Purkinje–muscle junction to modify the reentrant circuit, especially around the scar border area. During ablation, multiple PVCs with a right bundle branch block pattern and slightly different axes appeared; thus, we extended the ablation area from the left ventricular posteroseptum to the anteroseptum (Figure 1B). After 24 minutes of radiofrequency application, the spontaneous PVC and nonsustained VF disappeared. There was only a minor change (a small new s wave in lead II) in the QRS morphology after the procedure (Figure 3A). VF has not occurred for 2 months.

Discussion

To our knowledge, this is the first study to investigate the electrophysiological details of the Purkinje potential that initiates VF in patients with ischemic VF. The mechanism of VF-initiating PVC was presumably reentry involving the Purkinje network. Ablation targeting the distal Purkinje network, not the earliest activation site during PVC, successfully suppressed VF.

VF that occurs shortly after myocardial infarction mainly involves the Purkinje network. The Purkinje potential precedes the local myocardial potential during VF, and triggered activity or random reentry involving the Purkinje network is the suggested mechanism. Normally, the VF-initiating PVC is due to triggered activity or abnormal automaticity from the injured Purkinje network. The preceding time of the earliest potentials was up to 160 ms.[5] This latency is due to the anatomical distance between the foci in the Purkinje network and the breakout site of the PVC. In this case, however, the Purkinje potential recorded during PVC was linked with the preceding QRS. A decremental property was observed in the conduction from the QRS to the Purkinje potential.

Furthermore, a gradual conduction delay reproducibly facilitated PVC and nonsustained VF occurrences. Positioning the Octaray catheter along the left fascicle during PVC and nonsustained VF was difficult, and we could not record the sequential Purkinje potentials preceding PVC and nonsustained VF. However, from these findings, we speculated that the VF-initiating PVC in the present case originated from reentry involving the Purkinje network rather than triggered activity or abnormal automaticity. The action potential duration of the peripheral Purkinje system gradually increases from proximal to distal fibers and normally prevents retrograde conduction at short coupling intervals.[6,7] However, this protective mechanism can be overcome in certain situations, and inhomogeneous and slowed antegrade conduction at the Purkinje–muscle junction can result in a functional antegrade block, allowing for retrograde conduction and reentry.[8,9] In our case, sinus conduction invaded the Purkinje system retrogradely, and the conduction delay that occurred in this retrograde conduction facilitated antegrade conduction from the Purkinje network to the local myocardium, resulting in PVC (Figure 3B). When this reentrant activation continued inside the Purkinje network, VF was sustained. This case demonstrates that some ischemic VF can be initiated by PVC generated from reentry involving the Purkinje network.

Overdrive pacing is recommended for recurrent ventricular arrhythmias in patients with ischemia.[1,10,11] Although heart rate stabilization is suggested to help reduce action potential dispersion, the mechanism by which rapid pacing can suppress VF is not completely understood. In this case, atrial pacing suppressed the VF storms. Atrial pacing at a high rate could block retrograde conduction into the Purkinje system (Figure 2B), potentially suppressing reentry involving the Purkinje network.

Catheter ablation targeting VF-triggering PVC is effective in preventing recurrent VF storms.[1,2] Determining the earliest Purkinje potentials preceding the PVC is key to

area showing Purkinje potential at Octaray C3–4 and D1–2. A delayed potential was recorded at the Purkinje potential recording site 144 ms after the QRS (*arrow*). (Right) A gradual conduction delay between the QRS and the abnormal potential (*arrows*) (first beat: 144 ms, second beat: 172 ms, third beat: 208 ms) was observed, resulting in PVC.

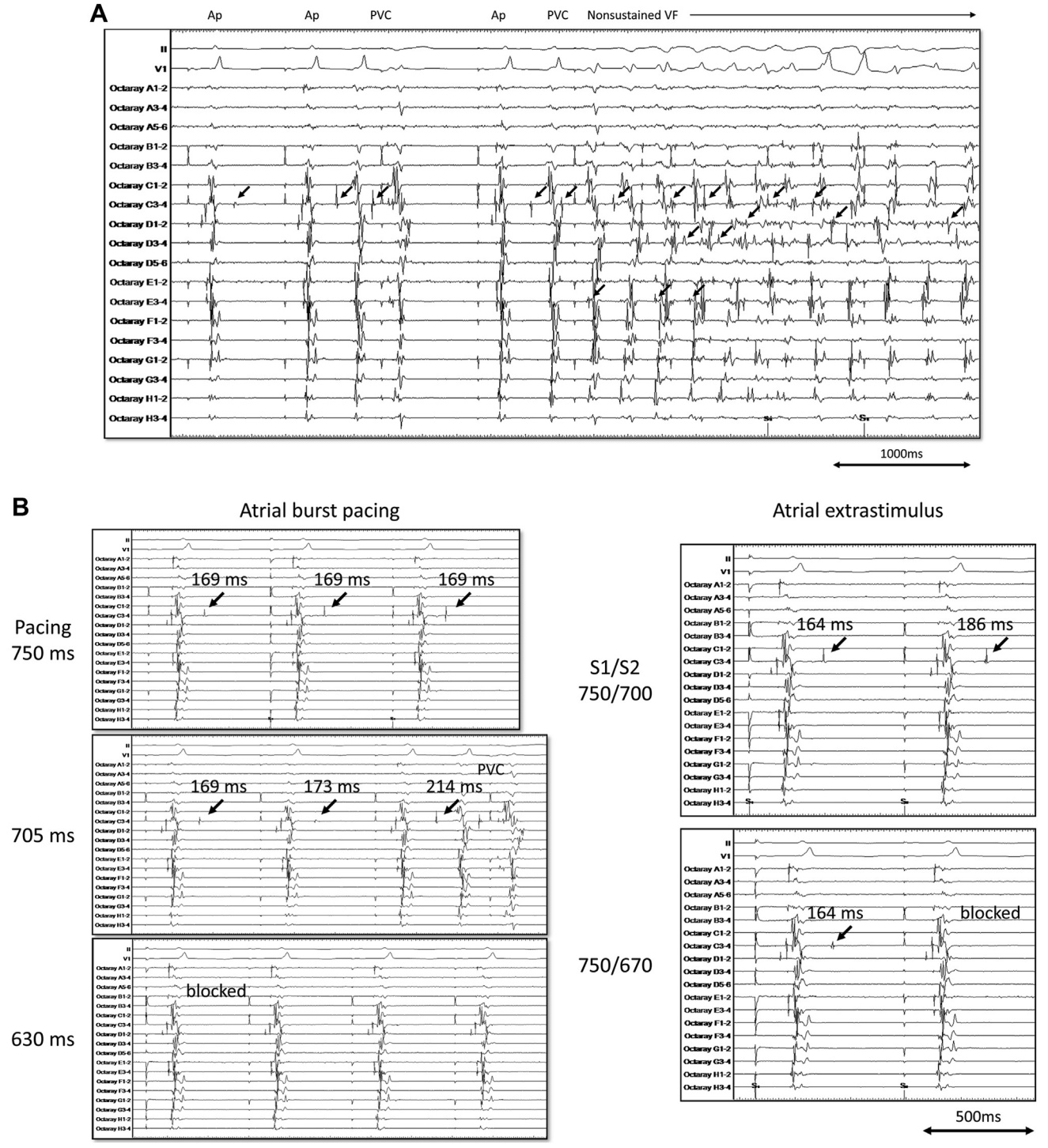

Figure 2 **A:** During atrial pacing (Ap), Octaray (Biosense Webster, Diamond Bar, CA) positioned along the left bundle branch area showed Purkinje potential. Following the second sinus beat, 2 premature ventricular contractions (PVC) occurred with the prolongation of the QRS to the Purkinje potential. After the third sinus beat, nonsustained ventricular fibrillation (VF) occurred. During nonsustained VF episodes, the Purkinje potential preceded the local myocardial potential (*arrows*), suggesting the involvement of the Purkinje network in VF maintenance. **B:** (Left) Recordings during atrial burst pacing showed decremental conduction. During atrial burst pacing at a rate of more than 750 ms, the conduction time between the QRS and the Purkinje potential was constant, with a coupling interval of 169 ms. At a rate of 705 ms, gradual prolongation of the conduction time was observed (first beat: 169 ms, second beat: 173 ms, third beat: 214 ms), which led to PVC. Finally, the QRS to the Purkinje conduction was blocked at a rate of more than 630 ms. (Right) Recordings during atrial extrastimulus showing decremental conduction between the QRS and delayed Purkinje potential (*arrows*). The basic atrial pacing cycle length was 750 ms, and atrial extrastimulus was delivered in a decremental fashion for 10 ms. The conduction time between the QRS and the Purkinje potential was 164 ms until delivering extrastimulus with a coupling interval of 710 ms. This conduction time started to be prolonged by extrastimuli with a coupling interval of 700 ms (186 ms) and was finally blocked by extrastimuli with a coupling interval of 670 ms.

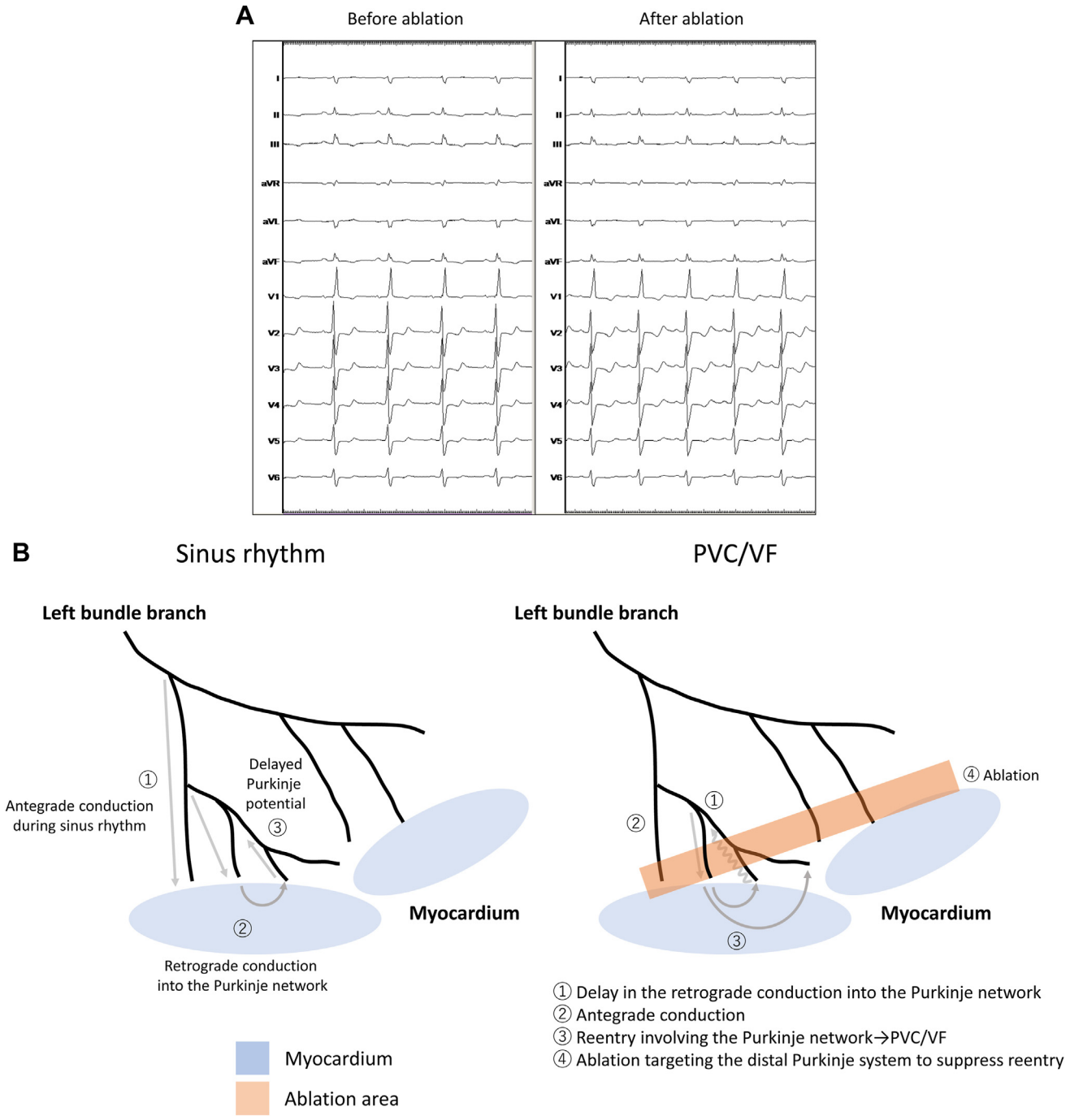

Figure 3 **A:** Twelve-lead electrocardiograms (ECGs) before and after the procedure show almost identical QRS morphology, except for a small s wave in lead II in the ECG after the procedure. **B:** Possible mechanism of premature ventricular contraction (PVC) and ventricular fibrillation (VF) in this case.

successful ablation. However, clinical mapping of PVCs in this situation is challenging because they mostly constitute the first beat of VF. In a large series of patients, atrioventricular block and left bundle branch block occurred in >10% of patients.[2] Since the Purkinje–muscle junction plays an important role in maintaining VF, recent reports have shown that an ablation strategy targeting the Purkinje–muscle junction can suppress VF without targeting the earliest activation site during VF-initiating PVC.[4,12] If the mechanism of PVC and VF is reentry, as in this case, this strategy can suppress VF without targeting the earliest activation site, which may prevent the risk of atrioventricular block.

Conclusion

We experienced a case in which the mechanism of the VF-initiating PVC seemed to originate from reentry involving the Purkinje network. An ablation targeting the distal Purkinje network successfully suppressed VF. This case demonstrates a novel mechanism for Purkinje-related VF after ischemia.

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Data Availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

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