Right atrial parasystole originating from isolated activities in the right inferior pulmonary vein with an epicardial connection

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Introduction
Pulmonary vein (PV) isolation for atrial fibrillation (AF) can now be safely and efficiently achieved thanks to the development of technologies such as 3-dimensional mapping, irrigation-tip catheters, contact force monitoring, and balloon ablation technologies. However, electrophysiologists sometimes encounter PVS that are difficult to isolate, especially when the antral ablation line is designed to be wider in the right-sided PVS, in which ablation in the PV carinal area is commonly effective.1,2 A recent study suggested the presence of an epicardial connection between the right-sided PV carina and right atrium (RA) by showing the close relation between carina breakthrough during sinus rhythm mapping and the requirement of carina ablation to isolate right-sided PVS.3,4 We report a case of RA parasystole originating from activities in the right inferior PV (RIPV) disconnected from the left atrium (LA) by ablation for AF under the presence of an epicardial connection with unidirectional conduction property.

Case report
A 60-year-old man was referred to our institution for catheter ablation of symptomatic, drug-resistant, paroxysmal AF. In the index procedure, right-sided PV carinal ablation following circumferential ablation was required to isolate the right-sided PVS. In a follow-up clinic 1 month after the procedure, the patient complained of paroxysmal palpitations, and bigeminy of atrial premature contractions (APCs) and atrial tachycardia were documented on the 12-lead electrocardiogram (Figure 1A). Immediately after the blanking period of 3 months, a redo procedure was performed under CARTO guidance (Biosense Webster Inc, Diamond Bar, CA). The patient presented to the electrophysiology laboratory in sinus rhythm with the APCs. During sinus rhythm mapping, there was no PV reconnection in any of the PVS except the RIPV. It was difficult to depict a detailed activation map during sinus rhythm because of scarring or a low-voltage area (bipolar voltage of <0.2 mV) created by the prior PV isolation. However, a tiny island with high amplitude (1.45 mV) and discrete potentials was noted in the RIPV (Figure 1B). Circumferential ablation was performed, and ablation at the PV floor achieved a bidirectional conduction block between the RIPV and LA and eliminated the APCs (Figure 2B).

Although frequent dissociated potentials recorded by a Lasso catheter (Biosense Webster) were observed in the RIPV, other APCs were still present. These dissociated activities and sinus rhythm had a regular rhythm of cycle lengths of 2520 ms and 1170 ms, respectively. At this time, we were aware that the dissociated PV activities could conduct to the
atrium only when the atrium depolarized by sinus impulses was outside its refractory period (red closed stars in Figure 2C), and, as a result, the coupling intervals between the P wave of the sinus impulses and the P' wave of the APCs (red arrows in Figure 2C) varied between the APCs. The intervals (ms) from the RA potential of the sinus impulse to the PV potential in the nonconductive activities (red open stars and numbers in parentheses in Figure 2C) also varied, indicating no conduction from the atrium to PV. This suggested a phenomenon of parasystole resulting from the coexistence of an automaticity and a unidirectional conduction from the PV to the atrium. We thought that the presence of residual gaps on the antral ablation line was unlikely because linear and continuous radiofrequency applications targeting an ablation index (Biosense Webster) of 500 were repeated in the first and second ablation procedures. So, we decided to depict an activation map of the APCs also in the RA using a PENTARAY catheter (Biosense Webster), which revealed RA posterior wall breakthrough of the RIPV activities, ie, the exit of the APCs (Figure 3). Taken together, we concluded that this phenomenon was RA parasystole originating from the dissociated activities in the RIPV that were connected to the RA posterior wall through the epicardial connection with unidirectional conduction property. Subsequently, a discrete potential with its earliest activation in the RA was ablated (left panel in Figure 2D), and the APCs immediately disappeared despite residual dissociated activities in the RIPV (right panel in Figure 2D). The distance between the

Figure 1  A: Twelve-lead electrocardiogram of the bigeminy of atrial premature contractions (APC) and atrial tachycardia. B: Activation map of the left atrium during sinus rhythm in the index procedure (left) and voltage map of the left atrium at the second procedure (right). A discrete potential observed in the right inferior pulmonary vein (RIPV) is shown. LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; RSPV = right superior pulmonary vein.
RA ablation site and the site with a discrete potential within the RIPV was 17.8 mm. Pacing maneuvers and isoproterenol infusion did not induce any further APCs, AF, or atrial tachycardia after ablation. The patient has remained free from any atrial tachyarrhythmias without any antiarrhythmic drugs for 8 months.

**Discussion**

From the 1900s, several anatomical studies have indicated the presence of muscular connections between the primary divisions of the heart, including an intercaval bundle connecting between the right-sided PVs and RA. A case series by Patel and colleagues provided the evidence of such a bypass tract in 4 patients undergoing catheter ablation of AF. Both antral ablation encircling the PVs and ablation at the RA entrance site for the epicardial connection were required to isolate the PVs in all patients, although only disconnection between the RIPV and LA terminated the macroreentrant atrial tachycardia, potentially involving the LA, PV, epicardial connection, and RA in their reentrant circuit. Although the atrial tachycardia clinically observed in our patient was not induced during the ablation procedure, we assumed that it had a reentrant mechanism involving the residual conduction gap in the RIPV and epicardial connection.

Recently, our group advanced this consideration by retrospectively analyzing the LA breakthrough sites during sinus rhythm in patients undergoing PV isolation. The presence of carina breakthrough at the right-sided PVs highly predicted the necessity of additional carina ablation following circumferential ablation to isolate the PVs ($P < .0001$), suggesting the presence of an epicardial connection between the right-sided PVs and the RA. The present case further supported our consideration by detailed mapping of the RA and close observation of the relation between the dissociated PV activities and their breakthrough to the RA. Furthermore, this is the first report, to the best of our knowledge, to show that this epicardial connection has a unidirectional conduction property from the PV to RA and can cause an iatrogenic parasystole in association with PV isolation. Although the mechanisms of the unidirectional conduction are unclear, “source-sink mismatches” may be the most likely explanation. Tissue branching and propagation through a narrow isthmus can create an abrupt change in electrical load, resulting in an imbalance between the current available upstream.
and the actual current required to excite cells downstream (source-sink mismatch), thus forming the unidirectional conduction or block. An epicardial connection could be a fine muscular fiber with regional heterogeneities of tissue structure and cellular ionic properties such as a bypass tract in Wolff-Parkinson-White syndrome that provides a favorable milieu for the occurrence of source-sink mismatch and unidirectional conduction. Another explanation might be that carina ablation in the index procedure and ablation of the anterior antrum of the RIPV in the index and second procedures could have altered the conduction properties of the epicardial connection (Figure 1B).

The precise diagnosis of an epicardial connection conducting to the RA and an occurrence of unidirectional conduction is clinically important. We must consider that there are a significant number of patients in whom circumferential ablation cannot achieve right-sided PV isolation without carina ablation to avoid excessive ablation on the antral ablation lines, especially when the antral ablation line is designed to be wider. Fortunately, because RA parasystole spontaneously and frequently occurred in this patient, we could create a map of its activation. However, RA mapping during pacing within the right-sided PV may be recommended to identify the RA breakthrough site in patients with a possible epicardial connection. Moreover, although ablation at the RA breakthrough site may be a more efficient and safer way than carina ablation to eliminate conduction of the epicardial connection, the possible lower efficacy of RA ablation than of carina ablation owing to the broader attachment of the epicardial connection to the RA than to the carina must be considered.

Conclusions
The presence of an epicardial connection between the right-sided PVs and RA was supported by observation of the electrophysiological findings and ablation results. This is the first report of this bypass tract showing a unidirectional conduction property and causing RA parasystole following disconnection between the PVs and LA. Ablation at the RA breakthrough successfully eliminated it and the AF without carina ablation, but further investigation is required to determine the best strategy for treating this entity.

Acknowledgment
We thank Ms Mari Ebine and Ms Yoshiko Uehara for their technical support during the ablation procedure.

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