Bigeminal Pulmonary Vein Ectopy Suppressed by Pulmonary Vein Isolation

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SUMMARY

A 58-year-old man with atrial fibrillation underwent pulmonary vein (PV) isolation (PVI). Bigeminal atrial premature beats persisted from the beginning of the PVI. The cardiac recordings from a basket catheter (BC) revealed the PV ectopic origin in the distal right superior PV. Successful PVI with the guidance of BC was confirmed by the appearance of concealed ectopy. Surprisingly, the PV ectopy completely disappeared immediately after the successful PVI. The findings suggest that the generation of PV trigger is sometimes dependent on left atrial input and that the underlying mechanism of the PV trigger may have been triggered activity or reentry. (Int Heart J 2008; 49: 129-132)

Key words: Premature atrial contraction, Bigeminy, Pulmonary vein, Isolation

THE pulmonary veins (PVs) have been demonstrated to be the major source of ectopic beats triggering paroxysmal atrial fibrillation (AF) and the electrical activity generating the ectopic beats can be recorded from the PV sleeve.1-3) Although previous studies of PV electrophysiology have proposed that triggered activity or reentry favored by left atrial inputs may cause PV tachycardia during AF,4-6) the details of the mechanism of PV ectopic beats triggering AF are still unknown.

CASE REPORT

A 58-year-old Japanese man with idiopathic, drug-refractory paroxysmal AF was referred for an electrophysiologic study (EPS) and radiofrequency catheter ablation. He complained of palpitations from AF which continued for a few hours, 2 or 3 times a week despite taking antiarrhythmic drugs. There was no echocardiographic evidence of structural heart disease. The left atrial dimension

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Received for publication July 17, 2007.
Revised and accepted December 27, 2007.
and left ventricular ejection fraction were 31 mm and 0.69, respectively. Informed consent was obtained, and an EPS was performed after all antiarrhythmic drugs had been discontinued for at least 5 half-lives prior to the study.

At the beginning of the EPS, a simple mapping technique using multielectrode catheters placed in the posterior right atrium (PRA), esophagus and coronary sinus (CS) was performed to identify the AF foci. A 7-French decapolar catheter with 1-5-1-mm interelectrode spacing between each electrode pair (St. Jude Medical, AF Division, Minnetonka, MN, USA) was introduced into the CS via the subclavian vein. The position of the most proximal electrode pair at the CS ostium was confirmed with a contrast injection. A 7-French duodecapolar catheter with 1-3-1-mm interelectrode spacing (CRISTA CATH™, Cordis Webster, Baldwin Park, CA, USA) was positioned along the PRA with the distal electrode pairs in the superior vena cava. A 10.5-French octapolar catheter (6-mm electrode width and 15-mm interelectrode spacing) (TO 8™, Dr. Osypka, Grenzach-Wyhlen, Germany) was inserted into the esophagus. AF was induced by

**Figure.** Spontaneous bigeminal pulmonary vein (PV) ectopy originating from the distal right superior PV (arrowheads) persisted from the beginning of the PV isolation procedure. The earliest activation of the ectopy was recorded from electrode pair A5-6. The activation during sinus rhythm conducted into the PV via spline D (entrance) and the activation during the ectopy exited the PV via the same spline (exit). Radiofrequency application around electrode pair D3-4 (electrical connection between the left atrium and PV) could achieve successful PV isolation, which was confirmed by the appearance of concealed PV ectopy (dotted arrows). Thereafter, the PV ectopy as well as the passive PV activation from the left atrium (solid arrows) completely disappeared. A to H indicate the splines of the multielectrode basket catheter; ESO, the transesophageal lead; ABLd, the distal electrode pair of the ablation catheter; and RF, radiofrequency.
intermittent atrial pacing, from 10 to 15 beats at a cycle length between 200 to 300 ms from the distal CS. The simple mapping technique identified AF triggered by frequent atrial premature beats (APBs) with a short coupling interval and suggested that the APBs originated from the right superior PV (RSPV). After transseptal catheterization of the left atrium, a 31-mm multielectrode basket catheter (MBC) (Constellation™, EP Technologies, Boston Scientific Corporation, San Jose, USA) was deployed within the RSPV coaxially to its long axis and with its most proximal electrodes positioned at the left atrial-RSPV junction. Thereafter, bigeminal APBs persisted throughout the RSPV isolation procedure. The MBC recordings revealed that bigeminal PV ectopic beats originated from a site around MBC electrode pair A5-6 (Figure). Segmental PV ostial isolation with the guidance of an MBC was performed by our previously reported technique.8,9) Successful PV isolation was achieved during radiofrequency application delivered around MBC electrode pair D3-4 away from the presumed ectopic origin, and it was confirmed by the appearance of concealed PV ectopy (Figure). Surprisingly, the bigeminal PV ectopy completely disappeared immediately after the successful PV isolation (Figure). Following this, isolation of the other 3 PVs was successfully achieved by the same technique above. Thereafter, neither AF nor APBs could be induced despite programmed electrical stimulation as well as isoproterenol infusion. Follow-up was performed at 2 weeks, 1 month and every month thereafter, using 24-Holter and cardiac recordings. When the patient reported symptoms, he was given an event monitor to document the cause of the symptoms. During more than one year of follow-up, this patient has been free of symptomatic AF without any antiarrhythmic drugs. No complications occurred.

**DISCUSSION**

Basic studies on PV electrophysiology have proposed the mechanism of AF generation as below.6) Repetitive PV activity due to either reentry or triggered activity would give rise to PV tachycardias. PV tachycardias could produce AF directly by activating the atria more rapidly than all regions could follow (fibrillatory conduction) or engaging an appropriate atrial substrate to initiate a multiple circuit atrial reentry. PV tachycardias with a shorter cycle length than the adjacent left atrium appear to maintain AF by serving as a driver during AF.4) However, PV tachycardias in turn depend on the atrial input because they largely disappear when PVs are electrically disconnected from the left atrium.4) Thus, AF may produce positive feedback on the PV activity by potentiating the PV triggered activity.6) In our case, the PV ectopy ceased immediately after isolation of the PV. Therefore, this case suggested that the generation of the PV ectopy triggering the AF also depended on the left atrial input and that the underlying mechanism of
the PV trigger might have been triggered activity or reentry.

It is known that some types of activity such as intrinsic slow PV activity with automaticity is sometimes observed within the isolated PVs. A case report demonstrated that PVs that are electrically disconnected from the atria could show paroxysmal tachyarrhythmias with a fibrillatory appearance. In that case, automaticity could favor triggered activity or reentry serving as a trigger and then the triggered activity or reentry could cause PV tachyarrhythmias. The underlying mechanism of the PV trigger in such a case would differ from that in our case.

Reithmann, et al also reported that achievement of ostial PV isolation was associated with the elimination of the bigeminal PV ectopy. However, we believe that they cannot exclude mechanisms other than a left atrial input, such as a change in the autonomic tone or hemodynamics, because they did not show a dynamic disappearance of the bigeminal PV ectopy. Therefore, we believe that this case report provides evidence for resolving the underlying mechanism of the PV trigger.

REFERENCES