Case Report

Use of Catheter Ablation in the Treatment of Ventricular Tachycardia Triggered by Premature Ventricular Contraction

Isao Kato MD, Toru Iwa MD, Yasushi Suzuki MD, Takayuki Ito MD

Division of Cardiology, Aichi Medical University School of Medicine

A 50-year-old man who had suffered from old myocardial infarction presented with an episode of syncope. DC shock was required for the interruption of frequent pleomorphic ventricular tachycardia (VT). Although the treatment for heart failure decreased the frequency of VT attacks, hemodynamically unstable VT occurred several times. A 12-lead Holter electrocardiogram was used to determine the triggering premature ventricular contraction (PVC) and catheter ablation was performed by targeting this PVC. The site of origin of the triggering PVC was considered to be located between damaged cardiac muscle and intact Purkinje's fiber. No episode of PVC and VT was observed after a few days of ablation. An implantable cardioverter defibrillator was implanted but VT did not recur for more than 20 months.
(J Arrhythmia 2006; 22: 174–179)

Key words: Ventricular tachycardia storm, Purkinje potential, Left posterior fascicle, Old myocardial infarction, 12-lead Holter electrocardiogram

Introduction

Recent reports indicate that catheter ablation for the elimination of triggering premature ventricular contraction (PVC) might be useful for the treatment of ventricular tachycardia (VT) or ventricular fibrillation.[1–4] We present a case in which catheter ablation succeeded by first assessing the origin of triggering PVC of hemodynamically unstable VT using a 12-lead Holter electrocardiogram (ECG).

Case Report

A 50-year-old man was admitted to our hospital for assessment of syncope. VT occurred frequently, accompanied with hemodynamic instability. Although antiarrhythmic drugs such as amiodarone and nifekalant were administered, VT could not be prevented and DC shock was frequently required at hourly intervals. VT episodes showed several different QRS morphologies indicating the existence of several different reentrant circuits (**Figure 1**). Deep sedation with endotracheal intubation prevented VT storm. However, VT storms occurred when sedation was decreased. Coronary angiography revealed total occlusion of segment (Seg.) 6. Left ventriculography revealed akinesis of Seg. 2 and dyskinesis of Seg. 3, which suggested an old myocardial infarction ac-

Received 5, December, 2005: accepted in final form 15, September, 2006.
Address for correspondence: Isao Kato MD, Division of Cardiology, Aichi Medical University School of Medicine, 21 Karimata Yazako, Nagakute, Aichi 480-1195. Tel: 0561-62-3311 Fax: 0561-63-8482

companied by ventricular aneurysm. As the heart condition deteriorated, PVCs that triggered VTs emerged from various sites due to increased end-diastolic pressure.

Deteriorating symptoms (CTR 66% and EF 27%) improved (CTR 55% and EF 31%) as a result of the treatment for heart failure, and VT storms did not occur even by decreasing the sedation. Hemody-

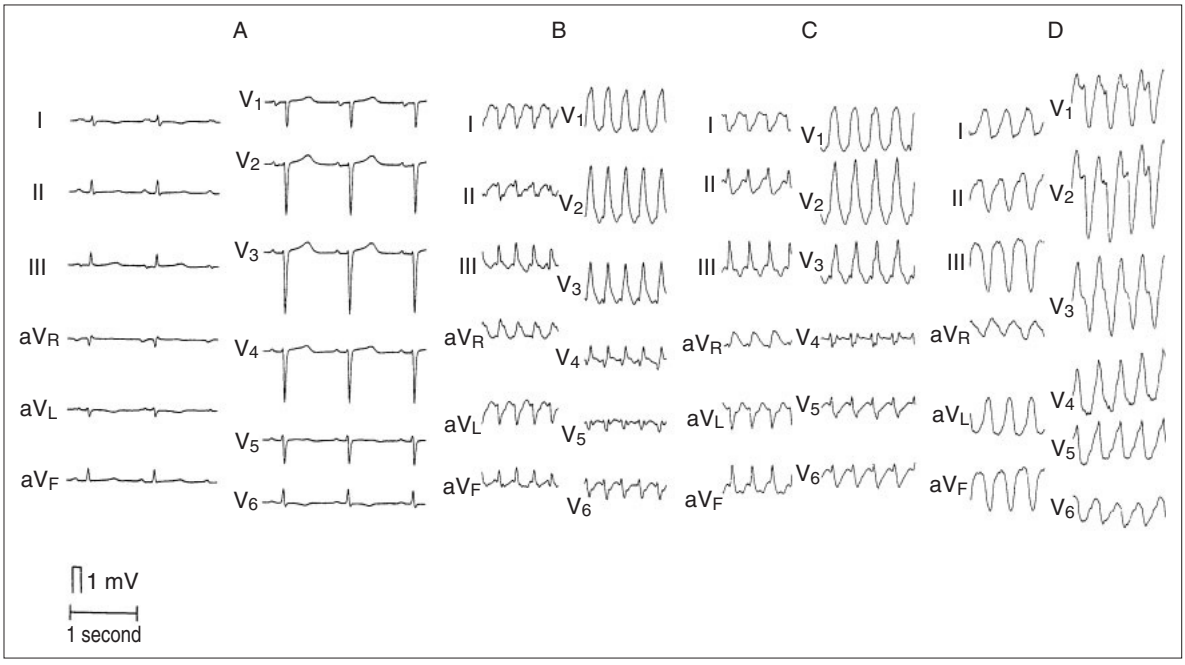

Figure 1 12-lead ECG at rest (A) and during VT (B: 222 bpm, C: 190 bpm, D: 180 bpm). The VTs were pleomorphic and several reentrant circuits were present.

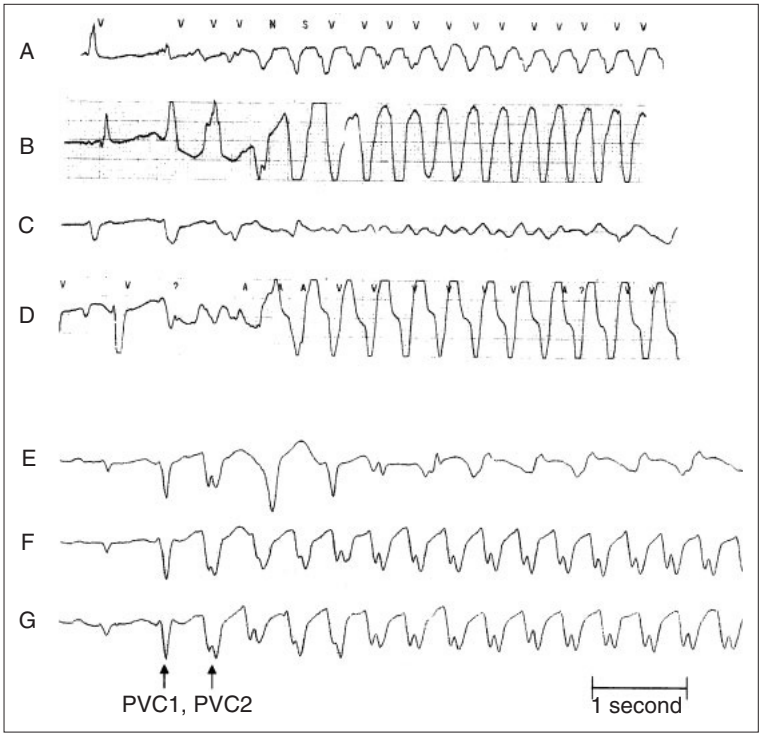

Figure 2 Traces of ECG monitoring. Electrodes were maintained on the same position and were recorded using the same lead. A, B, C and D: VT during early days of hospitalization. Fast VTs and ventricular fibrillation were caused by PVCs due to various triggers. E, F and G: VT after heart failure was improved by medication. VTs emerged from two PVCs (PVC1, PVC2) with the same consecutive QRS morphologies.

namically unstable VT appeared several times, however. Although antiarrhythmic drugs including β-blockers were administered, sufficient control of VT was not achieved.

ECG monitoring after the improvement of heart failure showed two triggering PVCs for VT episodes, each with a different QRS morphology (**Figure 2**).

Twelve lead Holter electrocardiogram

Since the PVC originated at a specific point, ECG monitoring and 12-lead Holter ECG were recorded simultaneously to identify the PVC morphology leading to the VT. PVCs with the same QRS morphology were recorded on 12-lead Holter ECG analysis. The first PVC showed left axis deviation and right bundle branch block pattern with relatively narrow QRS. The second PVC showed left axis deviation and right bundle branch block pattern with a wider QRS interval than that of the first PVC (**Figure 3**). From the 12-lead Holter ECG, the origin of PVC was assumed to be in the posteroseptal area of the left ventricle (LV) by the polarity of the QRS from each lead.

Because hemodynamically unstable VT could not be prevented, catheter ablation was performed for the PVC that triggered the VT.

Cardioelectrophysiologic study and catheter ablation

When the patient entered the electrophysiological laboratory, idioventricular rhythm (heart rate, 80 beats per minute) was observed (**Figure 4**). First, LV mapping was performed using the electroanatomical mapping system during constant pacing (100 paces per minute) from the high right atrium. Purkinje potential was recorded at the LV mid-posteroseptal area. At the same site, the Purkinje potential preceding the PVC was also recorded (**Figure 5**). The voltage map recorded an extensive low voltage area in the anteroseptal region (**Figure 6**, left).

Since the QRS morphology of the idioventricular rhythm was the same as that of the second triggering PVC, mapping of the earliest activation site of idioventricular rhythm was performed. The earliest

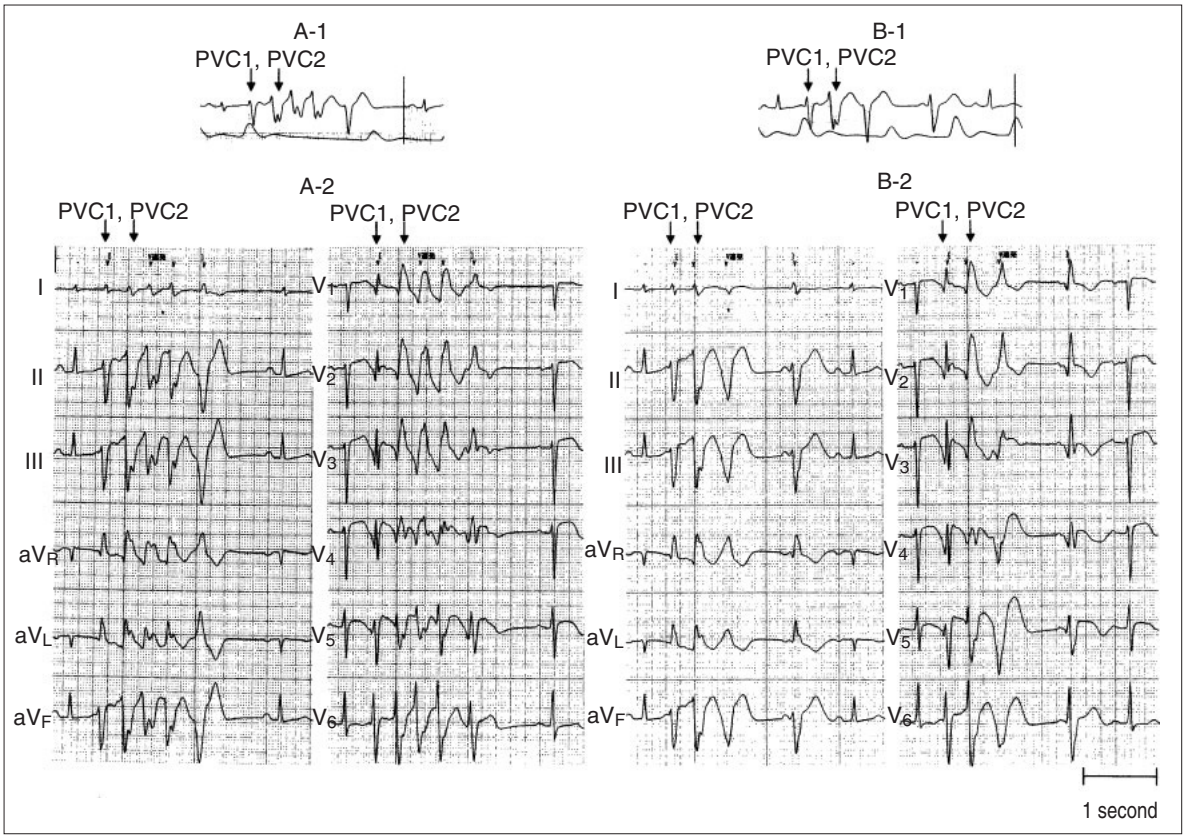

Figure 3 Simultaneous recording of ECG monitoring and 12-lead Holter ECG.
A-1: Monitor ECG recorded during PVC run. A-2: 12-lead Holter ECG recorded at the same time as A-1. B-1: ECG monitoring recorded during PVC run. B-2: 12-lead Holter ECG recorded at the same time as B-1. PVC1, 2 showed left axis deviation, right bundle branch block pattern and were indicated to emerge from the left posterior fascicle.

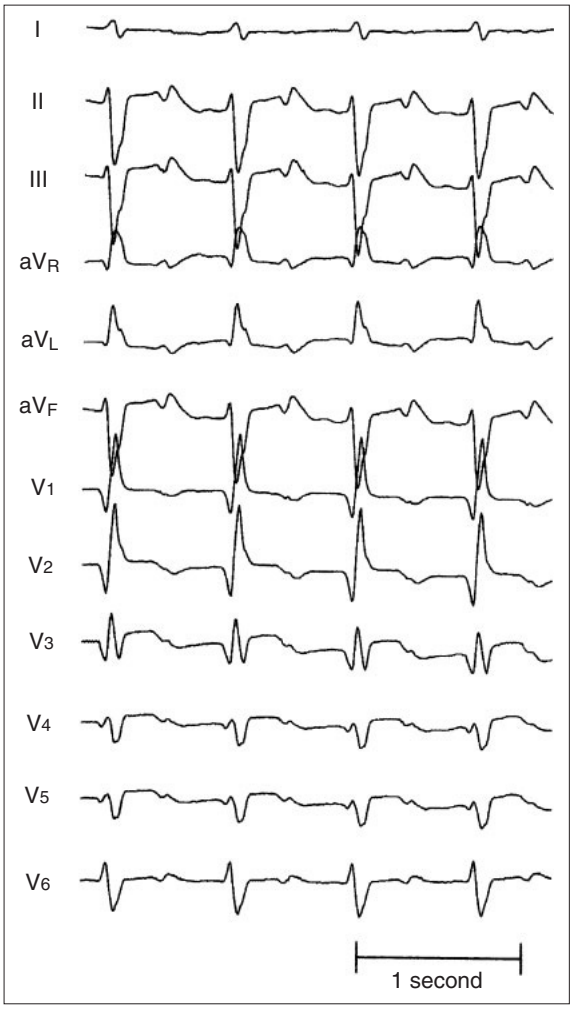

Figure 4 ECG results at the time of the electrophysiologic study.
ECG shows idioventricular rhythm, which was the same QRS morphology as PVC2.

activation site was the LV mid-posteroseptal area, observed on the boundary between low and normal voltage areas. The earliest activation site was identical to where the Purkinje potential was recorded during constant pacing from the high right atrium. It was considered that the idioventricular rhythm may have originated from the left posterior fascicle. The sites with Purkinje potentials were located linearly (**Figure 6**, blue tags). However, the pacing from these sites except for the earliest activation site of idioventricular rhythm could not reproduce the QRS morphology of trigger PVC and they were not on the boundary between low and normal voltage areas. Therefore, the ablation was performed at the earliest activation site of the idioventricular rhythm and additional ablation was performed to the surrounding area, especially for the low voltage area (**Figure 6**, right).

During the ablation, an automatic excitation was observed. The idioventricular rhythm and PVC disappeared after ablation on this area. After confirming no recurrence of PVC for one hour, the session was terminated. Although hemodynamically unstable VT occurred several times in the first three days after catheter ablation, the QRS morphology of triggering PVC was slightly different from that before the catheter ablation. Since ablation may cause inflammation or edema in surrounding tissues and may result in enhanced automaticity, the patient was observed without adding further therapy for a while. Then VT as well as PVC disappeared four days after ablation. After that, an implantable cardioverter defibrillator (ICD) was implanted and the patient was discharged from our hospital. VT did not recur for more than 20 months.

Discussion

In the present case of a patient with hemodynamically unstable pleomorphic VT, it was technically difficult to localize the origins of the VTs in routine electrophysiologic study. We utilized the 12-lead Holter ECG to determine the triggering PVC for VT episodes and two different QRS morphologies were identified. In accordance with the polarity of these QRS morphologies of PVCs, they were speculated to be located in the LV posteroseptal area. Because the first PVC showed a relatively narrow QRS complex, the origin was considered to be located in an area close to the left posterior fascicle. The second PVC showed relatively wider QRS complex and it was the same morphology as that of idioventricular rhythm observed in the ablation session. Because the width of QRS complex of the second PVC was wider than that of the first PVC, the origin of the second PVC was considered to be located in an area relatively far from the Purkinje system. In the ablation session, radiofrequency ablation targeting the earliest activation site of the second PVC and idioventricular rhythm successfully eliminated the second PVC, and interestingly, it also eliminated the first relatively narrow PVC. The precise mechanism of this result was unclear, but it was speculated that two PVCs were located in a relatively small area and extended ablation for the origin of PVC including surrounding area might eliminate both the foci. By identification of VT triggering PVC by utilizing 12-lead Holter ECG recording, catheter ablation could be performed effectively even in a case with hemodynamically unstable VT without direct activation mapping of the VT.

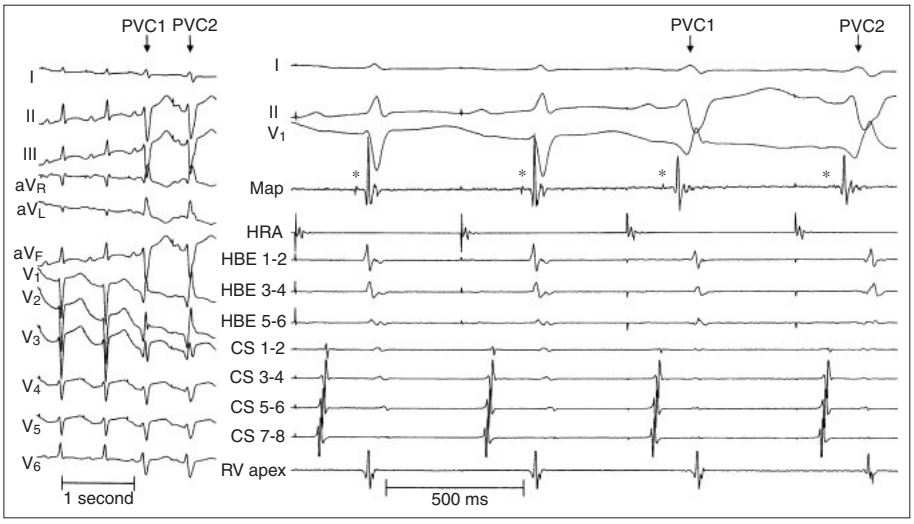

Figure 5 PVC during constant pacing from the high right atrium (HRA).
Left: Surface ECG at the time of recording two PVCs with the same QRS morphology as PVC1 and PVC2 recorded with 12-lead Holter ECG during electrophysiologic study. Right: Intracardiac electrogram recorded at the same time. Mapping catheter placed on the mid-posteroseptal area of the left ventricular recorded Purkinje potential. While the time interval from the Purkinje potential to the initial rise of QRS was 36 milliseconds (ms) during constant pacing from HRA, those of the first and the second PVC were 45 ms and 40 ms, respectively. Purkinje potential preceding PVC was recorded at the left posterior fascicle area.
∗: Purkinje potential
Map: mapping catheter, HBE: His bundle electrogram, CS: coronary sinus, RV: right ventricle

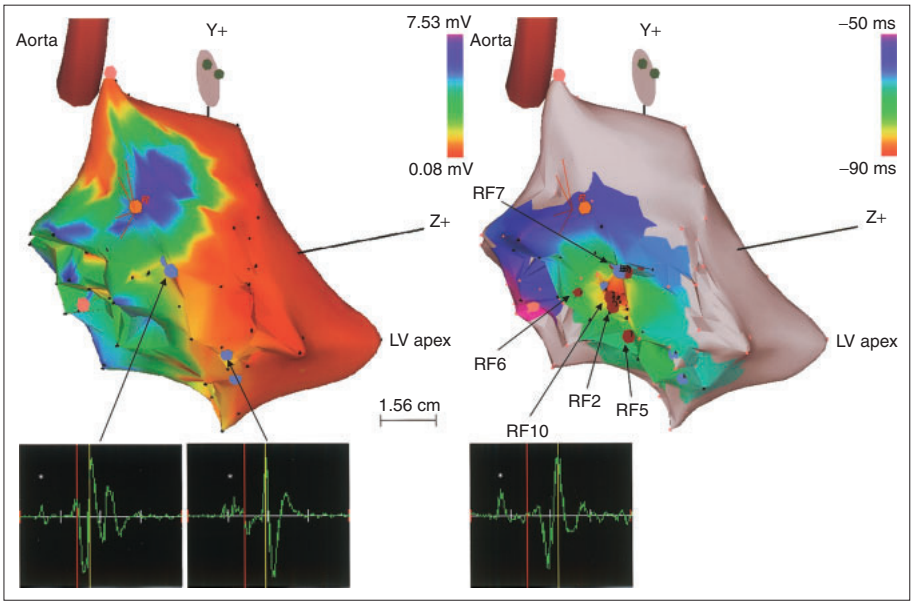

Figure 6 Electroanatomical mapping.
Left: Bipolar voltage map performed during constant pacing from HRA. Purkinje potential was recorded from the mid-posteroseptal area of the left ventricular (LV) to the apex side. An extensive low voltage area was recorded at the anteroseptal area of the LV. The red area shows an ischemic area with low voltage. Blue tags are the sites where the Purkinje potential was recorded. Below left: Purkinje potential.
∗: Purkinje potential
Right: Activation map performed during the idioventricular rhythm. The earliest activation site was the mid-posteroseptal area of the LV defined in red. The earliest activation site was located on the boundary between the low voltage (red) and the normal voltage area (green), and also the site where the Purkinje potential was recorded. Brown tags indicate ablation site. The left posterior fascicle was cauterized.
RF: radio frequency (the number indicates RF No.), LV: left ventricle

The role of the Purkinje system in this case was unclear. The localization of the PVC on the border zone between a normal and low voltage area indicates the involvement of reentry as the mechanism, but spontaneous and frequent appearance of PVC and increased firing during ablation indicates enhanced automaticity as the mechanism. Because a spikey-prepotential was observed at an area close to the ablation site only during the PVC, abnormal automaticity of Purkinje system might be involved in the mechanism.[5,6] Heterogeneity of the ventricular muscle between normal and low voltage area might play a role in this case. Our reasoning was that an electric potential difference was generated between the cardiac muscle damaged by myocardial infarction and Purkinje's fiber that was relatively resistant to ischemia.[7]

Although several VT episodes were observed in early days after the ablation, the VT and PVC disappeared later on. ICD was implanted in this case to prevent sudden death due to hemodynamically unstable VT, but no episode was observed for more than one and half years. In this case, a treatment of the PVC that caused VT was performed. Several different reentrant circuits remained. We may try to perform substrate mapping utilizing a voltage map in an electroanatomical mapping system which should be planed if VT recur frequently in this patient.[8] Currently the patient is well controlled with antiarrhythmic drugs and anti-heart failure drugs. If heart failure dose not turn worse, a reappearance of new PVC that caused VT may be prevented.

By an identification of VT triggering PVC utilizing 12-lead Holter ECG recording, catheter ablation could be performed safely and effectively even in a case with hemodynamically unstable VT without direct activation mapping of the VT. The simultaneous recording of ECG monitoring and 12-lead Holter ECG were useful for diagnosis before catheter ablation.

References

1) Haissaguerre M, Shah DC, Jais P, Shoda M, Kautzner J, Arentz T, Kalushe D, Kadish A, Griffith M, Gaita F, Yamane T, Garrigue S, Hocini M, Clementy J: Role of Purkinje conducting system in triggering of idiopathic ventricular fibrillation. Lancet 2002; 359: 677–678
2) Haissaguerre M, Shoda M, Jais P, Nogami A, Shah DC, Kautzner J, Arentz T, Kalushe D, Lamaison D, Griffith M, Cruz F, de Paola A, Gaita F, Hocini M, Garrigue S, Macle L, Weerasooriya R, Clementy J: Mapping and ablation of idiopathic ventricular fibrillation. Circulation 2002; 106: 962–967
3) Szumowski L, Sanders P, Walczak F, Hocini M, Jais P, Kepski R, Szufladowicz E, Urbanek P, Derejko P, Bodalski R, Haissaguerre M: Mapping and ablation of polymorphic ventricular tachycardia after myocardial infarction. J Am Coll Cardiol 2004; 44: 1700–1706
4) Nogami A: Catheter ablation of primary ventricular fibrillation. Heart View. 2005; 9: 579–587
5) Gilmour RFJ, Moise NS: Triggered activity as a mechanism for inherited ventricular arrhythmias in German shepherd Dogs. J Am Coll Cardiol 1996; 27: 1526–1533
6) Moise NS, Gilmour RFJ, Riccio ML: An animal model of spontaneous arrhythmic death. J Cardiovasc Electrophysiol 1997; 8: 98–103
7) Friedman PL, Stewart JR, Wit AL: Spontaneous and induced cardiac arrhythmias in subendocardial Purkinje fibers surviving extensive myocardial infarction in dogs. Circ Res 1973; 33: 612–626
8) Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM: Electrically unexcitable scar mapping based on pacing threshold for identification of the reentry circuit isthmus: feasibility for guiding ventricular tachycardia ablation. Circulation 2002; 106: 1678–1683