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CASE REPORT

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Characteristics of systolic and diastolic potentials recorded in the left interventricular septum in verapamil-sensitive left ventricular tachycardia

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Abstract

We studied the electrophysiological characteristics of systolic (SP) and diastolic (DP) potentials recorded during sinus rhythm (SR) in the left interventricular septum of a 27 year-old woman presenting with verapamil-sensitive idiopathic left ventricular tachycardia (VT). During SR, and during VT, SP was activated from ventricular base-to-apex, and DP from apex-to-base. SP and DP were both detected at the site of successful ablation during SR, whereas during VT, DP was detected away from the earliest activation site. Thus, SP apparently reflected a critical component of the reentrant circuit, while DP reflected the activation of a bystander pathway. (Cardiol J 2012; 19, 4: 418–423)

Key words: Purkinje fiber, bystander pathway, essential pathway, catheter ablation, idiopathic ventricular tachycardia

Introduction

Verapamil-sensitive idiopathic left ventricular tachycardia (VT) is uniquely characterized by right bundle-branch block QRS morphology, superior axis deviation and responsiveness to verapamil [1–3]. It often originates from the left interventricular septum (LIVS), where several investigators have detected pre-systolic potentials during ongoing tachycardia, which may reflect a critical segment of the reentrant circuit supporting the arrhythmia [4–10].

We recently reported the presence of systolic (SP) and diastolic (DP) potentials during sinus rhythm (SR) on the LIVS in structurally normal hearts and in the absence of ventricular arrhythmias [11]. The characteristics of SP and DP, including their a) basal or midseptal location, b) morphology and activation sequence, and c) slow conduction properties, are strikingly similar to those of the pre-systolic potentials detected in patients with verapamil-sensitive VT, though the role they play in the arrhythmic mechanism remains to be clarified.

The aim of this study was to define the characteristics and electrophysiological properties of SP and DP in a patient presenting with verapamil-sensitive VT.

Case report

A 27 year-old woman without apparent structural heart disease was admitted to our hospital for catheter ablation of a paroxysmal tachycardia with right bundle branch QRS morphology and left axis deviation, terminated by intravenous administration of verapamil, 2.5 mg, i.v.

Transthoracic two-dimensional echocardiography revealed the absence of left ventricular false tendons. The patient, who had granted her written informed consent, underwent electrophysiological

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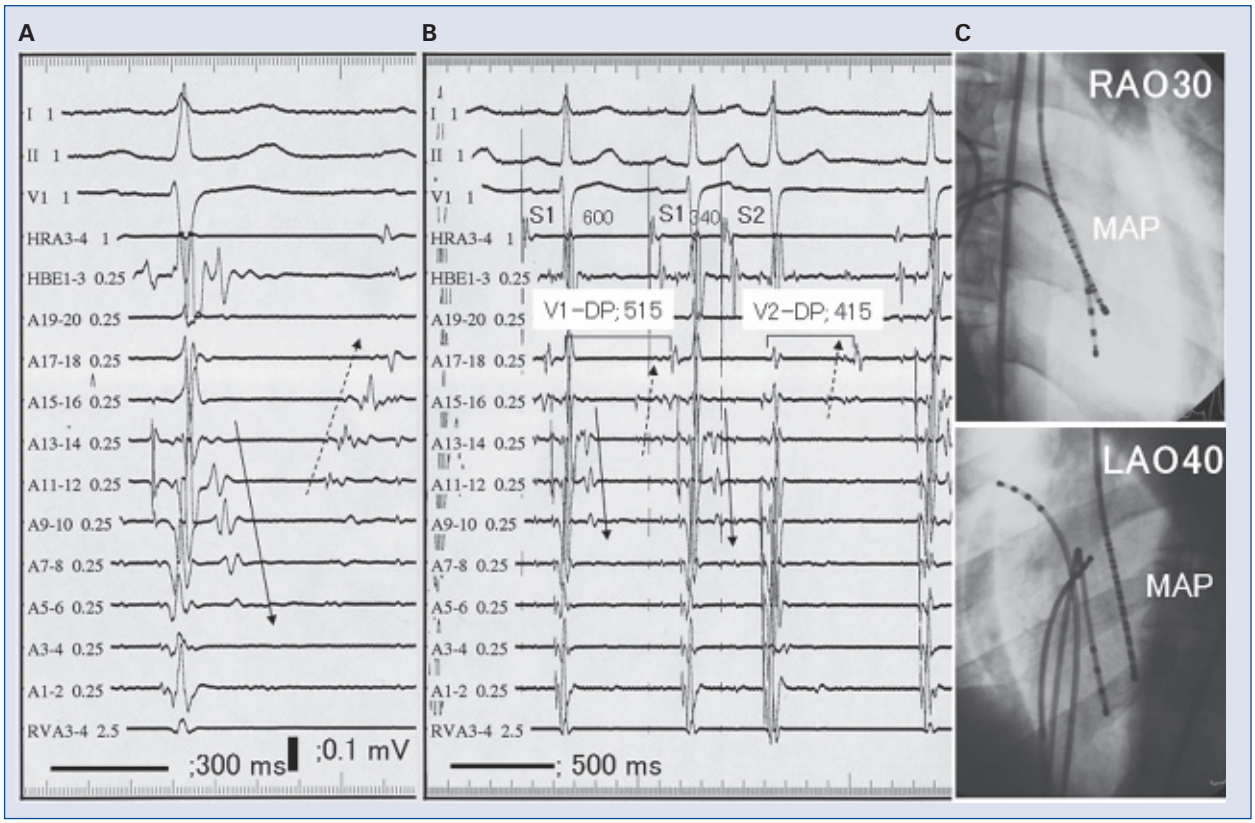

Figure 1. Systolic and diastolic potentials recorded during sinus rhythm (**A**) and high right atrial extrastimulation (**B**), and right (RAO) and left (LAO) anterior oblique fluoroscopic views (**C**) showing the position of the mapping catheter (MAP); **A.** Activation of the left bundle branch potentials in a basal-to-apical sequence, preceding activation of the ventricular myocardium from apex to base suggests that the Purkinje-muscle junction was located near the fusion point of the two potentials. Solid and dashed arrows indicate the direction of systolic (SP) and diastolic (DP) potentials activation, respectively; AH = 78 ms; HV = 45 ms; QRS onset to SP = 98–143 ms; SP amplitude = 0.20––0.86 mV; QRS onset to DP = 370–498 ms; SP amplitude = 0.37–0.86 mV; **B**: At an S1–S2 coupling interval of 340 ms and S1–S1 basic cycle length of 600 ms, the V2–DP interval is shorter than the V1–DP interval, suggesting incremental conduction properties of the tissue between the site of ventricular pacing and DP. SP is no longer visible after S2, as the effective refractory period of the tissue between the site of ventricular pacing and SP has been reached; **C.** Recordings of SP and DP (A17–18 to A5–6) are from the basal septum just below the aortic valve to the mid septum; I, II, V1 = surface ECG leads; HRA — high right atrium; HBE — His-bundle electrogram; A19–20 to A1–2 — left ventricular mapping sites from base to apex

study and catheter ablation procedure after discintinuation of all antiarrhythmic drugs for > 5 half--lives. Multi-electrode catheters were placed transvenously in the high right atrium (HRA), His-bundle region and at the right ventricular apex (RVA) or the right ventricular outflow tract (RVOT). Bipolar intracardiac electrograms were filtered between 30 and 400 Hz and stored with the 12-lead surface electrocardiogram on electronic medium (EPLab/EP Amp™, Quinton Electrophysiology Co., Seattle, WA, USA) for further analysis. A 20-pole, 7 F, A-20™ steerable electrode catheter (Biosense Webster, Inc., Diamond Bar, CA, USA) with 1-mm interelectrode spacing and 3-mm distance between adjacent electrode pairs was introduced retrogradely into the left venricle. The LIVS surface was meticulously mapped during sinus rhythm in search of potentials following the ventricular electrogram. We identified two types of low-frequency late potentials along a narrow base-to-apex line at the base or mid segment of the left posterior fascicle. The first (SP) was recorded during systole, in a ventricular base-to-apex direction; the second (DP) was recorded during diastole, in an apex-to-base direction (Fig. 1A). The position of the mapping catheter recording these potentials, viewed in the right and left anterior oblique fluoroscopic projections, is shown in Figure 1C.

Single extrastimuli, and trains of stimuli, were delivered from the HRA, RVA and RVOT during

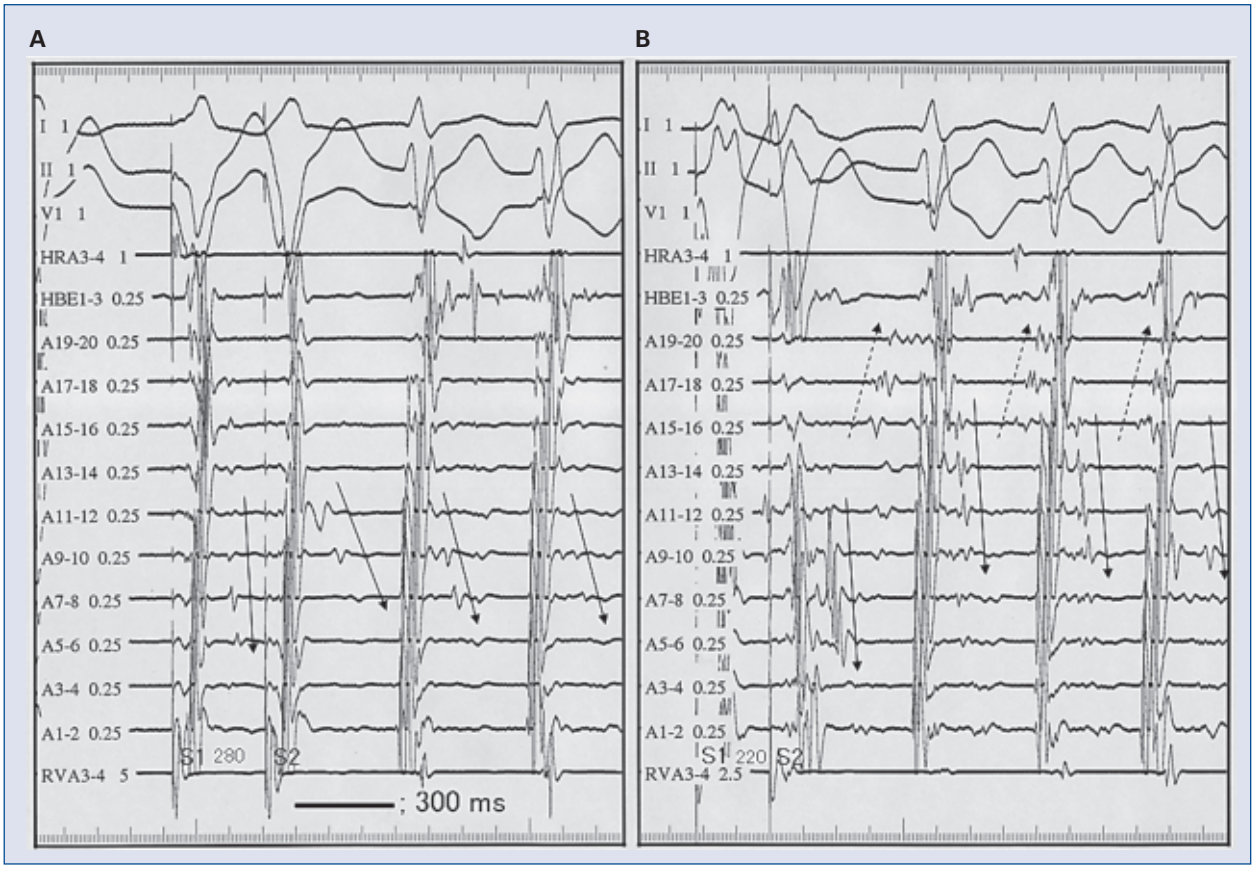

Figure 2. Systolic (SP) and diastolic (DP) potentials recorded during ventricular tachycardia induced by extrastimulation at the right ventricular apex (**A**) and right ventricular outflow tract (**B**). Solid and dashed arrows show the direction of activation of SP and DP, respectively; **A.** At an S1–S2 coupling interval of 280 ms and S1–S1 basic cycle length of 500 ms, S2–SP is distinctly longer than S1–SP, particularly at the apical end of SP (A9–10), indicating the presence of decremental conduction between ventricular myocardium and SP activation; **B.** Induction of ventricular tachycardia by ventricular extrastimulation at an S1–S2 interval of 220 ms and S1–S1 basic cycle length of 500 ms (not shown). The activation sequence of SP and DP during ongoing ventricular tachycardia is similar to that shown during sinus rhythm in Figure 1A. Note that the earliest ventricular activation is at A1–2, in the mid septum, away from the recording of DP. Other abbreviations are as in Figure 1.

stable recordings of SP and DP in SR. Atrial and ventricular pacing reproduced the same SP and DP morphology and activation sequence observed during SR (Figs. 1B, 2). Atrial or ventricular extrastimulation, or increasingly faster atrial or ventricular overdrive pacing, were associated with a gradually longer delay between ventricular activation and SP, consistent with decremental conduction properties of the tissue between ventricular capture and SP activation (Fig. 2A). Furthermore, the disappearance of SP after atrial or ventricular extrastimuli at critically short coupling intervals suggested that the effective refractory period of the tissue between the ventricular myocardium and the recording site of SP had been reached (Fig. 1B). In contrast to SP, atrial or ventricular pacing was associated with incremental conduction properties of DP (Fig. 1B). Single extrastimuli or burst stimulation from the RVA or RVOT reproducibly induced verapamil-sensitive VT with a 105-ms QRS duration and A-V dissociation (Fig. 2), terminated by ventricular burst stimulation. During VT (Fig. 2B) and entrainment pacing from the HRA, the activation sequence of SP and DP was similar to that observed during SR. The site of earliest ventricular activation was in the mid septum, away from the recording of DP (Fig. 2B). Entrainment pacing from the RVOT confirmed a reentrant mechanism of VT, capturing the ventricular myocardium, followed by SP, which was activated in a base-to-apex sequence, similar to that observed during VT. VT was induced while both SP and DP were being recorded during SR (Fig. 3A) from an ablation catheter located in the inferior mid septum (Fig. 3C). Entrainment from that site sug-

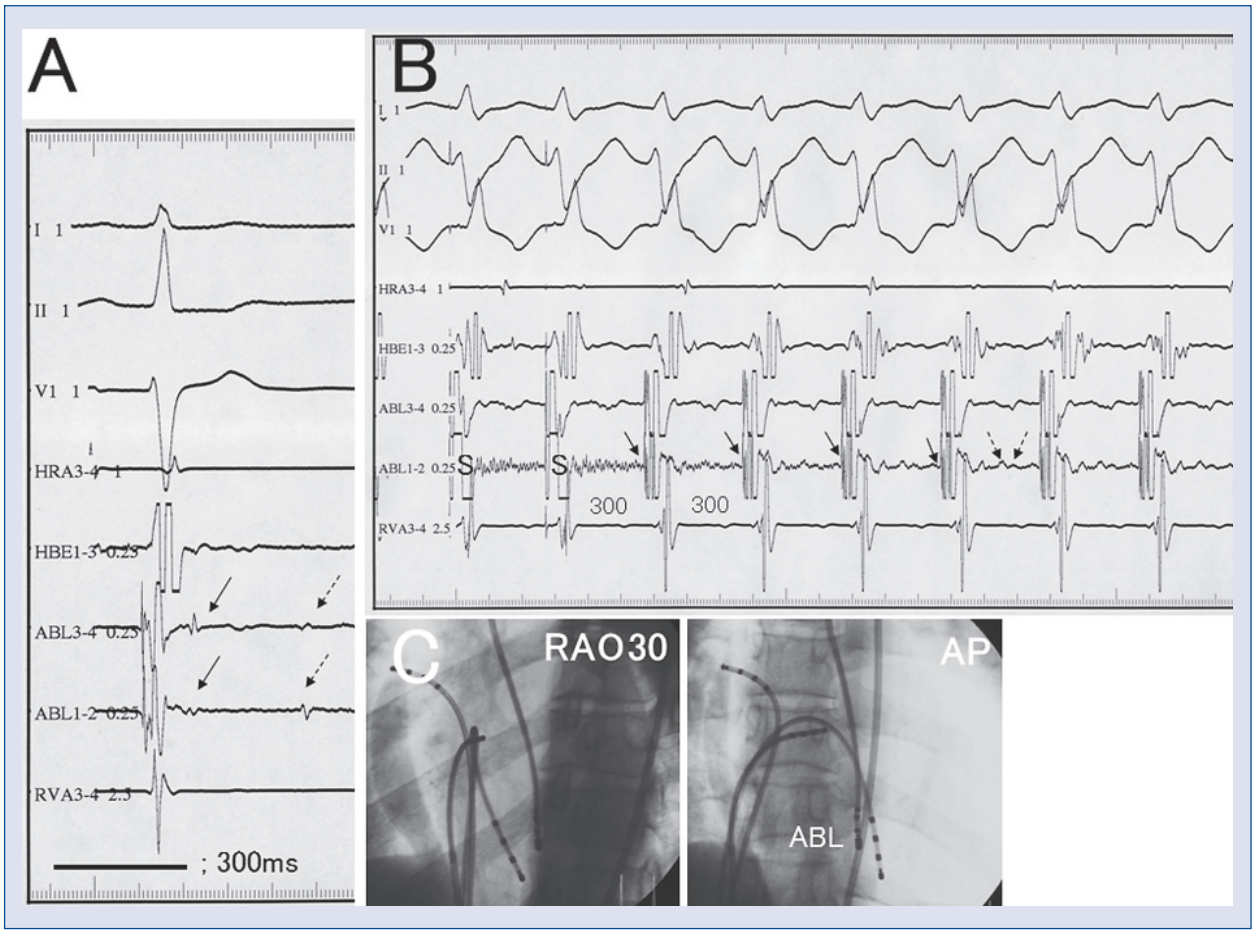

Figure 3. Intracardiac electrograms at the site of successful ablation during sinus rhythm (SR) (**A**) and entrainment pacing (**B**). Catheter positions during pace-mapping of ventricular tachycardia (VT) with the ablation catheter (ABL) at the site of successful ablation, in the right anterior oblique (RAO) (**C**) and antero-posterior (AP) (**D**) fluoroscopic views; **A.** A spiky Purkinje potentials preceded the ventricular electrograms at the site of successful ablation during SR. Note that SP (solid arrows) and DP (dashed arrows) were both recorded at that site; **B.** Entrainment pacing at a cycle length of 290 ms shows constant fusion. At the site of successful ablation (ABL1–2) during VT, a Purkinje potential preceded the onset of the QRS complex by –13 ms (solid arrows) and low-amplitude, low-frequency SP or DP (dashed arrows) were recorded. The 300-ms post-pacing interval is equal to the tachycardia cycle length. These findings suggest that the entrainment site (ABL1–2) is located near the exit of a critical segment of the reentrant circuit, which entrainment pacing captured simultaneously with the surrounding ventricular myocardium, thus exhibiting constant fusion; **C, D.** The ablation catheter (ABL) at the site of successful ablation was in the inferior mid septum. ABL1–2 and 3–4 — distal and proximal ablation catheter recordings. Other abbreviations are as in Figure 1.

gested that the pacing site was located on a critical segment of the reentry circuit (Fig. 3B). VT terminated approximately two seconds after a single delivery of radiofrequency at that site, and was no longer inducible thereafter. These combined observations confirmed the diagnosis of left ventricular verapamil-sensitive VT.

Discussion

The main observations made in the analysis of this case of verapamil-sensitive VT were: 1) SP and DP were detected during SR along a basal-to-apical line on the LIVS surface; 2) both SP and DP were also recorded during VT; 3) during SR, SP and DP were both recorded at the site of successful ablation, whereas during VT, DP was recorded away from the site of earliest ventricular activation.

The characteristics of SP and DP, including a) their anatomical location, b) morphology and activation sequence, c) the decremental conduction properties and effective refractory period of SP, and d) the incremental conduction properties of DP, were similar to those found in subjects with struc-

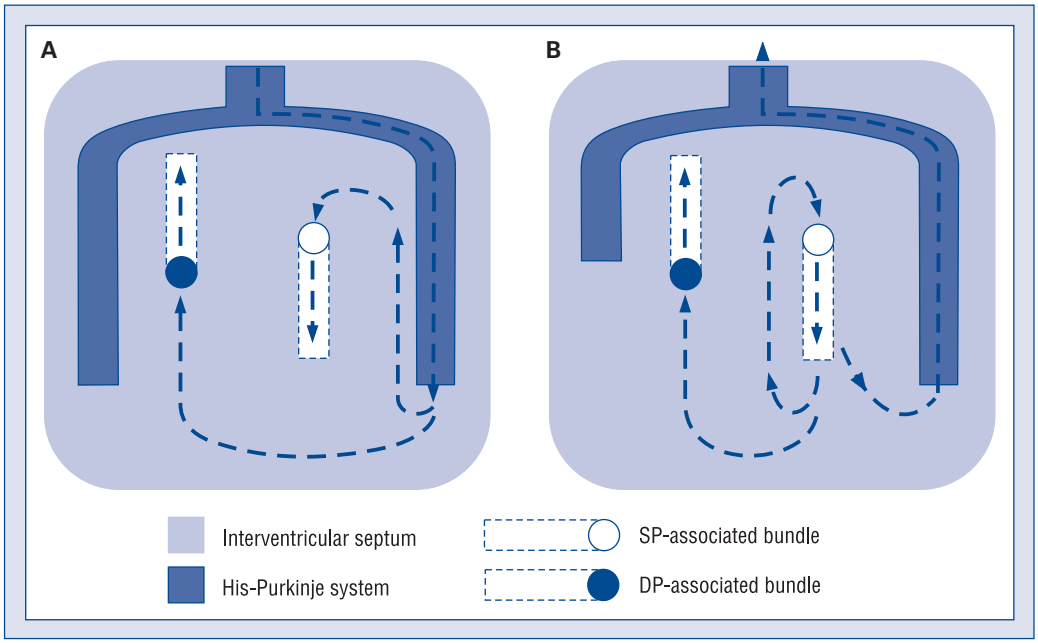

Figure 4. Hypothetical representation of activation of systolic (SP) and diastolic (DP) potentials, His-Purkinje system and interventricular septum, during sinus rhythm (**A**) and ventricular tachycardia (**B**). See text for detailed explanation.

turally normal hearts and no ventricular arrhythmias, as described in our previous report [11]. We believe that these potentials are not specifically found in patients with verapamil-sensitive VT.

We hypothesized that the substrates responsible for SP and DP are separate thin bundles, connected to the Purkinje network in a single direction [11]. Figure 4 is a schematic representation of the impulse propagation during SR and during VT. During SR, SP is activated from base-to-apex and DP from apex-to--base. Both bundles are penetrated by wavefronts propagating through the interventricular septal myocardium. SP and DP were both recorded at the site of successful ablation during SR, whereas during VT, DP was recorded away from that site (Fig. 2B).

This observation suggests that SP was a critical component of the reentrant circuit, whereas DP was a bystander pathway passively activated outside the main reentry circuit. During VT, the SP--associated bundle was penetrated in a base-to-apex direction, activating the whole heart through Purkinje fibers connected to the SP-associated bundle and His-Purkinje system. The interval between ventricular activation and DP was shorter during VT than during SR, probably because of incremental conduction properties of the DP.

Pre-systolic potentials preceding the Purkinje or bundle branch potentials have been described in patients with verapamil-sensitive idiopathic left VT, which may reflect a critical segment of the reentrant circuit on the basis of high success rates of catheter ablation or from observations made from entrainment studies [4–10]. The basal or mid septal recording sites, morphology and activation sequence of the potentials, and slow conduction properties described, are characteristics strikingly similar to those associated with our SP or DP. Although the SP or DP are frequently found not only in patients with verapamil-sensitive VT but also in normal subjects as described earlier, further studies are needed to determine whether the SP or DP during SR may be indicative of successful ablation site of verapamil-sensitive VT.

Conflict of interest: none declared

Conclusions

In a patient with verapamil-sensitive VT, SP and DP were detected on the LIVS surface during SR and during VT. This seemed to represent a critical component of the reentrant circuit, and a bystander pathway, respectively.

References

1. Belhassen B, Rotmensch HH, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. Br Heart J, 1981; 46: 679–682.

2. Lin FC, Finley CD, Rahimtoola SH, Wu D. Idiopathic paroxysmal ventricular tachycardia with a QRS pattern of right bundle branch block and left axis deviation: A unique clinical entity with specific properties. Am J Cardiol, 1983; 52: 95–100.
3. Ohe T, Shimomura K, Aihara N et al. Idiopathic sustained left ventricular tachycardia: Clinical and electrophysiologic characteristics. Circulation, 1988; 77: 560–568.
4. Ouyang F, Cappato R, Ernst S et al. Electroanatomic substrate of idiopathic left ventricular tachycardia: Unidirectional block and macroreentry within the Purkinje network. Circulation, 2002; 105: 462–469.
5. Nogami A, Naito S, Tada H et al. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. J Am Coll Cardiol, 2000; 36: 811–823.
6. Aiba T, Suyama K, Matsuo K et al. Mid-diastolic potential is related to the reentrant circuit in a patient with verapamil-sensitive idiopathic left ventricular tachycardia. J Cardiovasc Electrophysiol, 1998; 9: 1004–1007.
7. Tsuchiya T, Okumura K, Honda T et al. Significance of late diastolic potential preceding Purkinje potential in verapamil-sensitive idiopathic left ventricular tachycardia. Circulation, 1999; 99: 2408–2413.
8. Tsuchiya T, Okumura K, Honda T, Iwasa A, Ashikaga K. Effects of verapamil and lidocaine on two components of the re-entry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. J Am Coll Cardiol, 2001; 37: 1415–1421.
9. Aiba T, Suyama K, Aihara N et al. The role of Purkinje and pre-Purkinje potentials in the reentrant circuit of verapamil-sensitive idiopathic LV tachycardia. Pacing Clin Electrophysiol, 2001; 24: 333–344.
10. Maruyama M, Tadera T, Miyamoto S, Ino T. Demonstration of the reentrant circuit of verapamil-sensitive idiopathic left ventricular tachycardia: Direct evidence for macroreentry as the underlying mechanism. J Cardiovasc Electrophysiol, 2001; 12: 968–972.
11. Kaneko Y, Taniguchi Y, Nakajima T et al. Myocardial bundles with slow conduction properties are present on the left interventricular septal surface of normal human hearts. J Cardiovasc Electrophysiol, 2004; 15: 1010–1018.