Pleomorphic ventricular tachycardia originating from Purkinje fiber network of left anterior fascicle

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Short title: Pleomorphic VT from Purkinje Fiber Network

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Abstract

A 55-year old woman with recurrent syncope and palpitation experienced polymorphic ventricular tachycardia (VT) and more than three monomorphic VTs with a right bundle branch block configuration, either inferior-, middle-, and superior-axis. During the pleomorphic VT, the diastolic potential (dp) was recorded at the antero-lateral left ventricle. Changes in the QRS morphology were associated with the time between dp and onset of QRS complex (dp-V interval), and prolongation of dp-V interval terminated the VT. In addition, the delayed potentials were seen during sinus rhythm around this area. Delivery of radiofrequency current targeting the delayed potentials abolished all the VTs. Different exits from relatively large area of slow conduction in the left anterior fascicle might have produced the pleomorphic VTs.

Key Words: pleomorphic ventricular tachycardia, polymorphic ventricular tachycardia, diastolic potential, delayed potential, left anterior fascicle, Purkinje fiber network, syncope, radiofrequency ablation

Introduction

Idiopathic ventricular tachycardia (VT) with a right bundle branch block (RBBB) and left-axis deviation originating from the area of left posterior fascicle has been well-known[1-5]. In rare instances, idiopathic left anterior fascicular VT has also been demonstrated[6]. Both VTs are verapamil-sensitive, and ablation of the diastolic potential representing the Purkinje potential has been effective in eliminating the VTs[3-8]. In the present case report, a patient with recurrent syncope and palpitation demonstrated pleomorphic VTs[9] originating from the Purkinje fiber network of left anterior fascicle, in which the critical diastolic potentials and delayed potentials were identified during the VTs and sinus rhythm.

Case Report

A 55-year-old woman who had experienced syncope while walking in December 2007 was referred to our hospital in February 2008. She had experienced another episode of syncope in March 2006, and recurrent palpitation attacks in January, April, and May 2007, when monomorphic VT with her blood pressure of 78 / 50 mmHg was documented (Fig. 1A, left). During sinus rhythm with a heart rate of 56 / min, the 12-lead electrocardiogram (ECG) showed no apparent abnormal findings including QT interval (0.42 sec) (not shown). Although mild mitral regurgitation due to mitral valve prolapse of anterior leaflet was found by echocardiography, left ventricular ejection fraction was 71% and the patient had no signs of heart failure. Exercise stress test provoked pleomorphic VTs with a RBBB configuration, either inferior-, middle-, and superior-axis (Figs. 1A and 1B). A 24-hour Holter ECG demonstrated non-sustained polymorphic VT (Fig. 1C).

After obtaining written informed consent, the electrophysiological study was performed with no antiarrhythmic agents in the fasting state. Four quadripolar catheters and a 20-polar catheter were positioned in the right atrium, His bundle area, right ventricular apex (RVA), coronary sinus, and the antero-lateral left ventricle (LV), respectively (Fig. 2A, left). Intraveous isoproterenol administration (0.5 μg / minute) provoked a non-sustained pleomorphic VT (Figs. 2A, middle and 2B). During the pleomorphic VT, the diastolic potentials (dp) were recorded at the distal poles (Map 2, Map 3) of the 20-polar catheter covering the region between the basal left bundle branch (LB) and antero-lateral LV, and the LB potentials at the proximal poles (Map 7, Map 8) (Fig. 2B). The QRS morphology of the VT was RBBB pattern with superior-axis initially, became pleomorphic with changing its QRS axis, and termination of the VT was observed in association with the prolongation of the time between dp and onset of QRS complex (dp-V interval) (Figs. 2A, middle, right and 2B). The time between the onset of QRS complex and LB (V-LB interval) were relatively stable, ranging from 26 ms to 38 ms (Fig. 2B). These results suggest that the dp may reflect the antegrade slow conduction in the abnormal Purkinje fiber of peripheral left anterior fascicle and that the LB could be activated retrogradely through the intact Purkinje fiber.

In the presence of isoproterenol, programmed ventricular stimulation from the RVA induced the pleomorphic VTs reproducibly. Therefore, the 20-polar catheter was changed to a 4 mm-tip ablation catheter, for LV mapping and ablation. During sinus rhythm, the delayed potential immediately after QRS complex was detected in the region of antero-lateral LV, presumably representing the abnormal Purkinje potential of peripheral left anterior fascicle (Fig. 3C). Induction of VT converted the delayed potential

to the dp, and the morphology of QRS complex was altered in accordance with the change in the dp-V interval (Figs. 3A and 3B). In contrast, the dp-V interval was constant during the monomorphic VT (the last four QRS complexes in Figs. 3A and 3B). That is, when the dp-V interval was constant, stable monomorphic VT was maintained (Fig. 4). Incremental burst pacing at a cycle length of 250 ms from the site where the delayed potential was recorded reproduced the pleomorphic VTs (not shown). Because the placement of the ablation catheter at this area prevented the induction of sustained VT at this moment, the radiofrequency catheter ablation (RFCA) targeting the delayed potential was performed, during which transient repetitive ventricular response was observed (not shown). After this first RF delivery, the potential was further delayed (Fig. 3C, Post). Additional application of RF energy (total 7 times) eliminated these delayed potentials around this site, which extended to the circular area of about 1.5 cm diameter, and all the VTs were no longer inducible. The duration and configuration of QRS complex during sinus rhythm were not affected by these RFCA. The patient was discharged with no medications and the exercise stress test at 1, 6, and 12 months after discharge induced no sustained VTs. She has been free of syncope and palpitations for 19 months.

Discussion

The left posterior fascicular Purkinje fiber-related reentrant VT is one of the most popular idiopathic VTs[1-5]. This VT is verapamil-sensitive and its QRS morphology is a RBBB configuration with left axis deviation. During the idiopathic verapamil-sensitive VT, catheter ablation at the site, where the diastolic potential (dp) preceding LB potential around the left posterior fascicle was recorded, was reported to be effective for

eliminating this VT[3-5, 7, 8]. Thus, the dp has been proposed to be consisted of the slow conducting Purkinje fiber potential that could be essential for this reentrant VT[3-5, 7, 8]. It was also reported by Nogami et al. that verapamil-sensitive left anterior fascicular VT with a RBBB configuration and right-axis deviation can be abolished by RF ablation of the zone of slow conduction where the Purkinje potential was recorded in the diastolic phase[6]. In the present case report, the dp(s) during the VTs resembled that of the verapamil-sensitive VT, and were recorded around the Purkinje fiber network of peripheral left anterior fascicle. In addition, (1) termination of the VTs was preceded by prolongation of the dp-V interval, (2) alteration of the QRS morphology was associated with change in the dp-V interval, (3) stable monomorphic VT was maintained only when the dp-V interval was constant (the last four QRS complexes in Figs. 3A and 3B, and Fig. 4), and (4) the dp during the VTs was related to the delayed potential during the sinus rhythm (see below) whose elimination abolished all the VTs. Therefore, the dp(s) and the delayed potentials may be consisted of the slow conducting abnormal Purkinje potentials of the peripheral left anterior fascicle which would be essential substrate for the VTs in this report.

Experimental findings revealed that the core drifting of the spiral wave exhibited QRS complexes with an undulating axis (i.e., polymorphic VT) and that the core became stationary by anchoring to a small discontinuity (anatomical obstacles), resulting in monomorphic activation[10, 11]. It was reported that such anatomical obstacles included epicardial vessels, ridges of endocardial trabeculae and papillary muscle insertions[12]. This patient had at least three monomorphic VTs (Figs. 1A and 4), pleomorphic VTs (Figs 1B, 2A and 3A) and polymorphic VT (Fig. 1C) (Definitions are based on the European Heart

Rhythm Association / the Heart Rhythm Socirty Expert Consensus Document[9]). Although initial RF delivery at the site of the delayed potential slowed the onset of the potential, non-sustained VTs were still inducible. For complete elimination of all the VTs, additional RF energy applications were required around the circular area of about 1.5 cm diameter in which the delayed potentials were present. We propose that the mechanisms of pleomorphism and/or polymorphism may be due to the alteration of exit site after the antegrade slow conduction over degenerated Purkinje fiber network, which covers substantial amount of the area (Fig. 5).

Ouyang et al. has reported that the earliest diastolic potential during the sinus rhythm detected around the left posterior fascicle can be substrate for idiopathic left ventricular tachycardia with a RBBB configuration and left-axis deviation[5]. They called that potential as retro PP, because these abnormal potentials may represent a retrograde Purkinje activation with slow conduction over a Purkinje-Purkinje or Purkinje-ventricular-Purkinje connection attributable to unidirectional block[5]. In our patient, the delayed potentials in the early diastolic phase of sinus rhythm were identified in the peripheral left anterior fascicular region. In agreement with the study by Ouyang et al. [5], the delayed potentials converted to the dp(s) during the VTs (Figures 3B and 3C) and ablation targeting the delayed potentials eliminated all the VTs. Moreover, it was recently reported that the delayed Purkinje potential during sinus rhythm was target for successful catheter ablation of verapamil-sensitive left anterior fascicular VT associated with a healed myocardial infarction[13]. These[5, 13] and the present reports could support our postulate depicted in Fig. 5.

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Figure legends

Fig. 1. Pleomorphic and polymorphic ventricular tachycardias (VTs). **A,** Twelve-lead ECGs of VTs recorded in May 2007 (VT1) and induced by exercise stress test (VT2 and VT3). **B,** One example of the VT during exercise stress test. **C,** Holter ECG recording of polymorphic VT.

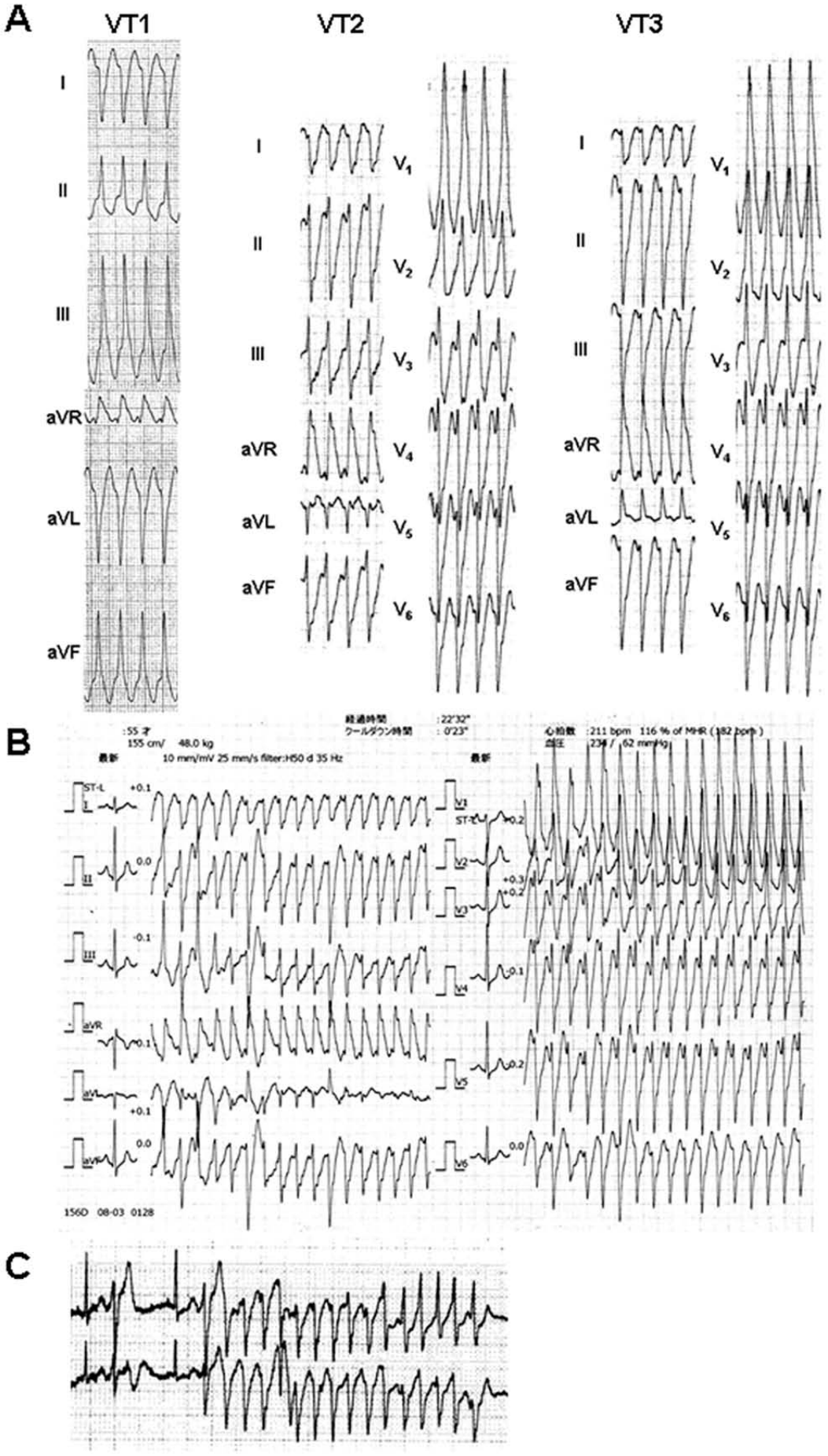
Fig. 2. Mappings of pleomorphic VT in presence of isoproterenol. **A,** Right anterior oblique (RAO) and left anterior oblique (LAO) radiographic imagings are given (**left)**. Twelve-lead ECG of the pleomorphic VT is shown (**middle**). The relationship between the preceding dp-V interval and its QRS axis is depicted (**right**). The direction and length of each arrow indicates the QRS axis and the preceding dp-V interval (relative value), respectively (**right**). **B,** Intracardiac mappings of the pleomorphic VT. Shown are the surface ECGs, leads I, II, and V1, and intracardiac electrograms recorded from high right atrium (HRA d and HRA p), His-bundle region (His d and His p), coronary sinus (CS d and CS p), right ventricular apex (RVA d and RVA p), mapping catheter (Map 1 - 10) which was located the antero-lateral LV. dp = diastolic potential during VT, LB = left bundle branch potential. dp-V = time between dp and onset of QRS complex.

Fig. 3. Delayed potential and diastolic potential. **A,** Surface ECG during the VT. The dp-V intervals are given before the QRS complex of the surface ECG tracings. **B,** Intracardiac electrograms during the VT. Note that the preceding dp of the last four ventricular activations exhibited the double potentials (denoted as ***dp*** in the italic form), indicating a local conduction block or a further delayed conduction within the zone of the slow conduction. **C,** Cardiac tracings before (**left**) and after (**right**) RF current delivery

are given. At the ablation site (i.e., the peripheral Purkinje fiber of left anterior fascicle), the delayed potential (indicated by arrow) was recorded, and it became further delayed after the initial RF current delivery. ABL d and ABL p = distal and proximal pairs of electrodes of ablation catheter. The other abbreviations are the same as in Fig. 2.

Fig. 4. Diastolic potential during monomorphic VT. **A,** Surface ECG during the monomorphic VT. **B,** Intracardiac electrograms during the monomorphic VT. The dp-V interval was stable (105 ms). The abbreviations are the same as in Fig. 3.

Fig. 5. The proposed mechanism of the pleomorphic and polymorphic VTs. The delayed potentials during sinus rhythm could consist of the retrograde conduction of the degenerated Purkinje fibers. On the other hand, the diastolic potentials (dp) during the VT may represent the antegrade slow conduction over the abnormal Purkinje fiber network, which covers substantial amount of the area. The proposed mechanisms of pleomorphism and/or polymorphism may be due to the alteration of exit site after the antegrade slow conduction over the diseased Purkinje fiber network.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10 mm/mV 25 mm/s filter:H50 d 35 Hz
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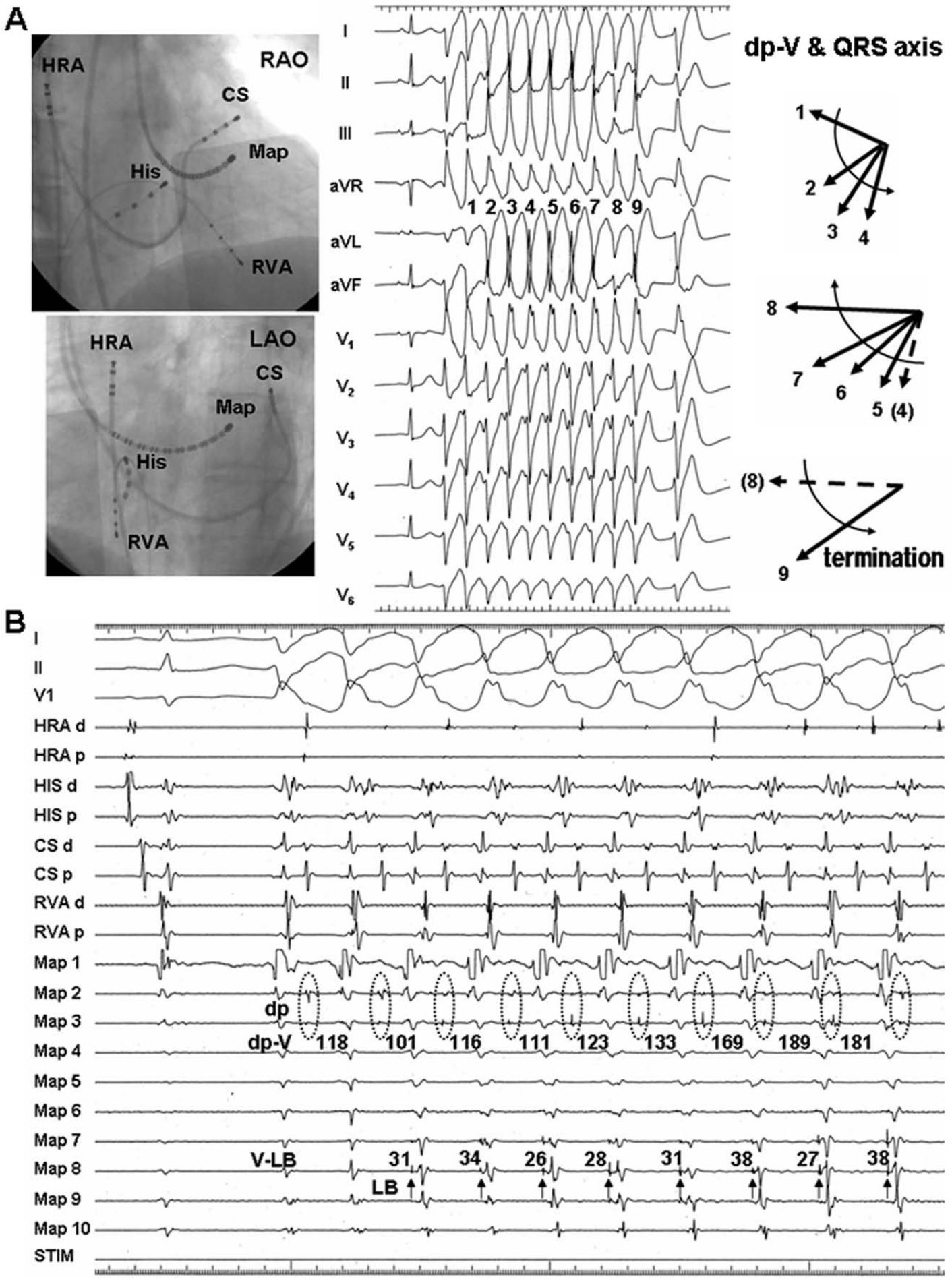
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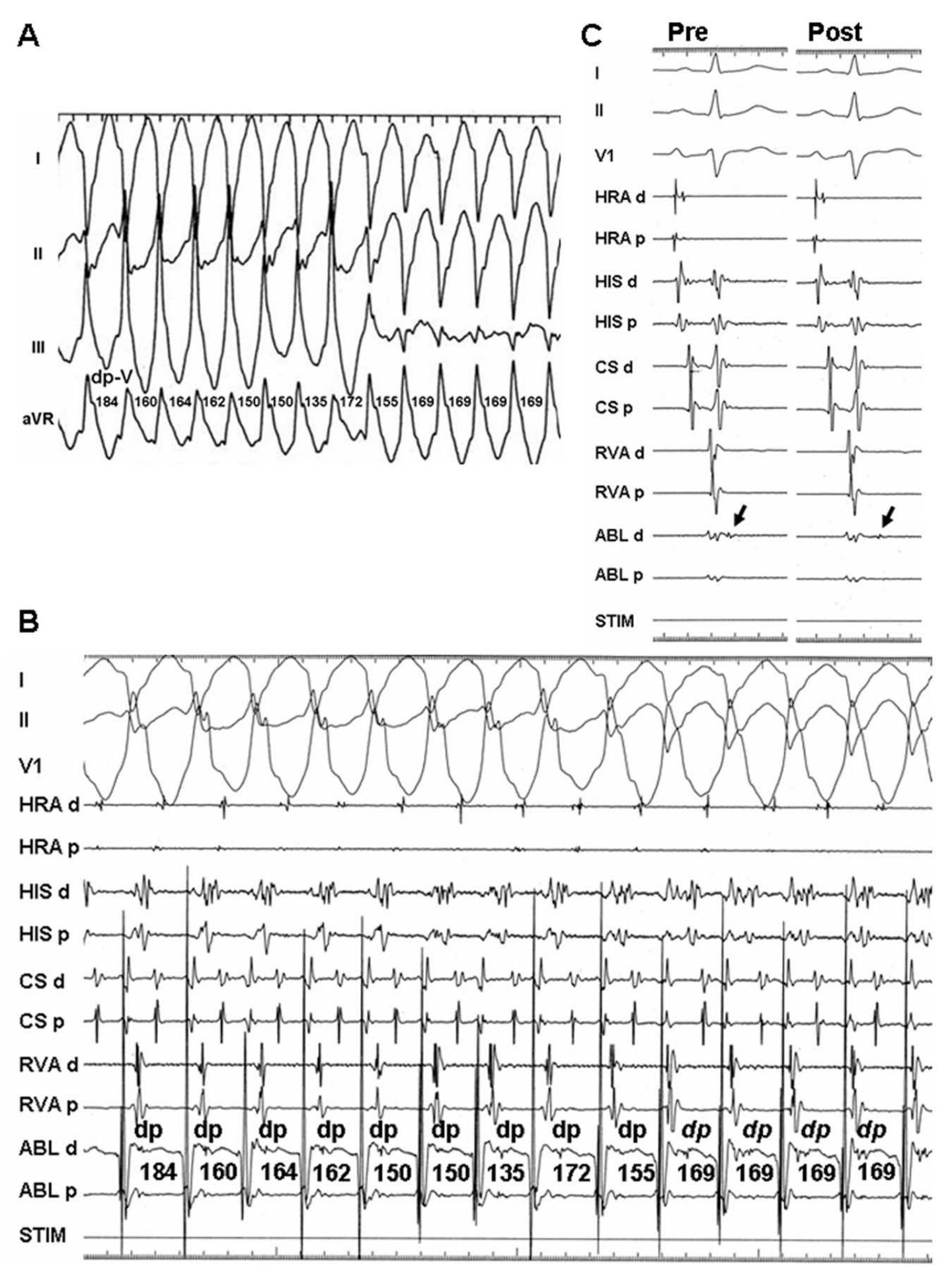
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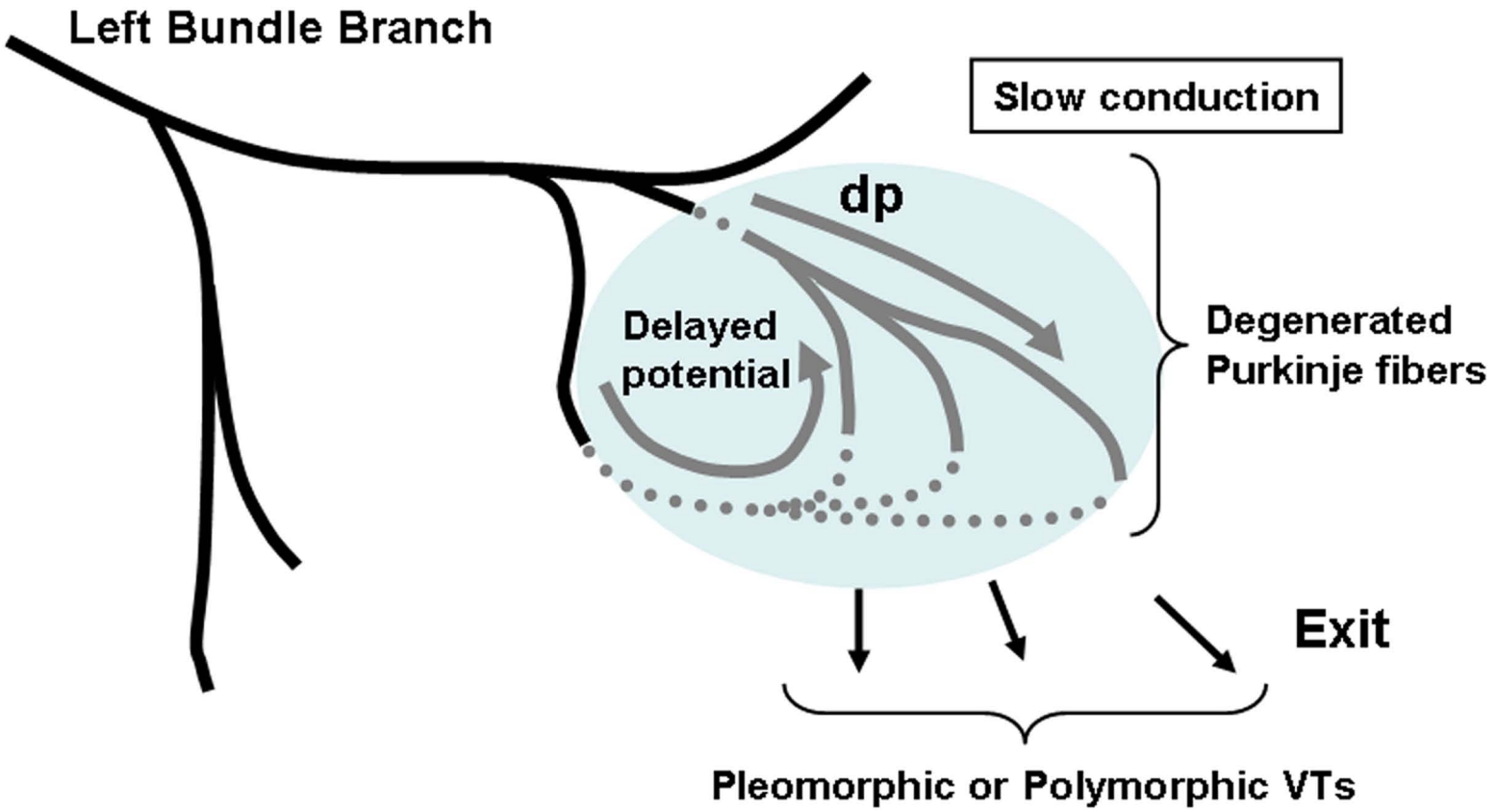
Left Bundle Branch
Slow conduction
dp
Delayed potential
Degenerated Purkinje fibers
Exit
Pleomorphic or Polymorphic VTs