Reentrant Ventricular Tachycardia Originating in the Right Ventricular Outflow Tract — Slow Conduction Identified by Right Coronary Artery Ostium Pacing —

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A case of reentrant ventricular tachycardia (VT) originating from the right ventricular outflow tract (RVOT) is described. An electrophysiological study revealed that programmed stimulation from the right ventricle apex induced 2 types of VT with similar left bundle branch block configuration and inferior axis. Yet, VT cycle length (CL) was different; one was stable, sustained VT with a CL of 360 ms and the other was hemodynamically intolerable VT with a CL of 330 ms. Similarly for both VTs, perfect pace mapping was obtained at the anterior septum beneath the pulmonary valve in the RVOT, and exits of both VTs were very close. Entrainment mapping during stable VT was performed and the anterior septum RVOT was designated as the exit for the stable VT. Intriguingly, entrainment pacing from the ostium of the right coronary artery showed that the post-pacing interval was identical to VTCL. The stimulus to QRS interval was very long (340 ms) during entrainment with concealed fusion, and the right coronary artery ostium was therefore consistent with the VT reentry circuit inner loop or the upper portion of the VT reentry circuit exit. These findings suggest that the stable VT reentry circuit had a slow conduction zone from the ostium of the right coronary artery to the exit in the anterior septum RVOT. When radiofrequency catheter ablation was performed at the 2 exits of the anterior septum RVOT, both VTs then could not be induced. (Circ J 2008; 72: 855–860)

Key Words: Reentry; Right ventricular outflow tract; Ventricular tachycardia

In general, ventricular tachycardia (VT) originating from the right ventricular outflow tract (RVOT) is not associated with organic heart disease. The origin of RVOT tachycardia is almost in the anterior septum of the right ventricle.1,2 The mechanism is mainly due to triggered activity and it is rare for there to be a reentry. A case of 2 types of reentrant VT originating from RVOT induced by electrophysiological study (EPS) in a patient with mitral regurgitation, atrial fibrillation with complete atrioventricular block and pacemaker implantation is reported. The exit of the VT reentry circuit and slow conduction zone were examined using an entrainment mapping technique and a pace mapping technique.

Case Report

A 72-year-old woman with a history of mitral regurgitation, hypertension, atrial fibrillation, complete atrioventricular block and pacemaker implantation was admitted to hospital for treatment of symptomatic VT, which was recorded by Holter electrocardiogram (ECG) monitoring, and complaining of edema in the legs and dyspnea. An ECG recorded during atrial fibrillation showed a right ventricular (RV) pacing rhythm of 80 beats/min, and a chest X-ray revealed cardiac enlargement (59.9%) with slight lung congestion. Echocardiography revealed the following: decreased left ventricular (LV) wall motion, especially in the anterior septum; LV diastole/systole of 50/43 mm; ejection fraction of 36%; aorta/left atrial size of 29/55 mm; ventricular septum/posterior wall of 12/11 mm; and mitral and tricuspid regurgitation levels of IV and II, respectively (27.5 mmHg).

Coronary angiography (CAG) revealed no significant stenosis. Symptoms were improved with diuretics and PDE III inhibitors after admission. A monitoring ECG recorded VT that lasted for 1 min, and this was considered to be the contributing cause of palpitation.

EPS and Radiofrequency Catheter Ablation: First Session

After written informed consent was obtained from the patient and her family to undertake all procedures associated with this study, a standard EPS, endocardial catheter mapping and radiofrequency catheter ablation were performed. Electrode catheters were inserted percutaneously into the femoral vein and positioned in the RV apex, outflow and His bundle position. Ventricular mapping and radiofrequen-
Fig 1. Hemodynamically stable ventricular tachycardia (VT) morphology and perfect pace mapping. (a) VT was left bundle branch block (LBBB) configuration and inferior axis (VT cycle length; VTCL = 360 ms). (b) Perfect pace mapping of VT obtained by pacing from the anterior septum right ventricular outflow tract (RVOT). Pacing cycle length (PCL) was 400 ms. S, stimulation; I-V6, body surface standard lead.

Fig 2. Hemodynamically intolerable VT morphology and perfect pace mapping. (a) VT was LBBB configuration and inferior axis (VTCL = 330 ms). (b) Perfect pace mapping of VT obtained by pacing from the right aortic sinus of Valsalva (PCL = 400 ms). The stimulus to QRS (S–QRS) interval was prolonged to 130 ms. Abbreviations see in Fig 1.
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Catheter ablation were performed with a 7-Fr steerable catheter (EP Technologies). This catheter had a 4-mm distal tip electrode and 2 mm between the distal 2 electrodes. Hemodynamically stable VT was repeatedly induced by double extrastimuli from the RV apex, with left bundle branch block (LBBB) configuration and inferior axis, and the R–R interval was 360 ms (Fig 1a).

In addition, double extrastimuli from the RVOT induced other non-sustained VT with a different frequency; with LBBB and inferior axis, and was hemodynamically intolerable with a R–R interval of 330 ms (Fig 2a).

As these 2 types of VT were comparatively similar in a 12-leads ECG, it was assumed that the RVOT was a reentry circuit exit. Pace mapping was performed to localize the exit, as pace mapping from the anterior septum RVOT allowed perfect pace mapping (12/12) of the stable VT (Fig 1b). The delivery of radiofrequency energy to the site of perfect pace mapping was performed during pacemaker rhythm. But the output power was insufficient despite the temperature reaching 60°C. The same stable VT recurred by programmed stimulation. After VT was terminated by RV pacing, an alternative transaortic approach was taken and pace mapping was performed from the LV outflow tract to the right coronary cusp. Thereafter, the QRS configuration during pace mapping at the right coronary cusp resembled that of hemodynamically intolerable VT and the stimulus
to QRS interval (S–QRS) was 130 ms, which is consistent with slow conduction (Fig 2b). The mapping catheter was then maintained at the site, and VT was initiated by programmed stimulation from the RV apex.

The induced VT assumed the same form as stable VT, and a fractionated electrogram was recorded within the ablation catheter in the right aortic sinus of Valsalva (Fig 3).

The post-pacing interval (PPI) was matched to the VT cycle length (CL) (410 ms) with concealed fusion, and the S–QRS interval was 340 ms, which is greater than 70% of...
Entrainment mapping at this site revealed entrainment with concealed fusion, and the PPI, which was matched to the VTCL, is consistent with a reentry circuit site (Fig 3). The interval from the stimulus to the onset of the following QRS complex during entrainment was 340 ms, which is greater than 70% of the tachycardia CL and, therefore, is consistent with the inner loop or the upper portion of the slow conduction zone within the stable VT reentry circuit.3 A CAG revealed that this site was at the right coronary artery ostium and, hence, radiofrequency energy could not be applied (Fig 4).

During stable VT, entrainment mapping from the anterior septum RVOT was re-performed. Entrainment pacing from this site revealed entrainment with concealed fusion with a very short S–QRS interval (20 ms), and was considered to be the stable VT reentry circuit exit (Figs 5, 7A). Delivery of 40 W of radiofrequency energy to the site resulted in ceasing VT. Subsequently, the stable VT could not be re-induced by RV programmed stimulation.

**Second Session**

The second EPS session was performed 40 days after administering amiodarone (200 mg/day) to estimate the effect of the drug against the remaining hemodynamically intolerable VT. Extra stimuli from RV could not induce the stable VT at all, and the induced VT (CL, 385 ms) had the same the VTCL.

Fig 7. Successful ablation sites. (a) Successful ablation site at the hemodynamically stable VT exit was located in the anterior septum RVOT by entrainment mapping (see Fig 5). (b) Successful ablation site at the hemodynamically intolerable VT exit was located in the anterior septum RVOT by pace mapping about 1 cm above the exit of the stable VT reentry circuit. Abbreviations see in Figs 1, 4.

**Fig 7. Successful ablation sites.** (a) Successful ablation site at the hemodynamically stable VT exit was located in the anterior septum RVOT by entrainment mapping (see Fig 5). (b) Successful ablation site at the hemodynamically intolerable VT exit was located in the anterior septum RVOT by pace mapping about 1 cm above the exit of the stable VT reentry circuit. Abbreviations see in Figs 1, 4.

Second Session

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QRS configuration as hemodynamically intolerable VT that was recorded in the first EPS session (Fig 6). Since the VT during this time was hemodynamically intolerable, pace mapping and ablation during sinus rhythm were performed rather than entrainment mapping during VT. The perfect pace mapping site for this VT (Fig 6B) was located in the anterior septum RVOT about 1 cm above the exit of the stable VT reentry circuit (Fig 7B); however, VT could not be induced after ablation at this site.

**Discussion**

The 2 types of VTs had different VTCL, but had similar LBBB configurations and inferior axes. They were supposed to originate from the RVOT. In general, the onset mechanism of RVOT–VT is an abnormal automaticity, including for triggered activity, and reentrant VT is extremely rare! The mechanism of reentrant VT in the present study was proven by the following findings: 2 different VTs could be induced and terminated by programmed stimulation; one stable VT QRS configuration and VTCL changed spontaneously to another VT; and the entrainment phenomenon was observed by constant pacing from RV during the sustained VT. Pace mapping was performed to identify the VT exit and matched both of the VTs at a RVOT site that was very similar. As for hemodynamically intolerable VT, a slow conduction was shown from pace mapping at the right coronary artery ostium (Fig 2), therefore, it is suggested that the critical slow conduction zone was from the right coronary artery ostium to the RVOT exit site. Pacing from the aortic sinus cusp involving myocardium has been reported but there is no report for pacing of the right coronary artery ostium. Although entrainment mapping could not be performed for hemodynamically intolerable VT, it was shown that the reentry circuit exit of stable VT was located in the RVOT, and that a slow conduction zone was present, extending from the right coronary artery ostium to the anterior septum RVOT. Such findings are extremely rare and, to our knowledge, this report is the first case of VT that has a slow conduction from the right coronary artery ostium. Should a radiofrequency current be delivered to a slow conduction zone without confirming the surrounding anatomical features, the right coronary artery might be occluded. A CAG should be performed to prevent serious complications whenever ablation is performed in the aortic sinus of Valsalva. During stable VT, entrainment mapping identified the reentry circuit exit at the anterior septum RVOT. Stable VT was ceased by catheter ablation at this site. As for hemodynamically intolerable VT, entrainment mapping could not be performed during tachycardia, and details of the reentry circuit could not be analyzed.

In the second EPS session, pace mapping at the anterior septum RVOT, the stimulus to QRS was short (20 ms) and the paced QRS configuration was similar to that of hemodynamically intolerable VT. Hemodynamically intolerable VT could not be induced after ablation at this exit site.

Accordingly, we presumed that the 2 types of VT exits were different but they might have a common slow conduction zone from the right coronary artery ostium to the anterior septum RVOT (Fig 8).

Ischemic heart disease was ruled out as the basis of the VT because coronary stenosis was not shown on CAG. The patient had a history of mitral regurgitation, hypertension, atrial fibrillation, complete atrioventricular block and pacemaker implantation. Echocardiography revealed left atrial and ventricle dilatation and mitral regurgitation level IV. In this patient, VT was LBBB QRS morphology and the mechanism of VT was considered by EPS to be reentry. At first we suspected arrhythmogenic RV cardiomyopathy (ARVC) in the background.

ARVC is a heart muscle disease characterized by the replacement of RV myocardium by fibrofatty tissue and LBBB VT? However, it was finally thought that the patient could not be diagnosed with ARVC because of the following findings: (1) Echocardiography showed the moderator band, but did not show RV chamber dilatation and a reduction in RV ejection fraction; (2) computed tomography also presented no fatty infiltration in the intraventricular septum and right ventricle dilatation; and (3) Pacemaker RV apex pacing threshold (0.6 V; 0.4 ms) had not changed at all since the pacemaker was implanted 8 years ago. Furthermore, regarding the clinical course, the patient's initial cardiac symptom was complete heart block and atrial fibrillation. The appearance of VT was diagnosed at the age of 72 years, 6 years after undergoing a pacemaker implantation. In the case of ARVC, it is reported that an initial cardiac symptom is usually VT and such symptoms appear in middle age.

It is assumed that various factors (eg, valve insufficiency atrial fibrillation and complete heart block) were involved in the development of myocardial abnormalities associated with the reentry circuit originating in an abnormal potential in the RVOT of this patient. Entrainment mapping between the ventricular outflow and aortic sinus cusp is important for elucidating VT reentry circuit and performing an ablation.

**References**


