Critical Slow Conduction Zone Located between the Tricuspid Annulus and a Myocardial Infarct Scar in a Patient with Multiple Ventricular Tachycardia Late after Myocardial Infarction

Emi Nakano MD, Tomoo Harada MD, Kiyoshi Nakazawa MD, Keizou Osada MD, Akihiko Takagi MD, Toshio Sasaki MD, Kihei Yoneyama MD, Hiroyaumigaakikomiyo MD, Osamu Tanaka MD, Kouichi Mizuno MD, Nobuyuki Azuma MD, Ryouji Kishi MD, Fumihiko Miyake MD

Department of Cardiology, St. Marianna University School of Medicine

A patient underwent an electrophysiological study (EPS) and catheter ablation (CA) for ventricular tachycardia (VT) that developed 11 years after myocardial infarction. Entrainment mapping (EM) of the left ventricle did not identify the origin of the VT during induction. Electro-anatomical mapping revealed a low voltage area from the anterior to the inferior left ventricular wall. Another EPS was performed for right ventricular mapping. A basket catheter was placed in the right ventricle to evaluate impulse propagation and we attempted to identify the VT circuit with EM during the same VT (complete left bundle branch block configuration, inferior axis) as the previous EPS. Eight different VT appeared, three of which were sustained during CA. The reentry circuit exit was identified on the tricuspid annulus (TA) at the 9:00 site and radiofrequency (RF) ablation was performed, but the axis transformed from the inferior to the superior and another VT appeared. Despite RF ablation to the VT circuit exit at the 5:00 site, another sustained VT appeared. Finally, it was terminated by ablation to a critical slow conduction zone (SCZ) located near the 7:00 site one year later. Electro-anatomical voltage mapping detected a critical isthmus between TA and right ventricle infarct scar.

Here, we describe a rare SCZ proximal to the TA in a patient with VT late after myocardial infarction.

(J Arrhythmia 2007; 23: 157–163)

Key words: Myocardial infarction, Ventricular tachycardia, Tricuspid annulus

Introduction

Ventricular tachycardia (VT) that develops late after myocardial infarction is generally caused by a reentry circuit around scar tissue and mostly originates in the left ventricle (LV). Identifying a critical slow conduction zone by entrainment mapping is important for terminating VT when hemodynamics...
are stable. However, frequent radiofrequency catheter ablations (CA) are usually required because of complicated circuits.\(^1\)\(^2\)

Furthermore, the appearance of various VT forms often causes difficulties with identifying and terminating the circuit.

The patient underwent an electrophysiological study (EPS) and CA of VT late after myocardial infarction. Eight different VTs were induced using the right ventricle extra stimulus method. The configuration of all VTs was that of a complete left bundle branch block (CLBBB), and we considered that they originated from the right ventricle.

We investigated mainly 3 of the sustained VTs to identify a reentry circuit in the right ventricle using entrainment mapping and a basket catheter placed near the tricuspid annulus. Only a few authors have described belated VT circuits that developed after myocardial infarction in the right ventricle.\(^3\)\(^–\)\(^5\)

Here, a detailed examination identified the critical slow conduction zone, and ablation completely terminated VT that originated near the tricuspid annulus.

**Case Report**

A 63-year-old man presented with palpitation. He had survived a myocardial infarction at the age of 52 (1993) (#1, complete occlusion; #6, 75%; and #11, 99% stenosis. The latter was treated by conventional balloon angioplasty in 1993 and percutaneous coronary intervention (PCI) in 2002).

In June 2002, VT appeared that mexiletine administration could not completely control. He developed VT with CLBBB configuration and a heart rate of 220 bpm again in April 2004, and DC shock was performed at 100 J. Although CA was performed based upon an electro-anatomical mapping (CARTO) evaluation of LV, it was ineffective. The patient was initially administered with 400 mg/day of amiodarone, followed by 200 mg/day and remained under close observation.

However, VT appeared again in July 2004 and he was admitted to our hospital for EPS and CA.

Upon admission, his blood pressure was 114/59 mmHg, he was 162 cm tall and weighed 62 Kg. Hematological analysis showed T-CHO, 177 mg/dl; TG, 198 mg/dl; LDL, 112 mg/dl; HDL, 40 mg/dl and BNP, 681.8 pg/ml. A chest X-ray revealed cardiac enlargement (CTR: 62%) and slight lung congestion.

Resting ECG upon admission revealed ventricular conduction delay according to the following findings: sinus rhythm of 59 bpm; rSr type in V1 lead; transition zone in V4; abnormal Q waves in II, III and aVF leads; and expanded QRS duration of 180 msec (Figure 1). Echocardiography revealed reduced LV ejection fraction (EF) (28%), diffuse hypokinesis of the LV wall motion especially from the inferior wall to the tricuspid valve, LV and left atrial dilation.

**ECG findings of ventricular conduction delay revealed sinus rhythm, 59 beats/min; rSr type in V1 lead; transition zone in V4; abnormal Q waves in II, III and aVF leads and QRS duration of 180 msec.**

---

**Figure 1** Twelve-lead rest ECG during sinus rhythm.

ECG findings of ventricular conduction delay revealed sinus rhythm, 59 beats/min; rSr type in V1 lead; transition zone in V4; abnormal Q waves in II, III and aVF leads and QRS duration of 180 msec.
On the 27th of April 2004, the patient and his family provided written informed consent to undertake all procedures associated with this study; EPS, coronary angiography and left ventriculography proceeded while the patient was on 200 mg/day of amiodarone.

Apical right ventricle programmed extrastimulation was performed to induce VT using 5-Fr 6 polar electrode catheters (Daig Co., Minnetonka, MN, USA) inserted through the right femoral vein. Electrode catheters were also inserted into the right ventricular outflow and high right atrium, and into the bundle of His.

An EP Lab (Quinton Instrument Co., Bothell, WA, USA) recorded 12-lead surface ECG and intracardiac electrograms at 100 to 200 mm/sec. The extra stimulation from the right ventricular outflow tract induced sustained VT, but entrainment mapping was not obtained in the LV. In addition, the VT origin in the LV was not identified because the ventricular cycle length (VTCL) and the post-pacing interval (PPI) did not match during extra stimulation.

Since VT recurred on July 2004, EPS and CA were repeated. During the second EPS session, we suspected that the VT originated in the right

![Figure 2 Eight VT forms during 2nd EPS session.](image)

We applied RF ablation to sustained VT3, VT7 and VT8.
ventricle, and placed a 64-pole basket catheter (EPT Co., 60 mm, 55° sheath) into the right ventricle immediately under the tricuspid valve in addition to the catheters applied in the first EPS session. We then evaluated the pathway of VT conduction and performed entrainment mapping.

Under fluoroscopic guidance, an ablation catheter with 8-mm tip (Ablaze; Japan Lifeline Co., Ltd.) was introduced into the right ventricle via the right femoral vein.

We then identified the VT reentry circuit by entrainment mapping and ablated that region.

We observed 8 VT forms with a CLBBB configuration and a superior or inferior axis after spontaneous premature ventricular contraction or during right ventricle extra stimulation (Figure 2). The spontaneous VT had a CLBBB configuration with an inferior axis. The waveform in the precordial leads remained essentially the same, but the positive component of the II, III and aVf leads gradually changed to low voltage, shifted to VT3 (VT3CL, 658 msec) and persisted.

Figure 3A shows the sustained VT3 activation mapping obtained from the 64-pole basket catheter. When a marker was set to QRS onset, B-spline 3–4, which indicated a fractionated electrogram, proceeded at 100 msec, and C-spline 1–2 matched the QRS onset. Accordingly, we considered that the C-spline 1–2 electrogram was located around the VT circuit exit, and that the VT3 critical slow conduction zone was located around B-spline 3–4 forward from the exit.

The impulse was propagated from the lateral wall to the septal side and came off at the C-spline 1–2 exit. Entrainment mapping around the C-spline revealed the entrainment of matched PPI and VTCL. Accordingly, the VT circuit exit was determined at the 9:00 site on the tricuspid annulus (Figure 3B).

![Figure 3](image-url)

**Figure 3**

(A) VT3 intracardiac electrograms measured using 64-pole basket catheter. Onset of QRS (↓) matched C-spline 1–2 (+), and site were considered the reentry circuit exit. II, VI and V5, body surface ECG. A–G, paired electrodes of 64-pole basket catheters. HBE, His bundle electrogram.

(B) Entrainment mapping with RF ablation from C-spline 1–2. Concealed entrainment revealed that post-pacing interval (PPI) matched ventricular cycle length (658 msec) and was identified as a circuit exit. S, stimulation; ABL1–2, ABL2–3, paired electrodes of ablation catheters.

(C) Left anterior oblique (LAO) projection at 45° angle. Basket catheter (64-pole) placed outside RV immediately under tricuspid valve. EM revealed VT3 circuit exit at 9:00 site on TA. VT3 was terminated by catheter ablation from 9:00 to outside of apical site.
Figure 3C shows the left anterior oblique (LAO) view at the VT3 exit obtained by angiography. The VT axis was transferred from the inferior to the superior, and the VT form changed over time after radiofrequency ablation of the VT3 exit. Only CLBBB VT7 with a superior axis (VT7CL, 660 msec) persisted. The VT7 intracardiac electrograms measured with the basket catheter revealed that H-spline 3–4 matched the QRS onset, indicating that the VT7 circuit exit was located at the 5:00 site on the tricuspid annulus. We therefore performed entrainment mapping (Figure 4A). The VT7 reentry circuit was stretched from the tricuspid valve to the apex (ABL #56) and a slow conduction zone was located near the tricuspid valve (Figure 4B; VT7; ABL #44). Although radiofrequency catheter ablation terminated the VT7, another VT form, VT8 (VT8CL, 663 msec), appeared and persisted. The electrical potential of VT8 measured using a basket catheter located the QRS onset on A-spline 7–8, and entrainment mapping revealed the exit at the 6:00 site on the tricuspid annulus (ABL #40) (Figure 4B). A critical slow conduction zone was located near the 7:00 site on the tricuspid annulus (ABL #58) and VT8 was terminated 7 sec after radiofrequency catheter ablation. The relationship between the

![Diagram of VT7 and VT8 reentry circuit exits and slow conduction zones.](image)

(A) EM performed near 5:00 site on TA revealed VT7 reentry circuit exit (ABL #56) and slow conduction zone (central: ABL #44).

(B) VT8 reentry circuit exit and slow conduction zone. EM revealed VT8 reentry circuit exit at 6:00 site on TA (ABL #40) and slow conduction zone at 7:00 site (central: ABL #58). VT8 was terminated 7 seconds after RF ablation to slow conduction zone.

(C) Basket catheter (64-pole) and schemas of tachycardia circuit and ablation site (LAO at 45°). Reentry circuit exit (E) of VT3. VT7 and VT8 located at 9:00 to 5:00 site on TA.
ablation site and each VT circuit in the basket catheter were shown in Figure 4C.

Neither right ventricular extra stimulation nor burst pacing induced VT thereafter. On September 2005, EPS did not induce VT and we drew a voltage map in the right ventricle using CARTO. The voltage amplitude of the complete scar, colored gray was $\geq 0.1$ mV, the dense scar in red was $\geq 0.1$ mV and $\leq 1.5$ mV, and that of the normal area in purple was $\geq 1.5$ mV.$^{6,7}$

Figure 5 shows the distribution of island-shaped scar tissue from the upper to the middle part of the right ventricle wall. We assumed that an isthmus was located between the scar tissue and the tricuspid valve at the 6:00 to 9:00 site, so we delivered radiofrequency energy to a location close to where the EPS session had been conducted one year previously. None of the VTs were induced after CA. We therefore presumed that the VT reentry circuit encircled the isthmus and scar tissue.

**Discussion**

Most chronic ventricular tachycardia that arises after myocardial infarction originate in the LV, and some reports have described that reentry around scar tissue causes VT.$^{1,2}$

From the first EPS session we speculated that the VT originated from the LV, and we performed LV endomyocardial and voltage mapping. However, catheter ablation could not identify the VT reentry circuit. We considered at the second EPS session that the VT originated in the right ventricle and we tried to identify a tachycardia circuit in right ventricle maps using a multielectrode basket catheter and entrainment mapping.

During EPS, eight different VT forms with a CLBBB configuration developed, of which three that persisted (VT3, VT7 and VT8) were ablated. The diastolic potential of each of these VTs was marked on basket spline electrodes.

During EPS, eight different VT with the CLBBB configuration appeared. We tried to ablate the three among them that were sustained (VT3, VT7 and VT8). The diastolic potential of each preceding QRS of these VTs was marked on basket spline electrodes. A diastolic potential often arises in a conduction zone of reentrant VT.

Fractionated electrograms with low amplitude and long duration are obtained from sites where normal cardiac muscles are scattered in fibrous or fatty tissue, and they indicate delayed conduction due to

---

**Figure 5** Electroanatomical voltage map.
Red indicates areas of lowest voltage and orange, yellow, green, blue, navy blue and purple indicate progressively increased voltage. Normal voltage area (>1.5 mV) is shown in purple. Island-shaped scar tissue (gray, voltage < 0.1 mV) extends from upper to middle part of right ventricular wall.
poor bonding among tissues. We performed entrainment mapping with CA based on the diastolic potential index to identify the reentry circuit exit and slow-conduction zone.

Impulse conduction varied with radiofrequency application to the exit of the VT3 reentry circuit and VT3 changed into VT7. Radiofrequency ablation of the central pathway and exit of the reentry circuit of VT7 changed this into VT8, which in turn was stopped by radiofrequency ablation at the central pathway and exit of the VT8 reentry circuit. Thereafter, VTs could not be induced.

We supposed that a critical slow conduction zone and the reentry circuit exit of VT3, VT7 and VT8 were located near the tricuspid valve based on the entrainment mapping findings (Figure 4C).

Reports describing VT ablation late after myocardial infarction that indicate right ventricle origin are rare, and ablation with a basket catheter and entrainment mapping has not been achieved until now. Furthermore, a reentry circuit exit located near the tricuspid annulus has not been considered.

A follow-up EPS study 6 months later confirmed that VT could not be induced and an infarct in the right ventricle was evaluated by simultaneous voltage mapping using CARTO. We identified a broad slow-conduction zone.

The isthmus appears between the mitral valve and post-infarction scars during posterior myocardial infarction. The mitral annulus is thought to become the anatomical obstacle, and impulses seem to easily traverse the narrow pathway between the mitral annulus and post-infarction area in normal tissue. However, whether an isthmus locates between the tricuspid annulus and a low voltage area. We postulated that this isthmus contributed to the right ventricle VT reentry circuit (Figure 5).

The patient is presently stable without syncope, VT, or Vf with deleterious hemodynamics since CA and remains under close observation as an outpatient under amiodarone administration.

References
6) Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM: Electr...