Fusion with Postpaced Return Cycle Identical to Tachycardia Cycle Length during Transient Entrainment of Ventricular Tachycardia and its Implications

Hitoshi Kitazawa, MD, Takashi Washizuka, MD, Hirohide Uchiyama, MD, Masaomi Chinushi, MD, Shin-ichi Niwano, MD, and Yoshifusa Aizawa, MD

SUMMARY

In reentrant ventricular tachycardia (VT), the postpaced return cycle (RC) during transient entrainment at a pacing site far from the central common pathway is longer than the VT cycle length (VTCL), when VT is represented by a figure-eight model. However, the reentrant circuit has not been fully elucidated.

The purpose of this study was to present VT in which the postpaced RC became identical to VTCL during transient entrainment while fusion is evident in the surface electrocardiogram (ECG).

Among 38 patients with inducible reentrant VTs who underwent electrophysiologic study (EPS), 10 VTs of six patients were selected. All patients had underlying heart diseases: dilated cardiomyopathy (n = 2), coronary artery disease (n = 1), postoperative tetralogy of Fallot (TOF; n = 2), and arrhythmogenic right ventricular dysplasia (n = 1). Catheter mapping was performed to demonstrate that the site of origin was distant from the pacing site.

The cycle length of induced VT (n = 10 VTs) was 380 ± 41 msec. Five patients (83%) had two morphological VTs; one a left bundle branch block (LBBB) and the other a right bundle branch block (RBBB) pattern. During rapid pacing, constant fusion was observed in all VTs, but the postpaced RC was identical to VTCL. In 2 patients (4 VTs), the revolution of wavefronts around an anatomical obstacle (scar of myotomy in TOF, and infarction) was demonstrated.

The fact that the postpaced RC was identical to VTCL but showed fusion in the surface ECG can be explained by macro-reentry. The pacing site must be located at the preferential route of the macroreentrant circuit. (Jpn Heart J 1997; 38: 369–378)

Key words: Ventricular tachycardia, Macroreentry, Transient entrainment, Postpaced return cycle length

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Received for publication November 18, 1996.

Accepted December 9, 1996.

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THE mechanism of sustained ventricular tachycardia (VT) induced by programmed electrical stimulation has been considered to be reentry and as such, VT can be often entrained with rapid ventricular pacing during VT.\(^1-^5\) The reentrant circuit might be represented most often by a figure-eight model,\(^7-^9\) and during transient entrainment or resetting, the postpaced return cycle (RC) must be longer than the VT cycle length (VTCL). The sum of the two conduction times: the conduction time from the pacing site to the entrance of the central common pathway (CCP), and that from the exit of CCP to the pacing site will be longer than the conduction time through the outer loop in the figure-eight model.\(^7-^9\)

Recently, we experienced 6 VTs in which the postpaced RC was identical to VTCL while the fusion complex was confirmed in the surface electrocardiogram (ECG). The postpaced RC at the pacing site would be identical to VTCL if we paced directly from the CCP of the reentry circuit, but this was not the case because fusion was clearly observed during rapid pacing.

In the present study, the incidence and mechanisms in which RC is identical to VTCL with a fusion complex during transient entrainment are discussed.

**PATIENTS AND METHODS**

**Patient selection:** Among 38 consecutive patients who underwent electrophysiologic study (EPS) for symptomatic VTs in the last 3 years, patients who fulfilled the following criteria were selected; 1) VT was induced by programmed ventricular stimulation using 1–3 extrastimuli or rapid pacing,\(^10\) and 2) VT was entrained by rapid ventricular pacing from a site far from the reentry circuit. The characteristics of the patients are presented in Table I.

**Induction of ventricular tachycardia:** After informed consent was obtained, EPS was performed primordially to determine the mechanism of VT and drug efficacy.\(^5,11-13\)

Under the fluoroscopic guidance, 3 quadripolar electrode catheters (USCI Co. Boston, USA, Josephson multipurpose electrode catheter 6F, interelectrode distance 5 mm) were inserted from the right femoral vein and femoral artery, and placed at the RVA, RV outflow tract (RVOT), and left ventricle (LV), and used for stimulation and recording of the intracardiac electrograms. A tripolar electrode catheter (interelectrode distance 10 mm 6F) was also placed at the His bundle area to record the His bundle electrogram.

Standard protocol\(^10-13\) was used for the induction of VT: 1–2 (3 if necessary) ventricular extrastimuli and rapid ventricular pacing up to 210 beats/min were applied. When VT could not be induced from RVA, the same induction protocol was repeated at the RV outflow tract (RVOT) and the left ventricle (LV). If VT
Table I. Characteristics of Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>UHD</th>
<th>VT morphology</th>
<th>VTCL (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>67F</td>
<td>LBBB + LAD</td>
<td>430</td>
</tr>
<tr>
<td></td>
<td>DCM</td>
<td>RBBB + RAD</td>
<td>430</td>
</tr>
<tr>
<td>2.</td>
<td>51F</td>
<td>LBBB + LAD</td>
<td>380</td>
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<td>RBBB + RAD</td>
<td>380</td>
</tr>
<tr>
<td>3.</td>
<td>47F</td>
<td>LBBB + LAD</td>
<td>340</td>
</tr>
<tr>
<td></td>
<td>OMI</td>
<td>RBBB + RAD</td>
<td>340</td>
</tr>
<tr>
<td>4.</td>
<td>16M</td>
<td>LBBB + LAD</td>
<td>400</td>
</tr>
<tr>
<td></td>
<td>TOF</td>
<td>RBBB + NA</td>
<td>380</td>
</tr>
<tr>
<td>5.</td>
<td>27F</td>
<td>LBBB + LAD</td>
<td>350</td>
</tr>
<tr>
<td></td>
<td>TOF</td>
<td>RBBB + NA</td>
<td>350</td>
</tr>
<tr>
<td>6.</td>
<td>17F</td>
<td>LBBB + NA</td>
<td>350</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>mean</th>
<th>RBBB-VT</th>
<th>376 ± 31....</th>
<th>(p &gt; 0.10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBBB-VT</td>
<td>380 ± 22....</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td></td>
<td>376 ± 29....</td>
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</table>

UHD = underlying heart disease; VTCL = VT cycle length; DCM = dilated cardiomyopathy; OMI = old myocardial infarction; TOF = tetralogy of Fallot; ARVD = arrhythmogenic right ventricular dysplasia.

could not be induced by electrical stimuli alone, the induction protocol was performed during isoproterenol infusion.

Stimuli were given at twice the diastolic threshold (Cardiac Stimulator BC-02A, Fukuda Denshi Co., Tokyo, Japan), and recordings were made on an ink jet recorder at a paper speed of 100 mm/sec (Mingograf 82, Siemens Elema Co., Germany). All data were stored on magnetic tape (TEAC Data Recorder XR-5000, Japan) for subsequent retrieval. Twelve-lead ECG was recorded for all induced VT and during rapid pacing of VT.

Mapping and entrainment of VT: Endocardial catheter mapping was performed according to a technique described by Josephson et al.\(^1\)\(^4\) The exit of the reentry circuit was presumed to be the site of the earliest activation. After induction of VT, rapid pacing was performed from RVA at a paced cycle length 10–20 msec shorter than VTCL. Transient entrainment was confirmed by the following criteria\(^1\)\(^–\)\(^6\); (a) demonstration of constant fusion during rapid pacing, (b) demonstration of a progressive fusion during pacing at shorter cycle lengths, (c) demonstration of interruption of VT by a localized conduction block to a site followed by activation of that site from a different direction, (d) advancement of the local electrogram at the exit without change in the morphology.

Measurement of the postpaced return cycle: Electrical stimuli were delivered using the distal and 3rd electrodes, while the 2nd and 4th electrodes were used to record of intracardiac electrograms.

RC was measured at the pacing site as the interval between the last stimulus and the onset of the local electrogram of VT which resumed after cessation of rapid pacing.
Data analysis: The incidence of which RC was identical to VTCL during transient entrainment of VT was determined. Clinical characteristics were also analyzed.

The values are presented as mean ± SD. Statistical analysis was performed using a t-test, and a p value of < 0.05 was considered significant.

RESULTS

Postpaced return cycle of VT: Forty-nine monomorphic sustained VTs were induced in 38 patients, and all VTs were entrained with rapid pacing. Six patients exhibited a postpaced RC which was identical to VTCL. All patients had underlying heart diseases: dilated cardiomyopathy (n = 2), coronary heart disease (n = 1), postoperative tetralogy of Fallot (TOF) (n = 2), and arrhythmogenic right ventricular dysplasia (n = 1) (Table I).

During the study, class-I antiarrhythmic drugs: procainamide (n = 2 VTs), disopyramide (n = 1 VT), cibenzoline (n = 2 VTs), and mexiletine (n = 1 VT) were administrated, and the mean VTCL was 392 ± 34 msec.

Mapping and transient entrainment: The site at which rapid pacing resulted in entrainment without fusion could be confirmed at RVOT (7 VTs) and the posterior free wall of LV (2 VTs) (Table II). The other 2 VTs could not be mapped because of hemodynamic intolerance or spontaneous termination of VT.

In three VTs in two patients (Cases 1 and 3), VT was entrained from other

<table>
<thead>
<tr>
<th>Case</th>
<th>Morphology</th>
<th>VTCL</th>
<th>SCZ</th>
<th>Pacing</th>
<th>RC</th>
<th>Resetting</th>
<th>Drug</th>
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<tbody>
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<td>#17–18 (RV)</td>
<td>RVA</td>
<td>430</td>
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<tr>
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<td>RBBB</td>
<td>430</td>
<td>#6 (LV)</td>
<td>RVA</td>
<td>430</td>
<td>(+)</td>
<td>(−)</td>
</tr>
<tr>
<td>2</td>
<td>LBBB</td>
<td>440</td>
<td>#17 (RV)</td>
<td>RVA</td>
<td>440</td>
<td>(+)</td>
<td>disopyramide</td>
</tr>
<tr>
<td></td>
<td>RBBB</td>
<td>380</td>
<td>spontaneous termination</td>
<td>RVA</td>
<td>340</td>
<td>N.E.</td>
<td>cibenzoline</td>
</tr>
<tr>
<td></td>
<td></td>
<td>340</td>
<td>#6 (LV)</td>
<td>RVO</td>
<td>340</td>
<td>N.E.</td>
<td>(−)</td>
</tr>
<tr>
<td>4</td>
<td>LBBB</td>
<td>420</td>
<td>#17 (RV)</td>
<td>RVA</td>
<td>420</td>
<td>(+)</td>
<td>mexiletine</td>
</tr>
<tr>
<td></td>
<td>RBBB</td>
<td>420</td>
<td>#17 (RV)</td>
<td>RVA</td>
<td>400</td>
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<td>#17 (RV)</td>
<td>RVA</td>
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<td>(+)</td>
<td>(−)</td>
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<td></td>
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<td>#17 (RV)</td>
<td>RVA</td>
<td>350</td>
<td>(+)</td>
<td>(−)</td>
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<td>6</td>
<td>LBBB</td>
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<td>#13 (RV)</td>
<td>RVA</td>
<td>350</td>
<td>N.E.</td>
<td>(−)</td>
</tr>
</tbody>
</table>

380 ± 41

387 ± 20 (p > 0.10)

VTCL = VT cycle length; SCZ = slow conduction zone; Pacing = pacing site during transient entrainment; RC = return cycle; LBBB = left bundle branch block; RBBB = right bundle branch block; RV = right ventricle; LV = left ventricle; RVA = right ventricular apex; RVO = right ventricular outflow tract; # = endocardial mapping site according to Josephson et al.; N.E. = not examined.
Figure 1. Transient entrainment with an identical return cycle to VT cycle length (Case 2). Upper panel: After the administration of disopyramide, the LBBB-VT was induced (VTCL: 440 msec), and entrained by rapid pacing from the RVA. Pacing at 410 msec, constant fusion was observed and the intracardiac electrogram at the RVOT was captured orthodromically (arrow). The postpaced return cycle was 440 msec, which was identical to VTCL. Lower panel: At shorter paced cycle length (380 msec), progressive fusion was observed, but the postpaced return cycle was 440 msec. I, II, V1 = leads I, II, V1 of surface electrocardiograms; RVA = right ventricular apex; RVOT = right ventricular outflow tract.

pacing sites (RVOT and LV) but the postpaced RC was identical to VTCL. In a patient with an inferior myocardial infarction (Case 3), VT was entrained without fusion from the left posterior free wall, but it could also be entrained from both RVOT and RVA in which the fusion complex was evident but showing an identical RC to VTCL (Figure 2). Furthermore, in a patient after repair of TOF (case 4), LBBB-VT which was entrained without fusion by rapid pacing from RVOT could be entrained from RVA with an identical RC to VTCL, however, fusion was evident. Another RBBB-VT was also entrained and the postpaced RC was identical to VTCL (Table II).

**Activation sequence of VT:** Circulating wavefronts were confirmed around a myocardial infarction scar within the left ventricle in Case 3 (Figure 3). The local electrogram at the left posterior wall showed fragmentation, and rapid pacing
Figure 2. Constant fusion with identical return cycle to VT cycle length. A: Rapid pacing from the RVOT. Rapid ventricular pacing at 310 msec entrained the LBBB-VT. The postpaced return cycle was 340 msec, which is identical to VTCL (340 msec). B: Rapid pacing from the RVA. Rapid ventricular pacing at 310 msec entrained the same VT with constant fusion in surface ECG. The postpaced return cycle was 340 msec. I, II, V1 = leads I, II, V1 of surface electrocardiograms; RVA = right ventricular apex; RVOT = right ventricular outflow tract.

from this area resulted in entrainment without fusion.

In a patient with postoperative TOF (Case 4), a counterclockwise rotation of wavefronts was demonstrated in LBBB-VT (Figure 4 left), and a reverse revolution in RBBB-VT. A fragmented electrogram was recorded below the pulmonary valve in two VTs (Figure 4, right).

In the other three patients: dilated cardiomyopathy (Cases 1 and 2), and arrhythmogenic right ventricular dysplasia (Case 6), the full activation sequence could not be determined.
Figure 3. Endocardial mapping for VT in a patient with inferior myocardial infarction. Using sequential left ventricular endocardial mapping, a circulating wavefront around the presumed infarction (shaded area) can be seen. I, II, V1 = leads I, II, V1 of surface electrocardiograms; RVA = right ventricular apex.

Figure 4. Endocardial mapping for VT after the repair of tetralogy of Fallot. Left = LBBB-VT. The revolving wavefront of LBBB-VT was counterclockwise around the scar of myotomy in the right ventricle. Right = RBBB-VT. The revolving wavefront of LBBB-VT was clockwise around the scar of right ventricle. The fractionated electrical activity was recorded below the pulmonary valve during two VTs. I, II, V1 = lead I, II, V1 of surface electrocardiograms.
DISCUSSION

**Demonstration of macroreentry:** In intraoperative mapping, Miller et al.\textsuperscript{15} confirmed the activation patterns of the wavefront and so-called monoregional spread was most often recorded during VT.\textsuperscript{15,18} The other pattern; continuous loop reentry which is considered to be macroreentry, was rarely recorded in their study.\textsuperscript{15} Furthermore, de Bakker et al. demonstrated that the macroreentry was rare with respect to being a mechanism of VT in coronary artery disease.\textsuperscript{18}

The underlying heart diseases showing macroreentrant VT were postoperative TOF,\textsuperscript{19-22} coronary artery disease,\textsuperscript{16,26} and dilated cardiomyopathy.\textsuperscript{23-25} In dilated cardiomyopathy, the bundle branch reentrant VT has sometimes been demonstrated,\textsuperscript{23-25} however, an anatomic obstacle would be unnecessary for the revolving wavefronts in this type of VT. In postoperative TOF and coronary artery disease, the anatomical obstacle of macroreentry has been confirmed to be a scar of myotomy\textsuperscript{19-22} or a scar of myocardial infarction,\textsuperscript{26} respectively. In the present study, circulating wavefronts around an anatomical obstacle were demonstrated in 2 patients; one with a postoperative TOF, and one with an old inferior myocardial infarction.

**Electrophysiologic characteristics of macroreentry:** When VT is entrained with rapid pacing outside of CCP, the surface ECG will show a fusion complex, and in this instance the postpaced RC will be longer than VTCL.\textsuperscript{3-7} However, in the present six cases, the surface ECG showed constant fusion, while the postpaced RC was identical to VTCL. In the figure-eight model, the postpaced

![Microreentry vs. Macroreentry diagram](image_url)

**Figure 5.** Mechanism in which the postpaced return cycle is identical to VT cycle length. Left: Rapid pacing (asterisk) was delivered at the right ventricular apex (RVA), and the circuit is presumed to be located in the RV outflow tract. The postpaced return cycle would include the conduction time between the reentry and pacing site, so that the return cycle should be longer than the VT cycle length. Right: Pacing site is the RVA, but the macroreentrant circuit was presented. The distance between the pacing site and reentrant pathway is negligible, and the return cycle would be identical to the VT cycle length.
RC would be identical to VTCL when rapid pacing was undertaken within CCP and this is known as entrainment without fusion.\textsuperscript{5,6} In our cases, fusion was evident while the postpaced RC was identical to VTCL. If we had paced the outer loop of the reentrant pathway as shown in Figure 5, this finding could be explained.

In VT with postoperative TOF, the two different directions of the circulating wavefronts around an anatomical obstacle\textsuperscript{19-22} were responsible for the RBBB and LBBB-VT. Recently, similar macroreentrant VT was also demonstrated in an inferior myocardial infarction,\textsuperscript{26} and the VT-morphology (RBBB or LBBB) of macroreentrant VT was shown to be dependent on the direction of the circulating wavefront around the infarction scar as an anatomical obstacle.\textsuperscript{25} Wang et al.\textsuperscript{25} reported VT in which the direction of the wavefront was reversed when the morphology of the VT changed in bundle branch reentry.

**CONCLUSION**

During transient entrainment of VT, the postpaced RC was identical to VTCL when fusion was evident in the surface ECG. The pacing site was not within the central common pathway of the reentrant circuit, and this finding could be explained as entrainment by rapid pacing at the preferential route of macroreentrant VT.

**REFERENCES**


