Abrupt Loss of Constant Fusion During Entrainment of Ventricular Tachycardia at a Critical Paced Cycle Length

Masayuki Yamaura, MD, Yoshifusa Aizawa, MD, Masaomi Chinushi, MD, Takashi Washizuka, MD, Hirohide Uchiyama, MD, and Hitoshi Kitazawa, MD

SUMMARY

Sustained monomorphic ventricular tachycardia (VT) can be frequently entrained and interrupted with rapid pacing and the mechanism of the pacing-induced interruption is considered to be due to orthodromic block.

This study focused on the incidence of VT which was interrupted at a critical cycle length and was characterized by an abrupt loss of constant fusion in the surface electrocardiogram (ECG), and the role of orthodromic block as the cause of such characteristic change and interruption of VT was analyzed.

Among 45 consecutive patients with symptomatic VT, rapid pacing was performed in 43 VTs of 39 patients. The exit was mapped as the earliest site of the activation during VT and an electrode catheter was located at the site. Rapid pacing was performed at progressively shorter cycle lengths in steps of 10 msec until VT was interrupted and the timing of the orthodromic and direct capture was compared at the exit.

Abrupt loss of constant fusion was observed in 25 of 39 patients (64.1%): and the loss was invariably associated with interruption of VT. When the timings of the activation of the exit were compared, which were measured from the preceding \( (n-1) \) stimulus as the time reference, the direct capture was relatively delayed compared to that of the orthodromic capture. This finding suggests that orthodromic block is the cause of the direct capture as well as the pacing-induced interruption of VT.

In the remaining 13 patients (35.9%), the surface ECG showed a gradual transition into the fully paced QRS morphology. The direct capture was confirmed in the non-fused beats, but it was not necessarily associated with interruption of VT. The interval from the stimulus to the entrained electrogram at the exit showed a gradual prolongation until the exit was finally captured directly from the pacing site.

The confirmation of constant fusion followed by abrupt loss in ECG can be a reliable hallmark of orthodromic block as the cause of the interruption of VT during transient entrainment at a critical paced cycle length. (Jpn Heart J 2001; 42: 67-78)
Key words: Sustained ventricular tachycardia, Transient entrainment, Loss of constant fusion, Orthodromic block

If sustained monomorphic ventricular tachycardia (VT) can be entrained with rapid pacing, the mechanism of such VT has been considered to be reentry with an excitable gap.1-8)

When VT is entrained and rapid pacing repeated at progressively shorter cycle lengths, it is often interrupted at a critical cycle length.1-3,7-10 Several mechanisms have been shown or considered for the pacing-induced interruption of VT: orthodromic block within the area of slow conduction,11-14 the production of echo-waves,13,14 a disruption of the functional block by rapid pacing,12 and the capture of the exit and the subsequent antidromic invasion of the wave front into the area of slow conduction. The differentiation of these mechanisms during electrophysiologic study is limited, however, in certain circumstances, we might be able to address this problem.

As an extension of our earlier studies,7-9 we focused on the special circumstance in which VT was interrupted at a critical paced cycle length, while at longer cycle lengths VT was entrained. The interruption of VT was further characterized by an abrupt loss of constant fusion in the surface electrocardiogram during rapid pacing at that critical cycle length.

In this paper, we determined the incidence of VT in which the pacing-induced interruption was associated with an abrupt loss of constant fusion. Then, we measured the timing of the activation of the exit in the fused and non-fused beats. A relatively delayed activation was observed for the direct capture compared to the orthodromic capture when constant fusion was lost during rapid pacing at the critical cycle length. The mechanisms are also discussed.

Patients and Methods

Patients: During the past three years, 45 patients with symptomatic monomorphic sustained VT underwent electrophysiologic study. The mean age (± SD) was 49 ± 18 years and the male to female ratio was 34:11. The underlying heart diseases are shown in Table I.

Electrophysiologic study: After written, informed consent was obtained, an electrophysiologic study was performed in the postabsorptive and non-sedated state. Antiarrhythmic agents were discontinued for two to three days before the study. No patient had been treated with amiodarone. Standard protocol was employed for the induction of VT as reported in pre-
Previous studies,\(^7\,9\) The earliest site of activation of VT was determined by endocardial mapping\(^7\,15\) and was considered to be the exit from the central common pathway of the reentrant circuit (the VT circuit). Pace-mapping was used to facilitate the determination of the exit.

### Rapid pacing and transient entrainment:

Rapid pacing was performed at a cycle length which was 10 - 20 msec shorter than the cycle length of VT, and if VT was entrained, it was repeated after a decrement of the paced cycle length in steps of 10 msec until VT was interrupted.\(^7\,9\) If rapid pacing produced an acceleration of the VT rate, direct-current shock was used for termination.

Rapid pacing was first attempted at the apex and then at the outflow tract of the right ventricle when the first site failed to entrain VT. The first stimulus was given 5 msec after triggering of the local electrogram at the pacing site so that the relation between the first stimulus and the local electrogram of VT was constant in each pacing. Rapid pacing was attempted in the left ventricle for those VTs which were not entrained from the right ventricle. The duration of each rapid pacing was 5-10 seconds.

The following criteria for transient entrainment were used to diagnose whether VT was entrained or not:\(^1\,16\,17\)

1. Demonstration of constant fusion in the electrocardiogram during pacing at a constant rate faster than VT except for the last captured beat which occurs at the pacing rate in the surface electrocardiogram (ECG) or in the local electrogram at the exit with the same non-fused configuration (non-fusion of the last captured beat).
2. Demonstration of a constant but different degree of fusion in the QRS complex during pacing at a different pacing rate (progressive fusion).
3. Interruption of VT with a

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<td>7</td>
</tr>
<tr>
<td>Non-ischemic LV aneurysm</td>
<td>1</td>
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<tr>
<td>Verapamil responsive idiopathic VT</td>
<td>6</td>
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<td>Total</td>
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HD = heart disease; ARVD = arrhythmogenic right ventricular dysplasia.

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### Table I. Clinical Characteristics of the Patients

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HD = heart disease; ARVD = arrhythmogenic right ventricular dysplasia.
localized block to the exit from the VT circuit followed by activation of that site from a different direction with a shorter conduction time by the next paced impulse (localized block). (4) An advancement of the local electrogram at the exit without change in the morphology by stimulus which resulted in a fused complex in the surface electrocardiogram (advancement of the local electrogram at the exit without a change in the morphology).

**Definitions:** VT was diagnosed by confirming a dissociation between the His bundle electrogram and ventricular activity or when a His deflection was found before the QRS complex, an HV interval shorter than that seen during sinus rhythm.18)

Sustained monomorphic VT was defined as that lasting > 30 seconds or requiring immediate termination because of hemodynamic deterioration.

Orthodromic capture of the exit was diagnosed if the local electrogram showed a morphology identical to that seen during VT and direct capture was diagnosed if the local electrogram at the exit showed a morphology which was identical to that seen during pacing of the heart during sinus rhythm.

Abrupt loss of constant fusion in the surface electrocardiogram was defined as follows: (1) the QRS configuration changes from that of constant fusion into a fully paced one without an intermediate morphology, (2) constant fusion is present at least for 5 beats prior to the loss of fusion.

The activation time of the orthodromic capture was measured as X in Figure 1: from the pacing artifact to the orthodromically captured electrogram at the exit, and that of the direct capture was measured as Y: from the stimulus to the electrogram of the non-fused beats. Relative timing was compared between the orthodromic and direct capture in which the activation time of the direct capture was measured from the preceding (n-1) stimulus: Y’ as shown in Figure 1.

**Data analysis:** The incidence of abrupt loss of constant fusion and its relation to the pacing-induced interruption of VT were analyzed. The relative timings of the activation of the exit were then compared between the orthodromic and direct capture: X versus Y’ as shown in Figure 1. Values are presented as mean ± SD and were statistically analyzed by the t-test; paired or unpaired if appropriate. A p value less than 0.05 was considered significant.
RESULTS

Initiation and rapid pacing of ventricular tachycardia: Sustained monomorphic VT was induced in 42 of 45 patients (93.3%): a left bundle branch block-like (LBBB) pattern in 19 VTs and a right bundle branch block-like (RBBB) pattern in 28 VTs. Two VTs with different morphologies were induced in 5 patients. Sustained polymophid VT was induced in 3 other patients (6.7%).

Immediate direct-current shock had to be used for termination of polymorphic VT and for rapid monomorphic VTs in an additional 3 patients. In the remaining 39 patients with 42 VTs, rapid pacing was performed at progressively shorter cycle lengths.

Incidence of abrupt loss of constant fusion: The pacing site was the right ventricular apex in 32 VTs, the outflow tract in 8, and the left ventricle in 2 and VT was interrupted in 35 patients at a critically short paced cycle length. In 25 patients (64.1%): 28 VTs, abrupt loss of constant fusion was observed in the surface ECG during pacing at a critical paced cycle length that interrupted VT (Figure 2). The mean cycle length of the VTs

Figure 1. Schema of measurement of the timings of the orthodromic and direct capture of the exit of the reentry circuit. In this schema, the initial three beats represent those showing constant fusion and at the fourth beat, fusion was lost. The local electrogram at the exit (Exit) shows changes called localized block by Waldo.1) In the beats with constant fusion, the exit is activated with a time interval X and in the non-fused beat, the site is activated directly with a much shorter time interval Y. However, if the activation time of the direct capture was measured from the preceding (n-1) stimulus denoted as Y’ and compared with X, Y’ is longer than X. Orthodromic block will explain the association of interruption of VT with an abrupt loss of constant fusion and the direct capture (Figure 2). If X is progressively prolonged and exceeds Y’ as the paced cycle length is progressively decreased or as the number of pacing stimuli increased at a certain cycle length, the exit might be eventually captured directly from the paced site. However in this instance, VT will not necessarily be interrupted (as shown in Figure 3). S: stimulus.
Figure 2. Abrupt loss of fusion during rapid pacing at a critical cycle length. A: The patient had been operated on for double outlet right ventricle and VT with a cycle length of 300 msec was initiated and the earliest site of activation (the exit) was mapped in the outflow tract of the right ventricle (RVO). Rapid pacing was performed between 290-270 msec. Constant fusion was observed at each pacing (A). At 270 msec, an abrupt loss of constant fusion and localized block was observed which was associated with interruption of VT (B). The change in the local electrogram is shown in C. $Y'$ was 50 msec longer than $X$. The appearance of a single non-fused beat was associated with interruption of VT. I, II, V1: Surface leads. RVA: right ventricular apex.
was 359 ± 59 msec and VT was interrupted at 305 ± 51 msec. VT showed an RBBB pattern in 20 VTs and an LBBB pattern in 8 VTs. At longer paced cycle lengths, constant and progressive fusions were confirmed.

In the other 14 patients (35.9%), these were 8 VTs with an RBBB pattern and 6 VTs with an LBBB pattern. Of these, VT was interrupted in 9 patients (23.1%) but the QRS complex showed a gradual change into a fully paced one (Figure 3). The cycle length of VT was 363 ± 79 msec, and was interrupted at 309 ± 87 msec. In the remaining 5 patients (12.8%), rapid pacing was terminated at 190-200 msec due to hemodynamic deterioration.

**Timing of orthodromic versus direct capture at exit:** When VT-interruption was associated with an abrupt loss of constant fusion, the activation time of the last orthodromic capture, X in Figure 1, was 342 ± 62 msec and the activation time Y was 65 ± 29 msec (Figure 2). When the latter was measured from the preceding (n-1) stimulus: Y' in Figure 1, the exit was activated 378 ± 70 msec after the preceding stimuli and it was longer than that of the orthodromic captor X: 342 ± 62 msec (p < 0.01). The difference ranged from between 10-90 msec (mean = 31 ± 22 msec).

**Figure 3.** Gradual change into the fully paced morphology during rapid pacing. The patient had VT with a cycle length of 390 msec and rapid pacing was performed between 380-240 msec from the right ventricle. At relatively longer paced cycle lengths, constant fusion was observed in the surface ECG (not shown). At 290 msec or shorter, rapid pacing resulted in the direct capture of the exit and the QRS changed into the fully paced morphology soon after rapid pacing. In the figure, the second and third stimuli reset VT and an advancement of the electrogram of the exit is apparent. The initial part of the electrogram is identical to that of VT and the late part to that of direct capture. The local electrogram shows a fusion complex in the two beats and thereafter, the site was activated directly from the pacing site with the activation timing of 115-120 (Y) msec and Y' is then 370 msec. After cessation of rapid pacing, VT resumed. VT was entrained during pacing and the orthodromic conduction can be seen from the last stimulus to the local electrogram at the exit in the first resumed VT: X = 380 msec. This prolongation of X makes direct capture possible which occurs at 370msec after the stimulus. I,II, V1 = surface leads; RV = right ventricle; LV = left ventricle.
In VTs exhibiting a gradual change into the fully paced QRS complexes, the activation time of direct capture Y was 74 ± 23 msec, and Y' was 384 ± 51 msec which was shorter than X: 420 ± 47 msec (p < 0.05).

The difference ranged from 10 to 70 msec: the mean was 23 ± 19 msec.

A representative case is shown in Figure 3. This is because orthodromic activation time X was prolonged significantly from 380 ± 63 to 420 ± 47 msec for the first and last entrained beats at each rapid pacing (p < 0.01).

The timings of activation in the two groups are shown Figure 4.

**Figure 4.** Comparison of timings of activation between VT showing abrupt loss of fusion (Y) and that without (N). Between the two groups, the cycle length of VT was not different. In VT showing an abrupt loss of fusion, the orthodromic activation time X was shorter than the direct activation time when measured from the preceding stimulus as denoted by Y' (see text and Figure 1). Because of this time difference, the exit cannot be captured directly unless the site remained un-captured because of block or delay in orthodromic conduction. On the contrary, in VT without an abrupt loss of fusion, X was prolonged and became significantly longer than Y'. Because of this delay in orthodromic activation, the exit can be captured directly before the arrival of the orthodromic wave fronts from the slow conduction area.

In VTs exhibiting a gradual change into the fully paced QRS complexes, the activation time of direct capture Y was 74 ± 23 msec, and Y' was 384 ± 51 msec which was shorter than X: 420 ± 47 msec (p < 0.05). The difference ranged from 10 to 70 msec: the mean was 23 ± 19 msec. A representative case is shown in Figure 3. This is because orthodromic activation time X was prolonged significantly from 380 ± 63 to 420 ± 47 msec for the first and last entrained beats at each rapid pacing (p < 0.01). The timings of activation in the two groups are shown Figure 4.

**DISCUSSION**

Though the majority of the patients in the present study had VT unrelated to coronary artery disease, we were able to entrain and interrupt VTs with rapid pacing and the mechanism of VT would most likely be reentry having an excitable gap. Different mechanisms have been demonstrated for the pacing-induced interruption of VT by high resolution mapping. These include a diminution of the lines of functional block, and a
sudden block in the central common pathway with or without production of an echo-wave.\textsuperscript{11,12,14} Of these, the most common mechanism for interruption of VT has been shown to be orthodromic block within the area of slow conduction\textsuperscript{13,14} though Waldecker, \textit{et al.}\textsuperscript{12} demonstrated that conduction block may develop in front of the entrance to the central common pathway.\textsuperscript{11,13,14} High resolution mapping has, furthermore, demonstrated that antidromic invasion from the exit occurs only at a length of 8\%, and interruption of VT from direct capture and antidromic invasion into the area of slow conduction seems unlikely as the cause of pacing-induced interruption.\textsuperscript{14} The analysis of the timing of activation of capture at the exit will allow us to assess the role of direct capture and retrograde invasion into the slow conduction area.

Clinical findings derived from a single electrogram recorded at the exit and the surface ECG have provided indirect criteria for orthodromic block.\textsuperscript{1-3,7,19,20} It is of note that in the data of high resolution mapping in animal models,\textsuperscript{12,14} the electrogram recorded distal to the orthodromic block was shown to follow a pattern similar to that recorded at the exit in clinical VT.\textsuperscript{1-3,7,9} The local electrogram at the exit was advanced without a change in the morphology when the surface ECG showed constant fusion and when constant fusion was lost, the exit was captured directly.\textsuperscript{21} The exclusion of direct capture as the cause of interruption would be certain if we could show that the timing of the direct capture is relatively delayed compared to the orthodromic capture: $Y'$ > $X$ in Figure 1.

As schematically shown in Figures 1 and 2, the activation time of the orthodromic capture: $X$, was 342 ± 62 msec and after an abrupt loss of constant fusion, the exit was activated directly with a shorter time $Y$: 65 ± 29 msec.

When the activation time of the direct capture was measured from the preceding ($n$-1) stimulus: $Y'$ in Figure 1, the direct capture is expected to have occurred at 378 ± 70 msec after the preceding stimulus which is relatively delayed compared to the orthodromic capture: $Y'$ > $X$ as shown in Figure 2.

This finding would mean that the direct capture is possible because the exit remains unactivated by the orthodromic wave fronts when the wave fronts from pacing site arrive at the exit. A similar difference in the timing of activation between the orthodromic and direct captures can be seen in the data of high-resolution mapping of Waldecker and Boersma.\textsuperscript{12,14} Not a mere delay but a block must be responsible for the direct capture and interruption since a delay in orthodromic conduction
will not result in interruption of VT after the cessation of rapid pacing. Even a single non-fused complex seen during rapid pacing (evidence of loss of fusion) is associated with interruption of VT (Figure 2).

Direct capture would be facilitated under the following conditions: (1) the proximity of the pacing site to the exit, (2) higher pacing rate, and (3) a decremental conduction within an area of slow conduction. The last mechanism was the case in VT which showed a gradual change into the fully paced QRS complexes with or without interruption.

In 9 patients, the exit was captured directly from the pacing site in the non-fused beats but before the direct capture, the QRS complexes with fusion were associated with an advancement of the electrogram at the exit without a change in the morphology (Figure 3). In this instance, the activation time X (Figure 1) showed a gradual prolongation as the paced cycle length decreased or as the number of stimuli increased at certain cycle lengths, and the orthodromic capture of the exit was replaced by direct capture when X exceeded Y' (Figure 3). The mode of the capture of the exit was observed in 35.9% during entrainment of VT but direct capture was not necessarily associated with an interruption of VT.

Limitations: The present study has several limitations. VTs with various underlying heart disease were treated together. However, the fact that the VTs were initiated with programmed stimulation and could be entrained with rapid pacing is best explained by the reentrant mechanism. The site of the orthodromic block could not be specified in this study for technical reasons, however the area of slow conduction could be actually localized in a limited number of cases. To overcome this problem, high resolution mapping would be required but it has been demonstrated that the whole reentrant circuit is rarely demonstrated in human VT which limits the usefulness of this technique. Finally, it has not been determined if orthodromic block is caused by conduction block or by production of echo-waves within an area of slow conduction. However, in both instances, the block occurs within an area of slow conduction.

Conclusion: VT which showed an abrupt loss of constant fusion at a critical paced cycle length was associated with interruption of VT. The exit was captured directly but the timing of the direct capture was relatively delayed compared to the orthodromic one. This finding suggests that direct capture of the exit and retrograde invasion into the slow conduction area is unlikely as a cause of interruption. The decremental properties of the slow conduction area will facilitate a direct capture but without interruption of VT.
The confirmation of constant fusion in the surface ECG and its abrupt loss can be the hallmark of orthodromic block as the mechanism of pacing-induced interruption of VT.

ACKNOWLEDGMENT

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