Evidence for Slow Conduction Areas during Pacing in Patients with Sinus Rhythm, and their Relation to the Site of VT Origin

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SUMMARY

In 52 patients with reentrant monomorphic sustained ventricular tachycardia (VT), the site of VT origin was determined by endocardial mapping and the interval from stimulus artifact to the onset of QRS complex (St-QRS) was measured during pace-mapping. Eleven patients had remote myocardial infarction (group 1), 25 patients had other underlying heart diseases (group 2), and 16 had idiopathic VT (group 3). A St-QRS interval of 40 msec or longer was defined as abnormal. (1) Long St-QRS interval: Thirteen sites with long St-QRS intervals were detected in 13 (25.0%) of 52 patients: 5 patients in group 1 (45.5%), 7 (28.0%) patients in group 2 and one (6.3%) in group 3. (2) Local electrogram: The local electrogram at sites with a long St-QRS interval was wide and 113±38 msec in duration during sinus rhythm which increased to 159±64 msec during VT (p<0.05). In sinus rhythm, an abnormally prolonged local electrogram was observed in 11 of 13 sites with a long St-QRS interval, and mid-diastolic potential or continuous activity was observed in 3 sites during VT. (3) Relation to VT origin: At sites with a long St-QRS interval, concealed entrainment was observed in 3 patients, and the earliest activated local electrogram during VT in 5 patients. Conclusion: Sites with a long St-QRS interval were observed in 25% of the patients with VT, and their incidence tended to be higher in patients with ischemic heart disease. Such sites were associated with abnormal local electrograms and some of the sites were considered to be the active limb of the reentry circuit. (Jpn Heart J 35: 1-13, 1994)

Key words: Sustained ventricular tachycardia Slow conduction area Pace-mapping Long St-QRS interval

Slow conduction areas have been found in patients with recurrent sustained ventricular tachycardia (VT),1-4 and such areas can participate in forming an essential part of a VT reentry circuit.1-4 Sometimes the site with long interval...
from the stimulus artifact to the onset of QRS complex (St – QRS) might be observed during pace-mapping in patients with VT, and such long intervals would mean delayed conduction via a slow pathway from the pacing site to the entire myocardium.\cite{5,6} Although these abnormal sites might be participating in the formation of the reentrant circuit, demonstration of such a slow pathway alone is not sufficient to prove that the site is essential for the initiation and maintenance of the reentrant VT.\cite{6,7} In this study, we determined those sites with a long St – QRS interval during pace-mapping and assessed their relation to the origin of VT.

**Subjects and Methods**

**Patient selection:** From July 1982 through December 1992, we performed electrophysiologic studies (EPS) on 78 patients who had inducible monomorphic sustained VT. Among them, transient entrainment\cite{1-3} was confirmed in 86 VTs in 52 patients. The most likely mechanism of these VTs was reentry,\cite{1-4} and these were the subjects in this study. As shown in Table I, 40 patients were male and 12 were female. The ages ranged from 12–75 years (mean 46.5±18.0). Thirty-six patients had underlying heart diseases: remote myocardial infarction in 11, arrhythmogenic right ventricular dysplasia in 9,\cite{8,9} idiopathic left ventricular aneurysm, possibly post-myocarditis in 5,\cite{10} post-operative stage of congenital heart disease in 5, idiopathic dilated cardiomyopathy in 4, alcoholic cardiomyopathy in 1, and cardiomyopathy of undetermined cause in 1. In the other 16 patients, underlying heart disease could not be detected by conventional cardiac examinations: these were designated idiopathic VT.\cite{11-13} We divided the 52 patients into 3 groups according to underlying heart diseases: group 1 consisted of 11 patients (23 VTs) with remote myocardial infarction, group 2 included 25 patients with non-ischemic heart disease (42 VTs), and group 3 included 16 patients without underlying heart disease (21 VTs) (Table I). Five patients (5 VTs) with verapamil sensitive VT were included in group 3.

**Electrophysiologic study (EPS):** After informed consent was obtained the electrophysiologic study was performed in the non-sedated and postabsorptive state. Three 6F quadripolar catheters with interelectrode distances of 5 mm were positioned within the heart under fluoroscopic guidance. Stimulations were performed using a pair of first (distal tip) and third electrodes, and the intracardiac electrogram was recorded using second and fourth electrodes. Otherwise, the local electrogram was recorded using a distal pair of electrodes. For the recording of the His-bundle electrogram, a 6F tripolar catheter with an interelectrode distance of 10 mm was used. Intracardiac electrograms were recorded on an ink-jet recorder (Siemens Elema Co. Ltd. Mingograf 7) with three surface leads; I, II, VI
Protocol for VT induction: Programmed electrical stimuli with a rectangular pulse of 2 milliseconds duration were given at twice the late diastolic threshold by a cardiac stimulator (Fukuda Denshi Co, Cardiac Stimulator BC02). Our standard stimulation protocol consisted of programmed electrical stimulations; single to triple extrastimuli were delivered after eight basic stimuli at two different basic cycle lengths (400 and 600 milliseconds), and rapid pacings at cycle lengths between 667 and 286 milliseconds for 5 to 15 seconds. Stimulations were performed first at the apex followed by the outflow tract of the right ventricle when VT was not induced from the first site. If the whole protocol was unable to induce VT, isoproterenol was infused until the sinus rate was increased by 20%, and the protocol was repeated at two sites in the right ventricle and at one site in the left ventricle.

Endocardial mapping: The interval from the stimulus artifact to the onset of QRS activation (St-QRS) was measured during the pace-mapping at a paper speed of 100 mm/second and considered abnormally prolonged when the interval was 40 milliseconds or longer. According to Josephson's methods, we performed pace-mapping at a minimum of 10 sites in one ventricle and repeated it more extensively at the site of VT origin. If VT originated from the interventricular septum, pace-mapping was attempted at each segment of both ventricles. Otherwise the pacing was applied to only the ventricle of VT origin. Rapid pacing was added at a cycle length similar to that of VT (ranging from 500 to 316 milliseconds).

When a site with a long St-QRS interval was detected during pace-mapping, its relation to the site of VT origin was evaluated by electrophysiologic findings during VT. The site of origin was defined according to the criteria
outlined below. The electrograms of such sites were analyzed for duration.

**Determination of the site of VT origin:** Extensive endocardial mappings were performed during VT and the site of VT origin defined as: (1) the earliest ventricular activation site during VT\(^{15-17}\) (the site with the minimum time interval between local activation and onset of QRS complex during VT), (2) the site showing optimal pace-mapping during rapid pacing\(^{14-17}\) (the morphology of the paced QRS complex was identical to that of the VT in at least 11 of 12 leads of the electrocardiogram), (3) evidence of a critical slow pathway\(^{2,7,15-18}\) in the reentry circuit. A critical slow pathway in the reentry circuit was defined as follows\(^7\): Fixed pacing during VT resulted in concealed entrainment\(^{2,7,15-18}\) and the first post-paced return cycle at the pacing site was identical in cycle length to the VT. If a distinct local electrogram prior to the surface QRS complex, usually mid-diastolic, was obtained during VT, the interval between the pacing stimulus and onset of the QRS complex during pacing was identical to the interval between the local electrogram and the onset of the surface QRS complex during VT.

**Definitions:** A fractionated electrogram was defined as one showing a duration of >133 milliseconds with low peak to peak amplitude (<0.5 mV) deflections, and an abnormal local electrogram was defined as one showing a duration of >70 milliseconds with peak to peak amplitude of less than 3.0 mV.\(^{19-21}\) An electrogram spanning the whole cardiac cycle was defined as continuous activity.\(^{22}\) Mid-diastolic potential\(^7\) was defined as a discrete potential separated by an isoelectric line between the QRS complexes of VT. Late potential was defined as discrete activity existing at least 10 milliseconds after inscription of the surface QRS.\(^{23}\)

A sustained VT was defined as a VT lasting more than 30 seconds, or one requiring emergency intervention for termination. A nonsustained VT was defined as a VT lasting more than 5 beats but terminating spontaneously within 30 seconds.

**Statistical analysis:** Statistical analysis was performed by Student’s t test and a multiple comparison test by Ryan. A p value of <0.05 was considered significant. Values are presented as mean±SD.

**Results**

**Site of VT origin:** In group 1, the site of VT origin was determined at the free wall of the right ventricle in 5 VTs, at the right ventricular septum in 2, at the left ventricular septum in 1, and at the free wall of the left ventricle in 15. In group 2, VT originated from the free wall of the right ventricle in 25 instances from the septum of the right ventricle in 5, and from the free wall of the left ventricle in
Table II. Electrophysiologic Findings

<table>
<thead>
<tr>
<th>Case</th>
<th>UHD</th>
<th>St–QRS (ms)</th>
<th>Sinus (ms)</th>
<th>VT (ms)</th>
<th>Relation</th>
<th>Findings</th>
<th>Paced QRS vs VT-QRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>72M</td>
<td>140</td>
<td>170**</td>
<td>210**</td>
<td>MD +</td>
<td>Concealed ent</td>
<td>Different</td>
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<tr>
<td>2</td>
<td>66M</td>
<td>60</td>
<td>80*</td>
<td>80*</td>
<td>+</td>
<td>EAS (~40ms)</td>
<td>Same</td>
</tr>
<tr>
<td>3</td>
<td>67M</td>
<td>50</td>
<td>90*</td>
<td>120*</td>
<td>+</td>
<td>EAS (~50ms)</td>
<td>Different</td>
</tr>
<tr>
<td>4</td>
<td>71M</td>
<td>80</td>
<td>120*</td>
<td>250**</td>
<td>-</td>
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<td>Different</td>
</tr>
<tr>
<td>5</td>
<td>58F</td>
<td>120</td>
<td>130*</td>
<td>140**</td>
<td>-</td>
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<td>Different</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90±35</td>
<td>118±32</td>
<td>160±62</td>
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<tr>
<td>6</td>
<td>62M</td>
<td>LVan 100</td>
<td>135**</td>
<td>-</td>
<td>U.D.</td>
<td></td>
<td>Same</td>
</tr>
<tr>
<td>7</td>
<td>67M</td>
<td>LVan 50</td>
<td>90*</td>
<td>100*</td>
<td>-</td>
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<td>Different</td>
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<tr>
<td>8</td>
<td>58M</td>
<td>DCM 50</td>
<td>100*</td>
<td>100*</td>
<td>+</td>
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<td>Different</td>
</tr>
<tr>
<td>9</td>
<td>48M</td>
<td>DCM 90</td>
<td>170**</td>
<td>210**</td>
<td>+</td>
<td>EAS (~90ms)</td>
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<tr>
<td>10</td>
<td>51F</td>
<td>DCM 100</td>
<td>170**</td>
<td>280**</td>
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<td>EAS (~100ms)</td>
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<tr>
<td>11</td>
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<td>CM 50</td>
<td>100*</td>
<td>140**</td>
<td>MD +</td>
<td>Concealed ent</td>
<td>Different</td>
</tr>
<tr>
<td>12</td>
<td>17F</td>
<td>DORV 70</td>
<td>60</td>
<td>-</td>
<td>CA U.D.</td>
<td></td>
<td>Same</td>
</tr>
<tr>
<td>13</td>
<td>67M</td>
<td>Idio 60</td>
<td>60</td>
<td>120*</td>
<td>+</td>
<td>EAS (~25ms)</td>
<td>Different</td>
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<tr>
<td></td>
<td></td>
<td>78±29</td>
<td>113±38</td>
<td>159±64</td>
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</table>

Thirteen patients who had sites with a long St–QRS interval are shown in this table. M=male; F=female; UHD=underlying heart disease; OMI=old myocardial infarction; LVan=idiopathic left ventricular aneurysm; DCM=idiopathic dilated cardiomyopathy; CM=cardiomyopathy of undetermined cause; DORV=post operation of double outlet right ventricle; Idio=Idiopathic VT; MD=Mid-diastolic potential; CA=continuous activity; Relation=relationship between the site with a long St–QRS interval and the site of VT origin; U.D.=undetermined; Concealed ent=concealed entrainment; EAS=the earliest ventricular activation site. An asterisk (*) indicates a site with an abnormally prolonged local electrogram and double asterisks (**) a site with fragmentation.

12. In group 3, the origin was confirmed at the free wall of the right ventricle in 9 cases, at the septum of the right ventricle in 5, at the septum of the left ventricle in 3, and at the free wall of the left ventricle in 4.

Sites with a prolonged St–QRS interval: Thirteen sites with a long St-QRS interval were determined in 13 of 52 patients (25.0%), and they ranged from 50 to 140 milliseconds (mean 78±29 milliseconds). As summarized in Table I, 5 of 13 patients had remote myocardial infarction. The 5 patients were 45.5% of the 11 patients in group 1, and the mean St–QRS interval was 90±35 milliseconds (Tables I and II). These 5 sites with a long St–QRS interval were located at the free wall of the left ventricle in 4 (posterior 2, lateral 2) and at the septum of the right ventricle near the His electrogram recording area in 1. Seven other patients had other underlying heart diseases (group 2): idiopathic left ventricular aneurysm in 2 patients, cardiomyopathies in 4, and post-operative stage of congenital heart disease in 1. These 7 patients accounted for 28.0% of the 25 patients in group 2, and the mean St–QRS interval was 73±22 milliseconds. These 7 sites with a long St–QRS interval were confirmed to be located at the free wall of the left ventricle in 4 (lateral 3, posterior 1), at the outflow tract of the right ventricle.
Figure 1. Electrophysiologic findings. A 51-year-old woman with idiopathic dilated cardiomyopathy (case 10). During sinus rhythm (A), a local electrogram at the site of the outflow tract of the right ventricle (site 17) showed fragmentation with a duration of 170 milliseconds. When rapid pacing was added to the site (B), paced QRS morphology, showing a left bundle branch block pattern, was observed with a St-QRS interval of 100 milliseconds. This paced QRS morphology was identical to that of VT and local electrical activity was confirmed at the pacing site immediately after the stimulus artifact. During VT (C), a local electrogram at the site was observed 100 milliseconds before the onset of QRS activation, and this site was considered the earliest during VT. Radiofrequency current of 50 Watts × 30 seconds was applied to the site and VT was terminated in 2.5 seconds. After the fluguration (D), VT became non-inducible. RVA=right ventricular apex; RVO=outflow tract of the right ventricle; MAP=mapping site; RF=radiofrequency current ablation. The endocardial mapping site was expressed according to the method of Josephson et al.6,14 Other abbreviations are the same as in Table II.

in 2, and at the septum of the right ventricle in 1. A long St-QRS interval of 60 milliseconds was observed at the septum of the right ventricle near the His electrogram recording area in 1 (6.3%) of the 16 patients with idiopathic VT. There was no statistical difference in the St-QRS intervals among these three groups.

There was no statistical difference in the incidence of sites with long St-QRS intervals among the 3 groups (Table I), but the incidence in group 1 tended to be higher than that in group 3 (5/11=45.5% vs 1/16=6.3%, p<0.1).

Local electrogram at the site with a long St-QRS interval: During sinus rhythm, the duration of the local electrograms at sites with a long St-QRS interval was 113±38 milliseconds, and 11 (84.6%) of 13 sites showed a long duration of more than 70 milliseconds and 4 (30.8%) of 13 sites showed fragment-
Figure 2. Electrophysiologic findings. A 48-year-old man with idiopathic dilated cardiomyopathy (case 9). During sinus rhythm (A), a local electrogram at the site of the left ventricle (site 7–9) showed fragmentation with a duration of 170 milliseconds. Rapid pacing at the site resulted in the same QRS morphology as that of VT with a St–QRS interval of 90 milliseconds (B). During VT (C), a local electrogram at the site showed fragmentation (210 milliseconds) and the initiation of the local electrogram was confirmed 90 milliseconds before the onset of QRS activation. This site was considered the earliest during VT. LV=left ventricle. Other abbreviations are the same as in the Tables and Figure 1.

The duration of the local electrogram of group 1 was 118±32 milliseconds (n=5) and that of group 2 was 118±39 milliseconds (n=7), and 60 milliseconds in one patient in group 3. There was no statistical difference in the duration of the local electrograms between groups 1 and 2. Late potential was not observed in any site with a long St–QRS interval.

When VT was induced, the duration of the local electrogram at the site with a long St–QRS interval was prolonged from 113±38 to 159±64 milliseconds (p<0.05). The durations of the electrograms during VT were 160±62 milliseconds in group 1 (n=5), and 166±70 milliseconds in group 2 (n=5). Continuous local electrical activity was observed in 1 patient of group 2, and mid-diastolic potential was confirmed in 2 patients, one each in groups 1 and 2. In one patient of group 2 (case 6), we could not record the local electrogram during VT. During VT, the local electrogram lasted for 120 milliseconds in one patient in group 3. There was no statistical difference in the durations of the local electrograms among the 3 groups.

Relationship between the site with a long St–QRS interval and the site of VT origin: In 3 patients (cases 1, 8, and 11), concealed entrainment was confirmed when the rapid pacings were added from the site with a long St–QRS interval (Figure 3). In 5 other patients (cases 2, 3, 9, 10, and 13), the local electrograms at the sites were found prior to the onset of the QRS activation (40, 50, 90, 100, and 60 milliseconds, respectively) and these electrograms were con-
Figure 3. Electrophysiologic findings. A 66-year-old woman with cardiomyopathy (case 11). During sinus rhythm (A), QRS morphology showed complete right bundle branch block, and frontal electrical axis deviated toward the right. The local electrogram at the outflow tract of the right ventricle was 70 milliseconds in duration. Rapid pacing to the site resulted in a QRS morphology with a long St-QRS interval (50 milliseconds), but this QRS morphology was quite different from that of VT. Local electrical activity was confirmed at the pacing site immediately after the pacing artifact (B). During the VT (C), mid-diastolic potential was observed at the outflow tract of the right ventricle. When rapid pacing was added from the site, the cycle length of VT accelerated to the paced cycle length without a change in the QRS complex, and the post-paced return cycle at the stimulus site was identical to the cycle length of VT. The interval between the mid-diastolic potential and the onset of QRS complex was 190 milliseconds during VT, and this interval was the same as the interval from the stimulus artifact to the onset of the QRS complex during rapid pacing. The interval from the stimulus artifact to the onset of the QRS complex during VT (190 milliseconds) was longer than that in sinus rhythm (50 milliseconds). Functional block during VT and/or change in the slow pathways from the pacing site might explain the prolongation. The duration of the local electrogram at the pacing site was 140 milliseconds. The cycle length of VT was 440 milliseconds and the pacing cycle length was 380 milliseconds. HBE=His bundle electrogram recording area. Other abbreviations are the same as in the Tables and Figures 1 & 2.

sidered the earliest during VT.\textsuperscript{15-17} In these 8 patients, the sites with a long St-QRS interval seemed to be identical to the sites of VT origin, and low energy electrical catheter ablation (100 Joules) or radiofrequency current ablation (30–50 Watts) to the sites was effective in preventing the induction of VT in 5 patients\textsuperscript{17,24} (Figures 1 and 4).

In 5 patients (cases 2, 6, 9, 10, and 12), paced QRS morphology at the site with a long St-QRS interval showed identical configuration to that of the clinical VT. In 4 of those patients, the St-QRS interval was identical to the interval between the initiation of the local electrogram and the onset of the QRS complex during VT as described in Figures 1 and 2. Therefore, these findings also supported our conclusion that the site was located at the site of VT origin or
Figure 4. Radiofrequency ablation. This case is the same as that in Figure 2. Radiofrequency ablation at an energy of 40 Watts was applied to the earliest ventricular activation site as determined in Figure 2-C. 7.5 seconds after the flutteration, the VT was terminated and became non-inducible. Abbreviations are the same as in the Tables and Figures 1-3.

in its vicinity.

In the remaining three patients (cases 4, 5, and 7), the local electrogram of the site with a long St-QRS interval was observed after the onset of the QRS complex, and the site did not show mid-diastolic potential or local continuous electrical activity during VT. Furthermore, the paced QRS morphology at the site was different from that of the clinical VT and these sites were considered to be far from the VT origin.

Therefore, the site with a long St-QRS interval was considered to participate in the formation of the reentry circuit in 8 (cases 1–3, 8–11, and 13) of 13 patients (61.5%). In 2 other patients (cases 6 & 12), the paced QRS morphology was the same as that of the VT, but the relationship between such sites and the site of VT origin was undetermined.

**DISCUSSION**

Slow conduction areas are essential for the initiation and maintenance of reentrant VTs,1-5) and the existence of such areas was confirmed by the demonstration of a long conduction time from the stimulus artifact to the orthodromically captured electrogram during VT.18,25-27) On the other hand, sites with a long St – QRS interval might be observed during sinus rhythm,5,6) and in those cases the long St – QRS interval would be due to a delayed conduction from the site of stimulation to the entire myocardium. Recently, Stevenson et al6) reported that some sites with a long St – QRS interval were participating in the formation of the reentrant circuit. However, the relationship between the site
with a long St–QRS interval and the VT origin has not been examined in a systematic manner.

**Underlying heart diseases:** In this study, detailed mapping showed sites with a long St–QRS interval occurring often, but there was no statistical difference in the incidence of sites with a long St–QRS interval among the 3 groups. However, the incidence seemed to be higher in the groups with structural heart disease than in the group without heart disease. These findings reflect the anatomical and/or histological differences in the subgroups. In the patients with VT associated with organic heart diseases, fibrosis, calcification and/or fatty infiltration provided the basis of delayed conduction and such a histological abnormality seems unlikely in patients with idiopathic VT as almost all conventional examinations showed normal findings.

**Local electrocardiogram at the site with a long St–QRS interval:** Fragmented or continuous activities which were considered to represent slowed or nonuniform anisotropic conduction were observed in patients with organic heart diseases, but were rare in patients with idiopathic VT. In this study the sites with a long St–QRS interval were closely associated with an abnormal or fragmented local electrogram in sinus rhythm (11/13 sites), so sites with a long St–QRS interval include the slowed conductive property even in sinus rhythm. The conduction delay would be the common mechanism for the long St-QRS interval and fragmentation, and the fragmentation was exaggerated during VT; the duration of the local electrogram was prolonged during VT from 113±38 to 159±64 milliseconds. Frequency-dependent conduction delay and/or functional block during VT might explain this prolongation.

**Relation between the site with a long St–QRS interval and the site of VT origin:** If the pacing in sinus rhythm from the site with a long St–QRS interval resulted in a QRS morphology identical to that of the VT, the site can be considered to be the critical portion of the reentry circuit. The exception was when the site with a long St–QRS interval had a bystander connected to the slow pathway of the active limb of the reentry circuit. In this situation, St–QRS interval in sinus rhythm would be longer than the interval between the initiation of the local electrogram and the onset of the QRS complex during VT. In this study, these two intervals were equal in 4 of 5 patients, but pacing during VT was required to determine whether the site was essential to the reentry circuit or not.

On the other hand, even if the pacing from the site with a long St–QRS interval showed a different paced QRS morphology from that of the VT, the site may be the critical part of the reentry circuit of VT. This would be possible in the following situations. (1) Functional block during VT would alter the direction of the conduction of the wavefronts resulting in a paced QRS morphology differ-
ent from that in the VT. (2) If the site with a long St – QRS interval has multiple slow conducting pathways, the QRS morphology would be affected by the conducting pathway. In this situation, the paced QRS morphology might change depending on the paced cycle length or stimulus output. These two mechanisms might explain the observation that concealed entrainment was observed even from the site at which paced QRS morphology in sinus rhythm was different from that of VT.

At the earliest ventricular activation site, paced QRS morphology in sinus rhythm was considered to be identical to VT morphology, but these two morphologies were different in 2 patients (cases 3 and 13). However, catheter ablation to the earliest ventricular activation site was effective in preventing the induction of VT in both cases, so the site must act as the active limb of the reentry circuit. Due to the constitution of the functional block, propagation of the wavefronts during VT must be different from that of pacing in sinus rhythm, even if the pacing site was identical to the exit from the reentry circuit.

Therefore, to prove that the site with a long St – QRS interval is participating as an active limb of the reentry circuit, rapid pacing at the site during VT must fulfill the criteria of Fontaine et al. Additional evidence is the finding that the local electrogram at the site with a long St – QRS interval showed the earliest ventricular activation during VT. In this study, concealed entrainment was observed in 3 patients, and the earliest activated electrogram at the site with a long St – QRS interval was observed in 5 other patients.

**Limitations:** Several limitations should be emphasized. There was a difference in the number of pace-mapping sites in each ventricle. Although pace-mapping was performed at a minimum of 10 sites in each ventricle, pacing was usually done near the site of VT origin. Therefore, it is possible to have missed a remote site which might show a long St – QRS interval. Although rapid pacing at a shorter cycle length might result in a longer St – QRS interval, the effect of the paced cycle length on the St – QRS interval was not evaluated in a systematic way. However, it was felt that such intervention would alter the present results. A large number of patients might be needed to establish the incidence of a long St – QRS interval and the relationship between such sites and the VT origin. Finally, the sites were not examined histologically or anatomically.

**Clinical implication:** Endocardial mapping during VT is essential to determine the site of VT origin. However, this approach was unsuitable for patients with hemodynamically unstable VT or with severe cardiac dysfunction. Pace-mapping is a useful method in such patients, and a long St – QRS interval might be another landmark to identify the sites with arrhythmogenicity.
CONCLUSION

In this study, we focused on those sites with a long St-QRS interval during pace-mapping.

1) Sites with a long St–QRS interval were observed in 25.0% of the patients with sustained ventricular tachycardia, and the incidence tended to be higher in patients with remote myocardial infarction.

2) The sites with a long St–QRS interval were associated with fragmented local electrograms and were related to the site of VT origin. In some cases, the site was considered to be the active limb of the reentry circuit.

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