Alternation in the Fusion Complex during Constant Pacing of Sustained Ventricular Tachycardia

Masaomi CHINUSHI, M.D., Yoshifusa AIZAWA, M.D.,
Yoriko KUSANO, M.D., Hitoshi KITAZAWA, M.D.,
Takefumi MIYAJIMA, M.D., and Akira SHIBATA, M.D.

SUMMARY

In a patient with sustained ventricular tachycardia (VT), we observed two different conduction times through the reentry circuit at the critical paced cycle length. The cycle length of the VT was 420 msec and overdrive pacing initially performed at a paced cycle length of 400 msec and repeated at decrements of 10 msec until the VT was interrupted at a paced cycle length of 320 msec. During rapid pacing, constant fusion and progressive fusion were confirmed. The first post-pacing return cycle was identical to each paced cycle length. The conduction time between the stimulus artifact and the orthodromically captured electrogram at the left ventricle was constant at 350 msec in each paced cycle length. However, only at a pacing cycle length of 360 msec two conduction times were alternatively observed, one of 350 msec and the other of 365 msec. When the conduction time changed from 350 msec to 365 msec, morphological alternation both in the surface QRS complex and in the orthodromically captured electrogram was evident. Dual slow pathways or a single slow pathway with plural exits from the reentry circuit is a likely mechanism of the alternation. (*Jpn Heart J* 34: 227-234, 1993.)

Key Words:

Ventricular tachycardia Dual pathways Slow conducting pathway
Entrainment

THE conductive properties of the atrioventricular (AV) node and the accessory pathway are relatively well known,¹⁾⁻⁴⁾ and dual pathways have been demonstrated as the cause of reentry in the AV node.⁴⁾⁻⁶⁾

In ventricular tachycardia (VT), resetting by a single extrastimulus^{7),8)} or transient entrainment^{9),10)} might be used to disclose the conductive characteristic of the slow conduction zone, but data concerning the electrophysiologic properties of reentry circuits in human ventricular tachycardia have been recently accumulating.

Recently, we observed an alternation both in the surface QRS complex

From the First Department of Internal Medicine, Niigata University School of Medicine, Niigata, Japan.

Mailing address: Masaomi Chinushi, M.D., First Department Internal Medicine, Niigata University School of Medicine, 1-754 Asahimachi, Niigata 951, Japan.

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and in the orthodromically captured electrogram during entrainment of VT at a critical paced cycle length. Change in the time interval between the stimulus and the electrograms near the site of exit was related to such alternations. Dual slow pathways and a single slow pathway with plural exits from the reentry circuit were considered to be likely mechanisms of the alternation.

CASE REPORT

The patient was a 66-year-old female. She developed dizziness while walking 3 months before admission and she was diagnosed as having a complete AV block (Fig. 1). She did not have symptoms suggesting inflammatory disease or ischemic heart disease.

After admission to a hospital, she underwent temporary pacing for AV block, but VT with a right bundle branch block pattern (Fig. 2) occurred during the procedure and she was referred to our hospital.

Fig. 1. Twelve-lead electrocardiogram showing complete AV block. The rate of the QRS complex was 45 beats per minute, and that of the P wave was 77 beats per minute. Arrows show the position of the P wave.

On admission, she was 154 cm in height and weighed 57.0 kg. The physical examination was non-contributory. The hematological and serological examinations showed no abnormalities. Her blood chemistry was also normal. The chest x-ray showed a mildly enlarged heart but no pulmonary congestion.

Her electrocardiogram showed a complete dissociation between the P wave and the QRS complex (Fig. 1). The QT interval was 0.48 sec.

The two-dimensional echocardiogram showed reduced wall motion in the apical portion of the intraventricular septum. M-mode echocardiogram showed a dilated right ventricle, 39 mm in end-diastolic dimension. Mild mitral and pulmonary regurgitation was observed by Doppler echocardiogram. The coronary angiograms showed normal vessels.

After obtaining informed consent from her and her family, the electrophysiologic study was performed in the nonsedated and postabsorptive state. Three 6F quadripolar catheters with interelectrode distances of 5 mm were positioned against the apex of the right ventricle, at the His-bundle electrogram recording area and at the left ventricle, under fluoroscopic guidance. The stimulation was performed using electrodes 1 (distal tip) and 3, and the intracardiac electrogram from the pacing site was recorded using electrodes 2 and 4. Otherwise, the local electrogram was recorded using the two distal electrodes, 1 and 2.

Fig. 2. Twelve-lead electrocardiogram showing ventricular tachycardia. The VT shows a right bundle branch block pattern with a cycle length of 420 msec.

The heart was stimulated at twice diastolic threshold with a 2 msec rectangular pulse by a programmable stimulator (Fukuda Denshi Co., Cardiac Stimulator BC02). A standard stimulation protocol was used for the induction of VT.^{11,12} Intracardiac electrograms were recorded on an ink-jet recorder (Siemens Elema Co. Ltd., Mingograf 7) with three surface leads, I, II, and V₁, at a paper speed of 100 mm/sec. The band-pass filter was set at 50 to 300 Hz.

In the drug-free state, VT with the same configuration as that of clinical VT was induced by double extrastimuli from the apex of the right ventricle. The cycle length of VT was 420 msec. Endocardial activation was mapped extensively, and the earliest site of activation was determined at the lateral wall of the left ventricle. The local electrogram at the site was 10 msec before the onset of the QRS complex.

Then, rapid pacings were performed from the apex of the right ventricle to entrain tachycardia starting at a cycle length 20 msec less than the cycle length of VT and after decrements of 10 msec until VT was interrupted at a paced

Fig. 3. Overdrive pacing during the VT.

The VT cycle length was 420 msec. Overdrive pacing was performed at cycle lengths of 370 msec (A) and 350 msec (B). Constant fusion was observed in the QRS complex and constant fusion with different degrees at different paced cycle lengths was also confirmed. The interval from the stimulus to the entrained local electrogram at the left ventricle was constant: 350 msec, and the local electrogram showed the same morphology at each paced cycle length. The first post-pacing return cycle was identical to each paced cycle length.

RVA = right ventricular apex; HBE = the tricuspid ring in the His-bundle area; LV = lateral wall of the left ventricle; PCL = paced cycle length.

Fig. 4. Overdrive pacing during VT.

Overdrive pacing was performed at a cycle length of 360 msec during the VT (A). During the last part of the pacing, two conduction times from the stimulus artifact to local electrogram at the left ventricle were alternatively observed. As the conduction time changed, the local electrogram at the left ventricle also showed slight alternation in the morphology and in the cycle length: one was 375 msec and the other was 345 msec. At the same time, the QRS complex on the surface electrogram changed in morphology. During the pacing in the escape rhythm (B), the interval from the stimulus artifact to the local electrogram at the left ventricle was 80 msec. Abbreviations are the same as in Fig. 3.

cycle length of 320 msec. During rapid pacing, constant fusion and constant fusion with different degrees at different paced cycle lengths were confirmed^{13,14} (Fig. 3). The first post-pacing return cycle was identical to each paced cycle length.¹⁴ These findings fulfilled the criteria of transient entrainment established by earlier workers.¹³⁻¹⁵

As reported earlier,¹⁰ the conduction time through the reentry circuit was measured as the time interval from the stimulus (apex of the right ventricle) to the orthodromically captured electrogram (lateral wall of the left ventricle) near the site of exit from the slow conduction zone. As the paced cycle length decreased from 400 to 370 msec and from 350 to 320 msec, the conduction time was fixed at 350 msec. The fusion QRS complex was stable during the pascings (Fig. 3).

At the intermediate paced cycle length of 360 msec, the conduction time was found to alternate between 350 msec and 365 msec. The alternation in conduction time was accompanied by a slight change in the morphology of the local electrogram recorded at the lateral wall of the left ventricle (Fig. 4A). Alternation in the QRS morphology on the surface electrogram was also observed during the alternation of the conduction times. We did not add extrastimulation during the VT. During the escape rhythm, the conduction time from the stimulus artifact to the lateral wall of the left ventricle was 80 msec (Fig. 4B).

DISCUSSION

Recently, electrophysiologic data concerning the slow conduction zone have been accumulating.^{7)-10),16),17)} The evaluation of the conductive properties of the slow conduction zone has been performed by two approaches. One is the resetting of the VT by extrastimulation^{7),8),16)} and the other is the use of transient entrainment of VT with rapid pacing.^{9),10),17)}

In the present case, we measured the conduction time as the time interval between the stimulus and the orthodromically captured electrogram at the left ventricle near the site of exit from the reentry circuit. Obviously, additional time is involved in this conduction time which is required for the wave fronts to propagate between the pacing site and the entrance of the reentry circuit. The conduction time was constant, 350 msec at each cycle length, except for the pacing at a cycle length of 360 msec. At this critical cycle length, the conduction times alternated between 350 and 365 msec.

There are several possible explanations for the alternation of the conduction time: (1) There are alternating pathways within the reentry circuit. (2) A latency at the pacing site or two conducting pathways in the tissue intervening between the pacing site and the entrance of the reentry circuit. (3) There are two entrances or exits in the reentry circuit.¹⁸⁾ (4) A slight shift of the catheter position might cause a change in the conduction time and morphology; this possibility was felt to be unlikely because this alternation occurred only at the critical paced cycle length.

When the conduction time changed from 350 to 365 msec, the local electrogram at the left ventricle showed a slight change in morphology. This finding suggests a change in the activation sequence from the exit of the reentry circuit. The antidromically propagating wave front did not seem to relate to the change in the morphology, because a stimulus artifact was found in the orthodromically captured electrogram at the site during the entrainment, and the conduction time from the stimulus artifact to the local electrogram at this site was 80 msec during the escape rhythm (Fig. 4B). These findings suggested that the antidromically activating wavefront could not reach this site during the entrainment and this site was activated orthodromically. Therefore, we think that alteration in QRS morphology is a secondary phenomenon due to alternating conduction over two slow pathways or due to changing the two exits of one slow pathway from one to the other.

Although the existence of two pathways between the stimulus site and the entrance of the reentry circuit could not be excluded, the change in the morphology of the local electrogram could not be explained by this mechanism. Also, a latency at the pacing site was not observed during the entrainment.

We were unable to determine the precise mechanism of the initiation of such an alternating conduction, and linking between two slow conducting pathways or so-called supernormal conduction^{19),20)} might be related to the finding. However, this phenomenon was an unusual feature seen during rapid pacing in VT, and it would be important to examine the conductive characteristics in the reentry circuit.

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