A Study on Entrainment in Various Forms of Tachycardias

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In order to study the entrainment phenomenon in various forms of clinical and experimental tachycardia, overdrive pacing was performed in clinical paroxysmal supraventricular tachycardia (PSVT) of Wolff-Parkinson-White syndrome (6 cases), atrial flutter (AF-6 cases), ventricular tachycardia (VT-2 cases), experimental AF induced by Rosenblueth's method (5 dogs), and in VT induced by aconitine (5 dogs). Progressive fusion was demonstrated in all but aconitine-induced VT. After cessation of pacing, the first tachycardia complex showed no fusion at all, but the timing of the complex varied depending on the recording sites of the electrogram. The tachycardia complex occurred at the pacing rate only when the electrogram was recorded at the upstream of the pacing site along the reentry circuit in PSVT and experimental AF. In clinical VT, the complex appeared progressively later as the pacing rate was increased. In aconitine-induced experimental VT, the complex occurred at its original tachycardia rate originating from the site where aconitine was applied.

In reentrant tachycardia, entrainment could be observed even when all three of the diagnostic criteria proposed by Brugada et al. were not fulfilled. The mechanism of tachycardia can be assessed by entrainment, although the proposed criteria should be reevaluated.

SINCE Waldo et al. first described the entrainment phenomenon of atrial flutter,¹ entrainment has been observed in various forms of tachycardia, such as atrial flutter (AF),¹⁻⁴ paroxysmal supraventricular tachycardia (PSVT) of Wolff-Parkinson-White (WPW) syndrome,⁵⁻⁸ AV nodal reentry,⁹ and ventricular tachycardia (VT).¹⁰⁻¹³

Entrainment is defined as "an increase in the rate of the tachycardia to a faster pacing rate, with resumption of the intrinsic rate of tachy-

cardia upon either abrupt cessation of pacing or slowing of the pacing rate below the intrinsic rate of the tachycardia."^{6,11,13}

Although the mechanism of entrainment has not been clearly explained, demonstration of entrainment strongly suggests that the mechanism of tachycardia is reentry. Among the electrophysiological phenomena which can occur during rapid pacing of a tachycardia, Brugada et al. proposed three diagnostic criteria of entrainment.¹⁴ First, there occurs progressive fusion between paced and tachycardia complexes at different rates of pacing. Second, after cessation of pacing the first tachycardia complex occurs at the pacing rate but shows no fusion. Third, if the local block occurs the distal site of the block is excited by the impulse via the different route.

Key Words:

Entrainment
Electrophysiology
Tachycardia
Reentry
Enhanced automaticity

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This work was partly supported by a Research Grant for Cardiovascular Diseases (59C-5) from the Ministry of Health and Welfare.

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INTERVALS

from the nearest potentials
of the last pacing stimulus
to the next potentials

PCL	330	310
R-R	330	310
H-H	330	310
A _{Eso} - A _{Eso}	330	360
A _{HRA} - A _{HRA}	410	420
A _{HBE} - A _{HBE}	360	370

Fig.1. Entrainment of reentrant tachycardia of WPW syndrome.

During reentrant tachycardia using AV node as an antegrade limb and left-sided accessory bypass pathway as a retrograde limb, overdrive pacing from high right atrium was carried out. Entrainment was demonstrated at pacing cycle lengths of both 330 msec and 310 msec. But the configuration of the left atrial potential and intervals from the nearest potentials of the last pacing stimulus to the next potential varied depending on the pacing cycle length. See text for discussion.

Abbreviations: Eso = esophageal lead; HRA = high right atrium; HBE = His bundle electrogram; RV = right ventricle; St = stimulus artifact; PCL = pacing cycle length

In order to reevaluate the proposed criteria of entrainment in determining the mechanism of tachycardia, we performed overdrive pacing in various forms of clinical and experimental tachycardia.

MATERIALS AND METHODS

1) Clinical tachycardia

There were 6 cases of PSVT of WPW syndrome, 6 cases of AF and 2 cases of VT. After informed consent was obtained from each patient, an electrophysiological study was performed. Three 6F electrode catheters were advanced from the femoral vein to the high right atrium (HRA), His bundle position and right ventricle (RV) under fluoroscopic control. In order to record the electrogram of the left

Fig.2. Entrainment of experimental atrial flutter.

Seven electrodes were attached to the right and left atria and atrial flutter was induced by Rosenbleuth's method. During atrial flutter with a cycle length of 160 msec, overdrive pacing was carried out from the site near electrode 5 at a pacing cycle length of 140 msec. See text for discussion.

Abbreviation: LA = left atrium; RA = right atrium; SVC = superior vena cava; IVC = inferior vena cava

atrium (LA), an esophageal lead was inserted. In patients with AF, a 4F semifloating catheter was inserted from the right antecubital vein and advanced to the low lateral right atrium, and a 6F electrode catheter was advanced from the femoral vein to the HRA. In all cases, three surface ECG (I, aV_F, V₁ or I, II, III) were recorded simultaneously along with the intracardiac electrograms. All tachycardias could be initiated and terminated by electrical stimu-

lation.

2) Experimental tachycardia

Eleven mongrel dogs were anesthetized with phentobarbital (30 mg/kg body weight). Under artificial respiration, the heart was exposed through a median sternotomy.

In 6 dogs, seven bipolar electrodes were attached to the right and left atria and AF was induced by rapid electrical stimulation according

Fig.3. Entrainment of clinical ventricular tachycardia.

Rapid pacing from right ventricle was performed during ventricular tachycardia with a cycle length of 320 msec. As the pacing cycle length was shortened, progressive fusion was observed, while the interval between the last paced beat and the first tachycardia complex prolonged gradually.

Abbreviations are the same as in Fig. 2.

to the method of Rosenblueth and Garcia Ramos.¹⁵ In 5 dogs, five electrodes were attached to the right and left ventricles. VT was induced by application of aconitine to the RV surface, the mechanism of which is considered to be the same as in AF.¹⁶ The surface ECG and contiguous atrial or ventricular electrograms were recorded simultaneously.

3) Stimulation protocol

Overdrive pacing during tachycardia was initiated at a pacing cycle length 10 msec shorter than that of respective tachycardia, continued for at least 5 seconds, and terminated abruptly. If the tachycardia was not terminated, the pacing cycle length was shortened by 10 msec until either tachycardia was terminated or it turned into other forms of arrhythmia. Configurations and timing of the atrial or ventricular electrograms recorded before, during and after overdrive pacing were compared to one another at different pacing rates.

RESULTS

1) PSVT of WPW syndrome

All the PSVT induced by electrical stimulation was of macro-reentry, using the AV node as an antegrade limb and the accessory pathway as a retrograde limb. By the overdrive pacing from HRA during tachycardia, entrainment was observed in all cases at pacing cycle lengths of 10 to 50 msec shorter than those of original tachycardia.

Figure 1 shows the typical pattern of entrainment of left-sided bypass pathway-type PSVT. At a relatively longer pacing cycle length, LA potential via the esophageal lead (Eso) was located after the ventricular potential at the same timing as during the tachycardia. But as the pacing cycle length was shortened, LA was excited by the impulse from HRA, and the timing and the configuration of the potential changed. The ventricle was excited by the pacing stimulus appearing near the QRS one beat previously due to the delay in the AV node. The ventricular interval was the same as the pacing interval at the cessation of pacing and the following intervals returned to the original tachycardia interval. Intervals of HRA electrogram ($A_{HRA}-A_{HRA}$) and A wave in the His bundle electrogram ($A_{HBE}-A_{HBE}$), however, were

Fig.4. Entrainment of aconitine-induced ventricular tachycardia.

Five electrodes were attached to both ventricles and aconitine was applied near electrode 5. During VT, excitation spread from electrode 5 with an interval of 210 msec. Overdrive pacing from electrode 2 with an interval of 190 msec was performed. At the cessation of pacing, original tachycardia occurred from the site where aconitine was applied with the original interval.

RV = right ventricle; LV = left ventricle

prolonged by one beat before returning to their original tachycardia intervals. The interval of LA was the same as for the ventricle when it was excited orthodromically. But once it was excited antidromically at a shorter pacing cycle, it showed prolongation as A wave in HBE. This different pattern of interval of LA electrograms depending on the pacing cycle length was observed only in left-sided bypass-type PSVT. When the pacing cycle length reached the critical interval, PSVT was terminated by AH block in all cases.

2) AF

In clinical AF, entrainment was observed in all cases at pacing cycle lengths of 10 to 80 msec shorter than that of intrinsic flutter cycle length. As the pacing cycle length was shortened, AF turned into sinus rhythm in one case and atrial fibrillation in five cases. On the cessation of overdrive pacing, the cycle length of which was not short enough to terminate AF, the timing of the appearance of the first tachycardia complex varied depending on the recording sites of the

electrogram in relation to the pacing site.

In order to explain this difference, an experimental study was performed. Seven electrodes were attached to the right and left atria of the dog's heart, and overdrive pacing during AF was carried out (Fig. 2). A typical case is shown in Fig. 2. During AF with flutter cycle length of 160 msec, overdrive pacing at a pacing cycle length of 140 msec was performed from the site near electrode 5. During entrainment, the pacing impulse entered into the reentry circuit and advanced from electrode 5 to 4 antidromically and electrode 5 to 7 orthodromically. The antidromic wave front collided with the orthodromic wave front of the previous paced beat at the site between electrodes 3 and 4. As for the intervals just after the cessation of pacing, they returned to the original flutter interval at the recording sites of 1 to 4. But at the sites of 5 to 7, they were prolonged by one cycle before returning to the original flutter interval.

3) VT

In two clinical cases, VT was initiated and

Fig.5. Schema of entrainment of reentrant tachycardia of WPW syndrome.

A: Reentrant tachycardia using AV node as an antegrade limb and bypass pathway as a retrograde limb.

B: Atrial pacing is performed during tachycardia. Pacing impulse enters into reentry circuit and advances in two directions. The antidromic wave front collides with the oncoming orthodromic wave front of the previous beat.

C: The next pacing impulse also enters into the reentry loop. And in this case, the antidromic wave front collides with the orthodromic wave front of the previous beat. Thus continuous resetting of the reentry circuit occurs.

D: When atrial pacing is stopped, the orthodromic wave front of the last paced beat is unopposed by the antidromic wave front. So this impulse restarts the tachycardia. A = atrium; V = ventricle

terminated by reproducible electrical stimulation. Overdrive pacing from the right ventricle was performed during VT, and entrainment was demonstrated in both cases at pacing cycle lengths of 10 to 100 msec shorter than that of original VT. Progressive fusion was observed in both cases. The first tachycardia complex after cessation of pacing, which showed no fusion, appeared progressively later as the pacing interval was shortened (Fig. 4). And when the pacing cycle length reached a critical level, VT was terminated.

In aconitine-induced experimental VT, the mechanism of which was considered to be either enhanced automaticity or triggered activity, the excitation wave front spread out radially from the site where aconitine was applied. During tachycardia, overdrive pacing from the site near electrode 2 at cycle lengths of 10 to 80 msec shorter than that of original tachycardia was performed. Overdrive pacing caused the tachycardia rate to increase to pacing rate, but progressive fusion could not be observed. After cessation of pacing, the tachycardia complex resumed its original rate originating from the site where aconitine was applied (Fig. 4). The

increase in the pacing rate resulted in ventricular fibrillation.

DISCUSSION

1) PSVT of WPW syndrome

Entrainment was observed in all PSVT, the mechanism of which was macro-reentry using the AV node as an antegrade limb and an accessory pathway as a retrograde limb.

The schema of entrainment is shown in Fig. 5. Figure 5-A shows the AV reentrant tachycardia. When atrial pacing is initiated, pacing impulse enters into the reentry circuit and advances in two ways, orthodromic and antidromic. The antidromic wave front ($n+1$) collides with the oncoming orthodromic wave front from the previous beat (n) (Fig. 5-B). The same phenomenon occurs with the next pacing ($n+2$) (Fig. 5-C). Thus the resetting of the reentry circuit occurs at each atrial pacing and the tachycardia rate increases to the pacing rate. When the pacing is stopped, the orthodromic wave front of the last paced beat has no antidromic wave front to collide with. So it enters into the AV node and PSVT continues at its

Fig. 6. Schema of entrainment of experimental atrial flutter.

Pacing impulse delivered during atrial flutter enters into the reentry circuit and the same phenomenon occurs as observed in WPW tachycardia.

intrinsic rate.

In PSVT using a left sided accessory pathway, the configuration of the electrogram recorded at LA depends on the pacing rate. At a relatively longer pacing cycle length, LA is excited orthodromically, i.e. the orthodromic wave front excites the LA and then collides with the antidromic wave front of the next pacing impulse. So the LA potential is the same as in PSVT. When the pacing cycle length is shortened, the pacing impulse penetrates deeper into the reentry circuit. Therefore, LA is excited antidromically and the configuration of LA potential differs from that observed at a longer pacing cycle length. In WPW syndrome with right-sided accessory pathway, atrial potential near the accessory pathway was not recorded, so this phenomenon could not be demonstrated.

2) AF

By overdrive pacing during clinical AF, progressive fusion between paced and tachycardia complexes at different rates of pacing was observed. This fulfills the proposed first criterion. But the F-F interval at the cessation of pacing varied depending on the recording sites. This fact does not fulfill the second criterion.

The study on experimental AF, the mechanism of which was macro-reentry, demonstrated that after cessation of pacing the first tachycardia complex appeared at the pacing interval only when the electrogram was recorded at upstream of the pacing site along the reentry circuit. This was due to the delay between recording sites 3 and 4. It is possible that the conduction velocity differs during pacing and during tachycardia because of the change in the

direction along which the excitation proceeds. This finding indicates that the second criterion cannot be fulfilled when the electrogram is recorded at sites downstream along the reentry circuit.

Thus it was demonstrated that entrainment occurs in clinical AF without fulfilling the second proposed criterion.

The schema of entrainment of AF is shown in Fig. 6. The mechanism is the same as that of PSVT in Fig. 5.

3) VT

In clinical VT, tachycardia could be reproducibly initiated and terminated by electrical stimulation. Therefore, the mechanism was suggested to be either reentry or triggered activity. During overdrive pacing, progressive fusion was demonstrated but the interval between the last paced beat and the first tachycardia complex at the cessation of pacing gradually prolonged as the pacing cycle length was shortened. This is not compatible with the second criterion. Although only one criterion was fulfilled in clinical VT, the entrainment phenomenon did occur and it indicated the mechanism as reentry.

In contrast, none of the proposed diagnostic criteria of entrainment were fulfilled in aconitine-induced experimental VT with a mechanism other than reentry.

4) Reevaluation of the Proposed Criteria of Entrainment

In reentrant tachycardia, the response to rapid pacing fulfilled the first criterion of entrainment. But the second criterion could not be applied when the electrogram was recorded at sites downstream of the pacing site along the reentry circuit. So careful observation of the relationship between the pacing site and the recording site is necessary.

On the other hand, in tachycardia caused by enhanced automaticity, the response to overdrive pacing did not fulfill any of the three criteria.

In conclusion, the mechanism of tachycardia can be assessed by the entrainment phenomenon. However, the proposed criteria of entrainment should be reevaluated.

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