The Electrophysiological Characteristics of Various Types of Paroxysmal Tachycardias

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There are various types of paroxysmal tachycardia in clinical medicine and it is quite possible that the mechanism responsible for them might be different in different types or patterns of tachycardia, the origin of tachycardia, and underlying heart diseases with which tachycardias are associated. Recent developments in clinical and basic electrophysiological studies have delineated the mechanisms responsible for some of the paroxysmal tachycardias. For example, the atrioventricular re-entrant tachycardia in patients with Wolff-Parkinson-White syndrome (WPW tachycardia) is now considered to be the prototype of re-entrant tachycardia.\(^1\)\(^-\)\(^3\) Also, most of the sustained junctional tachycardia are considered to be due to atrioventricular nodal re-entrant tachycardia (AVNRT).\(^4\) Furthermore, electrophysiological studies during surgery and those in animal studies suggest that the mechanism responsible for ventricular tachycardia (VT) associated with old myocardial infarction (OMI) is also reentry.\(^5\)\(^-\)\(^6\) But the mechanisms responsible for other paroxysmal tachycardias remain obscure.

In this report, we studied 1) the electrophysiological characteristics of WPW tachycardia whose mechanism have been proven to be reentry, 2) the electrophysiological characteristics of AVNRT and VT in patients with OMI whose mechanism are probably reentry, 3) the electrophysiological characteristics of VT in patients with dilated cardiomyopathy (DCM) and those without apparent organic heart disease whose mechanism are still unknown.

First, we delineated the electrophysiological characteristics of reentry by studying electrophysiological characteristics of WPW tachycardia, AVNRT, and VT in OMI; then we compared the electrophysiological characteristics of each paroxysmal tachycardia to those of WPW tachycardia, AVNRT, and VT in OMI in order to identify the mechanisms of the paroxysmal tachycardia.

Patients selection:

The study consisted of 258 patients who underwent electrophysiological studies between January 1979 and June 1984 at the National Cardiovascular Center at Osaka, Japan. Of these, 52 patients (mean age: 51.4 years) had AVNRT, 66 patients (mean age: 36.9 years) had VT, and 140 patients (mean age: 43.0 years) had WPW tachycardia. Patients with VT were also classified according to the underlying heart disease. VT associated with old myocardial infarction (OMI): 11 patients, VT with dilated cardiomyopathy (DCM): 12 patients, and VT without apparent structural heart disease (IdP): 43 patients. IdP VT were then divided according to the type of VT: sustained (15 patients) vs repetitive (28 patients). Repetitive VT represents frequent episodes of non-sustained tachycardia interposed between few sinus beats. Sustained VT represents the tachycardia lasting

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- Paroxysmal supraventricular tachycardia
- Ventricular tachycardia
- Re-entry
- Triggered activity

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more than 3 minutes without spontaneous termination. While, as to VT in OMI and DCM, only patients with sustained VT were included in this study.

Electrophysiological Methods:

Patients underwent electrophysiological studies after giving informed consent. All patients were sedated with diazepam and all antiarrhythmic medications were discontinued at least three days before the studies. His bundle electrograms were recorded using a standard technique. WPW tachycardia and AVNRT were diagnosed by characteristic atrioventricular function curves obtained during extrastimulus testing and by patterns of atrial depolarization of the tachycardia recorded by multiple intracardiac electrograms as previously reported. VT was diagnosed by demonstrating atrioventricular dissociation during the tachycardia.

Stimulation was done with a programmed digital stimulator (Fukuda Denshi Cardiac Stimulator: BC-02A) which produced electrical impulse of a 2.0 ms duration at twice diastolic threshold. As to the initiation of WPW tachycardia and AVNRT, atrial extrastimuli and pacings were used. Stimulation was done at the high right atrium first and if stimulation at the high right atrium failed to initiate the tachycardia, then stimulation at the coronary sinus was repeated. To initiate VT, a single extrastimulus at 2 different cycle lengths (600 and 400 msec) was initially delivered to the ventricle. If a single ventricular extrastimulus failed to elicit VT, then double ventricular extrastimuli were delivered until VT was provoked or until all extrastimuli failed to evoke ventricular responses. Stimulation was first performed at the right ventricular apex, but when VT was not induced by stimulation at the right ventricular apex, the
Fig. 2. Figure 2 shows the electrophysiological characteristics of the initiation and termination of the tachycardia and those in response to atrial pacings during episodes in patients with atrioventricular re-entrant tachycardia (AVNRT). Initiation (+) indicates that the tachycardia was initiated by atrial extrastimuli or bursts of atrial pacings. Termination (+) indicates that the tachycardia was terminated by atrial pacings. "Inverse" indicates that the relationship between the premature coupling interval and the echo interval is inverse. While "direct" indicates a direct relationship. Numerical numbers indicate number of patients. Dominators indicate number of patients tried and numerators indicate number of patients in whom the electrophysiological characteristic in question was present. The right upper figure represents the schematic figure of re-entrant circuit in AVNRT. "**" indicates the site of stimulation.

same stimulation protocols were repeated at the right ventricular outflow tract. If VT was not induced with the extrastimulus methods described above, incremental ventricular pacings from cycle lengths of 400 msec to 240 ms for periods of 5 to 30 seconds were performed. The premature coupling interval which induced tachycardia and the echo interval (the interval between the extrastimuli and the first beat of the tachycardia) was measured to determine the relationship between the premature coupling interval and echo interval (inverse vs direct) in patients in whom relatively wide tachycardia zones were recognized during extrastimulus testing.

Next, to examine the effects of pacings on the tachycardia, atrial pacings in cases of WPW tachycardia and AVNRT and ventricular pacings in cases of VT were done during the tachycardia. Pacings were started from the cycle length slightly shorter than that of the tachycardia to the cycle length of 250 msec for duration of 10 seconds. The following effects were determined: 1) the ability to terminate the tachycardia by pacings; 2) the pattern of termination of the tachycardia (abrupt termination or delayed termination); 3) the presence of constant fusion between the pacing and the tachycardia; 4) the presence of overdrive acceleration; 5) the presence of the change of the configuration of QRS wave or P wave; and 6) the presence of overdrive suppression.

In this study, delayed termination was defined as termination of the tachycardia after several beats of the tachycardia following the cessation of pacings. The presence of constant fusion was examined by comparing configurations of P wave (in cases of WPW tachycardia and AVNRT; or QRS waves (in cases of VT) of the tachycardia (depolarization exclusively by the tachycardia) with those of the pacing during normal sinus rhythm (depolarization exclusively by pacing).
VT in OMI

Initiation

Termination

Pacing during the tachycardia

100%

50%

9
11
3
4

8
9

2
3

0
4

0
4

F: Constant Fusion
C: Change of QRS wave
A: Overdrive Acceleration
O: Overdrive Suppression

(+)

Direct Inverse

Delayed Termination

F C A O

0 0 8 8

Fig. 3. Figure 3 shows the electrophysiological characteristics of the initiation and termination of the tachycardia and those in response to ventricular pacings during ventricular tachycardia (VT) in patients with old myocardial infarction (OMI). Initiation (+) indicates that the tachycardia was initiated by ventricular extra-stimuli or bursts of ventricular pacings. Termination (+) indicates that the tachycardia was terminated by ventricular pacings. "Inverse" indicates that the relationship between the premature coupling interval and the echo interval is inverse, while "direct" indicates a direct relationship. Numerical numbers indicate number of patients. Denominators indicate number of patients tried and numerators indicate number of patients in whom the electrophysiological characteristic in question was present. The right upper figure represents the schematic figure of re-entrant circuit of VT in patients with OMI. "*" indicates the site of stimulation.

and with those of pacing during the tachycardia (depolarization partially by pacing and partially by tachycardia: fusion). Constant fusion was determined to be present when fusion between pacing and tachycardia persisted as long as pacings continued. Overdrive suppression was determined to be present when the gradual shortening of cycle length of the tachycardia occurred for at least 3 beats upon termination of pacing. Overdrive acceleration was defined as the shortening of the cycle length of the tachycardia for more than 50 msec upon termination of pacing without change in the QRS and the P wave configurations in the surface electrocardiogram and without change in the atrial and ventricular conduction patterns examined by intracardiac electrogram. The change of QRS configuration was determined by a change in pattern upon termination of pacings which differed from that observed prior to the pacing.

RESULTS

Electrophysiological characteristics of WPW tachycardia

Electrophysiological characteristics of WPW tachycardia are shown in Fig. 1. Atrial extra-stimuli induced the tachycardia in 122 out of 140 patients (85%). Of 122 patients induced by extrastimuli, the relationship between a coupling interval and an echo interval could be determined in 98 patients. An inverse relationship was demonstrated in all of them.

Atrial pacing terminated the tachycardia in all 122 patients in whom the tachycardia was induced during the electrophysiological studies. Delayed termination was recognized in 2 of 27 patients (7%).

Constant fusion was noted in 25 of 34 patients (74%); change of QRS configuration was noted in 3 of 34 patients (9%); overdrive acceler-
VT in DCM

Fig. 4. Figure 4 shows the electrophysiological characteristics of the initiation and termination of the tachycardia and those in response to ventricular pacings during ventricular tachycardia (VT) in patients with dilated cardiomyopathy (DCM). Initiation (+) indicates that the tachycardia was initiated by ventricular extrastimuli or bursts of ventricular pacings. Termination (+) indicates that the tachycardia was terminated by ventricular pacings. "Inverse" indicates that the relationship between the premature coupling interval and the echo interval is inverse, while "direct" indicates a direct relationship. Numerical numbers indicate number of patients. Dominators indicate number of patients tried and numerators indicate number of patients in whom the electrophysiological characteristic in question was present. The right upper figure shows electrophysiological characteristics of WPW tachycardia for comparison. Note that electrophysiological characteristics of VT in DCM are similar to those of WPW tachycardia, which suggests that the mechanism of VT in DCM is re-entry.

atation was recognized in 1 of 34 patients (3%); but overdrive suppression was recognized in none of the 34 patients.

Electrophysiological characteristics of AVNRT

Electrophysiological characteristics of AVNRT are shown in Fig. 2. Atrial extrastimuli induced the tachycardia in 42 of 52 patients (81%). The relationship between the coupling interval and the echo interval could be examined in 41 patients. Thirty seven of them showed an inverse relationship, while none of them showed a direct relationship. Atrial pacing terminated the tachycardia in all 42 patients in whom the tachycardia could be induced during electrophysiological studies. Delayed termination was recognized in 2 of 19 patients (11%). The effects of atrial pacing on the tachycardia could be examined in 19 patients during the electrophysiological studies. Constant fusion, change of QRS configuration, overdrive acceleration, and overdrive suppression were seen in none of them.

Electrophysiological characteristics of VT in patients with OMI

Electrophysiological characteristics of VT in patients with OMI are shown in Fig. 3. Ventricular stimulation induced VT in 9 of 11 patients. In 4 of them, the relationship between the coupling interval and the echo interval could be examined. An inverse relationship was recognized in 3 of them while none of them showed a direct relationship.

Ventricular pacing terminated VT in 8 of 9 patients. Delayed termination was recognized in none.

Constant fusion was seen in 2 of 3 patients, a change of QRS configuration of VT upon
Sustained Idiopathic VT

Fig. 5. Figure 5 shows the electrophysiological characteristics of the initiation and termination of the tachycardia and those in response to pacings during the tachycardia in patients with sustained idiopathic ventricular tachycardia (VT). Initiation (+) indicates that the tachycardia was initiated by ventricular extrastimuli or bursts of ventricular pacings. Termination (+) indicates that the tachycardia was terminated by ventricular pacings. "Inverse" indicates that the relationship between the premature coupling interval and the echo interval is inverse, while "direct" indicates a direct relationship. Numerical numbers indicate number of patients. Dominators indicate number of patients tried and numerators indicate number of patients in whom the electrophysiological characteristic in question was present. The right upper figure shows the electrophysiological characteristics of AVNRT for comparison. Note that AVNRT and sustained idiopathic VT have similar electrophysiological characteristics suggesting the mechanism of these tachycardia is same.

termination of ventricular pacing was noted in 3 of 9 patients, while overdrive acceleration and overdrive suppression were recognized in none of them.

Electrophysiological characteristics of VT in patients with DCM

Electrophysiological characteristics of VT in patients with DCM are shown in Fig. 4. Ventricular stimulation initiated VT in 10 of 12 patients. In 4 of them, the relationship between the coupling interval and echo interval could be determined. An inverse relationship was recognized in 3 of them, while the direct relationship was noted in none of them.

Ventricular pacing terminated VT in 9 of 10 patients. Delayed termination was noted in only 1 of 9 patients.

Ventricular pacing during VT showed that constant fusion was noted in 3 of 6 patients, and that a change of QRS configuration of VT upon termination of pacing was recognized in 3 of 11 patients. Overdrive acceleration and overdrive suppression was recognized in none of 12 patients.

Electrophysiological characteristics of idiopathic sustained VT

Electrophysiological characteristics of idiopathic sustained VT are shown in Fig. 5. Ventricular stimulation induced VT in 13 of 15 patients. The relationship between the coupling interval and the echo interval could be examined in 10 patients. Eight of them showed an inverse relationship while a direct relationship was recognized in none of them.

Ventricular pacing terminated VT in all 13 patients in whom it was tried. Delayed termination was noted in 2 of 13 patients.

The effect of ventricular pacing on the tachy-
Repetitive Idiopathic VT

Fig. 6. Figure 6 shows the electrophysiological characteristics of the initiation and termination of the tachycardia and those in response to ventricular pacings during episodes in patients with repetitive idiopathic ventricular tachycardia (VT). Initiation (+) indicates that the tachycardia was initiated by ventricular extra-stimuli or bursts of ventricular pacings. Termination (+) indicates that the tachycardia was terminated by ventricular pacings. “Inverse” indicates that the relationship between the premature coupling interval and the echo interval is inverse, while “direct” indicates a direct relationship. Numerical numbers indicate number of patients. Dominators indicate number of patients tried and numerators indicate number of patients in whom the electrophysiological characteristics in question was present. The right upper figures show electrophysiological characteristics of AVNRT and WPW tachycardia for comparison. Note that electrophysiological characteristics of repetitive idiopathic VT are quite different from those of AVNRT or WPW tachycardia suggesting the mechanism responsible for repetitive idiopathic VT is different from that of AVNRT or WPW tachycardia.

Cardiac could be examined in 13 patients. Constant fusion, change of QRS configuration of VT upon termination of pacing, overdrive acceleration and overdrive suppression were noted in none of them.

Electrophysiological characteristics of repetitive idiopathic VT

Electrophysiological characteristics of idiopathic repetitive VT are shown in Fig. 6. Ventricular stimulation induced VT in 4 of 28 patients. The relationship between the coupling interval and the echo interval was direct in all 4 patients in whom VT was repeatedly initiated by ventricular premature beats.

In these 4 patients, ventricular pacing repeatedly terminated VT and delayed termination was noted in 2 of them.

In the 4 patients in whom VT was induced during the electrophysiological studies, the effect of ventricular pacing on VT could be examined. None of the 4 patients showed constant fusion, change of QRS configuration, and overdrive suppression, while one of them showed overdrive acceleration.

DISCUSSION

WPW tachycardia is one of a few paroxysmal tachycardias whose mechanism is proved to be reentry and is probably the only paroxysmal tachycardia that satisfies the Mines criteria for re-entry during clinical electrophysiological studies. These criteria are: 1) proof of a re-entrant pathway during the tachycardia and 2) the termination of the tachycardia by inter-
rupturing a part of the re-entrant pathway to prove that it was actively involved during the tachycardia. In WPW tachycardia, the all parts of the re-entrant pathway can be identified by recording multiple intracardiac electrograms during the episode, and it can be proven that all actively participate in the tachycardia by producing a block in the atrioventricular node, the His bundle, or the accessory pathway during the tachycardia. Thus, WPW tachycardia is now believed to be a prototype of re-entry. In the present study, electrophysiological characteristics of re-entry were deduced mainly from the WPW tachycardia model.

Re-entry vs Automaticity

Usually the mechanism of tachycardia is classified into two main groups: tachycardias due to abnormal automaticity and those due to re-entry. Over the years physicians have attempted to differentiate clinically between the two types. The differentiation was based on the belief that tachycardia due to automaticity could not be initiated by single or short burst of premature beats and is suppressed for a period of time after cessation of the overdrive pacing, but eventually increase their rate of firing and return to their previous rate gradually (overdrive suppression). On the other hand, re-entrant tachycardia could be initiated and terminated by a single or short burst of premature depolarization and does not have property of overdrive suppression. In the present study, none of the patients with WPW tachycardia, AVNRT and VT in OMI demonstrated overdrive suppression. Although these considerations help to differentiate re-entrant tachycardia from normal automatic tachycardia, they do not effectively distinguish tachycardia due to triggered activity from re-entrant tachycardia. Electrophysiological characteristics of triggered activity obtained from basic electrophysiological studies showed that triggered activity can also be initiated and terminated by a premature impulse or pacing. Furthermore, it has been reported to undergo overdrive suppression, depending on the duration and rate of pacing.

Recently Waldo et al. suggested other criteria for re-entrant tachycardia based on their observations during pacing. The criteria are: 1) constant fusion between depolarization by pacing and tachycardia; 2) a degree of fusion between pacing and tachycardia increasing as pacing rate increase (progressive fusion); 3) interruption of the tachycardia associated with conduction block in one limb of the re-entrant pathway. They demonstrated these phenomena in cases of WPW tachycardia and proposed the same criteria for establishing the diagnosis of re-entry in other tachycardias.

However, to satisfy all three criteria requires several additional conditions, i.e. 1) to demonstrate progressive fusion, a circuit should have a relatively wide excitable gap zone, 2) to demonstrate that the termination of a tachycardia is associated with the block in one of the re-entrant limb, requires the identification of the re-entrant circuit. Thus, only the presence of constant fusion was examined in the present study.

Constant fusion occurred in 50% to 70% of the patients with WPW, VT in OMI, and VT in DCM, while none in patients with AVNRT nor idiopathic sustained VT. Patients with WPW tachycardia who did not have constant fusion probably had very narrow excitable gap zones, so that even a pacing cycle length slightly shorter than that of the tachycardia terminated the tachycardia. While a different mechanism is responsible for the absence of constant fusion in AVNRT, it is most likely due to the fact that the site of fusion in AVNRT is within the atrioventricular node, which could not be detected by standard electrogram or intracardiac electrogram. The reasons for the presence or absence of constant fusion in other tachycardias remain to be clarified.

Re-entry vs triggered activity

Triggered activity is impulse generated by afterdepolarization which occurs during repolarization (referred to as an early afterdepolarization) or after repolarization is complete or nearly complete (referred to as a delayed afterdepolarization). Delayed afterdepolarizations occur under a number of conditions—one of the most widely recognized causes is digitalis toxicity and another the presence of high concentrations of catecholamines. Also, delayed afterdepolarizations have been found to occur in fibers in the upper pectinate muscle in the rabbit heart, in human atrial myocardium, and in Purkinje fibers surviving on the subendocardial surface of canine infarcted hearts. Recently electrophysiological characteristics of triggered activity obtained from basic electrophysiological studies have been reported. Delayed afterdepolarization which does not reach the threshold may cause triggered activity if the rate
at which the fiber is driven is increased, because the amplitude of the after depolarizations increases as the drive rate increase. A premature impulse may also increase the amplitude of the afterdepolarization of the action potential and initiate triggered activity. Triggered activity can also be terminated by overdrive pacing. Thus, it is difficult to differentiate tachycardia due to triggered activity from that due to reentry just by demonstrate that a premature beat or bursts of pacing initiate or terminate the tachycardia.

However, electrophysiological characteristics of triggered activity obtained from basic electrophysiological studies showed several other characteristics. First, the relationship between the premature coupling interval which initiates tachycardia and the echo interval is usually direct and second, following a period of overdrive, the rate of triggered activity may be faster than it was before overdrive (overdrive acceleration). Third, termination by overdrive stimulation sometimes occurred within several beats after the cessation of overdrive pacing (delayed termination). In the present study, the relationship between premature coupling interval and echo interval showed a direct relationship in none of the patients with WPW tachycardia, AVNRT and VT in OMI. Thus, its presence in a paroxysmal tachycardia suggests that the mechanism of the tachycardia is not re-entry. However, the present studies also showed that other characteristics (overdrive acceleration, delayed termination) may occur in re-entrant tachycardia, as delayed termination and overdrive acceleration were recognized in patients in WPW tachycardia and AVNRT.

Electrophysiological characteristics of WPW tachycardia, AVNRT, and VT in OMI

The present study showed that WPW tachycardia could be initiated and terminated by a premature beat or bursts of pacing in the majority of the patients. Also, the relationship between the premature coupling interval and the echo interval was inverse in the majority of the patients, but a direct relationship was not seen in any of the patients with WPW tachycardia. Thus, these characteristics were probably compatible with re-entry, although they might not be specific for re-entry.

The present study also showed that AVNRT and VT in OMI had similar electrophysiological characteristics to WPW tachycardia. As extrastimuli or pacing induced the paroxysmal tachycardia in 90% of them, and rapid pacing terminated it in almost all patients in whom it was tried. Also none of the patients in whom premature coupling interval and echo interval could be determined showed a direct relationship, while 90% of them showed an inverse relationship. It is noteworthy that in none of the patients with WPW tachycardia, AVNRT and VT in OMI showed overdrive suppression. Thus, clinical electrophysiological studies in the present study also confirmed that the mechanism responsible for these paroxysmal tachycardias (WPW tachycardia, AVNRT, and VT in OMI) is re-entry, as they all had similar electrophysiological characteristics.

However, it should be noted that constant fusion was recognized in none of the patients with AVNRT, while it was demonstrated in 70% of those with WPW tachycardia or VT in OMI. The explanation of the difference of the presence of constant fusion between these re-entrant tachycardias is probably because the site of fusion should be at the atrium or ventricle in order to demonstrate constant fusion by standard electrograms or intracardiac electrogram. As the site of fusion in AVNRT is within the AVN, constant fusion could not be demonstrated.

Electrophysiological characteristics of VT in DCM, sustained idiopathic VT, and repetitive idiopathic VT

In contrast to WPW tachycardia, AVNRT and VT in OMI which were believed to be due to re-entry, the mechanism responsible for VT in DCM and VT in patients without apparent heart diseases are still remained to be solved. But that the electrophysiological characteristics of VT in DCM were similar to those of WPW tachycardia and VT in OMI in the present study suggests that VT in DCM is also due to re-entry. The present studies showed that the electrophysiological characteristics of sustained idiopathic VT resembled to those of AVNRT suggesting it was also due to re-entry. But considering constant fusion was not present in any of the patients observed, its circuit is confined to a relatively small area as in case of AVNRT.

Electrophysiological characteristics of repetitive idiopathic VT were quite different from those of WPW tachycardia, VT in OMI, VT in DCM, and AVNRT. In the majority of these patients, neither pacing nor extrastimuli to
initiated the tachycardia and even in those patients in whom premature beats initiated VT, the relationship between premature interval and echo interval was direct. The fact that patients with repetitive idiopathic VT had quite different electrophysiological characteristics compared with those with WPW tachycardia, AVNRT and VT with OMI suggests that the mechanism responsible for repetitive idiopathic VT was not re-entry.

In conclusion, 1) AVNRT, VT in OMI, VT in DCM, and sustained idiopathic VT had electrophysiological characteristics similar to those of WPW tachycardia, suggesting their mechanisms are re-entry. 2) Repetitive idiopathic VT had quite different electrophysiological characteristics to those of WPW tachycardia, suggesting its mechanism is not re-entry. 3) Overdrive suppression or a direct relationship between coupling intervals and echo intervals were not recognized in any of the patients with WPW tachycardia, AVNRT, VT in OMI, VT in DCM, and sustained idiopathic VT. This suggests that these electrophysiological characteristics are counter to re-entry. 4) Constant fusion was recognized in WPW tachycardia, VT in OMI, or VT in DCM but not in AVNRT or sustained idiopathic VT which suggests that the site or the size of re-entry are important to demonstrate constant fusion. 5) Delayed termination and overdrive acceleration which were noted in triggered activity were also observed in a few patients with WPW tachycardia and AVNRT. This suggests these electrophysiological characteristics are not specific for triggered activity.

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