CONDUCTION THROUGH THE REENTRANT CIRCUIT IN RECURRENT SUSTAINED VENTRICULAR TACHYCARDIA EVALUATED BY USE OF TRANSIENT ENTRAINMENT

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Using transient entrainment, the effect of the paced cycle length on the conduction through the reentrant circuit was assessed in recurrent sustained ventricular tachycardia (VT). Fourteen patients were included in the present study and their VTs were paced at multiple cycle lengths while the criteria of entrainment were confirmed at each paced cycle length. Then, the effect of the paced cycle length upon the conduction time, which was evaluated by the measurement of the time interval from stimulus to the entrained electrogram, was analyzed. In the overdrive pacings of VT, 3 response patterns in conduction time were observed: an increasing pattern ($n=8$), a flat pattern ($n=5$) and a decreasing pattern ($n=1$) while the local conduction time outside the reentrant circuit remained unchanged at comparable paced cycle lengths. A decremental property is the likely mechanism responsible for the paced cycle length-dependent prolongation. As for the flat pattern, the existence of a fully excitable gap may be responsible. A paced cycle length related change in the reentrant circuit may account for the decreasing pattern.

By using transient entrainment, the electrophysiological characteristics of the reentrant circuit can be evaluated and the information so gathered may be valuable in analyzing the action of antiarrhythmic drugs on the slow pathway.

THE mechanism of the most recurrent sustained ventricular tachycardia (VT) which can be initiated by programmed electrical stimulation is believed to be re-entry¹⁻² and either transient entrainment³⁻⁶ or continuous local electrical activity supports the re-entrant mechanism⁷⁻⁸. However, little is known about the property of the reentrant circuit. Only in a limited number of cases has the property of the component of the reentrant circuit been assessed by electrophysiologic studies⁹⁻¹⁰.

Key words:

Ventricular tachycardia
Reentrant circuit
Slow conduction
Entrainment

In this paper, 14 patients were studied because their VT could be induced by electrical stimulation and they were able to undergo the overdrive pacings. Fusion in different degree in the QRS complex was observed at multiple paced cycle lengths, hence both constant fusion and progressive fusion were observed in all patients using first and second criteria of Waldo⁴ and first criterion of Brugada⁶.

The conduction property of the pathway of the re-entrant circuit was evaluated by analyzing the conduction time between the stimulus artifact and the entrained beat.

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TABLE I CLINICAL CHARACTERISTICS OF PATIENTS

No.	Name	Age/Sex	Diagnosis	VT morphology	Origin	PS
1.	M.H.	28/F	post-op T/F	LBBB & N	RVO	RVA
2.	M.Y.	41/F	idiopathic	RBBB & LAD	LVA	RVO
3.	M.W.	47/M	ARVD	LBBB & N	RV & IVS	RVA
4.	T.T.	72/M	OMI	LBBB & LAD	LV	RVA
5.	T.O.	59/F	DCM	RBBB & RAD	LV	RVA
6.	N.O.	52/M	ARVD	LBBB & N	RVO	RVA
7.	H.M.	13/M	post-op T/F	LBBB & LAD	RVO	RVA
8.	T.H.	72/M	OMI	RBBB & N	LV	RVA
9.	S.H.	58/M	ARVD	LBBB & LAD	RVA	RVA
10.	A.Y.	59/M	OMI	LBB & LAD	LV	RVA
11.	S.K.	71/M	Alcoholic CM	LBBB & LAD	LV	RVA
12.	Y.Y.	58/M	DCM	LBBB & LAD	LV	LV
13.	H.M.	16/F	Post-op DORV	LBBB & LAD	IVS	RVO
14.	M.O.	61/M	LV aneurysm*	LBBB & LAD	LV	RVO

PS = pacing site; T/F = tetralogy of Fallot; DORV = double outlet right ventricle; ARVD = arrhythmogenic right ventricular dysplasia; OMI = old myocardial infarction; DCM = dilated cardiomyopathy; LBBB = left bundle branch block; RBBB = right bundle branch block; N = normal axis; LAD = left axis deviation; LV = left ventricle; RVA and RVO = apex and outflow tract of the right ventricle. *: non-ischemic aneurysm of the left ventricle reported in ref.⁵

Fig. 1. Schema of measurement of conduction time during transient entrainment.

During entrainment, the wave front from the pacing site enters the reentrant circuit and emerges at the exit resulting in fusion complex (*). On cessation of the pacing, VT resumes. Then, the conduction time (CT) obtained by measurement of the time interval from the last stimulus artifact to the first non-paced (=entrained) local electrogram at exit as shown by EAS (=earliest activation site). This conduction time include the local conduction time between the pacing site and the entrance.

**: the first non-paced QRS complex.

S-ECG: surface ECG. f.r. and p.r.: fully and partially responsible state from the preceding depolarization, respectively.

SUBJECTS AND METHODS

Of the 70 patients who had recurrent sustained VT, 14 cases were selected for the present study because all fulfilled the criteria of entrainment at multiple paced cycle lengths⁴⁻⁶. Their ages ranged from 13 to 72 years and their clinical characteristics are

presented in Table I. Three patients had old myocardial infarction and their coronary angiograms showed occluded arteries. One male developed VT from a left ventricular aneurysm unrelated to the coronary occlusion but possibly related to myocarditis as reported in another paper!¹ Two patients had received corrective operations for tetralogy

of Fallot and one had been operated on for a double outlet right ventricle. Three patients had VT of right ventricular origin due to

right ventricular dysplasia.¹² One female patient had VT with a right bundle branch block and left axis deviation pattern of the QRS complex which responded to verapamil.^{9,13,14} One patient had alcoholic cardiomyopathy and the 2 remaining cases had idiopathic dilated cardiomyopathy.

The electrophysiologic study.

After written consent was obtained, electrophysiologic studies were performed in the postabsorptive and non-sedated state in the standard manner.¹⁻² The site of VT origin was determined by endocardial mapping and by paced mapping.¹⁵⁻¹⁷ Rectangular electrical stimuli of 2 msec were delivered at twice the diastolic threshold by a programmable stimulator (Fukuda Denshi Co., Cardiac Stimulator BCO2).

When VT was induced, the overdrive pacing was performed for 5 to 10 seconds at a cycle length which was 10-20 msec shorter than the VT cycle length. Then, the paced cycle length was shortened by 10 msec and the overdrive pacing was repeated 5-8 times, or until VT was terminated.

The conduction time through the reentrant circuit.

The conduction property of the reentrant circuit was evaluated by the measurement of conduction time between the stimulus artifact and the entrained electrogram (or alternatively the onset of the entrained QRS complex) as shown in Fig. 1. This conduction time was most clearly obtained by measuring the time interval between the last stimulus artifact and the onset of the first non-paced beat when the overdrive pacings were abruptly terminated.

The characteristics of the conduction time in relation to the intervening tissue outside the reentrant circuit, was evaluated by the measurement of the conduction time from the pacing site to some remote sites when the heart was paced at the comparable rates during the sinus rhythm. There was no isoelectric line between the stimulus artifact and the local response and the latency from the stimulus to the local response was negative in all patients.

Intracavitary electrograms were recorded simultaneously with the surface lead I, II and V1 on an ink-jet mingograph (Siemens-Ele-

Fig. 2. Entrainment of VT with a prolonged interval of stimulus to entrained beat.

VT in case 1 originated at the right ventricular outflow tract. The VT cycle length was 370 msec. The overdrive pacing was performed at cycle length 330 through 290 msec while constant fusion was observed in the QRS complex at each paced cycle length. The interval from stimulus to the entrained local electrogram (measured at the initial deflection) was prolonged from 300 msec to 330 msec. The post-paced return cycles were identical to the paced cycle lengths. VT was terminated by the pacing at 280 msec (the lowest column). I, II, V1: surface leads. S: stimulus. RV: local electrogram from the right ventricle.

Fig. 3. Entrainment of VT with a shortening of stimulus to entrained beat.

Patient no. 14 was diagnosed as having idiopathic left ventricular aneurysm (possibly postmyocarditic) but the coronary arteries were normal (5). The post-paced return cycles were identical to the paced cycle lengths. However, the interval from stimulus to the entrained electrogram shortened from 294 to 278 msec when VT was paced at 330 to 270 msec of cycle length. Abbreviations are same as Fig. 2.

ma Mingograf 82) at the paper speed of 100 mm/sec and they were also stored on magnetic tape (TEAC Cassette Data Recorder XR-5000) and retrieved on a thermal recorder (Fukuda Denshi Co, Thermal Recorder RF-85). The surface 12-lead electrocardiogram (ECG) was always obtained during the induction or pacings of VT.

During this study, disopyramide and a small dose of procainamide was given to 2 patients to control the VT rate when it recurred.

Values were expressed as mean $\pm$ SD.

RESULTS

Induction and entrainment

VT was induced by single ($n=1$) or by double ventricular extrastimuli ($n=13$). The VT cycle length ranged from 250 to 400

msec. Patients who had rapid VT of more than 200 beats per minute complained of palpitation or sometimes light-headedness. Sites of origin of VT were at the right ventricular apex in 1 patient, at the outflow tract of the right ventricle in 3, in the left ventricular free wall in 7, at the apex of the left ventricle in 1, and in the intramural layer of the interventricular septum in 2 patients.

Two patients required DC shock to restore sinus rhythm. One patient had old myocardial infarction in the inferior wall and at each session of the electrophysiologic study, VT degenerated into ventricular fibrillation. In the other patient, all modes of electrical stimulation failed to terminate the induced or spontaneous VT.

The overdrive pacing during VT.

The overdrive pacings were performed pri-

TABLE II VT CYCLE LENGTH, PACED CYCLE LENGTH AND CONDUCTION TIME

Case No.	VTCL	Paced CL	St-entrained electrogram	Dru
1.	340	330 – 280	300 – 360	PA*
2.	330	290 – 260	300 – 360	—
3.	250	230 – 190	375 – 310	—
4.	340	330 – 290	310 – 360	—
5.	400	390 – 290	380 – 420	—
6.	400	290 – 260	335 – 370	—
7.	380	370 – 300	420 – 480	—
8.	375	360 – 310	418 – 440	—
	351 ± 46	324 ± 49 – 273 ± 35	354 ± 47 – 388 ± 510	
9.	310	280 – 250	280 – 270	—
10.	340	320 – 240	380 – 380	—
11.	380	340 – 280	375 – 375	—
12.	320	310 – 290	355 – 350	D
13.	320	280 – 260	330 – 330	—
	(420	410 – 360	320 – 310	+PA 600 mg**)
	342 ± 29	312 ± 25 – 267 ± 18	349 ± 35 – 347 ± 38	
14.	340	330 – 270	294 – 278	—
	(420	380 – 340	310 – 310	+Amio***)

Values were expressed in msec. VTCL = ventricular tachycardia cycle length; D = disopyramide; PA* = procainamide in 750 mg given to control VT rate; PA** = after procainamide of 600 mg IV; Amio*** = amiodarone in 200 mg/day × 7 days following 400 mg/day × 7 days. Other abbreviations are same as Table I.

marily to terminate the induced VT. They were performed from the right ventricle in all but 1 patient. VT of the outflow tract origin of the right ventricle was entrained in 2 patients from the right ventricular apex. VT of the apical origin of the right ventricle was able to be entrained from the ipsilateral apex. VTs of the left ventricular origin were entrained from the right ventricle; from the apex in 5 and from the outflow tract in 2. One VT with left ventricular origin required the pacing from the left ventricle. Constant fusion in the QRS complex was observed in the surface ECG at multiple paced cycle lengths with evidence of progressive fusion at shorter paced cycle lengths (Fig. 2 and 3); therefore, the first and second criteria of transient entrainment were observed.⁴ In 2 patients, the third criterion of entrainment: local conduction block, was also observed.⁴ The post-pacing return cycles were also identical to the paced cycle lengths in 12 cases.⁶

The paced cycle length vs. conduction time through reentrant circuit.

By pacing at multiple cycle lengths, the time interval from stimulus to the entrained local electrogram or alternatively to the onset of the first non-paced QRS complex of VT showed a prolongation (n=8), no change (n=5) or shortening (n=1).

In 8 patients, the interval from stimulus to the local electrogram or to the onset of the first entrained QRS complex (=conduction time) showed a prolongation by 47 ± 11 msec (ranging from 35 to 65 msec) when the paced cycle length was shortened by 30 to 100 msec (Table II and Fig. 2 and 4). In this group, the cycle length of VT was 351 ± 46 msec and the starting paced cycle length was 32 ± 49 msec and the final one was 273 ± 35 msec.

Five patients showed no change in the conduction time as shown in Fig. 4. The cycle length of VT was 342 ± 29 msec which did not differ from that of the former 8 patients. The overdrive pacing was started at a cycle length of 312 ± 25 msec and ended at 267 ± 18 msec (Table II).

One patient who had a left ventricular aneurysm unrelated to coronary occlusion¹¹

Fig. 4. Changes of conduction time through the reentrant circuit.

A: A paced cycle dependent prologation was observed in 8 of the 14 patients (57%). B: A flat pattern was observed in 5 patients, one of whom showed a slight shortening by 5 to 10 msec for multiple paced cycle lengths and was included in this group. The rest showed a shortening as shown in Fig. 3.

showed a shortening in the conduction time (Fig. 3). The patient was later studied while he was taking oral amiodarone but VT was still able to be initiated by the programmed stimulations and the cycle length of VT lengthened from 340 msec to 420 msec. The conduction time again showed a shortening with an increasingly shorter paced cycle length (Table II).

Local conduction time outside the circuit.

When the heart was paced at almost similar cycle lengths as the overdrive pacings, the local conduction time from the pacing site to

some remote sites: from the apex to the out-flow tract of the right ventricle, from the right to the left ventricle, or vice versa, little change was observed (Fig. 5).

Therefore, a change in the local conduction time from the site of pacing to the entrance of the reentrant circuit was not responsible for the change of the conduction time measured from stimulus to the entrained beat (Fig. 1).

DISCUSSION

Recurrent sustained VT, induced and terminated by electrical stimulation, was usually caused by a reentrant mechanism¹⁻² and transient entrainment by the overdrive pacing of VT is best explained by reentry^{3-6,8-10,18-21}

To better understand the electrophysiological characteristics of the reentrant circuit, 14 patients were examined in the present study. In each, both a constant fusion and progressive fusion were observed which are prerequisites for the present analysis (Fig. 1).

In order for fusion to occur, 2 wave fronts have to be present simultaneously during pacing; one is obviously the wave front coming from the pacing site, and the other is from the entrained tachycardia from the exit of the reentrant circuit. The latter is entrained by the preceding pacing. Therefore, when transient entrainment is confirmed, the pacing impulse is certainly conducted into the reentrant circuit and then emerges from the exit of the reentrant circuit. Therefore, the time interval between the stimulus artifact and the entrained electrogram will represent the conduction time between the 2 sites. This conduction time is most clearly measurable at the cessation of the rapid pacing as schematically shown in Fig. 1.

The conduction time measured between the pacing site and the entrained electrogram apparently includes the conduction time through the intervening tissue outside the reentrant circuit. When the time interval from the stimulus artifact to the onset of the QRS complex is measured, an additional conduction time between the exit and the breakthrough point on the epicardial surface would be involved. However, the conduction time outside the reentrant circuit was constant when the heart was paced at compara-

Fig.5. Rapid pacings during sinus rhythm. Local conduction time from the pacing site to the site of the ipsilateral or contralateral ventricle was constant at variable paced cycle lengths. RV, LV: local electrogram from the right and left ventricle respectively. Other abbreviations are same as Fig-2 and -3.

ble paced cycle lengths as shown in Fig. 5. During the overdrive pacings of VT, the conduction time to the site which was activated in an antidromic manner was constant?

With an increase in the frequency of pacings, it is well known that the action potential duration shortens²² and this may facilitate the conduction through the intervening normal tissue between the pacing site and the entrance of the reentrant circuit. This fact may be the reason for the widening of the excitatory gap when the second premature stimulus was applied following the first premature stimulus in the resetting study of VT²⁰. Therefore, if the conduction time from the pacing site to the entrained electrogram changes, the main site of the change must be within the reentrant circuit.

An increasing pattern (=prolongation) was most common in the present study (8/14)

and the conduction time was prolonged by 47 ± 11 msec when the paced cycle length was shortened from 324 ± 49 msec to 273 ± 35 msec. From the above discussion, the main site of delay must occur within the circuit.

In 3 patients with VT of idiopathic etiology, Okumura et al. observed a rate-dependent prolongation in the time interval from stimulus to the earliest activation site. They postulated that this prolongation was caused by the decremental property of the slow pathway of the reentrant circuit⁹.

Given single or double ventricular extrastimuli which reset the VT, Almendal et al.²⁰ observed 3 patterns of responses in the first post-pacing return cycle to the coupling intervals of the extrastimuli. As to the mechanisms of this increasing pattern, it was postulated that the wave front encounters relatively refractory tissue within the reentrant

circuit, or at the vicinity of the circuit, or the site of entrance varies depending on the coupling interval resulting in a longer pathway for the impulse to travel.

In the cases which showed the paced-rate dependent prolongation of the conduction time, similar results were observed when VT was paced during mexiletine therapy.²³ If mexiletine depresses the conduction velocity and shortens the action potential duration of the tissue within the reentrant circuit,²⁴ the wave length (=action potential duration x conduction velocity) will shorten.²⁵ As long as the length of the reentrant circuit is fixed, a shorter wave length will allow for the tissue of the reentrant circuit to recover which will then improve the conduction if the paced cycle length-dependent prolongation of the conduction time is due to the relative refractory period. However, this was not the case in our study and a prolongation was observed in the conduction time even after mexiletine therapy.

A decremental property is the likely mechanism for the paced cycle length-dependent prolongation of the conduction time through the reentrant circuit. However, more precise electrophysiological and experimental information is required to obtain a final conclusion.^{26,27} A Wenckebach type exit block during VT is an example of the complexity of the reentrant circuit and its connection with the surrounding normal tissue.²⁸

Unless a complex compensatory mechanism is operating within the reentrant circuit, the flat pattern of the conduction time in the present study, or the return cycle by Almendral et al²⁰ must mean that the reentrant circuit contains fully responsive tissue to which the paced impulse encounters within the circuit.

One patient showed a shortening in the conduction time as the paced cycle length shortened (Fig. 3). A change in the site of entrance, or change in the pathway within the circuit may be responsible.

In all patients studied, the configuration of the first non-paced QRS complex was identical to that of VT which was confirmed by 6-12 channel ECG. From these findings, it is assumed that the exit of the reentrant circuit is spatially fixed.

As long as the change in the local conduc-

tion time outside the reentrant circuit is negligible, the change in the conduction time determined by the present method would represent the conduction property of the slow pathway. Ideally, we should pace at the entrance and record at the exit of the reentrant circuit to obtain precise data. However, it is not possible to place electrode catheters at the site of entrance and the exit of the reentrant circuit in every case. More detailed data will be obtained during the intraoperative mappings and will contribute to the understanding of the electrophysiological characteristics of the reentrant circuit.

Finally, it is not known if it is possible to predict the efficacy of antiarrhythmic drugs by understanding the conduction property while using overdrive pacing. Among the 14 patients in the present study, 6 VTs responded to drugs but they belonged to groups which showed a prolongation, or no change in the conduction time.

However, knowledge concerning the electrophysiological characteristics of the reentrant circuit might provide a basis for a rational pharmacological approach since the drug selection on a electrophysiological basis has been tried in an empirical manner and has attained only a limited success rate of 50% or less.²⁹⁻³²

CONCLUSION

VT which was induced and terminated by electrical stimulation and also entrained by overdrive pacing, has reentry as the causal mechanism. Using the technique of entrainment, the conduction property through the reentrant circuit was analyzed at multiple paced cycle lengths and different responses in the conduction time through the reentrant circuit were observed. In 57% of patients, a decremental property of the slow pathway was suggested, but more detailed data from the inside of the reentrant circuit will be helpful to understand the mechanism of the paced cycle length-dependent prolongation of the conduction time as well of other response patterns.

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