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Electrophysiologic Characteristics of Electrically Induced Nonsustained Ventricular Tachycardia in the Late Stage of Canine Myocardial Infarction

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SUMMARY: To examine electrophysiologic characteristics of electrically induced nonsustained ventricular tachycardia (NSVT), open chest electrophysiologic study was performed on 21 dogs with 25-day-old anteroapical myocardial infarction. Six of the 21 dogs had reproducibly inducible NSVT and five had reproducibly inducible sustained ventricular tachycardia (SuVT) in response to programmed ventricular stimulation. Remaining 10 dogs had no reproducibly inducible ventricular tachycardia or ventricular fibrillation (VF) in response to programmed ventricular stimulation. Twelve NSVTs in the 6 dogs were characterized by beat-to-beat variations of cycle length and QRS morphology of surface lead and also by wide disparity of local activation of the left ventricle, whereas 10 SuVTs in the five dogs did not show these electrophysiologic characteristics except during the first three to four beats. Three of the 12 NSVTs transformed into VF. Cycle lengths of these three NSVTs showed progressive shortening before transformation into VF. None of the 10 SuVTs transformed into VF. Continuous disorganized electrical activity was recorded on endocardial electrograms of the left ventricle in four of the 12 NSVTs, but not in the 10 SuVTs. It is concluded that in the late stage of canine myocardial infarction nonsustained ventricular tachycardia is an unstable ventricular tachyarrhythmia with some differences in electrophysiologic manifestation from sustained ventricular tachycardia.

INTRODUCTION

Ventricular arrhythmias are the most important cause of sudden cardiac death in patients with acute or old myocardial infarction^{1, 5)}. Reliable and effective therapies are expected to be developed for the prevention of sudden cardiac death due to ventricular arrhythmias. In order to make a progress in this field, better understanding of the electrophysiologic characteristics and mechanisms underlying the ventricular arrhythmia in myocardial infarction is

necessary. Many reports are available regarding electrophysiologic mechanisms of ventricular arrhythmia during acute myocardial ischemia^{6, 9)} but there are very few regarding ventricular arrhythmias in the late stage of myocardial infarction^{10, 12)}. It has been shown experimentally that reentry in small anatomic pathways might be the underlying mechanism of sustained ventricular tachycardia (SuVT) in the late stage of myocardial infarction¹⁰⁾. However, electrophysiologic characteristics and underlying mechanisms of nonsustained ventricular tachycardia (NSVT) in the late stage

of myocardial infarction have not been fully investigated. The present study was designed to elucidate the electrophysiologic characteristics of NSVT in the late stage of canine myocardial infarction created by ligation of the left anterior descending coronary artery. NSVT and SuVT were induced by programmed ventricular stimulation and electrophysiologic features of the two types of ventricular tachycardia were compared each other.

MATERIALS AND METHODS

Creation of myocardial infarction

Twenty three mongrel dogs of either sex, weighing eight to 12 kg, were anesthetized with intravenous sodium pentobarbital (25 mg/kg) and ventilated with room air through a tracheal tube using a Harvard respirator. Surgery was performed under proper aseptic conditions. A left lateral thoracotomy was performed in the fifth intercostal space and the heart was exposed and placed in the pericardial cradle. Anteroapical myocardial infarction was created by ligation of the left anterior descending coronary artery just distal to the first diagonal branch. The chest was closed 60 minutes after the ligation of the left anterior descending coronary artery and the dogs were allowed to recover.

Open chest electrophysiologic study in the late stage of myocardial infarction

An average of 25.4 ± 6.1 days after initial surgery the dogs were anesthetized with intravenous sodium pentobarbital (25 mg/kg) and ventilated with room air through a tracheal tube using a Harvard respirator. The heart was approached through a median sternotomy. Systemic arterial pressure was measured with a catheter introduced into the right femoral artery. Pressure measurements were obtained with Statham P32Db pressure transducer and displayed on an oscillograph (Fukuda Denshi, Monitorscope CS-800). Teflon-coated stainless steel plunge wires (0.15 mm in diameter) were used to record electrical activity of the left ventricular endocardium and to stimulate the left ventricular endocardium. The teflon coat was removed from the tip of the wires exposing

approximately two mm at the tip of each wire and wires were positioned in the endocardium of the left ventricle using 22 gauge hypodermic needles. The points of insertion of two wires constituting a pair (bipolar electrode) were approximately three mm apart. Six pairs of bipolar plunge electrodes were used for the study; two pairs were positioned in the noninfarcted basal anterolateral region of the left ventricle, two pairs in the anteroapical infarct zone and remaining two pairs around the margin of the infarct zone. Their positions were confirmed by postmortem examination of the heart after completion of the electrophysiologic study. The bipolar plunge electrodes were connected through a switch box to a programmable electrical stimulator (Fukuda Denshi, Cardiac Stimulator BC-02A), an oscillograph (Fukuda Denshi, Monitorscope CS-800) and a multichannel recorder (Siemens, Mingograf 800). The surface electrocardiogram and endocardial electrogram of the left ventricle were recorded simultaneously. The input from the bipolar plunge electrodes was filtered through amplifiers at 30 to 500 Hz. The electrophysiologic data were stored on FM magnetic tape (Sony, Instrumentation Taperecorder, UN930) for later retrieval and analysis, and were also recorded on a multichannel recorder at a paper speed of 100 or 200 mm/sec with identical gain settings. Arterial pressure was monitored continuously throughout the electrophysiologic study.

Programmed ventricular stimulation

Programmed electrical stimulation at six sites of the left ventricular endocardium was performed with a rectangular pulse of two msec duration and stimulus strength was kept at twice the diastolic threshold throughout the electrophysiologic study. Premature ventricular stimuli of increasing prematurity (S2) were given after ventricular pacing of eight beats with cycle length of 250 to 300 msec until the stimulus failed to capture the ventricle. Then, a second premature stimulus (S3) was applied at varying S1-S2 interval and S2-S3 interval was progressively shortened by pitches of 10 msec until S3 failed to capture the ventricle. After completion of this procedure, brief bursts (three

to five beats) of rapid ventricular pacing were introduced through each pair of bipolar electrode. Cycle length of brief burst of rapid ventricular pacing was shortened by pitches of 10 msec until the stimuli failed to achieve 1 to 1 ventricular capture and ventricular fibrillation (VF) occurred. If ventricular stimulation from one site of the left ventricle induced any form of ventricular tachycardia or VF, an identical mode of ventricular stimulation was repeated at the same site to demonstrate the reproducibility of electrical induction of ventricular tachycardia. Electrically induced SuVT was terminated with a brief burst of rapid ventricular pacing. Direct current countershock (10 to 20 joules) was applied to the epicardium of the ventricles to terminate VF by using a defibrillator (Fukuda Denshi, Model FC-600).

NSVT was defined as a run of four or more successive beats in response to any form of ventricular stimulation, which reverted spontaneously to sinus rhythm within 30 seconds. SuVT was defined as a ventricular tachycardia which persisted for at least 30 seconds or, in the event of hemodynamic compromise, required a pacing technique for termination. Finally, VF was defined as a ventricular arrhythmia with nonuniform morphology of the ventricular electrograms and an extremely short and variable cycle length.

After completion of the electrophysiologic study, each dog was sacrificed and the heart was excised. Then, the heart was stained with triphenyltetrazolium chloride (TTC) and cut transversely into slices of about five mm thickness. The percentage of infarcted myocardium was determined from the size of the infarcted tissue in each slice by the planimetric method. The slices were preserved in 10 % formalin.

Data are presented as the mean \pm SEM. Statistical analysis was performed by two sided unpaired t test and chi-square test, as appropriate. Values were considered significant at the level of $p < 0.05$.

RESULTS

Two of the 23 dogs died soon after creation of myocardial infarction. The remaining 21 dogs survived and open chest electrophysiologic

study was performed. All of the 21 dogs were in normal sinus rhythm at the beginning of the electrophysiologic study. They had a mean heart rate of 112 ± 14 beats/min and mean arterial pressure of 96 ± 4 mmHg.

Incidence of electrically induced ventricular arrhythmias

As shown in **Table 1**, eight of the 21 dogs did not develop any form of ventricular arrhythmias in response to programmed ventricular stimulation. Nonreproducible NSVT was induced in two of the 21 dogs. Reproducibly inducible ventricular tachycardias such as NSVT and SuVT were observed in 11 (52.3%) of the 21 dogs. Six of these 11 dogs had reproducible NSVT. Four of these six dogs also had electrically induced VF, but none of these dogs had electrically induced SuVT. Remaining five of the 11 dogs had reproducibly inducible SuVT.

Electrophysiologic characteristics of electrically induced ventricular arrhythmias

To examine electrophysiologic characteristics of electrically induced ventricular arrhythmias we selected reproducibly induced NSVT and SuVT from each dog. Thus, 12 NSVTs from the six dogs and 10 SuVTs from the five dogs were used for electrophysiologic analysis.

Figure 1 shows two examples of electrically induced NSVT in a dog. Upper panel (A) shows NSVT induced by double premature stimuli. This NSVT reverted spontaneously to sinus rhythm after seven successive beats. Cycle length of this ventricular tachycardia and QRS morphology of surface lead show marked variations during the entire period of ventricular tachycardia. Morphology of endocardial electrograms during the ventricular tachycardia also shows marked beat-to-beat variation. Furthermore, continuous disorganized electrical activities are recorded transiently on endocardial leads (INFARCT I and II) in the infarct zone during the ventricular tachycardia. Lower panel (B) shows another example of NSVT induced with the identical mode of ventricular stimulation. This NSVT transformed into VF during the course of ventricular tachycardia. QRS morphology of surface lead, cycle length of ventricular tachycardia and morphology of

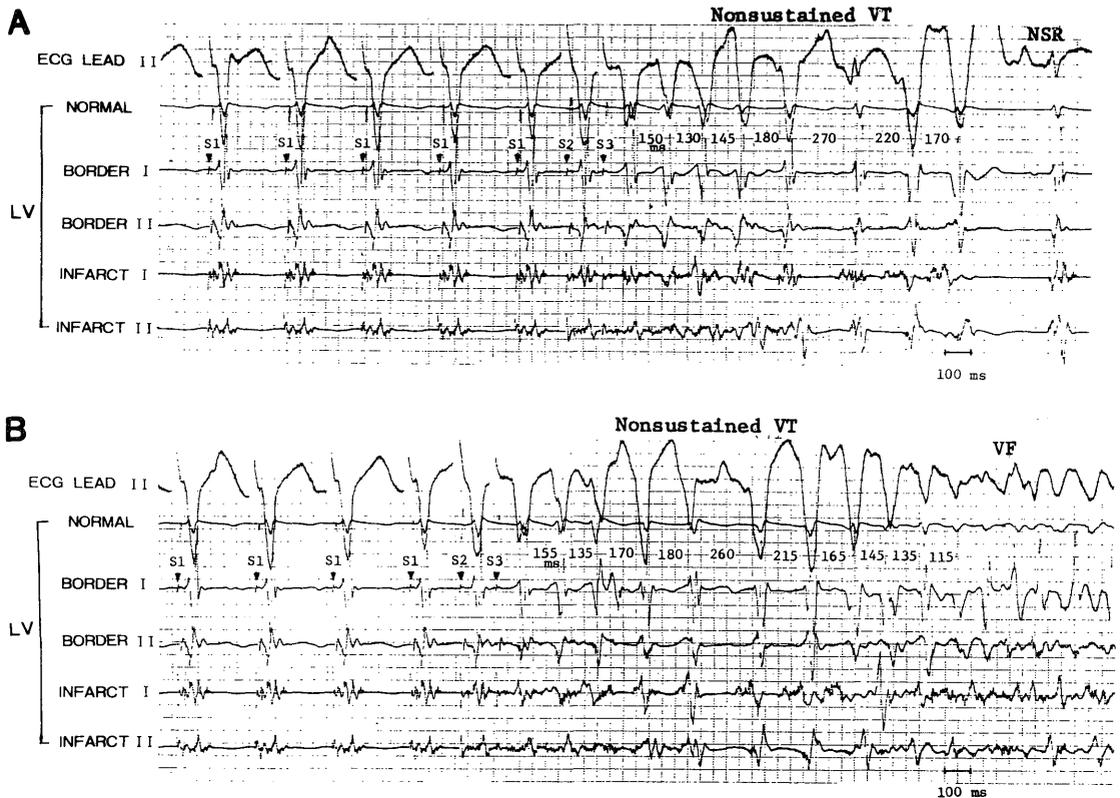


Fig. 1. A: Nonsustained ventricular tachycardia induced by double premature ventricular stimuli (S2-S3) during fixed rate ventricular pacing (S1) at a cycle length of 300 msec in a dog with old experimental myocardial infarction. The ventricular tachycardia reverted spontaneously to normal sinus rhythm (NSR) after seven consecutive beats.

B: Another nonsustained ventricular tachycardia induced by the same mode of programmed ventricular stimulation in the same dog. Electrically induced ventricular tachycardia transformed into ventricular fibrillation (VF) during its course.

LV=left ventricle, VT=ventricular tachycardia, ECG lead II=electrocardiographic lead II, NORMAL indicates the noninfarcted basal anterolateral region of the left ventricle, BORDER I and II the margin of the anteroapical infarct zone, and INFARCT I and II the anteroapical infarct zone. See text for details.

endocardial electrograms immediately before transformation into VF show considerable beat-to-beat variations. Continuous disorganized electrical activity was also recorded on endocardial leads (INFARCT I and II) in the infarct zone during the first two to four beats of the ventricular tachycardia and then disappeared. This continuous disorganized electrical activity reappeared first on an endocardial lead (INFARCT I) in the infarct zone and then spread over other endocardial leads with transformation of the ventricular tachycardia into VF. Remaining 10 electrically induced NSVTs also

showed similar electrophysiologic features. **Figure 2** shows a representative example of SuVT. QRS morphology of surface lead and cycle length of ventricular tachycardia show only small variation except the first two beats of the ventricular tachycardia. Endocardial electrograms have monomorphic configuration in four leads except in a lead (INFARCT II) in the infarct zone from which electrogram shows polymorphic configuration without continuous disorganized electrical activity. Remaining 9 electrically induced SuTVs showed electrophysiologic features similar to those of this

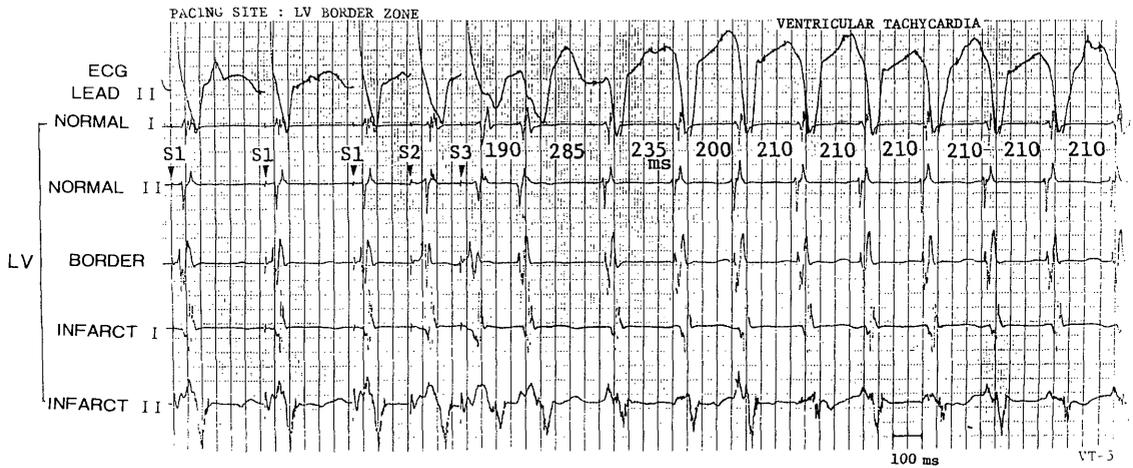


Fig. 2. Sustained ventricular tachycardia induced by double premature ventricular stimuli (S2-S3) during fixed rate ventricular pacing (S1) at a cycle length of 300 msec in a dog with old experimental myocardial infarction. Unidentified abbreviations as in Fig. 1. See text for details.

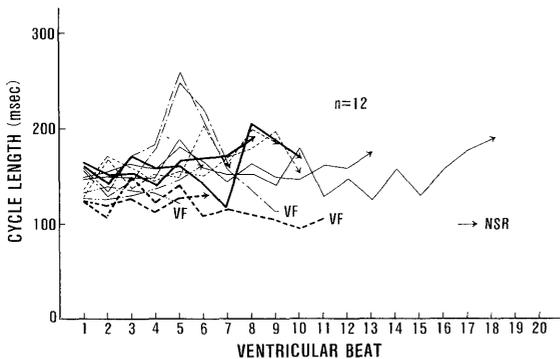


Fig. 3. Cycle length of nonsustained ventricular tachycardia (NSVT) reproducibly induced by programmed ventricular stimulation in six dogs with old experimental myocardial infarction. Cycle lengths of NSVT in the same dog are shown with lines of the same type. Duration of NSVT ranged from five to 18 beats. Nine of 12 NSVTs reverted spontaneously to normal sinus rhythm (NSR) and remaining three transformed into ventricular fibrillation (VF). See text for details.

example. **Figure 3** shows cycle length of electrically induced NSVTs. Duration of electrically induced NSVT varied markedly and ranged from five to 18 beats. Nine of the 12 electrically induced NSVTs reverted spontaneously to sinus rhythm after six to 18 consecutive beats and remaining three electrically induced NSVTs transformed into VF within five

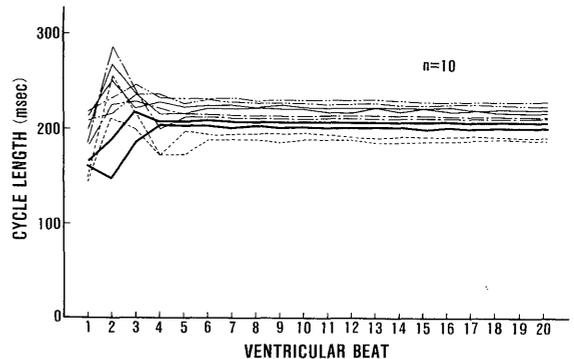


Fig. 4. Cycle length of the first 20 beats of sustained ventricular tachycardia (SuVT) reproducibly induced by programmed ventricular stimulation in five dogs with old experimental myocardial infarction. Cycle lengths of the reproducibly induced ventricular tachycardia in the same dog are shown with lines of the same type. See text for details.

to 11 beats. Cycle length of electrically induced NSVT was considerably short and had marked beat-to-beat variation. Cycle lengths of the three electrically induced NSVTs which transformed into VF were particularly short and progressively shortened immediately before transformation into VF. **Figure 4** shows cycle lengths of the first 20 beats of electrically induced SuVT. In all of the 10 electrically induced SuVTs cycle length of ventricular tachycardia showed small variation except the first three to four beats.

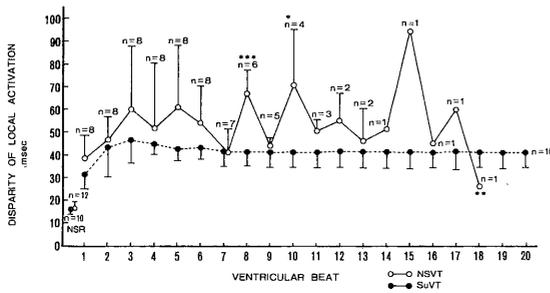


Fig. 5. Disparity of local activation of the left ventricle during normal sinus rhythm (NSR) immediately before induction of NSVT and SuVT and during reproducibly induced NSVT (open circle) and SuVT (filled circle). NSVT=nonsustained ventricular tachycardia, SuVT=sustained ventricular tachycardia. Significance of difference between NSVT and SuVT groups at each respective beat is indicated as $p < 0.05^{*}$, and $p < 0.001^{***}$. No comparison was made after the 11th beat because the number of variables in the NSVT group was too small. See text for details.

Continuous disorganized electrical activity was recorded on at least one of the endocardial leads in four of the 12 electrically induced NSVTs and in none of the 10 electrically induced SuVTs. Three NSVTs which transformed into VF had continuous disorganized electrical activity on an endocardial lead spreading over all of the other leads.

Disparity of local activation of the left ventricle was estimated during electrically induced NSVT and SuVT. Disparity of local activation was defined as the time difference between the earliest and latest onsets of local activation in six sites of the left ventricle. Ventricular tachycardia with continuous disorganized electrical activity was excluded from the estimation of disparity of local activation because the onset of local activation could not be determined in that case. The data are summarized in **Figure 5**. Disparity of local activation during normal sinus rhythm immediately before electrical induction of NSVT or SuVT was 16.4 ± 3.2 msec

Table 1. Electrically induced ventricular arrhythmias, age of myocardial infarction, the size of myocardial infarction and the weight of left ventricle.

Dog	Reproducible VT	Days post-MI	MI size (% total LV mass)	Weight of LV (g)
1	NSVT	12	16.9	46.2
2	SuVT	24	18.6	52.5
3	NSVT	19	14.9	42.3
4	(-)	21	9.1	45.7
5	SuVT	28	16.7	47.4
6	(-)	15	6.6	37.7
7	(-)	21	7.6	38.0
8	SuVT	21	13.2	67.6
9	(-)	28	9.8	55.8
10	NSVT	26	11.7	55.1
11	(-)	28	7.6	52.4
12	NSVT	28	12.9	53.2
13	(-)	34	5.1	35.5
14	SuVT	41	22.7	34.8
15	(-)	29	6.8	38.5
16	NSVT	28	12.9	48.2
17	SuVT	21	13.6	53.4
18	(-)	28	11.3	42.3
19	NSVT	27	16.2	38.5
20	(-)	28	8.6	41.5
21	(-)	26	9.4	36.4
Mean \pm SEM		25.4 \pm 6.1	12.0 \pm 4.4	45.8 \pm 8.3

VT=ventricular tachycardia, MI=myocardial infarction, LV=left ventricle, NSVT=nonsustained ventricular tachycardia, SuVT=sustained ventricular tachycardia.

in the 12 electrically induced NSVTs and 15.8 ± 2.4 msec in the 10 electrically induced SuVTs. There was no significant difference between the two values. As shown in **Figure 5**, disparity of local activation during electrically induced NSVT and SuVT was greater than that during normal sinus rhythm. Disparity of local activation during electrically induced NSVT had greater beat-to-beat variation and magnitude as compared to that during electrically induced SuVT.

Postmortem examination of the heart in all of the 21 dogs showed transmural anteroapical infarction. Infarct size of each dog is listed in **Table 1**. Average infarct size in all of the 21 dogs was 12.0 ± 4.4 (5.1 to 22.7) percent of the total ventricular mass. Average infarct size of the three subgroups was 14.3 ± 1.9 (11.7 to 16.9) per cent in the six dogs with reproducibly inducible NSVT, 16.9 ± 3.5 (13.2 to 22.7) per cent in the five dogs with reproducibly inducible SuVT and 8.2 ± 1.7 (5.1 to 11.3) per cent in the 10 dogs without reproducibly inducible NSVT or SuVT. There was no significant difference in the infarct size between the NSVT and SuVT groups. However, infarct size in these two subgroups was significantly greater than that in the 10 dogs without reproducible NSVT or SuVT ($p < 0.01$).

DISCUSSION

In contrast to acute myocardial ischemia, only limited observations on electrophysiologic features of the several week old experimental myocardial infarction have been reported¹⁰. The present study describes the electrophysiologic characteristics observed during open chest electrophysiologic study in dogs with 25-day-old transmural anteroapical infarction. Fifty-two per cent of the 21 dogs in the present study manifested reproducibly inducible ventricular tachyarrhythmias in response to programmed ventricular stimulation. Nonsustained ventricular tachycardia was induced reproducibly in six dogs and sustained ventricular tachycardia was also induced reproducibly in five dogs.

In the dog model of the present study, several major characteristics of nonsustained ventric-

ular tachycardia that distinguished those from sustained ventricular tachycardia were demonstrated as follows: 1) Nonsustained ventricular tachycardia had marked beat-to-beat variations of its cycle length and QRS morphology of surface lead, whereas sustained ventricular tachycardia showed much less variations of them except during the first three to four beats. 2) Nonsustained ventricular tachycardia terminated spontaneously or transformed into ventricular fibrillation within five to 18 beats of its onset. Cycle length of nonsustained ventricular tachycardia in association with transformation into ventricular fibrillation shortened progressively until the onset of ventricular fibrillation. In contrast, none of sustained ventricular tachycardia transformed into ventricular fibrillation. 3) Nonsustained ventricular tachycardia had marked beat-to-beat variation and greater magnitude of disparity of local activation of the left ventricle, whereas sustained ventricular tachycardia had small variation and magnitude of disparity of local activation. 4) Continuous disorganized electrical activity was recorded in four nonsustained ventricular tachycardias and in none of sustained ventricular tachycardia. Three of these four nonsustained ventricular tachycardia having continuous disorganized electrical activity transformed into ventricular fibrillation.

Although electrophysiologic mechanisms underlying nonsustained and sustained ventricular tachycardias described in the present study are not known with certainty, the ability to reproducibly initiate and terminate ventricular tachycardia by programmed ventricular stimulation and the appearance of continuous disorganized electrical activity during nonsustained ventricular tachycardia in our dog model suggest the presence of reentry mechanism^{11, 12}. A single reentrant circuit with a single exit site seems to be likely as the electrophysiologic mechanism underlying sustained ventricular tachycardia with small variations of its cycle length and QRS morphology of surface lead. In contrast, there seems to be several possibilities of underlying electrophysiologic mechanism which are responsible for variable cycle length of ventricular tachycardia, polymorphic QRS of surface lead and wide

disparity of local activation of the left ventricle during nonsustained ventricular tachycardia in our dog model. One possibility is beat-to-beat variation of the size of reentrant circuit and conduction velocity in the reentrant circuit^{12, 14}. Another possibility is the existence of multiple circuits of reentry with variable exit sites and ventricular activation pattern^{15, 16}. Further investigation is required to clarify the exact mechanism underlying nonsustained ventricular tachycardia. As for transformation of nonsustained ventricular tachycardia into ventricular fibrillation, our findings are consistent with the findings of previous study of Fujimoto *et al* who demonstrated that rate of ischemia induced spontaneous ventricular tachycardia accelerates prior to the onset of ventricular fibrillation¹⁷.

In conclusion, nonsustained ventricular tachycardia in the late stage of experimental myocardial infarction has several electrophysiologic characteristics different from those of sustained ventricular tachycardia and is an unstable ventricular tachyarrhythmia having a propensity to transform into ventricular fibrillation. It is anticipated that further investigation of the underlying mechanism of nonsustained ventricular tachycardia will shed light on the electrophysiologic mechanisms responsible for the development of ventricular fibrillation and sudden cardiac death in the clinical settings of old myocardial infarction.

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