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Endocardial Substrate Mapping for Monomorphic Ventricular Tachycardia Ablation in Ischemic and Non-Ischemic Cardiomyopathy

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We investigated the differences in the endocardial substrates between ischemic cardiomyopathy (ICM) and non-ICM (NICM) by using electro-anatomical mapping and pace-mapping. We studied 18 patients (ICM and NICM, 9 each) with monomorphic ventricular tachycardia (VT) documented by 12-leads ECG. Low voltage area was defined by signal amplitude <1.5 mV. A pace-map QRS morphology that matched VT in >10 of the 12-leads ECG was regarded as a pace-map match. And conduction delay during pace-mapping was defined as the stimulus to QRS interval ≥40 ms. Low voltage area was 53.8 ± 21.5 and 20.8 ± 16.7 cm^2 in ICM and NICM patients, respectively (P = 0.002). Pace-mapping was assessed in 6 ICM and 9 NICM. Pace-map match with conduction delay were obtained in all the 6 ICM patients. But in NICM patients, pace-map match with conduction delay was obtained in 3 patients. Pace-map match sites where conduction delay was not observed were obtained in 5 patients. Pace-map match could not be obtained in 1 patient. We attempted ablation in 6 ICM and 7 NICM patients. Subsequently, VT recurrence was not observed in ICM but it was observed in 6 of 7 NICM patients (log-rank P = 0.0016). In NICM patients, the arrhythmogenic substrate that represented the abnormal electrogram and conduction delay was observed less within the endocardial surface when compared with that observed in ICM. VT recurrence rate subsequent to endocardial ablation was higher in NICM than in ICM patients.

Sustained monomorphic ventricular tachycardia (VT) in patients with ischemic cardiomyopathy (ICM) is often associated with areas of infarction-related scar and border zone(14, 20, 28); using substrate mapping, the reentry circuits of VT can be ablated from the endocardium(13, 28). However reentry within the myocardium is the most common cause in non-ICM (NICM). Identifying the VT circuits on the endocardial surface in NICM is more difficult. Generally, conventional endocardial catheter ablation is reportedly more difficult to perform in NICM than in ICM patients(19, 25). An epicardial approach has been attempted in NICM cases that cannot be ablated using the endocardial approach(25, 27). However, the

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epicardial mapping procedure is complicated, and the indications require careful consideration.

Implantable cardioverter defibrillator (ICD) therapy reduces mortality in patients with sustained VT and structural heart disease(1, 5, 11). However, electrical storm after ICD implantation remains a major concern (4, 12, 16). And at least 50% of the patients with ICD received concomitant anti-arrhythmic drug therapy to decrease the frequency of episodes of ventricular arrhythmia(24).

The purpose of this study was to assess the differences with regard to the endocardial characteristics between ICM and NICM patients by using 3-dimensional (3D) voltage mapping and pace-mapping. Additionally, this study aimed to evaluate the efficacy of endocardial catheter ablation, as an adjuvant therapy to ICD, for clinically documented monomorphic VT.

MATERIAL AND METHODS

Patients (**Table I**)**.** Subjects comprised 18 consecutive patients (16 men and 2 women; mean age, 69 ± 12 years) with monomorphic VT referred for VT ablation between March 2003 and February 2007. ICM was observed in 9 patients (8 men and 1 woman; mean age, 69 ± 14 years; mean ejection fraction (EF), 33 ± 9%). All ICM patients experienced remote (>2 months before procedure) myocardial infarction; anterior infarction was observed in 6 patients; inferior or posterior infarction, in 2 patients; and anterior and inferior infarction, in 1 patient. NICM was observed in 9 patients (8 men and 1 woman; mean age, 69 ± 9 years; mean EF, 33 ± 8%). None of the NICM patients had a history of myocardial infarction, electrocardiographic (ECG) evidence of myocardial infarction, and coronary stenosis >50% diameter.

Of these, 5 patients were treated with an ICD prior to the ablation and had suffered from multiple VT episodes that required ICD interventions (102 ± 71 times per patient).

For the remaining 13 patients, ICD implantation was planned irrespective of the results of the ablation. Ablation was attempted because of frequent VTs in 10 patients despite the use of oral anti-arrhythmic drugs. Of these 10 patients, 6 required intravenous infusion of anti-arrhythmic drugs. Although VT episodes were not very frequent prior to ablation in 3 patients, ablation was attempted before ICD implantation because the patients refused to follow long-term anti-arrhythmic treatment.

All anti-arrhythmic medications with the exception of amiodarone were discontinued at least 5 half-lives before the study. Written informed consent was obtained from each patient before the procedure.

Electro-anatomical mapping. Systemic anticoagulation with heparin was adjusted to an activated clotting time >250 seconds. Left ventricular electro-anatomical mapping (CARTO; Biosense Webster, Diamond Bar, California) was performed in order to identify the endocardial voltage map during sinus or right ventricular pacing. Access to the left ventricle was achieved by using a retrograde and trans-aortic approach. Ventricular mapping was performed with a 7-Fr steerable catheter and using a 4-mm distal tip electrode and 2-mm ring electrode; the inter-electrode distance of 4 mm (Cordis-Webster, Diamond Bar, California). Bipolar electrograms filtered at 30-400 Hz were recorded on the electro-anatomical mapping system and a separate digital system (Prucka Engineering, Houston, Texas) with filtering at 30–500 Hz. The peak-to-peak signal amplitude of the bipolar electrogram was measured automatically.

Table I. Patient Characteristics-1

Patient	Gender	Age (years)	Heart Disease (MI site)	LVDd (mm)	EF (%)	Clinical VT-CL (ms)	Medication before Ablation
1	Male	80	ICM (ant.)	62	29	305	BB
2	Male	79	ICM (ant.)	70	30	340	A+BB+AM
3	Male	63	ICM (ant.)	63	32	280	A+BB+AM
4	Male	68	ICM (ant.)	63	23	320	None
5	Female	36	ICM (inf.)	52	52	280	None
6	Male	77	ICM (inf.)	56	34	330	A+BB
7	Male	71	ICM (ant.)	61	41	250	A+BB+AM
8	Male	63	ICM (ant.inf.)	65	28	300	A+BB+AM
9	Male	84	ICM (ant.)	62	26	380-400	A+BB+SOT
10	Male	53	NICM	69	34	390-430	A+BB+AM
11	Male	75	NICM	68	33	460-480	A+BB+AM
12	Male	73	NICM	63	15	370-410	A+BB+AM
13	Female	61	NICM	58	42	300-340	BB
14	Male	64	NICM	66	38	300	A+AM
15	Male	72	NICM	66	36	320	A+BB
16	Male	66	NICM	72	28	320	A+BB+AM
17	Male	86	NICM	58	41	370	A+BB+AM
18	Male	69	NICM	62	26	340-390	A+BB+SOT
mean ± SD		69 ± 12		63 ± 5	32 ± 9		

A indicates angiotensin converting enzyme inhibitor or angiotensin II receptor blocker; AM, amiodarone, Ant. indicates anterior wall; BB, beta blocker; EF, ejection fraction; ICM, ischemic cardiomyopathy; inf., inferior wall; LVDd, left ventricular diastolic dimension; NICM, nonischemic cardiomyopathy.

Pace-mapping and entrainment mapping. Pace-mapping using unipolar stimuli that were obtained from the distal electrode of the mapping catheter was performed at sites demonstrating fractionated electrograms and the VT exit site as obtained from the 12-lead ECG. The amplitude and pulse width of the stimuli were 10 mA and 2 ms, respectively(26). The stimulus to QRS interval (S-QRS) was estimated manually from stimulus spike to the onset of QRS in the ECG lead. Based on the previous studies, the S-QRS delays indicating conduction delays were defined as ≥40 ms(10, 17, 29, 30). The morphology of the paced QRS complex was compared with those of clinically documented VTs and/or induced VT during the session. A pace-map QRS morphology that matched VT in more than 10 of the 12 ECG leads was regarded as a pace-map match. A mapping catheter was placed on the presumed central common pathway or the exit sites; VT induction through triple extrastimuli from the right ventricular apex and the outflow tract was performed for entrainment mapping.

Ablation. Radiofrequency (RF) ablation was attempted by using a 7-Fr catheter having a 4 mm distal tip electrode (Cordis-Webster). RF energy was delivered in a temperature-controlled mode for 30-90 seconds at each ablation site; the maximal temperature target was 60°C, and the maximum power was 40 W. In the event that the isthmus of the VT circuit could be detected, each RF point was applied in a direction that was perpendicular to that of the isthmus. In cases where only the presumed exit site of the VT course was defined, RF points were applied by drawing a line including the VT exit site. In either case, the RF line was

drawn across the defined scar or from the scar to the mitral annulus as much as possible. Due to the instabilities with regard to inducibility, VT induction after ablation was not evaluated systematically.

Estimation of abnormal endocardium. The endocardial surface of 3D maps was divided into multiple non-overlapping triangular segments. The extent of the whole surface was the sum of all the triangular segments with the exception of the aortic and mitral valve annuli. Low-voltage area (LVA) was defined as the sum of areas with local bipolar amplitude <1.5 mV(21), and the scar was defined as an area with no reproducible local bipolar potential and no local capture during pacing. Extent of LVA and the areas of the dense scar were estimated by using the same method.

Follow-up. After the procedure, ICD implantation was recommended for all the patients. All the patients were followed up on an outpatient basis; ICD interrogation was performed every 3 months after the procedure. VT recurrence was defined as sustained VT that warranted device therapy. In patients who refused ICD implantation, recurrence was detected by ECG recording.

Statistics. Continuous data are expressed as mean ± SD. Continuous variables were analyzed by using the student's *t*-test for unpaired data, and categorical variables were analyzed by using Fisher's exact test. Free from VT recurrence was analyzed by using the Kaplan-Meier method with log-rank test. A Wilcoxon test was used to compare the number of ICD firing before and after ablation. Values of $P < 0.05$ were considered as statistically significant.

RESULTS (TABLE I AND II)

All the patients experienced ≥1 spontaneous VT as demonstrated on a 12-lead ECG. At the time of the procedure, 4 ICM and 6 NICM patients were treated with amiodarone. There were no significant differences with regard to the left ventricular diastolic diameter and left ventricular EF between the 2 patient groups.

Voltage mapping (**Table III**)**.** Left ventricular 3D maps were constructed using 145 ± 78 and 126 ± 69 points in ICM and NICM patients, respectively (P = n.s.). The total endocardial surface areas were 135.5 ± 21.7 cm^2 and 134.1 ± 25.0 cm^2 in ICM and NICM patients, respectively (P = n.s.), while total extent of the LVA was 53.8 ± 21.5 cm^2 and 20.8 ± 16.7 cm^2 in ICM and NICM patients, respectively (40.2 ± 17.3% vs. 14.5 ± 11.0% of the total endocardial surface, P = 0.002). LVA was not detected in 1 NICM patient. In ICM patients, a single large LVA was located at the infarcted site. However, in the NICM patients, a mean of 3.0 ± 2.3 (range, 2-8) scattered LVAs were observed. Of the 27 LVAs, 17 were located near the ventricular base. The extent of the scar tissue in ICM and NICM patients accounted for 15.2 ± 10.7% and 7.6 ± 8.2% of the respective LVA (P = n.s.).

VT induction (**Table IV**)**.** A total of 28 monomorphic VTs were induced in 16 patients. Only ventricular fibrillation was induced in 1 patient, and any sustained ventricular arrhythmia was not induced in 1 patient.

Five VTs, which were induced in 2 ICM and 2 NICM patients, were stable and adequate for whole chamber activation and entrainment mapping. The activation sequence of 2 VT induced in 2 ICM patient was consistent with a large reentry circuit. The activation sequence

in the remaining 3 stable VTs induced in 2 NICM patients demonstrated a focal pattern. However, these 3 VTs could be entrained and mechanisms underlying these VTs were considered to occur due to myocardial reentry.

The mechanisms underlying 23 unstable VTs were analyzed by limited activation and entrainment mapping. Of these, 1 had a focal origin, 2 VTs occurred due to bundle branch reentry, and 17 VTs occurred due to myocardial reentry(15, 18). The mechanism of the remaining 3 VTs could not be determined because of poor reproducibility with regard to the initiation or spontaneous changes from one VT to another.

Table II. Patient characteristics-2

	ICM	NICM	p
N	9	9	
Gender (Male)	8	8	n.s.
Age (years)	69 ± 14	69 ± 9	n.s.
LVDd (mm)	61 ± 5	64 ± 5	n.s.
EF (%)	33 ± 9	33 ± 8	n.s.
Medication before Ab			
AM	4	6	n.s.
SOT	1	1	n.s.
BB	7	8	n.s.
A	6	8	n.s.
Medication after Ab			
AM	3	4	n.s.
SOT	2	2	n.s.
BB	7	8	n.s.
A	6	8	n.s.

Data are expressed as mean ± SD or number of patients. A indicates angiotensin converting enzyme inhibitor or angiotensin II receptor blocker; Ab, ablation; AM, amiodarone; BB, beta blocker; EF, ejection fraction; ICM, ischemic cardiomyopathy; LVDd, left ventricular diastolic dimension; NICM, nonischemic cardiomyopathy; SOT, sotalol.

Table III. Voltage Mapping

Pt.	No. of mapping points	mapped rhythm	total surface (cm^2)	LVA surface (cm^2)	% of LVA (%)
1	154	sinus	139.7	43.7	31.3
2	91	sinus	156.9	64.2	40.9
3	75	paced	94.7	41.0	43.3
4	181	sinus	147.3	35.5	24.1
5	148	sinus	119.1	24.0	20.2
6	112	paced	157.2	82.9	52.5
7	62	sinus	147.9	57.5	38.8
8	162	sinus	113.2	88.6	78.3
9	320	sinus	143.2	47.0	32.8
10	170	VT	154.1	26.8	17.4
11	42	AF	123.2	36.2	29.4
12	163	sinus	149.9	15.5	10.4
13	254	paced	103.6	7.6	7.3
14	168	paced	172.2	31.4	18.3
15	102	sinus	112.8	0	0
16	38	paced	103.7	5.0	4.8
17	100	paced	131.7	13.6	10.3
18	97	paced	155.6	50.8	32.7
mean ± SD	135 ± 57		134.8 ± 22.7	37.3 ± 25.3	27.4 ± 19.3

AF indicates atrial fibrillation; LVA indicates low voltage area; VT, ventricular tachycardia.

Table IV. VT Induction, Ablation, and Follow-up Results

Pt	Induced VT	Stable C-VT n, (CL, ms)	Unstable C-VT n, (CL, ms)	Stable NC-VT (CL, ms)	Unstable NC-VT (CL, ms)	Ablation Attempted n	Medication after procedure	ICD	VT recurrence
1	1	0	0	0	1(376)	0	BB	yes	yes
2	1	0	0	0	1(300)	0	A+BB+AM	yes	yes
3	0	0	0	0	0	1	A+BB+AM	yes	no
4	1	0	1(290)	0	0	1	SOT	yes	no
5	1	0	0	0	1(280)	1	None	yes	no
6	1	0	1(320)	0	0	1	A+BB	yes	no
7	1	0	0	0	1(340)	0	A+BB+AM	no	no
8	2	0	0	1(340)	1(290)	2	A+BB	no	no
9	3	1(370)	0	0	2(380/400)	4	A+BB+SOT	yes	no
10	4	2(400/410)	2(390/440)	0	0	3	A+BB+AM	yes	yes
11	1	0	0	0	1(360)	0	A+BB+AM	yes	yes
12	4	0	1(350)	0	3(340/300/280)	1	A+BB+AM	yes	yes
13	2	0	0	0	2(340/280)	2	BB	yes	yes
14	2	0	0	0	2(340/440)	2	A+AM	yes	yes
15	0#	0	0	0	0	1	A+BB	yes	yes
16	1	0	0	0	1(260)	0	A+BB+SOT	yes	no
17	2	1(370)	0	0	1(310)	1	A+BB	yes	no
18	1	0	1(390)	0	0	1	A+BB+SOT	yes	yes

A indicates angiotensin converting enzyme inhibitor or angiotensin II receptor blocker; AF, atrial fibrillation; AM, amiodarone; BB, beta blocker; CL, cycle length; C-VT, clinically documented ventricular tachycardia; ICD, implantable cardioverter defibrillator; LVA indicates low voltage area; NC-VT, non-clinical ventricular tachycardia; Pt., patient; SOT, sotalol. #: only ventricular fibrillation was induced.

Pace-mapping and S-QRS interval. Pace-mapping was assessed for 6 VTs in 6 ICM patients and for 11 VTs in 9 NICM patients (**Fig. 1.** and **Fig. 2.**). Pace-mapping could not be assessed in 2 ICM patients who were assessed early in this series and in 1 ICM patient for whom clinical VT occurred due to bundle branch reentry.

In ICM patients, pace-mapping was performed at 257 sites (43 ± 17 sites per patient), and capture was present at 183 sites (71%). In NICM patients, pace-mapping was performed at 239 sites (27 ± 15 sites per patient), and capture was present at 216 sites (90%). Pace-map match sites with conduction delay (>40 ms) were obtained for all 6 VTs in the 6 ICM patients (**Fig. 3.**) and for 3 VTs in 3 NICM patients. The pace-map match sites were recorded; however, conduction delay was not observed for 7 VTs in 5 NICM patients (**Fig. 4.**). Pace-map match could not be obtained anywhere on the endocardial surface for 1 VT in 1 NICM patient.

Pace-map matching was obtained at 52 and 50 sites in ICM and NICM patients, respectively. Moreover, 39 of the 52 sites (75%) in ICM patients and 25 of the 50 sites (50%) in NICM patients were located in LVAs (<1.5 mV) (**Fig. 2.**). In ICM patients, the remaining 13 sites (25%) with pace-map matching were observed in areas close to LVAs. Additionally, at the sites with pace-map matching, the mean S-QRS interval in ICM patients was longer than that in NICM patients (65 ± 31 ms vs. 43 ± 25 ms, $P = 0.0002$) and the mean local bipolar amplitude in ICM patients was lower than that in NICM patients (0.7 ± 0.8 mV vs. 3.1 ± 3.2 mV, $P < 0.0001$). Moreover, the longest S-QRS interval at the pace-map match sites in each group patient was 109 ± 37 ms (range 67–168 ms) and 55 ± 39 ms (range 23–123 ms) in ICM and NICM patients, respectively ($P = 0.02$).

Fig. 1.

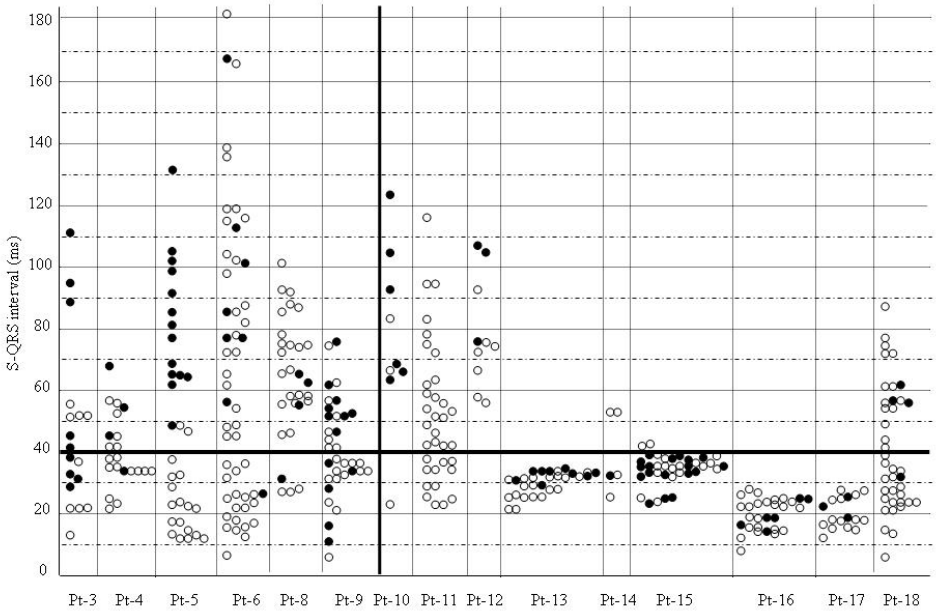

Fig. 1. The S-QRS interval during pace-mapping in each patient is shown. The black dot indicates S-QRS interval at the site with pace-map matching; open circle, without pace-map matching. The longest S-QRS interval at the pace-map match site was ≥40 ms in all 6 ICM patients. However, pace-map match sites with conduction delays (≥40 ms) were observed in 3 NICM patients. Furthermore, pace-map match sites were recorded, but conduction delay was not observed in 5 NICM patients. Pace-map match could not be obtained anywhere on the endocardial surface in 1 NICM patient. Conduction delays were observed in 3 NICM patients at the sites without pace-map matching. However, conduction delays were not detected in 3 NICM patients at any of the attempted pace-mapping sites. Pt indicates patient; S-QRS, stimulus to QRS.

Fig. 2.

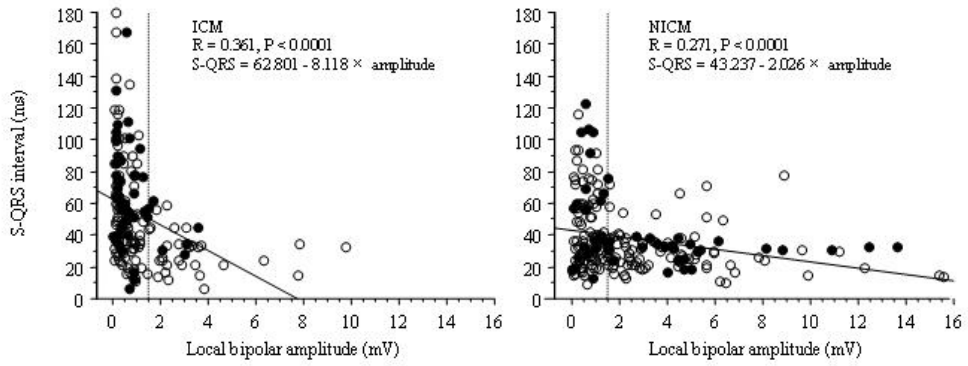

Fig. 2. The relationship between the local bipolar amplitude and S-QRS interval at sites with and without pace-map matching is presented. The black dot indicates S-QRS interval at the site with pace-map matching; open circle, without pace-map matching.
Correlation between the local bipolar amplitude and S-QRS interval was present in both ICM (left panel) and NICM (right panel) patients. The local bipolar amplitude at pace-map match sites was <1.5 mV at 39 of 52 sites (75%) in ICM patients and 25 of 50 sites (50%) in NICM patients. Half of the pace-map match sites in NICM patients were located at normal voltage areas (>1.5 mV). At the pace-map matching sites, the S-QRS interval was longer, and local bipolar amplitude, lower in the ICM patients than those in the NICM patients (65 ± 31 ms vs. 43 ± 25 ms, P = 0.0002; 0.7 ± 0.8 mV vs. 3.1 ± 3.2 mV, P < 0.0001)

Fig. 3.

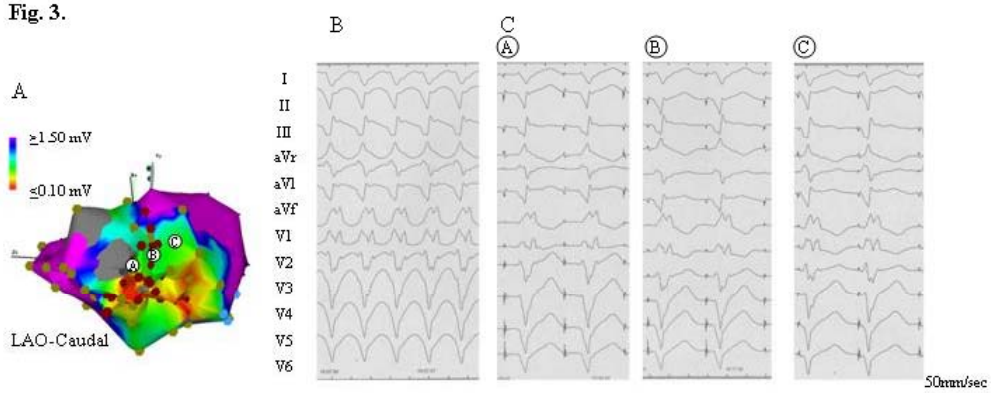

Fig. 3. Voltage map and 3 examples of pace-mapping in Patient 6 (ICM) are shown. (A) Modified left anterior oblique (LAO)-caudal view of the voltage map of the left ventricle during right ventricular pacing (pacing cycle length = 600 ms) in Patient 6. The purple area represents normal endocardium (>1.5 mV). The gray areas represent scars. Border zones (0.1–1.5 mV) are defined as areas with color gradients between red and purple. (B) Twelve-leads ECG of clinical VT induced during the session (cycle length = 330 ms). (C) Twelve-leads ECG during pace-mapping at sites A, B, and C; the QRS morphology at these sites was identical to those of induced clinical VTs. The S-QRS interval was 168 ms at site A, 87 ms at site B, and 56 ms at site C. After analyzing these S-QRS intervals, the entrance of the VT circuit was thought to be located at site A, and the exit, at site C. A linear radiofrequency (RF) lesion perpendicular to the VT circuit was created (red tag indicates ablation point).

Fig. 4.

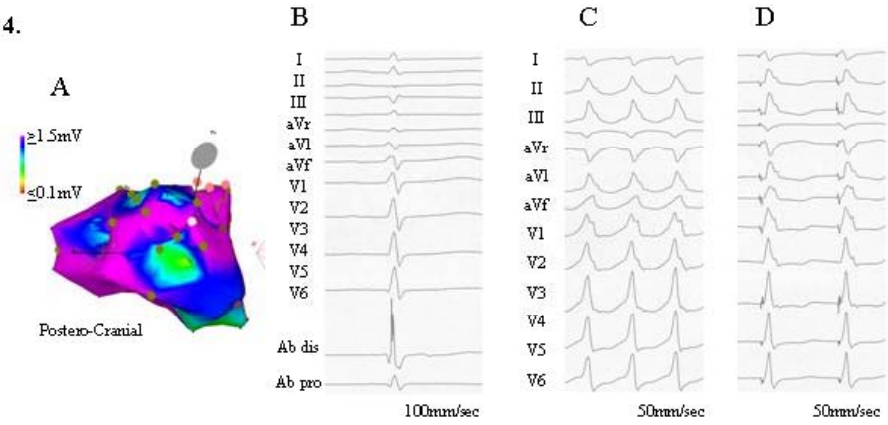

Fig. 4. Voltage map and pace-mapping in Patient 17 (NICM) was given. (A) Postero-cranial view of left ventricular voltage map during right ventricular pacing (pacing cycle length = 600ms) Color gradient corresponds to the local bipolar amplitude as described in **Fig. 3.** Pace-map matching was obtained at white dot site. Local potential at pace-map match site (B), induced clinical VT (cycle length = 370ms) (C), and pace-map QRS morphology were shown (D). The QRS morphology of pace-mapping is close match to that of induced clinical VT but S-QRS interval during pace-mapping is 27ms (without conduction delay).

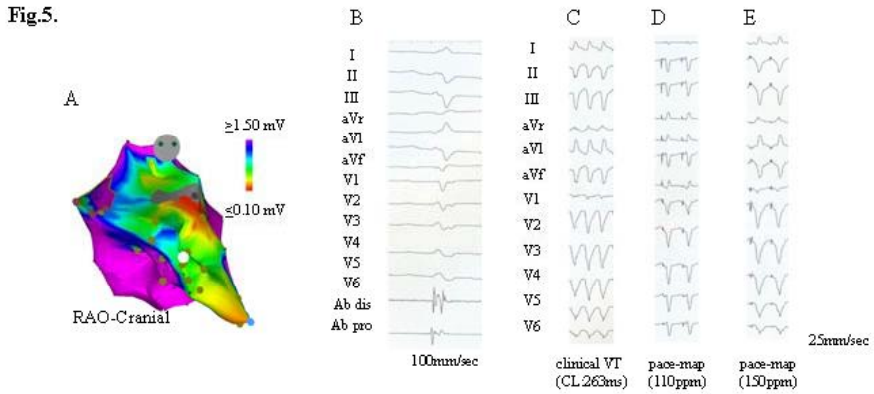

Fig. 5. Voltage map and pace-mapping in Patient-4 (ICM) was given. (A) RAO-cranial view of left ventricular voltage map in Patient-4 was shown. Color gradient corresponds to the local bipolar amplitude as described in **Fig. 3.** (B) Local potential at pace-map site (white dot), 12-lead ECG of induced clinical VT (cycle length = 290ms) (C), pace-mapping at 110ppm (D), and pace-map at 150ppm (E) were shown. QRS morphology produced by pace-map at 110ppm was not similar to that of clinical VT, QRS morphology produced by pace-map at 150ppm was perfect match to that of clinical VT.

Ablation (**Table IV**)**.** Ablation was attempted in 6 ICM patients and 7 NICM patients. Ablation could not be performed in the remaining 5 patients due to the following reasons: pace-map was not assessed in 2 ICM patients; clinical VT was considered to occur due to bundle branch reentry in 1 ICM patient; pace-map match could not be obtained in 1 NICM patient; and in 1 NICM patient, pace-map match was obtained within the normal voltage area; however, the scar and anatomical obstacle suitable for drawing linear ablation were not observed around this area. The mean numbers of RF pulses were 21 ± 15 and 22 ± 14 in ICM and NICM patients, respectively. The mean duration of the procedure was 184 ± 69 min, and the fluoroscopy time was 66 ± 39 min. In 1 ICM patient, cardiac tamponade occurred that required surgical intervention. However, no other major complications were observed in the present patient series.

Follow-up (**Table IV**)**.** After the procedure, ICD was implanted in 6 NICM patients and 5 ICM patients. Two ICM patients refused ICD implantation.

Amiodarone therapy was continued in 3 ICM patients and 4 NICM patients subsequent to the ablation. All the patients have been followed up in the outpatients clinic during the mean follow-up period of 397 ± 277 days. In the 2 ICM patients and 1 NICM patient who did not undergo RF ablation, VT recurrence was observed at 408 days, 433 days, and 44 days after ICD implantation, respectively. Among those treated with ablation, no VT recurrence was

observed in 6 ICM patients. However, 6 of the 7 NICM patients experienced VT recurrence despite ablation therapies (range; 46–391 days after ablation, log-rank P = 0.0016). Four NICM patients had undergone ICD implantation prior to ablation; subsequent to the ablation in the present study, they were treated with same oral drug as that provided before. The number of VT episodes in these 4 NICM patients decreased from 5 to 1, 60 to 1, 111 to 22,and 150 to 4, respectively, subsequent to ablation therapy (P = 0.08, follow up duration; 451, 466, 512, and 180 days in each patient). One NICM patient expired 512 days after the procedure due to gastrointestinal bleeding; another NICM patient expired 466 days after the procedure due to the progression of congestive heart failure.

DISCUSSION

Abnormal endocardial electrograms were observed in a smaller region in the NICM patients than in the ICM patients. Isthmus of the VT circuit could be detected by the combination of substrate mapping and pace-mapping in ICM. However, in NICM, it was more difficult to detect the isthmus on the endocardial surface. VT recurrence after ablation was not observed in ICM but it was observed in NICM patients. Endocardial RF ablation for NICM patients was effective in decreasing the frequency of the VT episodes.

Voltage mapping. Single large LVA was observed at the area with infarction in ICM. However, in NICM, multiple patchy LVA were detected. And as previously reported by Hsia et al (19), abnormal electrograms were frequently located in proximity to the ventricular base in perivalvular lesions in NICM patients. In several recent study, the area with abnormal electrogram have ranged 41 cm^2 to 71 cm^2 in ICM(21, 22, 26) and 7.9 cm^2 to 41 cm^2 in NICM (19, 21, 25). Although there is some dispersion in the size of the area with abnormal electrograms, our measured data of the area with LVA (53.8 ± 21.5 cm^2 in ICM and 20.8 ± 16.7 cm^2 in NICM) agrees with these reports. This dispersion was thought to be derived from the differences among these reports in LVEF (ranged 23% to 31% in ICM and 30% to 45% in NICM), location of infarction, and the cut off value of abnormal electrograms (ranged 1.5 mV to 1.8 mV).

The criteria of abnormal electrograms have varied in the previous reports(2, 19, 21); however, the cutoff value that is used to define an area of abnormality has ranged from <1.5 mV to 1.8 mV (19, 21). The ideal criteria remain unclear; however, a low-voltage limit of <1.5 mV appears reasonable according to the previous reports (9, 21).

Pace-mapping and S-QRS interval. In this study, pace-mapping was performed to determine the critical isthmus of VT; pace-mapping was performed because induced VTs were poorly tolerated, and the inducibility of the target VTs was unstable(6, 20, 21). Kottkamp et al. (20) reported that the pace-map QRS morphology combined with the S-QRS interval analysis was helpful in detecting the critical isthmus of VT, and an RF lesion at this site could suppress the VT recurrence in patients with un-mappable VT.

In the present study, pace-mapping was performed at 90–110/min; therefore, the effect of pacing rate on the QRS morphology should be considered. In patient-4 (**Fig. 5.**), no pace-map matching was obtained at a pacing rate of 110/min from the presumed exit site of the left ventricle and the opposite site of the right ventricle. However, at 150/min, pace-map matching was obtained at the same site from which a pace-map match could not be obtained at a pacing rate of 110/min. As a high-rate pacing resulted in VT induction, pacing at the same rate as that of clinical VT was not assessed routinely in this study. In the regions

around the presumed exit site where pace-map matching could not be obtained at ≤110/min, pacing at the rate of clinical VT was considered as necessary.

The differences of endocardial arrhythmogenic substrates and the ablation outcomes. The areas with abnormal endocardial electrograms and sites with conduction delays during pace-mapping were located at the area with infarction in ICM patients, however, in NICM patients, as multiple and patchy lesions.

In some NICM patients, pace-map match sites without conduction delays were detected during pace-mapping. These data may be explained by previous reports that slow conduction of reentry circuits in NICM often exists deep in the endocardium and on the epicardium rather than on the endocardial surface (25,27).

Both the multiple patchy substrates on the endocardial surface and the substrates located deep in the endocardium and on the epicardium are considered to be the causes of the difficulty in endocardial VT ablation in NICM patients.

Out of the 28 induced VTs in our study, 22 (79%) demonstrated entrainment in response to overdrive pacing (18). Based on our findings and previous reports (7,15,19-21,25), myocardial reentry appears to be the most common cause underlying the mechanism of sustained monomorphic VT in both ICM and NICM patients.

The effectiveness of substrate-guided ablation to the isthmus of VT has been reported previously (20,21,26,31). Since the VT isthmus could not be detected within the endocardium in all the candidates for VT ablation, linear ablation including VT exit site was considered to be the ideal method in patients for whom only the exit of the VT course was detected.

In general, the reentry circuits of VT in ICM can be detected by substrate mapping and can be ablated from endocardial surface. But the VT recurrences during follow-up have ranged 11% to 36% (3,20-22,26). Since the patients in these reports had more frequent VT before ablation compared with our patients, to compare their results with ours is unsuitable. Recently, the effectiveness of prophylactic VT ablation for the candidates for ICD was studied in ICM (23). VT recurred in 12% of ablated patients. The frequency of VT before ablation was nearly equal to ours. Our outcome is better than theirs, although our study populations were small compared with theirs.

In NICM, the rate of VT recurrences of our study (86%) was relatively higher than those (ranged 46% to 73%) of previous reports (19,21,25). In Japan, only conventional ablation catheter can be available. Our undesirable ablation outcome might be caused by the limitation of available catheter. Because VT circuit of NICM commonly exists deep in the endocardium and on the epicardium, conventional catheter inferiority that it is difficult to create transmural lesion tends to affect insufficient ablation outcomes of NICM than ICM. And our target was limited to clinically VT documented by 12-leads ECG. It is controversial whether all inducible VT should be ablated (including “non-clinical VT”). Aggressive ablation strategy raises the risk of complication for the long procedure time and of repeated VT induction. One multicenter trial reported that acute success was not predictive of VT recurrence during follow-up (8). Therefore, target and endpoint should be decided according to the clinical characteristics of ablation candidates and the consideration based on the experience of each institute.

Because the beneficial effects to suppress ICD discharge were obtained in our methods, endocardial ablation with conventional system is thought to be useful treatment option in NICM patients with frequent ICD discharge.

Limitations of the study. This study used a relatively small sample size. Although the influence of anti-arrhythmic drugs such as amiodarone on the local voltage and the S-QRS interval must be considered as well, they were not assessed due to the small sample size of the study. Although there are some reports suggesting that non-inducibility of any monomorphic VT subsequent to ablation is important, VT induction was not routinely performed in this study because inducibility of clinical VT was very poor prior to ablation.

Because VT was terminated by ICD intervention, a 12-lead ECG during VT could not be recorded. It is difficult to recognize whether VT recurrence has occurred due to ablated, failed.

CONCLUSIONS

In NICM, the arrhythmogenic substrate that represented the abnormal electrogram and conduction delay was observed less within the endocardial surface when compared with that observed in ICM. Isthmus of the VT circuit could be detected by combining substrate mapping with pace-mapping in ICM patients. However, detection of the isthmus on the endocardial surface in the NICM patients was difficult. Additionally, VT recurrence rate subsequent to endocardial ablation was higher in NICM than in ICM.

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