Conversion to Purkinje-Related Monomorphic Ventricular Tachycardia After Ablation of Ventricular Fibrillation in Ischemic Heart Disease

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Conversion to Purkinje-related Monomorphic Ventricular Tachycardia after Ablation of Ventricular Fibrillation in Ischemic Heart Disease

Masuda, Purkinje SMVT Conversion after VF Ablation

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Abstract

Background: Catheter ablation is an effective therapy for ventricular fibrillation (VF) arising from the Purkinje system in ischemic heart disease. However, some patients experience newly emergent monomorphic ventricular tachycardia (VT) after the ablation of VF. We evaluated the prevalence and mechanism of monomorphic VT after VF ablation.

Methods and Results: Twenty-one consecutive patients with primary VF due to ischemic heart disease who underwent catheter ablation were retrospectively analyzed. Twenty of 21 patients were in electrical storm. Ventricular premature contractions triggering VF arose from the left Purkinje system and were targeted for ablation. Before the ablation, 14 of 21 patients had only VF, and the other 7 had VF and concomitant monomorphic VT. Four of the 14 patients with only VF (29%) exhibited newly emergent monomorphic VT after VF ablation. Three of these patients had Purkinje-related VTs, which were successfully eliminated by ablation of a Purkinje network located in the same low-voltage area as the site of prior successful VF ablation. During a median follow-up of 28 months (interquartile range: 16–68 months), VF recurred in 6 of 21 patients (29%); however, there were no electrical storms or monomorphic VT, and all recurring arrhythmias were controlled by medical therapy alone.

Conclusions: Over a fifth of patients with primary ischemic VF experienced newly emergent Purkinje-related monomorphic VT after VF ablation. The circuit of the monomorphic VT associated with the Purkinje network was located in the same low-voltage area as the Purkinje tissue that triggered VF, and could be suppressed by additional ablation.

Key Words: ablation, ischemic heart disease, Purkinje network, ventricular fibrillation, ventricular tachycardia

Introduction

Ventricular fibrillation (VF) is a life-threatening arrhythmia that often occurs in patients with ischemic heart disease (IHD). The Purkinje system has been reported to play a pivotal role in triggering and perpetuating VF,[1] and catheter ablation targeting Purkinje potentials can suppress this arrhythmia.[2-9] Catheter ablation is an effective therapy and appears to have a high success rate for the treatment of VF associated with Purkinje system in general. However, some patients experience newly emergent monomorphic ventricular tachycardia (VT) after VF ablation.

The mechanism of VF conversion to monomorphic VT is unclear, but we hypothesized that catheter ablation of cardiac tissue results in conduction delay within the Purkinje network to generate new VT circuits. The aim of this study was to evaluate the prevalence and underlying mechanism of monomorphic VT after VF ablation, and its outcome over long-term follow-up.

Methods

Patients

Data regarding 21 consecutive patients who underwent catheter ablation of primary VF due to IHD between December 2003 and May 2014

were retrospectively analyzed. This study was approved by the Institutional Review Board of each of the 6 participating hospitals. Twenty of 21 patients were in electrical storm (more than 3 VF episodes per 24 hours). The diagnosis of IHD was based on past medical history and clinical findings. Coronary angiography was performed in all patients. Prior to VF ablation, various therapies were attempted, including coronary revascularization, heart failure treatment, antiarrhythmic medications, deep sedation, and overdrive pacing. However, VF was refractory to those therapies, and an urgent ablation was then performed. We obtained written informed consent for VF ablation from each patient and/or their family. Antiarrhythmic drugs were continued in almost all cases during the ablation.

Mapping and Ablation

Multielectrode catheters and an ablation catheter were introduced percutaneously, and a three-dimensional (3-D) mapping system (CARTO, Biosense Webster, Diamond Bar, CA, USA; or EnSite NavX, St. Jude Medical, St Paul, MN, USA) was used in all patients. A 3-D voltage map of the left ventricle was constructed before ablation, and the size of low-voltage areas was measured. However, this step was completed for only 8 patients, as the rest of the patients exhibited hemodynamic instability, or an older version of the 3-D mapping system was used, which lacked the functionality

to calculate the low-voltage area. We defined regions with an electrogram voltage <1.5 mV as low-voltage areas. VF was triggered by the same ventricular premature contraction (VPC) in each patient, and the origin was identified by determining the earliest activation and/or pace mapping. A Purkinje potential, a sharp and high-frequency deflection, always preceded the onset of the trigger VPC, and its localization was carefully mapped. The earliest Purkinje potential during the trigger VPC was targeted for ablation. If monomorphic VT was induced and hemodynamically stable, entrainment and/or activation mapping using the 3-D mapping system were performed to determine the mechanism and circuit of the VT. Among the induced VTs, we defined those with the involvement of a bundle branch, fascicle, or distal Purkinje network in the VT circuit as Purkinje-related VTs,[10] and those with the critical isthmus in the low-voltage area and lacking the involvement of the Purkinje system as scar-related VTs. Ablation was performed with a maximum power of up to 50 W and a target temperature of 42°C for the irrigation catheter, and 58°C for the non-irrigated 4-mm- or 8-mm-tip catheter.

The endpoint of the ablation was the elimination of VF and VT. With the exception of 3 patients with hemodynamic instability (patients no. 2, 17, and 20), we confirmed the non-inducibility of VF and VT by an

isoproterenol infusion and electrical stimulation from the right ventricle and/or left ventricle in all patients at the end of the session.

Follow-up

The patients were followed-up in the outpatient clinic every 1 to 2 months and/or in the device clinic every 6 months after the ablation; symptoms, electrocardiograms, and implantable cardioverter defibrillator (ICD) interrogation logs were checked at every visit. An ICD was implanted in 17 of 21 patients before or after the ablation. Three patients refused an ICD, and implantation was avoided in one patient due to their high risk of skin infection. Clinical success was defined as no recurrence of any sustained VT or VF during the follow-up period. Fifteen of 21 patients continued to take class III antiarrhythmic drugs after the ablation. If an arrhythmia recurred, a change in medication, reprogramming of the ICD parameters, and re-ablation were considered.

Statistical Analysis

Categorical variables were expressed as numbers and percentages. Continuous variables were expressed as the mean ± standard deviation, or median with 25% to 75% interquartile range (IQR). The variables were compared using the Student’s t-test or Fisher's exact test. The threshold for statistical significance was $p < 0.05$.

Results

Patient Characteristics

Patient characteristics are summarized in Table 1. The mean age was 66 ± 12 years, and 91% of the patients were male. The rates of comorbid diabetes mellitus, hypertension, and chronic kidney disease were relatively high: 57%, 67%, and 62%, respectively. Sixteen patients had already undergone complete revascularization before the occurrence of VF. The interval from the onset of an infarction to VF occurrence was 1 to 59 days (median 7 days) in 14 patients with acute or subacute myocardial infarction (MI), and 1.4 to 20 years (median 9.5 years) in 6 patients with remote MI. Among 14 patients with acute or subacute MI, 5 had undergone coronary revascularization after VF events. The other 9 had experienced VF at an interval of 1 to 8 days (median 3 days) after revascularization.

The patients in this study were divided into two groups: 14 patients who only had VF before ablation, and 7 patients who had both VF and concomitant monomorphic VT before ablation. Monomorphic VT was sustained in all cases. The two groups demonstrated no significant differences in patient characteristics except for total radiofrequency (RF)

time. The patients with both VF and monomorphic VT had significantly longer total RF time.

VF and/or nonsustained polymorphic VT were repeatedly initiated by the trigger VPC with the same QRS morphology in all patients. The morphology of the VPC was a right bundle branch block (RBBB) configuration with a superior axis in 15 patients, an RBBB configuration with an inferior axis in 5 patients, and an atypical left bundle branch block (LBBB) configuration with a superior axis in 1 patient. The mean number of failed antiarrhythmic drugs was 2.9 ± 1.2, corresponding to 62%, 62%, 95%, and 19% failed antiarrhythmic drugs with class IB, class II, class III, and class IV (verapamil), respectively. Nine patients required mechanical support such as an intraaortic balloon pump (patients no. 9, 12, 13, 15, 16, 17, 19, 20, and 21) and extracorporeal membrane oxygenation (patients no. 13, 15, and 20) to assist their hemodynamic state.

Mapping and Ablation

The site of the target VPC was located in the left ventricular septum in 18 patients, and in the anterolateral area in 3 patients, which was within the border of the low-voltage areas of a prior MI. In all patients, VF was acutely suppressed, and the electrical storm subsided after RF energy applications targeting the Purkinje potentials that preceded the onset of the

trigger VPC. No major complications were associated with the ablation procedures.

Conversion of VF to New Monomorphic VT

The therapies used in this study cohort are outlined in the flowchart (Figure 1). Among the 21 patients with primary VF, 14 had only VF before the ablation. In these 14 patient, the first ablation for VF was performed successfully, and the non-inducibility of any ventricular arrhythmias was confirmed by programmed stimulation at the end of the session. After this first session of ablation, total elimination of VF was achieved during the follow-up period in 6 of the 14 patients who had only VF (and no monomorphic VT) before ablation. New occurrence of monomorphic VTs after the first ablation was seen in 4 patients (29% of patients who had only VF before ablation), while VF recurred in 4 patients. The interval of time between the ablation and the occurrence of the new VT ranged from 4 hours to 29 days (median 4 days) (Table 2). These new monomorphic VTs were successfully suppressed by a second ablation session in all 4 patients. Two patients with recurrent VF underwent a second ablation, which achieved acute suppression of the VF; however, in both of these patients, VF recurred even after the second ablation session. The other two patients with recurrent

VF received only medical therapies and did not undergo re-ablation because the electrical storm subsided.

Concurrence of Monomorphic VT before VF Ablation

Seven patients demonstrated concurrent monomorphic VT before the ablation (Figure 1). In 2 of them, both VT and VF were successfully eliminated after the first ablation session. Of the remaining 5 patients, both VF and VT recurred in 2 patients, and VF alone recurred in 3 patients. Two patients with VF and VT recurrence, and 1 patient with only VF recurrence underwent a second ablation, which achieved complete arrhythmia suppression. The remaining 2 patients with VF recurrence did not undergo re-ablation and received only medical therapy due to the absence of electrical storm.

Mechanism of Monomorphic VTs

Monomorphic VTs were documented in 11 patients: newly emergent monomorphic VT after the first ablation for VF was noted in 4 patients, and concomitant monomorphic VTs before the ablation in 7 (Table 3). Three of 4 patients with new monomorphic VTs had Purkinje-related VTs: an inter-fascicular VT in 1 case (patient no. 3), and Purkinje VTs arising from the left distal Purkinje network in 2 cases (patients no. 4 and 13). The other VT (in patient no. 18) was scar-related, and unrelated to the Purkinje system.

All 7 cases of concomitant monomorphic VT before the ablation were Purkinje-related VTs. All 10 Purkinje-related VTs (i.e., previously present and newly developed) represented cases of very fast VT (mean cycle length [CL], 286 ± 53 ms), and were unmappable due to hemodynamic compromise; nonetheless, all were successfully ablated, guided by pace-mapping, at the site where Purkinje potentials were recorded during sinus rhythm.

Figure 2 shows an example of the conversion of VF into sustained monomorphic VT after VF ablation (patient no. 4). During a VF storm, two types of VPC were present (Figure 2A). Both VPCs had an RBBB configuration with a superior axis. While VPC2 was always isolated, VPC1 exclusively induced VF or nonsustained polymorphic VT. The ventricular tachyarrhythmia was always polymorphic VT or VF, and monomorphic VT was not observed. The trigger VPC1 was successfully eliminated by ablation at the scar border zone with preceding Purkinje potentials (Figure 2C). One month after the VF ablation, an ICD intervention for the “VF zone” occurred. Electrocardiogram monitoring and ICD interrogation revealed that a rapid sustained monomorphic VT (CL, 240 ms) was initiated by VPC2 and terminated by the ICD shock (Figure 2B). The QRS configurations of the monomorphic VT and VPC2 were similar. This rapid monomorphic VT was

successfully ablated at a single site with a diastolic Purkinje potential during the VT, and this site was located within the same low-voltage area as the Purkinje network responsible for the VF that was successfully ablated in the 1st session (Figure 2C). After the ablation, the VT became noninducible, and VPC2 was also abolished.

Conversion from VF to Purkinje-related monomorphic VT sometimes occurred after a very short interval from the VF ablation. Figures 3–5 show an example of the conversion from VF to monomorphic VT shortly after the VF ablation (patient no. 13). An urgent ablation was performed for a primary VF storm (Figure 3A); however, it resulted in a recurrent electrical storm of a newly emergent monomorphic VT 4 hours after the first ablation session (Figure 3B). On the following day, a second urgent ablation was performed. The target VPC had an RBBB configuration with an inferior axis and was similar to the QRS configuration during monomorphic VT. In the second session, sustained monomorphic VT, not VF, was repeatedly induced by the VPC. Diastolic Purkinje potentials were recorded from the left ventricular mid-septum during the VPC that preceded the onset of the QRS by 150 ms (Figure 4A). The coupling interval from the prior conducted QRS in sinus rhythm to the trigger Purkinje potential was 320 ms. A diastolic Purkinje potential was also recorded during the

monomorphic VT. From this site, sustained monomorphic VT was reproducibly induced by a single stimulus with the same coupling interval (Figure 4B). Pacing from this site terminated the VT without global capture (Figure 5). After the RF energy application to this site, which was in the same low-voltage area as the sites of the first successful ablation, the VT became noninducible (Figure 6), and the trigger VPC was eliminated.

Long-term Follow-up after Ablation

Bailout from a VF/VT storm was successfully achieved in one or two procedures (mean, 1.4 ± 0.5 procedures) in all patients. There were no VF storms after the ablation during the median follow-up of 28 months (IQR: 16–68 months). Patient no. 2 died from heart failure 1 month after the procedure and was excluded from the calculation of follow-up periods because of insufficient follow-up data.

Six patients (29% of the total) experienced arrhythmia recurrences. In 4 cases (patients no. 12, 15, 16, and 20), one to three episodes of VF occurred within 5 days after the ablation and resolved spontaneously with medical therapy alone. In the other 2 patients (patients no. 6 and 14), VF recurred at 25 and 35 months, respectively. These patients received additional medical therapies and no subsequent ICD intervention. During the follow-up period, 10 patients died: 3 from heart failure, 2 from stroke, 2

from sepsis, 2 from cancer, and 1 from pneumonia. There were no deaths from arrhythmia.

Discussion

Previous investigators reported that ablation targeting Purkinje potentials could suppress VF in ischemic cardiomyopathy.[2-9] Compared with previous studies, the present study has reviewed data regarding more cases of VF ablation in patients with IHD, and collected over a longer follow-up period. To our knowledge, no previous study has analyzed the conversion of VF into monomorphic VT by catheter ablation.

Monomorphic VT including Purkinje Fibers in the Circuit

Three of 4 newly emergent monomorphic VTs seen after VF ablation were Purkinje-related VTs, as were all 7 concomitant monomorphic VTs before VF ablation. In all patients, the diagnosis of these Purkinje-related monomorphic VTs was determined by mid-diastolic Purkinje potentials, not by entrainment pacing. These monomorphic VTs included the Purkinje system in part of the circuit and were successfully suppressed by ablation with guidance of Purkinje potentials during sinus rhythm and pace mapping. Therefore, they were diagnosed as Purkinje-related VTs.

This study included a relatively small number of scar-related VTs. We speculate that there are two possible reasons. First, the majority of patients had acute or subacute MI, and different arrhythmic substrates might be involved with remote MI. That is, compared to the scar-related isthmus, the Purkinje network had greater involvement in the establishment of the VT circuit. Purkinje fibers are relatively resistant to ischemia.[11] Second, 95% of patients had already taken class III antiarrhythmic drugs before ablation, which might have suppressed the circuit of the scar-related VT in these patients.

Focal tachycardia and macroreentry have been reported as mechanisms of Purkinje-related VT.[12,13] Because the Purkinje-related VTs in this study were very fast (mean CL, 286 ± 53 ms), a detailed electrophysiological study was difficult to perform. However, we could detect a reentrant mechanism in some patients (e.g., in patient no. 14).

Bogun et al.[13] reported that VTs that included Purkinje fibers as part of the re-entry circuit in patients with a MI exhibited a narrow QRS configuration of less than 145 ms. However, the mean QRS duration of the Purkinje-related VT in this study was wider (184 ± 17 ms). We speculate that one of the reasons for the wider QRS is the shorter VT-CL observed in

this study. While the mean VT-CL in the study by Bogun et al. was 402 ± 82 ms, the VT-CL in the present study was 286 ± 53 ms.

Conversion to Monomorphic VT after VF Ablation

In 4 of 14 patients with only VF before ablation, conversion to Purkinje-related monomorphic VT was observed; monomorphic VT, which was never observed before the VF ablation in these patients based on their records from ICD interrogation or stay in the intensive care department, became dominant after the VF ablation. Tsuchiya et al. reported the case of a patient who exhibited a transition from idiopathic Purkinje-related polymorphic VT to monomorphic VT after the administration of class IC drugs[14]; however, to the best of our knowledge, the present study is the first retrospective evaluation of conversion to Purkinje-related monomorphic VT by ablation of ischemic VF.

Although the hypotheses of mother rotors, multiple wavelets, and focal sources[15-17] have been advocated as mechanisms of VF, there is no single hypothesis explaining the maintenance of VF. In any case, it is clear that the Purkinje network is closely related to the trigger and maintenance of VF.[3, 18, 19] As a possible mechanism of VT conversion, we speculate that creating a unidirectional conduction block or conduction delay within the Purkinje network by ablation organized the unstable reentry circuit into a

stable one, which caused a newly emergent monomorphic VT. Hayase et al. reported that the localization of the VF rotor was consistent with the critical isthmus of the monomorphic VT.[20] The VT and VF originated from nearby regions, which may have created a condition whereby the ablation easily modified the arrhythmic substrates. In fact, 3 of 4 patients with new monomorphic VT in this study had Purkinje-related VTs, and the circuits of these VTs were located within the same low-voltage area, and likely within the same Purkinje network, as the circuits responsible for the VF that was ablated during the 1st session. It is important to recognize the existence of Purkinje-related VT because the ablation strategy for Purkinje-related VT might differ from that of scar-related VT. That is, the strategy of ablation for Purkinje-related VT is simply to identify and ablate the Purkinje potential, which was often located near the VF ablation site. On the other hand, electroanatomical mapping of the entire affected region is often required in the ablation of scar-related VT, and all of the possible critical isthmuses must be eliminated within the wide scar areas.

Outcome after VF Ablation

The efficacy of VF storm ablation[6,21] and urgent ablation for ventricular arrhythmias during acute heart failure[22] has already been reported. In this study, VF recurrence was present in 6 patients; however, the

electrical storms had subsided and all the recurrent arrhythmias were controlled by medical therapy alone. The results of our study seem to be comparable to those of previous reports.[5, 6, 21, 22]

Study Limitations

This study included a relatively small cohort of patients. It is unclear whether concomitant monomorphic VT was absent before the VF ablation in patients without ICDs. However, all patients with VF storms were monitored in the intensive care unit, and no monomorphic VTs were observed. Therefore, we believe these 4 patients had newly emergent monomorphic VTs after the VF ablation. It is noteworthy that there was no VF recurrence concomitant with the newly emergent monomorphic VT after the VF ablation.

Conclusion

VF ablation in patients with IHD was feasible and effective as a bailout therapy for electrical storm, and resulted in a good outcome in the majority of patients. However, over a fifth of patients included in this study, who did not have monomorphic VT prior to ablation, developed Purkinje-related monomorphic VT after the VF ablation. These newly emergent VTs were related to the same Purkinje network that had been

successfully ablated in the 1st session to resolve VF, and were also successfully suppressed by ablation of the Purkinje system in the 2nd session. Although VF ablation in IHD appears to have a high success rate in general, the possibility of conversion from VF to Purkinje-related monomorphic VT and the existence of new VT circuits in the same Purkinje network at the initial VF ablation site should always be kept in mind.

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Disclosures

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Tables

Table 1. Patient Characteristics

	Overall (n = 21)	Original VA		*p*-value
		VF only (n = 14)	VF and VT (n = 7)	
Age (years)	66 ± 12	66 ± 14	65 ± 6	0.84
Male sex	19 (91%)	12 (86%)	7 (100%)	0.43
Diabetes mellitus	12 (57%)	8 (57%)	4 (57%)	0.68
Hypertension	14 (67%)	11 (79%)	3 (43%)	0.13
Chronic kidney disease	13 (62%)	9 (64%)	4 (57%)	0.56
Left ventricular ejection fraction (%)	36 ± 10	37 ± 10	29 ± 8	0.082
NYHA class	2.6 ± 1.0	2.6 ± 0.9	2.7 ± 1.1	0.76
Type of ischemic heart disease				
acute MI	7	5	2	
subacute MI	7	4	3	
remote MI	6	4	2	
myocardial ischemia (non-MI)	1	1	0	
Coronary status				
3-VD	12	9	3	
2-VD	5	3	2	

1-VD	4	2	2	
Complete revascularization	16 (76%)	10 (71%)	6 (86%)	0.44
Failed antiarrhythmic drugs before ablation	2.9 ± 1.2	2.6 ± 1.2	3.3 ± 1.4	0.27
Electrical storm	20 (95%)	13 (93%)	7 (100%)	0.67
Electroanatomical map				
Number of points	257 ± 130	300 ± 121	128 ± 16	0.10
Size of low-voltage area (cm^2)*	100.9 ± 64.7	104.1 ± 70.9	91.2 ± 62.9	0.83*
Size of low-voltage area (%)*	54.2 ± 19.7	54.5 ± 20.5	53.4 ± 25.1	0.95*
Ablation data				
No. of RF sites	17.6 ± 13.2	16.9 ± 14.7	19.3 ± 10.0	0.71
Total RF time (seconds)	998 ± 568	836 ± 570	1377 ± 368	0.048

*Data recorded only for 8 patients, due to technical limitations.

MI indicates myocardial infarction; NYHA, New York Heart Association; RF, radiofrequency; VD, vessel disease; VA, ventricular arrhythmia; VF, ventricular fibrillation; and VT, ventricular tachycardia.

Table 2. Details Regarding the Catheter Ablation Procedure

Patient	Original VA	Electrical storm	Mechanism of the original VA	Conversion from VF to SMVT	Interval from VF ablation to SMVT	Number of required procedures	ICD implantation (pre or post ablation)
Patients who had only VF before ablation							
1	VF only	Yes	Purkinje VF			1	post
3	VF only	Yes	Purkinje VF	Yes	7 days	2	pre
4	VF only	Yes	Purkinje VF	Yes	29 days	2	pre
6	VF only	Yes	Purkinje VF			1	post
7	VF only		Purkinje VF			1	none
9	VF only	Yes	Purkinje VF			1	post
11	VF only	Yes	Purkinje VF			1	post

13	VF only	Yes	Purkinje VF	Yes	4 hours	2	post
14	VF only	Yes	Purkinje VF			2	pre
15	VF only	Yes	Purkinje VF			1	post
16	VF only	Yes	Purkinje VF			2	none
18	VF only	Yes	Purkinje VF	Yes	1 day	2	post
19	VF only	Yes	Purkinje VF			1	post
21	VF only	Yes	Purkinje VF			1	post
Patients who had both VF and VT before ablation							
2	VF and VT	Yes	Purkinje VF Purkinje VT			1	post
5	VF and VT	Yes	Purkinje VF Purkinje VT			2	post

8	VF and VT	Yes	Purkinje VF Purkinje VT	1	post
10	VF and VT	Yes	Purkinje VF Purkinje VT	2	pre
12	VF and VT	Yes	Purkinje VF Purkinje VT	1	post
17	VF and VT	Yes	Purkinje VF Purkinje VT	2	none
20	VF and VT	Yes	Purkinje VF Purkinje VT	1	none

1

2 ICD indicates implantable cardioverter defibrillator; SMVT, sustained monomorphic ventricular tachycardia;

3 VA, ventricular arrhythmia; VF, ventricular fibrillation; and VT, ventricular tachycardia.

1 Table 3. Details Regarding Monomorphic Ventricular Tachycardia

Patient	VT emergence phase (before or after VF ablation)	VT morphology	QRS duration (ms)	Diagnosis of VT	VT-CL (ms)	Hemodynamic collapse during VT
Patients who had only VF before ablation						
3	After	SA + RBBB	210	interfascicular VT	325	Yes
4	After	SA + RBBB	180	Purkinje VT	240	Yes
13	After	IA + RBBB	175	Purkinje VT	280	Yes
18	After	IA + LBBB	160	Scar-related VT	440	No
Patients who had both VF and VT before ablation						
2	Before	N/A	165	Purkinje VT	272	Yes
5	Before	SA +	163	Purkinje VT	239	Yes

		Negative concordance				
8	Before	SA + RBBB	195	Purkinje VT	289	No
10	Before	SA + Negative concordance	195	Purkinje VT	278	Yes
12	Before	SA + RBBB	170	Purkinje VT	246	Yes
17	Before	IA + RBBB	208	Purkinje VT	419	Yes
20	Before	SA + RBBB	180	Purkinje VT	270	Yes

CL indicates cycle length; IA, inferior axis; LBBB, left bundle branch block; N/A, not available; RBBB, right bundle branch block; SA, superior axis; VF, ventricular fibrillation; and VT, ventricular tachycardia.

Figure legends

Figure 1. Flowchart of arrhythmia management in the study cohort. Twenty-one patients were referred for VF ablation. Fourteen patients had only VF, and the other 7 had concomitant monomorphic VT with VF. They were treated by 1 or 2 ablation procedures. SMVT indicates sustained monomorphic ventricular tachycardia; VF, ventricular fibrillation; and VT, ventricular tachycardia.

Figure 2. Conversion from VF to rapid sustained monomorphic VT after the VF ablation (patient no. 4).

A, There were two kinds of VPCs. While VPC2 was always isolated, VPC1 exclusively induced VF or nonsustained polymorphic VT. No monomorphic VT was observed. The trigger VPC1 was successfully eliminated by ablation at the scar border zone with preceding Purkinje potentials. **B,** One month after the VF ablation, monomorphic VT (CL, 240 ms) was initiated by VPC2 and terminated by the ICD shock. This rapid monomorphic VT was successfully ablated at the single site with diastolic Purkinje potentials during VT. After the ablation, the VT became noninducible, and VPC2 was also abolished. **C,** Electroanatomical voltage map obtained in the first session for VF ablation. The red tags indicate the ablation sites for VF. The

blue tags indicate the Purkinje potentials. The successful ablation site for the monomorphic VT is superimposed as a white tag positioned at the site of the Purkinje network, and appears to lie in the same low-voltage area as the original VF ablation sites. CL indicates cycle length; ICD, implantable cardioverter defibrillator; VF, ventricular fibrillation; VPC, ventricular premature contraction; and VT, ventricular tachycardia.

Figure 3. Conversion from VF to rapid sustained monomorphic VT shortly after the VF ablation (patient no. 13).

A, Twelve-lead electrocardiogram during the first ablation session. VF was initiated by the trigger VPC. **B,** Twelve-lead electrocardiogram during the second ablation session, which was performed the next day. SMVT (CL, 280 ms) was initiated by the trigger VPC. CL indicates cycle length; SMVT, sustained monomorphic ventricular tachycardia; VF, ventricular fibrillation; VPC, ventricular premature contraction; and VT, ventricular tachycardia.

Figure 4. Successful ablation site of a monomorphic VT (patient no. 13).

A, On the left ventricular septum, Purkinje potentials (P) were recorded before the QRS during sinus rhythm and the trigger VPC. While Purkinje firing with a coupling interval of 320 ms (arrow) conducted to the

ventricular muscle and produced the VPC, the firing with a shorter coupling interval (270 ms) did not conduct to the ventricular muscle (arrowhead). A diastolic Purkinje potential was recorded during VT. **B,** A single stimulus (S) from this site with a coupling interval of 320 ms repeatedly induced sustained monomorphic VT. VPC indicates ventricular premature contraction; and VT, ventricular tachycardia.

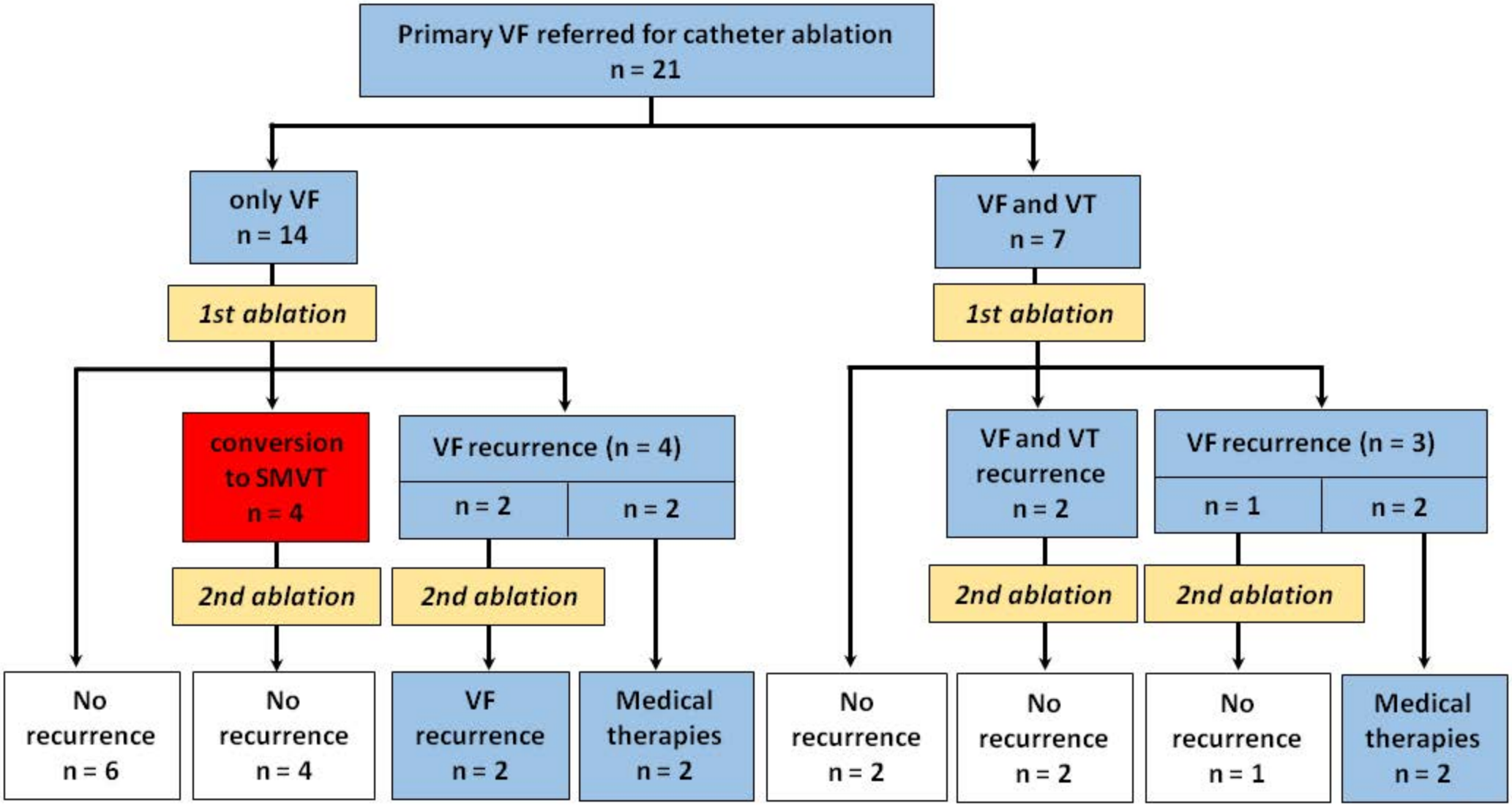
Figure 5. Termination of the VT without global capture at the successful ablation site (patient no. 13).

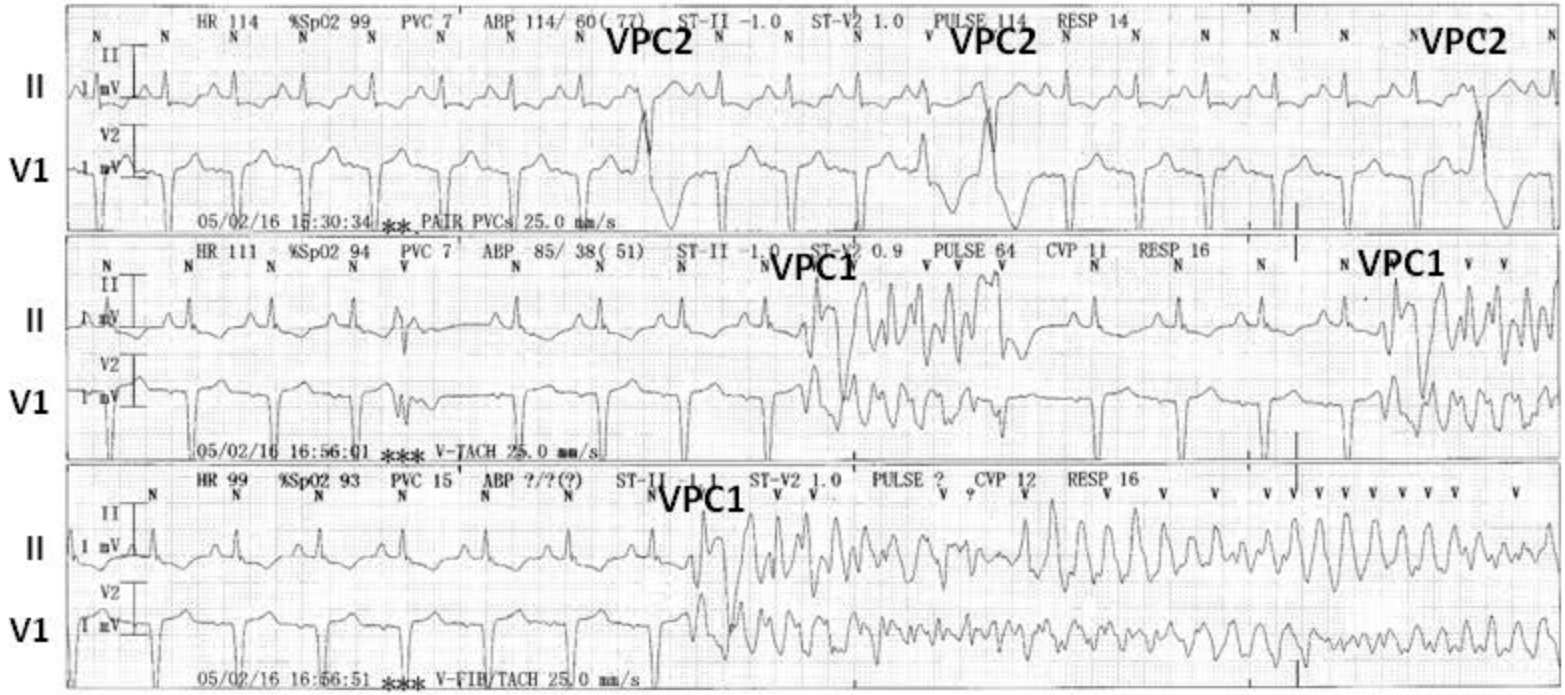
Pacing from the successful ablation site (Figure 4B) could not capture the ventricle. However, it terminated VT. CL indicates cycle length; and VT, ventricular tachycardia.

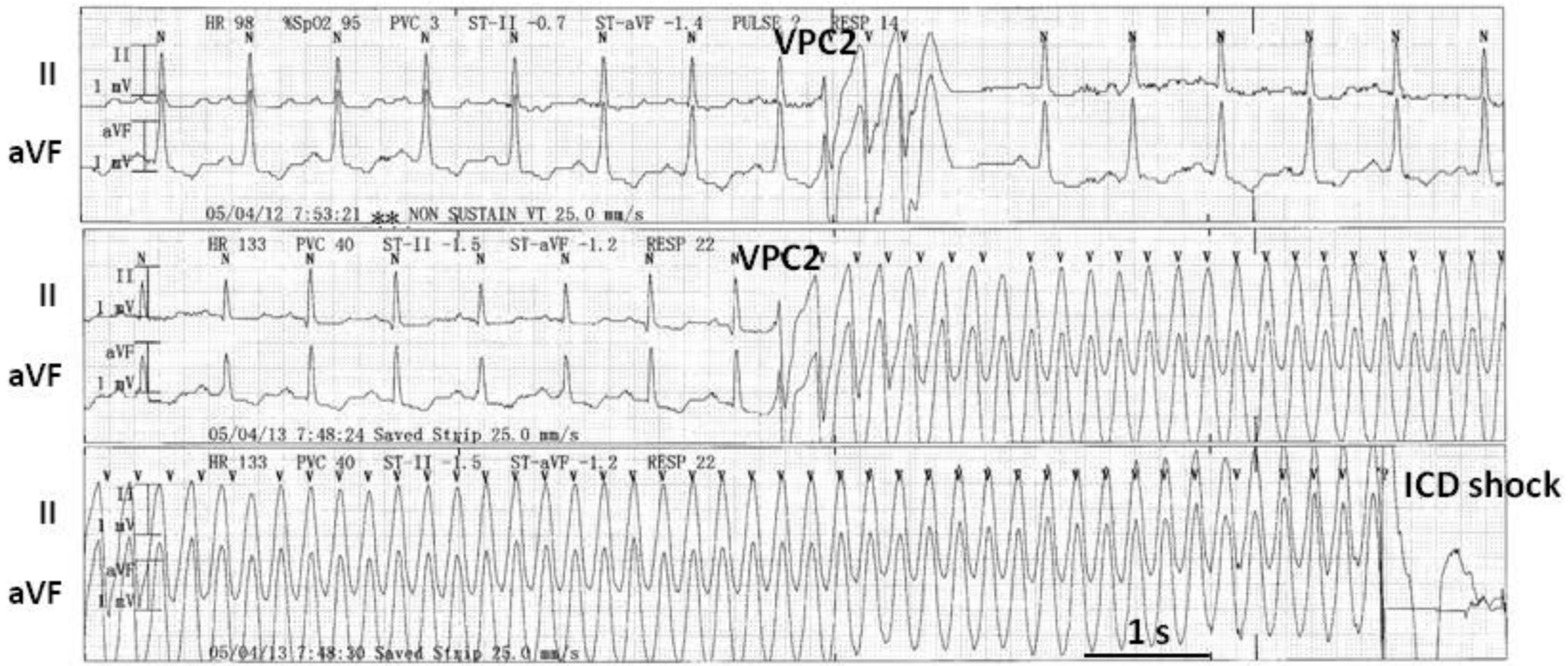
Figure 6. Electroanatomical mapping (patient no. 13)

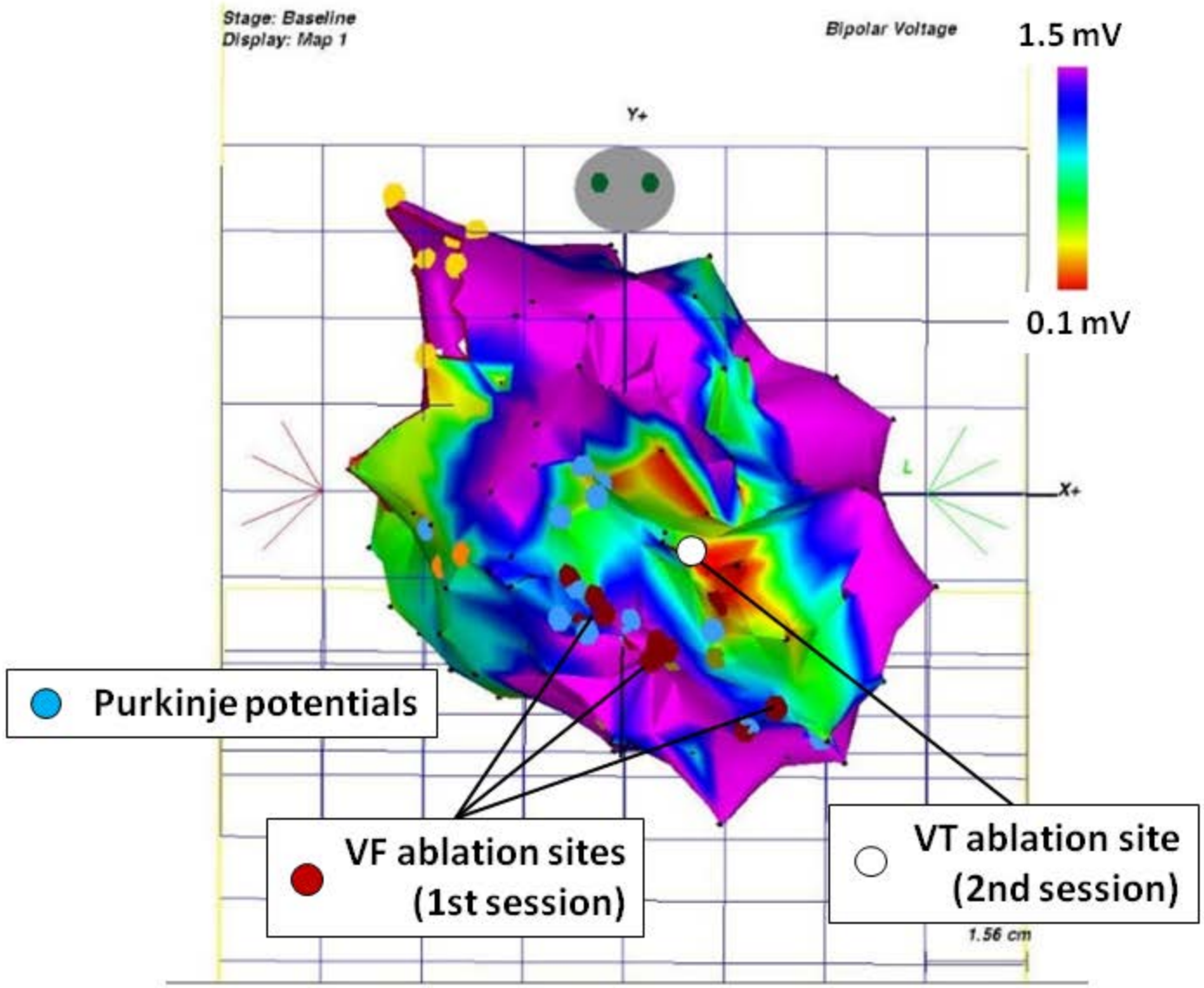
This electroanatomical map was created during the 2nd session for VT ablation. The red tag indicates the ablation site for VF in the 1st session, superimposed on the same map. The white tag indicates the successful ablation site for VT in the 2nd session. The brown tags represent additional ablation sites to prevent the recurrence of ventricular arrhythmias, and the

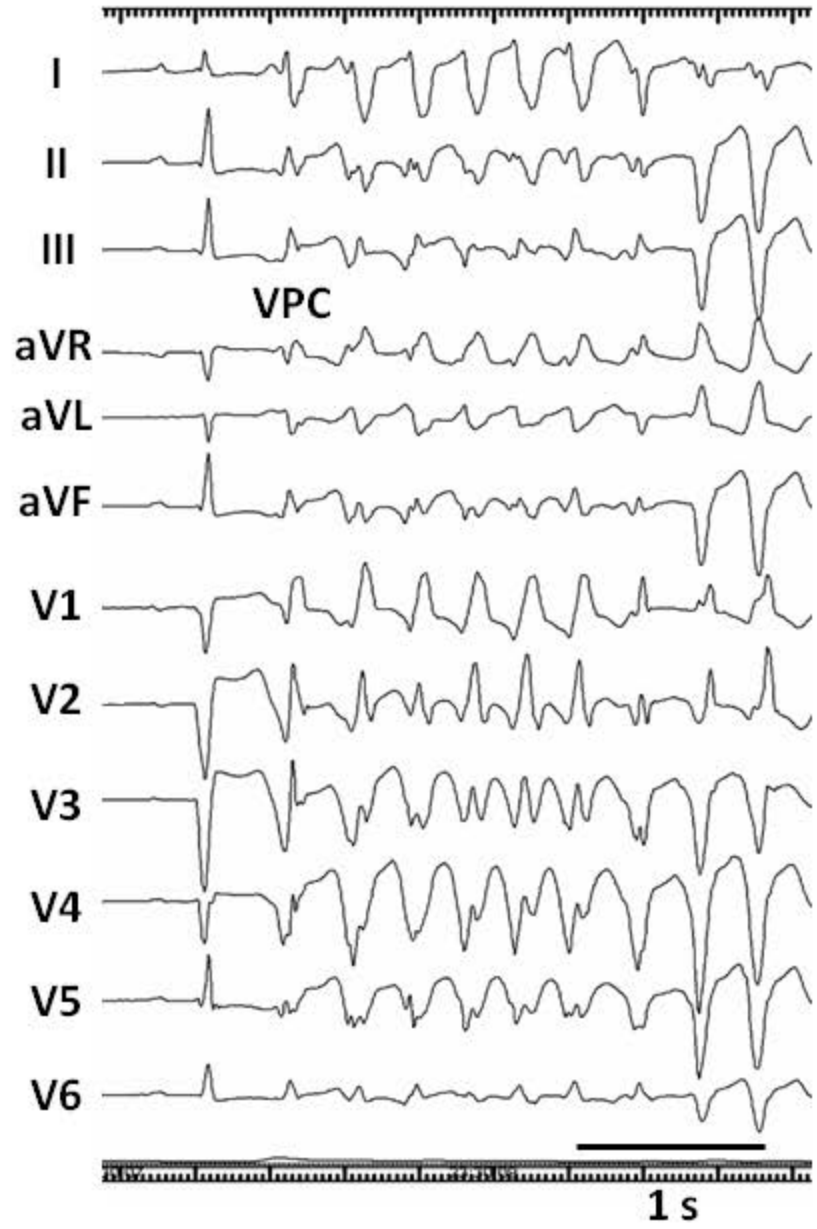
blue tags indicate the Purkinje potentials during sinus rhythm. VF indicates ventricular fibrillation and VT, ventricular tachycardia.

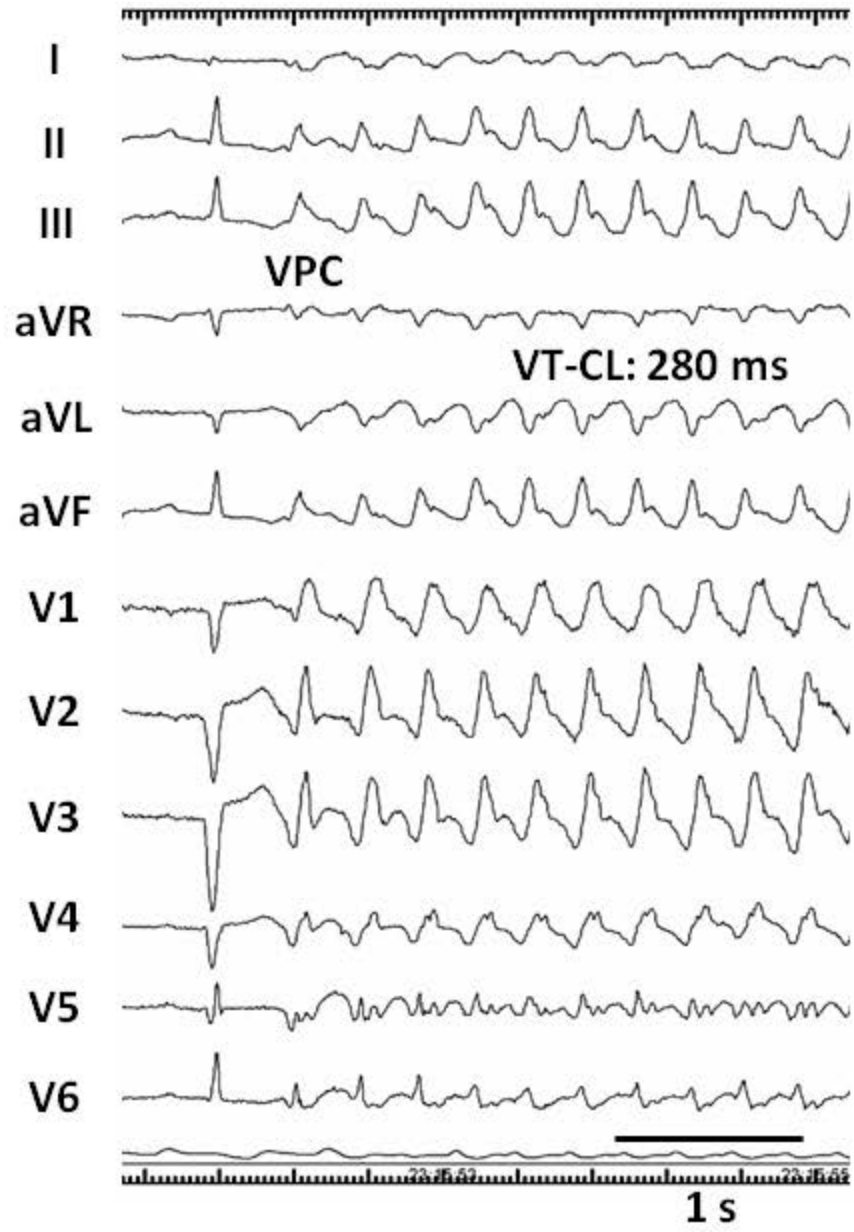

Primary VF referred for catheter ablation
n = 21
only VF
n = 14
VF and VT
n = 7
1st ablation
1st ablation
conversion to SMVT
n = 4
VF recurrence (n = 4)
n = 2
n = 2
VF and VT recurrence
n = 2
VF recurrence (n = 3)
n = 1
n = 2
2nd ablation
2nd ablation
2nd ablation
2nd ablation
No recurrence
n = 6
No recurrence
n = 4
VF recurrence
n = 2
Medical therapies
n = 2
No recurrence
n = 2
No recurrence
n = 2
No recurrence
n = 1
Medical therapies
n = 2

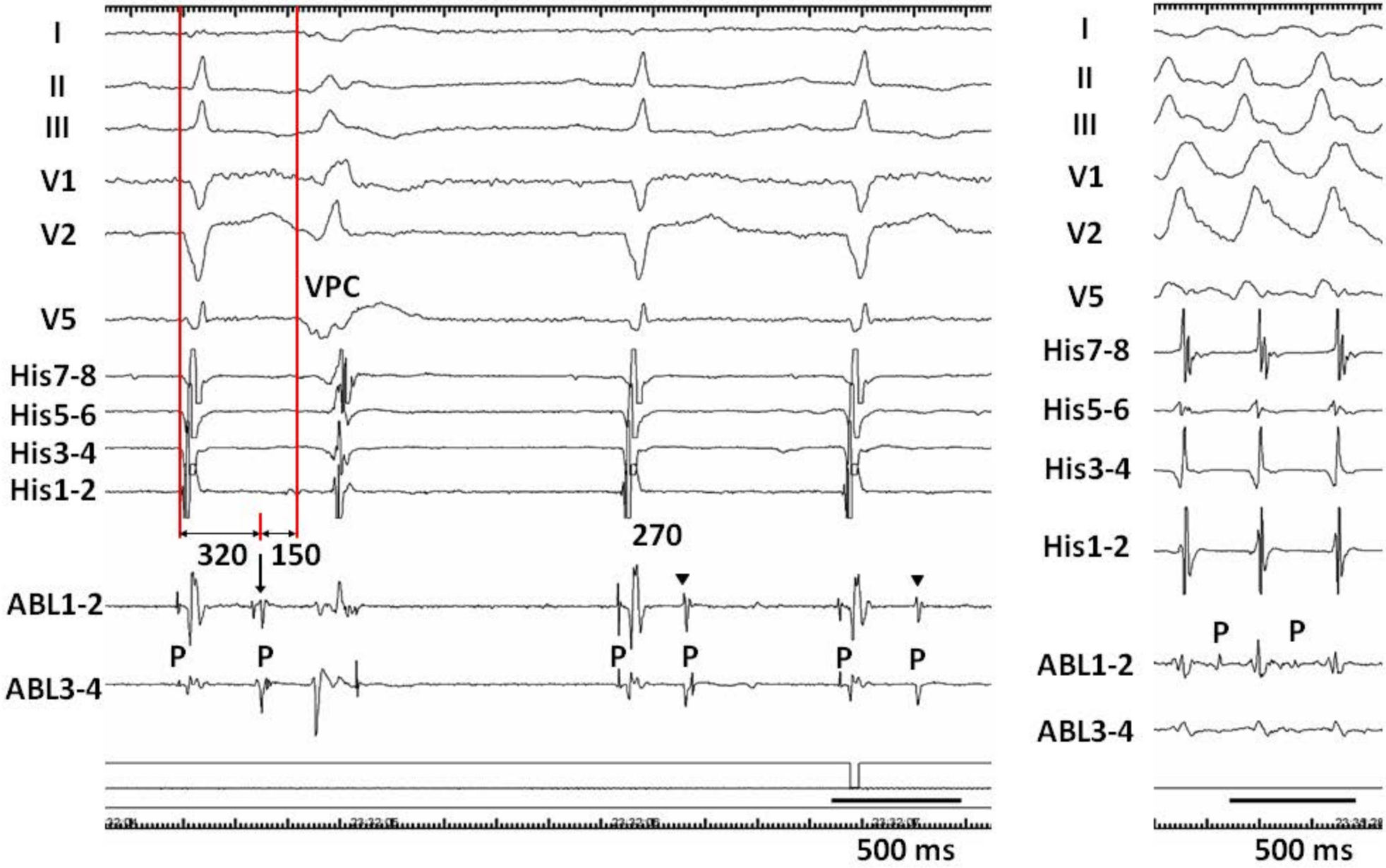

II
V1
VPC2
VPC2
VPC2
VPC1
VPC1
VPC1
II
V1
II
V1

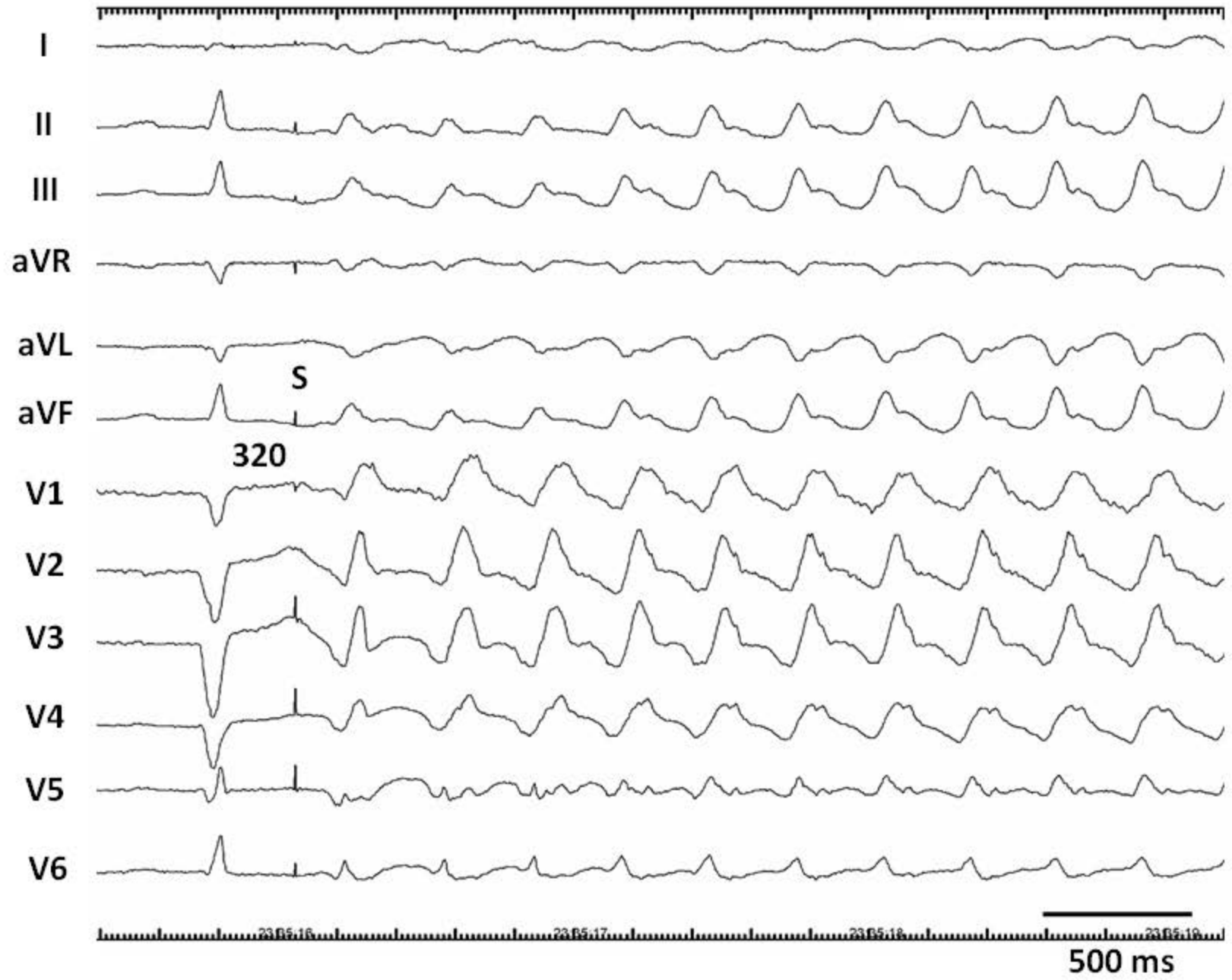

II
aVF
HR 98 %SpO2 95 PVC 3 ST-II -0.7 ST-aVF -1.4 PULSE 2 RESP 14
VPC2
05/04/12 7:53:21 ** NON SUSTAIN VT 25.0 mm/s
II
aVF
HR 133 PVC 40 ST-II -1.5 ST-aVF -1.2 RESP 22
VPC2
05/04/13 7:48:24 Saved Strip 25.0 mm/s
II
aVF
HR 133 PVC 40 ST-II -1.5 ST-aVF -1.2 RESP 22
ICD shock
1 s
05/04/13 7:48:30 Saved Strip 25.0 mm/s

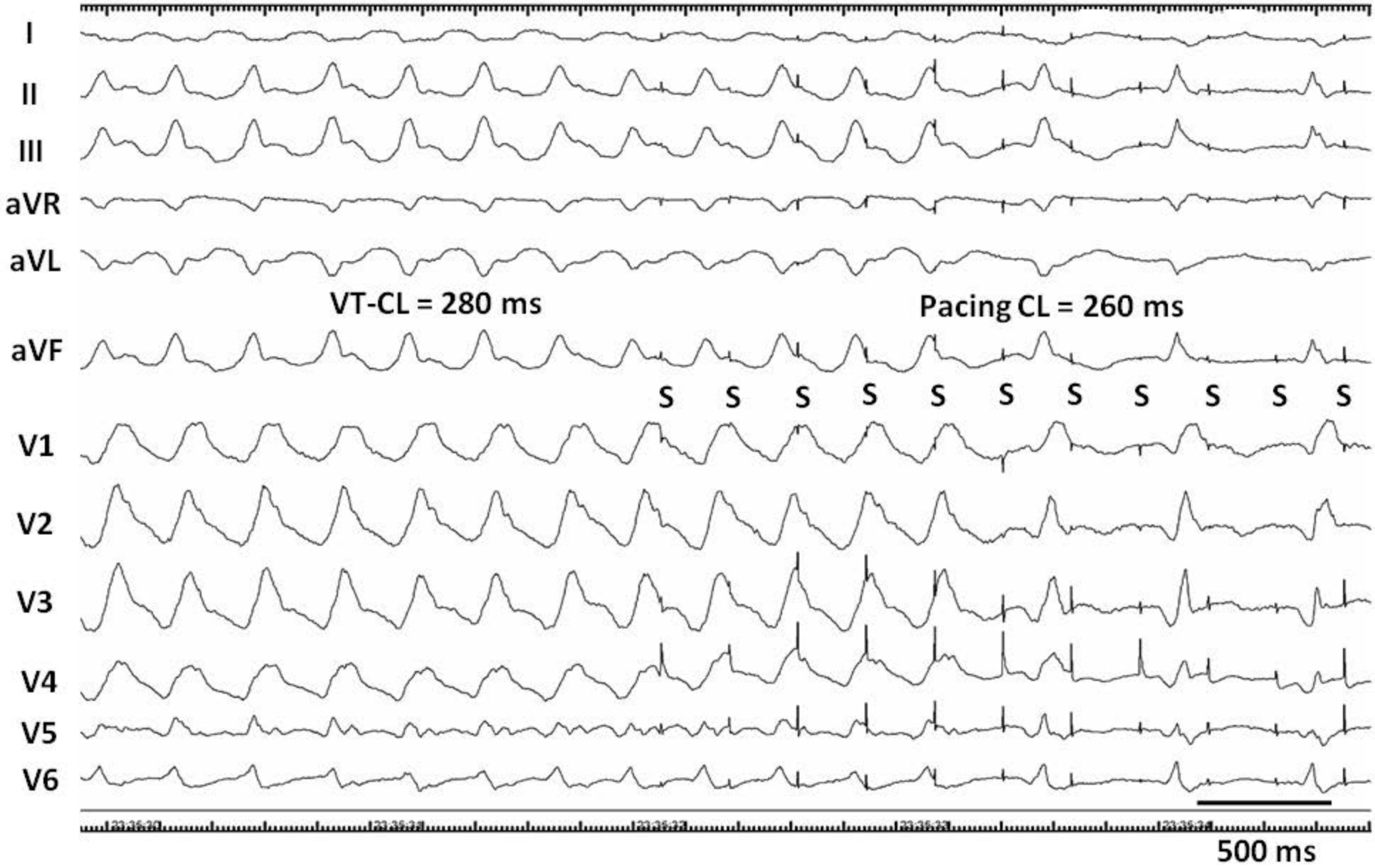

Stage: Baseline
Display: Map 1
Bipolar Voltage
1.5 mV
0.1 mV
Y+
X+
L
Purkinje potentials
VF ablation sites
(1st session)
VT ablation site
(2nd session)
1.56 cm

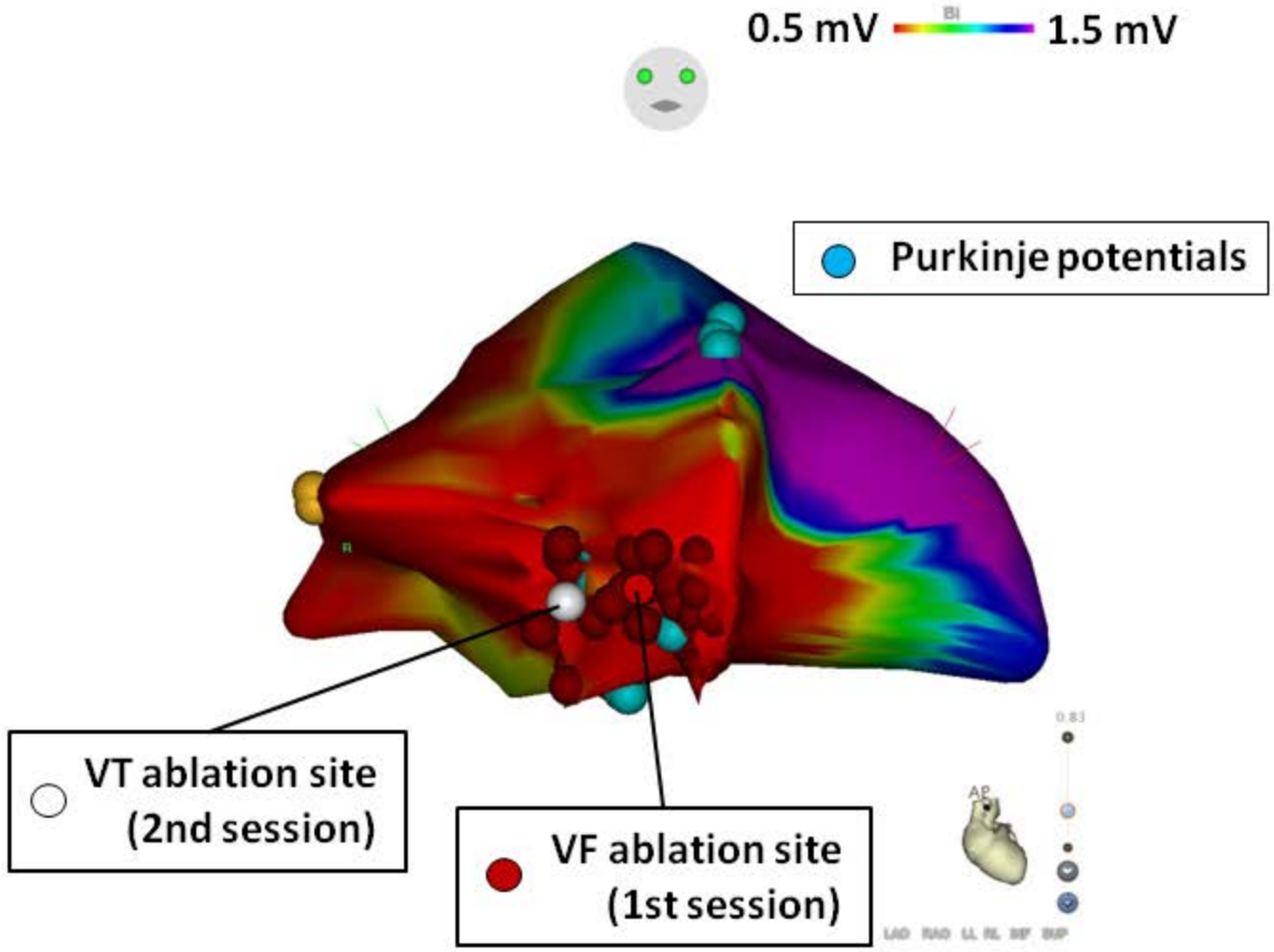

I
II
III
VPC
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
1 s


I
II
III
VPC
aVR
VT-CL: 280 ms
aVL
aVF
V1
V2
V3
V4
V5
V6
1 s


I
II
III
V1
V2
V5
VPC
His7-8
His5-6
His3-4
His1-2
320
150
270
ABL1-2
ABL3-4
P
P
P
P
P
P
500 ms
I
II
III
V1
V2
V5
His7-8
His5-6
His3-4
His1-2
P
P
ABL1-2
ABL3-4
500 ms


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
S
320
500 ms


I
II
III
aVR
aVL
VT-CL = 280 ms
Pacing CL = 260 ms
aVF
S S S S S S S S S S S
V1
V2
V3
V4
V5
V6
500 ms


0.5 mV
BI
1.5 mV
Purkinje potentials
VT ablation site
(2nd session)
VF ablation site
(1st session)
AP
LAO RAO LL RL INF SUP