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Original Article

Analysis of the spatial and transmural dispersion of repolarization and late potentials derived using signal-averaged vector-projected 187-channel high-resolution electrocardiogram in patients with early repolarization pattern

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ABSTRACT

Background: Electrophysiological characteristics of early repolarization syndrome (ERS), i.e., the spatial and transmural dispersion of ventricular repolarization and ventricular late potentials can be evaluated using a signal-averaged vector-projected 187-channel high-resolution electrocardiogram (187-ch SAVP-ECG). We investigated these characteristics as markers of ventricular fibrillation and sudden cardiac arrest in patients presenting with an ER pattern.

Methods: The 187-ch SAVP-ECGs were recorded for 8 patients with idiopathic ventricular fibrillation associated with ERS (ERS patients), and 5 patients with an ER pattern without arrhythmic events (ER pattern patients).

Results: The ER pattern was located in the inferior leads ($n=7$), lateral leads ($n=1$), or both inferior and lateral leads ($n=5$). The corrected RT(RT_c) (peak point of the R wave – positive maximum peak of the first derivative of the T wave interval corrected using the Bazett formula) interval and $T_{(peak\text{-}end)\text{-}c}$ interval from the 187 channels were calculated. Late potentials were positive in 7 of 8 ERS patients and in 3 of 5ER pattern patients ($P=0.25$). The average RT_c was shorter in patients with ERS (192.6 ± 29.8 ms vs. 234.0 ± 25.5 ms, $P=0.04$). However, average $T_{(peak\text{-}end)\text{-}c}$ interval did not differ between the 2 groups.

Conclusion: Late ventricular potentials were common in ERS and ER pattern patients. Lethal arrhythmia in ERS patients appeared to be related to the relatively short average repolarization time rather than the spatial and transmural dispersion of repolarization.

1. Introduction

The early repolarization (ER) pattern has been considered a benign electrocardiographic phenomenon affecting 2–5% of the general population and is most commonly seen in healthy young men and athletes [1–3]. Recent studies have challenged the view of this electrocardiogram (ECG) variant being benign, and instead implicate it as an important marker of vulnerability for ventricular fibrillation (VF) and sudden cardiac death (SCD).

Haïssaguerre et al. were the first to document a high incidence of ER in the inferolateral leads in patients with idiopathic VF [4]. Similarly, Rosso et al. showed that J-point elevation is more common among patients with idiopathic VF than among healthy individuals [5]. Recent studies have provided evidence supporting an association between the ER pattern and life-threatening arrhythmias, such as ER syndrome (ERS) or Brugada syndrome, depending on the region of the heart responsible for the arrhythmogenic substrate.

Although Brugada syndrome and ERS differ with respect to the magnitude and location of the leads in the abnormal ECG J waves, the 2 disorders are thought to represent a continuous spectrum of phenotypes, termed J wave syndromes by Antzelevitch and Yan [6]. There is growing evidence that amplifications of the spatial and transmural dispersion of repolarization underlies the development of life-threatening ventricular arrhythmias associated with inherited ion channelopathies, such as the long-QT, short-QT, and Brugada syndromes, as well as catecholaminergic polymorphic ventricular tachycardia [7].

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The present study retrospectively compared various ECG markers of ventricular depolarization and repolarization, obtained using the novel signal-averaged vector-projected 187 channel high-resolution electrocardiogram (187-ch SAVP-ECG) in patients with ERS and ER pattern.

2. Material and methods

2.1. Patients

Thirteen consecutive men (mean age, 42.2 ± 19.5 years) with the ER ECG pattern (Figs. 3 and 4a) were enrolled in the study. ER was defined as J-point elevation of ≥0.1 mV in at least 2 consecutive inferior or lateral leads. Structural heart disease was ruled out in all patients. Patients with a history of documented VF or aborted SCD were classified as ERS patients ($n=8$), and asymptomatic patients were classified as ER pattern patients ($n=5$). The ER ECG was classified into type 1 (I, V4–V6), type 2 (II, III, aVF), or type 3 (global) [6]. Brugada syndrome was ruled out by a negative pilsicainide test.

The study was approved by the Institutional Review Committee of the Nihon University Hospital (IRB Approval #33, June 1, 2008). Patients signed informed consent forms to allow 187-channel SAVP-ECG recordings, mutation screening for the SCN5A gene, and invasive electrophysiological studies.

2.2. Signal-averaged vector-projected 187-channel high-resolution electrocardiography

Details of the 187-ch SAVP-ECG system have been reported previously [8–10]. The 187-ch SAVP-ECG system consisted of an electrode lead system, an input box, a high-precision amplifier (HRES-1000, Fukuda Denshi Co. Ltd., Tokyo, Japan), and a personal computer. The input box generated a modified X, Y, Z-lead ECG, and the vector-projected 187-channel synthesized ECGs via a Mason-Likar lead system. The input signal (±550 mV) was digitized at 2 kHz by an analog to a digital converter with a resolution of 0.076 μV. Electrodes were attached to the patient's right shoulder, left shoulder, left lower abdomen, right lower abdomen, and at positions V1 through V6 according to the 10-lead Mason-Likar system. The 187-ch SAVP-ECG recording was performed with each patient in the resting position.

2.2.1. Repolarization

The repolarization time (RT) interval was defined as the time between the peak of the R wave and the occurrence of the maximum positive derivative of the T wave (Fig. 1). The T_{peak} and T_{end} were defined the peak (or the nadir in negative or biphasic *T* waves) and as the end of the ECG T wave, respectively. The RT_{end} interval was defined as the time between the peak of the R wave and the occurrence of the maximum derivative of the T wave between T_{peak} (T_{nadir}) and T_{end}. The $T_{peak\text{-}end}$ ($T_{(p\text{-}e)}$) interval was defined as the time between the occurrence of the T_{peak} and the occurrence of the maximum derivative of the T wave between T_{peak} and T_{end} of the ECG.

A previous report has demonstrated that there is a good correlation between the corrected RT (RT_c) and corrected $T_{p\text{-}e}$ dispersion values determined using 187-ch SAVP-ECG and 64-channel magnetocardiographic modalities [9]. Therefore, these values were calculated in this study. The RT dispersion was automatically calculated as the difference between the maximum RT interval (RT_{max}) and the minimum RT interval (RT_{min}). The RT_{end} dispersion was automatically calculated as the difference between the maximum RT_{end} interval ($RT_{end,\ max}$) and the minimum RT_{end} interval ($RT_{end,\ min}$). The $T_{peak\text{-}end}$ dispersion was automatically calculated as the difference between the maximum $T_{peak\text{-}end}$ interval ($T_{p\text{-}e,\ max}$) and the minimum $T_{peak\text{-}end}$ interval ($T_{p\text{-}e,\ min}$). Corrected RT interval (RT_c), corrected RT_{end} interval ($RT_{end\text{-}c}$), and corrected $T_{peak\text{-}end}$ interval ($T_{(p\text{-}e)\text{-}c}$) were calculated using the Bazett formula. Average RT_c, average $RT_{end\text{-}c}$, and average $T_{(p\text{-}e)\text{-}c}$ were calculated as the respective average values of the RT_c, $RT_{end\text{-}c}$, and $T_{(p\text{-}e)\text{-}c}$ intervals obtained from the 187-channels.

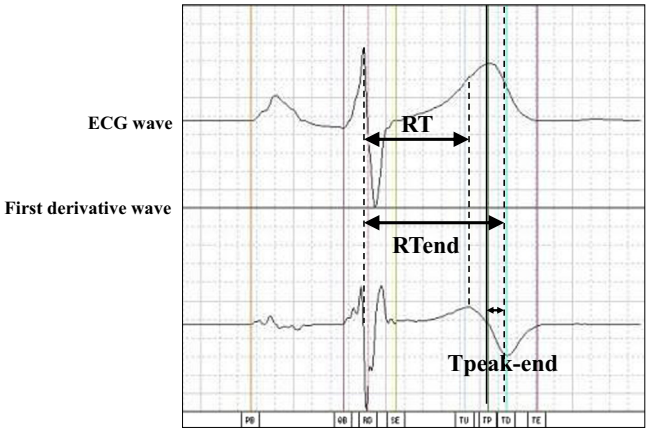

Fig. 1. Reference points for RT interval, $T_{peak\text{-}end}$ interval, and RT_{end} interval. The RT interval was defined as the time between the peak point of the R wave and the maximum positive peak of the first derivative of the T wave. The $T_{peak\text{-}end}$ ($T_{(p\text{-}e)}$) interval was defined as the time between the occurrence of the T_{peak} and the occurrence of the maximum derivative of the T wave between T_{peak} and T_{end} of the ECG. The RT_{end} interval was defined the time between the peak of the R wave and the occurrence of the maximum derivative of the T wave between T_{peak} (T_{nadir}) and T_{end}.

To summarize:

RT_c dispersion = $RT_{c,\ max} - RT_{c,\ min}$.
$RT_{end\text{-}c}$ dispersion = $RT_{end\text{-}c,\ max} - RT_{endc,\ min}$.
$T_{(p\text{-}e)\text{-}c}$ dispersion = $T_{(p\text{-}e)\text{-}c,\ max} - T_{(p\text{-}e)\text{-}c,\ min}$.
Average RT_c = average of RT_c from the 187-channel ECG.
Average $RT_{end\text{-}c}$ = average of $RT_{end\text{-}c}$ from the 187-channel ECG.
Average $T_{(p\text{-}c)\text{-}c}$ = average of $T_{(p\text{-}c)\text{-}c}$ from the 187-channel ECG.

The RT_c, $RT_{end\text{-}c}$, and $T_{(p\text{-}e)\text{-}c}$ maps were displayed as 256-color coordinated image according to the length of time intervals. The blue color indicated intervals <40 ms; green–yellow: 40–79 ms; orange: 80–99 ms; and red: ≥100 ms.

2.2.2. Depolarization

Modified X, Y, Z-lead ECG signals and the synthesized signals from the 187-ch SAVP-ECGs were amplified and passed through a finite impulse response (FIR) digital filter (frequency characterization: 27th order) with a low frequency of 45 Hz and a high frequency of 280 Hz. They were converted from analog to digital signals with 12-bit accuracy at a rate of 2000 samples/s. After elimination of ectopic beats, the ECG signals were averaged over 10 min by means of a signal-processing system. The non-filtered X, Y, Z-lead ECG, filtered(f) X, Y, Z-lead ECG, and vector-magnitude ECG were displayed on the same time scale (Fig. 2).

The time interval of the fQRS was determined automatically using the beginning and end of the vector-magnitude ECG considering the points that exceeded 5 times the noise levels. The root mean square voltage over the last 40 ms of the QRS (RMS_{40}) was calculated as the integral of the vector-magnitude ECG at 40 ms before the end of the QRS (QRS_{end}). In addition, we measured the duration of low-amplitude signals under 40 μV (LAS_{40}) on the vector-magnitude ECG.

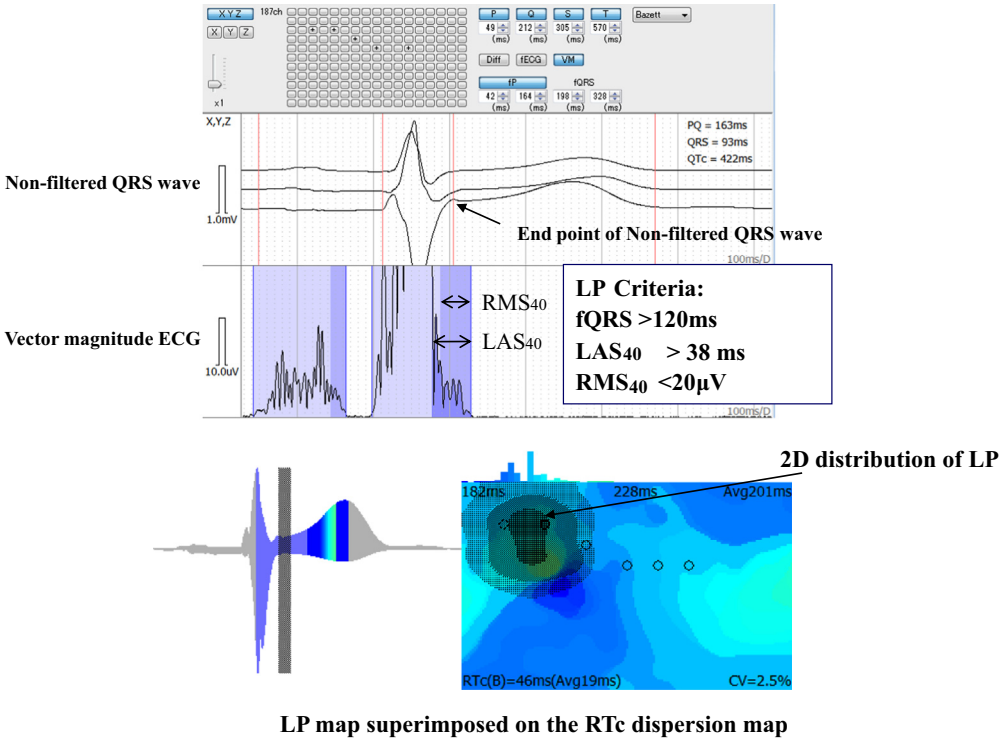

Fig. 2. Spatial distribution of ventricular high-frequency late potentials (HFLPs) as defined by the gray scale of the RT dispersion map derived from the 187-channel signal-averaged vector-projected high-resolution ECG. HFLPS was generated graphically by dividing voltages that exceeded 5 times the mean noise level, and represented by an increasing gray scale. HFLPs area is variable depending by the threshold voltage, which can be changed by 0.5 μV or its integer multiple. Note the ventricular high frequency late potentials located at the right anterior lesion. LP=late ventricular potential, fQRS=filtered QRS duration, RMS_{40}=root mean square voltage of the last 40 ms, LAS_{40}=duration of low amplitude signals under 40 μV, and RT_c=time between the peak points of the R wave and the occurrence of the positive maximum peak of the first derivative of the T wave corrected using the Bazett formula.

The spatial distribution of integrated high-frequency late potentials (HFLPs) on the 187-ch SAVP-ECGs was calculated as the integral of the electrical potentials of the fQRS in the time interval between 30 ms (initial offset) before QRS_{end} (QRS_{end-30}) and QRS_{end}. The endpoint in each channel was defined as the end point of the QRS, where the average voltage exceeded the mean plus 5 standard deviations of the noise sample. The integrated HFLPs map from the 187-ch SAVP-ECG was generated graphically by dividing the voltages that exceeded 5 times the mean noise level by 0.5 μV or its integer multiple. The spatial distribution of integrated HFLPs was represented by an increasing gray scale. The spatial distribution of integrated HFLPs from the 187-ch SAVP-ECG was superimposed on the RT_c and $T_{(p\text{-}e)\text{-}c}$ dispersion map (Fig. 2).

The signal averaged ECG was obtained from the vector magnitude of the X, Y, Z leads. Late ventricular potential was considered positive when two of three criteria (fQRS duration >130 ms, $RMS_{40}<20$ μV, and LAS_{40} duration >38 ms) were met.

2.3. Electrophysiological study

A comprehensive electrophysiological study was performed on the 8 fasting, drug-free, and non-sedated ERS patients. After access to the right femoral vein was obtained at 4 sites, 1 quadripolar catheter (Biosense-Webster, Diamond Bar, CA, USA) was positioned at the right atrial appendage, 1 octapolar catheter (Biosense-Webster) was positioned at the His bundle electrogram recording site, and 2 steerable quadripolar catheters (6-F) with an interelectrode distance of 2-5-2 mm (Biosense-Webster) were positioned in the right ventricular apex and outflow tract. Endocardial potentials were filtered to recording frequencies of 30–500 Hz and recorded on a BARD computer system (BARD Lab Pro, BARD Electrophysiology, Lowell, MA, USA).

Programmed electrical stimulation from the right ventricular apex and right ventricular outflow tract was performed at twice diastolic threshold strength and a pulse of 2-ms duration was achieved with a pulse generator (BD-02, Fukuda Denshi Co., Tokyo, Japan). An S_1-S_2 interval was applied after 8 beats of drive pacing (S_1) at basic cycle lengths of 600 ms and 400 ms. The S_1-S_2 interval was reduced in 10-ms decrements until the effective refractory period of the right ventricle was achieved. If VF lasting >5 s requiring DC shock was not induced with a single premature beat, 3 additional extrastimuli were delivered (S_2 until the effective refractory period was reached, S_3 and S_4 to 180 ms).

2.4. Follow-up

In general, patients attended follow-up examinations at 4- to 5-month intervals in our outpatient clinic. Examinations included assessment of subjective symptoms, as well as 12-lead ECG and device interrogation. Further examinations were performed in the event of symptom onset or device discharge. Follow-up examinations continued over a period of 18–122 months (mean, 47.4 ± 28.9 months; median, 53 months).

2.5. Statistical analysis

Continuous clinical and electrophysiological values are represented as mean $\pm$ SD in Table 1. The Mann–Whitney *U* test was employed to analyze the differences in the values between the groups. Categorical data were analyzed using the Fisher exact probability test. Multivariate Cox regression analyses were performed to determine the association between arrhythmic events and noninvasive parameters. A *P*-value of <0.05 was considered statistically significant. StatView 5.0 software (SAS Institute, Cary, NC, USA) was used for analysis.

Table 1
Clinical, genetic, electrocardiographic, and electrophysiological characteristics of ERS and ER pattern patients.

	ERS ($n=8$)	ER pattern ($n=5$)	P-value
Age (years)	39.1 ± 17.0	51.8 ± 23.2	0.281
Sex ratio (M/F)	8/0	5/0	0.502
Family history of SCD	2	0	0.223
ECG pattern (1/2/3)	6/0/2	1/1/3	0.008
EPS	8	0	–
Inducible VF/PVT at EPS	6	–	–
ICD implantation	5	0	0.003
Follow-up (months)	51.6 ± 29.6	30.0 ± 20.2	0.185
Arrhythmic event during follow-up	2	0	–

Number of patients is shown unless otherwise indicated.
ER: early repolarization; ERS: early repolarization syndrome; SCD: sudden cardiac death; BS: Brugada syndrome; EPS: electrophysiologic study; VF: ventricular fibrillation; PVT: polymorphic ventricular tachycardia; and ICD: implantable cardioverter defibrillator.

Table 2
Comparison of spatial and transmural repolarization times and dispersion of repolarization time in ERS and ER pattern patients.

	ERS ($n=8$)	ER pattern ($n=5$)	P-value
Avg. RT_c (ms)	201.7 ± 18.8	234.0 ± 25.5	0.0404
Avg. $RT_{end,c}$ (ms)	287.1 ± 10.3	325.8 ± 43.8	0.0481
Avg. $T_{(p\text{-}e)\text{-}c}$ (ms)	41.0 ± 9.9	42.6 ± 8.2	0.883
RT_c dispersion (ms)	71.9 ± 14.3	84.2 ± 11.9	0.223
$RT_{end\text{-}c}$ dispersion (ms)	52.7 ± 14.3	52.6 ± 11.3	0.807
$T_{(p\text{-}e)\text{-}c}$ dispersion (ms)	52.7 ± 14.3	52.6 ± 11.3	0.806
fQRS (ms)	114.1 ± 25.6	113.5 ± 18.2	0.851
RMS_{40} (μV)	28.6 ± 30.0	28.7 ± 39.1	0.753
LAS_{40} (ms)	48.8 ± 22.4	39.3 ± 14.5	0.806

Average RT_c=average of RT_c from the 187-channel ECG; Average $RT_{end,\ c}$=average of $RT_{end,\ c}$ from the 187-channel ECG; Average $T_{(p\text{-}c)\text{-}c}$=average of $T_{(p\text{-}c)\text{-}c}$ from the 187-channel ECG; RT_c dispersion=$RT_{c,\ max}-RT_{c,\ min}$; $RT_{end\text{-}c}$ dispersion=$RT_{end\text{-}c,\ max}-RT_{end\text{-}c,\ min}$; $T_{(p\text{-}e)\text{-}c}$ dispersion=$T_{(p\text{-}e)\text{-}c\ max}-T_{(p\text{-}e)\text{-}c\ min}$; fQRS=filtered QRS duration; RMS_{40}=root mean square voltage of the terminal 40 ms in the filtered QRS complex; and LAS_{40}=duration of low-amplitude signals <40 μV in the terminal filtered QRS complex.

3. Results

Clinical, electrocardiographic, and electrophysiological characteristics of the study patients are shown in Table 1. Two ERS patients had a family history of SCD ($P=0.22$). Six ERS patients had a type 1 ER ECG and two had a type 3 ER ECG. One ER pattern patient had a type 1 ER ECG, 1 had a type 2 ER ECG, and 3 had a type 3 ER ECG. Late ventricular potentials were positive in 7 of the 8 (87.5%) ERS patients and in 3 of the 5 (60.0%) ER pattern patients ($P=0.25$). Electrophysiological studies were performed in all 8 ERS patients, and VF was induced in 6 of the 8 (75.0%) patients. Electrophysiological studies were not performed in the ER pattern patients. An implantable cardioverter-defibrillator (ICD) was implanted in all 8 ERS patients, and appropriate ICD discharge occurred in 2 of these patients during the follow-up period of 47.4 ± 28.9 months. No arrhythmic event was noted in ER pattern patients during the follow-up period of 30.0 ± 20.2 months.

Average RT_c and average $RT_{end\text{-}c}$ were significantly shorter in ERS patients than in ER pattern patients (201.7 ± 18.8 ms vs. 234.0 ± 25.5 ms, $P=0.0404$; 287.1 ± 10.3 ms vs. 325.8 ms ± 42.8 ms, $P=0.0481$). The average $T_{(p\text{-}e)\text{-}c}$ did not differ between ERS and ER pattern patients (41.0 ± 9.9 ms vs. 42.4 ± 8.2 ms, $P=0.883$). The average spatial and transmural repolarization times (average RT_c, average $RT_{end\text{-}c}$, and average $T_{(p\text{-}e)\text{-}c}$) obtained from the 187-ch SAVP-ECG per group are shown in Table 2. There was no significant difference in the RT_c dispersion (71.9 ± 14.3 ms vs. 84.2 ± 11.9 ms, $P=0.223$), or $T_{(p\text{-}e)\text{-}c}$ dispersion $RT_{end\text{-}c}$ dispersion (52.7 ± 14.3 ms vs. 52.6 ± 11.3 ms, $P=0.807$) between the ERS and ER pattern patients, (Figs. 3a and b, 4a and b, Table 2). The fQRS, RMS_{40}, and LAS_{40} values (Table 2), as well as the prevalence of late potentials did not differ between ERS and ER pattern patients (7/8 vs. 3/5, $P=0.254$). No statistically significant differences were observed in the parameters on the spatial and transmural dispersion of repolarization between ERS and ER pattern patients.

4. Discussion

4.1. Major findings

We investigated the spatial and transmural dispersion of repolarization and late potentials in patients with ERS using 187-ch SAVP-ECG. We found that the spatial repolarization parameters (average RT_c and average $RT_{end\text{-}c}$) were significantly shorter in ERS patients than in ER pattern patients. In contrast, spatial repolarization dispersion parameters (RT_c dispersion, $RT_{end\text{-}c}$ dispersion) did not differ between the 2 groups. We also showed that parameters of transmural repolarization (average $T_{(p\text{-}e)\text{-}c}$) and transmural repolarization dispersion ($T_{(p\text{-}e)\text{-}c}$ dispersion) did not differ between the 2 groups. Furthermore, there was a high incidence of late ventricular potential and higher rate of VF induction in both groups of patients in ERS patients.

4.2. Electrophysiological abnormalities in J wave syndromes

Yan and Antzelevitch provided evidence that heterogeneous distribution of a transient outward current-mediated spike and dome morphology of the action potential across the ventricular wall underlies the electrocardiographic manifestation of J waves [11]. Recent clinical reports have also suggested that repolarization abnormalities contribute to life-threatening arrhythmic events.

Ghosh et al. suggested that the presence of regions with relatively short action potential, early repolarization, and abnormally large spatial repolarization gradients are causes of proarrhythmia in ERS patients [12]. Letsas et al. reported higher $T_{peak}-T_{end}$ intervals in lead V2, $T_{peak}-T_{end}$ dispersions in the precordial leads, and ($T_{peak}-T_{end}$)/QT ratios in lead V2 in ER pattern patients than patients without the ER pattern [13]. Furthermore, Talib et al. reported that $T_{peak\text{-}end}$ interval and $T_{peak\text{-}end}$/QT ratio in the ambulatory ECG CM5 lead were significantly higher in ERS patients than in ER pattern patients, but did not significantly differ between the control and ER pattern patients [14]. A prolonged $T_{peak}-T_{end}$ interval in V5 was also shown to be associated with SCD in patients with out-of-hospital cardiac arrest in the general population [15].

In the present study, spatial and transmural dispersion of repolarization did not significantly differ between ERS and ER pattern patients. Bastiaenen et al. reported that ER type may represent 2 different processes; classical benign ER may reflect earlier onset of repolarization but malignant ER may reflect abnormal depolarization, possibly due to underlying subtle structural disease [16]. Roten et al. reported that patients with recurrent VF have significantly higher inferior/global J wave amplitudes, inferior J wave amplitudes, high lateral J wave amplitudes, and lateral J wave amplitudes than patients without VF recurrences, but differences in ST segment morphology, $T_{peak}-T_{end}$ interval, and $T_{peak}-T_{end}$/QT ratio in lead V5 were not associated with recurrent VF [17]. Opthof et al. conducted in vivo canine experiments in which the $T_{peak}-T_{end}$ interval correlated with transmural dispersion of refractoriness and was an index of total dispersion of refractoriness [18]. Furthermore, Abe et al. reported that

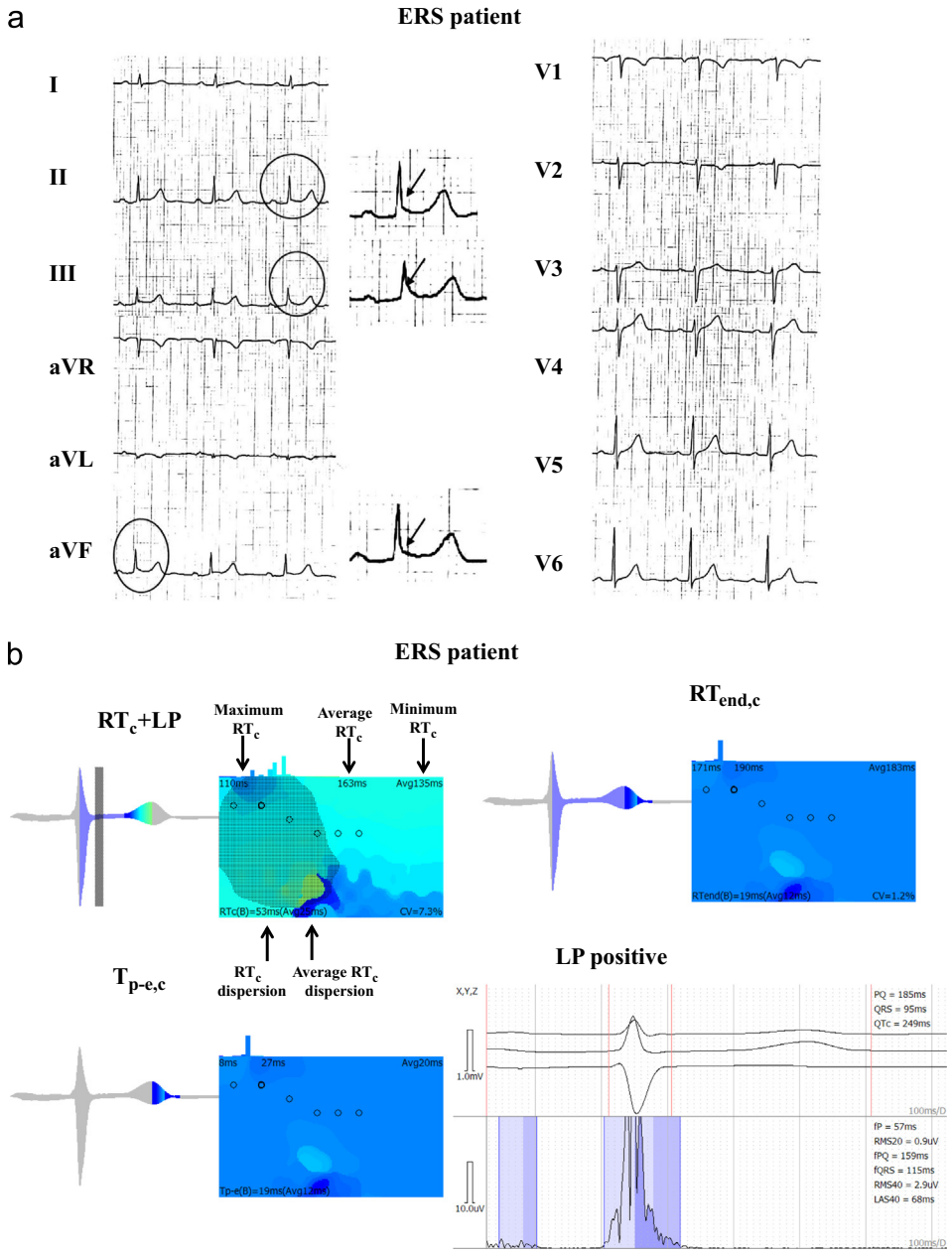

Fig. 3. (a) Twelve-lead ECG from a patient with early repolarization syndrome who was resuscitated from sudden cardiac death. Note the J wave in inferior limb leads II, III, and aVF. (b) Signal-averaged vector-projected 187-channel high-resolution ECG from the same patient with early repolarization syndrome (see Fig. 3a). The RT_c, RT_{end-c}, and $T_{(p-e)-c}$ maps were 256-color coordinated images according to the length of time intervals; blue color: < 40 ms, green–yellow: 40–79 ms.

repolarization abnormality markers (T-wave alternans, QT dispersion) did not differ between idiopathic VF patients with or without J waves.

In the present study, we found the average spatial repolarization interval to be shorter in ERS patients than in asymptomatic patients with the ER pattern, and we found a high incidence of positive late potentials in both ERS and ER pattern patients. Abe et al. reported that patients with idiopathic VF and the J wave had a high incidence of late potentials (86%) than those without the J wave, showing circadian variation with night ascendancy. However, repolarization abnormalities did not differ between the 2 types of patients, suggesting that the J wave is more closely associated with depolarization abnormalities and autonomic modulation than with repolarization abnormalities [19]. Therefore, the role of repolarization abnormalities and depolarization abnormalities in the pathogenesis of "malignant" forms of ER ECG pattern needs further investigation.

The induction of VF by programmed ventricular stimulation (6/8, 75%) was higher than that reported by Haissaguerre et al. (34%); however, the inducibility of VF with 3 extrastimuli increased VF induction to 81% [4].

4.3. Study limitations

The study was limited by the size of the study group. All patients enrolled in this study were from a single institution, and all ERS patients were survivors of VF or unexpected cardiac arrest. This may offer an explanation as to why results obtained in the present study contradicted the results of Antzelevitch et al., who reported that ER ECG type 2 is a significant marker of high risk and

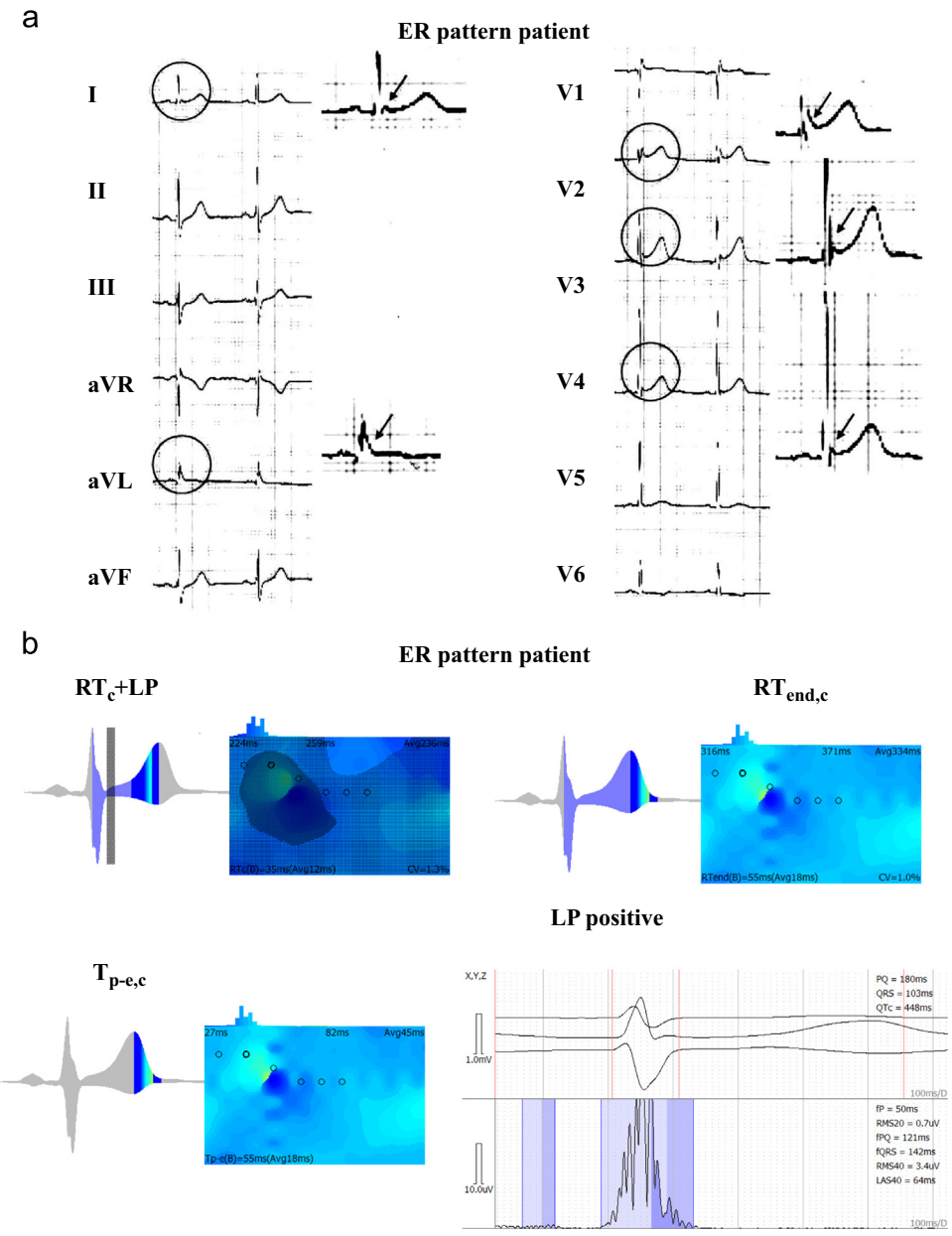

Fig. 4. (a) Twelve-lead ECG from an asymptomatic patient with an early repolarization ECG pattern. Note the J wave in inferior limb leads I, aVL, and V2-V4. (b) Signal-averaged vector-projected 187-channel high-resolution ECG from the same patient with early repolarization pattern (see Fig. 4a). The RT_c, RT_{end-c}, and $T_{(p-e)-c}$ maps were displayed as 256-color coordinated images according to the length of time intervals; blue color: <40 ms, green–yellow: 40–79 ms.

ER ECG type 1 is a marker of lower risk [6]. Therefore, our results need to be confirmed in a large-scale prospective study.

This study employed a novel technology for measuring spatial and transmural repolarization and its dispersion, which has so far only been employed by one group; they reported that the parameters analyzed using the 187-ch SAVP-ECG correlated with 64-channel magnetocardiography. However, the reliability of spatial and transmural repolarization and its dispersion using this technology needs to be evaluated in a multicenter study.

The investigators were not blinded to the group assignments, which could have introduced bias into the study. However, data were measured automatically, which reduced the potential for bias.

Finally, only one 187-ch SAVP-ECG was recorded for each patient; therefore, diurnal and daily variations in repolarization and depolarization parameters might have influenced the results [19].

5. Conclusion

An increased incidence of late potentials was noted in both ERS and ER pattern patients. Lethal arrhythmias in ERS patients appear to be related to the shorter average repolarization time, rather than the transmural repolarization time or spatial and transmural dispersion of repolarization. However, a large-scale study is necessary to identify the risk stratification in patients with an ER ECG pattern.

Conflicts of interest

The authors have no conflict of interest regarding the study described.

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