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ELECTROPHYSIOLOGY

The prognostic value of J-wave pattern for recurrence of ventricular tachycardia after catheter ablation in patients with myocardial infarction

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Abstract

Background: J-waves and fragmented QRS (fQRS) on surface ECGs have been associated with the occurrence of ventricular tachyarrhythmias. Whether these non-invasive parameters can also predict ventricular tachycardia (VT) recurrence after radiofrequency catheter ablation (RFCA) is unknown. Of interest, patients with a wide QRS-complex have been excluded from clinical studies on J-waves, although a J-wave like pattern has been described for wide QRS.

Methods: We retrospectively included 168 patients (67 ± 10 years; 146 men) who underwent RFCA of post-infarct VT. J-wave pattern were defined as J-point elevation ≥ 0.1 mV in at least two leads irrespective of QRS width. fQRS was defined as various RSR' pattern in patients with narrow QRS and more than two R wave in those with wide QRS. The primary endpoint was VT recurrence after RFCA up to 24 months.

Results: J-wave pattern and fQRS were present in 27 and 28 patients, respectively. Overlap of J-wave pattern and fQRS was observed in nine. During a median follow-up of 20 (interquartile range 9–24) months, 46 (27%) patients had VT recurrence. Kaplan–Meier curves revealed that both J-wave pattern and fQRS were associated with VT recurrence. Multivariate Cox regression analysis demonstrated that the presence of J-wave pattern (hazard ratio [HR] 2.84; 95% confidence interval [CI] 1.45–5.58; $P = .002$) and greater number of induced VT (HR 1.29; 95% CI 1.15–1.45; $P < .001$) were the independent predictors of VT recurrence.

Conclusions: A J-wave pattern—but not fQRS—is independently associated with an increased risk of post-infarct VT recurrence after RFCA irrespective of QRS width. This simple non-invasive parameter may identify patients who require additional treatment.

Abbreviations: CI, confidence interval; FQRS, fragmented QRS; HR, hazard ratio; ICD, implantable cardioverter defibrillator; IQR, interquartile range; LV, left ventricular; MI, myocardial infarction; RFCA, radiofrequency catheter ablation; RV, right ventricular; VT, ventricular tachycardia

KEYWORDS
fragmented QRS, J-waves, myocardial infarction, radiofrequency catheter ablation, ventricular tachycardia

1 | INTRODUCTION

Radiofrequency catheter ablation (RFCA) for ventricular tachycardia (VT) in post-myocardial infarction (MI) patients is increasingly employed. However, non-invasive parameters that may predict VT recurrence after RFCA are sparse. J-waves on 12-lead-ECGs have been associated with idiopathic ventricular fibrillation[1] and more recently with ventricular tachyarrhythmias in the acute[2] and chronic[3] phase of MI. Although J-waves have been recognized as early repolarization, the mechanisms of J-waves (early repolarization or delayed depolarization) are still controversial.[4] Slow conduction is a precondition for scar-related re-entry and may result in late activated myocardium contributing to J-wave pattern. Of note, prior clinical studies on J-waves have excluded patients with a wide QRS complex, which is frequently observed in patients referred for RFCA of VT, although, a J-wave like pattern has been described in patients with a wide QRS complex.[5] Fragmented QRS (fQRS) is another electrocardiographic parameter that has been associated with the substrate for ventricular tachyarrhythmias in patients with ischemic heart disease.[6] The purpose of the study is to evaluate if the presence of J-wave pattern and/or fQRS on pre-procedural ECGs as non-invasive surrogate for a VT substrate are associated with VT recurrence in patients with narrow and wide QRS complex undergoing RFCA of post-MI VT.

2 | METHODS

2.1 | Study subjects

We retrospectively included consecutive patients with prior MI and spontaneous sustained monomorphic VT who underwent a first RFCA procedure at the Leiden University Medical Center from 2009 to 2015. Patients who underwent surgical RFCA or presented with frequent premature ventricular contractions or non-sustained VT without documented spontaneous sustained VT were excluded. The diagnosis of MI was based on clinical history, the presence of Q waves, wall motion abnormalities, non-reversible perfusion defects, and/or subendocardial or transmural late gadolinium enhancement areas within the perfusion territory of a coronary artery with significant stenosis (>70%). MI was diagnosed if patients had not only one but a combination of the listed criteria.

On admission, patients underwent a comprehensive clinical evaluation. A transthoracic echocardiogram was routinely performed to assess the left ventricular (LV) function. Antiarrhythmic drugs except for amiodarone were discontinued for five half-lives before RFCA. All patients were treated according to our standard institutional protocol and provided informed consent for RFCA.

Written informed consent was not obtained. However, the Dutch Central Committee on Human-related Research (CCMO) permits use of anonymous data without prior approval of an Institutional Review Board, if the data are obtained for patient care and if the data do not contain identifiers that could be traced back to the individual patient.

2.2 | ECG analysis

In each patient, 12-lead ECGs were recorded on admission. No patients were excluded due to inability to assess a baseline ECG for J-wave pattern or fQRS. The median duration from pre-procedural ECG recording to VT ablation was 1 day (interquartile range [IQR] 0–1 days). The pre-procedural 12-lead ECG was independently analyzed by two experienced investigators (Y.N.; M.T.) blinded for all patient data. Details of the ECG analysis have been described previously.[3] Parameters included baseline rhythm (sinus rhythm or atrial fibrillation), heart rate, QRS duration, presence of J-wave pattern, and fQRS. J-wave pattern was defined as elevation of an end-QRS notch or slur on the downslope of a prominent R-wave of ≥0.1 mV in at least two contiguous inferior (II, III, and aVF) or lateral (I, aVL, and V4–V6) leads (Figure 1).[7] Of note, the same definition was applied to patients with a wide QRS complex of ≥120 milliseconds. Since the term "J-wave" is usually used in the presence of a narrow QRS complex, for the purpose of the current report, we will use the term "J-wave pattern" irrespective of QRS width.

fQRS was defined as the presence of an additional R wave (R'), or notching in the nadir of the R wave or the S wave in at least two contiguous inferior (II, III, and aVF), anterior (V1–V3), or lateral (I, aVL, and V4–6) leads in patients with a narrow QRS complex.[8] The terminal notching was categorized as both fQRS and J-wave pattern as this criterion meets both definitions. fQRS in patients with wide (≥120 milliseconds) QRS complex was defined as the presence of more than two notches in the R or the S wave in at least two contiguous leads[8] (examples are provided in Figure 2).

2.3 | Electrophysiological study and RFCA

Details of our mapping and ablation approach have been previously reported.[9] Briefly, studies were performed under conscious sedation or general anesthesia. The mapping and RFCA procedures were performed with an electroanatomic mapping system (CARTO, Biosense Webster, Diamond Bar, CA, USA) and an irrigated tip catheter (ThermoCool catheter, Biosense Webster). A total of 12-lead ECGs and intracardiac electrograms were displayed and recorded simultaneously on a 48-channel acquisition system (Prucka CardioLab EP system, GE Healthcare, USA). At the beginning of the procedure, an attempt to

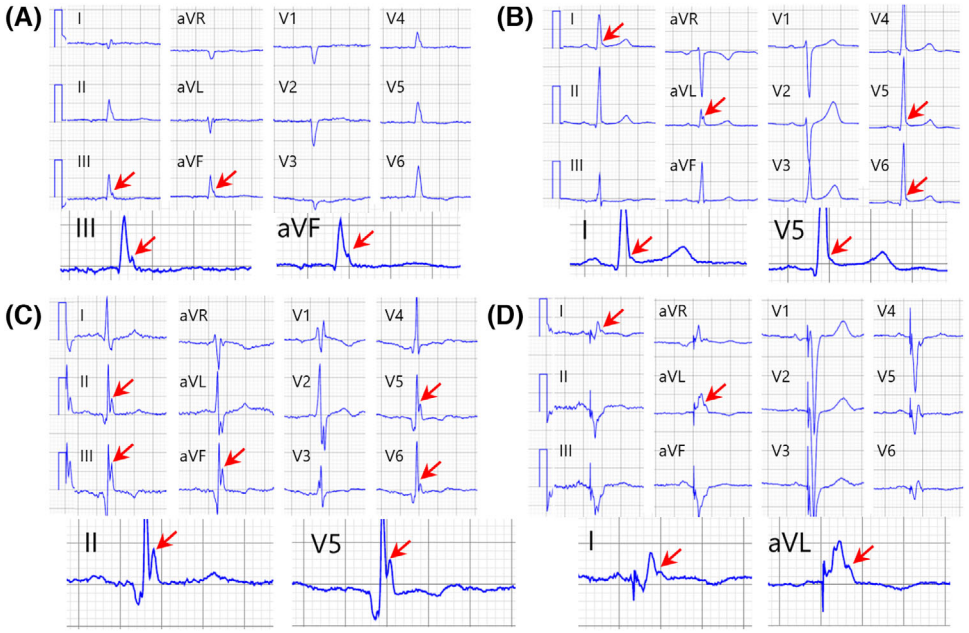

FIGURE 1 Examples of J-wave pattern on 12-lead ECGs. (A) Narrow QRS with notched J-wave pattern and horizontal/descending ST segment in the inferior leads after inferior myocardial infarction (MI). The terminal QRS notch also meets the definition of fQRS. (B) Narrow QRS with slurred J-wave pattern and horizontal/descending ST segment in the inferior and lateral leads after anterior MI. (C) A wide QRS with notched J-wave pattern with horizontal/descending ST segment in the inferior and lateral leads after inferior MI. (D) A wide QRS (pace14203d) with notched J-wave pattern with horizontal/descending ST segment in the lateral leads after a posterior MI. The arrows point to the J peak [Color figure can be viewed at wileyonlinelibrary.com]

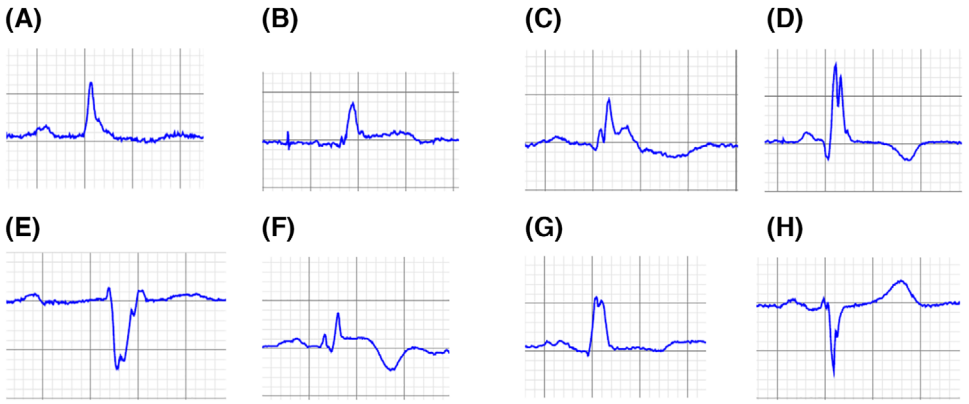

FIGURE 2 Examples of J-wave pattern and fQRS according to current definitions. (A) Slurred J-wave pattern with wide QRS. (B) Notched J-wave pattern with upsloping ST segment and narrow QRS. (C and D) fQRS with wide QRS. The examples also meet the definition of J-wave pattern. (E) fQRS with wide QRS (notching in S wave). (F, G, and H) fQRS with narrow QRS [Color figure can be viewed at wileyonlinelibrary.com]

VT induction was performed ([1] until October 2013: three drive cycle length [S1: cycle length = 600, 500, and 400 milliseconds], one to three extrastimuli until 200 milliseconds from two right ventricular (RV) sites and in addition from one LV site in selected patients and [2] since October 2013: four drive cycle lengths [S1: cycle length = 600, 500, 400, and 350 milliseconds], one to four ventricular extrastimuli [S2: ≥200 milliseconds] from two RV sites and more than or equal to one LV site). An induced VT was considered clinical when the 12-lead morphology and cycle length of the VT coincided with the clinically documented VT and presumed clinical VT when the cycle length was within 20 milliseconds of an implantable cardioverter defibrillator (ICD)-recorded VT. In all patients, detailed LV endocardial mapping (fill threshold < 15 mm) was performed through a retrograde or a combined retrograde and transseptal approach. The areas with bipolar voltages below 0.5 mV and between 0.5 and 1.5 mV were defined as dense scar and scar border zone, respectively.

All patients underwent substrate-based ablation. Between January 2009 and October 2013, ablation was based on elimination

of late potentials (onset after QRS, separated by an isoelectric line > 20 milliseconds) and fragmented electrograms. Since October 2013, a systematic pacing protocol was used for substrate identification.[9] In brief, EGMs located in the presumed infarct area, as derived from cardiac imaging and coronary anatomy, independently of their voltage or morphology during sinus rhythm were analyzed during RV pacing at a fixed rate of 500 milliseconds and during the application of a short-coupled RV extra-stimulus. Sites showing low voltage, near-field potentials that delayed >10 milliseconds or blocked in response to RV extra-stimulation were categorized as *evoked delayed potentials* and targeted for ablation. In addition, if hemodynamically stable VTs were repeatedly induced, activation and entrainment mapping was performed in an attempt to identify the critical VT isthmus and terminate the tachycardia by ablation. Radiofrequency energy was delivered (45–50 W, temperature limit 43°C, flow rate 20–30 mL/min) until stimulation with high output pacing (10 mA, 2 milliseconds) failed to capture. The same VT induction protocol was repeated at the end of the procedure. Complete procedural success was defined as non-inducibility of any sustained monomorphic VT; partial success as elimination of the (presumed) clinical VT but persistent inducibility of more than or equal to one nonclinical VT (including fast VT with a VTCL < 250 milliseconds considered of uncertain clinical relevance and not targeted by ablation); and failure as persistent inducibility of the (presumed) clinical VT.

2.4 | Acute and long-term outcome

Patients were followed 2 months after the procedure and every 6 months thereafter including clinical history for symptoms suggesting VT recurrence and ICD interrogation. Pre-procedural antiarrhythmic drugs were continued until the first follow-up visit. VT recurrence was defined as occurrence of any VT after RFCA lasting >30 seconds or requiring adequate therapy of ICD. In the absence of VT recurrence, discontinuation of antiarrhythmic drugs was recommended. The primary endpoint was VT recurrence up to 24 months after ablation.

2.5 | Statistical analysis

Continuous variables were expressed as means ± standard deviation or medians (IQR). Comparisons between groups were tested by an unpaired *t*-test or Mann–Whitney *U*-test according to the data distribution with or without normality. All categorical variables were presented as the number and percent in each group and were compared by a Fisher's exact test. A comparison of the probability of the freedom from VT recurrence between patients with and without J-wave pattern and fQRS was performed with Kaplan–Meier survival analysis tested by log-rank test. Univariable and multivariable Cox proportional regression analysis was performed to detect significant predictors for VT recurrence (reported as the hazard ratio [HR] with a 95% confidence interval [CI]). A *P*-value < .05 was considered statistically significant. The following variables were chosen as adjustment variables for multivariate analysis according to the literature reporting on clinical and procedural parameters associated with VT recurrence[10–13]: Number of clinical VT, LV ejection fraction, the number of induced VT, cycle length of induced VT, bipolar scar area, and non-inducibility. J-wave pattern and fQRS were also included in multivariate analysis based on our hypothesis. We constructed three multivariable models: model 1 including both pre-procedural and procedural parameters, model 2 including only pre-procedurally available parameters, and model 3 including only procedural parameters. All analyses were performed with R (The R Foundation for Statistical Computing, Vienna, Austria, version 3.1.1).

3 | RESULTS

3.1 | Baseline characteristics

The cohort consisted of 168 consecutive patients (87% men; age 67 ± 10 years) who underwent ablation for post-MI VT. The median time interval between the index MI and RFCA was 19 (IQR 13–26) years. A total of 31 (18%) patients had undergone acute reperfusion during the index MI. A total of 65 (39%) patients had an anterior MI. The mean EF was 33 ± 12% at the time of ablation. Baseline characteristics are provided in Table 1.

3.2 | ECG analysis

Mean heart rate during pre-procedural and postprocedural ECG analysis was 66.7 ± 11.9 and 66.8 ± 11.8 bpm, respectively. A total of 18 (11%) patients had atrial fibrillation at the time of the pre-procedural ECG. The mean QRS duration was 143 ± 37 milliseconds. Of note, 113 (67%) patients had a wide QRS (≥120 milliseconds), of which 41 had a pace14203d rhythm. Among the 41 patients with pace14203d rhythm, biventricular pacing was observed in 12 patients. J-wave pattern and fQRS were observed in 27 (16%) and 28 (17%) patients, respectively. Among the nine (5%) patients who had both J-wave pattern and fQRS, seven had a morphology that fulfilled both definitions for J-wave pattern and for fQRS (terminal QRS notch) and two patients had J-wave pattern at one location (e.g., inferior leads) and a fQRS at another (e.g., lateral leads). J-wave pattern and fQRS were observed in 19 (17%) and 16 (14%) of 113 patients with wide QRS complex, respectively. The prevalence of J-wave pattern and fQRS did not differ between the patients with narrow and wide QRS complex (15% vs. 17%; $P = .824$ and 14% vs. 22%; $P = .270$, respectively).

3.3 | Electrophysiological study and RFCA

A median of three (IQR 2–5) VTs with a mean cycle length of 354 ± 85 milliseconds were induced per patient. The clinical/presumed clinical VT was induced in 139 (83%) patients and only non-clinical

TABLE 1 Baseline characteristics of all patients

	All ($n = 168$)	VT recurrence ($n = 46$)	No VT recurrence ($n = 122$)	*P*-value
Age (year)	67 ± 10	67 ± 10	68 ± 10	.754
Male gender (*n* [%])	146 (87%)	44 (96%)	102 (84%)	.042
Comorbidity				
Hypertension (*n* [%])	70 (42%)	21 (46%)	49 (40%)	.599
Diabetes mellitus (*n* [%])	26 (15%)	6 (13%)	20 (16%)	.811
Hypercholesterolemia (*n* [%])	74 (44%)	20 (44%)	54 (44%)	1.000
Prior stroke/TIA (*n* [%])	14 (8%)	5 (11%)	9 (7%)	.533
Atrial fibrillation (*n* [%])	48 (29%)	14 (30%)	34 (28%)	.848
Heart failure (*n* [%])	75 (45%)	24 (52%)	51 (42%)	.297
Chronic kidney disease (*n* [%])	57 (34%)	14 (30%)	43 (35%)	.589
Anterior MI (*n* [%])	65 (39%)	15 (33%)	50 (41%)	.376
Time since MI (year)	19 (13–26)	19 (15–24)	19 (12–26)	.785
MI acute reperfusion (*n* [%])	31 (18%)	10 (22%)	21 (17%)	.509
Prior CABG (*n* [%])	62 (37%)	18 (39%)	44 (36%)	.723
Prior PCI (*n* [%])	67 (40%)	20 (44%)	47 (39%)	.598
Prior ICD (*n* [%])	119 (71%)	41 (89%)	78 (64%)	.001
LV ejection fraction (%)	33 ± 12	29 ± 11	35 ± 12	.004
Prior VT ablation (*n* [%])	26 (15%)	6 (13%)	20 (16%)	.811
Medication at admission				
Betablockers (*n* [%])	130 (77%)	38 (83%)	92 (75%)	.410
Amiodarone (*n* [%])	71 (42%)	22 (48%)	49 (40%)	.386
VT clinical presentation				
Number of clinical VT	1 (1–2)	1 (1–2)	1 (1–1)	.002
CL of clinical VT (ms)	387 ± 88	395 ± 84	384 ± 89	.512
Electrical storm (*n* [%])	28 (17%)	9 (20%)	19 (16%)	.643
Incessant VT (*n* [%])	18 (11%)	6 (13%)	12 (10%)	.580
Electrocardiographic findings				
Heart rate (bpm)	67 ± 12	64 ± 9	68 ± 13	.075
QRS duration (ms)	143 ± 37	153 ± 34	140 ± 37	.034
QRS duration ≥ 120 ms (*n* [%])	113 (67%)	40 (87%)	73 (60%)	.001
J-wave pattern (*n* [%])	27 (16%)	15 (33%)	12 (10%)	.001
Inferior J-wave pattern (*n* [%])	12 (7%)	6 (13%)	6 (5%)	.091
Lateral J-wave pattern (*n* [%])	9 (5%)	6 (13%)	3 (3%)	.014
Both inferior and lateral J-wave pattern (*n* [%])	6 (4%)	3 (7%)	3 (3%)	.347
fQRS (*n* [%])	28 (17%)	14 (30%)	14 (12%)	.005
Inferior fQRS (*n* [%])	13 (8%)	6 (13%)	7 (6%)	.191
Lateral fQRS (*n* [%])	6 (4%)	3 (7%)	3 (3%)	.347
Anterior fQRS (*n* [%])	3 (2%)	2 (4%)	1 (1%)	.182
Multiple fQRS (*n* [%])	6 (4%)	3 (7%)	3 (3%)	.347

(Continues)

TABLE 1 (Continued)

	All ($n = 168$)	VT recurrence ($n = 46$)	No VT recurrence ($n = 122$)	*P*-value
Either J-wave pattern or fQRS (*n* [%])	46 (27%)	22 (48%)	24 (20%)	<.001
Only J-wave pattern (*n* [%])	18 (11%)	8 (17%)	10 (8%)	.098
Only fQRS (*n* [%])	19 (11%)	7 (15%)	12 (10%)	.412
Both J-wave pattern and fQRS (*n* [%])	9 (5%)	7 (15%)	2 (2%)	.002

Values are reported as the mean ± standard deviation, median (interquartile range), or *n* (%).
Abbreviations: CABG, coronary artery bypass grafting; CL, cycle length; ICD, implantable cardioverter defibrillator; LV, left ventricle; MI, myocardial infarction; PCI, percutaneous coronary intervention; TIA, transient cerebral ischemic attack; VT, ventricular tachycardia.

TABLE 2 Procedural characteristics of all patients

	All ($n = 168$)	VT recurrence ($n = 46$)	No VT recurrence ($n = 122$)	*P*-value
Number of induced VTs	3 (2–5)	5 (3–6)	2 (1–4)	<.001
Inducibility before RFCA				.076
Non-inducible (*n* [%])	10 (6%)	0 (0%)	10 (8%)	
Inducible only non-clinical VTs (*n* [%])	16 (10%)	3 (7%)	13 (11%)	
Inducible clinical VT (*n* [%])	142 (85%)	43 (94%)	99 (81%)	
CL of induced VT (ms)	354 ± 86	367 ± 83	349 ± 87	.233
Bipolar scar area (cm^2)	65 (42–90)	86 (59–109)	58 (38–82)	.001
Dense scar area (cm^2)	24 (8–44)	33 (15–58)	22 (7–42)	.025
Border zone area (cm^2)	35 (23–48)	46 (34–57)	32 (21–42)	.001
Epicardial RFCA (*n* [%])	15 (9%)	4 (9%)	11 (9%)	1.000
Inducibility after RFCA				<.001
Non-inducible (*n* [%])	76 (45%)	12 (26%)	64 (53%)	
Non-clinical VTs remaining (*n* [%])	86 (51%)	33 (72%)	53 (43%)	
Clinical VTs remaining (*n* [%])	6 (4%)	1 (2%)	5 (4%)	
ICD after RFCA	145 (86%)	45 (98%)	100 (82%)	.005

Values are reported as the median (interquartile range) or *n* (%).
Abbreviations: CL, cycle length; ICD, implantable cardioverter defibrillator; RFCA, radiofrequency catheter ablation; VT, ventricular tachycardia.

VT in 15 (9%). LV endocardial mapping was performed in all patients (number of endocardial points 227 ± 76 points), right ventricular endocardial mapping in 18 (11%), and epicardial mapping in 15 (9%). The location of J-wave pattern and fQRS matched the infarct area in 24/27 (89%) and 15/28 (54%) patients, respectively. Complete procedural success was achieved in 76 (45%) patients, partial success in 86 (51%), and ablation failed in 6 (4%) (Table 2).

3.4 | Pre-procedural and procedural patient characteristics according to the presence of J-Wave pattern and fQRS

There were no significant differences in pre-procedural and procedural characteristics including age, gender, comorbidities, LV ejection fraction, ICD implantation rates, medication, number of induced VTs, bipolar scar area, and non-inducibility at the end of RFCA between patients with and without J-wave pattern. Only the prevalence of anterior MI was lower (11% vs. 44%; $P = .001$) in patients with J-wave pattern (Table S1).

Patients with fQRS had more often atrial fibrillation (46% vs. 25%; $P = .037$), a higher number of induced VTs (4 [3–5] vs. 3 [2–4]; $P = .014$), and a lower rate of non-inducibility after ablation (25% vs. 49%; $P = .039$) (Table S2).

3.5 | Long-term outcome and predictors of VT recurrence

During a median follow up of 20 (IQR 9–24) months, 46 (27%) patients had VT recurrence and 23 patients died. Kaplan–Meier analysis

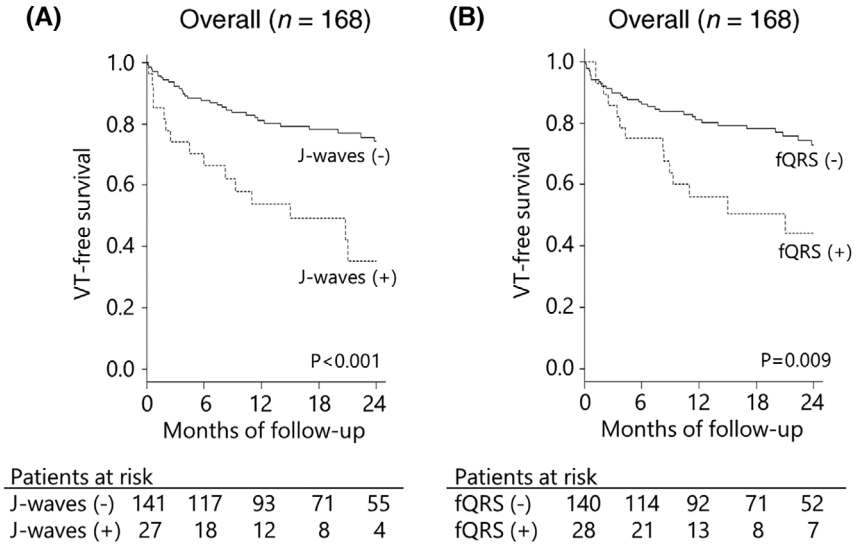

FIGURE 3 Kaplan–Meier curves. J-wave pattern (A) and fQRS (B) were associated with an increased risk of ventricular tachycardia (VT) recurrence after catheter ablation of post-MI VT

TABLE 3 Univariate and multivariate Cox proportional regression analyses of VT recurrence after RFCA

	Univariate		Multivariate	
Variables	**Hazard ratio(95% CI)**	***P*-value**	**Hazard ratio(95% CI)**	***P*-value**
LV ejection fraction (per 10% decrease)	1.45 (1.13–1.87)	.003	1.04 (0.75–1.45)	.807
J-wave pattern	3.23 (1.74–5.99)	<.001	2.81 (1.43–5.52)	.003
Fragmented QRS	2.48 (1.32–4.67)	.005	1.21 (0.59–2.48)	.605
Number of induced VT	1.35 (1.22–1.49)	<.001	1.27 (1.12–1.45)	<.001
Maximum CL of induced VT (per 10 ms increase)	1.04 (1.02–1.07)	.001	1.00 (0.97–1.04)	.875
Bipolar scar area (per 10 cm^2 increase)	1.12 (1.06–1.19)	<.001	1.08 (0.98–1.18)	.121
Non-inducible after RFCA	0.37 (0.19–0.72)	.003	0.86 (0.42–1.76)	.675

Abbreviations: CL, cycle length; LV, left ventricular; RFCA, radiofrequency catheter ablation; VT, ventricular tachycardia.

showed that patients with J-wave pattern had a significantly lower 24-month survival free from VT recurrence compared to those without J-wave pattern (35% [95% CI 15–56%] vs. 74% [95% CI 65–81%]; $P < .001$) (Figure 3). Similar, 24-month VT-free survival was lower in patients with fQRS than in those without fQRS (44% [95% CI 23–63%] vs. 73% [95% CI 64–80%]; $P = .003$) (Figure 3).

Multivariate Cox regression analysis revealed that the number of induced VT (HR 1.27 [95% CI 1.12–1.45]; $P < .001$) and the presence of J-wave pattern (HR 2.81 [95% CI 1.43–5.52]; $P = .003$) were independently associated with VT recurrence after ablation (Table 3).

When analyzing pre-procedural parameters separately, a lower LV ejection fraction (HR 1.30 [95% CI 1.10–1.66]; $P = .038$), the number of clinical VT (HR 1.34 [95% CI 1.00–1.78]; $P = .048$), and the presence of J-wave pattern (HR 2.33 [95% CI 1.18–4.59]; $P = .015$) were the pre-procedural independent predictors for VT (Table S3-1). The number of induced VT (HR 1.26; 95% CI 1.11–1.42, $P < .001$) and the bipolar scar area (HR 1.08; 95% CI 1.01–1.16, $P = .030$) were independently associated with VT recurrence after adjustment of procedural parameters (Table S3-2).

We performed a sub-analysis according to the ablation protocol (before and after the ablation was focused on identification of evoked delayed potentials). Out of 108 patients who underwent ablation without the guidance of evoked delayed potentials, 32 (30%) experienced VT recurrence. In these patients, the presence of J-wave pattern was associated with an increased risk of VT recurrence (HR 3.20 [95% CI 1.56–6.58]; $P = .002$). In 60 patients undergoing evoked delayed potentials ablation, VT recurrence occurred in 14 (23%) patients. Presence of J-wave pattern tended to be associated with the recurrence of VT (HR 3.05 [95% CI 0.85–10.97]; $P = .088$).

4 | DISCUSSION

4.1 | Main findings

To the best of our knowledge, this is the first study to evaluate the prevalence of J-wave pattern and fQRS on the baseline 12-lead ECG of patients with post-MI VT and to assess whether the presence of

J-wave pattern and/or fQRS are associated with higher rates of VT recurrence after ablation. The main findings can be summarized as follows: (1) The prevalence of J-wave pattern and fQRS, if determined irrespective of QRS widths, was relatively low in a post-MI population referred for catheter ablation of VT, in whom 68% had a QRS≥120 milliseconds. (2) In this population, the pre-procedural presence of J-wave pattern was an independent predictor for VT recurrence after endocardial ablation. The easily available, non-invasive parameters may identify patients who require additional treatment after ablation.

4.2 | J-wave pattern and VT substrates

Transmural dispersion of the cardiac action potential duration mediated by the I_{to}, $I_{K\text{-}ATP}$, and $I_{K\text{-}Ach}$ channels has been considered to be responsible for the inscription of the ECG J-waves and for the occurrence of phase 2 re-entry, which may induce scar-related VT.[4]

Although repolarization abnormalities were mainly recognized as a cause of J-waves, there is increasing evidence that J-waves may also be associated with delayed depolarization. Abe et al investigated 22 patients who experienced idiopathic ventricular fibrillation and showed that not repolarization abnormalities assessed by T-wave alternans and QT dispersion but depolarization abnormalities assessed by signal averaged ECG were associated with the presence of J-waves.[14] Others have demonstrated that fragmented late electrograms recorded from the right ventricular inferior subepicardium coincided with J-waves in patients with inferior J-wave syndrome.[15] In patients after MI, slow conduction as a precondition for scar-related re-entry results in areas of late activated myocardium that may also contribute to the genesis of J-wave pattern. Viable and late activated myocardium can be found in the scar border zone or at the subepicardium. Of interest, in our population, not the dense scar area but the size of the border zone tended to be larger in patients with J-wave pattern. VT recurrence after ablation may be due to lesion recovery or incomplete substrate elimination. It has been reported that an epicardial ablation approach is more often required in patients with inferior MI compared to anterior MI.[16] Therefore, it is interesting to speculate whether the higher incidence of J-wave pattern in patients with inferior MI in our cohort reflects a subepicardial substrate as potential explanation for the higher postprocedural VT recurrence. As we have performed epicardial mapping only in a minority of patients, we unfortunately cannot correlate the ECG findings with biventricular endo- and epicardial activation and voltage maps.

In particular in patients with a wide QRS complex, notching of the terminal portion of QRS complex is thought to be due to intraventricular conduction delay and not recognized as J-waves.[4] Prior reports have only focused on J-waves in patients with a narrow QRS complex. In the present study, we could demonstrate that J-wave pattern have prognostic implication on VT recurrence after RFCA also in patients with a wide QRS, which is a frequent finding in a population referred for RFCA of post-MI VT (67% in our cohort).

4.3 | fQRS

Previous studies have reported that an fQRS indicates myocardial scar in patients with MI and its presence has been associated with a higher mortality.[17] In MADIT II, an fQRS was present in 33% of patients with a normal QRS and in particular inferior fQRS location was found to be predictive of SCD/ICD shocks.[18] In an unselected population of patients with ischemic and non-ischemic cardiomyopathy who received ICDs for primary or secondary prevention, an fQRS was predictive for arrhythmic events.[6] These data support an association between fQRS and arrhythmogenic scar. In our cohort patients with fQRS had a higher number of inducible VTs during the procedure and remained more often inducible after ablation, suggesting a more complex VT substrate. Although Kaplan-Meier curve showed the association between fQRS and VT recurrence, fQRS was not independently associated with VT recurrence in the multivariate Cox regression analysis, most probably due to the low number of patients and the diagnostic overlap between J-wave pattern and fQRS.

4.4 | Clinical implication

Inducibility after RFCA, the number of induced VT and size of the scar area are known procedural factors associated with VT recurrence after ablation.[19,20] In line with prior reports, VT inducibility after ablation was indeed associated with VT recurrence in the univariate analysis. However, multivariate analysis failed to show that VT inducibility was an independent predictor for the recurrence of VT in this population. This finding is at variance with other reports in patients with post-infarct VT. One possible explanation is the introduction of an extensive, uniform reinduction protocol at the end of the procedure including up to four drive cycle lengths and up to four extrastimuli from RV and LV sites and the inclusion of patients with very fast induced VTs in the non-successful ablation group. This may contribute to a higher percentage of patients with non-procedural success compared to reports from other groups. Our study showed that the presence of J-wave pattern remains independently associated with an increased risk for VT recurrence after RFCA in post-MI patients. Pre-procedural non-invasive prognostic parameters that may contribute to patient selection or may identify patients who require additional treatment after ablation are sparse. The present study demonstrated that electrocardiographically recognized J-wave patterns were useful to identify patients with a higher risk for recurrence after ablation of post-infarct VT. This non-invasive parameter may identify patients who require additional treatment.

4.5 | Study limitations

Our study was a retrospective observational study with a relatively small sample size and should be considered as a hypothesis-generating

study rather than a conclusive trial. Further studies with a larger sample size are needed to confirm and enhance the present findings. The ablation protocol was changed during the study period. In 60 patients undergoing evoked delayed potentials ablation, the presence of J-wave pattern tended to be associated with the recurrence of VT, but did not reach statistical significance, likely due to the low number of patients and the low event rate in this subgroup.

The reported acute and long-term outcomes after RFCA come from a high-volume referral center and may therefore not apply for smaller less experienced centers. The antiarrhythmic regimen after RFCA was left at the discretion of the referral physician, and this might have influenced the outcome of some patients. The ventricular propagation wavefront could have influenced the presence or absence of J-wave pattern/fQRS, However, pacing mode was not systematically changed to assess the difference in the presence of J-wave pattern or fQRS among intrinsic QRS, RV pacing, and biventricular pacing in this study. We speculated that late activated viable myocardium could be responsible for J-wave pattern and could provide the link between the ECG parameters and VT recurrence but could not provide sufficient evidence due to the lack of whole heart activation mapping. Whether J-wave pattern reflects the presence of a subepicardial substrate requires additional studies correlating ECG findings with combined endo- and epicardial mapping data. Further studies are currently conducted to clarify this point.

5 | CONCLUSION

A J-wave pattern—but not fQRS—is independently associated with an increased risk of post-infarct VT recurrence after RFCA irrespective of QRS width. This simple non-invasive parameter may identify patients who require additional treatment.

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AUTHOR CONTRIBUTIONS

Yoshihisa Naruse has contributed to the conception and design of this work and to the drafting of the manuscript. Katja Zeppenfeld and Marta de Riva have contributed to data collection. Masaya Watanabe, Jeroen Venlet, and Marnix Timmer have contributed to the analysis and interpretation of the data. Marta de Riva, Adrianus P. Wijnmaalen, and Katja Zeppenfeld have contributed to critical revision of the manuscript for important intellectual content. Martin J. Schalij and Katja Zeppenfeld have contributed to final approval of the manuscript submitted.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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