Understanding ventricular tachycardia : towards individualized substrate-based therapy

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Chapter 6

Endocardial or Epicardial Ventricular Tachycardia in Nonischemic Cardiomyopathy?

The Role of 12-lead ECG Criteria in Clinical Practice

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ABSTRACT

Background
Specific 12-lead ECG criteria have been reported to predict an epicardial site of origin (SoO) of induced ventricular tachycardias (VTs) in left ventricular nonischemic cardio-myopathy (NICM).

Objectives
This study aimed to (1) determine the value of ECG criteria to predict an epicardial SoO of clinically documented VTs, (2) analyze the effect of VT cycle length (CL) and antiar-rhythmic drugs on the accuracy of ECG criteria, and (3) assess interobserver variability.

Methods
In 36 consecutive NICM patients (age 58±16y, 75% male) who underwent combined endocardial/epicardial VT ablation, all clinically documented and induced right bundle-branch block (RBBB) VTs were analyzed for previously reported ECG criteria to determine the SoO, as defined by ≥11/12 pacemap, concealed entrainment, and/or VT termination during ablation.

Results
In 21 patients with clinically documented (25mm/s) RBBB VT, none of the ECG criteria differentiated between patients with and without an epicardial SoO. In induced VTs (100mm/s), 2/4 interval criteria differentiated between an endocardial and epicardial SoO for slow VTs (CL>350ms) and 2/4 criteria in patients on amiodarone, but none for fast VTs (CL≤350ms) or those off amiodarone. The Q-wave in lead I was the most accurate criterion for an epicardial SoO (sensitivity, 88%; specificity, 80%).

In both clinically documented and induced VTs, interobserver agreement was poor for pseudodelta wave and moderate for other criteria.

Conclusion
When applied to clinically documented VTs, no ECG criterion could differentiate between patients with and without an epicardial SoO. Published interval-based ECG criteria do not apply to fast VTs and patients off amiodarone.

INTRODUCTION

In patients with ventricular tachycardia (VT) due to nonischemic cardiomyopathy (NICM), the substrate for VT is frequently—but not always—located intramurally or sub-epicardially.[1] Endocardial ablation and/or epicardial ablation may therefore be required to abolish VT. Prior studies have demonstrated that 12-lead ECG criteria can identify VTs with an epicardial site of origin (SoO), suggesting a need for epicardial access.[2-4]

Reported ECG criteria have been assessed for relatively slow VTs (average cycle length [CL] >390-400 ms[4,5]) on electrophysiology recording systems with electronic calipers at a sweep speed of 100 mm/s,[2-4] but their accuracy has not been evaluated on 25mm/s 12-lead ECGs of clinically documented VTs, which may be more relevant for planning the ablation approach.

In addition, the effect of VT CL and antiarrhythmic drug (AAD) use on the accuracy of ECG criteria has not been analyzed. In particular, interval criteria that depend on the determination of the VT QRS onset, preceded by an isoelectric interval, and the "earliest fast deflection" in precordial leads may be less accurate for fast VTs and prone to a high interobserver variability, thereby limiting its practical use.

The aims of the present study were to (1) determine the value of the reported ECG criteria when applied to 25 mm/s 12-lead ECGs of clinically documented VTs, (2) analyze the effect of VT CL and AAD use on the accuracy of the ECG criteria, and (3) assess the interobserver variability.

METHODS

Patients

Consecutive patients with NICM who underwent combined endocardial and epicardial VT ablation at the Leiden University Medical Center between 2007 and 2012 were included. The study excluded patients with coronary artery disease (>50% stenosis), congenital heart disease, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardio-myopathy, left ventricular noncompaction cardiomyopathy, restrictive cardiomyopathy, (sub)acute myocarditis, cardiac sarcoidosis, tachycardia-induced cardiomyopathy, or primary valvular disease. All patients were treated according to the clinical institutional protocol.

In patients undergoing elective VT ablation, AADs were discontinued for ≥5 half-lives, with the exception of amiodarone. Patients were admitted the day before the procedure and monitored with implantable cardioverter defibrillator therapy programmed off to allow recording of 12-lead ECGs of spontaneous VTs. Effort was taken to obtain 12-lead ECGs of all spontaneous VTs.

Electrophysiologic evaluation

Programmed electrical stimulation was performed under conscious sedation or general anesthesia (3 drive CL [600, 500, and 400ms], 1-3 ventricular extrastimuli [coupling interval ≥200ms], 2 right ventricular [RV] sites, and burst pacing, with isoproterenol [2-10µg/min] when necessary). The positive end point for stimulation was induction of a sustained monomorphic VT lasting for >30 seconds or requiring termination because of hemodynamic compromise.

Electroanatomical mapping and ablation

Epicardial access was obtained through a subxiphoid puncture. Electroanatomical mapping (EAM) was performed using a 3.5mm irrigated-tip NaviStar ThermoCool catheter (Biosense Webster Inc., Diamond Bar, CA, USA) and the CARTO™ system. Limited EAM of the aortic root was performed, and the left main coronary artery position, confirmed by undiluted contrast injection through the mapping catheter, was tagged on the map. The left main landmark and the surface registration tool were used to integrate the CT-derived coronary artery anatomy and epicardial fat thickness with the EAM.[6,7] The left ventricle (LV) was mapped retrogradely via the aorta, and the RV was mapped if indicated. After endocardial mapping, epicardial mapping of the region of interest was performed.

Ablation target sites were identified based on activation mapping and entrainment mapping for stable VT. For unstable VT, the area of interest was identified by substrate mapping and pace mapping. Then, VT was reinduced and briefly mapped in an attempt to identify diastolic activity and terminate the VT by ablation. In addition, limited substrate-based ablation was performed, targeting fragmented electrograms and late potentials in areas presumed to be related to VT based on pace, activation and entrainment mapping. Radiofrequency (RF) energy was applied at 30-45W (maximum temperature, 45°C; flow, 20-30 mL/min; 60 seconds) at endocardial sites and up to 50W (flow 20mL/min) at epicardial sites.

VT morphology and ECG criteria

Clinically documented and induced sustained monomorphic VTs were categorized as right or left bundle branch block-like (RBBB or LBBB) morphology (defined as predominant R or S in lead V_1), inferior or superior axis (predominant R or S in lead aVF), left or right axis (predominant R or S in lead I), and by precordial transition (first lead with a predominant R or S for LBBB and RBBB VTs, respectively). The ECG features assessed for all RBBB-like morphology VTs, as previously described[2-4], were:

- **QRS duration**: interval from the earliest ventricular activation to the offset of the QRS in the precordial leads.
- **Diastolic interval**: VT CL minus QRS duration.

- - *Pseudodelta wave (PDW)*: interval from the earliest ventricular activation to the onset of the earliest fast deflection in any precordial lead.
- - *Intrinsicoid deflection time in V_2 (IDT)*: interval from the earliest ventricular activation to the peak of the R wave in V_2.
- - *Shortest RS complex (SRS)*: interval from the earliest ventricular activation to the nadir of the first S wave in any precordial lead.
- - *Maximum deflection index (MDI)*: interval from the earliest ventricular activation to the peak of the largest amplitude deflection in each precordial lead (taking the lead with the shortest time) divided by the QRS duration, as described in patients with idiopathic VT.[5]
- - *Q wave in lead I (Q-I)*: initial negative deflection in lead I, occasionally preceded by a short isoelectric segment.
- - *Q waves in inferior leads (Q-INF)*: initial negative deflections in lead II, III, and aVF, occasionally preceded by a short isoelectric segment.
- - *Absence of Q waves in inferior leads (Abs-Q-INF)*: initial positive deflection in lead II, III, or aVF, occasionally preceded by a short isoelectric segment.

Previously published cutoff values were applied (≥34 ms for PDW, ≥85 ms for IDT, ≥121 ms for SRS, and ≥0.45 for MDI).[2] The PDW, IDT, SRS, and MDI are further referred to as "interval" criteria, and the Q-wave criteria are referred to as "morphology" criteria. For the Q-I and Abs-Q-INF criteria, only inferior-axis VTs were analyzed, and for the Q-INF criterion, only superior-axis VTs were analyzed.

Clinical VTs that were documented on a 25mm/s, 10mm/mV 12-lead ECG were analyzed using pens and manual calipers. Induced VTs were analyzed using electronic calipers on an electrophysiologic recording system (Prucka CardioLab EP; GE Healthcare, Waukesha, WI, USA) at a sweep speed of 100mm/s and an amplification of 10mm/mV. All measurements were performed by one observer (SP) and repeated by another independent observer (MRS) to analyze the interobserver reproducibility. Both observers were blinded to the SoO.

Site of origin

The SoO of a VT was defined as a site that was considered as ablation target site, with (1) the best pace map (≥11/12 leads match), (2) entrainment suggestive of an isthmus site (i.e., concealed entrainment with the postpacing interval equaling VT CL), and/or (3) VT slowing and termination during ablation. An induced VT was considered as a clinical VT if the 12-lead ECG showed an exact morphologic match with the clinically documented VT in ≥11/12 leads.

To analyze the value of ECG criteria, VTs with a SoO located at the epicardium or in the cardiac venous system were classified as epicardial VTs and those with a SoO at the LV endocardium as endocardial VTs. The analyses excluded VTs with a SoO located in the aortic sinus cusps and with both endocardial and epicardial sites fulfilling the definition of a SoO.

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ECG criteria and the need for an epicardial approach

To assess the value of the ECG criteria in standard 12-lead VT ECGs to predict the require-ment for an epicardial approach, the following analyses were performed: (1) the presence of an epicardial SoO was compared between clinical VTs with positive vs negative ECG, (2) the presence of an epicardial SoO of any spontaneous or induced VT was compared between patients with positive vs negative ECG criteria for the clinically documented VT, and (3) the number of epicardial RF applications was compared between patients with positive vs negative ECG criteria to analyze the potential therapeutic consequences of ECG criteria.

Statistics

Statistical analyses were performed with SPSS 20 software (IBM, Armonk, NY, USA). The categoric variables are expressed as number (percentage), and continuous variables as mean ± standard deviation, or median and interquartile range (IQR) where appropriate. Fisher exact tests, χ^2 tests and Mann-Whitney U tests were performed where applicable. For each ECG criterion, the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated.

To analyze the effect of VT CL and AADs on the accuracy of the ECG criteria, the VTs were categorized according to CL (prespecified as ≤350ms or >350ms) and according to amiodarone use.

The percentage interobserver agreement and the Cohen's Kappa value were calcu-lated for all criteria. The interobserver variability was analyzed by Bland-Altman plots for continuous parameters. The mean bias (mean difference between observer 1 and 2) and the 95% limits of agreement are reported. All p values are 2-tailed, and a value of $p < 0.05$ was considered to be statistically significant.

RESULTS

Baseline characteristics

A total of 36 NICM patients (age, 58±16 years; LV ejection fraction, 42±14%) underwent combined endocardial and epicardial VT ablation (Table 1); of these, 13 (36%) had VT recurrence after prior endocardial ablation, 6 (17%) were rescheduled for combined endocardial and epicardial VT ablation after a failed endocardial procedure, and the remaining 17 (47%) had no prior VT ablation.

Clinically documented VTs

Before ablation, 34 distinct VTs in 25 patients were documented on 12-lead ECGs (ex-amples in Figure 1). Of these VTs, 18 (53%) were recorded in our center and 16 (47%) in

Table 1. Baseline and procedural data

	N=36
Age, years	58±16
Male	27 (75%)
LV ejection fraction, %	42±14
LV end-diastolic volume, mL	162±48
History of atrial flutter/fibrillation	8 (22%)
eGFR < 60 mL/min/1.73m^2	2 (6%)
Anti-arrhythmic drugs	
Class I	5 (14%)
Class II	20 (56%)
Class III–Sotalol	8 (22%)
Class III–Amiodarone	16 (44%)
Class IV	0 (0%)
Number of VTs induced	3 (1–4)
BBR VT	1 (3%)
Unstable induced VT	27 (75%)
Mean CL of induced VTs, ms	346±74
Endocardial RF applications	25 (69%)
Epicardial RF applications	22 (61%)
Acute outcome	
Complete success	11 (31%)
Partial success	16 (44%)
Failure	9 (25%)

BBR, bundle-branch re-entry; CL, cycle length; LV, left ventricular; eGFR, estimated glomerular filtration rate; RF, radiofrequency energy; VT, ventricular tachycardia

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other centers. Six VTs (18%) were documented while the patient was on a class I AAD, 6 (18%) on sotalol, 20 (59%) on amiodarone, and 1 (3%) on a class IV AAD. Four patients (12%) were on 2 AADs, 25 (74%) on 1 ADD, and 5 (15%) were off AADs.

Mapping and ablation

A total of 111 distinct VTs (CL 342±89ms) were induced in 34 patients; of these, 66 VTs (59%) in 32 patients (94%) had RBBB-like morphology and were further analyzed. A SoO could be identified for 45 of these 66 VTs (68%) in 28 of 32 patients (88%). A ≥11/12 pace map was obtained for 34 VTs (52%), with a median S-QRS interval of 40ms (IQR, 15–65ms). Entrainment was concealed, and the postpacing interval was equal to the VT CL in 3 VTs (5%), with S-QRS intervals of 9%, 17%, and 46% of VT CL. During ablation, 21 VTs (32%) slowed and terminated without a premature ventricular contraction, with a median E-QRS percentage of VT CL before ablation of 24% (IQR 14%–35%) and median
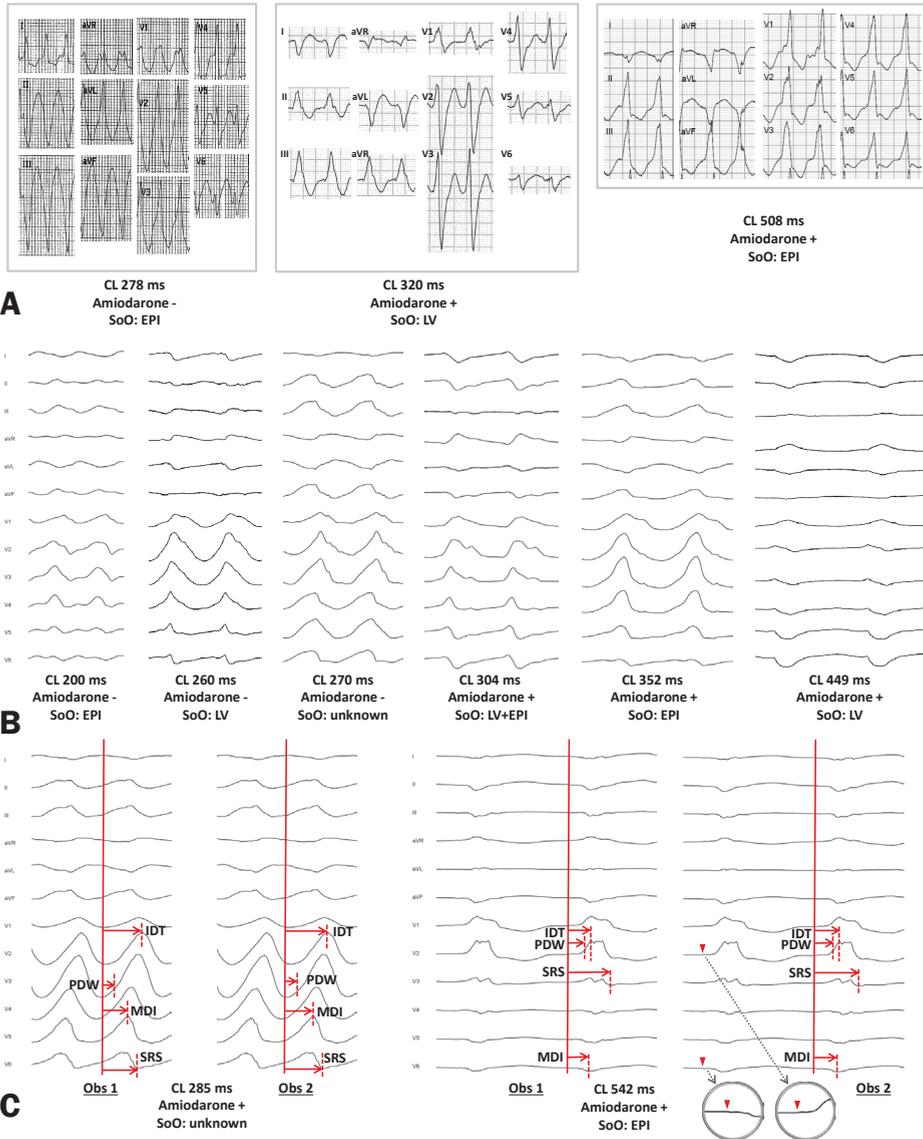
Figure 1. Examples of induced and clinically documented VTs

Panel A: Examples of clinically documented spontaneous VTs displayed at 25 mm/s. Panel B: Examples of induced VTs displayed at 100 mm/s. Panel C: Measurements of observers 1 and 2 for one fast and one slow VT. For the fast VT, the onset of the QRS complex is defined differently by observer 1 and 2, affecting all interval criteria. For the slow VT, a slurred onset of the QRS complex is observed (indicated with triangles), which may be difficult to detect if VT is faster, with a potential overlap with the preceding T-wave. CL, cycle length; SoO, site of origin.

termination time of 11 seconds (IQR, 8–29 seconds). The SoOs were classified as epicardial in 15 patients (54%), as endocardial in 6 (21%), and as endocardial and epicardial SoO in 7 (25%). Epicardial ablation was performed in 22 patients (61%).

ECG criteria applied to clinically documented VTs

Of 66 induced RBBB VTs, 19 (29%) corresponded to a clinically documented VT. Twelve of these 19 clinical VTs (63%) had a SoO at the LV epicardium, 3 (11%) at the LV endocardium, 1 (4%) in the LV and at the epicardium (termination In the LV, best pacemap at the epicardium), and 1 (4%) in the aortic sinus cusps. No SoO could be identified for 2 VTs (7%). The 15 VTs with a SoO categorized as LV endocardial or epicardial were further analyzed.

When applied to the clinically documented VTs, none of the interval criteria differed between endocardial and epicardial VTs (Table 2). The value of the Q-wave criteria could not reliably be assessed due to the limited number of endocardial and epicardial VTs in the inferior and superior axes subgroups.

The percentage of patients with an epicardial SoO of at least 1 RBBB VT was not significantly different between patients with positive or negative ECG criteria for the clinical VTs (Table 2). Depending on the applied criterion, 33% to 100% of patients with a negative criterion for the clinically documented VT had at least 1 epicardial RBBB VT necessitating an epicardial approach.

The number of epicardial RF applications tended to be higher in patients with positive vs negative criteria for clinical VTs, which was statistically significant for the Q-I criterion (Table 2), suggesting that the substrate may be more amenable to epicardial ablation in patients with a Q-wave in lead I during VT.

ECG criteria applied to induced VTs

For induced RBBB VTs, the SoO were located at the epicardium for 26 of 66 VTs (39%), at the endocardial LV for 11 VTs (17%), at the epicardium and at the endocardial LV for 4 VTs (6%), in the aortic root for 2 VTs (3%), and in the anterior cardiac vein for 2 VTs (3%). No SoO could be identified for the remaining 21 VTs (32%). The 39 VTs with a SoO categorized as LV endocardial or epicardial were further analyzed.

When measured in these induced VTs (examples in Figure 1), the PDW (p=0.040), IDT (p=0.008), SRS (p=0.022), and MDI (p=0.034), but not the QRS duration (p=0.32), differed significantly between endocardial and epicardial VTs (Figure 2). Of interest, the ECG criteria were similar for epicardial VTs and VTs of unknown origin (all p>0.05, interval criteria displayed in Figure 2). The most accurate ECG criteria to distinguish epicardial from endocardial VTs were Q-I for inferior-axis VTs (sensitivity, 88%; specificity, 80%) and Q-INF for superior-axis VTs (sensitivity, 73%; specificity, 67%). The diagnostic accuracy of all criteria is displayed in Figure 3.

**Table 2. ECG criteria and the need for an epicardial approach**

	Presence of an epicardial SoO of clinically documented VT if... (per VT)			Presence of an epicardial SoO of any clinically documented or induced VT if... (per patient)			Number of epicardial RF applications if... (per patient)		
	Criterion positive	Criterion negative	p	Criterion positive	Criterion negative	p	Criterion positive	Criterion negative	p
PDW ≥34 ms	3/3 (100%)	9/12 (75%)	1.00	4/5 (80%)	12/16 (75%)	1.00	8 (1-23)	8 (0-12)	0.77
IDT ≥85 ms	11/13 (85%)	1/2 (50%)	0.37	14/17 (82%)	2/4 (50%)	0.23	8 (1-17)	5 (0-11)	0.34
SRS ≥121 ms	11/13 (85%)	1/2 (50%)	0.37	14/17 (82%)	2/4 (50%)	0.23	10 (1-17)	4 (0-9)	0.20
MDI ≥0.45	7/8 (88%)	5/7 (71%)	0.57	9/11 (82%)	7/10 (70%)	0.64	11 (0-21)	5 (0-9)	0.20
Q-I*	1/2 (50%)	6/6 (100%)	0.25	4/4 (100%)	7/11 (64%)	0.52	12 (10-24)	0 (0-8)	0.043
Abs-Q-INF*	7/8 (88%)	N/A	N/A	10/12 (83%)	1/3 (33%)	0.15	8 (0-12)	0 (0-28)	0.66
Q-INF†	3/4 (75%)	2/3 (67%)	1.00	3/5 (60%)	4/4 (100%)	0.44	11 (1-26)	9 (7-18)	0.81

Abs-Q-INF, absence of Q-waves in inferior leads; IDT, intrinsicoid deflection time in V2; MDI, maximum deflection index; PDW, pseudodelta wave; Q-I, Q-wave in lead I; Q-INF, Q-wave in inferior leads; RF, radiofrequency energy; SoO, site of origin; SRS, shortest RS complex; VT, ventricular tachycardia.

*Only for inferior-axis VTs.

†Only for superior-axis VTs.
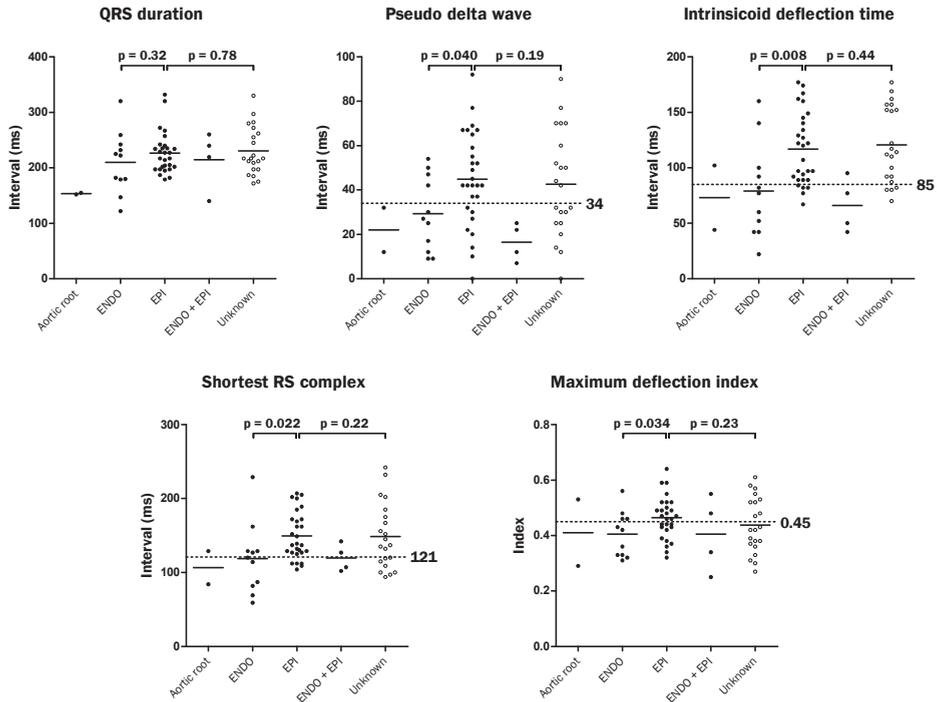
Figure 2. Interval criteria according to the site of origin of induced VTs

Effect of VT cycle length

For induced slow VTs (CL ≥350ms, n=16), the QRS duration, IDT and the SRS were significantly longer for epicardial VTs compared with endocardial VTs (Table 3). A trend was observed for a longer PDW and MDI. However, for fast VTs (CL <350ms, n=23), none of the interval criteria differed significantly between endocardial and epicardial VTs. The QRS duration was similar for slow and fast VTs (235±43 vs 213±41ms, p=0.19), but the diastolic interval was significantly longer in slow VTs than in fast VTs (median, 228ms [IQR, 174-302ms] vs 58ms [IQR, 27-92ms], p<0.001).

Effect of amiodarone use

For the 15 induced VTs in patients on amiodarone, the IDT and SRS were significantly longer in epicardial VTs than in endocardial VTs. The QRS duration, PDW and MDI also tended to be longer in epicardial VTs (Table 4). For the 24 VTs in patients off amiodarone, however, none of the interval criteria could distinguish epicardial from endocardial VTs. The QRS duration was similar in patients on and off amiodarone (228±53ms vs 218±36ms, p=0.62); however, compared with patients off amiodarone, patients on amiodarone had a significantly longer CL (422±91 ms vs 306±89ms, p<0.001) and median diastolic interval (205 ms [IQR 80-285ms] vs 62 ms [IQR 42-114ms], p=0.002).
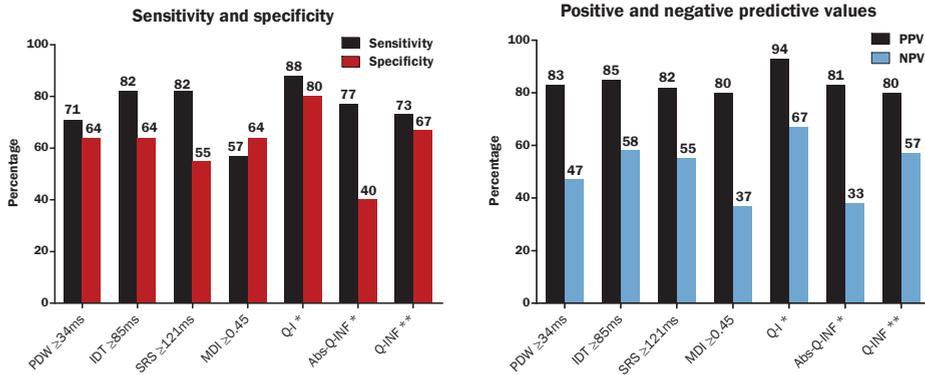
Figure 3. Accuracy of ECG criteria applied to induced VTs

NPV, negative predictive value; PPV, positive predictive value.

* Only for inferior-axis VTs   ** Only for superior-axis VTs

Table 3. Effect of VT cycle length on the accuracy of ECG criteria in induced VTs

	Slow VT (CL ≥350ms, n=16)			Fast VT (CL <350ms, n=23)		
	Epicardial vs. endocardial VTs (n=11 vs. n=5)		p	Epicardial vs. endocardial VTs (n=17 vs. n=6)		p
QRS, ms	247±45 vs. 208±26		0.038	214±25 vs. 212±75		0.92
PDW, ms	55±22 vs. 31±18		0.052	38±19 vs. 28±17		0.20
IDT, ms	130±38 vs. 61±19		0.002	108±26 vs. 94±52		0.81
SRS, ms	175±28 vs. 118±18		<0.001	133±23 vs. 119±65		0.32
MDI	0.46±0.08 vs. 0.39±0.07		0.12	0.47±0.07 vs. 0.42±0.09		0.16
Q-I, %*	7/8 (88%) vs. 1/3 (33%)		0.15	8/9 (89%) vs. 0/2 (0%)		0.055
Abs-Q-INF, %*	7/8 (88%) vs. 1/3 (33%)		0.15	6/9 (67%) vs. 2/2 (100%)		1.00
Q-INF, %†	2/3 (67%) vs. 1/2 (50%)		1.00	6/8 (75%) vs. 1/4 (25%)		0.22

Abbreviations as in Table 2.   *Only for inferior-axis VTs   †Only for superior-axis VTs

Table 4. Effect of amiodarone on the accuracy of ECG criteria in induced VTs

	VTs on amiodarone (n=15)			VTs off amiodarone (n=24)		
	Epicardial vs. endocardial VTs (n=8 vs. n=7)		p	Epicardial vs. endocardial VTs (n=20 vs. n=4)		p
QRS, ms	242±43 vs. 211±61		0.12	220±34 vs. 208±52		0.79
PDW, ms	53±24 vs. 28±17		0.054	42±20 vs. 32±18		0.39
IDT, ms	123±37 vs. 71±34		0.009	114±31 vs. 94±57		0.63
SRS, ms	176±25 vs. 127±51		0.040	139±29 vs. 104±45		0.16
MDI	0.48±0.10 vs. 0.40±0.07		0.072	0.46±0.07 vs. 0.42±0.11		0.48
Q-I, %*	4/5 (80%) vs. 1/3 (33%)		0.46	11/12 (92%) vs. 0/2 (0%)		0.033
Abs-Q-INF, %*	5/5 (100%) vs. 2/3 (67%)		0.11	8/12 (67%) vs. 2/2 (100%)		1.00
Q-INF, %†	2/3 (67%) vs. 2/4 (50%)		1.00	6/8 (75%) vs. 0/2 (0%)		0.13

Abbreviations as in Table 2.   *Only for inferior-axis VTs   †Only for superior-axis VTs

Because the subgroups of endocardial and epicardial VTs with inferior and superior axes were small, the effects of VT CL and amiodarone on the Q-wave criteria could not be reliably analyzed.

Interobserver variability of ECG criteria

Bland-Altman plots of interval criteria measurements are provided in Supplemental Figures 1 and 2. The mean bias for the interval criteria was high, and the 95% limits of agreement were wide for induced VTs and, in particular, for the clinically documented VTs (Table 5). The interobserver agreement for clinically documented and induced VTs was poor for the PDW, SRS, and MDI, but moderate or good for IDT, Q-I, and Q-INF.

Table 5. Interobserver variability and agreement

	Clinically documented VTs (n=27)					Induced VTs (n=66)				
	Mean±SD or %	Mean bias	95% limits of agreement	Interobserver agreement, %	Cohen's Kappa	Mean±SD or %	Mean bias	95% limits of agreement	Interobserver agreement, %	Cohen's Kappa
QRS, ms	265±51	43	-33–119	NA	NA	222±44	27	-41–96	NA	NA
PDW, ms	18±15	27	-88–34	44%	0.08	39±22	4	-48–55	68%	0.37
IDT, ms	132±54	24	-62–110	85%	0.51	107±39	7	-42–56	82%	0.61
SRS, ms	172±56	25	-88–139	74%	0.38	141±40	19	-48–86	74%	0.51
MDI	0.46±0.11	0.02	-0.20–0.24	63%	0.26	0.44±0.09	0.02	-0.18–0.23	68%	0.36
Q-I*	31%	NA	NA	77%	0.42	66%	NA	NA	74%	0.44
Abs-Q-INF*	0%	NA	NA	100%	1.00	23%	NA	NA	86%	0.53
Q-INF†	50%	NA	NA	93%	0.86	48%	NA	NA	84%	0.68

Abbreviations as in Table 2.
*Only for inferior-axis VTs (induced VTs, n=35; clinically documented VTs, n=13).
†Only for superior-axis VTs (induced VTs, n=31; clinically documented VTs, n=14).

DISCUSSION

The clinical VT documentation in the present study demonstrated that the suggested ECG criteria could not reliably identify NICM patients who needed an epicardial approach. When applied to induced VTs on 100 mm/s electrophysiology recording system tracings, some ECG criteria proved useful for the identification of epicardial VTs, but not for all VT subgroups. Two of 4 interval criteria differed between induced endocardial and epicardial slow VTs (CL >350ms) and 2 of 4 criteria in patients on amiodarone, but none when applied to fast VTs (CL ≤350ms) or in patients off amiodarone. The Q-wave in lead I (Q-I) was the most accurate criterion. In clinically documented and induced VTs, the interobserver agreement was poor for the PDW but moderate for IDT, Q-I, and Q-INF.

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ECG criteria applied to clinically documented VTs: the need for an epicardial approach

Applying ECG criteria to the 12-lead VT ECG for the identification of a potential epicardial SoO has been considered to be useful for selection of patients who may benefit from epicardial ablation.[2-4] To assist preprocedural planning, ECG criteria are typically applied to clinically documented VTs. However, this approach may have important limitations. First, measurements need to be performed on 25mm/s 12-lead ECGs, which may be less accurate than on 100mm/s recordings as used in the electrophysiology laboratory.

Second, not clinically documented VTs are frequently induced, which may originate from other parts of the scar and need to be targeted if noninducibility is pursued. In the current study, at least 1 VT originated from the epicardium in 79% of patients with induced RBBB VTs.

Third, 12-lead ECG morphology and thereby ECG criteria are particularly influenced by the VT exit site; however, the VT may be abolished by ablation at any part of the reentry circuit.

Based on the present data we conclude that, although potentially useful as a guide to the area of interest for VTs induced during the procedure, the ECG criteria do probably not allow identification of patients who are likely to benefit from an epicardial approach.

ECG criteria applied to induced VTs

When applied to induced VTs, most of the ECG criteria had a high PPV, indicating that an epicardial SoO may be likely when criteria are positive. All criteria have moderate and often even poor NPV, indicating that induced VTs may still originate and be targeted from the epicardium if the ECG criteria are negative. Interestingly, the ECG criteria did not differ between epicardial VTs and VTs of unknown origin, which is in line with prior observations,[4] perhaps suggesting that most of these VTs may also be of epicardial origin. Thick layers of epicardial fat and the coronary arteries may have prevented the identification of some epicardial SoO.

In NICM, the effect of the VT CL and AADs on the accuracy of ECG criteria has not been analyzed. The initial reports on ECG criteria have included VTs with a mean CL of >400ms[4] and 390ms.[5] However, in patients with NICM currently considered for VT abla-tion, fast VTs are frequently induced (CL 342±89ms in this study and CL 346±74ms in a recent report[8]), and therefore, evaluation of the accuracy of ECG criteria for fast VTs is important. All criteria rely on an accurate identification of the onset of the QRS complex, which may be particularly difficult to define when the QRS is broad, in the absence of an isoelectric interval, and when there is overlap with a previous T-wave, which is commonly observed in fast VTs. In the present study, the IDT and SRS differed between endocardial and epicardial slow (CL >350ms), but not fast, VTs (CL ≤350ms). The IDT and SRS also differed between endocardial and epicardial VTs in patients with amiodarone,

but not in patients without amiodarone. Although the number of RBBB inferior-axis VTs was limited, the Q-I criterion appeared not to be affected by VT CL or amiodarone use. The diastolic interval was significantly shorter in fast VTs and in patients off amiodarone, which may hamper determination of the QRS onset. Owing to the limited number of VTs, the independent effects of VT CL and amiodarone use could not be analyzed.

Interobserver agreement

Only one study in postmyocardial infarction patients reported interobserver agreement of ECG criteria for epicardial VTs.[9] The interobserver agreement in the present study was particularly poor for the PDW criterion, as has been observed in postmyocardial infarction patients,[9] and for the MDI, which relies on an accurate identification of the QRS onset, QRS offset, and maximum deflection in all precordial leads. In contrast, the IDT relies only on the identification of the onset of the QRS and the well-defined peak of the R wave in V_2, perhaps explaining the relatively high interobserver agreement. The Q-wave criteria are typically easier to assess, with moderate to good interobserver agreement, but may be less clear when the onset of the QRS is difficult to define or when a putative initial R wave is very small.

Limitations

The number of patients and VTs was too small for analysis in some subgroups. Owing to inclusion of patients after endocardial ablation failure, it may be expected that the number of NICM patients with an epicardial SoO will be overrepresented. However, among the 32 patients with inducible RBBB VT, 9 of 14 patients (64%) who had not undergone prior ablation had an epicardial SoO compared with 13 of 18 patients (72%) who had undergone prior endocardial ablation (p=0.71).

The SoO could be identified for only 68% of induced RBBB VTs, with ≥1 identified SoO in 88% of patients with inducible RBBB VTs, which may be explained by the strict definition of a SoO, intramural reentry circuits, epicardial SoOs covered with epicardial fat, fast VTs without a reasonable pacemap, and other reasons.

Limb leads are typically placed on the torso in the electrophysiology lab, while the clinically documented VT may be recorded with the leads placed on the extremities. The resulting differences in extremity lead vectors may affect Q-waves, but were not analyzed in the present study.

CONCLUSION

When applied to 25mm/s ECGs of clinically documented VTs, none of the ECG criteria could differentiate between patients with and without epicardial VTs. These data sug-

gest that the ECG criteria do not allow identification of patients who are likely to benefit from a primary epicardial ablation approach. The VT CL and the use of AADs affect the accuracy of ECG criteria in induced VTs.

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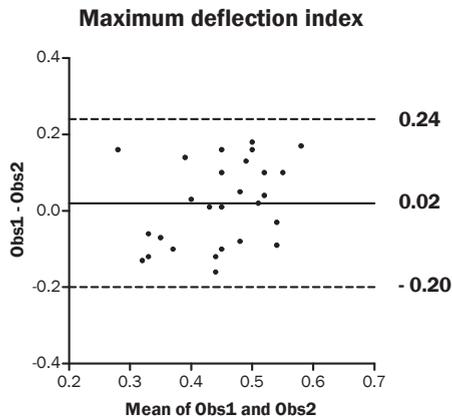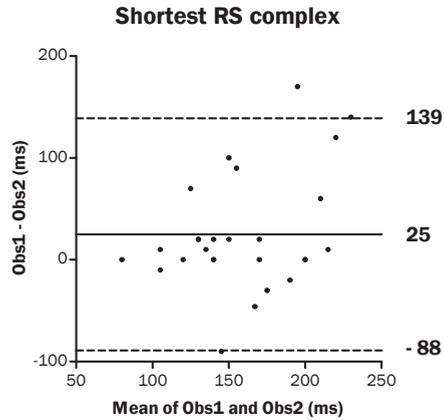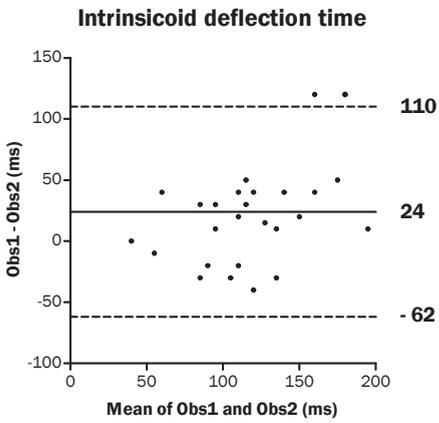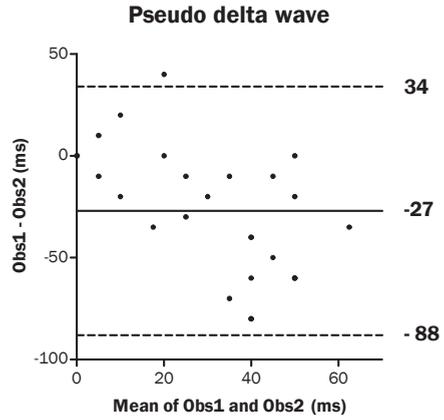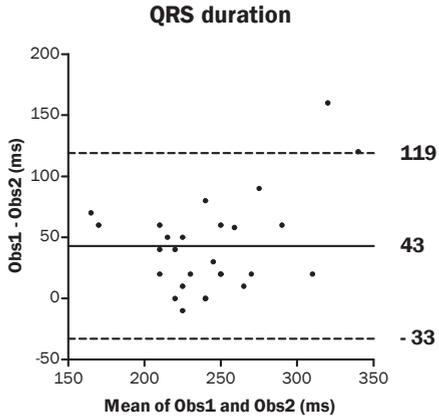
Supplemental Figure 1. Interobserver variability of ECG criteria for clinically documented VTs
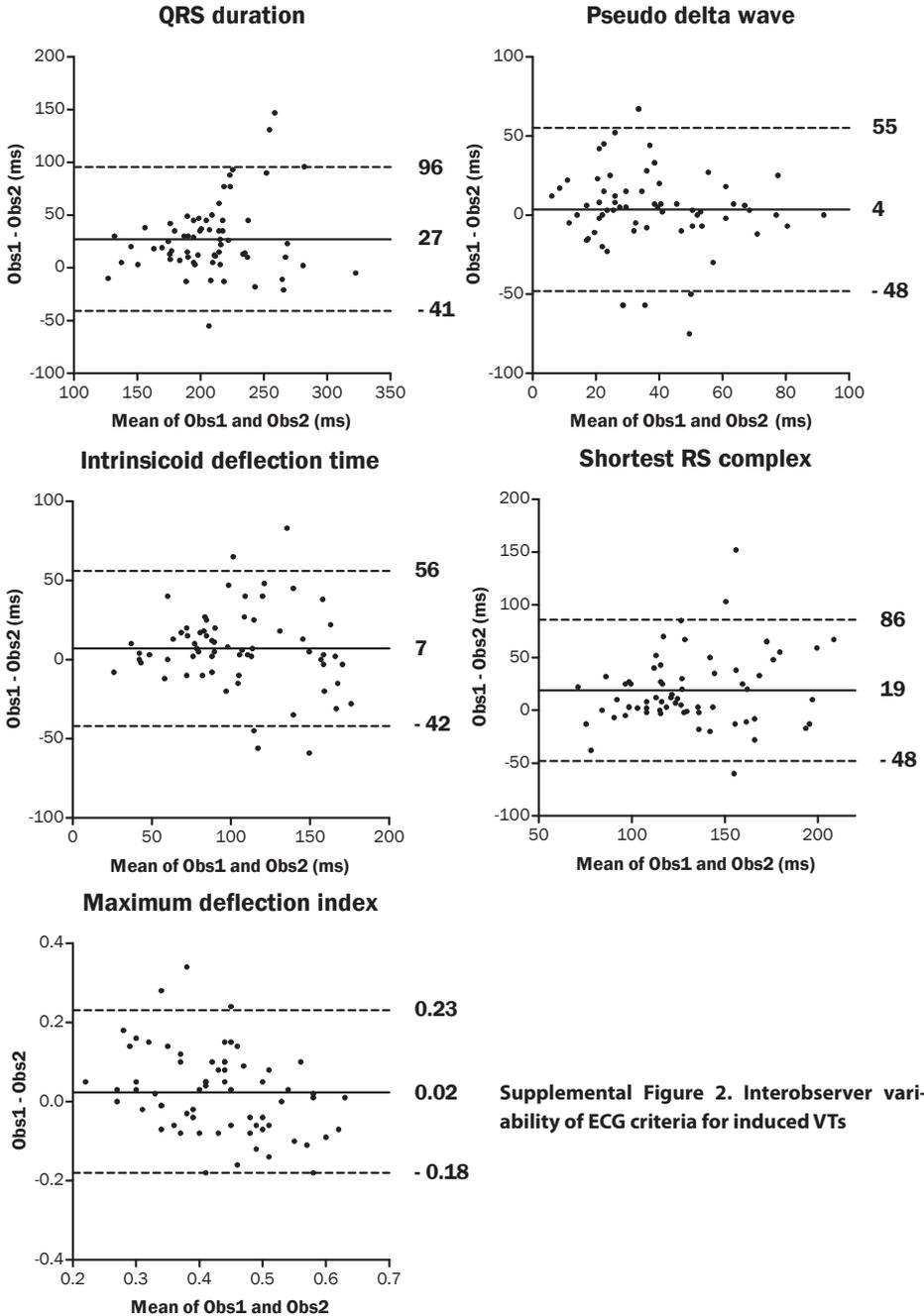
Supplemental Figure 2. Interobserver variability of ECG criteria for induced VTs