Endocardial or epicardial ventricular tachycardia in nonischemic cardiomyopathy?: The role of 12-lead ECG criteria in clinical practice

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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 cardiomyopathy.

OBJECTIVE The purpose of this study was 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 antiarrhythmic drugs on the accuracy of ECG criteria, and (3) assess interobserver variability.

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

RESULTS In 21 patients with clinically documented (25 mm/s) right bundle branch block VT, none of the ECG criteria differentiated between patients with and those without an epicardial SoO. In induced VTs (100 mm/s), 2 of 4 interval criteria differentiated between an endocardial and epicardial SoO for slow VTs (CL >350 ms) and 2 of 4 criteria in patients on amiodarone, but none for fast VTs (CL ≤350 ms) or patients 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 those without an epicardial SoO. Published interval-based ECG criteria do not apply to fast VTs and patients off amiodarone.

KEYWORDS Ventricular tachycardia; Catheter ablation; Nonischemic cardiomyopathy; Electrocardiography; Epicardial

ABBREVIATIONS **Abs-Q-INF** = absence of Q waves in inferior leads; **AAD** = antiarrhythmic drug; **CL** = cycle length; **EAM** = electroanatomic mapping; **IDT** = intrinsicoid deflection time to R wave in V_2; **IQR** = interquartile range; **LV** = left ventricle; **MDI** = maximum deflection index; **NICM** = nonischemic left ventricular cardiomyopathy; **PDW** = pseudodelta wave; **Q-I** = Q wave in lead I; **Q-INF** = Q waves in inferior leads; **RBBB** = right bundle branch block; **RF** = radiofrequency; **SoO** = site of origin; **SRS** = shortest RS complex; **VT** = ventricular tachycardia

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Introduction

In patients with ventricular tachycardia (VT) due to nonischemic cardiomyopathy (NICM), the substrate for VT is frequently—but not always—located intramurally or subepicardially.[1] Therefore, endocardial ablation and/or epicardial ablation may be required to abolish VT. Previous 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 electrophysiologic recording systems with electronic calipers at a sweep speed of 100 mm/s,[2–4] but their accuracy has not been evaluated on 25 mm/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 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

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12-lead ECGs of clinically documented VTs, (2) analyze the effect of VT CL and AAD use on the accuracy of 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 Centre between 2007 and 2012 were included. The study excluded patients with coronary artery disease (>50% stenosis), congenital heart disease, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, left ventricular (LV) 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 with the patient under conscious sedation or general anesthesia (3 drive CL [600, 500, and 400 ms], 1–3 ventricular extrastimuli [coupling interval ≥200 ms], 2 right ventricular sites, and burst pacing, with isoproterenol [2–10 μg/min] when necessary). The positive end-point for stimulation was induction of a sustained monomorphic VT lasting >30 seconds or requiring termination because of hemodynamic compromise.

Electroanatomic mapping and ablation

Epicardial access was obtained through a subxiphoid puncture. Electroanatomic mapping (EAM) was performed using a 3.5-mm irrigated-tip NaviStar ThermoCool catheter (Biosense Webster, Diamond Bar, CA) and the CARTO system (Biosense Webster). 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 computer tomography–derived coronary artery anatomy and epicardial fat thickness with the EAM.[6,7] The LV was mapped retrogradely via the aorta, and the right ventricle 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 to 45 W (maximum temperature 45°C, flow 20–30 mL/min, 60 seconds) at endocardial sites and up to 50 W (flow 20 mL/min) at epicardial sites.

VT morphology and ECG criteria

Clinically documented and induced sustained monomorphic VTs were categorized as right bundle branch (RBBB)-like or left bundle branch block-like 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 left bundle branch block and RBBB VTs, respectively). The ECG features assessed for all RBBB-like morphology VTs, as previously described,[2–4] were as follows:

QRS duration: Interval from earliest ventricular activation to offset of the QRS in the precordial leads

Diastolic interval: VT CL minus QRS duration

Pseudodelta wave (PDW): Interval from earliest ventricular activation to onset of the earliest fast deflection in any precordial lead

Intrinsicoid deflection time in V_2 (IDT): Interval from earliest ventricular activation to peak of the R wave in V_2

Shortest RS complex (SRS): Interval from earliest ventricular activation to the nadir of the first S wave in any precordial lead

Maximum deflection index (MDI): Interval from earliest ventricular activation to the peak of the largest amplitude deflection in each precordial lead (taking the lead with the shortest time) divided by 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 leads 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] 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. For the Q-INF criterion, only superior-axis VTs were analyzed.

Clinical VTs that were documented on 25 mm/s, 10 mm/mV 12-lead ECG were analyzed using pens and manual calipers. Induced VTs were analyzed using electronic calipers on