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ORIGINAL PAPER

Electrocardiographic criteria of epicardial ventricular tachycardia with anterior origin

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Abstract

Background ECG criteria for identifying an epicardial origin of ventricular tachycardia (VT) have mainly been described for VTs with basal-superior and lateral origin.

Objective The aim of this study was to determine ECG criteria for epicardial VTs with anterior origin as a guide for trans-pericardial ablation.

Results Among 22 patients undergoing successful ablation of VTs from the anterior myocardial wall, 14 patients underwent endocardial ablation and 8 patients underwent epicardial ablation. VTs with anterior origin ablated epicardially had widened QS complexes in precordial leads with staircase-shaped notching and slowing of the descent to the nadir of S. In comparison, endocardial VTs with anterior origin usually had narrower QS complexes with a smooth and fast downstroke to the nadir of S. The duration of the negative pseudodelta wave was longer in epicardial VTs (55 ± 12 ms) compared to endocardial VTs (22 ± 12 ms). The interval "time to the nadir of S" in patients with anterior VT origin was longer in epicardial VTs (121 ± 16 ms) than in endocardial VTs (80 ± 22 ms). The QRS duration was also longer in patients with epicardial origin (212 ± 19 ms) than with endocardial VT origin (166 ± 30 ms).

Conclusions Epicardial origin of VTs arising from the anterior myocardial wall produces a slowing, widening and staircase-shaped notching in the initial VT–QS complex. Thus, the morphology of the initial part of the QS complex in precordial leads can be used as a guide for trans-pericardial ablation of VTs with anterior origin.

Keywords Ventricular tachycardia · ECG · Epicardial ablation · Anterior myocardial infarction · Nonischemic cardiomyopathy

Introduction

Percutaneous epicardial radiofrequency (RF) ablation of ventricular tachycardia (VT) has been introduced as an approach for the treatment of recurrent VT refractory to endocardial ablation [1–4]. The ECG criteria for identifying an epicardial origin of VT include slowing and unusual notching in the initial portion of QRS [5–7]. These criteria are region-specific and morphological ECG features and cut-off intervals have mainly been described for epicardial VTs with basal-superior and lateral origin [5–7]. ECG criteria identifying an epicardial origin of VTs arising from the anterior wall of the left ventricle (LV) or right ventricle (RV) have not been described up to date. The aim of this study was to compare 12-lead surface VT-ECGs with successful epicardial vs. endocardial VT ablation in the anterior myocardial wall in order to describe ECG criteria suggesting an epicardial anterior VT origin.

Patients and methods

Study population

We analyzed 12-lead ECGs of 22 patients (15 male, 69 ± 11 years) during monomorphic VT with anterior or anterior apical VT substrate who underwent successful RF ablation. In 14 consecutive patients with prior anterior myocardial infarction, an endocardial VT substrate was detected and the VT could be successfully eliminated by

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endocardial ablation. An endocardial VT substrate was defined by the presence of endocardial reentry circuit isthmus sites with presystolic or diastolic electrograms within an area of very low voltage, entrainment at these sites without QRS fusion and a postpacing interval within 30 milliseconds of the tachycardia cycle length [8]. 8 patients (prior anterior myocardial infarction: $n=5$; arrhythmogenic right ventricular cardiomyopathy (ARVC): $n=1$; hypertrophic obstructive cardiomyopathy (HOCM): $n=1$; nonischemic cardiomyopathy (NICM) after prior myocarditis: $n=1$) underwent successful trans-pericardial epicardial ablation after previous unsuccessful endocardial ablation ($n=5$) or as a primary ablation approach ($n=3$). Three patients undergoing epicardial VT ablation had an endocardial thrombus in the LV apex. In the 8 patients undergoing successful epicardial ablation, an epicardial VT substrate was identified by the presence of epicardial presystolic or diastolic potentials during the VT (in 5 patients) or by the presence of isolated diastolic potentials within an area of low or very low voltage during sinus rhythm (in 3 patients). In the case of epicardial mapping during sinus rhythm, epicardial pacemapping at high output power (12 V, 2 ms duration) was used in and around the abnormal substrate to define the approximate exit site of the VT circuit and to identify sites with a long stimulus to QRS interval with a good QRS match suggesting an epicardial critical isthmus of the VT reentry circuit.

Cardiac magnetic resonance imaging (MRI) using a 3 T magnetic imaging scanner was performed as recently described [9]. Transmural scar and epicardial-only DE MRI substrate was identified in each one patient undergoing successful epicardial VT ablation.

Electrophysiologic study and mapping

After giving informed consent, all patients underwent electrophysiologic study. All antiarrhythmic drugs including beta blockers except amiodarone were discontinued before the procedure for at least five half lives. ICD therapies were de-activated during the procedures in all patients. Endocardial electrode catheters for programmed ventricular stimulation, mapping and ablation were placed into the RV and LV through the right and left femoral veins and the right femoral artery. Endocardial mapping was performed with a three-dimensional mapping system (CARTO, Biosense Webster Inc., Baldwin Park, CA, USA) according to standard criteria [10]. Epicardial transcoronary microelectrode catheter mapping was performed as previously described [11]. Epicardial mapping through a trans-pericardial access was performed using the technique of Sosa [1]. In most cases, a 22 mm 8,5 F sheath was used to introduce the mapping catheter in the pericardial space. A posterior pericardial approach was performed in all patients with epicardial anterior VT substrate. Epicardial mapping and ablation was performed under general anesthesia in all patients.

An endocardial voltage map during sinus rhythm was constructed in all patients without LV thrombus. An epicardial voltage map during sinus rhythm was constructed in all 8 patients undergoing trans-pericardial epicardial mapping. The stimulation protocol consisted of a programmed ventricular stimulation from the RV apex and the RV outflow tract with up to three extrastimuli at four basic drive cycle lengths (five captured beats at 600, 500, 400 and 330 ms cycle length). If the VT was not inducible by endocardial stimulation, stimulation was repeated under orciprenaline infusion. Pace mapping and entrainment mapping was attempted in all patients using standard criteria [10]. During mapping and ablation, 5000 IU heparin was given as a bolus, followed by 1000 IU as an infusion. In the case of epicardial mapping, heparin was given after the pericardial puncture.

Catheter ablation

A 500-kHz RF ablation unit (Stockert Cordis) was used for ablation. The ablation catheter was a 3.5 or 4 mm tipped irrigated ablation catheter (Cordis Webster or Biosense Webster). The current was initially applied at a power output of 20 W and was increased by 10 W every 5–10 s up to a maximum of 50 W for endocardial ablation and 40 W from the pericardial space. RF energy application from the pericardial space was performed at sites where the distance to the adjacent coronary artery was > 5 mm detected by simultaneous coronary angiography [9]. Endocardial ablation was performed in most patients during the clinical VT according to standard entrainment and pace mapping criteria [10]. The endpoint of endocardial ablation was termination and non-inducibility of the clinical VT and of VTs with a similar or longer cycle length. Due to the poor inducibility of the clinical VT under general anesthesia in some patients, epicardial RF energy delivery was performed during sinus rhythm in 3 patients. Detailed epicardial voltage mapping in the region of the arrhythmia substrate was performed, and all sites demonstrating late potentials or isolated diastolic potentials were identified. The combination of late or isolated diastolic potentials during sinus rhythm, a good pacemap QRS match and a long stimulus-QRS interval during pace mapping suggested the likelihood of proximity to VT origin/circuit and was used to define the target of substrate-based ablation. In addition to non-inducibility of the clinical VT, the

endpoint of epicardial ablation was complete elimination of all potentials with late or isolated diastolic electrograms in a region of epicardial low (< 1.5 mV) or very low voltage (< 0.5 mV) that was identified as an appropriate target for ablation by pace mapping.

Analysis of the ECG

Three distinct intervals of ventricular activation during the VT were defined. The intervals were measured using digitized tracings with calipers allowing 1 ms resolution at a screen velocity of 100 mm/s and with the signal amplified at 10 mm/mV.

Negative pseudodelta wave

The negative pseudodelta wave was measured from the earliest negative ventricular activation to the earliest fast deflection in any precordial lead (except V1).

Time to the nadir of S

The time to the nadir of S was defined as the interval measured from the earliest ventricular activation to the nadir of S in any precordial lead (except V1).

QRS complex duration

The QRS complex duration was defined as the interval measured from the earliest ventricular activation to the offset of QRS in the precordial leads.

Statistical analysis

Continuous data are expressed as mean ± standard deviation. Means were compared by use of Student´s t-test for paired data. A value of < 0.05 was considered significant.

Results

Identification of epicardial VT origin

Eight patients (6 male, 65 ± 14 years) had a successful epicardial VT ablation in the anterior or anterior apical wall of the LV ($n = 7$) and RV ($n = 1$). Among them, 5 patients had a prior anterior myocardial infarction (Fig. 1), and each one had an ARVC (Fig. 2), HOCM (Fig. 3) and NICM after prior myocarditis. Five patients had a prior unsuccessful endocardial ablation in our ($n = 3$) or another institution ($n = 2$). Diastolic or presystolic potentials during the VT ($n = 5$) or isolated diastolic potentials during sinus rhythm ($n = 3$) were found epicardially in all patients. The results of entrainment and pace mapping and successful ablation at epicardial sites indicated that these VTs had critical reentry circuit isthmus sites at the epicardial surface. 14 consecutive patients (11 male, 71 ± 9 years) had an endocardial VT origin as determined by activation and entrainment mapping criteria and underwent successful VT ablation at endocardial sites.

Intervals in VT

Epicardially ablated VTs had QS complexes in precordial leads with slower and often staircase-shaped notching in the descent to the nadir of the S wave (Fig. 4). Endocardially ablated VTs often had a smooth and fast descent to the nadir of S (Fig. 4). The initial slower part preceding the subsequent fast downstroke to the nadir of the S wave termed negative pseudodelta wave was markedly more pronounced in patients with epicardial VT origin than with endocardial VT origin (Fig. 5). The duration of the negative pseudodelta wave was 55 ± 12 ms in epicardially ablated VTs compared to 22 ± 12 ms in endocardially ablated VTs ($p < 0.05$). The interval "time to the nadir of S" in VT patients with anterior VT substrate was longer in VTs with anterior epicardial origin (121 ± 16 ms) than with anterior endocardial origin (80 ± 22 ms) ($p < 0.05$). The QRS duration was also longer in patients with epicardial origin (212 ± 19 ms) than with anterior endocardial origin (166 ± 30 ms) ($p < 0.05$).

Epicardial pacing

Epicardial pacing from the suspected region of VT origin was performed in all 8 patients undergoing trans-pericardial mapping (Fig. 6). Epicardial capture was obtained in 6 patients and produced a widening of the QRS complex as a result of slowing and notching in the initial part of QS similar to the QS morphology during the VT. In comparison, QS complexes were narrower and notching was less pronounced in the same patients during endocardial pacing from a corresponding endocardial location ($n = 5$) and during sinus rhythm (n = 3) (Fig. 6).

Follow-up after ablation

VT ablation was successfully performed in all patients included in this study. During the follow-up, 5 of 8 patients with primarily successful epicardial ablation and 8 of 14 patients with primarily successful endocardial ablation remained on amiodarone (200 mg daily). The 52-year-old patient with ARVC who underwent successful epicardial ablation (Fig. 2) had a prolonged stay of 6 days at the intensive care unit due to temporary right ventricular failure.

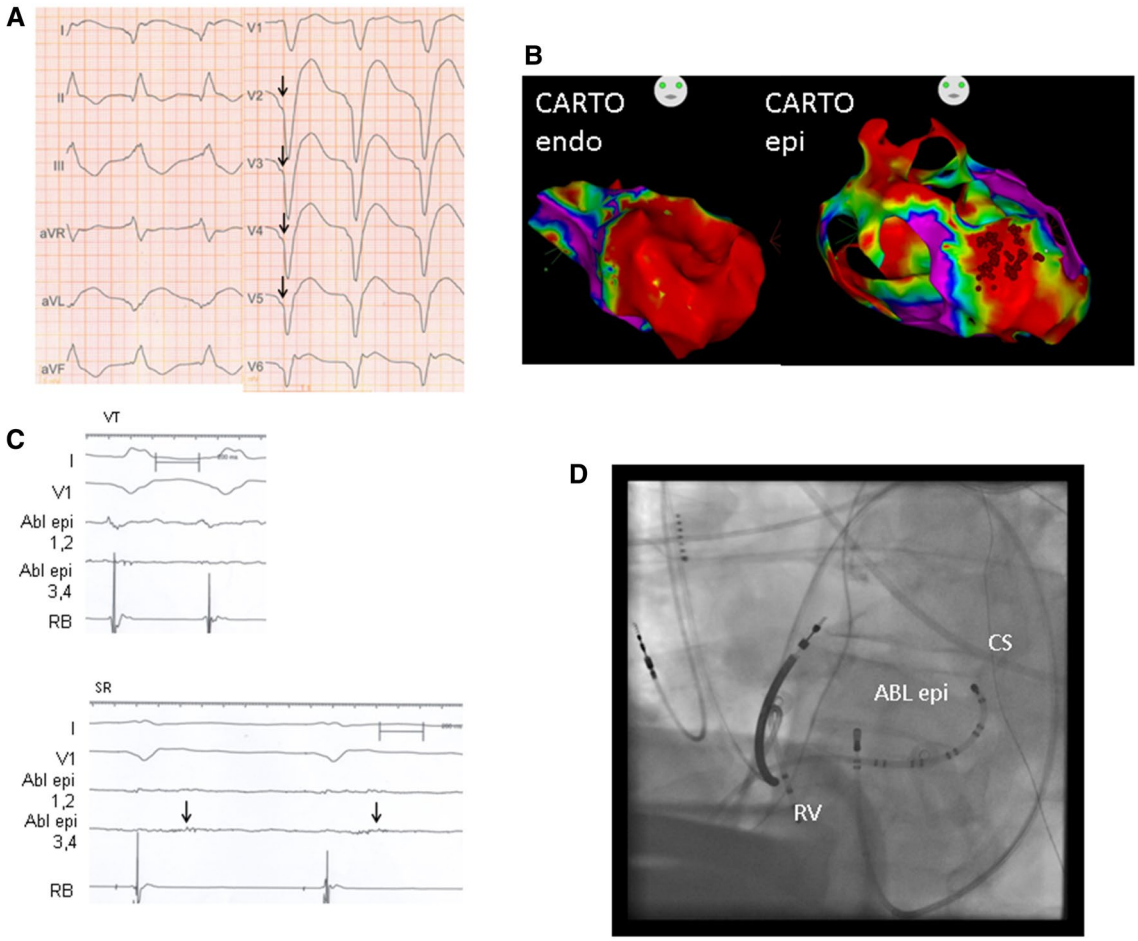

Fig. 1 VT refractory to endocardial ablation after prior myocardial ablation in a 70-year-old patient. **a** 12-lead ECG of the clinical VT showing a notched descent to the nadir of S in the precordial leads (arrows). **b** Endocardial and epicardial CARTO voltage map of LV showing a large very low voltage zone (< 0.5 mV) (red) in the anterior and apical LV. The red dots in the epicardial CARTO voltage map indicate sites where the VT could be successfully ablated after initial unsuccessful endocardial ablation. **c** Epicardial electrograms during the VT and sinus rhythm (SR) from the distal (Abl epi 1,2) and proximal (Abl epi 3,4) electrode pair of the ablation catheter at the successful epicardial ablation site at the anterior LV. RB, right bundle. **d** Fluoroscopic position of the epicardial ablation catheter (ABL epi) at the successful epicardial ablation site in a posterior anterior projection; *CS* coronary sinus; *RV* right ventricular apex

There was no in-hospital death but a 81-year-old patient died 2 months after epicardial ablation and subsequent endocardial ablation due to deteriorating heart failure. During the follow-up of 42 ± 38 months after ablation, 3 of the 8 patients undergoing epicardial ablation had at least one recurrence of sustained VT and two of them underwent successful endocardial re-ablation of monomorphic VT with other QRS morphology. Among the 14 patients with primarily successful endocardial VT ablation, 5 had at least one VT recurrence and 3 underwent endocardial re-ablation.

Discussion

Electrocardiographic criteria proposed for the identification of epicardial VT

A number of electrocardiographic parameters were defined for the diagnosis of epicardial VT. They are mainly based on the concept that when ventricular activation starts at the epicardial level, the initial part of the wavefront progresses slowly through the myocardium until reaching the Purkinje system at the subendocardium [12]. This initial slow transmural activation is reflected as slow onset on the surface electrocardiogram [5]. The duration of this pseudodelta wave measured from the earliest ventricular activation to the earliest fast deflection in any precordial lead coincided with the duration of the

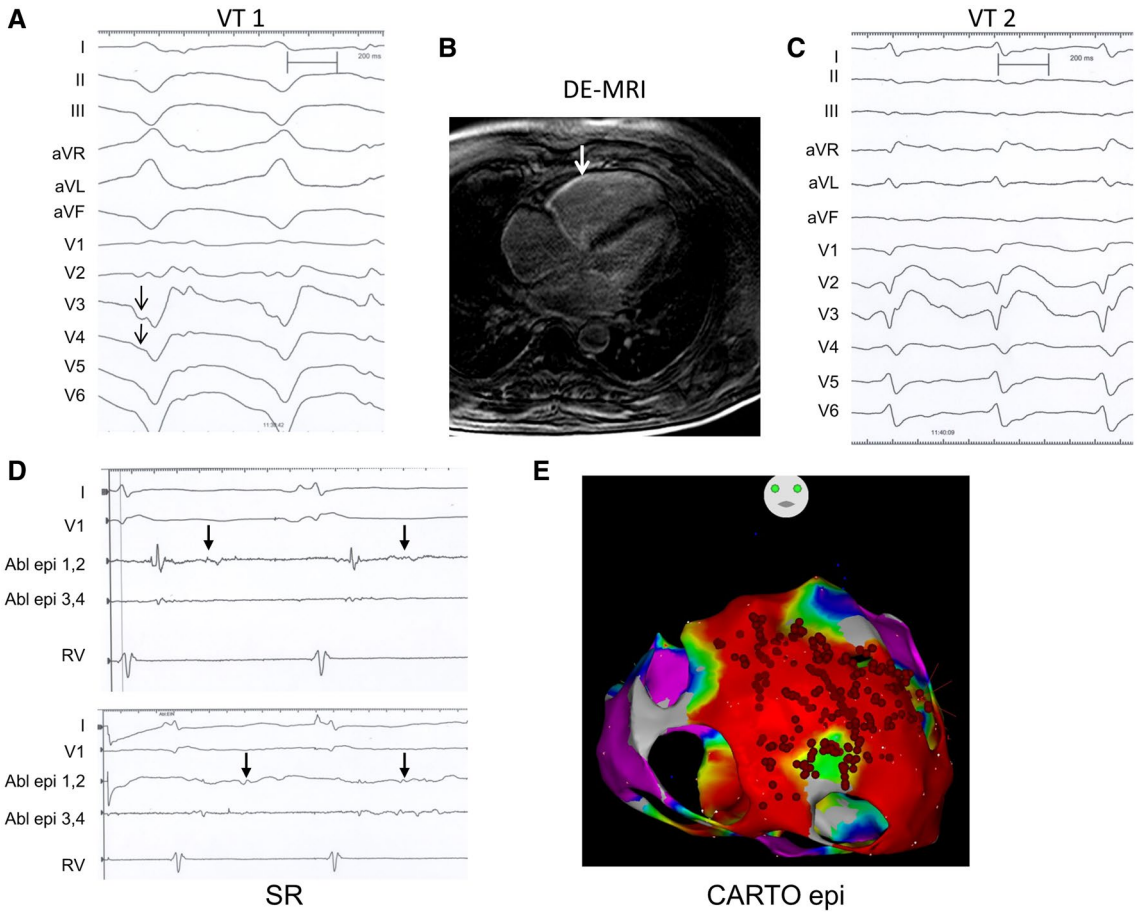

Fig. 2 VT refractory to endocardial ablation in a 53 year-old patient with arrhythmogenic right ventricular cardiomyopathy. **a** 12-lead ECG of the predominant clinical VT1 showing a staircase-shaped notched descent to the nadir of S in the precordial leads V3 and V4 (arrows). **b** Delayed enhanced magnetic resonance imaging (DE-MRI) in a four-chamber view showing a transmural scar in the anterior wall of the RV. **c** 12-Lead ECG of VT2 showing a fast and smooth descent to the nadir of S and a narrower QS in the precordial leads V1–V4. VT2 had an endocardial origin and could be successfully ablated from the endocardial RV. **d** Typical epicardial electrograms during sinus rhythm from the distal (Abl epi 1,2) and proximal (Abl epi 3,4) electrode pair of the ablation catheter at the epicardial wall of the RV. Epicardial isolated diastolic electrograms are indicated by arrows. **e** Epicardial CARTO voltage map in an anterior posterior projection. Red areas indicate areas of very low voltage (< 0.5 mV). The red dots indicate sites where VT1 could be successfully ablated by targeting all epicardial sites with isolated diastolic electrograms within very low voltage regions at the epicardial anterior wall

transmural epi- to endocardial activation time [5]. The intrinsicoid deflection time defined as the interval measured from the earliest ventricular activation to the peak of the R wave in V2 and the shortest RS complex interval in precordial leads were also reported to predict an epicardial VT origin with high sensitivity and specificity [5, 12]. Valles et al. showed that the presence of q waves in lead I and the absence of q waves in inferior leads helped to identify basal-superior and lateral epicardial VTs in nonischemic cardiomyopathy [6].

In epicardial VTs with anterior origin, the initial slower part in the descent to S in the QS complex in precordial leads apparently represents the epi-to-endocardial activation arising from the epicardial site of origin in the anterior wall. Similarly to the results obtained from epicardial VTs with superior-basal and lateral origin, the duration of the negative pseudodelta wave, the interval "time to the nadir of S" and the QRS duration were significantly longer in anterior VTs with epicardial than with endocardial origin. The suggestion that this morphology of the initial descending part of QS in the precordial leads was due to the epicardial VT origin were substantiated by epicardial pacing that produced a widening and notching of the QS complexes similar than during the spontaneous VT.

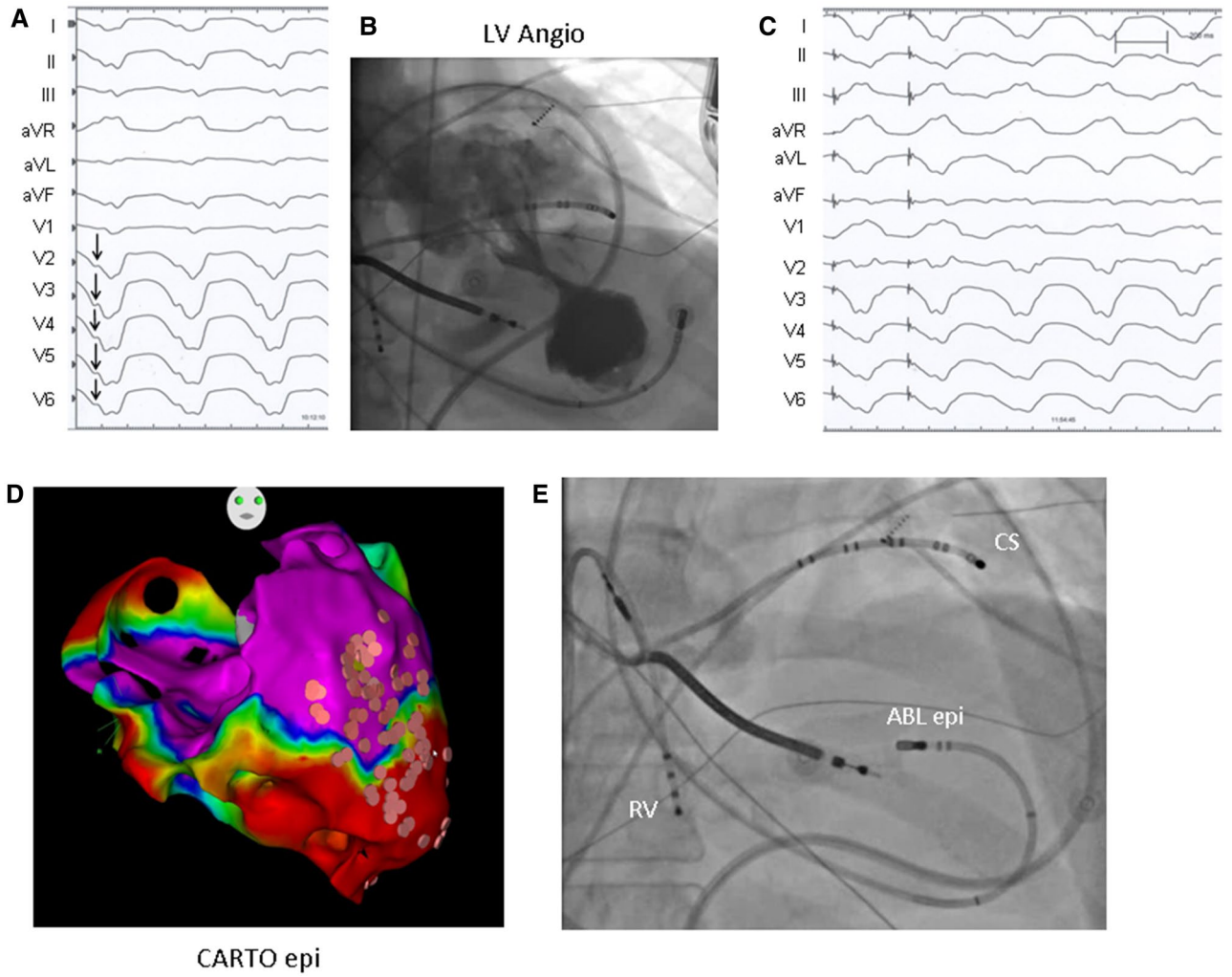

Fig. 3 VT refractory to endocardial ablation in a 37-year-old female patient with hypertrophic obstructive cardiomyopathy and secondary aneurysm formation. **a** 12-lead ECG showing a marked staircase-shaped notching in the descending part of QS in the precordial leads (arrows). **b** LV angiogram in posterior anterior projection showing a hypertrophic obstructive cardiomyopathy with LV aneurysm formation and an apical LV thrombus. **c** Concealed entrainment without fusion from the epicardial anterior surface at the neck of the LV aneurysm. The site of pacing from the ablation catheter in shown in **e. d** Epicardial CARTO voltage map in a modified right anterior oblique projection. Red areas in the epicardial part of the LV aneurysm represent areas of very low voltage (< 0.5 mV). Purple areas in the epicardial map represent areas of normal voltage (> 1.5 mV) in the basal LV. The line of red and purple dots indicates epicardial ablation sites in the anterior wall resulting in successful ablation of the VT. **e** Fluoroscopic position of the epicardial ablation catheter (ABL epi) in a posterior anterior projection. *CS* coronary sinus, *RV* right ventricular apex

Ischemic versus nonischemic cardiomyopathy

The success rate of endocardial ablation is lower in patients with NICM than in patients after myocardial infarction [10]. Cardiac MRI with delayed enhancement has demonstrated that scars are often located intramurally and epicardially in NICM [9]. Earlier studies with endocardial and epicardial mapping during surgical treatment of post-myocardial infarction VTs showed that 20% of VTs have earliest epicardial activation rather than endocardial activation [13]. In other series, around 15% of circuits in ischemic cardiomyopathy were complete subepicardial circuits [14]. The presence of epicardial circuits has been recognized as a cause of recurrence after endocardial ablation for VT after myocardial infarction [15]. On the other hand, the presence of an epicardial origin

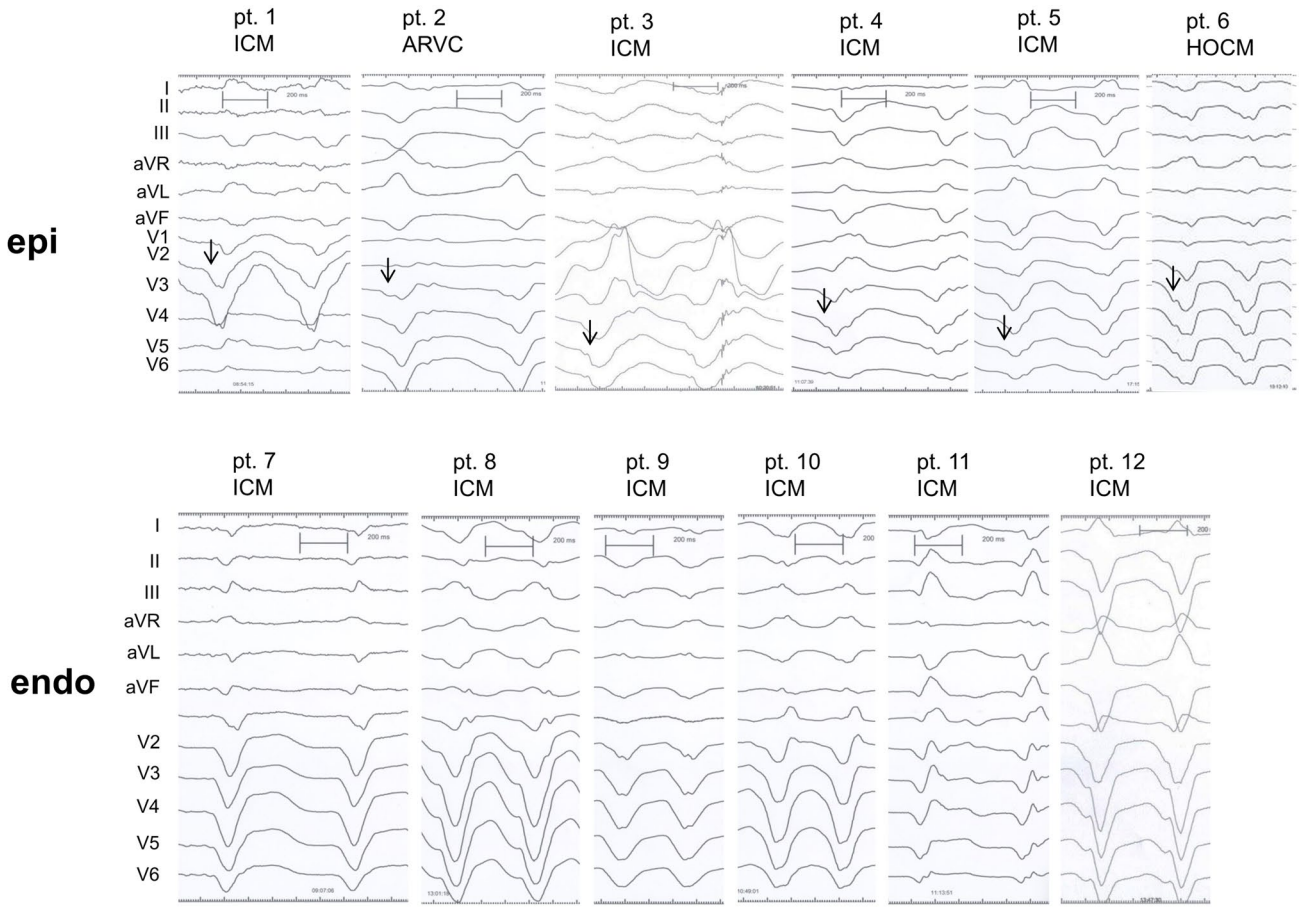

Fig. 4 12-lead ECGs of each six patients with VTs with anterior origin undergoing successful epicardial (epi) and successful endocardial (endo) ablation. Epicardial VTs with anterior origin usually showed a slowing and notching in the descent to the nadir of S in the precordial leads. Endocardial VTs with anterior origin usually showed narrower QS complexes in the precordial leads with a smooth and fast descent to the nadir of S

does not exclude successful VT ablation from the endocardium due to wall thinning in ischemic cardiomyopathy [16]. In our patients, 5 of the 8 patients with successful epicardial VT ablation had prior large anterior myocardial infarction, and three patients had different forms of NICM. The results suggest that successful epicardial ablation after previous unsuccessful endocardial ablation in ischemic cardiomyopathy identifies VT reentry circuits in patients with large transmural infarcts where critical VT isthmuses apparently involved subepicardial layers of the infarcted myocardial wall. We hypothesize that the three patients who did not undergo endocardial ablation due to LV thrombi had transmural scar involving subepicardial isthmus sites of the VT reentry circuits accessible to epicardial ablation.

Limitations

Sometimes, it is difficult to measure the duration of the pseudodelta wave; in these cases, measurement of the interval "time to the nadir of S" may be easier and more reliable. Due to the relatively low number of patients with epicardial ablated VTs with anterior origin, it was not possible to determine reliable cut-off values for identifying an epicardial origin of ventricular activation in the anterior wall. As endocardial ablation can be effective in ablating epicardial local abnormal ventricular activities [16], some of the patients with successful endocardial ablation may have transmural substrates with deep endocardial or even subepicardial substrates. It must also be taken into account that the electrocardiogram pattern only indicates the exit of the VT circuit and that the VT isthmuses can be complex involving subendocardial and

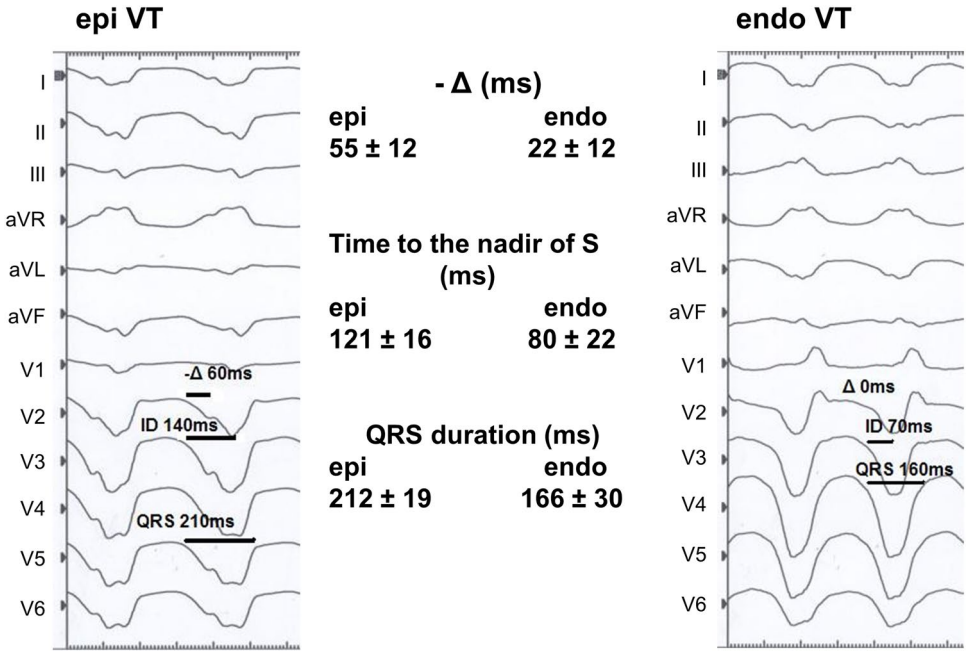

Fig. 5 12-lead ECG during epicardial VT (epi VT) and endocardial VT (endo VT) with anterior origin. Example of intervals measured in VTs from each group. Negative pseudodelta wave (-Δ) was measured from the earliest negative ventricular activation to the earliest fast deflection in any precordial lead (except V1). Time to the nadir of S was defined as the interval measured from the earliest ventricular activation to the nadir of S in any precordial lead (except V1). QRS complex duration was defined as the interval measured from the earliest ventricular activation to the offset of QRS in the precordial leads. Means ± standard deviation were given for epicardial VTs (epi VT) ($n = 8$) compared to endocardial VTs (endo VT) ($n = 14$)

subepicardial layers. Finally, a possible explanation for the need for an epicardial approach after primarily unsuccessful endocardial ablation is that the VT origin was not identified correctly during the initial procedure. The fact that patients with different types of structural heart disease were enrolled might limit the reliability of the analyzed ECG patterns because the ECG characteristics identifying an epicardial origin might not be the same in different types of structural heart disease.

Clinical considerations

It is generally established that a failed endocardial ablation procedure is sufficient to attempt an epicardial ablation in an experienced center [12]. Most infarct-related VTs can be successfully ablated from the endocardium. Unsuccessful endocardial VT ablation in patients after myocardial infarction led us to suspect transmural infarction with epicardial circuits. Although it is reasonable to be more conservative in the epicardial mapping and ablation, especially in patients with ischemic cardiomyopathy, a typical QS morphology in precordial leads during the VT describing an initial epi- to endocardial activation can be useful as a guide for earlier epicardial ablation.

Conclusions

The epicardial origin of ventricular activation in the anterior ventricular myocardium wall produces a slowing, widening and notching in the initial part of the VT–QS complex visible as a negative pseudodelta wave. This QS morphology in precordial leads during the VT can be a guide for earlier epicardial ablation in patients after anterior myocardial infarction and in different forms of nonischemic cardiomyopathy.

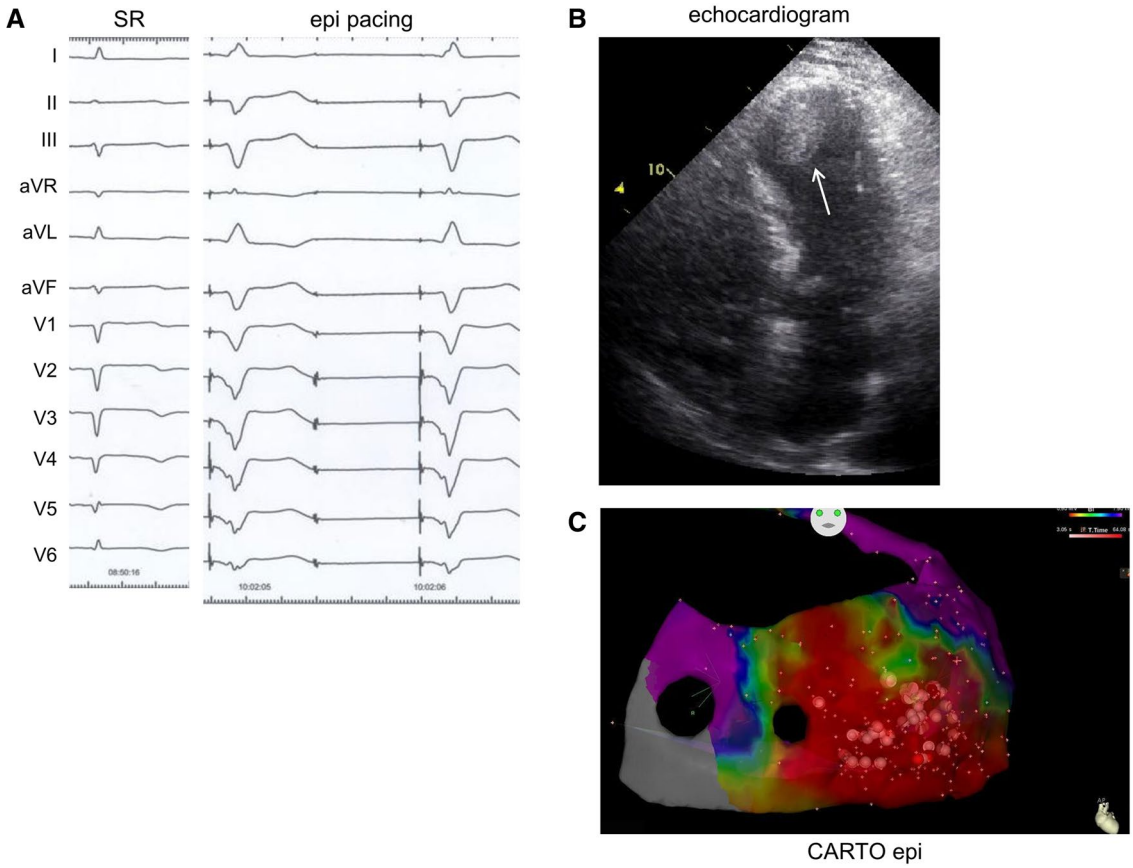

Fig. 6 Epicardial pacing and successful epicardial ablation of a VT originating from the anterior apical LV in a 70-year-old patient after prior myocardial infarction with nodular LV thrombus. **a** 12-Lead ECG during sinus rhythm and epicardial pacing (2:1 capture) from the low-voltage zone in the anterior apical LV (position of the epicardial pacing site is seen in **c**). During epicardial pacing a staircase-shaped notching in the descent to the nadir of S is seen. **b** Echocardiogram showing a large nodular thrombus in the LV apex (arrow) that did not allow endocardial mapping, pacing and ablation. **c** Epicardial electroanatomical map (CARTO) in an anterior posterior projection. The sites of ablation (red and purple dots) in a zone of epicardial very low voltage (< 0.05 mV) are indicated

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