Recording of low-amplitude diastolic electrograms through the coronary veins: a guide for epicardial ventricular tachycardia ablation

Christopher Reithmann1*, Michael Fiek1, Anton Hahnefeld2, Michael Ulbrich2, and Gerhard Steinbeck2

1Medizinische Klinik I, Klinikum München Pasing, Steinerweg 5, 81241 München, Germany; and 2Medizinische Klinik I, Klinikum Grosshadern, Universität München, Marchioninistr. 15, 81377 München, Germany

Received 17 July 2011; accepted after revision 16 November 2011; online publish-ahead-of-print 23 December 2011

Aims
The purpose of the study was to evaluate the role of coronary venous mapping to identify epicardial ventricular tachycardia (VT) in patients with structural heart disease.

Methods and results
Epicardial mapping of the electrophysiological substrate through the coronary vein branches using a 2.2F, 16-pole microelectrode catheter was performed in 33 consecutive patients undergoing VT ablation. Twenty-six patients had a history of myocardial infarction and seven had a non-ischaemic cardiomyopathy. Endocardial ablation was successful in 19 of the 33 patients (58%). Low-amplitude fractionated diastolic electrograms with an electrogram–QRS interval amounting to 30–70% of the VT cycle length were recorded during the VT in the coronary vein branches in eight patients (24%). Endocardial ablation failed in seven of the eight patients with diastolic electrograms in the coronary veins, suggesting an epicardial involvement of the VT re-entry circuit. Among the patients with a suspected epicardial VT origin, four patients underwent epicardial ablation using a pericardial access after unsuccessful endocardial ablation which eliminated mappable VTs in all.

Conclusion
Recording of low-amplitude fractionated diastolic electrograms through the coronary veins facilitates the identification of VTs with an epicardial origin requiring mapping and ablation through a pericardial access.

Keywords
Catheter ablation • Coronary venous mapping • Epicardial ablation • Ventricular tachycardia • Pericardial puncture

Introduction
In the majority of patients with ventricular tachycardia (VT), at least a portion of the re-entrant circuit involves the endocardium.1 However, intramyocardial and epicardial regions of slow conduction can also be critical components of the re-entrant circuits.2 Recent studies have suggested that some VTs originating from the epicardium are not suitable for endocardial ablation. Epicardial ablation is required for 10–30% of post-infarct VTs and for more than 30% of VTs due to non-ischaemic cardiomyopathy.3 Characteristics from the 12-lead surface electrocardiogram (ECG) such as the presence of a pseudodelta wave4 and visualization of epicardial scar tissue using cardiac magnetic resonance imaging5 can help to identify VTs with an epicardial origin. Recently, nonsurgical epicardial mapping through a pericardial access has been introduced as a new technique that opened the possibility for mapping and ablation from the pericardial space.6 Activation mapping through the coronary veins and their tributaries using multipolar microelectrode catheters has been introduced as a tool for epicardial mapping of the electrophysiological substrate.7 The aim of the present study was to systematically evaluate the use of microelectrode catheter coronary venous mapping as a guide for potential epicardial ablation in a consecutive series of patients with structural heart disease referred for VT ablation.

* Corresponding author. Tel: +49 89 8892 2307; fax: +49 89 8892 2274. Email: christopher.reithmann@kliniken-pasing-perlach.de
Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2011. For permissions please email: journals.permissions@oup.com.
Methods

Patients
Thirty-three consecutive patients with sustained clinical VT and structural heart disease were admitted for catheter ablation due to frequent episodes of VT (>3 sustained VT with an interval of >5 min within 24 h) or incessant VT. Written informed consent was obtained from all patients. There were 30 men and three women with a mean age of 66 ± 12 years. Thirty-one patients had an implantable cardioverter defibrillator (ICD). Twenty-six patients had a history of myocardial infarction. Seven patients had a non-isaemic cardiomyopathy (including dilated cardiomyopathy in six patients and cardiac sarcoidosis in one patient). The mean left ventricular (LV) ejection fraction was 24%. All patients had a 12-lead ECG of the clinical VT, electrophysiological study, coronary and left ventricular angiography, and coronary venous angiography.

Microelectrode catheter placement
Coronary venous angiography was performed through the right femoral vein using an Amplatz left coronary (AL II, 7F) catheter. Five to ten millilitres of contrast were delivered in the proximal and distal segments of the coronary sinus. For angiography of the posterior interventricular vein (PIV), we attempted to cannulate this vein selectively. Angiography was recorded in two projections, mostly right anterior oblique (30°) and left anterior oblique (45°). After coronary venous anatomy was defined, a 2.2F 16-polar (2.6-2mm interelectrode spacing) microelectrode catheter (Pathfinder; Cardima, Fremont, CA, USA) was manoeuvred into one of the tributaries of the coronary sinus (anterior interventricular vein (AIV), PIV, anterolateral vein, or posterolateral vein). Left ventricular angiography and the 12-lead ECG during the VT were the determinants for placement of the micro-electrode catheter into the target coronary venous branch aiming to record electrograms from the epicardial surface of an LV aneurysm or region with marked wall motion abnormalities where a slow conduction zone critical for the VT re-entry circuit was suspected.

Diastolic and pre-systolic electrograms
Endo- and epicardial electrograms were defined as diastolic if the electrogram to QRS interval (EG–QRS) per VT cycle length (VTCL) was 0.30–0.70. Electrograms were classified as pre-systolic if the EG–QRS/VTCL was <0.3.

Ventricular tachycardia mapping
Endocardial electrode catheters for programmed ventricular stimulation, mapping, and ablation were placed in the right and left ventricle through the right and left femoral veins and the right femoral artery. Left ventricular mapping was performed using a retrograde trans-aortic approach in most patients and a transseptal approach was used in four patients. Endocardial activation mapping was performed using a 3D mapping system (CARTO; Biosense-Webster Inc., Baldwin Park, CA, USA or Ensite-NavX, St Jude Medical, St Paul, NM, USA) in 18 patients. Simultaneously, microelectrode epicardial activation mapping was performed through the target coronary vein. Bipolar epicardial and endocardial electrograms filtered at 30 and 500 Hz during the VT were recorded on an Electrophysiology LAB system (Prucka Engineering, Houston, TX, USA).

Induction of the VT was attempted by programmed stimulation with up to three extrastimuli at four basic drive cycle lengths (five captured beats at 600, 500, 400, and 330 ms cycle lengths) and at two different RV sites (apex and outflow tract). Mappable VT was defined as sustained, haemodynamically tolerated, and reproducibly inducible. Unmappable VT changed to another VT during attempted mapping or could not be reproducibly initiated or it required termination within 30 s because of haemodynamic compromise. Incessant VT was defined as continued VT despite attempted electric or pharmacologic cardioversion so that VT was present >50% of the time for a period >12 h.

At the endocardial or epicardial sites where earliest pre-systolic or diastolic electrograms were recorded during the VT, pacing (starting with stimuli twice the diastolic threshold and 2 ms in duration) was performed at cycle lengths of 20 ms shorter than the VT cycle length and decreasing by 20 ms until the VT was entrained. Endocardial and epicardial sites, where concealed entrainment of the VT with a post-pacing interval ± 30 ms of the VT cycle length could be demonstrated, were visualized in the 3D electroanatomical maps as isthmus sites of the re-entry circuit. During mapping and ablation, 5000–7500 IU intravenous heparine was given as a bolus, followed by 1000 IU/h as an infusion.

Epiperial mapping through a pericardial access
Pericardial puncture was performed using the technique of Sosa et al. Under general anaesthesia, an introducer needle was advanced from a subternal position into the pericardial space under fluoroscopic guidance. Contrast medium was injected while advancing the needle to demonstrate the position of the needle tip and a guidewire was introduced into the pericardial space. After a 7F sheath was advanced, an irrigated 4 mm tip CARTO mapping and ablation catheter was introduced into the pericardial space. Activation mapping, CARTO voltage mapping, and entrainment mapping of the VT was attempted from the pericardial space.

Catheter ablation
All induced mappable VTs were targeted by ablation. A 4 mm tipped irrigated ablation catheter (Cordis Webster or Biosense Webster) was used as the ablation catheter. A 500 kHz radiofrequency (RF) ablation unit (Stockert Cordis) was used for ablation. The current was initially applied at a power output of 10 W and was increased by 5–10 W every 5–10 s up to a maximum power of 60 W for endocardial ablation. 20 W for ablation from the aortic root, and 30 W from the pericardial space. Delivery of impulses was guided by impedance control and was immediately stopped if an impedance rise occurred or if the catheter was displaced. The following recordings guided the individual ablation strategy: (i) the point-to-point endocardial activation mapping, (ii) the coronary venous epicardial multipolar electrode activation mapping, (iii) the endocardial and epicardial 3D voltage mapping, and (iv) entrainment mapping manoeuvres. In most endocardial ablation procedures, ablation consisted in creating a linear lesion between an area of unexitable scar and an anatomic barrier such as the mitral annulus or between a dense scar and normal endocardium as proposed by Marchlinski et al.6 Catheter ablation from the coronary cusp/aortic root was only performed if the distance from the electrode tip to the ostium of each left and right coronary artery was >8 mm as determined by simultaneous coronary angiography. Radiofrequency energy application from the pericardial space was performed at sites where the distance to the adjacent coronary artery was >5 mm as determined by simultaneous coronary angiography.

The endpoint of the ablation procedure was the absence of inducible, clinically relevant sustained monomorphic VT with the same stimulation protocol used for VT initiation at the beginning of the ablation procedure. Clinically relevant VTs were defined as sustained monomorphic VTs independent from their morphology with equal,
similar, or longer cycle lengths compared with the documented clinical VT.

Follow-up
Implantable cardioverter defibrillator interrogation was performed every 3 months. Patients without ICD were seen every 3 months in our outpatient clinic.

Results
Microelectrode catheter placement in target coronary vein branches
The predominant clinical VT was induced in 28 patients and incessant VT was present in 5 patients. A total of 54 VTs was induced by programmed ventricular stimulation in the 33 patients. In eight patients more than three different VT morphologies were induced. Mean VT cycle length was 436 ± 88 ms. The multipolar microelectrode catheter could be manoeuvred into the target coronary vein branch (AIV, n = 16; PIV, n = 10; anastomoses between AIV and PIV, n = 2; anterolateral or posterolateral vein, n = 2) in 30 patients. In three patients, it was not possible to place the microelectrode catheter into the target coronary venous branch due to technical reasons or to occlusion of the target vein. Venous mapping usually extended the procedure time by less than 15 min.

Coronary venous mapping during the ventricular tachycardia
Simultaneous endocardial and epicardial activation mapping was attempted during the VT in all patients. In eight patients, low-amplitude fractionated diastolic electrograms were recorded in the coronary vein branches during the VT (Figure 1). The diastolic potentials were found in the AIV (n = 3), PIV (n = 4), and in an anterolateral vein (n = 1). Recording of low-amplitude fractionated diastolic potentials in the coronary veins suggested that at least one part of the VT re-entry circuit may involve the epicardium. Entrainment mapping by pacing from the microelectrode catheter positioned in the target coronary vein branch was attempted during the VT in all cases. Concealed entrainment of the VT could not be demonstrated in any case due to lack of capture or to different QRS morphology. Endocardial electrograms recorded facing epicardial areas with low-amplitude fractionated potentials were pre-systolic electrograms in four patients (Figure 1B, Map d endo), diastolic electrograms in three patients, and electrograms not preceding the onset of the QRS complex in one patient.
Endocardial mapping and ablation

In all patients, VT ablation was first attempted endocardially (Figure 2). Endocardial sites characterized as isthmus sites of the VT re-entry circuit by activation mapping and entrainment mapping were found in 20 of 33 patients. The endocardial ablation strategy included encircling of the scar in 4 patients, a linear lesion between dense scar and the mitral annulus in 8 patients, a linear lesion between dense scar and normal endocardium in 7 patients, a linear lesion between two scars in 3 patients, and an electrophysiologically guided ablation targeting critical re-entry circuit isthmuses with a small number of RF applications in 10 patients. Among the patients with low-amplitude fractionated diastolic electrograms in the coronary venous recordings (Group 1), endocardial ablation targeted sites with pre-systolic or diastolic electrograms facing epicardial areas with low-amplitude fractionated diastolic signals (Figure 18). In these patients, endocardial ablation was successful in one patient and was unsuccessful in seven patients (marked slowing down of the VT cycle length without termination in one patient). Among the patients without diastolic electrograms in the coronary venous recordings (Group 2), endocardial ablation was successful in 18 of 25 patients (72%).

Epicardial mapping and ablation

In four highly symptomatic patients presenting with VT storm or incessant VT, in whom low-amplitude fractionated diastolic electrograms were recorded in the coronary veins, mapping and ablation from the pericardial space was performed after prior unsuccessful endocardial ablation. After pericardial puncture, pacing from the pericardial space demonstrated concealed entrainment (in two patients) and entrainment with a QRS match in 9 of 12 ECG leads in two patients. Epicardial ablation was mainly performed during sinus rhythm based on the epicardial voltage map in combination with entrainment manoeuvres due to the different VT morphologies and the haemodynamic impairment of these patients. Epicardial ablation consisted in a linear lesion transecting an epicardial low-voltage zone in three patients (Figure 3) and a focal ablation with a small number of RF applications in one patient. The combination of endocardial and epicardial ablation eliminated all mappable VTs in the four patients and led to non-inducibility of all ventricular tachyarrhythmia in two patients (Figure 4). One patient with epicardial ablation died of heart failure 4 weeks after the ablation.

Follow-up

A total of 56 ablation procedures (mean 1.6 procedures per patient, range 1–4) were performed to treat all mappable monomorphic VTs. After a follow-up of 28 ± 22 months after ablation, clinical success (freedom of VTs or reduction of VT episodes by >95%) was achieved in 26 patients (78%). Complete freedom from VT was obtained in 17 patients (51%). Three patients with unsuccessful ablation or VT recurrence died of heart failure due to incessant VT, one ICD patient could be treated pharmacologically (flecainide), one patient had to undergo heart surgery (aneurysmectomy), and two patients underwent successful heart transplantation.

Discussion

Recording of low-amplitude fractionated diastolic electrograms using microelectrode catheters manoeuvred into coronary vein branches adjacent to an aneurysma or scar can identify VTs with an epicardial origin.

Epicardial ventricular tachycardia origin

In VT re-entry circuits, the circulatory excitation wavefront propagates through regions of scar, and their depolarization is not detectable on the standard ECG recorded from the body surface. Electrograms recorded directly from these regions are of low amplitude and have fractionated potentials consistent with asynchronous activation of myofibre bundles and slow conduction. In the majority of VT patients with ischaemic cardiomyopathy, some portion of the re-entry circuit exists in the subendocardial accessible to endocardial entrainment and ablation. Endocardial sites, where concealed entrainment can be demonstrated with a stimulus to QRS (S–QRS) interval <70% of the VTCL, have the highest incidence of tachycardia termination and are referred to as isthmus sites. In re-entry circuit isthmus sites, the S–QRS interval matches the electrogram–QRS interval (EG–QRS) during tachycardia. Sites with concealed entrainment and an S–QRS interval amounting to 30–70% of the VTCL are termed as central and proximal isthmus sites.

In our study, low-amplitude fractionated electrograms in the coronary veins with an EG–QRS interval of 30–70% of the VTCL were recorded in 8 of 33 patients and were associated with a poor outcome of endocardial catheter ablation. As concealed entrainment by pacing from the coronary veins could not

---

Figure 2 Flow chart of endocardial and epicardial ventricular tachycardia ablation (DPs, low-amplitude fractionated diastolic electrograms).

In five patients, earliest pre-systolic coronary venous electrograms preceded earliest pre-systolic endocardial electrograms without recording low-amplitude fractionated diastolic potentials (AIV, n = 3; PIV, n = 2).

---
be demonstrated in any patient, it was not possible to determine whether these epicardial sites represented a critical part of the VT re-entry circuit or were only bystander sites unrelated to the re-entry circuit. However, the following findings suggest that the low-amplitude fractionated diastolic electrograms recorded in the coronary veins identify epicardial isthmus sites critical for the VT re-entry circuit. (i) Concealed entrainment of the VT could be demonstrated by pacing from the pericardial space close to the site where low-amplitude fractionated diastolic potentials were recorded through the coronary veins. (ii) Successful epicardial ablation was performed in all four patients undergoing the pericardial access after unsuccessful endocardial ablation.

Impact on the ablation strategy

Epicardial ablation is associated in experienced centres with a risk of 5 and 2% of acute and delayed major complications related to epicardial access. After unsuccessful endocardial ablation, microelectrode catheter mapping through the coronary veins can help to detect an epicardial involvement of the VT re-entry circuit. Owing to the distribution of the coronary venous branches, epicardial ablation through a pericardial access is typically performed with a risk of acute complications of 5% and of delayed complications of 2%.

Figure 3 Epicardial ablation through a pericardial access. (A) Twelve-lead electrocardiogram of the ventricular tachycardia in a 69-year-old patient after prior anterior infarction. (B) Fluoroscopic position of the multipolar microelectrode catheter in the anterior interventricular vein (arrows) and of the epicardial mapping and ablation catheter advanced through a pericardial access after substernal puncture (Abl epi). In parallel, coronary angiography was performed. The anterior interventricular vein runs in parallel with the left anterior decendent (LAD). (C) Tracings from the multipolar microelectrode catheter positioned in the anterior interventricular vein. Low-amplitude fractionated diastolic electrograms were recorded in anterior interventricular vein 3/4 and anterior interventricular vein 5/6. (D) Epicardial CARTO voltage map (CARTO epi) was performed using a pericardial access. The green dot marks the epicardial site where concealed entrainment with a post-pacing interval equal to the ventricular tachycardia cycle length could be demonstrated. The fluoroscopic position of the epicardial ablation catheter at the site of concealed entrainment (Abl epi) is shown in (B). An epicardial linear lesion (red dots) intersecting the epicardial low-voltage zone eliminated the ventricular tachycardia. The patient required another endocardial ablation session and subsequently remained free from further ventricular tachycardias during the 12-month follow-up.
mapping through the coronary veins can only be performed in defined regions of the LV and the sensitivity of this method to detect an epicardial VT origin is limited. In our study, recording of low-amplitude fractionated diastolic electrograms in the coronary veins characterized a subgroup of patients with a poor outcome of endocardial ablation requiring epicardial ablation. Our experience suggests that the presence of diastolic potentials in the coronary veins makes it highly likely that epicardial ablation is going to be required for successful ablation. The successful epicardial ablation sites were in close proximity to the positions where low-amplitude fractionated diastolic electrograms were recorded through the coronary veins. The findings suggest that the diastolic potentials recorded in the coronary veins can be used as a landmark for successful ablation from the pericardial space.

**Limitations**

Concealed entrainment of the VT by pacing from the coronary veins could not be performed in any of the eight patients with a suspected epicardial VT origin. The most probable explanation is that the pacing stimuli were not strong enough to capture the myocardium due to epicardial fat or fibrosis. Low-amplitude fractionated electrograms may also be found in epicardial bystander regions of the VT re-entry circuit and does not necessarily represent an epicardial isthmus site critical for the re-entry circuit. Only four patients underwent the epicardial approach and further randomized studies are necessary to study whether the detection of low-amplitude fractionated diastolic potentials in the coronary venous recordings is associated with a high rate of successful epicardial ablation in larger series of patients. None of these patients who had no venous diastolic potentials had pericardial mapping. We suggest that the results of ablation would be better if they had.

**Conclusions**

Recording of low-amplitude fractionated diastolic potentials through the coronary veins in patients with structural heart
disease can help to identify patients with recurrent VT who are candidates for an epicardial ablation if endocardial ablation fails.

Conflict of interest: none declared

References