Mechanisms that initiate ventricular tachycardia in the infarcted human heart

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BACKGROUND Precise mechanisms that initiate ventricular tachycardia (VT) in the intact infarcted human heart have not been defined.

OBJECTIVE The purpose of this study was to investigate the mechanisms that underlie human postinfarct VT initiation.

METHODS Noncontact mapping of the left ventricle was performed in 9 patients (age 67.1 ± 7.8 years, ejection fraction 34.4% ± 5%) with previous myocardial infarction and sustained monomorphic VT.

RESULTS Circuits in which ≥30% of the diastolic pathway (DP) could be defined were identified in 12 VTs (cycle length 357 ± 60 ms). Eighteen VT episodes were initiated with pacing, and one occurred spontaneously. Ten complete and two partial circuits were mapped (89% ± 25% of the DP). In all complete circuits, pacing led to the development of unidirectional conduction block at the location of the subsequent VT exit site and the formation of functional block creating a border(s) for subsequent DP. Wavefront velocity in the DP region slowed from 1.22 ± 0.2 m/s during sinus rhythm to 0.59 ± 0.14 m/s during VT ($P < .005$). In 11 initiation episodes, lines of functional block and areas of slow conduction developed progressively over one to six reentrant cycles before a stable DP was established and sustained monomorphic VT ensued. The formation of unidirectional or functional lines of block was not identified during identical pacing protocols that failed to initiate VT (n = 14).

CONCLUSION Initiation of sustained monomorphic VT requires the development of unidirectional block and formation of lines of functional block creating borders for a DP in areas of slow conduction. A transitional stage often exists during the initiation process before a stable VT circuit is established.

KEYWORDS Arrhythmia; Electrophysiology; Infarction; Reentry; Tachycardia; Ventricles

ABBREVIATIONS **DP** = diastolic pathway; **ECG** = electrocardiogram; **ICD** = implantable cardioverter defibrillator; **LV** = left ventricle; **MI** = myocardial infarction; **VT** = ventricular tachycardia

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Introduction

Sustained monomorphic ventricular tachycardia (VT) affects up to 5% of patients with remote myocardial infarction,[1] of which the underlying mechanism in the majority of cases is reentry.[2–4] Programmed ventricular stimulation is widely used for the initiation of sustained monomorphic VT and traditionally was considered a useful predictor of future arrhythmic events and mortality following myocardial infarction.[5] Premature extrastimuli delivered to the right ventricle can induce VT in up to 70% of patients with documented spontaneous VT,[6] increasing to 90% with left ventricular (LV) stimulation.[7] Experimental and animal models have implicated a number of different factors necessary for the initiation of stable monomorphic VT, including formation of lines of functional block,[3,8] development of a region of slow conduction,[4] formation of unidirectional block,[9] anisotropy,[10] and dispersion of tissue refractoriness leading to reentry.[11] The precise role of these factors in the initiation of spontaneous or pacing-induced VT in the intact human heart in patients with remote myocardial infarction remains to be fully determined. Greater insight into the mechanisms that underlie VT initiation may improve our ability to identify patients likely to develop VT in the future, permit more selective prescription of implantable cardioverter-defibrillator therapy, and help devise interventions to treat or prevent the spontaneous occurrence of VT.

Methods

Consecutive patients with a history of remote myocardial infarction (>6 months) and spontaneous episodes of sustained monomorphic VT despite treatment with antiarrhythmic drugs underwent noncontact mapping–guided ablation. The study was approved by the local ethics committee, and patients provided written informed consent.

Mapping procedure

High-resolution activation maps of the entire LV endocardium were created using noncontact mapping (EnSite 3000,

Drs. Segal and Chow were funded for this research by grants from the British Heart Foundation, including RG/05/009. We acknowledge support from the NIHR Biomedical Research Centre funding scheme. **Address reprint requests and correspondence:** Dr. Oliver Segal, The Heart Hospital, 16-18 Westmoreland Street, London W1G 8PH, United Kingdom. E-mail address: oliver.segal@uclh.nhs.uk (Received July 28, 2009; accepted September 16, 2009.)

1547-5271/$ -see front matter © 2010 Heart Rhythm Society. All rights reserved. doi:10.1016/j.hrthm.2009.09.025

St. Jude, St. Paul, MN, USA),[12] with the multielectrode array deployed retrogradely via the aorta. Intracardiac contact electrograms and surface 12-lead ECG were recorded on a conventional electrophysiology system ((Bard EP Lab System, Lowell, MA, USA) and bipolar electrograms filtered at 30 to 500 Hz. A quadripolar catheter deployed in the right ventricle was used to induce VT using burst pacing, double ventricular extrastimuli, or up to three extrastimuli following eight-beat drive trains (S1 600 and 400 ms). Intracardiac 4-mm nonirrigated mapping/ablation catheters (Cordis, Biosense Webster, Diamond Bar, CA, USA) were deployed retrogradely and via transseptal puncture. Patients were given heparin, with the activated clotting time maintained between 300 and 400 seconds.

Isopotential and isochronal maps and electrograms were analyzed at multiple filter settings to ensure the consistency of patterns of activation and electrogram fidelity. Distance measurements obtained using the noncontact system were calculated to account for surface curvature, as described previously.[13] VT circuits in which >30% of the diastolic pathway could be identified were included for analysis.

Definitions

Regions of scar were defined as areas of endocardium with absent or very-low-amplitude reconstructed electrograms during sinus rhythm, pacing, *and* VT. Scar was confirmed in these regions using bipolar contact catheters demonstrating electrogram amplitudes <0.5 mV. Although some investigators have defined normal endocardium as areas with bipolar electrograms >1.5 mV,[14] other investigators have chosen <0.5 mV to define true scar, with infarct border zone tissue lying between these two values.[15,16] Using the former definition would have reduced the infarct border zone size, the principal area of interest for the study. We previously used a similar definition.[17]

VT exit sites were defined as points of rapidly expanding systolic activation on the isopotential map synchronous with or just prior to QRS onset.

Lines of functional block were defined as lines that divided activation between adjacent endocardial areas by >50 ms, were not fixed (seen with pacing or tachycardia only), and varied with different rates of ventricular activation. When present, they produced dissociated activation in adjacent regions and electrograms with double potentials. They were confirmed by identification with pacing from different sites.

Diastolic pathways (DPs) were defined as regions of the LV that activated between QRS complexes during VT and were protected from systolic activation by lines of block or scar.

Transitional beats were defined as cycles of ventricular activation occurring immediately prior to the establishment of sustained monomorphic VT with different surface QRS morphology and endocardial activation patterns from monomorphic VT.

Statistical analysis

Continuous data are presented as mean and standard deviation and were compared using the Wilcoxon paired test. Data analysis was performed using SSPS 10.0 statistical software (SPSS, Inc., Chicago, IL, USA). $P < .05$ was considered significant.

Results

In 9 patients (7 men and 2 women; age 67.1 ± 7.8 years) with myocardial infarction (4 anterior, 3 inferior, 1 posterior, 1 anterior and inferior) and impaired LV function (ejection fraction 34.4% ± 5%), complete episodes of VT initiation were recorded and at least 30% of the DP, including entry and exit sites, identified (Table 1). Only in two VT circuits could <100% of the DP be identified. All patients were treated with amiodarone, and four had been treated with mexiletine (Table 1).

Episodes of VT initiation

Nineteen episodes of VT initiation (18 induced by programmed stimulation, 1 occurred spontaneously) with 12 different VT morphologies (cycle length 357 ± 60 ms) were recorded. Four of the VTs were initiated during continuous pacing (420 ± 98 ms, range 300–500 ms), 2 were initiated after delivery of closely coupled ventricular extrastimuli during sinus rhythm (280 ms/280 ms and 280 ms/260 ms), and the remaining 12 were induced with one to three premature ventricular extrastimuli (range 240–360 ms) following a drive train of eight paced beats (S1 400–600 ms). Complete circuits (100% of DP) were mapped in 10 VTs, and partial DPs (>30%) were identified in 2 VTs (overall 89% ± 25% of all DPs, range 31%–100%). Completely mapped DPs were 5.2 ± 2.1 cm long.

Figure 1 shows a schematic representation of the LV endocardium in all nine patients. Infarct scar is shown in gray. Black pacing symbols show pacing site locations. Patients 2, 3, and 9 have ≥2 pacing symbols representing the position of pacing for separate VT episodes. Paired black lines represent DPs. Numbered black dots indicate sites of the corresponding evolving transitional beat DP.

DP formation

Borders of the DPs of monomorphic VT were formed by a mixture of anatomic (infarct scar) and functional block in 7 VTs, purely by lines of functional block in 4 VTs, and entirely bordered by two areas of fixed block in 1 VT.

In all cases of VT initiation, unidirectional block formed at the same location as the subsequent monomorphic VT exit site, thereby protecting an area of myocardium that formed the DP. The activation wavefront was forced to propagate around this line of block, enter the protected area at the same location as the subsequent entry site of the monomorphic VT DP, and then propagate through this region toward the exit site region. Upon reaching the distal end of this protected region, the wavefront of activation was able to continue because either conduction block was uni-

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