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Obstacle-Induced Transition from Ventricular Fibrillation to Tachycardia in Isolated Swine Right Ventricles

Insights into the Transition Dynamics and Implications for the Critical Mass

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OBJECTIVES The study was done to test the hypothesis that an artificial anatomical obstacle prevents the maintenance of ventricular fibrillation (VF) by stabilizing reentrant wavefronts (RWF) and increases the critical mass (CM) of myocardium required to sustain VF.

BACKGROUND Artificial obstacles can anchor RWF in simulated models of VF. Whether an artificial obstacle affects multiple-wavelet VF in real tissue is unclear.

METHODS The endocardial surfaces of seven isolated, perfused swine right ventricles were mapped using a plaque of 477 bipolar electrodes with 1.6-mm resolution. An 8-mm hole was punched in the tissue. The CM was reached by tissue mass reductions, at which VF converted to periodic activity (ventricular tachycardia, VT).

RESULTS After the creation of the obstacle, the VF cycle length increased from 71.6 ± 18.4 ms to 87.5 ± 13.0 ms ($p < 0.05$). The obstacle, together with the papillary muscle, facilitated the transition from VF to VT by serving as attachment sites for the RWF. When one RWF attaches to the obstacle and another attaches to the papillary muscle, it may result in stable VT with figure-eight patterns. The CM for VF in the presence of an 8-mm hole (28.7 ± 3.8 g) was higher than in the control group (swine right ventricles without holes, 24.0 ± 3.4 g, $p < 0.05$).

CONCLUSIONS An artificial anatomical obstacle induces slowing and regularization of VF, impairs the persistence of VF as judged by an increase of the CM, and can convert VF to VT by serving as an attachment site to reentrant excitation. (J Am Coll Cardiol 2000;36:2000–8) © 2000 by the American College of Cardiology

Obstacles have been used extensively to study reentry (1–7). Naturally occurring obstacles, like arteries and other anatomical inhomogeneities, can attach a drifting spiral wave and make it stationary (8,9). Similarly, artificial obstacles may convert a meandering (detached) reentrant wavefront (RWF) into a stable (attached) one in an experimental (5) and simulated (6,9) two-dimensional (2D) tissue. In simulated three-dimensional (3D) tissue a full-thickness obstacle is capable of attaching a *single* drifting scroll wave, whereas a partial-thickness obstacle produces unstable attachment (10). Whether this holds true in real 3D tissue that sustains multiple-wavelet ventricular fibrillation (VF) remains unclear. We (11) reported that persistent and stable multiple-wavelet VF may be induced in the isolated, perfused swine right ventricle (RV). Gradual reduction of the tissue mass resulted in the reduction of the number of wavelets and eventually the termination of VF or the conversion from VF to ventricular tachycardia (VT) when a critical mass (CM) (12) was reached. The papillary muscle (PM) played a key role as an attaching structure to RWF during VT (13). However, the presence of PM did not prevent the maintenance of VF. The purpose of the present study was 1) to assess the influence of a full-thickness artificial obstacle on the dynamics of multiple-wavelet VF; 2) to determine whether an artificial obstacle can convert VF to VT by stabilizing RWF; and 3) to determine the effects of an artificial obstacle on the CM for VF.

METHODS

Experimental preparation. The study protocol was approved by the Institutional Animal Care and Use Committee. The experimental model has been previously described (11). Briefly, the hearts of seven farm pigs were quickly removed and placed in oxygenated Tyrode's solution at 37°C. The right coronary artery was cannulated and perfused with Tyrode's solution. The RV was excised; VF occurred in vivo during excision, and persisted in vitro. The fibrillating RV was then placed in a tissue bath with the endocardial surface facing on a built-in electrode array on the bottom of the tissue bath with 477 bipolar electrodes, 1.6 mm apart, arranged in 20 columns and 25 rows. The

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Abbreviations and Acronyms

CL = cycle length
CM = critical mass
pECG = pseudoelectrocardiogram
PM = papillary muscle
RWF = reentrant wavefront
SD = standard deviation
2D = two-dimensional
VF = ventricular fibrillation
VT = ventricular tachycardia

PM was cut to improve electrode contact. Data were acquired by a computerized mapping system (Unemap, Uniservices, New Zealand) at 1,000 samples per second with 16 bits of accuracy (11). Pseudoelectrocardiograms (pECG) were recorded from two electrodes placed on the opposite edges of the tissue (Fig. 1).

An 8-mm-diameter cylindrical obstacle (hole) was then created using a skin biopsy punch (5). The specific location of the obstacle within the tissue was determined by the need to avoid major epicardial arteries, but in general the hole was created near the center of the tissue, at variable distances from the PM. Several 8-s recordings were acquired before and after the creation of the obstacle.

Progressive tissue mass reduction was achieved by cutting 0.3 g to 1.5 g from the boundary of the tissue (11). If VF continued, additional tissue was removed. A transition from VF to VT was judged to occur when a regular and largely repeated pattern of activation was present consistently. Polymorphic or transient (<30 s) VT was not considered a transition. The CM for spontaneous VF was defined as the tissue weight at which spontaneous VF converted to VT. The VF was reinduced by rapid pacing at different sites; VF was considered induced if it persisted for >5 s after pacing. After successful VF reinduction, additional tissue mass reductions were made until VF could not be induced. A minimum of four different pacing sites were used before considering VF as noninducible. At the end of the experiments, bipolar diastolic pacing thresholds were measured at a minimum of five sites in the region around the obstacle. Data were compared with baseline threshold determined at similar locations.

Control data. An additional tissue was used as control. The tissue preparation was as described, including obstacle creation, but no mass reductions were performed so that the effects of the obstacle at masses above the CM could be studied. To compare CM with and without the obstacle, we used our previous studies as a control (11). In that study, the tissue preparation and experimental procedures were exactly the same, but no obstacle was placed.

Data analysis. The methods of computerized mapping have been previously reported (14). Patterns of activation were displayed dynamically on the computer screen (15). We defined attachment of a RWF to the obstacle as a mode of activation when the tip of consecutive RWFs followed a path corresponding to the boundary of the obstacle (5). In this situation, the cross-sectional area of the obstacle was the size of the core. If attachment did not occur around the obstacle, then the size of the core was calculated by tracing the point on the RWF closest to the core. The area encircled by these points was the core size (15). The number of RWFs at a given instant of VF was defined as the number of activations separated from each other by recovered but nonactivated tissue (11). A mean VF cycle length (CL) was obtained for each channel by averaging the interactivation intervals. The VF CL of the entire tissue was calculated by averaging CLs of all channels. Standard deviation (SD) of the VF CL was used to assess the variability of VF CL recorded by each channel. Slowing and regularization of VF were defined as significant increases and decreases of VF CL and VF SD, respectively. For comparisons, data were pooled from two 8-s recordings before and after creating the obstacle.

All data are presented as mean ± SD and were compared using the Student *t* test when appropriate. Two-way repeated-measures analysis of variance (ANOVA) was used to compare VT CL before and after obstacle creation, and in and out of the surrounding rim of channels. Linear regression analysis was used to determine the relation between the core size and VT CL. A p value ≤0.05 was considered significant.

RESULTS

In one tissue, the placement of an obstacle caused immediate transition from VF to VT. In the remaining six tissues, tissue mass reductions were necessary to achieve transitions from VF to VT. Spontaneous annihilation of all electrical activity occurred at the end of the experiments, only after converting to VT (never from VF). The pacing thresholds at the end of the experiments did not differ significantly from that obtained before punching the hole (0.55 ± 0.36 mA vs. 0.47 ± 0.26 mA, p = NS).

Effects of the obstacle on VF. The characteristics of VF changed immediately after creation of the obstacle. The mean VF CL increased in all tissues. In all but one tissue, there was a decrease in the SD of VF CL (Table 1). In dynamic display, baseline VF was characterized by the presence of multiple wavelets coexisting in the tissue. The creation of an obstacle did not change significantly the number of wavelets present in the tissue at any given time (from 4.8 ± 0.7 to 4.4 ± 1.3), except in one tissue. During baseline VF, we observed RWFs attached to the PM insertion for an average of 1.5 ± 1.4 cycles (13) at a time in the span of two 8-s recordings. After creation of the obstacle, attaching to the obstacle was seen with a mean of 1.7 ± 1.5 cycles. Anchoring to either structure occurred at different times in a given tissue, but the coexistence of RWF attaching to both the PM and the obstacle simultaneously was not seen during VF.