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Epicardial mapping of ventricular fibrillation over the posterior descending artery and left posterior papillary muscle of the swine heart

Thomas D. Nielsen,

Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

Jian Huang,

Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

Jack M. Rogers,

Department of Biomedical Engineering, University of Alabama at Birmingham, Birmingham, AL, USA

Cheryl R. Killingsworth, and

Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

Raymond E. Ideker

Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

Department of Biomedical Engineering, University of Alabama at Birmingham, Birmingham, AL, USA

Department of Physiology, University of Alabama at Birmingham, Birmingham, AL, USA

Cardiac Rhythm Management Laboratory, 1670 Volker Hall B-140, Birmingham, AL 35294-0019, USA, e-mail: rei@crml.uab.edu

Abstract

Background—Recent studies suggest that during ventricular fibrillation (VF) epicardial vessels may be a site of conduction block and the posterior papillary muscle (PPM) in the left ventricle (LV) may be the location of a “mother rotor.” The goal of this study was to obtain evidence to support or refute these possibilities.

Methods—Epicardial activation over the posterior LV and right ventricle (RV) was mapped during the first 20 s of electrically induced VF in six open-chest pigs with a 504 electrode plaque covering a 20 cm² area centered over the posterior descending artery (PDA).

Results—The locations of epicardial breakthrough as well as reentry clustered in time and space during VF. Spatially, reentry occurred significantly more frequently over the LV than the RV in all 48 episodes, and breakthrough clustered near the PPM ($p < 0.001$). Significant temporal clustering occurred in 79% of breakthrough episodes and 100% of reentry episodes. These temporal clusters occurred at different times so that there was significantly less breakthrough when reentry was present ($p < 0.0001$). Conduction block occurred significantly more frequently near the PDA than elsewhere.

Conclusions—The PDA is a site of epicardial block which may contribute to VF maintenance. Epicardial breakthrough clusters near the PPM. Reentry also clusters in space but at a separate site. The fact that breakthrough and reentry cluster at different locations and at different times supports

the possibility of a drifting filament at the PPM so that at times reentry is present on the surface but at other times the reentrant wavefront breaks through to the epicardium.

Keywords

Ventricular fibrillation; Cardiac arrhythmias; Cardiac mapping; Reentrant arrhythmias; Papillary muscle; Conduction block; Animal model of arrhythmia

1 Background

The mother rotor hypothesis for ventricular fibrillation (VF) states that one or more stable periodic sources (“mother rotors”) spawn wavefronts that spread across the myocardium and maintain fibrillation [1]. These wavefronts block at functional or anatomic obstacles to cause the characteristic activation sequences of VF. The evidence for the mother rotor hypothesis comes from studies of optical mapping that imaged isolated rabbit hearts, sheep right ventricles (RVs) and left ventricles (LVs), and guinea pig hearts [2]. Sustained periodic sources in these small hearts or pieces of hearts have been found to be located in the fastest activating region, i.e., the dominant domain. Recent studies suggest that anatomic heterogeneities may be important causes of block and may contribute to the maintenance of VF, and specifically, the posterior papillary muscle may be an important structure contributing to the maintenance of VF [3,4]. Studies also suggest that epicardial vessels may be a common site of conduction block and that reentrant wavefronts may anchor around the insertions of papillary muscles into the ventricular free wall [3,5–7].

Global epicardial recordings from pigs have demonstrated that the basal LV has a higher activation rate than the remainder of the LV and RV [8,9]. Epicardial mapping has demonstrated that VF wavefronts tend to propagate from the anterior LV to the anterior RV [10]. These findings suggest that the LV could be the site for a dominant domain, and the LV posterior papillary muscle (PPM), which in the pig inserts into the LV free wall just adjacent to the septum, could be the location of a stable mother rotor that gives rise to wavefronts spreading up to the epicardium and out to more distant myocardium. However, extensive epicardial mapping in pigs has not demonstrated sustained reentry [11]. Recent optical mapping studies have concluded that epicardial mother rotors do not drive VF and are not strictly necessary for VF maintenance [12,13]. It has been hypothesized that sustained reentry may occur intramurally [6] and thus cannot be detected by epicardial mapping alone.

The goal of this study was to examine the role of anatomic structures in an in situ large animal heart model, the pig heart. First, the epicardial VF activation patterns over the PPM were examined for direct evidence of sustained reentry and indirect evidence of intramural reentry. Second, the epicardial activation patterns over the posterior descending artery (PDA) were examined for direct evidence of conduction block.

2 Methods

All animals were managed in accordance with guidelines established by the AHA on research animal use [14] and protocols were approved by the Animal Care and Use Committee at the University of Alabama at Birmingham.

2.1 Data collection

VF activation patterns from both the posterior LV and RV epicardium were mapped in six open-chest mixed breed pigs. The animals were injected intramuscularly with zolazepam–tiletamine 4.4 mg/kg, xylazine 4.4 mg/kg, and atropine 0.04 mg/kg for anesthetic induction. Anesthesia was maintained with isoflurane in 100% oxygen by inhalation. Core body

temperature, arterial blood pressure, arterial blood gases, surface electrocardiogram lead II, and serum electrolytes were monitored and maintained within normal ranges throughout the study. The heart was exposed through a median sternotomy and supported in a pericardial sling.

Data were recorded from 504 unipolar electrodes in a uniform rectangular array (24×21) separated by 2 mm. The electrode plaque was fabricated from 2 mm thick Silastic sheeting to conform to the curvature of the heart. The electrode plaque was centered over the PDA and sutured in place. The plaque covered approximately 20 cm² of the posterior epicardium. Unipolar signals were filtered with a high-pass filter of 0.05 Hz and a low-pass filter of 500 Hz and then digitized to 14 bits accuracy at 2,000 samples per second using a 528 channel mapping system.

A lead containing a pacing electrode and defibrillation coil was placed in the RV using fluoroscopic guidance. The diastolic pacing threshold was determined with a standard pacing protocol. The pacing current was set to the higher of twice the pacing threshold or 1.0 mA. A defibrillator can was implanted in the left chest wall.

VF was induced through application of a 9 V DC current on the anterior LV. Four VF episodes were mapped with the posterior LV/RV plaque. Each VF episode was allowed to continue for at least 20 s before a rescue shock was delivered. Biphasic defibrillation shocks were delivered using the RV coil and the defibrillator can. If internal shocks failed, epicardial paddles were used for defibrillation.

At the end of the experiment the heart was excised and fixed in formalin. The location of the epicardial plaque was marked using sutures around the edges. The location and anatomy of the PDA and PPM were determined by direct examination and the extent of the insertion of the PPM was extrapolated on the epicardial surface.

2.2 Statistics: VF activation patterns

VF episodes were examined quantitatively using quantitative pattern analysis techniques, which have been previously described [15,16]. A single temporal sample at a recording site was considered to represent an activation in the underlying tissue when $dV/dt < -0.5$ V/s. [17] A five-point digital filter was used to calculate the temporal derivative of each electrogram. The first 20 s of VF was divided into four epochs of 5 s duration and each epoch was analyzed individually.

Wavefront isolation methods were used to compute the following quantitative descriptors of VF organization: (1) the number of wavefronts, (2) the incidence of wavefronts that fractionate into two or more separate wavefronts, (3) the incidence of wavefronts that collide, (4) the incidence, location, and timing of new wavefronts that break through onto the epicardial surface, (5) the incidence and locations of wavefronts that block, (6) the multiplicity index (number of distinct activation pathways in the rhythm), (7) the repeatability index (number of times activation pathways are traversed), (8) the mean propagation velocity, (9) the mean area swept out by wavefronts, (10) the incidence of reentry, (11) the mean number of cycles for which reentrant circuits persist, (12) the mean area circumscribed by reentrant wavefronts (core area), (13) the reentrant wavefront locations, start, and end times, and (14) the locations of wavefronts propagating onto and off of the epicardium beneath the plaque [18].

A separate analysis comparing the VF activation patterns of the LV and RV was then performed. Although the plaque was centered over the PDA in each animal, there were variations in the anatomy of the PDA and the size of the fat pad surrounding the vessel between animals. Therefore, the center third of the plaque was excluded from this analysis, and only the data from the one-third of the plaque located exclusively over the LV and the one-third of

the plaque located exclusively over the RV were analyzed. Comparison of the LV and RV was performed using a two-tailed *t*-test. Bonferroni correction was applied to the analysis of VF activation patterns. Although many of the 15 measured parameters are likely interdependent, a conservative approach was used, and a *p* value of 0.003 (0.05/15) was considered significant.

2.3 Analysis of clustering of breakthrough and reentry

Clustering of times of breakthrough of wavefronts to the epicardium was analyzed using the Ederer–Myers–Mantel test [19–21]. A cumulative distribution function (CDF) was used to quantify the probability of a certain number of events occurring at particular points in time. A CDF of breakthrough events was constructed and compared with both a uniform distribution and the Poisson distribution, which expresses the probability of independent events. The electrode plaque was divided into a 3×3 grid and the number of breakthrough events in each of the nine sectors was compared to a uniform distribution.

Clustering of reentry in time was also analyzed with the Ederer–Myers–Mantel and scan tests [19–21]. The centroids (the center of mass of reentry circuits) of the reentrant cycles were calculated as previously described [15] and plotted. A two-dimensional Kolmogorov–Smirnov test was then used to analyze reentry centroids clustering in space.

For clustering analysis, a *p* value of 0.05 was considered significant.

2.4 Analysis of block

Sites of conduction block were determined using two separate methods: (1) using the locations where wavefronts terminated identified by the VF wavefront analysis techniques, and (2) finding locations in which there was significant activation delay between adjacent electrode sites, as has been previously described. [3] If the conduction time between two neighboring electrodes was >50 ms, block was considered to have occurred between the two electrodes.

3 Results

3.1 VF activation patterns

Analyses of the two sides of the plaque showed dramatic differences between wavefronts on the LV and RV (Table 1). The number of wavefronts, multiplicity, repeatability, and the incidence of reentry were higher on the LV than the RV epicardium. Reentrant wavefronts lasted a similar number of cycles but were larger on the RV. The mean wavefront speed was slightly faster and the mean wavefront size was larger on the LV.

3.2 Breakthrough

Analysis of breakthrough sites showed clustering in both space and time on the posterior epicardium (Figs. 1 and 2). Significant clustering of the breakthrough sites was found in all 48 VF episodes using the Kolmogorov–Smirnov test. Wavefronts broke through to the epicardium at a number of sites on the plaque, but the most commonly repeated pattern was a breakthrough near the PPM with later spread across the LV (Fig. 1). In 38 of the 48 VF episodes (79%), the times at which wavefronts broke through to the epicardium were not randomly distributed, but clustered temporally (Fig. 3) according to the Ederer–Myers–Mantel and scan tests.

3.3 Reentry

Reentry cycles also clustered in time and space. The centroids of the reentrant cycles showed significant clustering in space in all VF episodes (Fig. 2). The time intervals during which reentry was present on the epicardium were not random, but clustered in time for all 48 VF episodes using the Ederer–Myers–Mantel and scan tests.

3.4 Reentry-breakthrough comparison

While breakthrough times and reentry cycles were not mutually exclusive, breakthrough was significantly less ($p<0.0001$) common when reentrant wavefronts were present than when they were absent in all 48 VF episodes (Fig. 3). Locations of breakthrough sites and reentry centroids were not significantly correlated.

3.5 Wavefront direction

Wavefronts that propagated on and off the edges of the plaque were analyzed. More wavefronts propagated onto the apical side of the plaque vs. the basal side of the plaque (96 ± 32 vs. 45 ± 51 , $p<0.0001$) and onto the left ventricular side of the plaque vs. the right ventricular side of the plaque, (134 ± 62 vs. 37 ± 28 , $p<0.0001$). More wavefronts propagated off the basal side of the plaque vs. the apical side of the plaque (137 ± 55 vs. 28 ± 22 , $p<0.0001$), but equally off the LV and RV sides (84 ± 45 vs. 74 ± 44 , $p=0.08$). Thus, wavefronts propagated onto the plaque from the LV apex and midportion and propagated off the plaque at the LV base.

3.6 Block

Both methods of determining sites of conduction block showed block was significantly more prevalent in the epicardial myocardium adjacent to the PDA than over the remainder of the plaque (Fig. 4).

4 Discussion

The major findings in this study were that both breakthrough and reentry clustered in time and space over the posterior LV. Breakthrough and reentry occurred at different points in time, and although both clustered in space, the locations of these phenomena were not similar. Activation patterns differed dramatically between the left and right ventricular epicardium.

The finding that breakthrough and reentry both cluster in space supports the hypothesis that anatomical structures of the myocardium affect VF activation, [3,4] and hence may be important in the maintenance of VF. Breakthrough and reentry also clustered in time, but were prominent at different times in the fibrillation sequence. A possible explanation is that breakthrough and reentry may be part of a single mechanism which is maintaining VF, but may appear differently on the epicardium throughout the episode. Breakthrough clustered over the LV PPM, which may be an important anchor for fibrillation cycles [3–7]. Models of three-dimensional rotors have suggested that they may anchor to discontinuities in the cardiac muscle resulting in a stationary rotor, [22,23] and the PPM insertion site contains abrupt discontinuities in cardiac fiber orientation. Additional studies including intramural mapping and more extensive epicardial mapping of this area will be important to further explore the possibility of a three-dimensional rotor contributing to the maintenance of VF.

Another possibility is that the activity seen on the epicardium may represent intramural focal wave fronts and not reentry [24]. A recent study in pigs showed, however, that foci were far less common during the first stage of VF and increased with time [25]. Our study examined only the first 20 s of VF.

In this study, breakthrough and reentry showed a poor correlation in spatial clustering. Reentry did not tend to cluster over the papillary muscle. This may be due to the location of the epicardial plaque in this study, as the PPM was located under a corner of the electrode plaque. It is possible that reentrant wavefronts propagated onto and off of the plaque and the reentrant circuit was not recognized.

VF activation patterns in this study are consistent with previous studies from our group. Rogers, et al. studied separate electrode plaques on the left and right ventricular epicardium and found greater numbers of wavefronts on the LV [9]. This study showed similar findings on the posterior epicardium.

The PDA was found to be an important site of block, consistent with previous studies which have indicated that block occurs more frequently in the myocardium along the paths of blood vessels [3,7]. If a stable periodic source is driving VF near the PPM, this could be an important anatomic obstacle leading to more chaotic activation sequences of VF. An hypothesis that could explain increased block over the PDA is the effect of curvature of the cardiac fibers as they pass underneath epicardial vessels. However, convex curvature is thought to contribute to conduction block, not concave curvature as probably occurs beneath the vessel, which is thought to make block less likely [26]. It is also possible that conduction block near the coronary vessel is an artifact of the experimental preparation. Exposing the epicardium to air causes the heart to be surrounded by an electrical insulator that confines to the heart the portion of current which normally passes into the surrounding thorax. This should decrease the electrical load on the most epicardial portion of the wavefronts, increasing their ability to excite tissue as they propagate and decreasing their tendency to block [27]. However, even after the heart is exposed to air, the conductive blood in the epicardial vessels and the surrounding connective tissue continue to be available as a current sink. This could decrease the ability of wavefronts to excite tissue just in those regions and increase the local incidence of block relative to the rest of the epicardium in which the block has been suppressed.

Another hypothesis is that conduction block is caused because the epicardium away from the blood vessels is cooler than next to the vessels, [28] because it is exposed to air at room temperature in the open-chest experimental preparation while the coronary arteries are perfused with warm solution. Further studies are needed to determine whether the conduction block adjacent to the PDA is a real phenomenon or an artifact of the open-chest preparation.

4.1 Study limitations

This study was limited by the fact that mapping was confined to the epicardium. We propose a three-dimensional intramural mechanism to explain our findings. A full understanding of such a mechanism would require not only epicardial and endocardial recordings, but also transmural recordings which would likely have an effect on the patterns of VF. Also, while this in situ study may more directly represent the conditions of VF in intact animals than isolated perfused heart models, the amount of epicardium sampled was limited, and there may be artifacts induced by the preparation.

4.2 Clinical implications

Increased understanding of the mechanisms for VF maintenance may lead to better treatments during cardiac resuscitation in the future. The results of this study support the hypothesis that all parts of the ventricles do not contribute equally to the maintenance of VF. Rather, anatomic structures, such as papillary muscles and blood vessels, influence the frequency of conduction block and reentry during VF.

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Abbreviations

VF	ventricular fibrillation
PPM	posterior papillary muscle
LV	left ventricle
RV	right ventricle
PDA	posterior descending artery

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Fig. 1.
Example of epicardial activation pattern of a breakthrough wavefront. *PDA*: posterior descending artery, *PPM*: posterior papillary muscle

Fig. 2.

Sites of breakthrough and reentry during a 20 s episode of VF in one animal. Breakthrough clusters near the posterior papillary muscle (*PPM*). Reentry clusters in space (*dots*) but not at the same location as breakthrough (*color*). The figure shows the plaque as viewed from the posterior of the heart. The plaque was centered over the posterior descending artery (*PDA*)

Fig. 3. Timing of breakthrough and reentry in an example VF episode showing clustering in time. Breakthrough was significantly less likely when reentrant wavefronts were present. *Solid bars (top)* represent times when reentrant wavefronts were present. *Marks (bottom)* indicate times of breakthrough of wavefronts onto the plaque surface

Fig. 4.

The number of times conduction block occurred at each electrode position during a single 20 s VF episode in one animal. Block was most frequent over the posterior descending artery. The graph shows the number of occurrences of block at each electrode on the plaque, as indicated to the right

Table 1

Ventricular fibrillation wavefront characteristics in the left ventricular third and right ventricular third of the posterior epicardial plaque

	Right ventricle	Left ventricle	<i>p</i> value
Number of wavefronts per second	9.8±6.5	15.2±6.0	<0.0001
Fractionation (percent of wavefronts)	6.3±7.9	8.3±7.4	0.04
Collision (percent of wavefronts)	6.5±7.7	8.5±7.6	0.05
Breakthrough (percent of wavefronts)	14.6±16.7	15.1±12.1	0.83
Block (percent of wavefronts)	13.9±17.6	14.3±11.5	0.86
Multiplicity	1.9±1.6	3.1±1.3	<0.0001
Repeatability	4.1±2.2	4.9±1.2	<0.003
Speed (m/s)	0.33±0.16	0.38±0.14	0.05
Area swept out (mm ²)	188±101	211±90	0.05
Reentry (percent of wavefronts)	0.2±0.7	0.7±0.9	0.001
Number of reentry cycles	1.3±1.0	1.3±1.3	0.95
Reentry perimeter (mm)	18.8±6.7	14.7±6.5	<0.0001
Reentry area (mm ²)	17.2±11.2	10.8±9.3	<0.0001
Reentry duration (ms)	97.9±32.7	79.3±34.9	<0.0001
Peak dV/dt (V/s)	-1.5±0.3	-1.4±0.2	0.60

Multiplicity index quantifies the number of distinct activation pathways. Repeatability index quantifies the number of times activation pathways are traversed. After Bonferroni correction, a *p* value of 0.003 was considered statistically significant.