Spiral Wave Attachment to Millimeter-Sized Obstacles

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Background—Functional reentry in the heart takes the form of spiral waves. Drifting spiral waves can become pinned to anatomic obstacles and thus attain stability and persistence. Lidocaine is an antiarrhythmic agent commonly used to treat ventricular tachycardia clinically. We examined the ability of small obstacles to anchor spiral waves and the effect of lidocaine on their attachment.

Methods and Results—Spiral waves were electrically induced in confluent monolayers of cultured, neonatal rat cardiomyocytes. Small, circular anatomic obstacles (0.6 to 2.6 mm in diameter) were situated in the center of the monolayers to provide an anchoring site. Eighty reentry episodes consisting of at least 4 revolutions were studied. In 36 episodes, the spiral wave attached to the obstacle and became stationary and sustained, with a shorter reentry cycle length and higher rate. Spiral waves could attach to obstacles as small as 0.6 mm, with a likelihood for attachment that increased with obstacle size. After attachment, both conduction velocity of the wave-front tip and wavelength near the obstacle adapted from their pre-reentry values and increased linearly with obstacle size. In contrast, reentry cycle length did not correlate significantly with obstacle size. Addition of lidocaine 90 μmol/L depressed conduction velocity, increased reentry cycle length, and caused attached spiral waves to become quasi-attached to the obstacle or terminate.

Conclusions—Anchored spiral waves exhibit properties of both unattached spiral waves and anatomic reentry. Their behavior may be representative of functional reentry dynamics in cardiac tissue, particularly in the setting of monomorphic tachyarrhythmias. (Circulation. 2006;114:2113-2121.)

Key Words: action potentials ■ reentry ■ ventricles ■ arrhythmia ■ tachyarrhythmias ■ mapping ■ antiarrhythmic drugs

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The question then arises whether a spiral wave attached to small obstacles behaves more like a functional spiral wave that happens to be stationary or like anatomic reentry in a tissue ring.7 Thus, the goals of the present study were first, to track the process of spiral-wave induction by programmed stimulation; second, to determine the obstacle sizes sufficient for attachment; third, to compare the dynamics (specifically, cycle length [CL]) of the spiral wave when it is pinned compared with when it is free; fourth, to determine which reentry parameters of the pinned spiral wave are strongly influenced by the dimensions of the obstacle; and fifth, to determine how attachment of the spiral wave to the obstacle is affected by a reduction in cellular excitability.
Signals were low-pass filtered at 320 Hz and amplified with 8 custom-designed, 32-channel, printed circuit boards. Signals were sampled at 1 kHz and digitized with four 64-channel, 16-bit analog-to-digital boards (Sheldon Instruments, San Diego, Calif). Data were stored, displayed, and analyzed with software written in Visual C++ (Microsoft; Redmond, Wash), LabVIEW (National Instruments, Austin, Tex), and MATLAB (MathWorks, Natick, Mass).

Cell Culture

Neonatal rat ventricular myocytes were dissociated from 2-day-old Sprague-Dawley rats (Harlan, Indianapolis, Ind) with the use of trypsin (US Biochemicals, Cleveland, Ohio) and collagenase (Worthington, Lakewood, NJ), as described previously. Cells were resuspended in M199 culture medium (Life Technologies, Rockville, Md) supplemented with 10% heat-inactivated fetal bovine serum (Life Technologies), differentially preplated in two 45-minute steps, and plated in a 12-well culture plate (at 10^5 cells per well) that contained 22-mm-diameter, circular plastic coverslips previously coated with fibronectin (25 μg/mL) at room temperature for 2 hours. At day 2 after cell plating, serum was reduced to 2%. Experiments were performed on days 4 to 7 after plating. During experiments, the cell monolayers were stained with 10 μmol/L di-4-ANEPPS and continually superfused with warmed (36±0.5°C) oxygenated Tyrode’s solution (in mmol/L: 135 NaCl, 5.4 KCl, 1.8 CaCl₂, 1 MgCl₂, 0.33 NaH₂PO₄, 5 HEPES, 5 glucose).

Formation of Anatomic Obstacles

Holes of different sizes were drilled in the coverslips before fibronectin plating, and photomicrographs were taken of the holes to record their size. To create the smallest obstacles (0.6 mm), a microcontact printing method was used to pattern the obstacle onto the coverslips. Masks were designed in Photoshop (Adobe, San Jose, Calif) and printed on transparent acetate sheets at 1200-dpi resolution. Silicon wafers spin-coated with a 10-μm thickness of SU-8 photoresist were exposed to ultraviolet light through the mask. The photoresist was later developed, and polydimethylsiloxane was poured over the wafer and allowed to bake overnight. Polydymethylsiloxane stamps were later used to transfer 50 μg/mL human fibronectin (Sigma, St. Louis, Mo) onto the coverslips. Pluronic (0.2% wt/vol, F127, BASF, Florham Park, N.J.; 2114 Circulation November 14, 2006)

Figure 1. Contact fluorescence imaging system. Transmembrane voltage of cardiac cells is measured with voltage-sensitive dye. Emission signals from cells in the experimental chamber are passed through a fiber optic bundle and recorded and amplified via 253 channels over a 17-mm-diameter field of view. Excitation light source consists of a concentric array of green, light-emitting diodes.

Methods

Experimental Setup

A schematic of the experimental setup is shown in Figure 1. Maps of transmembrane voltage were recorded by contact fluorescence imaging. The experimental chamber was placed directly above a bundle of 253 optical fibers 1 mm in diameter, arranged in a tightly packed, 17-mm-diameter hexagonal array. An array of 26 high-power, green light-emitting diodes (Kingbright, Taipei, Taiwan) delivered excitation light. The light-emitting diodes were powered at 30 mA and were arranged in a concentric pattern behind a 35-mm-diameter interference filter (530±25 nm). A No. 1 glass coverslip spin-coated with 3 layers of red photoresist (PSCRed, Brewer Science, Rolla, Mo) was placed between the coverslips. Pluronic (0.2% wt/vol, F127, BASF, Florham Park, N.J.; 2114 Circulation November 14, 2006)}

Figure 2. Initiation of reentry. A, Schematic of stimulating electrodes. Bipolar line electrode serves as an S1 electrode. Area electrode serves as an S2 electrode and consists of 4 parallel pairs of line electrodes (the anodal return electrodes for the 4 cathodal electrodes are situated ~1 mm directly above their corresponding cathodal electrodes, similar to that for the single bipolar line electrode, but are not shown). B, Color bar on left indicates pseudocolor map of transmembrane potential. Cells in blue area are at resting potential or repolarized potential, whereas cells in red area are fully depolarized. Gray translucent lines in top frames show location of S1 and S2 electrodes. Numbers at bottom of each frame show time (milliseconds) relative to the make of the most recent S1 stimulus; time 40 corresponds to 0.332 seconds in Movie III in the Data Supplement). A planar action potential triggered by S1 propagates through the monolayer from top to bottom. Just before the third frame, an S2 stimulus is applied in the wake of the planar wave. Because cells in the bottom half of the monolayer are in a more refractory state, the action potential propagates upwards and to the right. By the fifth frame, these cells have recovered from refractoriness, and the action potential wave front curves around with the eventual formation of a spiral wave. CL eventually settled at 134 ms. C, Phase-contrast photomicrographs of obstacles used to anchor spiral waves: 1.35-mm-diameter obstacle with hole-drilling (left) and 0.6-mm obstacle fabricated by microcontact printing (right).
Park, NJ) was used to block areas where no cell attachment was desired.

**Data Analysis**

Two-second recordings were made at intervals as short as 1 minute during reentry. Baseline drift was corrected by subtraction of a fitted second-order polynomial curve from the optical signal. Animations of electrical propagation were generated from signals that were low-pass filtered between 0 and 100 Hz with a fourth-order elliptical filter. The activation time was defined as the instant of maximum positive slope. CL and action potential duration at 80% repolarization (APD80) were determined as the average CL and APD80 of individual beats over a 2-second interval for all episodes of sustained reentry. Diastolic interval (DI) was defined as (CL – APD80). Wavelength at the wave-front tip circumscribing the anatomic obstacle was defined as \( \text{APD}_{80} \times \text{CV}_{\text{tip}} \), where \( \text{CV}_{\text{tip}} \) is average conduction velocity (CV) of the wave-front tip (defined as obstacle circumference/RCL), and RCL is reentry CL. Linear interpolation was done between sampling points, and the location of the wave-front tip was visually determined at 1-ms intervals as the point of maximal wave-front curvature for unattached spiral waves. For the lidocaine experiments, we measured the path length of the tip (PLtip) by manually tracing the path and averaging over 3 cycles, and \( \text{CV}_{\text{tip}} \) was defined as \( \text{PL}_{\text{tip}} / \text{RCL} \). Signals from channels lying within the obstacle were excluded from the data analysis, and channels that had <40% of the maximum signal intensity obtained across all channels were rejected.

The relative activation times at each recording point of the mapping array were used to calculate CV before reentry. To compare velocities among different episodes in the same monolayer, CV was calculated along a selected path of recording sites and averaged over different stimulus responses. Paths were chosen to be sufficiently far away from the S1 stimulus electrode so that latency delays associated with excitation could be neglected. Data are expressed as mean ± SD unless stated otherwise.

The significance of hole size on various reentry parameters was determined by 1-way ANOVA followed by Scheffé’s post hoc test. Values of \( P < 0.05 \) were considered to be significant. Pearson’s correlation coefficient (\( r \)) between different reentry parameters and hole size was also determined with a single-parameter linear regression model and between RCL and multiple reentry parameters with a multivariable regression model.

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

**Results**

Spiral waves were initiated with a paired-pulse (S1-S2) protocol. Monolayers were paced (Figure 2A) with a bipolar line stimulus (S1) at 3 Hz for 30 beats before an area stimulus (S2) was applied in the wake of the last S1 stimulus. A plane wave was launched by the S1 electrode at time 0 to 10 ms and propagated from the top to the bottom of the monolayer (Figure 2B). At 40 ms, the wave front was one fourth of its way down the coverslip and moving at 24.1 cm/s. By 146 ms, the repolarizing tail was two thirds of the way down the coverslip. At 140 to 150 ms, an S2 stimulus was applied where the area electrodes are shown and resulted in a local area of excitation at 182 ms. At 224 ms, the S2-initiated wave could propagate only to the right and the top because of still refractory tissue in the lower half of the monolayer. The S2 wave then propagated downward into the formerly refractory tissue, and after 244 ms, it could propagate to the left. Finally, by 334 ms, the wave completed 1 clockwise rotation and continued to circulate (not shown). A full animation (Movie I) showing an initiation sequence with spiral wave drift in a monolayer without an obstacle can be found in the Data Supplement.

By changing the location of the coverslip with respect to the stimulus electrode, as well as the strength of the S2 pulses, we could guide the initiation point of the functional reentrant wave to be near the center of the coverslip, where an anatomic obstacle (Figure 2C) was placed. Eighty reentry episodes that consisted of at least 4 revolutions each were induced in this manner. Most of the spiral waves lasted for only a transient period before they drifted to the
too large to anchor the spiral wave.

When the characteristics of attached spiral waves were analyzed in terms of their pre-reentry and post-reentry waveform parameters in the subset of data for 3 different obstacle sizes (circumferences of 3.6, 6.0, and 8.2 mm), we found no significant differences in pre-reentry wavelength (measured at 3-Hz pacing) at the different sizes ($P=0.827$ overall; $r=-0.044$; Figure 5A). Pre-reentry wavelength also exceeded the obstacle circumference in all cases (line of unity-slope). However, after attachment, the reentry wavelength of the wave-front tip adapted and was both less than and linearly related to the obstacle circumference ($P<0.001$ overall with 1-way ANOVA and $P<0.001$ with Scheffé’s test for all pairwise comparisons of the 3 obstacle sizes; $r=0.985$; Figure 5B). Similarly, there was no correlation of pre-reentry CV with obstacle size ($P=0.528$ overall; $r=-0.125$; Figure 5C) but a strong correlation of CV$_{tip}$ with the different obstacle sizes ($P<0.001$ overall and $P<0.05$ for all pairwise comparisons with Scheffé’s test; $r=0.888$; Figure 5D). Finally, somewhat surprisingly, there was no correlation between RCL and obstacle size ($P=0.674$ overall; $r=0.009$; Figure 5E). Further analysis with a multivariable regression model showed that RCL correlated strongly with pre-reentry CV, pre-reentry APD$_{80}$, CV$_{tip}$, and reentry APD$_{80}$ ($R^2=0.932$) but with significance only for reentry APD$_{80}$ ($P<0.001$).

In a final series of experiments, we added lidocaine 90 $\mu$mol/L to depress the excitability of the monolayer. In 3 control experiments, this concentration of lidocaine did not have a significant effect on APD$_{80}$ (paired t test for data pooled over the range of stimulus rates tested; Figure 6A) but depressed CV by $\sim$10 cm/s at all stimulus rates (Figure 6B) or by $\sim$60% at reentry rates. These results are consistent with a selective depressive effect of lidocaine on Na channels, and little or no effect on Ca or K channels.

When lidocaine was applied to ongoing spiral waves, they became less stable and underwent augmented cycles of transient detachment and reattachment, as reflected in the isochrone maps (Figure 7A; see also Movies IV, V, and VI in the Data Supplement). The maximum separation of the wave-front tip from the obstacle was larger for the 1.9-mm-diameter (D1) obstacles relative to the obstacle size than that for the 2.6-mm-diameter (D2) obstacles (Figure 7B). For D1 obstacles, RCL at 130 seconds of drug exposure increased by an average of 14.6% to 11.6% (126.5$\pm$14.0 to 144.8$\pm$20.3 ms) in all 7 monolayers and increased further with time, in 1 case up to 156.0% after 10 minutes of drug exposure. PL$_{tip}$ increased by 12.7$\pm$8.4% at 130 seconds, whereas APD$_{80}$ and DI increased by 11.8$\pm$10.5% (87.5$\pm$6.9 to 97.9$\pm$12.5 ms) and 21.2$\pm$15.7% (37.1$\pm$6.2 to 44.7$\pm$8.4 ms), respectively.

There was no change in CV$_{tip}$ ($-1.0\pm$9.4%). The spiral waves terminated in 6 of 7 monolayers within 6 minutes of lidocaine exposure (presumably by detachment and collision with the tissue boundary). PL$_{tip}$ increased by 35.9$\pm$27.6% in the last measurement before termination.

However, when lidocaine was applied to spiral waves attached to D2 obstacles, the spiral wave terminated in only 1 of 5 monolayers even after 10 minutes, presumably after complete detachment. After 130 seconds of drug exposure, RCL increased by 9.2$\pm$6.3% (107.2$\pm$18.2 to 115.4$\pm$20.3 ms), PL$_{tip}$ by 10.4$\pm$7.7%, APD$_{80}$ by 7.9$\pm$8.8% (75.9$\pm$10.6% to 81.7$\pm$12.2 ms), and DI by 9.0$\pm$12.9% (29.9$\pm$7.8% to 32.4$\pm$8.3 ms) in all 5 monolayers. Again, there was no change in CV$_{tip}$ (2.5$\pm$4.3%). At 10 minutes, RCL in the surviving 4 reentries increased a total of 69.6$\pm$13.5% (103.4$\pm$18.7 to 174.6$\pm$28.5 ms), PL$_{tip}$ by 22.3$\pm$6.2%, APD$_{80}$ by 56.1$\pm$14.2% (73.3$\pm$10.3 to 114.2$\pm$17.4 ms), and DI by 105.7$\pm$23.4% (28.9$\pm$8.6 to 58.2$\pm$12.2 ms).
**Discussion**

The main findings of this study are as follows. First, spiral waves can attach to obstacles as small as 0.6 mm in diameter, with a likelihood for attachment that increases with obstacle size. Once attached, RCL decreases from its unattached value, and the spiral wave speeds up. CV and wavelength at the wave-front tip but not CL then become a function of obstacle size. Administration of lidocaine to ongoing, attached spiral waves increases RCL, tip-path length, APD80, and DI and can eventually lead to detachment and termination of the spiral wave. The likelihood for termination is higher for smaller (1.9-mm diameter) obstacles than for larger (2.6-mm diameter) obstacles.

**Properties of Attached Spiral Waves**

Purely anatomic reentries in a ring around an obstacle have certain distinguishing characteristics. First, the wave front is nearly planar (while revolving around a central locus). Second, when the obstacle size (and path length) is increased, CL increases. Third, if there is no wave-back–wave-front interaction (ie, a fully excitable gap), CV is independent of obstacle size. Finally, with wave-back–wave-front interactions, complicated dynamic behavior such as oscillations in APD and CL can arise that involve CV and APD restitution.

In contrast, with purely functional (spiral wave) reentry, the wave front is curved and has the approximate shape of...
an Archimedean spiral except near the tip. Both the front and back of the wave meet at a phase singularity point that is close to the tip. The wave-front tip circumscribes a core that is unexcited.13,14 Because of wave-front curvature, CV is slowest at the tip and faster on the arm, such that CL remains constant at all locations outside the spiral core. The wave-front tip can follow different trajectories (eg, circle, line, or cycloid pattern), depending on the excitability of the medium,14,15 and can circumscribe a core that is unexcited.6,13,14 It can also drift in the presence of parameter gradients (eg, in refractoriness, excitability, or fiber direction) in the medium.6,14,15 Complex dynamic behavior such as meandering, quasi-periodicity, scalloping, and breakup involving CV and APD restitution can occur.15–17

Spiral waves that are anchored to an anatomic obstacle represent a transition between functional and anatomic reentry where there is a mix of both kinds of behavior, such that there is a continuous transition in behavior as obstacle size is varied from zero (pure spiral wave) to very large (pure anatomic reentry).1,3 We have previously termed this type of reentry “2D anatomic reentry.”2 With unattached spiral waves, the wave-front tip encounters the greatest source-load mismatch, such that conduction fails completely in the direction of the core. In attaching to a small obstacle, the Archimedean shape of the spiral is preserved, but the loading effects of the core are largely removed from the wave-front tip. Therefore, conduction can proceed more quickly and shorten CL, provided that the obstacle is not so large as to negate this effect by substantially adding to the path length of the wave-front tip (as may have occurred in some previous studies5,6). The results of the present study show that CV of the wave-front tip correlates well with obstacle size, as expected for a curved wave front subject to different source-load ratios at its tip for different diameter obstacles. Furthermore, reduction of cellular excitability by lidocaine not only reduces CV but also augments the source-load effects at the wave-front tip and results in an increased path length of the tip and partial or complete detachment of the tip from the obstacle. The lidocaine-induced increase in path length is accompanied by an increase in RCL, an increase in APD (owing to the rate dependence of APD), and an increase in DI.

The increase in CVtip with an increase in obstacle size most likely explains why RCL did not increase with obstacle size (Figure 5D) despite the increase in tip path length. However, Ikeda et al5 showed that RCL of pinned spiral waves increases with obstacle size. A possible reason for the difference in results is that in their experiments, spiral waves attached to obstacles 6 to 10 mm in size, which may have less of a size-dependent increase in CVtip. Even so, their correlation between CL and obstacle size was not strong (P=0.07). Ikeda et al5 also did not observe stable pinning to obstacles smaller than 6 mm, whereas in the present experiments, spiral waves could attach with varying likelihoods to obstacles with sizes 10-fold smaller, down to 0.6 mm. Other factors such as CV, 2D versus 3D tissue substrate, or induction protocol may also have contributed to the difference in results. The induction protocol in the present study placed the tip of the nascent spiral wave close to the obstacle, which likely facilitated its attachment.

It appears that spiral waves attached to small obstacles have wave-back–wave-front interaction near the tip like that in anatomic reentries that lack a fully excitable gap. Several observations support this viewpoint. First, single-site recordings show that cells near the core do not repolarize fully and lack a clear diastolic potential during reentry (Figure 3C). Second, RCL varies significantly with reentry APD, as would be expected if the spiral wave front were to encroach on more refractory tissue and propagate more slowly as APD increases. Third, CV and wavelength at the wave-front tip adapt to the obstacle size during reentry. When the obstacle size becomes very small, the wave-front tip detaches from the obstacle for part or all of the cycle. However, we did not observe significant oscillations in APD or CV, as would be expected with strong wave-back–wave-front interaction and steep APD restitution.1

In summary, spiral waves anchored to small anatomic obstacles have a mix of spiral wave and anatomic reentry properties. Such behavior may be important to consider with regard to reentry dynamics in cardiac tissue, in conjunction with that of pure, unattached spiral waves,
which are typically assumed in theoretical and computational studies.

**Theoretical Basis for Spiral Wave Attachment**

Theoretical work suggests that spiral waves are attracted toward localized heterogeneity.\(^{18}\) The likelihood for attachment of the wave depends on the size of the obstacle and the impact parameter (the closest distance between the obstacle and the trajectory of the wave-front tip that would occur in the absence of the obstacle).\(^{18,19}\) Thus, a spiral wave drifting by an obstacle will either become trapped or pass by. Obstacles that are partially excitable as opposed to fully unexcitable will have lower trapping capacity, and spiral waves attached to such obstacles may meander.\(^{19}\)

Given that the intact myocardium is replete with small structures (eg, blood vessels) that may act as anatomic obstacles, the question arises as to why spiral waves are only occasionally stationary (giving rise to monomorphic tachycardia). According to the reconstruction of the coronary arterial tree by Kaimovitz et al,\(^{20}\) vessels of order 9 to 11 (diameter 700\(/\)H9262\(\) to 3 mm) are confined to the epicardium. Therefore, coronary vessels that would be expected, on the basis of the present study, to be significant attractors (diameter >0.6 mm) are located mainly along the epicardial surface, and spiral waves attached to such obstacles may meander.\(^{19}\)

On the other hand, sustained monomorphic ventricular tachycardias can be observed with local myocardial scarring and fibrosis. It is a common occurrence with sarcoidosis\(^{21}\) or Chagas’ cardiomyopathy\(^{22}\) and also occurs, although infrequently, with dilated cardiomyopathy.\(^{23}\) We hypothesize that with these forms of structural disease, the frequency of millimeter or larger obstacles increases substantially and increases the incidence of stationary spiral waves. Their ability to be sustained, however, is mitigated by factors that act to detach the waves from the obstacle, as discussed below.

**Detachment of Pinned Spiral Waves With Reduction in Excitability**

The detachment of spiral waves has not been studied previously for circular obstacles but has been characterized for wave fronts circumnavigating line obstacles that have a terminus end.\(^{14,24,25}\) The ability of the wave to remain attached depends on a source-load balance of charge, ie, the diffusive-reaction charge available within the wave-front versus the charge required to extend the wave-front tip.\(^{24}\) Reduced excitability leads to an increase in the pivoting radius of the spiral wave\(^{14}\) and, ultimately, detachment of the wave from the terminal end of the obstacle. Cabo and coworkers showed that after the application of tetrodotoxin to thin slices of sheep ventricle, the spiral wave detaches and can either undergo decremental conduction\(^{26}\) or, if it is sustained, break away from the tip of the line obstacle over a distance up to 1 cm and then either return along a line of block to the other side of the obstacle or initiate a spiral wave.\(^{25}\) Unlike line obstacles that have abrupt increases in source-load conditions at their ends, the circular obstacles in the present experiments do not possess sharp corners and hence might not be considered to be conducive for wave detachment. Nonetheless, under conditions of severely reduced excitability owing to rapid excitation during reentry in combination with block of sodium channels by lidocaine, we have

![Image](http://circ.ahajournals.org/)
shown that the detachment of spiral waves from millimeter-sized circular obstacles is enhanced and that the excursion of the wave from the obstacle can extend to several obstacle diameters (several millimeters; Figure 7A). Lines of block are important in functional reentrant arrhythmias, because the length of the line is influential in setting RCL, and changes in the length or location of the line of block can affect CL of the tachyarrhythmia. The present experiments support those of Cabo et al., which suggest the potential role of an anatomic obstacle toward the formation of a functional line of block under conditions of reduced excitability.

**Lidocaine as an Antiarrhythmic Agent**

Lidocaine is a class I drug that is used as an antiarrhythmic agent for patients with ventricular tachycardia. Its effects include decreased excitability, slowing of conduction and formation of conduction block, increase in refractoriness or refractory gradient, and oscillation in increase of reduced excitability.

**Funding for this work was provided by National Institutes of Health grant R01-HL62393 to Dr. Tung and a scholarship from the Public Service Commission, Singapore to Z.Y. Lim.**

**Acknowledgments**

We wish to thank M. Roselle Abraham for helpful discussions and Yibing Zhang for helping to prepare some of the supplemental material.

**Sources of Funding**

Funding for this work was provided by National Institutes of Health grant R01-HL62393 to Dr. Tung and a scholarship from the Public Service Commission, Singapore to Z.Y. Lim.

**Disclosures**

None.

**References**


**CLINICAL PERSPECTIVE**

Monomorphic ventricular tachycardia is often associated with local myocardial scarring or fibrosis and may be the result of a functional “spiral” wave that is anchored to a structural heterogeneity. This research demonstrates the feasibility of studying this form of cardiac arrhythmia in an in vitro experimental model. Anatomic obstacles (0.6 mm or larger) were situated in the center of monolayers of cultured neonatal rat ventricular cells. Using voltage-sensitive dyes and optical mapping, the authors show that spiral waves can be generated systematically, that the incidence of anchoring of spiral waves increases with obstacle size, and that the dynamics (cycle length) of the spiral wave depend on obstacle size. Furthermore, the authors show that the class 1B drug lidocaine, which reduces cellular excitability, facilitates the partial or complete detachment of anchored spiral waves so that they are no longer stationary. This finding reveals multifaceted effects of lidocaine. Partial detachment results in an increase in the tip trajectory of the spiral wave, thereby augmenting the slowing effect of lidocaine on conduction velocity, and increases cycle length, action potential duration, and diastolic interval. Complete detachment allows the spiral wave to move about, so that it may attach to a new anchoring site (change in ECG morphology), collide with other wavelets and form new wave breaks (a proarrhythmic effect), or collide with tissue boundaries and terminate (an antiarrhythmic effect).
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Circulation. 2006;114:2113-2121; originally published online November 6, 2006;
doi: 10.1161/CIRCULATIONAHA.105.598631
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2006 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circ.ahajournals.org/content/114/20/2113

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