Drift and breakup of spiral waves in reaction–diffusion–mechanics systems

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Rotating spiral waves organize excitation in various biological, physical, and chemical systems. They underpin a variety of important phenomena, such as cardiac arrhythmias, morphogenesis processes, and spatial patterns in chemical reactions. Important insights into spiral wave dynamics have been obtained from theoretical studies of the reaction–diffusion (RD) partial differential equations. However, most of these studies have ignored the fact that spiral wave rotation is often accompanied by substantial deformations of the medium. Here, we show that joint consideration of the RD equations with the equations of continuum mechanics for tissue deformations (RD–mechanics systems), yield important effects on spiral wave dynamics. We show that deformation can induce the breakup of spiral waves into complex spatiotemporal patterns. We also show that mechanics leads to spiral wave drift throughout the medium approaching dynamical attractors, which are determined by the parameters of the model and the size of the medium. We study mechanisms of these effects and discuss their applicability to the theory of cardiac arrhythmias. Overall, we demonstrate the importance of RD–mechanics systems for mathematics applied to life sciences.

Rotational dynamics can induce the breakup of spiral waves into complex spatiotemporal patterns. We also show that mechanics leads to spiral wave drift throughout the medium approaching dynamical attractors, which are determined by the parameters of the model and the size of the medium. We study mechanisms of these effects and discuss their applicability to the theory of cardiac arrhythmias. Overall, we demonstrate the importance of RD–mechanics systems for mathematics applied to life sciences.

Mathematical Model

Our RDM model is based on a three-variable Fenton–Karma RD model for cardiac excitation (15), coupled with the soft-tissue mechanics equations described in refs. 12 and 16:

\[ \frac{\partial u}{\partial t} = \nabla^2 u - I_d(u, v) - I_m(u) - I_s(u, w) - I_I(u, C), \]

\[ \frac{\partial v}{\partial t} = \frac{\Theta(u_e - u) - v}{\tau_v(u)}, \]

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Abbreviations: RD, reaction–diffusion; RDM, RD–mechanics; BZ, Belousov-Zhabotinsky; [t.u.], dimensionless time units; [s.u.], dimensionless space units.

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Is the delay between activation and active force development. Maximal conductance of $g_{fi}$ which represents the active stress generated by the medium. The $CMN$ thus the maximal value of active tension. Doubling mechanics are modulated by the variable function ($\frac{\partial W}{\partial E_{MN}}$, $\frac{\partial W}{\partial E_{NM}}$) together with the parameter stress components, and $uc_{i}$ may be regarded as general descriptions of the sodium, potassium, and calcium currents, respectively, of an excitable cardiac cell.

After excitation, the tissue in our model contracts, and the currents $I_{w}$ is the maximal conductance and reversal potential, respectively, of the stretch-activated channels. Following ref. 16, the current in Eq. 8 is present only if $\sqrt{C} > 1$ (which indicates stretch).

The value of $E_{s}$ in one of the main determinants of the effects of deformation on wave propagation and was varied in our computations. The complete list of parameters of the models used in this study is given in supporting information (SI) Table 1.

Numerical Integration Methods. The coupled RDM model was solved by using a hybrid approach that combines an explicit Euler scheme for the RD system, with nonlinear finite element techniques for large deformation mechanics. Full details are given in refs. 12 and 16. The numerical parameters were the following. Euler computations were performed on a deforming grid of up to $513 \times 513$ finite difference points by using no-flux boundary conditions. For all simulations, we used a time integration step of $\Delta t = 0.1$ (dimensionless time units [t.u.]) and a space integration step of $\Delta x = \Delta y = 0.8$ (dimensionless space units [s.u.]), consistent with previous studies involving a similar RD model (15). Each mechanical element contained between 6 × 6 and 33 × 33 electrical grid points, and the mechanics solution steps were separated by between 10 and 80 excitation integration steps (consistent with refs. 12 and 16). When solving Eq. 5, the boundaries of the medium were fixed in space, which is consistent with an isometric contraction regime, a standard experimental procedure for muscle mechanics, during which end points or edges of the tissue are fixed to maintain a constant overall dimension. Isometric contraction is appropriate for isovolumic phases of contraction and relaxation during the cardiac cycle, for which the overall physiological action of these channels is depolarization of the membrane in response to stretch, as shown in the majority of experimental observations from isolated cardiac tissue and the whole heart. Experimental studies of the electrophysiological properties of stretch-activated channels show that they are activated instantly by mechanical stimulation, and the current–voltage ($I-V$) relationship for the most important nonspecific cation channel is linear (18, 19). On the basis of these observations, linear ionic models for $I_{s}$ have been proposed (20, 21). These linear models have been used to study the effects of mechanical stretch on heart tissue by using detailed ionic models of the cardiac myocyte. Therefore, we believe that a linear time-independent description also will be sufficient for our low-dimensional formulation for cardiac cells. Thus, we use $I_{s} = G_{s}(\sqrt{C} - 1)(u - E_{s})$.

The Hodgkin–Huxley-type gating variable $v$ determines inactivation of $I_{f}$, with the time constant given by: $\tau_{v}(u) = \tau_{1}$ for $u \leq 0.085$; $\tau_{v}(u) = \tau_{2}$ for $0.085 < u < 0.125$; $\tau_{v}(u) = \tau_{3}$ for $0.125 \leq u < u_{c}$; and $\tau_{v}(u) = \tau_{4}$ for $u \geq u_{c}$. The gating variable $w$ determines activation of $I_{s}$, with the time constant $\tau_{w}(u) = 125$ for $u \leq 0.25$ and $\tau_{w}(u) = 170$ for $u > 0.25$. The currents $I_{f}$, $I_{w}$, and $I_{s}$ may be regarded as general descriptions of the sodium, potassium, and calcium currents, respectively, of an excitable cardiac cell.

After excitation, the tissue in our model contracts, and the mechanics are modulated by the variable $T_{w}$ (given by Eq. 4), which represents the active stress generated by the medium. The function $\varepsilon(u) = 1$ for $u \leq 0.05$ and $\varepsilon(u) = 0.1$ for $u > 0.05$ governs the delay between activation and active force development. $k_{T}$ governs the rate of tension development during excitation and thus the maximal value of active tension. Doubling $k_{T}$ results in an approximate two-fold increase in active tension. The mechanical part of our model is unchanged from ref. 16. The main equations here are the equations of stress equilibrium (Eq. 5) formulated by using the second Piola–Kirchhoff stress tensor, $T_{MN}$ in Eq. 6, which contains two parts: (i) the active stress components, $T_{w}C_{MN}$ where $C_{MN} = \frac{\partial x_{i}}{\partial x_{j}}\frac{\partial x_{j}}{\partial x_{k}}$ is the right Cauchy–Green deformation (metric) tensor and $C$ is its determinant and (ii) the passive elastic stress components, which are expressed in terms of the derivatives of a strain energy function ($W$) with respect to components of Green’s strain tensor, $E_{MN} = 1/2(C_{MN} - \delta_{MN})$, where $\delta_{MN}$ is the unitary tensor. For the purposes of this study, the strain energy function was chosen to be the isotropic Mooney–Rivlin constitutive law (17), $W = c_{1}(I_{1} - 3) + c_{2}(I_{2} - 3)$, where $I_{1}$ and $I_{2}$ are principal invariants of $C_{MN}$ and $c_{1}$ and $c_{2}$ are stiffness coefficients that, together with the parameter $k_{T}$ from Eq. 4, determine local deformation during contraction ($c_{1} = 2, c_{2} = 6$, and $k_{T} = 10$ for all simulations, chosen to give rise to relative local deformations of $\approx 15\%$).

The direct influence of deformation on the excitation properties is given by the stretch-activated current $I_{s}$. In general, there are three groups of mechanically activated channels in the heart, but only two of them (the cation nonselective channels and the potassium-selective channels) are activated by stretch (11). The overall physiological action of these channels is depolarization of the membrane in response to stretch, as shown in the majority of experimental observations from isolated cardiac tissue and the whole heart. Experimental studies of the electrophysiological properties of stretch-activated channels show that they are activated instantly by mechanical stimulation, and the current–voltage ($I-V$) relationship for the most important nonspecific cation channel is linear (18, 19). On the basis of these observations, linear ionic models for $I_{s}$ have been proposed (20, 21). These linear models have been used to study the effects of mechanical stretch on heart tissue by using detailed ionic models of the cardiac myocyte. Therefore, we believe that a linear time-independent description also will be sufficient for our low-dimensional formulation for cardiac cells. Thus, we use $I_{s} = G_{s}(\sqrt{C} - 1)(u - E_{s})$.  

\begin{align*}
\frac{\partial w}{\partial t} &= \Theta(u - 0.25) - w, \\
\frac{\partial T_{w}}{\partial t} &= \varepsilon(u)(k_{T} - T_{w}), \\
\frac{\partial}{\partial x_{M}}(T_{MN} \Delta x_{N}) &= 0, \\
T_{MN} &= \frac{1}{2} \left( \frac{\partial W}{\partial E_{MN}} + \frac{\partial W}{\partial E_{NM}} \right) + T_{s}C_{MN}^{-1}, \\
\nabla^{2}u &= \frac{\partial}{\partial x_{M}} \left( \sqrt{C}C_{MN}^{-1} \frac{\partial u}{\partial x_{N}} \right),
\end{align*}

where $\Theta(x)$ is the standard Heaviside step function: $\Theta(x) = 1$ for $x \geq 0$ and $\Theta(x) = 0$ for $x < 0$. Eqs. 1–3 provide a standard low-dimensional model of cardiac electrical propagation, which includes a qualitative description of three main ionic currents that modulate the activation of cardiac tissue: the fast inward current $I_{f}(u, v) = -g_{f}0\Theta(u - 0.25)(1 - u)(u - 0.25)$, with a maximal conductance of $g_{f}0 = 7.2$, determines the primary excitation of a cell; the slow outward current $I_{o}(u) = 0.05u^{4}\Theta(0.2 - u)$ accounts for recovery of cell properties after excitation; the slow inward current $I_{w}(u, w) = \Theta(u - 0.2)uw(0.46 + 0.085 \cdot \tanh[k(u - 0.5)])$ determines the duration of the excitation pulse; and $I_{s}(u, c)$ = the stretch-activated current, which will be described later. The variable $u$ represents the (nondimensional) transmembrane potential scaled to the interval [0, 1].
into complex spatiotemporal patterns (Fig. 1b) that persisted for the duration of our simulations (~50 rotations).

We investigated the factors underpinning the transition from a stable rotating spiral into spiral breakup. In Eqs. 1–3, the main influence of mechanical deformation on excitation appears in two ways: (i) via the stretch-activated current $I_s$ in Eq. 1 and (ii) caused by deformation of the tissue, as expressed in Eq. 7. We studied the relative contributions of these two factors to the spiral wave instability. We performed one simulation using the same parameter values and initial conditions as the simulation in Fig. 1b but in the absence of $I_s$. In this case, spiral wave stability persisted despite the tissue deformations (SI Fig. 6a).

In another simulation, $I_s$ was maintained similarly to the computation illustrated in Fig. 1b, but the effect of tissue deformation on wave propagation was neglected (i.e., rather than by using Eq. 7, the Laplacian was evaluated by using $\nabla^2 u = (\partial^2/\partial x^2) + (\partial^2/\partial y^2)$, as for the undeformed configuration). In this case, we observed that the onset of spiral breakup, and its subsequent complexity, was similar to the simulation in Fig. 1b (see SI Fig. 6b).

In additional sets of computations, we found that spiral breakup occurred only if the conductance of the stretch-activated channel was $G_s \geq 0.025$. We found that this threshold value (which we denote as $G_{sTH}$) was modulated by other parameters of the model that influence the stretch-activated current in Eq. 8. If the reversal potential for the stretch-activated current was decreased to $E_s = 0.75$, then the conductance threshold for breakup increased to $G_{sTH} = 0.039$, and a further decrease to $E_s = 0.5$ resulted in $G_{sTH} = 0.065$. Clearly, decreasing $E_s$ in Eq. 8 reduces the magnitude of the stretch-activated current, and thus a larger value of $G_s$ is necessary for breakup to occur. The complete dependence of $G_{sTH}$ on $E_s$ is illustrated in SI Fig. 7.

The observation that mechanically induced spiral wave breakup was primarily caused by the stretch-activated current was somewhat unexpected, because it is a depolarizing current, and such currents typically promote excitation in cardiac tissue. Thus, we investigated how propagation block could be caused by the stretch-activated current, and it turns out that the mechanism of this effect is related to the so-called “accommodation phenomenon,” whereby the threshold for activation increases as the rate of depolarization is decreased. This effect has been studied in electrophysiology since 1936 (22, 23).

We illustrate this effect by using an example that incorporates a recent detailed ionic model for human cardiac cells (24). This model uses a widely accepted biophysical description of the sodium current, for which conductance of the sodium channels is proportional to the product of activation ($m$) and inactivation ($h$) gates: $INa = m^3hj$. Following the Hodgkin–Huxley approach (23), the dynamics of the gating variables are given by equations of the form: $dm/dt = (m_m(u) - m)/\tau_m(u)$, where the parameters of the voltage-dependent functions $m_m(u)$ and $\tau_m(u)$ are fitted to experimental measurements. Similar exponential relaxation equations are used for the $h$ and $j$ gates. The steady-state values $m_s(u)$, $h_s(u)$, and $j_s(u)$ depend on the transmembrane voltage and are shown in Fig. 2a. As voltage increases, we see that the activation gate goes from 0 to 1, and the inactivation gate goes from 1 to 0. It is important to note that the inactivation curve approaches zero at a voltage of around $u \sim 0.15$, whereas the activation curve starts to increase for higher values of voltage above about $u \sim 0.25$. Thus, if cardiac tissue is depolarized slowly from the resting state $u = 0$ such that the gating variables approximately follow their steady-state values, then $INa$ will be inactivated (at $u = 0.15$) before the voltage reaches the activation threshold ($u = 0.25$). A similar situation will occur if cardiac tissue is incompletely repolarized after excitation such that the resting potential is above the inactivation value of $u = 0.15$. This type of $INa$ inactivation occurs in our simulations and results in spiral wave breakup.

This finding is illustrated in Fig. 3a, which shows the time course of the transmembrane voltage $u$, the Fenton–Karma variable $v$ (which accounts for inactivation of the fast inward current $I_n$), and the currents $I_n$ and $I_s$ at a point where the wave block occurs (marked by the filled square in Fig. 3b). The horizontal pink lines in Fig. 3a upper and c show the voltage above which $I_n$ is inactivated by the $v$ gating variable ($u = 0.15$). Because $I_n$ is responsible for excitation and thus wave propagation, inactivation of $I_n$ results in wave block.

Now let us explain the onset of new wave breaks. During spiral wave rotation (Fig. 3a upper and c inset), we see that the minimal diastolic value of the transmembrane potential initially is slightly below the pink (inactivation) line, resulting in recovery of the variable $v$ up to values of $\sim 0.6$ (Fig. 3a lower), which allows recovery of $I_n$ required for generation of a new action potential. However, we also observed that the minimal diastolic voltage increased, and after the third action potential the transmembrane potential did not decrease below the inactivation value. As a result, the inactivation variable $v$ was not recovered and remained at zero, and thus $I_n$ did not recover as well (around $u = 0.15$).
potential was caused by the stretch-activated current. We can see that $I_s$ was maximal during the late repolarization phase, which prevented the voltage from decreasing below the pink line. To confirm this effect, we performed a similar simulation but with $I_s$ blocked (Fig. 3d). In this case, repolarization continued below the inactivation threshold. Thus, the $I_s$ current was not inactivated, and the spiral wave did not break up.

The breakup we observed was caused by sodium current inactivation. However, although the representation of the sodium current in the Fenton–Karma model provides a qualitatively correct description of the activation–inactivation processes of this current, it is not based on experimental data of these processes. To underline the importance of the activation–inactivation processes for our mechanisms, we developed a modification of the Fenton–Karma model, which included biophysically based activation and inactivation curves of the fast sodium current. We replaced the Heaviside-based activation function $\Theta(u - 0.25)$ from Eq. 1 with the activation curve $n^3$ from the TNNP model (24), scaled to the interval $[0, 1]$ (Fig. 2a) and the Heaviside inactivation function $\Theta(u - u_c)$ from Eq. 2 with the voltage-dependent inactivation curve $h \times j$ (Fig. 2a). Thus, in this modified model, activation and inactivation processes are based on experimentally measured properties of the sodium current. We studied spiral wave rotation with this model and found that with these modifications we also obtained mechanically induced breakup of the spiral wave. As in the case of the Fenton–Karma model, after a few rotations the spiral wave broke down into a complex spatiotemporal pattern (Fig. 2b). Overall, the breakup process was similar to that observed in the Fenton–Karma model; however, our modification of $I_s$ resulted in some increase in the wavelength of the spiral waves.

**Spiral Wave Drift.** We studied the dynamics of spiral wave rotation by using parameter values for which spiral breakup was absent in the absence of deformation. Under these conditions, the spiral wave rotation was stationary with a circular motion of the spiral tip (Fig. 1a). Using the same parameter values in the presence of mechanical activity, we observed drift of the spiral wave to the center of the medium (Fig. 4a) and subsequent meander of the spiral around the center. The meander pattern in Fig. 4b resulted from Eq. 9 describes a cycloidal motion that is a superposition of a clockwise spiral wave rotation with frequency $f_0$ along a circle of radius $R_0$ and a counterclockwise circular motion along the radius $R_1$ with frequency $f_1$. For Fig. 4a, these parameters are $R_0 = 7.50$ [s.u.], $f_0 = 11.95 \times 10^{-3}$ [t.u. $]^{-1}$, $R_1 = 4.86$ [s.u.], $f_1 = 0.56 \times 10^{-3}$ [t.u. $]^{-1}$. We performed several simulations with different initial spiral wave locations, and in all cases, the spiral wave approached the center of the medium and meandered along a similar trajectory to that in Fig. 4.

We also have studied how the size of the medium affects spiral wave drift. Fig. 4b illustrates the behavior of a spiral wave in a larger medium ($151 \times 151$ grid points, compared with the $141 \times 141$ grid in Fig. 4b). In this case, the spiral wave also drifted to the center, but its meander pattern was of larger overall dimension. This meander also was reproduced by using Eq. 9 with $R_0 = 7.50$ [s.u.], $f_0 = 11.95 \times 10^{-3}$ [t.u. $]^{-1}$, $R_1 = 10.67$ [s.u.], $f_1 = 0.37 \times 10^{-3}$ [t.u. $]^{-1}$. Thus, the change in medium size did not affect the spiral wave rotation ($R_0$ and $f_0$ are the same for Fig. 4a and b); however, the radius and the period of the circular meander trajectory were greater for the larger medium.

Fig. 5a shows the effect of medium size on the characteristics of the meander pattern. We observed that $R_0$ and $f_0$ remained constant, whereas $R_1$ increased and $f_1$ decreased with the increase in medium size. As a result, the radius of the meander pattern increased, and the speed along the circular trajectory $2\pi \times R_1 \times f_1$ increased only slightly with size (data not shown).
In a similar manner to the spiral breakup analysis, we studied how this drift was modulated by the two feedback effects of deformation: the stretch-activated current \( I_a \) and the effect of tissue deformation of wave propagation. We observed that drift of the spiral wave occurred in the absence of \( I_a \), although the meander pattern was not cycloidal (SI Fig. 8a). However, the drift speed was much slower than that in Fig. 4a. After 120 rotations, the spiral wave had drifted approximately one-third of the medium width, whereas by the same time in Fig. 4b, the spiral had approached and made several rotations about the center of the medium. In SI Fig. 8b, we see that \( I_a \) alone induced drift and meander of the spiral wave similar to that in Fig. 4a. The characteristics of the meander trajectories also were similar \((R_0 = 7.36 \text{ s.u.}, f_0 = 12.07 \times 10^{-3} \text{ [t.u.]}^{-1}, R_1 = 4.23 \text{ [s.u.]} f_1 = 0.56 \times 10^{-3} \text{ [t.u.]}^{-1}\), although there was a small increase in the spiral wave frequency \( f_0 \) and a slight decrease in the radius of the circular motion \( R_1 \) compared with that of Fig. 4a. Thus, for these parameters, we conclude that the dominant factor driving spiral drift is the stretch-activated current.

We believe that the mechanism of this drift is similar to the resonant drift of spiral waves reported in refs. 25 and 26. As demonstrated in ref. 26, a periodic modulation of the properties of an excitable medium in synchrony with the period of a spiral wave resulted in drift and subsequent stable meandering of the spiral wave tip. In our model, deformation of the medium also produced a periodical modulation of the tissue properties in synchrony with the spiral wave period as a result of the excitation–contraction coupling. Thus, the effects of mechanics in the present study are likely to be similar to the effects of periodical forcing during resonant drift, leading to drift and meandering attractors for spiral wave rotation.

Discussion

We have demonstrated that deformation has a pronounced effect on spiral wave rotation and can induce either breakup or drift and meander of spiral waves.

By using very general descriptions of the excitation–mechanics properties, our modeling has demonstrated that stretch-activated channels can induce spiral wave breakup. This conclusion requires confirmation both experimentally and by using modeling studies involving detailed ionic descriptions of cardiac tissue. In support of the latter, we have shown that spiral breakup occurred as a result of inactivation of the fast inward current \( I_{in} \), which was caused by diastolic depolarization mediated by the stretch-activated current. Furthermore, the notion that the stretch-activated current can block action potential has been demonstrated by using the Beeler–Reuter ionic model for ventricular cells (20), for which it was shown that increasing the conductance of the stretch-activated current resulted in failure of excitation of cardiac cells (see figure 3 in ref. 20). Note also that inactivation of the fast sodium current by depolarization has been observed experimentally (e.g., ref. 23) and reproduced by using ionic models of cardiac tissue (27), for which it can cause block of propagation.

We suggest that the mechanism of spiral drift is similar to the resonant drift mechanism. Resonant drift of spiral waves in cardiac tissue has not been studied experimentally in biological tissues but has been shown to exist in detailed ionic models of cardiac tissue (28), as well as in experiments involving BZ reactions (26). Therefore, it is likely that these effects of mechanics on spiral wave dynamics also could be reproduced by using more detailed experimental and modeling studies in cardiac tissue and in BZ reaction.

Here, we have presented a general study of spiral wave dynamics in a deforming medium, but many potentially important factors have been neglected, such as the fibrous anisotropy of cardiac tissue, which is important both for the electrical and mechanical properties of the heart. We chose not to consider this factor because the main aim of this study was to investigate the basic effects of mechanics on a general RD system. The influence of cardiac anisotropy is likely to add additional effects and will need to be addressed in future studies.

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Corrections

IN THIS ISSUE, MEDICAL SCIENCES. For the “In This Issue” summary entitled “Carvedilol sidesteps G proteins,” appearing in issue 42, October 16, 2007, of Proc Natl Acad Sci USA (104:16392), the figure caption appeared incorrectly. The online version has been corrected. The figure and its corrected caption appear below.

Carvedilol recruits β-arrestin to the β2-adrenergic receptor. The β-arrestin2-GFP is shown in green.

www.pnas.org/cgi/doi/10.1073/pnas.071062104

PERSPECTIVE. For the article “Powering the planet: Chemical challenges in solar energy utilization,” by Nathan S. Lewis and Daniel G. Nocera, which appeared in issue 43, October 24, 2006, of Proc Natl Acad Sci USA (103:15729–15735; first published October 16, 2006; 10.1073/pnas.0603395103), the authors note that in Fig. 1, the charges shown in the solar fuel cell are on the wrong sides of the cell. The holes should be at the anode, and the electrons should be at the cathode. This error does not affect the conclusions of the article. The corrected figure and its legend appear below.

Fig. 1. H₂ and O₂ are combined in a fuel cell to generate a flow of electrons and protons across a membrane, producing electrical energy. The solar fuel cell uses light to run the electron and proton flow in reverse. Coupling the electrons and protons to catalysts breaks the bonds of water and makes the bonds H₂ and O₂ to effect solar fuel production.

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BIOPHYSICS. For the article “Drift and breakup of spiral waves in reaction–diffusion–mechanics systems,” by A. V. Panfilov, R. H. Keldermann, and M. P. Nash, which appeared in issue 19, May 8, 2007, of Proc Natl Acad Sci USA (104:7922–7926; first published April 27, 2007; 10.1073/pnas.0701895104), the authors note that on page 7922, right column, the first sentence in Mathematical Model, “Our RDM model is based on a three-variable Fenton–Karma RD model for cardiac excitation (15), coupled with the soft-tissue mechanics equations described in refs. 12 and 16...,” should instead read: “Our RDM model consists of RD equations developed by F. H. Fenton (personal communication) and is based on a three-variable Fenton–Karma RD model for cardiac excitation (15), coupled with the soft-tissue mechanics equations described in refs. 12 and 16...,” where \( \Theta(x) \) is the standard Heaviside step function: \( \Theta(x) = 1 \) for \( x \geq 0 \) and \( \Theta(x) = 0 \) for \( x < 0 \).” should instead read: “Our RDM model consists of RD equations developed by F. H. Fenton (personal communication) and is based on a three-variable Fenton–Karma RD model for cardiac excitation (15), coupled with the soft-tissue mechanics equations described in refs. 12 and 16...,” where \( \Theta(x) \) is the standard Heaviside step function: \( \Theta(x) = 1 \) for \( x \geq 0 \) and \( \Theta(x) = 0 \) for \( x < 0 \).” Additionally, on page 7923, left column, beginning on line 10 of the text, the formula for \( I_{si} \) is incorrect in part. The portion of the formula appearing as \( (0.46 + 0.085 \tanh[k(u - 0.5)]) \) should instead appear as: \( (0.23 + 0.085\tanh[10(u - 0.65)]) \).” Finally, on page 7926, in the first sentence of the Acknowledgments, the authors would like to more specifically acknowledge the assistance of Dr. Fenton. Therefore, “We thank Dr. F. Fenton, Prof. P. J. Hunter, and Dr. P. Kohl for valuable discussions” should instead read: “We are grateful to Dr. F. H. Fenton, who kindly provided equations used in the construction of our RDM model, and to Prof. P. J. Hunter and Dr. P. Kohl for valuable discussions.” These errors do not affect the conclusions of the article.

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