One-Dimensional Spiral Waves, Source Defects, and Initiation of Cardiac Arrhythmia

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Traveling Pulses and Threshold Solutions

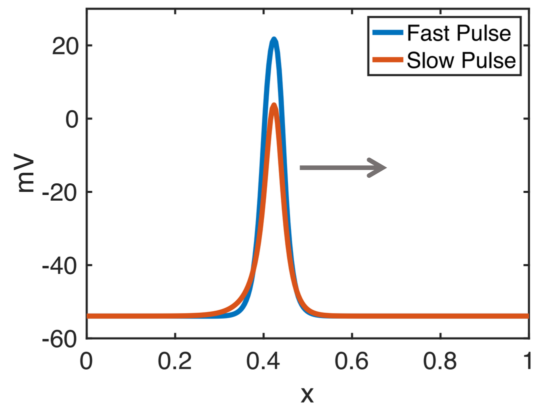
Morris-Lecar System

- Conductance model for voltage (V) and potassium gating (n)

$$V_t = \delta V_{xx} - [I_{Ca}(V) + I_K(V,n) + I_L(V)] + I_{\text{app}}$$
$$n_t = \epsilon\left(n_\infty(V) - n\right)/\tau_n(V)$$

$\rightarrow$ Reaction-diffusion system: $U_t = DU_{xx} + F(U), U = (V,n)^T$

- **Saddle-node bifurcation at $\epsilon = \epsilon_{SN}$:**
  - Stable fast pulse: $U_f(x,t)$
  - Unstable slow pulse: $U_s(x,t)$
- Cardiac tissue is an **excitable media**
  - *Subthreshold* Input: Decays to rest
  - *Superthreshold* Input: Traveling pulse



Threshold solution: separates basins of attraction for fast pulse and rest state

- $\epsilon_* < \epsilon < \epsilon_{SN}$ **:** Slow pulse
- $\epsilon < \epsilon_*$ **:** 1D spiral wave
  - Infinite series of "reflections"

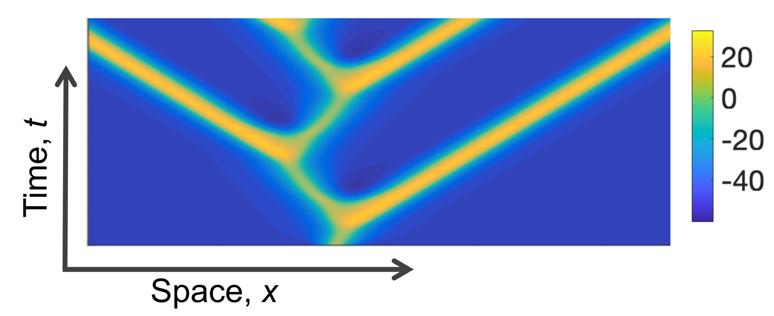


Figure 1. 1D spiral wave (unstable)

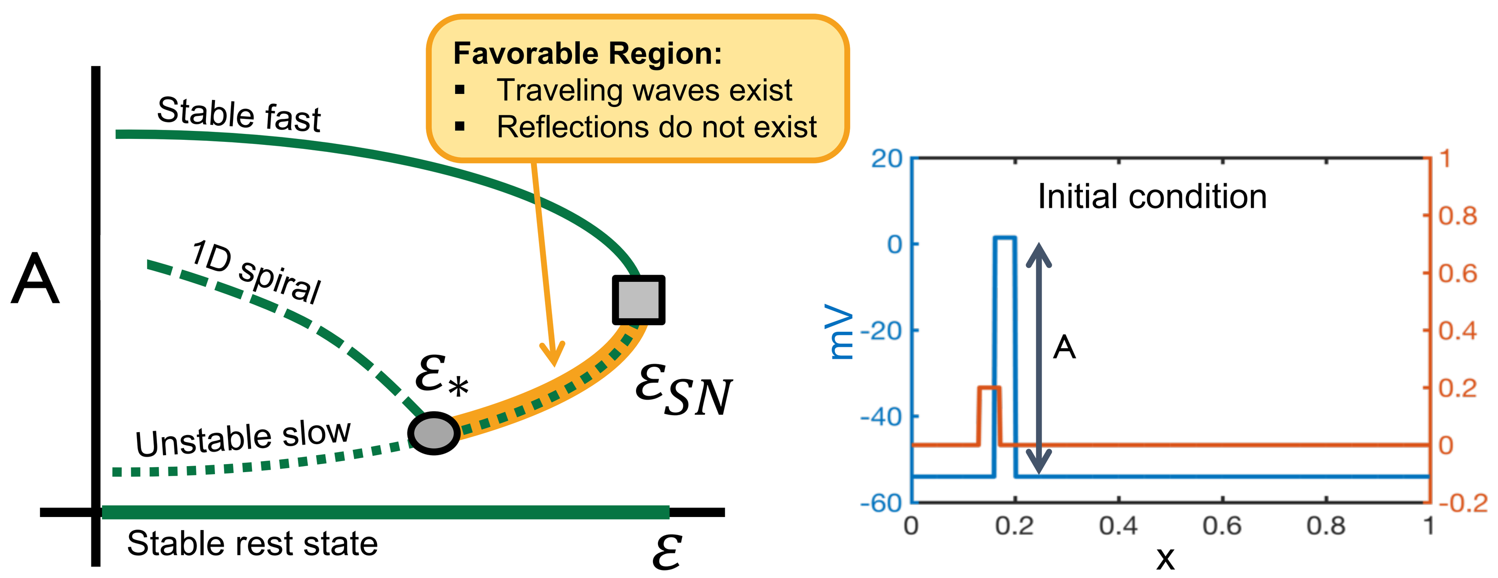


Figure 2. (left) Schematic of bifurcation diagram. (right) Initial conditions.

Ability for waves to reflect $\leftrightarrow$ Existence of 1D spiral

- Reflected waves associated with 1D spirals are thought to initiate **fatal cardiac arrhythmias**

Reseach Goals

The aim of this research is to investigate how biophysical processes influence the existence of the 1D spiral and thus contribute to the onset reflection mediated arrhythmia. Specifically,

- Determine how the "favorable region" depends on system parameters
- Understand the global bifurcation structure
- Identify how common drug therapies alter 1D spiral existence

One-Dimensional Spiral Wave as a Source Defect

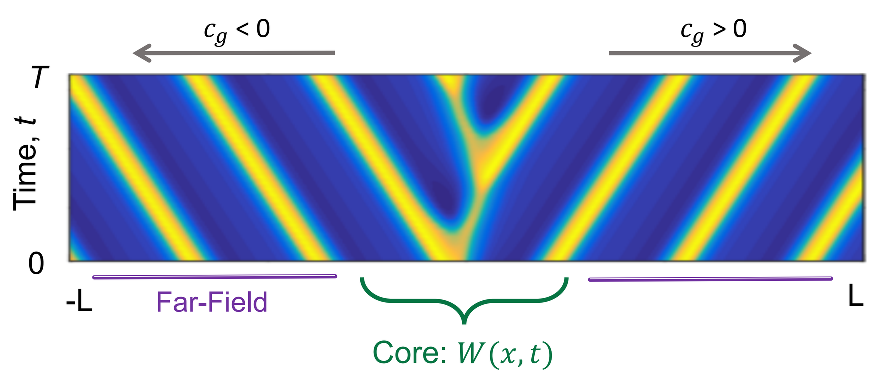


Figure 3. Diagram of antisymmetric source defect.

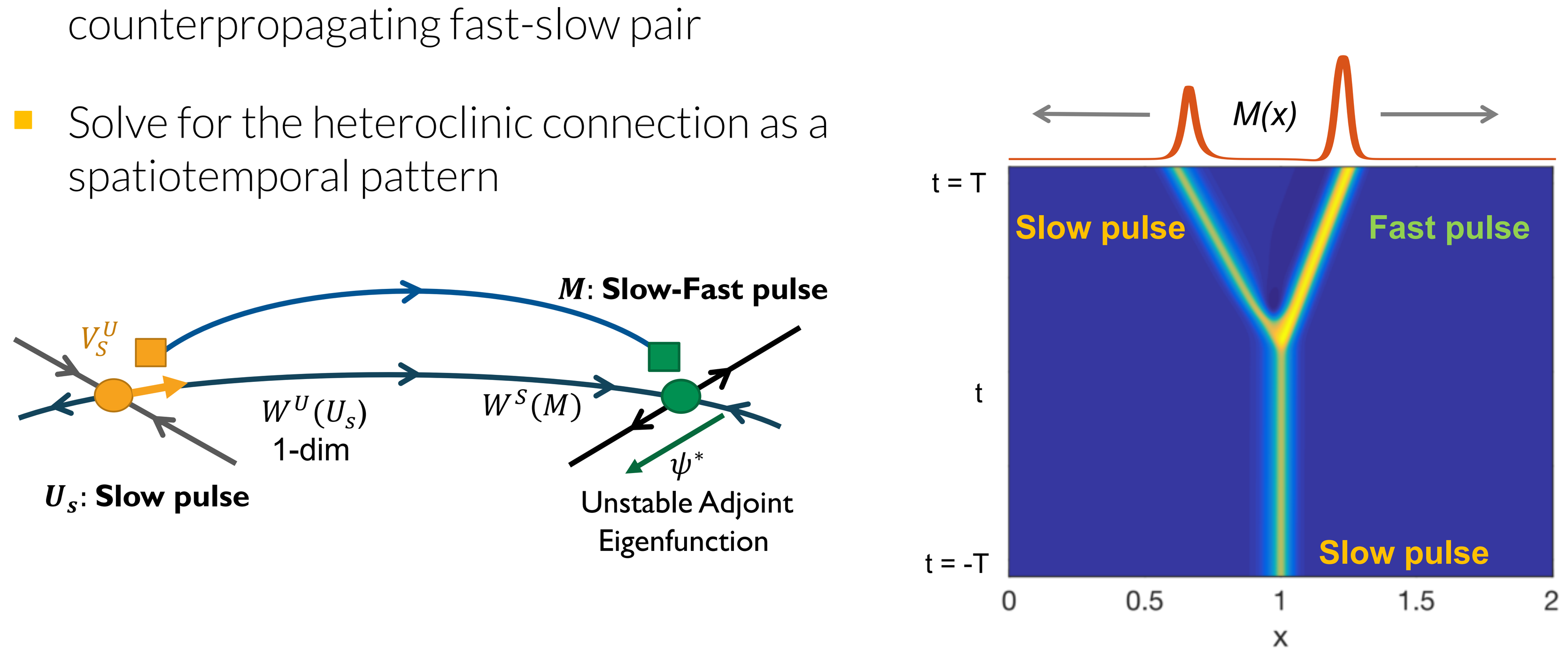
- 1D spiral wave is a T-periodic spatiotemporal **anti-symmetric source defect**
- Unstable core that alternately sheds stable periodic wave trains $U_\infty(\kappa x - \omega t)$ into the far-field

Numerical Computations

- Exploit core and far-field structure to **solve for source defect $\mathbf{U}_*(\mathbf{x},\mathbf{t})$** as solution to

$$\omega U_\tau = DU_{xx} + F(U), \;\; (x,\tau) \in [-L,L] \times \mathcal{S}^1$$
$$\text{with} \;\; U_*(x,t) = W(x,\omega t) + \chi(x) U_\infty(\kappa x - \omega t)$$

- **At bifurcation point $\epsilon = \epsilon_*$: Heteroclinic connection** forms between slow pulse and counterpropagating fast-slow pair
- Solve for the heteroclinic connection as a spatiotemporal pattern



Dependence of Bifurcations on System Parameters

- **Goal:** Find parameter range with a large **favorable region**: $\epsilon_* \ll \epsilon_{SN}$.

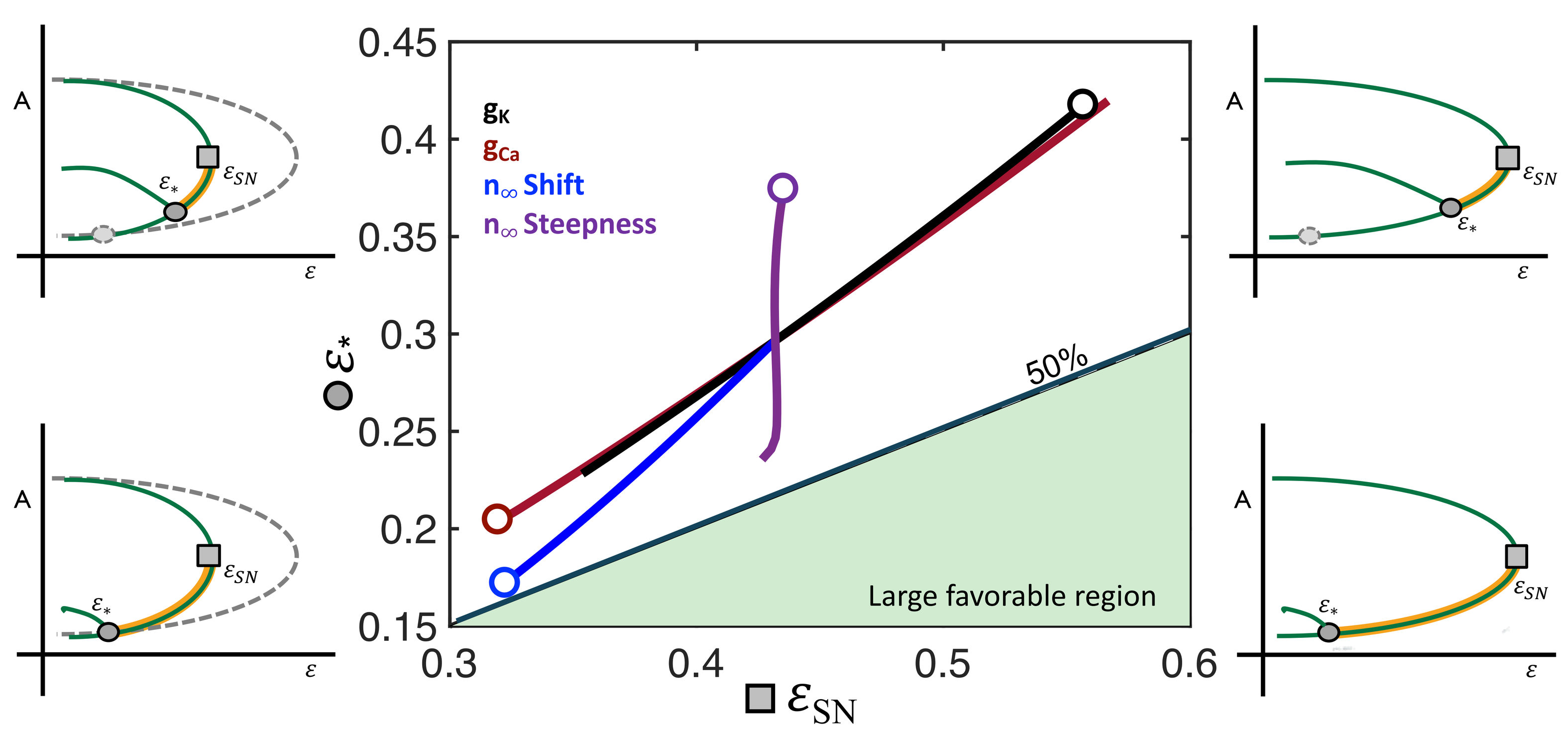


Figure 4. Central figure shows locations of ϵ_* and ϵ_{SN}, with circles indicating low parameter values. Green shaded area represents large favorable region, with $\epsilon_* < \frac{1}{2}\epsilon_{SN}$. Outside panels indicate relation of ϵ_* & ϵ_{SN}.

Proposed Heteroclinic Bifurcation Structure

- Source defect arises from rearrangement of $W^u(U_s)$
- $\dim W^u(U_s) = \dim W^u(U_*) = 1$

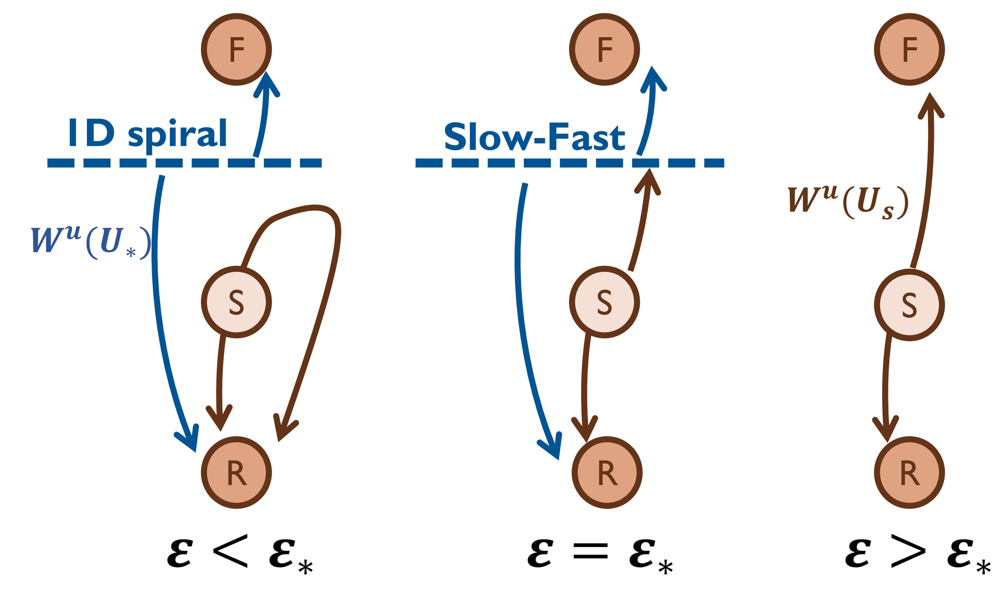


Figure 5. Schematic of predicted rearrangement of heteroclinic connections near $\epsilon = \epsilon_*$ on a periodic spatial domain.

Numerical Evidence of a Heteroclinic Bufurcation

- Period scaling consistent with heteroclinic bifurcation: $T \sim \log(\epsilon_* - \epsilon)$

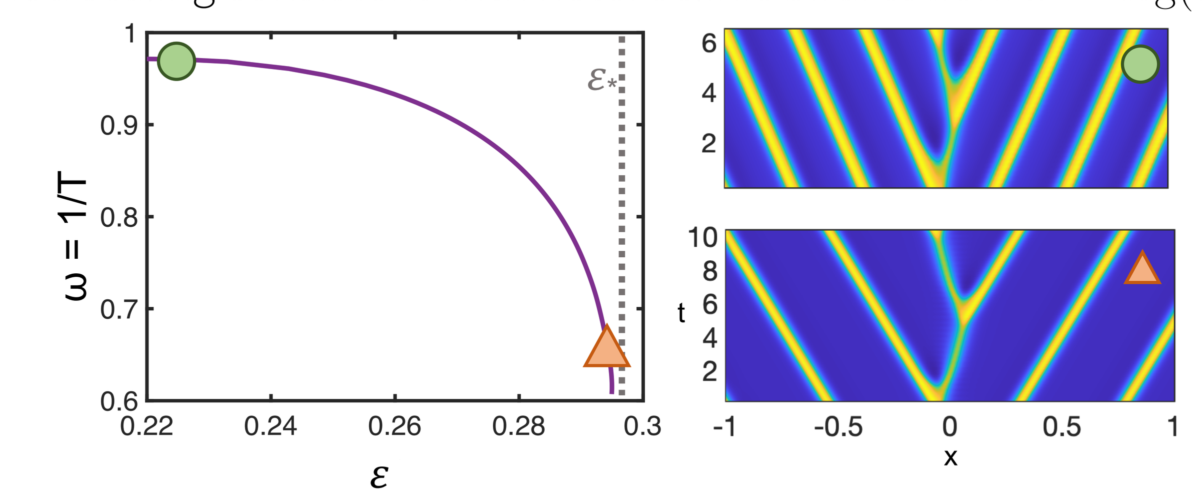


Figure 6. Temporal frequency approaching ϵ_*. Insets show spatiotemporal pattern at indicated point along the continuation curve.

Outcomes

- Additional evidence for global bifurcation: scaling of temporal period
- Preliminary understanding of dependence of ϵ_* and ϵ_{SN} on system parameters
- Numerical methods for computing source defect and bifurcation point

Next Steps

- Continue systematic study for favorable parameter regimes
- Extend results to biophysically realistic models of cardiac tissue
- Identify how drug therapies deter or promote reflections
- Confirm the structure of the global bifurcation

References

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