# Computation and Projection of Spiral Wave Trajectories During Atrial Fibrillation: A Computational Study

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**KEYWORDS**

• Cardiac electrophysiology • Atrial fibrillation • Body surface potential map • Phase mapping

**KEY POINTS**

- No strong relation was found between dominant frequency and phase singularity location for meandering rotors.
- Phases computed with the Hilbert transform method can produce spurious phase information if cells return to rest for extended periods between beats.
- Complexity of activity is increased on the convex hull of the atria.
- Loss of frequency content on the surface may limit the reconstruction of epicardial activity.

**Videos of spiral wave trajectories during atrial fibrillation accompany this article at http://www.cardiacep.theclinics.com/**

## INTRODUCTION

Phase analysis[1] has become a useful tool for the interpretation of fibrillatory activity.[2] It provides an amplitude-independent manner in which to characterize and visualize dynamic data, especially important for inverse mapping. This tool is helpful during fibrillation when activity is complicated because of wavefront interactions, leading to constant fractionation and collision. Beyond using phase statistics to simply classify arrhythmia, phase singularities (PSs) represent organizing centers and, thus, have become targets for ablation therapy.[3,4]

The concept that underlies phase is that the instant within the cell cycle can be characterized by an angle, which can be considered as time relative to the action potential onset normalized to the duration of the cellular action potential. It is convenient to take phase 0 as rest, just before the cell begins to depolarize, and the complete return to

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rest as occurring at a phase angle of $2\pi$. Several methods have been proposed to calculate phase, including time embedding[1] and the Hilbert transform.[5] Furthermore, the center of a functional reentry is identified by its core, which is a point of PS, meaning the phase at the point is itself undefined, but all phases are found encircling the point. These PSs may be located by spatial analysis of phase maps but may also be determined directly by the intersection of isolines.[6] However, which of the methods performs best for atrial fibrillation (AF) has yet to be resolved.

The relationship between activity on the atrial epicardium and activity recorded on the torso body remains to be fully elucidated. A recent study[7] has found, using simplified geometries for the atria and torso, that the number of PSs detected on the body surface potential map is reduced compared with the number found on the atrial surface. This finding could present a theoretic limitation of inverse mapping,[8] because filtering by the body could limit accurate reconstruction of potentials on the epicardium, and, hence, PSs. The study also found that when filtered at the correct frequency, the location of the dominant frequency (DF) on the torso is related to the position of the DF on the atria.

This is a computational study with 2 aims. The first is to compare the number and position of PSs as determined by 3 methods: time embedding, filament detection, and the Hilbert transform. The second aim is to see if a relationship exists between DF and PSs on different surfaces, starting from the epicardium and expanding to the torso, to understand how phase information is affected by volume conduction in a geometrically realistic situation.

## METHODS

### *Spiral Waves in Two Dimensions*

To study phase computation in a simpler geometry, spiral waves were induced in a 2-cm $\times$ 2-cm sheet discretized at 100 $\mu$m into a triangular finite element mesh. Simulations were performed with the CARP software[9] using a monodomain formulation with zero flux boundary condition using the Courtemanche human atrial cell model.[10] Cell properties inside a circular domain of radius 5 mm in the center of slab were changed to double intrinsic action potential duration (APD) (by increasing the background calcium current 5-fold and halving the rapid potassium current) to attract the phase singularity. Tissue conductivity was isotropic and set to 0.236 s/m everywhere (conduction velocity about 0.2 m/s). Reentry was initiated with a typical cross-field S1S2 protocol, as shown in Fig. 1. Simulations were run for 400 milliseconds to generate 1 spiral wave following a trajectory on the edge of circular domain starting at time 250 milliseconds. Results were output every 1 millisecond. Fig. 1 shows the schematic and approximate spiral wave core trajectory.

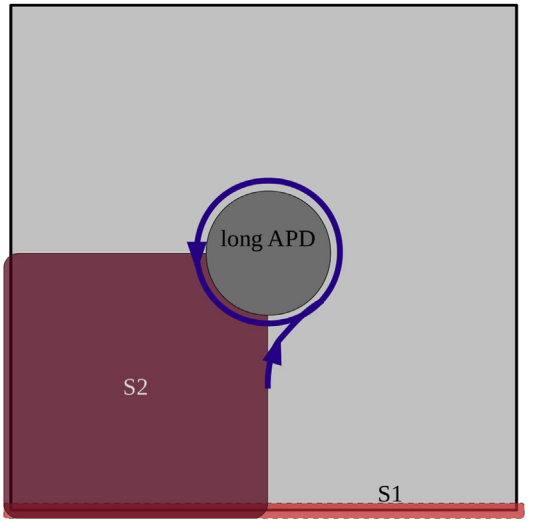


**Fig. 1.** The sheet model and approximate trajectory of the rotor core around the region with long APD (*dark gray*). The S1 stimulus along the bottom edge was applied at time 0 and S2 at 250 milliseconds.

### *Computer Model of Atria and Atrial Fibrillation*

A computed tomography (CT)-derived bilayer computer model of the atria was used, which incorporated structural and conductive elements, including left and right atria, discrete connection paths between the right and left atria, fiber orientation, pectinate muscles, and inflow vessels. A modified Courtemanche human atrial ionic model was used.[11] Tissue conductivities were set to closely match clinically measured values. Full details of the model are available in Ref.[12] Reentry was induced by an ectopic focus centered around the pulmonary vein delivered at 270 milliseconds after a normal beat originating from the sinoatrial node. The reentry, which developed, was sustained and persisted for the length of the simulation, 3.4 seconds. Output was sampled every 5 milliseconds.

To observe changes with distance from the epicardium, 10 surfaces were interpolated from the atria to the torso, on which phase maps were computed from surface potentials (Fig. 2). To begin, the convex hull of the atria (ie, the minimal elastic surface enclosing the atria) was generated using MeshLab (http://meshlab.sourceforge.net/).