IMAGE

Spiral wave breakup: Optical mapping in an explanted human heart shows the transition from ventricular tachycardia to ventricular fibrillation and self-termination

Ilija Uzelac, PhD, MBA,* Shahriar Iravanian, MD,† Neal K. Bhatia, MD,† Flavio H. Fenton, PhD*

*From the *School of Physics, Georgia Institute of Technology, Atlanta, Georgia, and †Division of Cardiology, Section of Electrophysiology, Emory University Hospital, Atlanta, Georgia.*

Since the first demonstration of ventricular fibrillation induction by electrical stimulation in 1850 by Hoffa and Ludwig,[1] many mechanisms[2,3] have been identified that initiate the complex dynamics of fibrillation. We report a high-resolution visualization of a spiral wave transitioning into multiple waves in human ventricles by using optical mapping (voltage and calcium) from the posterior epicardial surface ($\sim 7 \times 7$ cm^2) of an explanted heart from a 38-year-old female transplant recipient.

Online Supplemental Video 1 shows the transition from a relatively stable functional spiral wave of the 325-ms period lasting $\sim$50 rotations into multiple waves (fibrillation), which lasted $\sim$8 seconds (dominant period 250 ms) before self-termination. The video is slowed down during each transition.

While it is impossible to identify the mechanism acting without intramural information uniquely, the transition is consistent with the hypothesis of ventricular fibrillation induction by a twisted vortex filament[3,4] (Figure 1). Filaments

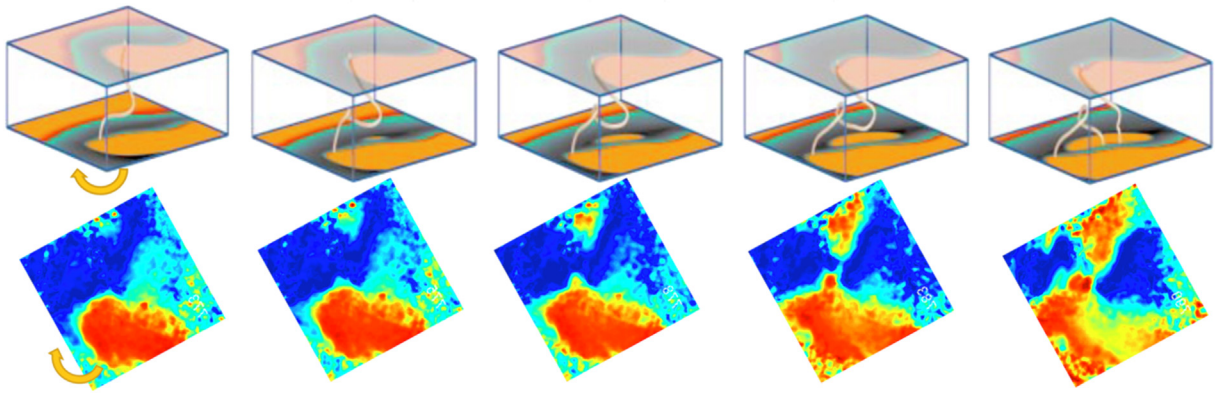

Figure 1 Top row, 3D simulation of a reentrant scroll wave on a slab of tissue with intramural fiber rotation. Top semitransparent surface is endocardium and bottom surface is epicardium. Bottom row, the spiral wave from the Online Supplemental Video 1 just before VF initiation.

KEYWORDS Spiral wave; Optical mapping; Ventricular fibrillation; Ventricular tachycardia; Human heart (Heart Rhythm 2022;19:1914–1915)

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are 3-dimensional extensions of spiral wave cores that can twist and bend.[3,4] Numerical simulations (Figure 1) show that the filament may twist because of intramural fiber rotation, producing an intramural activation that breaks through at the surface as a target pattern generating new spiral waves, resulting in fibrillation. Figure 1 (bottom panel) shows that the target pattern propagates faster than the rotation of the spiral wave, indicating that the target pattern is not produced by a point stimulus. This is consistent with the bending of a transmural vortex filament hypothesis but would require intramural recordings to confirm.

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Self-termination of fibrillation resulted from relatively large wavelengths occurring in this heart, which restricted the number of active waves in the domain. The critical mass required for fibrillation has been shown to depend not only on mass but also on dynamically changing wavelengths in human hearts.[5]

Appendix
Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2022.07.013.

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