Letter to the editor regarding: 'Mechanistic interpretations of sustained ventricular fibrillation: The role of mapping methodology, model and time'

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Article highlights

This letter illustrates how research is moving away from the traditional rigid debate between the 'multiple wavelet hypothesis' and the 'mother rotor framework.' Ventricular fibrillation (VF) is now known to be a dynamic, time-dependent process and not a static electrical event. This study introduces the concept of substrate-specific mechanistic phenotyping, hypothesizing that the underlying mechanisms that sustain VF may vary based on the specific cardiac tissue substrate. A major challenge remains the recognition of these complex VF phenotypes with standard clinical tools [e.g. surface electrocardiograms (ECGs)] during cardiac arrest and not by experimental 3D mapping. Future work should explore the use of surface ECG entropy metrics as accurate surrogates for 3D intramural dynamics during active cardiac arrest.

Dear Editor,

I read with great interest the recent comprehensive article by Tonko et al. regarding the dynamic and substrate-specific mechanisms that sustain ventricular fibrillation (VF).[1] The authors deserve credit for moving past the archaic dichotomy of the 'multiple wavelet hypothesis' versus the 'mother rotor framework,' rightfully framing VF as a duration-dependent, evolving process.

The authors propose an innovative and intellectually compelling concept of substrate-specific mechanistic phenotyping of VF. However, it remains unclear how clinically useful it is. In out-of-hospital cardiac arrest, treatment decisions must be made within minutes and are based primarily on surface electrocardiogram (ECG) interpretation and automated defibrillator algorithms. The article does not address how these proposed VF phenotypes could be recognized in real time using available clinical tools, as opposed to sophisticated 3D mapping technologies that remain largely experimental.[2] Hence, the translational pathway from mechanistic phenotyping to clinical implementation needs further elucidation.

The article concentrates on mechanistic insights from experimental models of VF but gives little attention to their external validity. Most of the available data are derived from healthy animal hearts; however, in clinical practice, sudden cardiac arrest occurs mostly in elderly patients with structural heart disease and multiple co-morbidities. Fibrosis, metabolic dysfunction and chronic cardiovascular remodeling may have a large impact on fibrillatory dynamics, and it remains unclear how the proposed mechanisms translate to real-world patient populations.[3] Further discussion of strategies to bridge this gap would strengthen the translational relevance of the article.

To implement these clinical realities into everyday care, future computational and experimental frameworks need to focus on how surface ECG entropy metrics can accurately surrogate these intramural 3D dynamics during cardiac arrest. ❑

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