Markers of self-organized criticality in atrial and ventricular fibrillation


Introduction: Atrial fibrillation (AF) and ventricular fibrillation (VF) are complex arrhythmias whose mechanisms remain elusive. We hypothesize that they may exhibit self-organized criticality (SOC), akin to processes observed in the brain. This could allow the heart to operate between stability and chaos, potentially enabling self-regulation of electrical activity, which may be crucial for the spontaneous termination of these arrhythmias.

Methods: Here, we aim to determine if AF and VF exhibit key features of SOC, specifically power law distributions in cluster sizes characterized by a critical exponent $\tau$. We analyzed AF and VF using the Aliev-Panfilov model (n=50 simulations), mouse AF (n=6), and human VF (n=12). For each episode, we created 2D binary maps to delineate refractory and non-refractory areas in the atria or ventricles. These enabled us to investigate the presence of power law distributions in refractory cluster sizes and calculate $\tau$ using Kolmogorov-Smirnov (KS) estimation and the log-likelihood ratio. To validate results, we compared $\tau$ values with those reported in established SOC systems.

Results: Distributions of refractory cluster sizes across model systems consistently conformed to a power law distribution, as evidenced by the Kolmogorov-Smirnov (KS) statistics for computer-simulated AF (median = 0.155, IQR = 0.021), mouse AF (median = 0.283, IQR = 0.037), and human VF (median = 0.145, IQR = 0.035), further supported by log-likelihood ratios (P<0.001 all cases). $\tau$ showed variability comparable to SOC in the brain (simulated AF: median = 1.464, IQR = 0.018; mouse AF: median = 1.155, IQR = 0.032; human VF: median = 1.310, IQR = 0.553).

Conclusions: AF and VF exhibit properties of SOC, with values of $\tau$ comparable to SOC in the brain. This suggests a potential mechanism in which the heart's electrical activity self-organizes to facilitate spontaneous AF/VF termination.