

Is Human Ventricular Fibrillation Different From Experimental Models?

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Introduction

There is a paucity of data on wavefront dynamics during human ventricular fibrillation (VF). Some experimental models in animal hearts show multiple small wavelets [1], whilst others implicate a single stationary rotor [2]. We have studied human VF using phase and wavefront analysis based on global epicardial mapping in order to compare and contrast human VF dynamics with animal models.

Methods

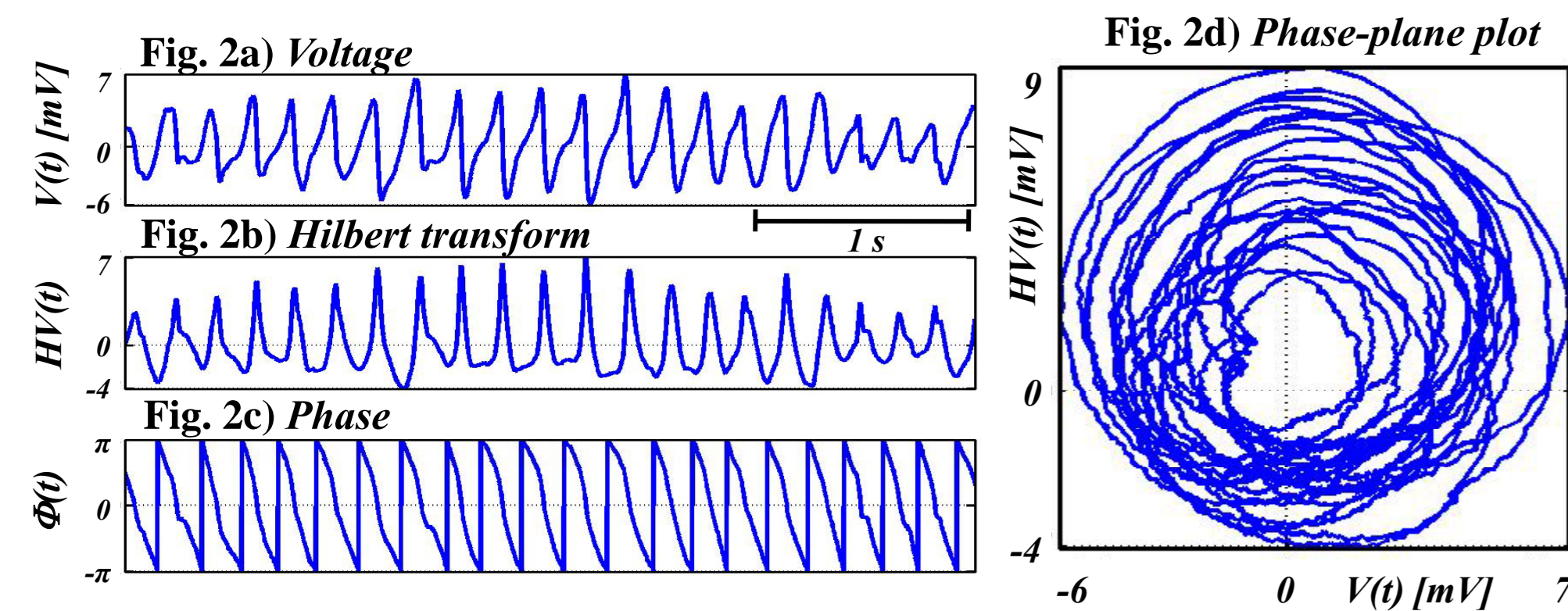
Experimental Procedure: VF was induced by burst pacing in 10 patients undergoing routine cardiac surgery. For each subject, a 20-40 s episode of VF activity was sampled at 1 kHz using an epicardial sock (see Fig. 1) containing 256 unipolar contact electrodes connected to a UnEmap system [3][4].



Fig. 1)

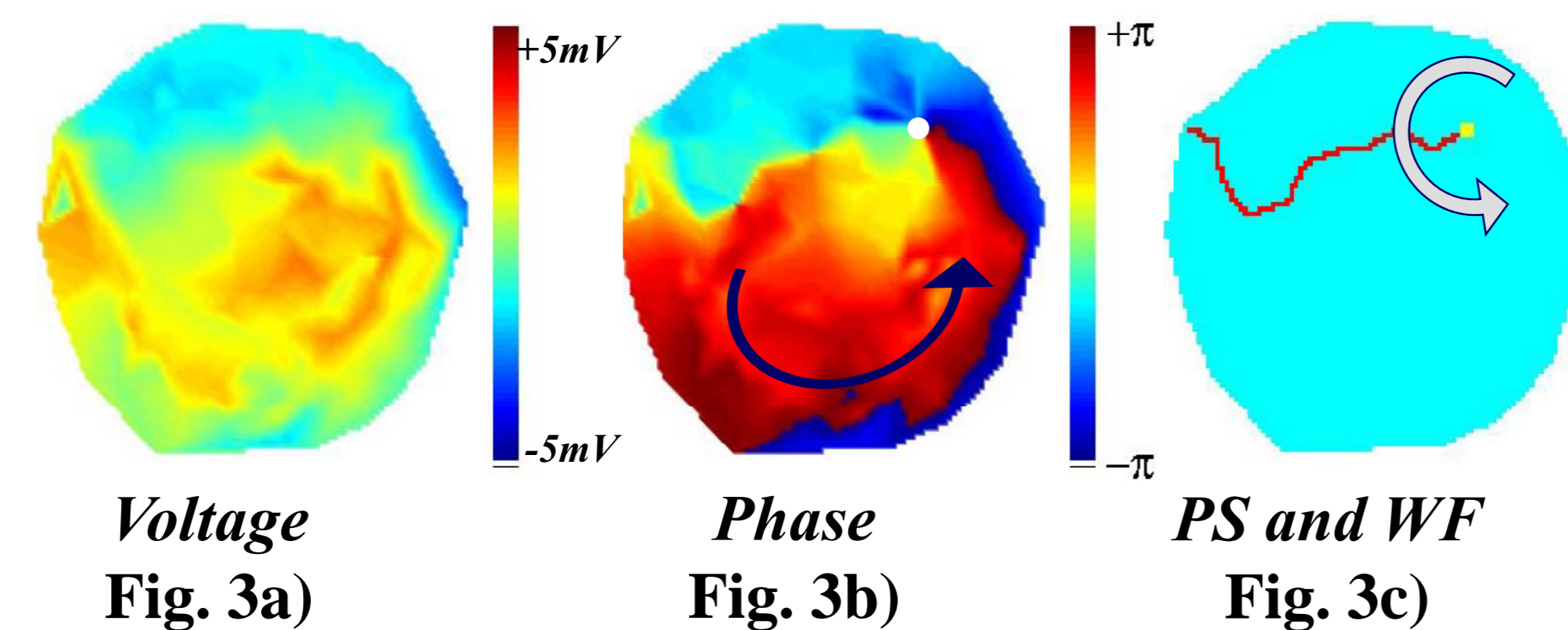
Activation times: were computed at the minimum negative slope of voltage [5]. We subsequently applied a signal de-trending algorithm [6] in order to set the voltages at the activation times to be zero.

Phase: was computed from the phase-plane plot (Fig. 2) using the Hilbert transform [7] for each de-trended signal, and phase maps were plotted using the 2D polar plot (Fig. 3b).



Phase singularities: (PS) are the tips of re-entrant waves on the epicardial surface (epicardial rotors). PS were identified using a method based on the topological charge [8]. Chirality is indicated by yellow dots (anticlockwise) and dark blue dots (clockwise) on the 2D polar maps (Fig. 3c).

Activation wavefronts: (WF) correspond to spatial isochrones of activation time. Due to signal de-trending, WF can equivalently be determined from the isolines of zero phase under the Hilbert transform. WF were identified using an active edge method [6] and illustrated as red lines on the 2D polar maps (Fig. 3c).



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References

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Results

Wavefronts are large

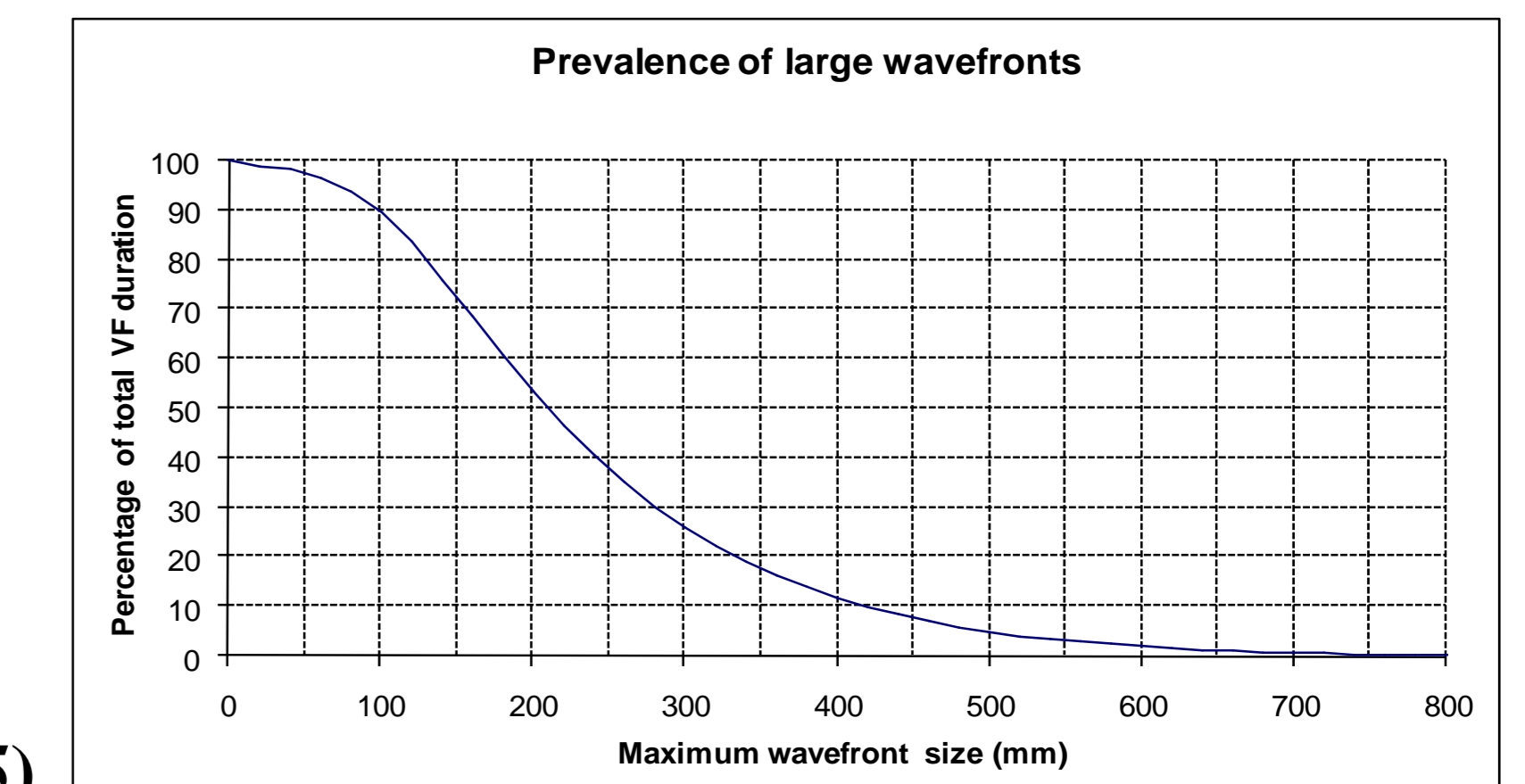
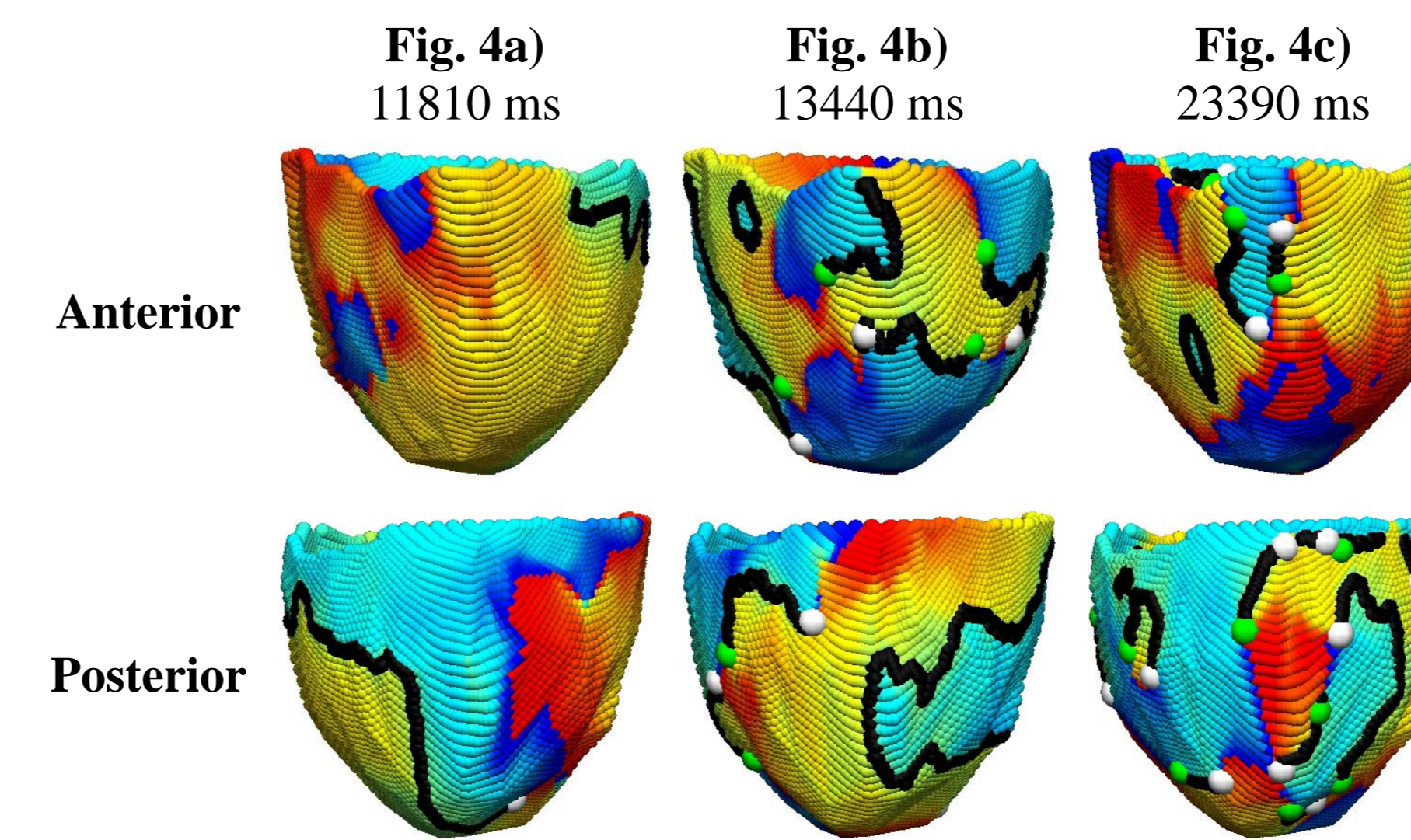


Fig. 5)

Wavefront morphology: The typical wavefront morphology observed during the course of VF was that of large wavefronts together with a range of smaller wavefronts. In a single patient we observed a range of wavefront sizes, from very large (Fig. 4a), to medium (Fig. 4b), to small (Fig. 4c).

Wavefront sizes: As illustrated in Fig. 5) one or more wavefronts of at least 100 mm in size were present for 90% of the VF duration. Large convoluted wavefronts were predominant with at least one wavefront of size > 200 mm present for over half of the VF duration.

Rotors are persistent

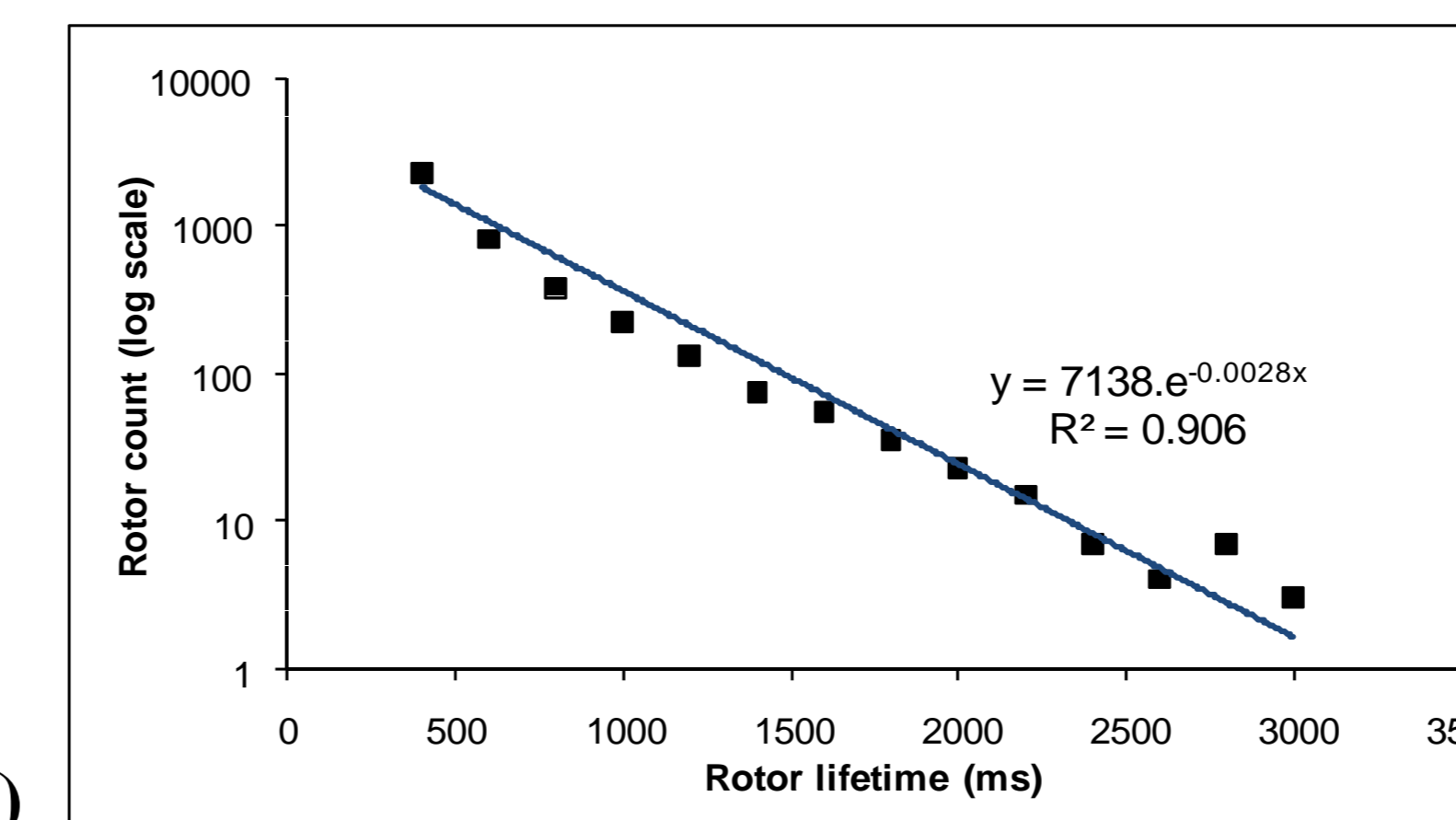


Fig. 6)

Rotor Lifetimes: As shown in Fig. 6) a number of epicardial rotors with moderate lifetimes were observed. The rotor lifetime exhibited an exponential decay. The rate of decay was 0.0028 ms^{-1} ($P < 0.001$) which corresponds to a mean lifetime of 356 ms.

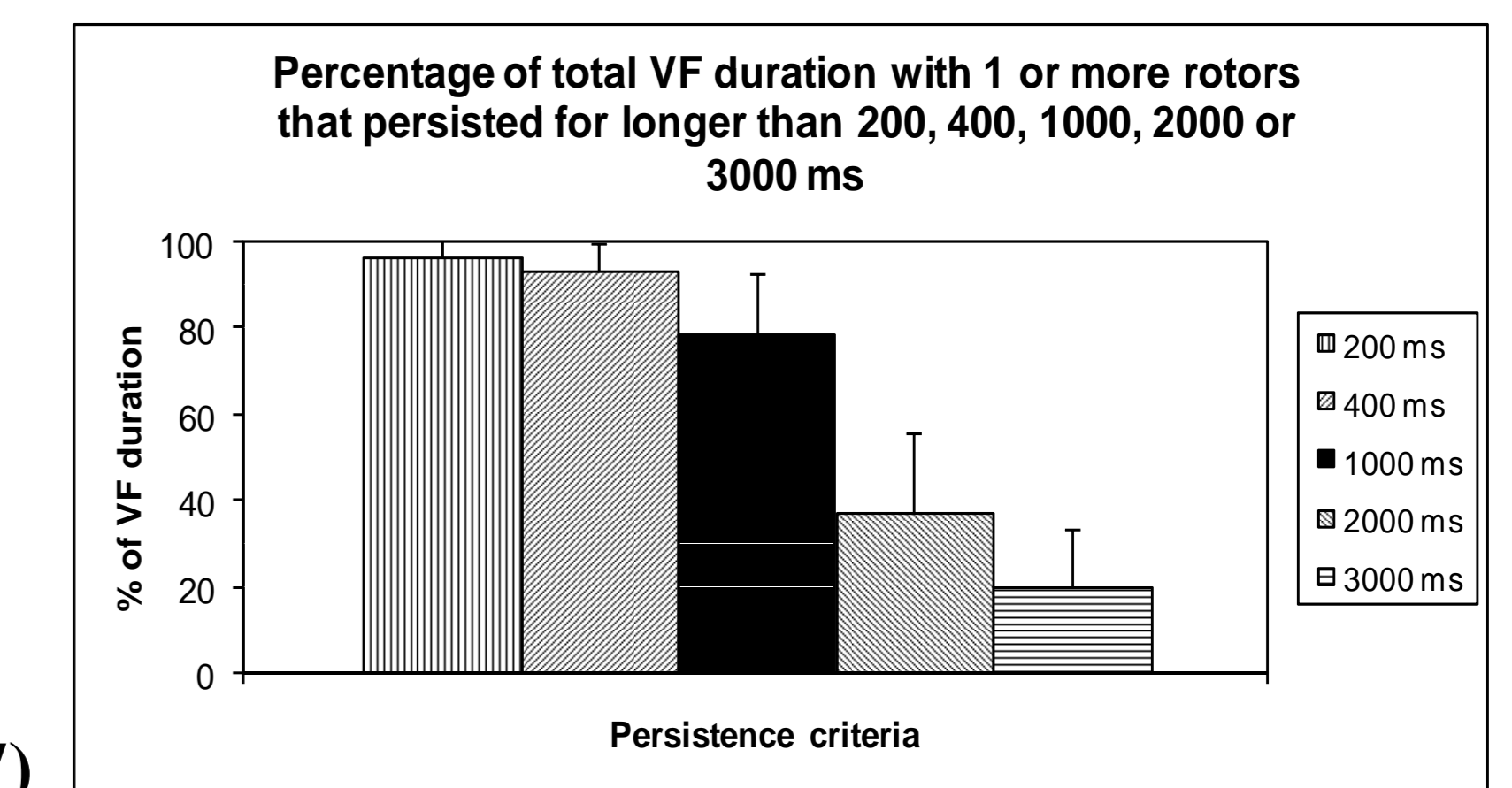


Fig. 7)

Rotor Persistence: Epicardial rotors with lifetimes greater than 400 ms were present for more than 90% of the total VF duration (Fig. 7). When the lifetime threshold was increased to 1000, 2000, and 3000 ms, at least one persistent rotor was present for more than 78%, 34% and 20%, respectively, of the total VF duration.

Rotors coexist

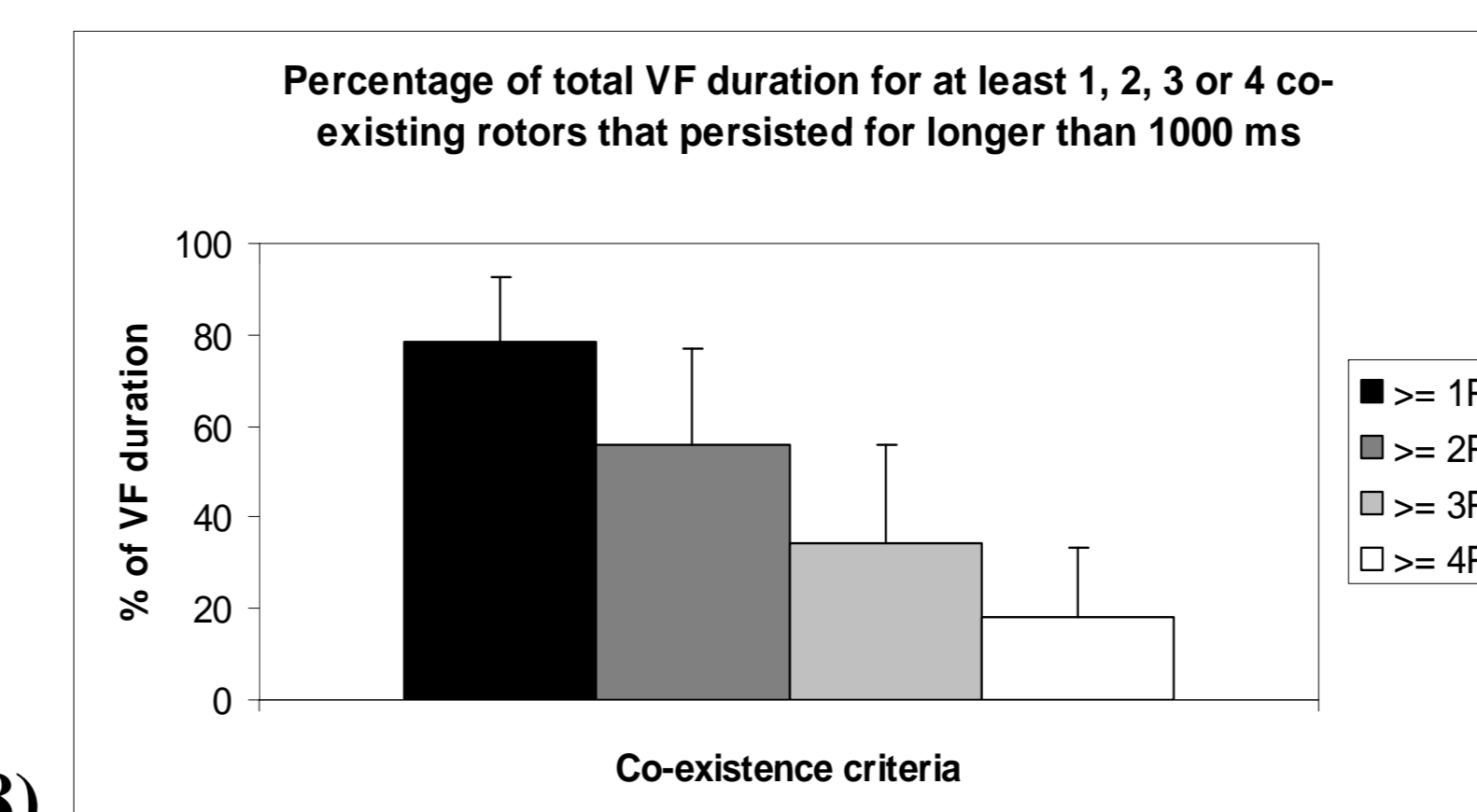


Fig. 8)

Rotor coexistence: When defining persistent rotors as those with lifetimes greater than 1000 ms (approximately 5 rotations), we found that 2 or more persistent rotors were present for 55% of the total VF duration, and 3 or more persistent rotors were present for 32% of the time (Fig. 8).

Conclusions

Epicardial mapping in humans suggests that neither multiple wavelets, as seen in some animal models, nor a single persistent stationary rotor, as seen in other animal models, drive VF. Instead, in all subjects the predominant characteristic was multiple rotors of moderate duration, which generated large make and break wavefronts. These results suggest that human VF is different from animal VF because we found that human VF is sustained by a small number of persistent rotors.