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Background: Complex fractionated atrial electrograms (CFAEs) have long been associated with proarrhythmic alterations in atrial structure or electrophysiology. Structural alterations disrupt and slow smoothly propagating wavefronts, leading to wavebreaks and electrogram (EGM) fractionation, but the exact nature and characteristics for arrhythmia remain unknown. Clinically, in AF patients, increases in frequency, whether by pacing or fibrillation, increase EGM fractionation and duration, and reentry can occur in relation with the conduction disturbance. Recently, percolation has been proposed as an arrhythmogenic mechanism but its role in AF has not been investigated.

Objective: We sought to determine if percolation can explain reentry formation and EGM behavior observed in AF patients.

Methods: Computer models of fibrotic tissue with different densities were generated based on late Gadolineum enhanced MRI images, using pixel intensity as a fibrosis probability to avoid an arbitrary binary threshold. Clinical pacing protocols were followed to induce AF, and EGMs were computed.

Results: Reentry could be elicited, with a biphasic behavior dependent on fibrotic density. CFAEs were recorded above fibrotic regions, and consistent with clinical data, EGM duration and fractionation increased with more rapid pacing.

Conclusion: These findings confirm percolation as a potential mechanism to explain AF in humans and give new insights into dynamics underlying conduction distortions and