KATP channel opening accelerates and stabilizes rotors in a swine heart model of ventricular fibrillation


**Aims**
The mechanisms underlying ventricular fibrillation (VF) are still disputed. Recent studies have highlighted the role of KATP-channels. We hypothesized that, under certain conditions, VF can be driven by stable and epicardially detectable rotors in large hearts. To test our hypothesis, we used a swine model of accelerated VF by opening KATP-channels with cromakalim.

**Methods and results**
Optical mapping, spectral analysis, and phase singularity tracking were performed in eight perfused swine hearts during VF. Pseudo-bipolar electrograms were computed. KATP-channel opening almost doubled the maximum dominant frequency (14.3±2.2 vs. 26.5±2.8 Hz, P<0.001) and increased the maximum regularity index (0.82±0.05 vs. 0.94±0.04, P<0.001), the density of rotors (2.0±1.4 vs. 16.0±7.0 rotors/cm²×s, P<0.001), and their maximum lifespans (medians: 368 vs. ≥3410 ms, P<0.001). Persistent rotors (≥1 movie ¼ 3410 ms) were found in all hearts after cromakalim (mostly coinciding with the fastest and highest organized areas), but they were not epicardially visible at baseline VF. A ‘beat phenomenon’ ruled by inter-
domain frequency gradients was observed in all hearts after cromakalim. Acceleration of VF did not reveal any significant regional preponderance. Complex fractionated electrograms were not found in areas near persistent rotors.

**Conclusion**

Upon KATP-channel opening, VF consisted of rapid and highly organized domains mainly due to stationary rotors, surrounded by poorly organized areas. A ‘beat phenomenon’ due to the quasi-periodic onset of drifting rotors was observed. These findings demonstrate the feasibility of AF driven by stable rotors in hearts whose size is similar to the human heart. Our model also showed that complex fractionation does not seem to localize stationary rotors.