

# Relation of surface T-wave to vulnerability to ventricular fibrillation in explanted structurally normal hearts

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**Introduction:** Repolarization dispersion can favor the onset of ventricular fibrillation (VF), but identifying vulnerable substrates on the body surface is challenging. We investigated if susceptibility to VF can be assessed from surface T-wave in an *ex-vivo* model of purely electrical repolarization dispersion.

**Methods:** Langendorff-perfused pig hearts (N=7) were suspended in a human-shaped 256-electrode torso tank. Tank potentials were recorded in sinus rhythm with/without repolarization gradients created by localized perfusion of dofetilide and/or pinacidil. VF inducibility was tested at each drug state by an S1 (right atrium) S2 (anterior left or right ventricle) protocol and quantified by the vulnerability window (VW), i.e. the total interval within which S2 ventricular extra-beats triggered VF. Susceptibility to VF was quantified on T-waves by markers of duration (the peak-to-end interval,  $T_{PEAK}-T_{END}$ ), and shape, that is, symmetry (the ratio of the areas under T-wave final and initial portions, with respect to the peak,  $Asy$ ), and flatness (measured by kurtosis,  $Kurt$ ). We fitted a linear mixed-effect model (LMM) to link T-wave markers (fixed effects) with VW (response), and with random effects on drug states and hearts (significant if  $p < 0.05$ ).

**Results:** VF was induced in 15/23 drug states from at least one ventricle. In vulnerable substrates (higher VW), T-waves were longer ( $T_{PEAK}-T_{END}$ ,  $p < 0.001$ ), less symmetric ( $Asy$ ,  $p < 0.0001$ ) and moderately flat ( $Kurt$ ,  $p = 0.01$ ) than in non-inducible cases (Figure). In a combined LMM model, significant effects were only explained by  $Asy$  ( $p < 0.0001$ ) and  $Kurt$  ( $p < 0.05$ ).

**Conclusions:** Vulnerability to VF due to abnormal repolarization can be assessed by surface T-wave analysis, and potentially applied to human VF.

