Letter to the Editor

T Wave Alternans Detected in Defibrillator Electrograms

To the Editor:

I read with great interest the contribution by Kim et al¹ regarding the prognostic value of T wave alternans (TWA), evaluated from stored cardiac electrograms, retrievable from implantable cardioverter defibrillators (ICD). What is of interest is that the amplitude of TWA increased more prior to the onset of spontaneous ventricular tachycardia (VT) than immediately after induced VT or inappropriate ICD shocks. Also, the amplitude of TWA in μV preceding spontaneous VT was larger in patients with ischemic rather than non-ischemic cardiomyopathy or in patients with idiopathic ventricular fibrillation. Furthermore, in patients with ischemic cardiomyopathy, the magnitude of TWA was larger before spontaneous VT than before induced VT. The authors felt that this behavior of TWA suggested a role for this electrophysiological phenomenon in the induction of spontaneous VT. Although this is possible, the increase in the amplitude of TWA prior to spontaneous VT may be merely an index, heralding the emergence of this catastrophe, but not necessarily the main or partial cause of the induction of VT. In the spirit of symmetry, I am curious about the amplitude of TWA immediately after the appropriate ICD shocks in the patients with spontaneous VT. The authors detected differences in the magnitude of TWA in the same patients when TWA was measured at different times. Recently, it has been theorized that the magnitude of TWA is T-wave amplitude dependent². Clinical experience from serial standard ECGs, ECG tracings during the course of exercise stress testing,

and in ambulatory Holter ECG recordings attests to the volatility of the T-wave in morphology, amplitude and polarity in both normal subjects and patients with a variety of pathologies. Although many of these T-wave alterations may be explainable, many more remain puzzling as to their genesis. One wonders about the relationship of the magnitude of the TWA in μV and the amplitude of the corresponding T-waves in the different subgroups that the authors reported on. This is reduced to the notion that the difference in the magnitude of the TWA that the authors noted was due to the differing amplitude of the corresponding T-waves, and not to a difference in vulnerability for VT. Also, an opportunity is afforded by the authors' material in the subgroup of patients who had measurements of TWA at different times with different TWA amplitude values; could this be traced to the different amplitudes of the corresponding T-waves? I will appreciate very much the response of the authors to my questions.

References

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2. Madias JE. The need for studies to evaluate the reproducibility of the T-wave alternans (TWA), and the rationale for a correction index of the TWA. *Indian Pacing Electrophysiol J* 2007; **7**: 176–183.

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