Effect of acute volume overload on the magnitude of T-wave alternans

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To the Editor: I read with great interest the contribution by Narayan et al. (1) on the effect of acute volume loading on the magnitude of the T-wave alternans (TWA) in five dogs without structural heart disease. The authors showed that the acute saline infusion led to a rise of pulmonary capillary wedge pressure and an increase of the magnitude of TWA in comparison with the value of TWA found prior to the acute volume infusion. Importantly, the increased magnitude of the TWA after the volume occurred at a lower atrial paced rate than the one noted at baseline. In addition, there was a linear relationship between the volume loading and the increased magnitude of TWA. Although these findings were noted in normal anesthetized dogs undergoing fluid loading, there may be clinical implications for patients with structural heart disease and associated fluid overload. The findings are also of interest in the light of the current climate that the magnitude of TWA and the heart rate at its onset matter and suggest a higher arrhythmic risk than the one associated with TWA of lower magnitude, elicited at higher heart rates. However, one wonders whether the increase in the TWA magnitude following the fluid overload was of primary nature (indicative of a more advanced electrophysiological destabilization), or secondary, due to changes in the overall T-wave magnitude/morphology/axis consequent to the volume overload and resultant heart enlargement. No data on heart volumes were acquired, but the magnitude of the administered fluid infusions would be expected to produce some heart dilation (indeed the authors refer repeatedly to a plausibly induced “stretch,” and its repolarization and arrhythmic consequences), and such increases in end-diastolic volumes elicit the “Brodie effect” (2), characterized electrocardiographically by an augmentation of the QRS complexes; also changes in the corresponding T-waves (secondary) may be noted. Were changes noted after volume infusion in the ECG QRS and/or T-waves, both before and during atrial pacing? Was the increase in magnitude of TWA after fluid infusion totally or partially secondary to the changes in the amplitude (or other attributes) of the T-waves, and could a “crude” adjustment (notwithstanding the complexity of the spectral TWA method) of the increased TWA magnitude values by possibly increased T-wave amplitude render the magnitude of the TWA after the fluid loading less impressively elevated?

REFERENCES


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