John J.V. McMurray
University of Glasgow
Cardiology Division
126 University Place
Glasgow G12 8TA
UK

doi:10.1093/eurheartj/ehp166

Prediction of fatal or near fatal arrhythmias in patients with a depressed left ventricular function after an acute myocardial infarction

The ambitious study by Huikuri et al.¹ for the CHARISMA study group deserves our attention, careful reading, and contemplation about our current practices in predicting malignant arrhythmias in patients with an acute myocardial infarction and depressed left ventricular function, and what we should go from here. Methodologies employed in the study of 312 patients included standard electrocardiogram, heart rate variability/turbulence, ambient arrhythmias, signal-averaged electrocardiogram, T-wave alternans (TWAs), and programmed electrical stimulation (EPS). Ventricular fibrillation or symptomatic sustained ventricular tachycardia, the primary endpoint, was documented by implantable ECG loop-recorder. During a follow-up of 2 years, measurement of heart rate variability was shown to be an independent non-invasive predictor of outcomes with the best performance. T-wave alternan was assessed by exercise stress testing, and for patients with incomplete or indeterminate tests, during the EPS by atrial and atrial/ventricular pacing. While many investigators have recommended the employment of quantitative results of TWA,²,³ the authors of this report have not reported actual values of TWA, but resorted to the currently prevailing dichotomy of positive/negative/intermediate and dichotomy of negative/intermediate/NST study results. In the context of previously expressed opinions,¹,²,³ it would be useful to have information about the contribution of quantitation of TWA results. Accordingly, was the magnitude of TWA a better predictor of the arrhythmic outcomes than mere characterization of negative/intermediate/NST results? What were the correlates (i.e. other test results) of patients with relatively high values of TWA? Another notion recently expressed⁴ is whether the magnitude of the TWA is T-wave amplitude dependent. In this vein, was there any relationship between the magnitude of the TWA values and the corresponding T-wave lead(s) used for the TWA calculations? The authors have employed all the 12 standard ECG leads, and X, Y, Z orthogonal leads for the TWA assessment, and thus an opportunity exists to evaluate whether such a relationship underlies TWA. Although the spectral assessment of TWA does not reflect only the amplitude of the corresponding T-wave, some insight on the above issue could be forthcoming from a correlation of the values of TWA magnitude in µV in the 15 leads used and the values of the amplitude of the corresponding T-waves in µV or mV. A response to this inquiry may provide an insight whether a different TWA magnitude in a repeat assessment in a particular patient is a reflection of changing of the degree of vulnerability to malignant arrhythmias, or merely the often unexplained change in the morphology, amplitude, or polarity of T-waves.

References

4. Madas 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.

John E. Madas
Mount Sinai School of Medicine
New York University
New York, NY 10029-6500
USA

Division of Cardiology
Elmhurst Hospital Center
79-01 Broadway
Elmhurst, NY 11373
USA
Tel: +1 718 3345005
Fax: +1 718 334 5990
E-mail: madasj@nychhc.org

Letters to the Editor

long-term experience from which safety can be inferred. While it is theoretically possible to identify patients who might be given a relatively selective beta-1 receptor antagonist, e.g. non-smokers with mild–moderate airways disease, no previous hospitalization, only Stage 1 or 2 treatment, a stable peak expiratory flow rate, no nocturnal symptoms, little wheeze, and the sense to know when to seek help, such advice is based upon clinical assumptions and not on evidence. What might happen to patients who develop an exacerbation of asthma while taking a beta-blocker? Additionally, the patients’ response to rescue beta-agonist therapy during that attack remains a concern. While in time recommendations may change, we believe that, in present, it is wise to recommend in a general guideline such as ours that relatively beta-1 selective beta-blockers be given to patients with asthma in the absence of convincing evidence of safety.

Although guidelines are evidence based, anecdotal experience frequently generates strong hypotheses. We would encourage Drs Shaw and Williams to address their hypothesis in a prospective, randomized trial with appropriate endpoints. We agree that the role of selective beta-blockade in patients with asthma and heart failure deserves more rigorous attention.

Albert Einstein put it well ‘In theory, theory and practice are the same. In practice they are different’...