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DISCUSSION FORUM

Identifying coronary artery disease patients at risk for sudden and/or arrhythmic death: remaining limitations of the electrocardiogram

Babken Asatryan , Laurent Roten, and Tobias Reichlin *

Department of Cardiology, Inselspital, Bern University Hospital, University of Bern, Freiburgstrasse 18, 3010 Bern, Switzerland

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This commentary refers to 'Simple electrocardiographic measures improve sudden arrhythmic death prediction in coronary disease', by N. A. Chatterjee *et al.*, pages 1988–1999.

The important study concerning the easy-to-use electrocardiogram (ECG) markers of sudden and/or arrhythmic death (SAD) in coronary artery disease (CAD) patients as presented by Chatterjee *et al.*[1] is a weighty contribution to an intricate and still rather puzzling topic. They implicate that in CAD patients, contiguous Q waves, left ventricular hypertrophy (LVH), QRS duration, and JTc prolongation provide incremental predictive value beyond traditional risk factors for SAD. While this study is unique in providing easily measurable markers that improve SAD risk stratification, it also reveals that these ECG markers do not identify the *entire* CAD population at risk for SAD. In particular, here proposed parameters correctly reclassified only one-third of patients at risk for SAD. Potential reasons for imperfect prediction are manifold: firstly, current cut-offs may not be optimal to detect sudden cardiac death (SCD) risk; second, single resting ECG may not allow to correct for potential confounders; third, there are several arrhythmia mechanisms underlying SCD in CAD patients, and the proposed ECG markers can only capture some of them.

With regard to the third point, all of the proposed ECG markers to some extent have been associated with myocardial scar/fibrosis. Q waves are a reflection of myocardial scar/fibrosis, and fibrosis is an essential component of hypertrophy. As to JTc, there is limited data published, but QTc has been found to predict cardiovascular mortality in subjects with CAD, diabetes mellitus, and hypertensive LVH.[2] A recent paper showed that clinically unrecognized myocardial scar is associated with QTc prolongation in patients with no history of CAD, indicating QTc alteration even with clinically negligible structural substrate.[3] Additionally, QRS duration has been shown to correlate with myocardial fibrosis in an early study.[4] It is therefore plausible that both components of QTc interval—QRS duration and JTc—carry the positive correlation of QTc with presence/extent of myocardial fibrosis.

The extent of late gadolinium enhancement-defined scar/fibrosis correlates closely with arrhythmic substrate and independently predicts adverse cardiovascular outcomes post-myocardial infarction. The proposed ECG markers therefore likely identify patients with myocardial fibrosis at risk for scar-related reentrant ventricular tachycardia. Conversely, non-reentrant ventricular arrhythmias due to triggered activity or enhanced automaticity, although insufficiently investigated, are not uncommon in CAD patients,[5] and may underlie SAD in some cases. Furthermore, ventricular fibrillation due to recurrent acute myocardial ischaemia is a relevant cause of SAD in CAD patients. These latter mechanisms may be more difficult to predict with the ECG and may in part explain the limitations of the proposed ECG score.

While identifying SCD mechanisms in large populations is challenging, reviewing the initiation and termination mechanisms and morphology of ventricular arrhythmias in implantable cardioverter defibrillator (ICD) recipients may provide some insights. Since non-reentrant ventricular arrhythmias may be largely responsible for arrhythmic deaths among patients determined to be at lower risk and thus not be implanted with an ICD, a prospective study with implantable cardiac monitors may ascertain whether risk groups for mechanistically different arrhythmias exist.

We commend the authors for their valuable contribution to the unceasing fight against SAD/SCD and invite their thoughts on our perspective.

Conflict of interest: none declared.

*Corresponding author. Tel: +41 31 664 00 50, Fax: +41 31 632 42 11, Email: tobias.reichlin@insel.ch

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