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Ventricular Repolarization in Hypertension: Beyond Bazett

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Nowadays, even though elaborate and sophisticated imaging facilities are used for assessing heart/circulation morphology and functioning, the electrocardiograph still remains the first-step approach in evaluating the patient. Being widely recognized for its diagnostic utility, the electrocardiograph remains indispensable for the prognostic stratification of cardiovascular patients, because of its low cost and noninvasiveness in investigating cardiac electric activity. In fact, electrocardiographic measurements of ventricular repolarization have shown that various congenital and acquired clinical conditions are associated with a higher risk of life-threatening arrhythmias and sudden death.

The time required for all ventricular depolarization and repolarization processes is reflected by the duration of the QT interval on the surface electrocardiograph. Among the many physiological and pathological factors that contribute to the length of the QT interval, heart rate plays a major role. The QT interval can be corrected through several approaches. The simplest is Bazett's formula (i.e., $QTc = QT/RR^{1/2}$) which overcorrects at fast heart rates and undercorrects at low heart rates.¹ In addition to QT duration, other indices associated with ventricular repolarization have been proposed. These measure QT dispersion, taking into account the fact that QT length is lead dependent.²

Systemic arterial hypertension, particularly if associated with left ventricular hypertrophy, increases the risk of ventricular arrhythmias.³ Moreover, hypertension is frequently associated with sympatho-vagal imbalance, which may lead to QT prolongation and trigger ventricular arrhythmias,⁴ and/or with left ventricular hypertrophy, which is an independent risk factor for sudden death.⁵

Nevertheless, QT duration and dispersion formulas have many technical and pathophysiological limitations. On this basis, in this issue of the Journal, Salles *et al.*⁶ assess two relatively novel indices of ventricular repolarization, spatial T-wave axis and T-wave peak-end interval duration, in patients who are affected by hypertension that is resistant to multidrug treatment, and have a significant prevalence of coronary artery

disease, diabetes, and obesity. The authors observed that the T-wave axis deviation alone was independently associated with left ventricular hypertrophy, even when adjusted for the presence of QT prolongation. They therefore suggest that this index could be a surrogate prognostic marker. In actual experience, studies in unselected populations challenge its predictive value,⁷ whereas it has been claimed to be relevant in healthy elderly people.⁸

Since it was first proposed in 1920, much evidence has been collected concerning the clinical usefulness of Bazett's formula. Nowadays it is incorporated in automatic digital ECG analysis as a friendly companion for the cardiologist. As for the spatial T-wave axis computation, there are concerns that its value is likely to be affected by stimuli that are constitutional (e.g., long-QT syndromes), physiological (e.g., insulin secretion and resistance, thyroid function, salt-water balance, wake-sleep cycle), pathological (e.g., altered systolic and diastolic left ventricular function, renal insufficiency, etc.), or pharmacological (e.g., drugs known to exert an adverse effect on ventricular repolarization). Further studies are needed to gain a deeper insight into its clinical significance and value. It may therefore be a long time before it finds general acceptance.

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