An ever-increasing number of individuals are being treated for non-ST-elevation acute coronary syndrome (NSTEACS). The clinical profile of these patients is highly heterogeneous, and the incidence of serious adverse events varies widely. Thus, stratification of these patients on the basis of prognosis is an enormous challenge because it should be rapid, accurate and efficient in order to enable the early identification of patients at significant risk for a poorer outcome. Their subsequent management should be adjusted to their initial prognostic profile, once it has been defined; more aggressive medical and interventional therapies are applied in those patients in whom the available scientific evidence is indicative, a priori, of a higher risk.

In this context, given its universal availability, low cost and simplicity, the electrocardiogram is a basic “bedside” tool that provides highly important, and early, information. Indeed, a number of reports have consistently demonstrated the negative short-term and long-term prognostic implications of the presence (and magnitude) of ST depression in the electrocardiogram performed at admission. We know, however, that in 34% to 54% of the patients with NSTEACS, the ST segment shows no evidence of changes upon their arrival at the hospital, and that, once again, the subsequent course is highly variable. This explains the interest in the study of other electrocardiographic parameters that could provide additional information that would complement the ST segment analysis, such as QRS complex duration, T wave abnormalities, or duration of the corrected QT interval, the utility of which has been demonstrated in some series, mostly involving smaller groups of patients from a single center.

**QT Interval and Myocardial Ischemia: From the Cell to the Patient’s Bedside**

The QT interval is determined in the electrocardiogram from the beginning of the QRS complex (whether it starts with a Q wave or an R wave) to the point at which the T wave (or the U wave, if present) returns to the isoelectric line. Thus, it includes the duration of ventricular depolarization and repolarization, and corresponds to the action potential duration.

In a number of epidemiological studies involving theoretically healthy individuals, the ventricular repolarization abnormalities in the electrocardiogram (not only the ST segment deviations, but changes in T wave morphology and QT interval prolongation as well) have been associated with an increased risk of sudden death and of cardiovascular death, probably because they could be markers of ventricular hypertrophy, left ventricular dysfunction or myocardial ischemia. Acute myocardial ischemia has been shown to modify the duration of the QT interval, increase repolarization heterogeneity (expressed as an increase in QT dispersion) and prolong the duration of the maximum electrocardiographic QT interval. Several mechanisms have been proposed to be involved in the prolongation of the QT interval secondary to acute myocardial ischemia: changes in the myocardial response to catecholamines or to cholinergic stimulation, perturbation of calcium or potassium ion channels, or induction of changes in the intracellular hydrogen concentration.

Beyond the basic mechanisms, clinical interest in this causal relationship is long-standing, and the observation that acute transmural myocardial ischemia prolonged the QT interval, and that this increase in the QT interval following Q wave acute myocardial infarction was associated with a significantly higher risk for sudden death, are findings that were reported in the eighties. More recently, Nowinski et al demonstrated that the myocardial ischemia occurring during balloon inflation in percutaneous transluminal coronary angioplasty immediately produced changes.
in ventricular repolarization, including a significant prolongation in the QT interval that persisted for minutes, or even hours. These findings suggest the possibility of employing the QT interval as an early marker of acute and transient myocardial ischemia.

However, the possible clinical applications of the previous experimental data in patients with NSTEACS arrived only a relatively short time ago. Recently, in 2 series involving consecutive, unselected patients with a diagnosis of NSTEACS, the prognostic value of the corrected QT (QTc) interval at admission and the maximum QTc interval during the first 48 hours were determined, and it was observed that values over 450 milliseconds were significantly associated with an increase in the incidence of major short-term and long-term adverse events. In contrast to the findings reported following transmural myocardial infarction, the negative impact on prognosis of a prolonged QTc interval in NSTEACS was not associated with a higher risk for sudden death presumably due to ventricular arrhythmias, but due to an increase in the incidence of major short-term and long-term adverse events.

It is a welcome development. It is a welcome development.

In agreement with the findings recently reported by our group, these authors demonstrate that a QTc interval with a duration of more than 458 milliseconds, detected within the first 24 hours of admission, with no electrocardiographic evidence of ST-T changes, serves as an independent risk marker and is related to a significant increase in the probability of adverse events (death, infarction, or coronary revascularization) within the first 28 days of the onset of the symptoms. In our opinion, there are 3 aspects of this report on which particular emphasis should be placed: on the one hand, the high incidence (38%) of adverse events observed, taking into account the selection criteria employed in the study, which highlights the degree of heterogeneity of populations with acute coronary syndrome and an isoelectric ST segment at admission; on the other hand, the reproducibility of the QTc value beyond which the risk for a complicated clinical course increases (around 450-460 milliseconds); finally, the correlation observed between the duration of the QTc interval and the maximum troponin T concentrations, which, as the authors themselves point out, makes it possible to move up a rung in the understanding of the complex pathophysiological relationship between the QT interval and acute myocardial ischemia and its severity.

Meanwhile, in these times of numerous biological markers and of increasingly sophisticated diagnostic tests, a simple, long recognized variable, easy to obtain in that old electrocardiogram, is making advances in the stratification of patients with NSTEACS according to prognosis. It is a welcome development.
REFERENCES


