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EDITORIAL COMMENT

Looking at late repolarization to see early disease[☆]

Olhar para a repolarização tardia para «ver» doença precoce?

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Cardiac pump function is the central physiological element of the cardiovascular system. Cardiac myocytes are highly specialized cells responsible for both conduction of electrical impulses and mechanical contraction.[1] For this mechanical function to work correctly, the myocardial cells must be electrically activated in a coordinated sequence.

The importance of cardiac arrhythmias comes from the potential for the mechanical function of cardiomyocytes to become disordered, which can reduce or (in the case of atrial or ventricular fibrillation) even eliminate cardiac output.

Arrhythmias can occur as a result of structural heart disease, but also in hearts without apparent structural defects. The underlying mechanisms can be classified as alterations in impulse formation (automaticity or triggered activity) or alterations in impulse conduction (reentry).[2]

Reentry circuits may be caused by an anatomical substrate or they may be functional rather than anatomical in nature, resulting from the electrophysiological properties of the tissue itself.[3]

Various studies over the last three decades have established that the ventricular myocardium is electrically heterogeneous, being composed of layers of at least three electrophysiologically and functionally different cell types: epicardial cells, M cells, and endocardial cells.[4,5]

In functionally determined circuits, heterogeneity or dispersion of repolarization is a prerequisite for unidirectional block, which is essential to reentry.[3]

Attempts have been made over the years to use the surface electrocardiogram (ECG) to predict arrhythmias by detecting regional differences in cardiac tissue by non-invasive means that will identify the presence of an arrhythmic substrate giving rise to reentry circuits. One such element of the ECG is QT interval dispersion, which indicates regional differences in duration of the action potential (recovery of excitability) and in certain circumstances can reflect risk of malignant arrhythmias.[6–8]

In this issue of the *Journal*, Kasapkara et al.[9] apply the hypothesis of Antzelevitch et al.[10] that the interval from the peak to the end of the electrocardiographic T wave (T-peak to T-end [Tpe] interval) may be used as an index of total dispersion of repolarization. They analyze a group of patients diagnosed with sarcoidosis but with no signs of cardiac involvement, using the Tpe/QT ratio.

The differences observed in these parameters between patients with sarcoidosis but without cardiac involvement and healthy controls give grounds for hope that such assessment may be able to detect cardiac involvement while still at a subclinical stage.

Although the authors designated the patients with sarcoidosis as having no signs of cardiac involvement, since they had similar left ventricular ejection fraction to the healthy controls, myocardial hypertrophy (greater interventricular septum thickness, posterior wall thickness, and relative wall thickness) was detected in the sarcoidosis group. This may indicate that the myocardium was in fact already affected by the disease process in these patients.

Despite this limitation, which could reduce the impact of the results, the authors' suggestion, that a non-invasive method (the surface ECG) can be used for risk stratification in patients with cardiac involvement in this systemic disease, is an interesting one.

Conflicts of interest

The author has no conflicts of interest to declare.

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