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Ventricular depolarization and repolarization for stratifying the risk of malignant ventricular arrhythmias and sudden death

Despolarización y repolarización ventriculares para estratificar riesgo de arritmias ventriculares malignas y muerte súbita

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To the Editor:

I would like to make some comments concerning the article published in CorSalud journal: Depolarization (QRS complex) or ventricular repolarization (QT interval): Which one adds further value to diagnosis and prognosis in different clinical scenarios?[1]

The title might cause some confusion: it seems that these two edges of the problem are being confronted and there is no reason to confront them but to use both to reach a greater approach to the reality of each patient in terms of risk stratification using electrical predictors of malignant ventricular arrhythmias (MVA) and sudden death (SD). We have to combine, not divide, add and not subtract, why pitting one process against the other? Moreover, the QT interval is not the only one that represents repolarization. Recognizing the true transcendence of each premonitory sign would be still difficult even if we use both processes, unless there was a previous event (MVA or aborted SD)[1-7].

Depolarization and repolarization share processes and phenomena, such as alternation, non-uniformity, heterogeneity, dispersion and electrical remodeling[8,9].

The conduction of the electrical impulse in the many structures of the cardiac system is neither homogeneous nor uniform, even under normal conditions and within a certain range. On the contrary, it is heterogeneous and uneven, since the impulse is formed until the end of the journey at the level of the Purkinje arborizations in the ventricular walls; this is observed in both depolarization and repolarization. The anatomical characteristics, physiology and physiopathology differ in each structure, sinus and atrio-ventricular nodes, atria, ventricles, bundle of His, branches and fascicles. Not only from one structure to another but in the same one. For example, at the atrial and atrio-ventricular node levels, there are different electrophysiological properties, conduction speed and recovery capacity in the various units; at the ventricular level, subepi-cardial, subendocardial, mediomyocardial areas; at the level of branches and fascicles, central zones vs. peripheral areas, with different conduction velocities and more or less far destination towards the ventricles. Of course both, heterogeneity and non-uniformity, are exacerbated in conditions of illness. It is stressed that, both depolarization and repolarization are not uniform or homogeneous processes, but quite the opposite. The anatomical and functional heterogeneities appear in diverse structures and are, along with dispersion, the mother of arrhythmogenesis[8-10].

Both processes, depolarization and repolarization, share another critical phenomenon, the dispersion (origin of anatomical or functional heterogeneity, or both, which in turn leads to arrhythmogenesis) at the level of any structure of the elec-

trical impulse conduction system. Spatial and temporal dispersion occurs between apex and base, septum and free walls, both ventricles (circumferential) and myocardial wall[8,11].

This property is observed in the atria, in the QRS complex, in the QT, T_{PEAK}-T_{END}, J-T_{PEAK}, and J-T_{END} intervals. There is ionic and electrical dispersion, as well as dispersion of refractory periods and action potential duration[8,11].

The alternation of electrical properties is also shared by depolarization and repolarization: action potential duration, conduction velocity, intracellular calcium concentrations, and spatial and temporal phenomena (pasting and changing QRS duration). Aspects that are observed in the QRS, in the T wave, in the various intervals, in the ST segment and in other electrical accidents, both in one process and the other[7].

Let's see other situations in which depolarization and repolarization are connected. Total cosine R-to-T is a vectocardiographic marker that reflects the spatial dispersion between depolarization and repolarization, and predicts SD in myocardial infarction, ventricular fibrillation and ventricular tachycardia; it results from averaging the QRS vector angle by reconstructing the electrocardiogram[12].

Another example is the Brugada syndrome where there are hypotheses invoked in its physiopathology, some linked to depolarization and others to repolarization. Several pathophysiological explanations for ST elevation in this syndrome have been proposed; disorders of repolarization due to a possible increase in the right ventricular transmural dispersion of repolarization or more specifically of its outflow tract, and depolarization disorder, hypothesis based on the evidence of activation delay of the right ventricle subepicardium or its outflow tract (late potentials, subdivisions). Again both phenomena are connected[13].

The premonitory electrical signs of MVA and SD are diverse, many, elusive, difficult to interpret (unless there has been a fatal event so there would be no discussion) and they cannot be given absolute value, nor can they be ruled out or forgotten, at the time of stratifying the risk of MVA or SD as a debut or as a relapse. In addition, some can range from normality and insignificance to become serious problems. It is easy to give a prognosis and adopt a therapeutic behavior after the disaster, but in those that have not yet presented, the risk can be applied by groups but it is very difficult, and often impossible, to stratify a particular individual[5-7, 14,15].

Let's see how many signs are related to depolarization and how many with repolarization, and we will understand that none can be underestimated (**Box 1**)[5-7,15,16]. Besides, in the torsades de pointes there are also premonitory electrical signs of their presentation related to these two processes (**Box 2**)[17].

For all that I have said, I think that there should be no competition between depolarization and ventricular repolarization as premonitory electrical

Box 1. Electrical signs that are related to depolarization and repolarization processes.

With depolarization
- Bundle branch block
- Ventricular ectopic QRS interval (VEQSI)
- QRS notches
- QRS higher or lower voltage
- High and narrow QRS
- QRS duration
- Interventricular dyssynchrony
- Prominent R in aVR
- Delayed QRS transition in the precordial leads
- Prolonged intrinsicoid deflection
- Alternation in the different elements of depolarization
- Total cosine R-to-T
- Terminal QRS distortion and fragmentation
- Zigzag conduction and unblocked QRS spikes
With repolarization
- Dispersion of the QT, J-T_{PEAK}, J-T_{END}, and T_{PEAK} -T_{END} intervals
- Alternation of ST segment and T wave
- Paradoxical and stunned QT
- Waves T, T1T2, TU, J, epsilon
- Late potentials
- Electrical memory
- T_{PEAK}-T_{END} interval
- TU discordance
- Giant TU
- Wilson's spatial ventricular gradient
- Post-extrasystolic T wave
- Brugada signs, and short and long QT
- Lower voltage, wide base, bimodal T wave

signs of MVA and SD risk, in order to stratify the risk for these events. Both processes must be combined to approach reality by groups (with greater success) and by individuals (much more difficult and sometimes impossible).

I have invited Dr. Elibet Chávez, a companion and a friend, who has worked on these issues for a long time, to put forward his view, which will be very well received.

Box 2. Premonitory electric signs of torsades de pointes related to depolarization and repolarization.

With depolarization
- Longer duration of the QRS in the first beat of the torsion
- Lower QRS angle
- Early post-depolarizations as triggers
- Slow rise of the QRS of the ventricular extrasystole
- QRS Fragmentation
With repolarization
- Abnormal TU waves
- Prominent U
- QT interval and T wave alternation (in duration, configuration, polarity and amplitude)
- Increased QT and T_{PEAK}-T_{END} intervals
- Start of arrhythmia with giant TU

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Ventricular depolarization and repolarization for stratifying the risk of malignant ventricular arrhythmias and sudden death. Reply

Despolarización y repolarización ventriculares para estratificar riesgo de arritmias ventriculares malignas y muerte súbita. Respuesta

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Key words: QRS complex, QT interval, QRS duration, Malignant ventricular arrhythmia, Sudden cardiac death, Electrocardiographic risk markers

To the Editor:

First of all, I would like to thank the respected teacher Margarita Dorantes[1] for her invitation, since, more than a confrontation, these are points of view and explanations to the readers of our journal. The Editorial title "Depolarization (QRS complex) or ventricular repolarization (QT interval): Which one adds further value to diagnosis and prognosis in different clinical scenarios?"[2] does not mention what I have been doing as a researcher. Though being responsible for the title, it just mentions what researchers worldwide have done while searching for different electrical signs in different clinical scenarios. A number of them have divided repolarization and depolarization, or at least they have considered them separately; for example, in the Brugada syndrome and ischemic heart disease[3-9].

In the latter, we have found both depolarization and repolarization to be predictors of malignant ventricular arrhythmias (MVA). These results, published in Medicina Intensiva from Spain[10], presented greater sensitivity and specificity for the QRS dispersion (QRSd) over that of the QT interval. Nevertheless, we study both processes (depolarization and repolarization), with no intention of giving more