CorSalud 2018 Jan-Mar;10(1):1-3

CorSalud

Cuban Society of Cardiology

Editorial

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Depolarization (QRS complex) or ventricular repolarization (QT interval): Which one adds further value to diagnosis and prognosis in different clinical scenarios?

Despolarización (complejo QRS) o repolarización ventricular (intervalo QT): ¿Quién aporta más al diagnóstico y pronóstico en diferentes escenarios clínicos?

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ARTICLE INFORMATION

Key words: Electrocardiography, QRS complex, QT interval, QRS duration, Prognosis
Palabras clave: Electrocardiografía, Complejo QRS, Intervalo QT, Duración del QRS, Pronóstico

Endo-epicardial excitation ends first than the total QRS traces on the electrocardiogram (ECG). In my opinion the phenomenon of synchrony versus electromechanical dyssynchrony began to be understood from the study of Durrer et al.[1] in 1969, published in 1970. Fourteen years later Cassidy et al.[2] mapped a left ventricular endocardial activation demonstrating that the duration of left ventricle (LV) endocardial activation only comprised 41% of the total surface QRS complex on ECG. The aforementioned studies describe ventricular depolarization or QRS duration.

Measurement of the QT-interval on ECG (which represents ventricular depolarization and repolarization) has been proposed for detecting ventricular repolarization. This interval and its dispersion have been more widely studied and spread. Their ionic implications and modifiers of these ion channels have been known. Likewise, thoroughly studies have been conducted on how to perform and interpret their measurements[3]. In the normal heart, M cells located in the middle myocardium, prolong their action potential much more than epicardial or endocardial cells, so there is an electrotonic coupling with the adjacent layers, which coincides with the end of the T wave on ECG[4].

The ischemic myocardial lesion allows these M cells-intrinsic expression, which are manifested in the surface ECG as a prolongation of the QT[4] interval. Although the risk of prolonged QT interval and its dispersion has been widely and well described in literature, I insist on separating depolarization from ventricular repolarization, much more when we refer to ischemic myocardium. Wiener *et al.*[5], demonstrated marked changes in the activation of akinetic and dyskinetic zones in patients with coronary artery disease, and observed that the activation ended, even after total QRS registration. I consider that separating the QRS complex from the JT interval in the diagnosis and prognosis of patients with myo-

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cardial disease may lead to a better understanding of these pathophysiological phenomena as in acute myocardial infarction, heart failure, left bundle branch block (LBBB) and even in cardiac resynchronization treatment.

I have read two original articles on the issue in CorSalud[6,7], which focused attention upon the ECG, emphasizing in the QRS duration variable for both research groups. Rodríguez Jiménez et al.[6] suggest that a QRS duration greater than 90 milliseconds (ms) was associated, in patients with acute myocardial infarction with ST-segment elevation, to an adequate discrimination capacity to predict a reduced left ventricular ejection fraction at discharge. On the other hand, López Ramírez et al.[7], in patients with coronary artery bypass graft surgery, found that the QRS complex duration was significantly associated (p=0.021) with higher mortality, with a mean 92.0± 22.0 ms in the live patients, compared with an average 100.0±24.5 ms in the deceased. These two publications are examples of the searching for the «problem» in depolarization.

QRS dispersion (QRSd) is another variable related to ventricular depolarization. The researches that have analyzed it in recent years have provided useful information in different scenarios of clinical practice[8]. In a letter to the editor, published in the Hellenic Journal of Cardiology[9], the authors suggest that the ECG could give us clues for the prediction of the response to cardiac resynchronization therapy, when the QRS duration has not been the best predictor for the response to this procedure. They argue this referring to a study where the QRSd variable was evaluated as a response to cardiac resynchronization therapy, and it was suggested that the greater the QRSd there is probably greater mechanical dyssynchrony and, after resynchronization, the responding patients had lower QRSd and, therefore, better mechanical synchrony[10]. My reflection about this links the physiology of the studies of Durrer et al.[1] and Cassidy et al.[2] with the knowledge of LBBB dyssynchrony and the response to cardiac resynchronization therapy described above. On the other hand, Tsagalou et al.[11], showed higher values of QRSd in patients with heart failure who suffered sudden death compared with survivors (56±13 vs. 46±16 ms [p <0.02]).

We have mentioned previous studies[10,11] to show how QRSd is a variable of interest in different scenarios of the diseased myocardium; because, if we return to ischemic heart disease, Chávez-González et al.[12] described that increases in the duration and dispersion of QRS showed a higher probability for the appearance of ventricular arrhythmias in the initial phases of acute myocardial infarction, than increases in the corrected QT interval and its dispersion. These ventricular arrhythmias were more likely to occur in patients with higher QRSd values with respect to the dispersion of the corrected QT (area under the curve): 0.942 vs. 0.660 for ventricular tachycardia, and 0.966 vs. 0.852 for ventricular fibrillation.

Fragmented QRS (fQRS) is another electrocardiographic finding described as the presence of a myocardial infarction scar and as a risk of cardiac events in these patients. It has been suggested that the mechanism of fQRS production is caused by a zigzag conduction around the scarred myocardium and has been associated with aneurysms of the ischemic area[13].

After reading the articles published in this issue of CorSalud[6,7], my aim has been to draw attention to the importance that the study of ventricular depolarization has shown. I call on the scientific community to discriminate the importance between depolarization and repolarization in multicenter studies, because perhaps new results would lead us to the search for a more advanced thought in other preventive or therapeutic forms in different clinical scenarios, where variables such as duration, dispersion and fragmentation of the QRS have proven diagnostic and prognostic value.

CONFLICTS OF INTERESTS

None declared

REFERENCES

1. Durrer D, van Dam RT, Freud GE, Janse MJ, Meijler FL, Arzbaecher RC. Total excitation of the isolated human heart. Circulation. 1970;41(6):899-912.
2. Cassidy DM, Vassallo JA, Marchlinski FE, Buxton AE, Untereker WJ, Josephson ME. Endocardial mapping in humans in sinus rhythm with normal left ventricles: Activation patterns and characteristics of electrograms. Circulation. 1984;70(1):37-42.
3. Locati ET, Bagliani G, Padeletti L. Normal ventricular repolarization and QT interval: Ionic background, modifiers, and measurements. Card Electrophysiol Clin. 2017;9(3):487-513.

4. Patel C, Burke JF, Patel H, Gupta P, Kowey PR, Antzelevitch C, *et al*. Is there a significant transmural gradient in repolarization time in the intact heart? Cellular basis of the T wave: A century of controversy. Circ Arrhythm Electrophysiol. 2009; 2(1):80-8.
5. Wiener I, Mindich B, Pitchon R, Pichard A, Kupersmith J, Estioko M, *et al*. Epicardial activation in patients with coronary artery disease: Effects of regional contraction abnormalities. Circulation. 1982;65(1):154-60.
6. Rodríguez Jiménez AE, Cruz Inerarity H, Valdés Arias B, Dr. Quintana Cañizares G, Toledo Rodríguez E. Duración del QRS como predictor de baja fracción de eyección en el infarto miocárdico con elevación del ST. CorSalud [Internet]. 2018 [citado 14 Ene 2018];10(1):13-20. Disponible en: http://www.revcorsalud.sld.cu/index.php/cors/article/view/191/622
7. López Ramírez M, Nafeh Abi-rezk M, Moya Liriano H, Gómez Sardiñas JA, Tamargo Barbeito TO, Valdés Álvarez J. Duración del QRS y su relación con la mortalidad postoperatoria de la cirugía de revascularización miocárdica. CorSalud [Internet]. 2018 [citado 14 Ene 2018];10(1):4-12. Disponible en: http://www.revcorsalud.sld.cu/index.php/cors/article/view/206/621
8. Donoiu I, Costinel Târtea G, Chávez-González E. Is there a utility for QRS dispersion in clinical practice? J Mind Med Sci [Internet]. 2017 [citado 14 Ene 2018];4(2):132-41. Disponible en: https://scholar.valpo.edu/cgi/viewcontent.cgi?article=1080&context=jmms
9. Chávez-González E, Moreno-Martínez FL. QRS dispersion is better than QRS duration for predicting response to cardiac resynchronization therapy. Hellenic J Cardiol. 2016;57(5):366-7.
10. Chávez-González E, Alonso Herrera A, Carmona Puerta R, Pérez Cabrera D, Ramos Ramírez RR, Gómez Paima W, *et al*. Dispersión del QRS como índice de disincronía en el bloqueo de rama izquierda y de sincronía tras la terapia de resincronización cardíaca, una variable de respuesta exitosa. CorSalud [Internet]. 2015 [14 Ene 2018]; 7(2):106-16. Disponible en: http://www.revcorsalud.sld.cu/index.php/cors/article/view/28/73
11. Tsagalou EP, Anastasiou-Nana MI, Karagounis LA, Alexopoulos GP, Batziou C, Toumanidis S, *et al*. Dispersion of QT and QRS in patients with severe congestive heart failure: Relation to cardiac and sudden death mortality. Hellenic J Cardiol. 2002; 43(6):209-15.
12. Chávez-González E, Rodríguez Jiménez AE, Moreno-Martínez FL. Duración y dispersión del QRS para predecir arritmias ventriculares en las fases iniciales del infarto agudo de miocardio. Med Intensiva. 2017;41(6):347-55.
13. Take Y, Morita H. Fragmented QRS: What is the meaning? Indian Pacing Electrophysiol. 2012; 12(5):213-25.