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Editorial

QRS Dispersion, a New Variable to Value the Prediction of Cardiovascular Risk in Hypertensive Patients

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Hypertension is one of the most devastating cardiovascular risk factors for death and disability, especially those related to ictus, heart disease and renal failure. Despite of this knowledge; due to its clinic and epidemiologic characteristics of silent behavior and high prevalence worldwide, hypertension is still a very difficult disease to be controlled affecting more than one billion people and causing an estimate of almost 10 million deaths per year [1]. Increasing in blood pressure is and early hall mark epidemiological transition. In addition, overall impact varies depending on treated or untreated hypertension and approximately 62% strokes and 49% ischemic heart disease are attributed to suboptimal blood pressure control [2,3].

The primary objective in the treatment of hypertension is to reduce fatal and nonfatal hypertension-related complications. A necessary first step in this endeavor is to prevent damage to target organs which is without questioning a critical point to evaluate the injury and the risk that this disease represents to affected population. Nowadays, science tries to determine this damage in advance [4]. Left ventricular hypertrophy (LVH), is one of the complications that results from uncontrolled hypertension. The growth of left ventricular mass is related with an increase of the oxygen consumption and it has been also associated to cardiac arrhythmias [5].

The electrocardiogram has become in a very valuable tool to assess target organ damage (TOD). The changes on the electrophysiology of the auricular wall are considered no homogeneous modifications of atrial depolarization; these alterations are expressed in the electrocardiogram as increment in P wave duration and dispersion. Chávez-González., *et al.* described this modifications in a population of children suffering from hypertension. They stated that these electrophysiological changes of the auricular wall can be considered TOD [6].

The electrocardiographic variable that has been widely studied in association to hypertension is the QT interval; the increment of this interval is related to the presence of LVH [7].

Furthermore, other electrocardiographic variables has been described in hypertensive patients such as the voltage in aVL lead, the index of Sokolow-Lyon, Cornell index and some others broadly reported in scientific literature [8]. They are all associated to the assume presence of LVH with low sensibility and specificity. However; we consider the electrical remodeling that may appear in the auricular wall associated to hypertension might developed itself as well in the ventricular wall of any patient that suffers hypertension, so we should study QRS duration and dispersion which represents the ventricular depolarization, as well as the P wave represents the auricular depolarization [9].

Additionally, QRS dispersion has been studied in different clinical and therapeutic scenarios where it has been related for example, with bad prognoses in systolic heart failure. The reduction of QRS dispersion after cardiac resynchronization therapy is considered a vari-

able of successful therapy [10]. Increments in QRS dispersion in Acute Coronary Syndromes have also been related with the presence of ventricular arrhythmias [11]. According to Turrini., *et al.* QRS dispersion (≥ 40 ms) remained an independent predictor of sudden death in Arrhythmogenic Right Ventricular Cardiomyopathy with a sensitivity and specificity of 90% and 77%, respectively [12].

Ferrer-Orozco., *et al.* [13], considering that QT interval includes ventricular depolarization (QRS complex) and ventricular repolarization (JT interval), separated these two electrophysiological phenomenas and demonstrated in a case-control study that the maximum duration of the JT interval was 328,21± 34,43 vs 329,29 ± 31,25 milliseconds respectively, p = 0.989. The highest QRS duration was 116, 07 ± 16,41 vs 101,43 ± 5,35 milliseconds respectively, p = 0.033. The QRS dispersion was 44,64 ± 19,72 vs 30,71 ± 6,16 milliseconds respectively for cases and controls, p = 0.033 with a cut point of 40 milliseconds for QRS dispersion. In their study Ferrer-Orozco., *et al.* [13], also demonstrated increments of the QT interval in hypertensive patients vs non hypertensive; however, with the results expressed above, it can be stated that the increments of QT interval depended, in the sample studied, on the increments in QRS duration. The latter supports the theory, that in the population studied, there are electrophysiological changes probably determined by the non-homogeneity of the ventricular depolarization which provides increments in QRS duration and dispersion.

The changes in the electrophysiology of the ventricular myocardium in hypertensive patients cause an increment in the heterogeneity of the depolarization and repolarization, which is associated to the appearance of arrhythmias such as premature ventricular complexes [7].

There are not similar studies to the one made by Ferrer-Orozco., *et al*. that relate hypertension with QRS dispersion. The design of population studies that demonstrate this association might support the theory of the electrical ventricular remodeling in people suffering from hypertension that would justify the increments in QRS duration and dispersion. Some other studies could be designed to reveal the presence of these changes in the electrocardiogram, without the presence of LVH which might support, even more, the theory of the electrical ventricular remodeling as TOD in hypertensive patients.

Conflict of Interest

None.

Bibliography

1. McNiece KL., *et al*. "Left ventricular hypertrophy in hypertensive adolescents. Analysis of risk by 2004 National High Blood Pressure Education Program Working Group staging criteria". *Hypertension* 50.2 (2007): 392-395.

2. Ávila JA., *et al*. "Considerations about the 2017 Cuban Guideline for High Blood Pressure". *CorSalud (Revistas de Enfermedades Cardiovasculares)* 10.3 (2018): 266-273.

3. Chakrapani BS and Mukul RF. "Cardiovascular disease and health". *EC Cardiology* 7.2 (2020): 1-8.

4. Cortese F., *et al*. "Vascular, cardiac and renal target organ damage associated to arterial hypertension: which noninvasive tools for detection?" *Journal of Human Hypertension* (2020): 1-12.

5. Aronow WS. "Hypertension and left ventricular hypertrophy". *Annals of Translational Medicine* 5.15 (2017): 310.

6. Chávez-González E., *et al*. "Dependence of P-wave dispersion on mean arterial pressure as an independent hemodynamic variable in school children". *Colombia Médica* 44.3 (2013): 178-183.

7. Satpathy S., *et al*. "Correlation of blood pressure and QT interval". *National Journal of Physiology, Pharmacy and Pharmacology* 8 (2018).

8. Cabezas M., *et al*. "Comparision of the sensitivity and specificity of electrocardiographic criteria for left ventricular hypertrophy according to the Romhilt-Estes, Sokolow-Lyon, Cornell and Rodriguez Padial methods". *Revista Española de Cardiología* 50.1 (1997): 31-35.

9. Chávez-González E., *et al*. "El electrocardiograma del paciente hipertenso. Dispersión de la onda P: nueva medida a tener en cuenta". *Medisur* 8.5 (2010): 71-75.

10. Chavez-González E and Moreno-Martínez FL. "QRS dispersion better than QRS duration for predicting response to cardiac resyncronization therapy". *Hellenic Journal of Cardiology* 57.5 (2016): 366-367.

11. Ionuț D., *et al*. "Is there a utility for QRS dispersion in clinical practice?" *Journal of Mind and Medical Sciences* 4.2 (2017): 132-141.

12. Turrini P., *et al*. "Dispersion of ventricular depolarization-repolarization: a noninvasive marker for risk stratification in Arrhythmogenic Right Ventricular Cardiomyopathy". *Circulation* 103.25 (2001): 3075-3080.

13. Ferrer-Orozco L., *et al*. "Electrocardiographic modifications in hypertensive patients without associated diseases". *Revista Cubana de Investigaciones Biomédicas* 38.1 (2019): e172.

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