

Electrocardiographic modifications in hypertensive patients without associated diseases

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ABSTRACT

Introduction: High Blood Pressure is considered a devastating disease due to its several complications, which do not manifest until there is severe damage established, reason why the electrocardiogram can be a tool easily accessible in clinical practice to detect cardiac complications that may happen in the future allowing us to act early in their treatment.

Objective: Identify the electrocardiographic modifications present in hypertensive patients without associated diseases.

Methods: An analytical observational study of case-control in hypertensive patients without associated diseases or supposedly healthy patients, belonging to the Family Medical Office # 17.30, of the "Chiqui Gómez Lubián" Polyclinic; Santa Clara municipality was carried out in the period from November 2017 to February 2018.

Results: Maximum P-wave mean was significantly higher in the case group (56.43 ms vs 35 ms control group, $p=0,000$), behaving similarly when comparing P-wave dispersion in both groups ($56, 43 \pm 19, 48$ ms vs $35, 00 \pm 16, 98$ $p=0,001$). Maximum corrected QT interval showed significant differences between both groups ($471, 75 \pm 32, 04$ ms vs $448, 36 \pm 40, 84$ ms, $p=0,020$), QT dispersion did not showed significant differences. The maximum JT segment and its dispersion did not showed to be significantly different.

Conclusions: P wave dispersion, maximum P wave, maximum QRS, its dispersion and the maximum QT interval showed significant differences between both groups; those results were

findings on hypertensive patients group. On this sample the increase QT intervals could be dependent on QRS but not on the JT interval.

Key words: electrocardiogram, hypertension, dispersion, P wave, QT interval, QRS complex.

INTRODUCTION

Systemic arterial hypertension (HT) is a multifactorial disease that includes the elevation of blood pressure levels, cardiovascular risk factors and left ventricular hypertrophy. It is a progressive silent disease, usually asymptomatic in its onset, progressing to target organ damage over time. When hypertension appears earlier the target organ damage is more severe. If hypertension is not controlled early the endothelial damage is greater.⁽¹⁾

Uncontrolled hypertension generates left ventricular hypertrophy (LVH), which causes increased myocardial oxygen consumption, and produces cardiac arrhythmias (example: atrial fibrillation).⁽¹⁾ Several studies has related the presence of hypertension and LVH with increases in P wave dispersion and QT interval, both alterations have been described associated with arrhythmias, supraventricular and ventricular. Atrial and ventricular arrhythmias, both, are considered hypertension comorbidity, the mechanisms involved are several and include LVH and relative myocardial ischemia. Atrial fibrillation (AF) is more frequently in hypertensive patients, around 57 % of patients who suffer AF are hypertensive.^(1,2)

Now day the science is interested on detect the target organ damage early in asymptomatic patients. Multiple electrocardiographic changes have been studied as predictors of target organ damage, principally on heart.⁽³⁾ Electrophysiological changes on the heart, it is expressed in the electrocardiogram by increases P wave dispersions, QRS complex and QT interval, it changes are suggested an increased risk for developing cardiac arrhythmias.⁽⁴⁾

Increases in P wave dispersion and QT interval associated with hypertension has been related to cardiac arrhythmias. Considering QT interval includes depolarization (QRS complex) plus ventricular repolarization (JT interval) of the ECG, we decided to find the relationship between the QRS complex and HT.⁽⁴⁾ However, we did not find in the literature reviewed studies that mention the disorders of ventricular depolarization (QRS complex) associated with HT, perhaps it represents a scientific novelty.

OBJECTIVE

To identify the electrocardiographic changes in the following waves, complexes and interval: P wave, QRS complex, QT and JT interval, present in hypertensive patients without associated diseases.

METHODS

An analytical observational study of case-control in hypertensive patients without associated diseases or supposedly healthy was carried out. The case group consisted of the patients diagnosed of arterial hypertension who fulfilled the inclusion criteria for this group (n = 28), through a non-probabilistic sampling based on the intentional sampling technique; in this group 11 men and 17 women participated, with an average age of 47.36 ± 11.94 . The control group (n = 14), patients dispensed as supposedly healthy, selected through non-probabilistic sampling using the technique of voluntary subjects; this group was formed by 7 men and 7 women, with an average age of 30.00 ± 12.27 years. Although the case group was under antihypertensive treatment in three blood pressure measurements, they showed mean blood pressure values of 104.57 ± 11.21 millimeters of mercury (mmHg) and the control group of 85.61 ± 6.00 (mmHg).

Case group inclusion criteria

Hypertensive patients without associated diseases such as: (Bronchial Asthma, Chronic Obstructive Pulmonary Disease, Diabetes Mellitus, Ischemic Heart Disease, Dilated Cardiomyopathy, Hypertrophic Cardiomyopathy, Hematological Diseases, Valvular Diseases, Hypothyroidism, Hyperthyroidism).

Hypertensive patients who do not consume antiarrhythmic drugs of group I or III.

Patients who wish to participate in the study. Inclusion criteria for the control group: Patients who are supposed to be healthy and who wish to participate in the study.

Control group inclusion criteria:

- Supposedly healthy patients who wish to participate in the study.

On both groups a 12-lead electrocardiogram was performed, the following waves and intervals: P wave, QRS complex, QT and JT intervals measurements were made, it was performed manually because it was considered to be the most used method in clinical practice. The variables were measured in each one of the 12-lead. Maximum and minor values in each ECG were used for calculating P wave dispersion ($PWD = P_{\text{maximum}} - P_{\text{minimum}}$) and QRS dispersion ($QRSd = QRS_{\text{maximum}} - QRS_{\text{minimum}}$). It was done by two observers, and the measure was taken when it was coincident.

When it was no coincidence, a third observer participated, but without knowing the measured values, he offered his measurement. Finally two closest measurements were taken and it was

taken into account if the difference between them did not exceed the different between means standard deviation values calculated for them. In this way it was possible because of the differences of these measurements, accepted to be included in the study, and would not exceed 5 milliseconds.

All data was collected in a form and recorded in a Microsoft Office Excel 2013 spreadsheet, and then exported to the SPSS statistical package (Statistical Packed for Social Science), version 20.0 for Windows for processing according to the type of variable.

Firstly, the analysis was carried out to determine the possible associations to the dependent variable (hypertensive yes / no) as well as the differences between the established groups; the homogeneity test based on the Chi-square distribution was used. Summary measures are used as mean, maximum, minimum and standard deviation to quantitative variables, Student's t test was used for comparison of means to independent groups. Finally the analysis with variables significant was done; we determined the operating range curves (ROC) to predictive capacity of the electrocardiographic variables studied, QRS duration and dispersion cut-off points were calculated.

ETHICS

All ethic principles were rigorous accomplished according to the Helsinki principles. Each individual signed an informed statement.

RESULTS

Table 1, maximum P wave was significantly higher in the case group, (56,43 milliseconds (ms) vs 35 ms; $p=0,000$). Comparing maximum corrected QT interval and its dispersion in both group, only the first showed significant differences ($471, 75 \pm 32, 04$ ms vs $448, 36 \pm 40, 84$ ms; $p=0,020$), while in its dispersion (97.86 ms vs 96.36 ms, $p = 0.522$).

Table 1: Statistical results of P wave and QT interval by group studied

| | Groups | | t | p |
|--------------------------------------|----------------|----------------|-------|--------|
| | Case group | Control group | | |
| Maximum P wave | | | | |
| X ± SD | 123,21 ± 11,56 | 101,43 ± 9,49 | 6,087 | 0,000* |
| mínimum | 100 | 80 | | |
| maximum | 150 | 120 | | |
| P wave dispersion | | | | |
| X ± SD | 56,43 ± 19,48 | 35,00 ± 16,98 | 3,500 | 0,001* |
| mínimum | 30 | 20 | | |
| maximum | 100 | 90 | | |
| Maximum corrected QT interval | | | | |
| X ± SD | 471,75 ± 32,04 | 448,36 ± 40,84 | 1,910 | 0,020* |
| mínimum | 421 | 400 | | |
| maximum | 574 | 526 | | |
| QT interval dispersion | | | | |
| X ± SD | 97,86 ± 31,73 | 96,36 ± 53,07 | 0,65 | 0,522 |
| mínimum | 55 | 44 | | |
| maximum | 184 | 235 | | |

Source: Form (X ± SD: mean ± standard deviation)

Maximum QRS complex in both groups, table 2 was compared. It was significantly higher in hypertensive group ($116,07 \pm 16,41$ vs $101,43 \pm 5,35$ $p=0,033$), QRS complex dispersion showed significantly difference in both groups, too (44.64 ms vs 30.71 ms, $p= 0,033$). The maximum JT interval (328.21 ms vs 329.29 ms, $p=.0,989$) and its dispersion ($83,93 \pm 32,81$ ms vs $85 \pm 37,77$ ms, $p= 0,873$) did not show to be significantly different comparing both groups.

Table 2. Statistical results to JT interval and QRS complex in each group studied

| | Groups | | t | p |
|-----------------------|----------------|----------------|-------|--------|
| | Case group | Control group | | |
| Maximum JT | | | | |
| X ± SD | 328,21 ± 34,43 | 329,29 ± 31,25 | 0,098 | 0,989 |
| minimum | 270 | 290 | | |
| maximum | 410 | 400 | | |
| JT Dispersion | | | | |
| X ± SD | 83,93 ± 32,81 | 85 ± 37,77 | 0,095 | 0,873 |
| minimum | 40 | 50 | | |
| maximum | 170 | 170 | | |
| Maximum QRS | | | | |
| X ± SD | 116,07 ± 16,41 | 101,43 ± 5,35 | 1,910 | 0,033* |
| minimum | 90 | 90 | | |
| maximum | 160 | 110 | | |
| QRS Dispersion | | | | |
| X ± SD | 44,64 ± 19,72 | 30,71 ± 6,16 | 0,65 | 0,033* |
| minimum | 20 | 20 | | |
| maximum | 110 | 40 | | |

Source: Form (X ± SD: mean ± standard deviation)

Maximum P wave, its dispersion, maximum corrected QT interval, the maximum QRS complex and its dispersion were found to be significantly higher in the group of hypertensive patients (Figure 1) showing the maximum P wave and its dispersion the largest areas under the curve, with 0.927 [0.845; 1,000] and 0.828 [0.683; 0.973] respectively. Corrected QT interval dispersion, the maximum JT and its dispersion did not turn out to be significantly different in both groups.

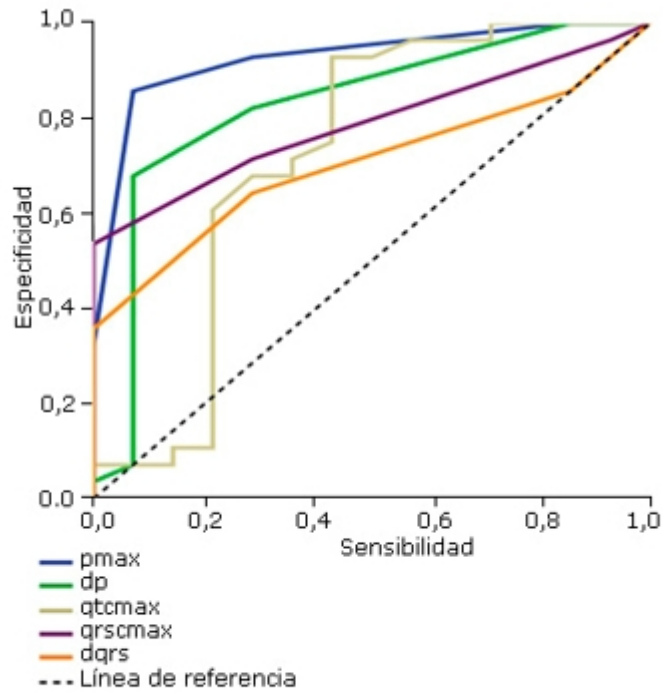


Fig. 1. ROC curve for electrocardiographic variables with statistical significance.

(pmax: Maximum P wave; dp: P wave dispersion; qtcmax: Maximum corrected QT interval; qrscmax: Maximum QRS; dqrs: QRS Dispersion)

Source: Statistical processing

Maximum QRS cut-off point was 116 ms. And its dispersion cut-off point was 40 ms. (Figure 2).

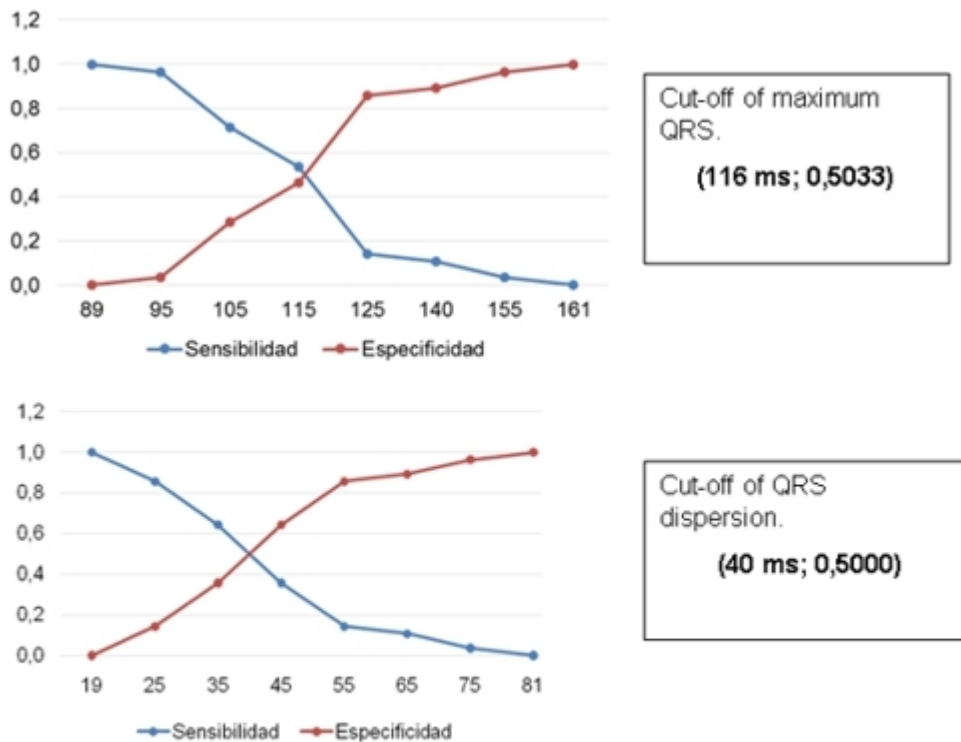


Fig. 2. Cut-off of QRS duration and its dispersion variables.

Source: Statistical processing

DISCUSSION

Hypertension and its anatomical and functional relationship with LVH, diastolic dysfunction and end-diastolic left ventricular pressure increase, can produce an increased pressure atrial with atrial enlargement. The angiotensin II action and others substances mediators can produce fibrotic and electrical changes on atrial electrical conduction, and in short time it can be evidenced in the ECG as an increase in P wave dispersion.⁽⁵⁾

Chávez et al. suggest that the pathophysiological bases of the P wave dispersion are related with the alterations on the atrial action potential, the delay on the electrical impulse and the atrial electrical remodeling on the atrial wall.⁽⁶⁾ The results of this work suggest in hypertensive patients studied, the presence of electrical atrial remodeling should be considered.

Dilaveris et al. suggest that fractionated and prolonged electrograms showed an intra and interatrial delay conduction have been demonstrated in patients with paroxysms of AF, while they are in sinus rhythm.⁽⁷⁾

This study did not determine the presence of LVH, by electrocardiogram or echocardiogram. It would be interesting to intend a study of these characteristics.

Coincident with these criteria, in the present study the values of the maximum P wave and its dispersion were significantly higher in hypertensive group, it could be demonstrate in hypertensive patients without associated diseases or complications, the presence of electrical changes in atrial wall. This criterion was raised, even in pediatric people, when Chávez-González et al demonstrated higher values of P wave dispersion in hypertensive and prehypertensive.⁽⁸⁾

AF risk can be reduced when P wave dispersion value decreases; when the control of hypertension is achieved the value of P wave dispersion can decrease. It could be reach with drugs capable of blocking the renin-angiotensin-aldosterone system are used.^(4,8) The group of hypertensive patients showed higher value of mean blood pressure perhaps they showed higher value of P wave dispersion. The above criteria suggests the effort for keeping control in this condition is necessary from primary care.

Hypertensive patients have a higher risk of presenting associated coronary disease, which must be distinguished from the cardiac involvement of hypertensive heart disease.⁽⁴⁾ Hypertensive heart disease contributes to the heterogeneity of the depolarization and repolarization of the cardiac muscle, it could be an anatomical substrate for the development of cardiac arrhythmias (eg: premature ventricular complex, AF). According to Satpathy S et al have mentioned with the knowledge about electrocardiogram markers such as: QT interval duration, QT dispersion (dQT), it can be predicted those patients who be able to suffer cardiac arrhythmias.⁽⁴⁾

The present study showed QT interval duration increases, but not in its dispersion. QT interval duration increases are well related to electrical heterogeneity and development of arrhythmias.

Regarding QRS measurement and its dispersion, is scarce on the literature, although in none of the reviewed publications was it studied in hypertensive patients. The dispersion of the QRS was studied for the first time in 2000 by Anastasiou-Nana et al.,⁽⁹⁾ who related it to sudden death in patients with advanced congestive heart failure.

A recent study about QRS complex duration and its dispersion as a predictor of ventricular arrhythmias around the initial phases of the infarction, demonstrated a greater risk of ventricular arrhythmias in patients with higher values of QRS duration and dispersion.⁽¹⁰⁾ The group of hypertensive patients studied showed greater values of QRS duration and dispersion, it may be related with arrhythmias risk in hypertensive patients and this result could has an inestimable value.

On the other hand, if there is a local cause to prolong the QRS, the QRS dispersion will be greater, affecting the myocardium electrophysiological properties as a whole and facilitating the appearance of potentially lethal ventricular arrhythmias. Hence, they have found a highly significant association more evident between QRS dispersion and ventricular arrhythmias.⁽¹⁰⁾

The theory could be raised, in hypertensive patients studied, the myocardium damage, secondary to hypertension, can determine areas of blockage and conduction delays, it can express greater values of QRS dispersion and duration. The present study showed significance difference for QRS duration and dispersion, but not JT interval. Therefore, we could state that in our sample of hypertensive patients, QT interval increases may depend on the QRS duration and not on the JT interval.

In hypertensive patients, asymptomatic or silent myocardial ischemia may occur in the absence of significant atherosclerotic obstructive coronary disease. This has various explanations, such as obstruction of epicardial arteries, intramyocardial pathway, alterations in the arteriolar wall or in its tone, subendocardial ischemia due to increased parietal stress, alterations between supply and demand of oxygen in hypertrophic myocardium, etc. It has been suggested the activation of the renin angiotensin aldosterone system can produce a greater degree of structural changes of the arterioles accompanied by coronary flow disorder.⁽¹⁰⁾ This could explain the statistically significant results of the maximum QRS complex and its dispersion when compared with non-hypertensive individuals, in the present study.

These results constitute a scientific novelty because the relationship between HT and QRS dispersion was observed for first time, which could become a useful tool for the prediction of ventricular arrhythmias in these patients. No scientific evidence was found in the literature that mentions the study of QRS dispersion in hypertensive patients. Finding in this study statistically significant differences between both groups that clearly show how QRS complex duration and dispersion are increase in hypertensive patients, suggest probably a variable can be considered to future studies for evaluating the risk to develop arrhythmias in hypertensive patients.

This can also be related to damage suffered by the conduction system and the ventricular myocardium in hypertensive cardiomyopathy, despite the absence of macroscopic evidence of complications of this disease in this group of patients. It could be considered that increases in QRS dispersion represent damage to the target organ.

CONCLUSIONS

P wave dispersion, maximum P wave, maximum QRS and its dispersion and the maximum corrected QT interval were the variables that showed significant differences between both groups, thus identifying the presence of these electrocardiographic changes in hypertensive patients. It turned out to be the P wave dispersion the variable of greatest statistical significance, while the QRS duration and its dispersion showed statistical superiority over the QT interval. On this sample the increase QT intervals could be dependent on QRS but not on the JT interval.

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AUTHOR CONTRIBUTIONS

Lisett Ferrer-Orozco: conception and design of the study, analysis of the data, interpretation of the data, drafting the article.

Yoandri Orozco-Martínez: collection of data, analysis of the data, interpretation of the data, critical revision of the manuscript.

Jesús Ignacio Medina-Morales: interpretation of the data, critical revision of the manuscript.

Beatriz Orozco Pérez de Prado: collection of data, critical revision of the manuscript.

Calixto Emanuel Orozco Pérez de Prado: collection of data, critical revision of the manuscript.

Rosa María Hernández Maldonado: analysis of the data, interpretation of the data and critical revision of the manuscript.

Elibet Chávez-González: conception and design of the study, critical revision of the manuscript.

All authors have read and approved the final manuscript.

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