

## The relation between arterial blood pressure variables and ventricular repolarization parameters

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### ABSTRACT

**Introduction:** Ventricular arrhythmia and sudden cardiac death risk are associated with prolonged electrocardiographic (ECG) QT and Tpeak-Tend intervals.

**Objective:** To evaluate the influence of blood pressure variables on ventricular repolarization parameters, especially QT and Tpeak-Tend intervals.

**Method:** Two groups of patients were enrolled in the study. The first group included 77 patients, with essential hypertension, aged 62±12 years, 40% males. The control group included 56 patients, age and sex matched, with optimal, normal and high normal blood pressure. They underwent 12-lead ECG and ventricular repolarization parameters were assessed. QT intervals: QTmax (maximal QT interval duration), QTc (heart rate corrected QTmax), QTm (mean QT interval duration in all leads), QTIIc (heart rate corrected QT interval duration in lead DII), and T wave variables: T0e (maximal T wave duration), Tpe (maximal Tpeak-Tend interval) and Ta (maximal T wave amplitude) were manually measured. Arterial blood pressure variables: systolic (SBP), diastolic (DBP), mean arterial (MAP) and pulse pressure (PP), were recorded.

**Result:** SBP was 139±24 mmHg, DBP 86±13 mmHg, MAP 103±15 mmHg, PP 53±16 mmHg, QTmax 430±51 ms, QTc 474±48 ms and Tpe 100±26 ms in the hypertensive group. Multiple regression analysis revealed significant associations (multiple R=0.985, significance F<0.01) of DBP with QTIIc (p<0.01) and Tpe (p<0.01). MAP was significantly associated (multiple R=0.986, F<0.01) with QTc (p<0.001) and QTm (p=0.014). Multiple regression analysis also revealed significant associations between blood pressure variables and ECG parameters in the control group. Arterial blood pressure values and mean arterial blood pressure were sensitive and specific predictors of prolonged QT intervals in the hypertensive group.

**Conclusion:** Elevated systolic and diastolic blood pressures and mean arterial pressure are predictors of prolonged QT intervals. Blood pressure variables are associated with the ventricular repolarization parameters.

**Keywords:** hypertension, QT interval, Tpeak-Tend interval, pulse pressure

## Introduction

Hypertension is a major risk factor for atherosclerotic disease<sup>1</sup>, and a significant association has been demonstrated between hypertension and arrhythmias<sup>2</sup>. Ventricular arrhythmias, common in hypertensive patients, are important causes of sudden cardiac death. The most important mechanisms by which hypertension predisposes to sudden cardiac death are: left ventricular hypertrophy, interstitial fibrosis, myocardial or subendocardial scars, silent myocardial ischemia, diastolic dysfunction and high sympathetic nervous activity<sup>1,3-5</sup>.

The risk of sudden cardiac death due to ventricular arrhythmias in hypertensive patients was demonstrated by prolonged QT intervals, QT dispersion, Tpeak-Tend interval and late ventricular potentials.

The QT interval, one of the most studied ECG parameters<sup>6</sup>, is associated with increased risk of torsades de pointes, cardiovascular and all-cause mortality<sup>7-9</sup>. Essential hypertension prolongs the QT interval, despite the blood pressure-lowering therapy<sup>4</sup>. A prolongation of the QT interval was found in patients with untreated essential hypertension as a risk factor for ischemic heart disease<sup>10</sup>. QT interval prolongation is also present in prehypertension, independent of left ventricular mass<sup>11</sup>.

QT dispersion (QTd) is the difference between maximal and minimal QT interval duration in all measurable ECG leads. It is considered a marker of arrhythmia and sudden cardiac death risk, and a quantitative noninvasive method to determine myocardial repolarization inhomogeneities<sup>12-14</sup>, although several trials deny the prognostic value of QT dispersion<sup>15</sup>.

The QT interval consists of a short depolarization (the QRS complex) and a longer repolarization part (the ST segment and the T wave)<sup>6</sup>. Thus, the majority of the QT interval reflects ventricular repolarization, and QT prolongation, predominantly, reflects delayed ventricular repolarization.

The Tpeak-T end interval is an index of total dispersion of repolarization, a marker of ventricular arrhythmia vulnerability and a better predictor of torsades de pointes, compared to the QT interval, in patients with acquired long QT interval<sup>16,17</sup>.

Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP) are easily obtainable arterial blood pressure variables. Pulse pressure was considered a powerful predictor of cardiovascular end points<sup>1</sup>.

The present research paper aimed to evaluate the impact of blood pressure variables on repolarization parameters: QT intervals and T wave parameters, especially Tpeak-Tend interval.

## Material and Method

### Patient selection

A total of 77 consecutive patients, with pharmacologically treated essential hypertension, were enrolled in the study. A total of 56 age and sex matched consecutive controls were also included in the study ( $58 \pm 23$  years,  $p=0.24$ ), 46% males. The patients and controls underwent standard 12-lead ECG and blood pressure measurements.

Essential hypertension was diagnosed considering the criteria of the European Society of Cardiology<sup>18</sup>. Inclusion criteria for the control group were: optimal, normal and high normal blood pressure, considering the same criteria.

The most important exclusion criteria for the hypertensive group were: secondary hypertension, atrial flutter or fibrillation, bundle branch block, electrolyte imbalances, history of myocardial infarction, active infections, peripheral edema, and chronic obstructive pulmonary disease. Patients with electrocardiographic abnormalities, including myocardial ischemia and bundle branch block were excluded from the control group.

Patients, who fulfilled the mentioned criteria and provided informed written consent, were included in the study.

The investigations conformed to the principles outlined in the Declaration of Helsinki (Cardiovascular Research 1997; 35:2-4) and were approved by the Ethics Committee of the University.

### Electrocardiographic measurements

The patients underwent standard 12-lead ECG at a paper speed of 25 mm/second, using a Cardioline Delta 3 Plus ECG unit. ECGs were high-quality digital recordings and all leads were recorded simultaneously. ECG measurements were performed in a blinded fashion, by two observers. All subjects were in sinus rhythm.

QT intervals: QTmax (maximal QT interval duration in the 12 ECG leads), QTc (heart rate corrected QTmax), QTm (mean QT interval duration in all measurable leads), QTd (QT interval dispersion), QTII (QT interval duration in lead DII), QTIIc (heart rate corrected QTII), and T wave variables: T0e (maximal T wave duration), Tpe (maximal Tpeak-Tend interval) and Ta (maximal T wave amplitude) were manually measured in all ECG leads.

The methodology for QTmax, QTc, QTm, QTd, QTII, QTIIc and T wave variables was previously described<sup>4</sup>.

### Blood pressure measurements

Systolic (SBP), diastolic (DBP), mean arterial (MAP) and pulse pressure (PP) were recorded. Patients were informed to not smoke, to consume caffeinated beverages or to perform excessive

physical activity 3 hours prior to blood pressure measurements. Blood pressure was measured in the morning, at stable room temperature, two times for each patient, with a standard mercury sphygmomanometer, on the right arm, in sitting position, following a 15-minute rest. Phase I and V Korotkoff sounds were used to determine systolic and diastolic blood pressure. The average of the two consecutive blood pressure measurements was used for data analysis.

Pulse pressure was calculated as the difference between systolic and diastolic blood pressure and mean arterial pressure was estimated from systolic and diastolic blood pressure, using the following equation:  $MAP = DBP + 1/3 (SBP - DBP)^{19}$ .

#### Additional data

Additional data were obtained from medical records, including history, clinical examination, major risk factors, use of medication and laboratory tests. Smoking status was self-reported.

#### Statistical analysis

Continuous variables were presented as means  $\pm$  SD (standard deviation). Linear and multiple regression analysis, t-Student test, sensitivity and specificity were used as statistical methods. A  $p < 0.05$  was considered statistically significant.

### Results

The clinical characteristics of the hypertensive patients were included in table 1. The values for SBP, DBP, MAP and PP, QT intervals and T wave variables for hypertensive patients and the control group were included in table 2. High normal blood pressure levels (SBP: 130-139 mm Hg and DBP: 85-89 mm Hg)<sup>18</sup> were measured in 23 patients of the control group (41%). The rest of the patients of the control group had normal blood pressure<sup>18</sup> (SBP: 120-129 mm Hg and DBP: 80-84 mm Hg; 30 patients - 54%) or optimal systolic and diastolic values<sup>18</sup> (<120 mm Hg and <80 mm Hg, respectively). QT was prolonged >450 ms in 52 hypertensive patients (68%).

QTmax, QTc, QTm, QTII and QTIIc values were significantly higher in the hypertensive compared to the control group ( $p < 0.05$ ) (table 2).

#### Linear regression analysis

Linear regression analysis revealed a significant association between elevated blood pressure values (>140/90 mmHg) and QT intervals, QTd and T wave variables in hypertensive patients (table 3). QT dispersion was significantly associated with blood pressure variables, and QTIIc with SBP and PP (table 4). High normal blood pressure levels were significantly associated with prolonged QTc (>450 ms) in the control group ( $p = 0.016$ , multiple  $R = 0.315$ ,  $R^2 = 0.09$  and adjusted  $R^2 = 0.08$ ).

### Multiple regression analysis

Multiple regression analysis revealed a significant association of DBP with QTIIc and Tpe in the hypertensive group. MAP was significantly associated with QTc and QTm and PP with T0e (table 5). No significant association with QT intervals and T wave variables were found for SBP in the hypertensive group.

Multiple regression analysis also revealed significant associations between blood pressure variables and ECG parameters in the control group (table 6).

High normal blood pressure levels were significantly associated with QTmax ( $p=0.037$ ) and QTc ( $p=0.031$ ) (significance  $F<0.01$ , multiple  $R=0.691$ ,  $R\text{ square}=0.478$ , adjusted  $R=0.44$ ).

### Sensitivity and specificity of blood pressure measurements as predictors of prolonged QT intervals

The values obtained for sensitivity and specificity of blood pressure measurements as predictors of prolonged QT intervals are included in tables 7-11.

The most sensitive and specific predictor of a prolonged QTmax was  $DBP > 90\text{ mmHg}$  (table 7). The most sensitive predictor of a prolonged QTc was  $MAP \geq 105\text{ mm Hg}$ , and the most specific,  $DBP > 90\text{ mm Hg}$  (table 8).  $SBP > 140\text{ mm Hg}$  was the most sensitive and specific predictor of an increased QTd ( $> 60\text{ ms}$ ) and a prolonged QTII (table 9, 10). The most sensitive predictor of a prolonged QTIIc was  $DBP > 90\text{ mmHg}$  and specificity was highest for both  $DBP > 90\text{ mmHg}$  and  $MAP \geq 105\text{ mm Hg}$  (table 11).

## Discussion

The main findings of our study are the associations between blood pressure variables and QT intervals and T wave parameters in hypertensive patients and controls. Prolonged QT intervals can be predicted by elevated blood pressure variables.

Pulse pressure arises as a consequence of the episodic nature of cardiac contraction and the properties of arterial circulation<sup>20</sup>. It is a major determinant of cardiovascular morbidity and mortality, left ventricular mass, arterial stiffness, and rises with advancing age<sup>21, 22</sup>. Elevated pulse pressure seems to reflect arterial stiffness and atherosclerosis, while a lower pulse pressure is closely related to impaired hemodynamic and reduced cardiac output<sup>23</sup>. A significant association between pulse pressure and T0e, in the present study, demonstrates the association between arterial stiffness and T wave duration in hypertensive patients.

QT intervals and T wave variables were not significantly associated with systolic blood pressure, but elevated SBP was the most sensitive predictor of  $QTd > 60\text{ms}$ . MAP and PP reflect the combined effects of systolic and diastolic blood pressure, and they were significantly associated with QT intervals and T wave variables in the present study. As far as we know, the present study is the first attempt to evaluate the combined effect of systolic and diastolic blood pressure values on ventricular repolarization. Combination of blood pressure indices could be superior to single components in predicting sudden cardiac death risk.

Peng et al.<sup>24</sup> demonstrated a significant positive relationship between the QTc interval duration and systolic and diastolic blood pressure in a Chinese hypertensive population. SBP was also significantly associated with QTc in elderly Japanese hypertensive patients<sup>25</sup>.

Therapeutic control of arterial hypertension remains a major unsolved problem in the prevention of cardiovascular morbidity and mortality. Only 34 hypertensive patients (44%) included in the present study, achieved blood pressure targets <140/90 mmHg<sup>26</sup>. Uncontrolled hypertension was significantly associated with QT intervals and T wave variables (table 3), as a link to sudden cardiac death risk.

### Limitations

The main limitations are due to additional sources of variability of the QT interval, the use of the Bazett formula, methodological difficulties in delineating the end of the T wave, lack of standardization for Tpe and QTd and no direct arrhythmia data.

The major limitations of our studies are iatrogenic. Similar associations to those found in the hypertensive group, between blood pressure measurements and ECG intervals, were obtained in the control group, as well, and no patient of the control group was receiving any therapy.

The use of the Bazett formula results in an overcorrection of the QT interval at higher heart rates and undercorrection at lower heart rates. Nevertheless, Bazett's correction formula is very frequently used in clinical practice or research<sup>6</sup>. The relation QT-HR is very individual<sup>27</sup> and, therefore, any generally valid QTc formula will not be probably found<sup>6</sup>. The Bazett method is appropriate for the average physiological heart rate. The heart rates of the patients included in the present study were: 75±19 beats/minute in the hypertensive group versus 71±11 beats/minute in the control group. Only 14 hypertensive patients (18%) and 6 patients of the control group (11%) had elevated heart rates (higher than 85 beats/minute). Bradycardia (between 50-60 beats/minute) was detected in 10 (13%) and 7 (12.5%) patients, respectively.

The QT interval has many sources of variability, including advanced age, drugs, body mass index, diabetes mellitus<sup>28</sup>, dyslipidemia<sup>29</sup>, smoking<sup>30</sup>, heart failure, impaired renal function<sup>4</sup>. The number of patients with comorbidities was low in the present study.

Beta-blockers are considered beneficial in long QT syndromes, the first choice of therapy in patients with congenital long QT<sup>31</sup>, reduce the prevalence of cardiac events and syncope<sup>17, 32</sup>, but there is a lack of prospective, placebo-controlled studies<sup>33</sup>. Beta blockers are also effective in suppressing ventricular ectopic beats and arrhythmias and reduction of sudden cardiac death<sup>32</sup>. Discrepancies between the results of different studies were noticed: some authors consider no direct effect of beta blockers on repolarization or QT interval<sup>34</sup>, others found a QT shortening or prolonging effect<sup>35,36</sup>. It seems that heart rate and heart rate correction formulas may influence the results, and beta-blockers decrease the abrupt lengthening of both QT and Tpe intervals at elevated heart rates (higher than 85 beats/minute), whereas QT intervals measured at steady-state conditions remained unchanged<sup>37</sup>. Iatrogenic QT interval shortening is caused by angiotensin converting enzyme inhibitors<sup>38</sup> and digitalis<sup>39</sup>. Angiotensin converting enzyme inhibitors and calcium channel blockers may be also associated with left ventricular hypertrophy regression,

independent of blood pressure reduction<sup>40</sup>. QT was prolonged in 68% of the hypertensive patients, despite therapy.

Difficulties in delineating the end of the T-wave in some leads are mentioned in several articles<sup>4,41</sup>. Accurate determination of the T-wave end may be difficult when the T wave is flat or bizarre. In the present study, leads in which the end of the T wave could not be assessed were eliminated.

The clinical use of Tpe requires further validation. No large studies, using Tpe, exist and there is no clear consensus about the normal values for Tpe and QTd. Tpe values in the present study are comparable to those of other studies<sup>4,17</sup>.

Surrogate markers of ventricular arrhythmia risk were used and no direct arrhythmia data were provided. Longitudinal studies are needed to confirm the association between elevated blood pressure variables and ventricular arrhythmias and sudden cardiac death.

## Conclusion

The reported findings demonstrate that elevated systolic, diastolic blood pressure and mean arterial pressure impair ventricular repolarization, are predictors of prolonged QT intervals and elevated QT dispersion. Blood pressure variables are associated with repolarization parameters, including T wave duration and Tpeak-Tend interval.

**Conflict of Interest:** None declared.

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**Table 1:** Clinical characteristics of the hypertensive patients

<b>Clinical Characteristics</b>	
Age (mean $\pm$ SD)	62 $\pm$ 12 years
Gender	31 (40%) male
Cardiovascular risk factors:	1-2 risk factors: 8 (10%) 3 or more risk factors: 5 (6%) Established cardiovascular or renal disease: 10 (13%) Dyslipidemia: 12 (15%) Obesity: 4 (5%) Diabetes mellitus type 2: 3 (4%) Family history of premature cardiovascular disease: 15 (20%) Current smokers: 15 (19%)
Associated pathology	Congestive heart failure: 5 (6%), Coronary heart disease: 20 (26%), Chronic renal failure: 2 (3%), Anemia: 5 (6%)
Therapy	Beta-blockers: 37 (48%); Calcium channel blockers: 40 (52%); Angiotensin converting enzyme inhibitors (ACEI): 47 (61%); Digoxin 4 (5%); Diuretics: 32 (42%)

**Table 2:** ECG parameters and arterial blood pressure variables in hypertensive patients and control group

Parameter	Values in hypertensive patients (means±SD)	Values in the control group (means±SD)	p
Heart rate (beats/minute)	75±19	71±11	0.126
QTmax (ms)	430±51	380±37	<0.01
QTc (ms)	474±48	409±30	<0.01
QTm (ms)	399±43	372±22	0.03
QTd (ms)	74±35	73±60	0.877
QTII (ms)	397±51	376±27	0.04
QTIIc (ms)	437±45	401±29	<0.01
T0e (ms)	237±48	254±28	0.08
Tpe (ms)	100±26	108±12	0.14
Ta (mm)	7.47±2	5.77±2.7	0.72
SBP (mm Hg)	139±24	125±8	<0.01
DBP (mm Hg)	86±13	78±8	<0.01
MAP (mm Hg)	103±15	94±7	<0.01
PP (mm Hg)	53±16	47±8	0.069

QTmax = maximal QT interval duration in all measurable leads, QTc = heart rate corrected, QTm = the mean QT interval duration in all leads, QTd = QT interval dispersion, QTII = the QT interval in lead DII, QTcII = heart rate corrected QT interval in lead DII, T0e = maximal T-wave duration in all measurable leads, Tpe = maximal Tpeak-Tend interval in all measurable leads, Ta = maximal T-wave amplitude in all leads, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, PP = pulse pressure.

Intervals are expressed as means ± SD (standard deviation). P<0.05 was considered significant.

**Table 3:** Linear regression analysis. Parameters significantly associated with elevated blood pressure values (>140/90 mmHg) in the hypertensive group

Parameter	p	Multiple R	R square	Adjusted R
QTmax	<0.01	0.734	0.539	0.526
QTc	<0.01	0.741	0.549	0.536
QTm	<0.01	0.737	0.543	0.530
QTd	<0.01	0.706	0.498	0.485
QTII	<0.01	0.734	0.539	0.526
QTIIc	<0.01	0.740	0.548	0.535
T0e	<0.01	0.718	0.516	0.503
Tpe	<0.01	0.725	0.526	0.513
Ta	0.0079	0.298	0.089	0.076

QTmax = maximal QT interval duration in all measurable leads, QTc = heart rate corrected, QTm = the mean QT interval duration in all leads, QTd = QT interval dispersion, QTII = the QT interval in lead DII, QTcII = heart rate corrected QT interval in lead DII, T0e = maximal T-wave duration in all measurable leads, Tpe = maximal Tpeak-Tend interval in all measurable leads, Ta = maximal T-wave amplitude in all leads, multiple R = multiple correlation coefficient, R square = coefficient of determination, adjusted R = the coefficient of determination adjusted for the number of independent variables in the regression model

**Table 4:** Linear regression analysis. Parameters significantly associated with QTd and QTIIc in hypertensive patients

Parameter	associated with	p	Multiple R	R square	Adjusted R
QTd	SBP	<0.01	0.905	0.819	0.806
	DBP	<0.01	0.903	0.816	0.803
	MAP	<0.01	0.905	0.82	0.807
	PP	<0.01	0.883	0.78	0.767
QTIIc	SBP	<0.01	0.982	0.964	0.951
	PP	<0.01	0.954	0.91	0.897

QTcII = heart rate corrected QT interval in lead DII, QTd = QT dispersion, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, PP = pulse pressure, multiple R = multiple correlation coefficient, R square = coefficient of determination, adjusted R = the coefficient of determination adjusted for the number of independent variables in the regression model

**Table 5:** Multiple regression analysis in hypertensive patients

Arterial blood pressure variable	Associated with	Multiple R	R square	Adjusted R	Significance (F)
DBP	QTIIc (p<0.01) Tpe (p<0.01)	0.985	0.971	0.957	<0.01
MAP	QTc (p<0.001) QTm (p=0.014)	0.986	0.972	0.958	<0.01
PP	T0e (p<0.01)	0.944	0.891	0.878	<0.01

DBP = diastolic blood pressure, MAP = mean arterial pressure, PP = pulse pressure, QTc = heart rate corrected, QTm = the mean QT interval duration in all leads, QTcII = heart rate corrected QT interval in lead DII, T0e = maximal T-wave duration in all measurable leads, Tpe = maximal Tpeak-Tend interval in all measurable leads, multiple R = multiple correlation coefficient, R square = coefficient of determination, adjusted R = the coefficient of determination adjusted for the number of independent variables in the regression model

**Table 6:** Multiple regression analysis in the control group

Arterial blood pressure variable	Associated with	Multiple R	R square	Adjusted R	Significance (F)
SBP	QTmax (p<0.01)  QTc (p=0.01)  HR (p<0.01)	0.997	0.995	0.976	<0.01
DBP	QTmax (p<0.01)  QTc (p=0.165)  HR (p<0.01)	0.995	0.990	0.971	<0.01
MAP	QTmax (p<0.01)  QTc (p<0.01)  HR (p<0.01)	0.997	0.994	0.975	<0.01

QTmax = maximal QT interval duration in all measurable leads, QTc = heart rate corrected, HR = heart rate, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, multiple R = multiple correlation coefficient, R square = coefficient of determination, adjusted R = the coefficient of determination adjusted for the number of independent variables in the regression model

**Table 7:** Sensitivity and specificity of arterial pressure variables as predictors of prolonged QTmax (QTmax  $\geq$  450 ms) in hypertensive patients

	<b>Sensitivity (95% CI)</b>	<b>Specificity (95% CI)</b>
SBP > 140 mm Hg	0.5 (0.29-0.71)	0.67 (0.53-0.79)
DBP > 90 mm Hg	0.95 (0.72-0.99)	0.81 (0.68-0.89)
MAP $\geq$ 105 mm Hg	0.34 (0.18-0.52)	0.59 (0.43-0.73)

SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, CI = confidence interval

**Table 8:** Sensitivity and specificity of arterial pressure variables as predictors of prolonged QTc (QTc  $\geq$  450 ms) in hypertensive patients

	<b>Sensitivity (95% CI)</b>	<b>Specificity (95% CI)</b>
SBP > 140 mm Hg	0.45 (0.25-0.67)	0.24 (0.14-0.37)
DBP > 90 mm Hg	0.58 (0.33-0.79)	0.29 (0.18-0.43)
MAP $\geq$ 105 mm Hg	0.59 (0.41-0.76)	0.27 (0.15-0.42)

SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, CI = confidence interval

**Table 9:** Sensitivity and specificity of arterial blood pressure variables as predictors of increased QTd (QTd >60 ms)

	<b>Sensitivity (95% CI)</b>	<b>Specificity (95% CI)</b>
SBP > 140 mm Hg	0.64 (0.41-0.82)	0.44 (0.31-0.58)
DBP > 90 mm Hg	0.63 (0.39-0.83)	0.43 (0.30-0.57)
MAP $\geq$ 105 mm Hg	0.63 (0.44-0.78)	0.44 (0.29-0.59)

SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, CI = confidence interval

**Table 10:** Sensitivity and specificity of arterial pressure variables as predictors of prolonged QTII (QTII  $\geq$  450 ms) in hypertensive patients

	<b>Sensitivity (95% CI)</b>	<b>Specificity (95% CI)</b>
SBP > 140 mm Hg	0.27 (0.12-0.50)	0.91 (0.79-0.97)
DBP > 90 mm Hg	0.26 (0.10-0.51)	0.89 (0.78-0.96)
MAP $\geq$ 105 mm Hg	0.19 (0.08-0.37)	0.89 (0.75-0.96)

SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, CI = confidence interval

**Table 11:** Sensitivity and specificity of arterial pressure variables as predictors of prolonged QTIIc (QTIIc  $\geq$  450 ms) in hypertensive patients

	<b>Sensitivity (95% CI)</b>	<b>Specificity (95% CI)</b>
SBP > 140 mm Hg	0.36 (0.18-0.59)	0.69 (0.55-0.80)
DBP > 90 mm Hg	0.42 (0.21-0.66)	0.71 (0.57-0.82)
MAP $\geq$ 105 mm Hg	0.38 (0.22-0.56)	0.71 (0.55-0.83)

SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, CI = confidence interval