Prognostic value of arrhythmogenic markers in systemic hypertension


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Objectives To evaluate the prognostic value of arrhythmogenic markers in hypertensive patients.

Design Two hundred and fourteen hypertensive patients without symptomatic coronary disease, systolic dysfunction, electrolyte disturbances or anti-arrhythmic therapy were included. Recordings were made of 12-lead standard ECGs with calculations of QT interval dispersion, 24 h Holter ECGs (204 patients), echocardiography (187 patients) and signal-averaged ECGs (125 patients).

Results Baseline data: echocardiographic left ventricular hypertrophy was found in 63 patients (33.7%), non-sustained ventricular tachycardia (Lown class IVb) in 33 patients (16.2%), ventricular late potentials in 27 patients (21.6%). Mortality: after a mean follow-up of 42.4 ± 26.8 months, global mortality was 11.2% (24 patients), cardiac mortality 7.9% (17 patients), sudden death 4.2% (nine patients). Univariate analysis: predictors of global, cardiac and sudden death were age ≥65 years, ECG strain pattern, Lown class IVb and QT interval dispersion >80 ms (P<0.01). Left ventricular mass index was closely related to cardiac mortality (P=0.002). Multivariate analysis: only Lown class IVb was an independent predictor of global (RR 2.6, 95% CI 1.2-6.0) and cardiac mortality (RR 3.5, 95% CI 1.2-9.7).

Conclusion In hypertensive patients, non-sustained ventricular tachycardia has a prognostic value.

Keywords: Hypertension, left ventricular hypertrophy, QT interval dispersion, 24 h Holter ECG, mortality.

Introduction

Left ventricular hypertrophy in hypertensive subjects carries a negative prognosis, being a recognized harbinger of ventricular arrhythmias and mortality. The initial reports of the Framingham Heart Study showed the deleterious effects on survival of left ventricular hypertrophy as demonstrated by electrocardiography and echocardiography.[3] Since then much effort has been put into evaluating mortality and morbidity risk factors in hypertension, as isolated hypertension increases sudden cardiac death risk threefold.[9] In the absence of coronary heart disease, which itself carries a negative survival prognosis, the probable cause of sudden cardiac death in hypertensive patients remains ventricular arrhythmias. Data from the same Framingham study showed that subjects with echocardiographic left ventricular hypertrophy and asymptomatic ventricular arrhythmias had a higher mortality, so that the latter should be considered an ominous sign when recorded on Holter ECGs[3].

Multiple risk markers for an arrhythmic substrate have been identified in different pathological cardiovascular conditions and which can be applied to hypertensive heart disease. Ventricular late potentials,[6-9] diminished heart rate variability[8] and, recently identified in hypertension, QT interval dispersion[10] have been evaluated for arrhythmia risk with controversial results. However, a prognostic evaluation of these markers has not been realized as yet in systemic hypertension. In the present prospective study, we evaluated the prognostic value of ventricular arrhythmias, QT interval dispersion and ventricular late potentials in hypertensive subjects according to the degree of left ventricular hypertrophy.

Methods

Patients

We included 214 hypertensive patients (127 men and 87 women), 59.11 ± 12.87 years old, admitted to the...
Table 1  General features of the 214 hypertensive patients

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Means ± SD</th>
<th>Characteristics</th>
<th>n</th>
<th>%</th>
</tr>
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<tbody>
<tr>
<td>Age (years)</td>
<td>59.11 ± 12.87</td>
<td>Bundle branch block</td>
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<tr>
<td>BMI (kg. m^2)</td>
<td>26.98 ± 4.64</td>
<td>Diuretics</td>
<td>81</td>
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<tr>
<td>LV mass (g)</td>
<td>211.73 ± 63.38</td>
<td>Beta-blockers</td>
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<td>25.7</td>
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<tr>
<td>LV mass index (g. m^2)</td>
<td>115.94 ± 33.33</td>
<td>Alpha-blockers</td>
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<tr>
<td>Ejection fraction (%)</td>
<td>75 ± 7.47</td>
<td>ACE inhibitors</td>
<td>106</td>
<td>49.53</td>
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<tr>
<td>Shortening fraction (%)</td>
<td>37.5 ± 6.71</td>
<td>Central</td>
<td>19</td>
<td>8.87</td>
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<tr>
<td>Kalaemia (mmol. 1⁻¹)</td>
<td>4.09 ± 0.4</td>
<td>Ca²⁺ blockers</td>
<td>113</td>
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</tr>
<tr>
<td>Cholesterolaemia (mmol. 1⁻¹)</td>
<td>4.48 ± 1.17</td>
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<tr>
<td>Trglycerides (mmol. 1⁻¹)</td>
<td>1.75 ± 1.14</td>
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BMI = body mass index; LV = left ventricular; ACE inhibitors = angiotensin converting enzyme inhibitors.

Cardiology Department of the Rangueil University Hospital between 1987 and 1993. All patients had primary systolic-diastolic systemic hypertension (systolic and diastolic arterial blood pressure >160/95 mmHg). Most of the patients were in sinus rhythm (209 i.e. 98.1%); five patients were in atrial fibrillation. General features of the study group are displayed in Table 1.

We excluded patients with secondary hypertension to avoid the potential influence of the cause of hypertension on proarrhythmic markers (i.e. QT interval prolongation induced by hypokalaemia in primary hyperaldosteronism). Hypertensive patients with symptomatic coronary disease and post infarction patients were also excluded because of the well known influence of myocardial ischaemia on the incidence of ventricular arrhythmias and other prognostic markers, such as ventricular late potentials. Patients with systolic left ventricular dysfunction, expressed by an echocardiographic ejection fraction less than 50%, were not enrolled, to eliminate the influence of diminished cardiac performance on the same arrhythmia risk indicators. Finally, electrolyte disturbances (serum potassium <3.5 mmol. 1⁻¹ or serum magnesium <0.7 mmol. 1⁻¹) and anti-arrhythmic treatment at the time of initial evaluation were considered exclusion criteria. Anti-hypertensive treatment was not considered an exclusion criterion at enrolment. The body mass index was calculated (mean 26.98 ± 4.64) and considered to be pathological if it was greater than 27 in men and 25 in women, but was not an exclusion criterion.

Potentially significant prognostic factors:

Surface 12 lead standard ECGs (25 mm. s⁻¹, 10 mm. mV⁻¹) were obtained in all patients for the diagnosis of left ventricular hypertrophy (Sokolow index ≥35 mm with or without strain pattern). The QT interval was measured manually by the same investigator in two adjacent cycles in each ECG lead, averaged and corrected for heart rate using Bazett's formula. The end of the T wave was defined by the intersection between the tangent of the descending part of the T wave and the baseline, and the U wave was excluded. QT interval dispersion was calculated as the difference between the longest and the shortest QT interval measured in each individual ECG lead. QT interval dispersion was considered pathological if it exceeded 80 ms, defined by previous studies as the superior limit in normal subjects.11 12

Echocardiographic examination was performed on a Hewlett-Packard SONOS 1000 machine and considered to be technically adequate in 187 patients. Left ventricular end-systolic and end-diastolic dimensions (LVID), and septal (SWT) and posterior wall (PWT) thickness were measured in accordance with the American Society of Echocardiography recommendations. Left ventricular volumes were calculated using the cube formula and left ventricular ejection using the Teicholz regression formula. Left ventricular mass was calculated using the Devereux formula:13

\[ \text{LV mass} = 0.8 \times \left[10 \times \left(\text{LVID} + \text{SWT} + \text{PWT}\right)^3 - \left(\text{LVID}\right)^3\right] + 0.6 \text{ g}. \]

Left ventricular mass index was calculated by dividing the left ventricular mass by body surface area. Left ventricular hypertrophy was considered when left ventricular mass index exceeded 134 g. m⁻² in men and 110 g. m⁻² in women.14

Twenty-four hour Holter ECG recordings were obtained from 204 patients, using a Marquette Electronics 8000 system. Premature ventricular beats, polymorphism, ventricular doublets and non-sustained ventricular tachycardia were noted and classified according to the Lown criteria.

Signal-averaged electrocardiograms were recorded in 125 patients, enrolled after 1988, on a Marquette MAC 15 machine, using about 300 QRS cycles and a high-pass digital filter of 40 Hz; a noise level of less than 0.4 uV was considered as acceptable. Ventricular late potentials were considered to be present if at least two of the following criteria existed: filtered QRS duration >120 ms; root-mean square voltage in the last 40 ms <20 uV; low-amplitude signals less than 40 uV >40 ms. Signal-averaged ECGs were considered not interpretable in patients with bundle branch block.

Follow-up

All-cause and cardiac mortality were considered as primary end-points; in the cardiac death group, patients...
with sudden death (death occurring less than 1 h after the onset of symptoms) were considered separately. Survival data were obtained by direct patient examination or from the general practitioners of the enrolled patients.

Statistical analysis

Quantitative values are reported as mean ± SD. Continuous variables (QT interval dispersion, ventricular premature beats, left ventricular mass index) were treated by linear regression and the correlation coefficient was calculated. Mean QT interval dispersion for every Lown class was tested by analysis of variance. When significant data emerged, the mean values were treated in pairs by Fisher’s PLSD test. The Student’s t-test for independent series was used to compare the mean QT interval dispersion values in relation to presence or absence of left ventricular hypertrophy or ventricular late potentials. The Chi-squared test was used to compare the distribution of Lown classes in relation to the presence of ventricular late potentials. The results of the tests are considered significant if a $P$ value of less than 0.05 is obtained.

Survival analysis

Survival time estimates were calculated by the method of Kaplan–Meier and statistical comparisons between survival curves were done using the log-rank test. The significance of each categorical variable was determined by a $P \leq 0.05$. The following continuous variables were transformed into dichotomized variables: age <65 or age ≥65 as a mean of social inactivity; the presence of left ventricular hypertrophy if the left ventricular mass index was >110 g·m² in women and >134 g·m² in men; the presence of abnormal QT duration if >80 ms; the presence or absence of ventricular late potentials; serum kalaemia >4 mosm·L⁻¹ or ≤4; serum cholesterol >6-2 or ≤6-2 mosm; triglycerides >1-7 or ≤1-7. The relative risk and the significance for each categorical variable were assessed using a discrete Cox model. Multivariate survival analysis was performed with the Cox proportional hazards model to determine which factors were significantly associated with global or cardiac death, after adjustment for the other variables. Because the number of events was not sufficient, sudden death was not treated in multivariate analysis. Variables selected to be tested in multivariate analysis were those with a $P \leq 0.05$ in the univariate model. A stepwise selection was done using a $P$ to remove from and a $P$ to enter into the model ≤0.05, with both prior backward selection after inclusion of all selected variables (saturated model) and then forward selection. The $P$ value refers to the likelihood ratio test of the hypothesis that the regression coefficient was zero. Results are expressed as relative risk with confidence intervals (CI 95%). A significant increase of risk is obtained if CI 95% excludes 1 and $P$ of Wald test ≤0.05 (computed with the Statview package).

Results

Baseline descriptive data

Electrocardiographic data

Left ventricular hypertrophy with a strain pattern was found in 42 patients (19.6%). When only electrical amplitude criteria were used to define left ventricular hypertrophy, this was identified in 64 patients (29.9%). The mean QT interval dispersion was 57.3 ± 32.9 ms and varied between 20 and 175 ms, the median value was 47.5 ms. The cut-off point of 80 ms corresponded to the value superior to the 75% percentile of QT interval dispersion distribution measured in our population. Left ventricular hypertrophy with a strain pattern, as revealed by electrocardiography, was closely related to QT interval dispersion ($P < 0.0001$).

Echocardiographic data

Mean left ventricular dimensions were normal in the study group (end-diastolic diameter was 48.6 ± 5.1 mm; end-systolic diameter 30.4 ± 5.2 mm). The mean ejection fraction was 75% with a lower limit of 54%. The mean shortening fraction was 37.5% with a lower value of 22.7%. Echocardiographic left ventricular hypertrophy was found in 63 patients (33.7%) compared to 124 patients (66.3%) with normal values for their gender. Significant correlations between QT interval dispersion and left ventricular mass index ($r = 0.34, P < 0.0001$), the thickness of the interventricular septum ($r = 0.37, P < 0.0001$) and the posterior wall ($r = 0.35, P < 0.0001$), and the left ventricular end-diastolic dimension ($r = 0.20, P < 0.0001$) were obtained. QT interval dispersion was significantly higher in patients with left ventricular hypertrophy (74.4 ± 36.9 ms) than in patients without left ventricular hypertrophy (50.5 ± 28.4 ms, $P < 0.0001$).

Holter data

The absolute number of ventricular premature beats in Holter ECG recordings varied between 0 and 18 500 with a mean of 757 ± 2464/24 h. When classified according to the Lown criteria, 27 patients (13.2%) were in class 0, 99 (48.5%) in class I, seven (3.4%) in class II, 11 (5.4%) in class III, 27 (13.2%) in class IVa and 33 (16.2%) in class IVb. QT interval dispersion showed a weak correlation with the absolute number of ventricular premature beats ($r = 0.18, P = 0.011$), a better correlation using the log of the number of ventricular premature beats plus one ($r = 0.39, P < 0.0001$), and a correlation with the Lown classes ($r = 0.41, P < 0.0001$) (Fig. 1). Lown classes were correlated with the left ventricular mass index ($r = 0.25, P = 0.001$).

Ventricular late potentials were found in 27 patients (21.6%), compared to 98 patients (78.4%) in whom they were considered as absent. Left ventricular late potentials were found in 27 patients (21.6%), compared to 98 patients (78.4%) in whom they were considered as absent. Left ventricular late potentials were found in 27 patients (21.6%), compared to 98 patients (78.4%) in whom they were considered as absent. Left ventricular late potentials were found in 27 patients (21.6%), compared to 98 patients (78.4%) in whom they were considered as absent.
mass index was not significantly different in patients with or without ventricular late potentials. The severity of ventricular arrhythmias was weakly related to the presence of ventricular late potentials ($P=0.02$). QT interval dispersion duration was not statistically different in patients with (58.6 ± 34.5 ms) or without ventricular late potentials (60.4 ± 24.3 ms).

**Survival data**

The 214 patients were followed for a mean of 42.4 ± 26.8 months. All-cause mortality was 11.2%, 24 patients dying of cardiac or non-cardiac causes. Total cardiac mortality was 7.94% (17 patients), because of sudden death (nine patients), acute myocardial infarction or severe acute heart failure. Among the initial 214 patients included in the study, for 14 patients (6.5%) no follow-up data could be obtained, even from the general practitioner or directly from the patient himself; these patients were excluded from the survival analysis and considered as lost from the study.

**Univariate survival analysis (Table 2)**

Age was a significant predictor of mortality, patients older than 65 years having a greater incidence of all-cause (Chi-squared = 17.63), cardiac (Chi-squared = 10.50) and sudden death (Chi-squared = 9.06). Electrocardiographical left ventricular hypertrophy with a strain pattern was closely related to all-cause mortality (Chi-squared = 19.89), cardiac (Chi-squared = 20.95) and sudden death (Chi-squared = 13.6). No significant relationship was found between isolated high amplitude QRS and global, cardiac or sudden death. QT interval dispersion longer than 80 ms was significantly related to global (Chi-squared = 7.39), cardiac (Chi-squared = 14.22) and sudden death (Chi-squared = 5.03) (Fig. 2). Patients with non-sustained ventricular tachycardia (Lown class IV b) showed a significant increase of all cause mortality (Chi-squared = 9.17), cardiac (Chi-squared = 9.21) and sudden death (Chi-squared = 12.55) (Fig. 3). Echocardiographic left ventricular hypertrophy was closely related to total cardiac mortality (Chi-squared = 9.23) but failed to correlate with all-cause mortality and sudden death.

Ejection fraction, as demonstrated by echocardiography, lacked significance for mortality (this could be easily explained by the inclusion criteria, because only patients with normal systolic function were considered eligible in the study). No difference in all cause mortality was found in the 27 patients with ventricular late

![Figure 1 Relationship between QT interval dispersion (QTd) and the Lown classes (LC 0, LC I, LC II, LC III, LC IV A, LC IV B). The QTd was significantly shorter in LC 0 to LC II than in LC III to LC IV B ($P<0.0001$).](image-url)
Figure 2 Kaplan–Meier cumulative survival plot for cardiac death: QT interval dispersion (QTd). QTd ≥ 80 ms was significantly related to cardiac death (P = 0.0002). —— = cumulative survival QTd ≥ 80 ms, ○ = event times; —— = cumulative survival QTd < 80 ms, □ = event times.

Figure 3 Kaplan–Meier cumulative survival plot for global death: Lown classes. Lown class IV b was significantly related to global death (P = 0.0025). —— = cumulative survival Lown class IV b, ○ = event times; —— = cumulative survival Lown classes 0 to IV a, □ = event times.

Among the treatments, the use of a diuretic, of an angiotensin converting enzyme inhibitor or of a beta-blocker was associated with a significant change in mortality risk.

Multivariate survival analysis

After testing the appropriateness of the Cox regression model for the different mortality classes, we selected for multivariate survival analysis different parameters which showed a significant relationship with mortality in univariate analysis: age, Lown classes, QT interval dispersion, left ventricular mass index, diuretics, angiotensin converting enzyme inhibitors and beta-blocker...
treatments. Because of an extremely close relationship between QT interval dispersion and the strain pattern of left ventricular hypertrophy, this latter was excluded from the analysis. For all-cause mortality, only age and Lown class IV b were retained as independent predictors of global mortality. An age greater than 65 years multiplied death risk by 4·9 (95% CI 1·8–13·6). A Lown class IV b increased the risk of death 2·6-fold (95% CI 1·2–6·0). For cardiac death, only echocardiographic left ventricular hypertrophy and Lown class IV b were retained as independent predictors of cardiac death. An increased left ventricular mass index multiplied cardiac death risk by 4·2 (95% CI 1·3–13·2). A Lown class IV b increased the cardiac death risk 3·5-fold (95% CI 1·2–9·7).

Discussion

The increased risk of cardiac death in hypertensive patients reflects the result of the interaction of pathological consequences of left ventricular hypertrophy and coronary artery disease. The Framingham study showed that left ventricular hypertrophy, as demonstrated by ECG and echocardiography, is associated with an increased risk of death in asymptomatic subjects. Although the cardiac death risk induced by left ventricular hypertrophy is independent of coronary heart disease, silent or clinically manifest, we cannot exclude an impact of coronary artery disease in our data. The absence of a history of coronary events in our patients does not exclude the possibility that coronary artery disease interacts with left ventricular hypertrophy in the genesis of ventricular arrhythmias and cardiac mortality. In fact, in the absence of stress imaging studies in our patients, we cannot eliminate the existence of silent ischaemia at inclusion. Silent ischaemia is an independent predictor for the development of ventricular arrhythmias in hypertensive patients and may be a predictor for sudden death. Whatever the mechanisms involved in the genesis of ventricular arrhythmias in hypertension, there is no clear evidence yet that the ventricular ectopic activity seen in hypertensive patients is a marker for sudden death.

Arrhythmogenic markers in systemic hypertension

By the concomitant study of pro-arrhythmic markers, we showed that some of these parameters are strongly correlated, such as abnormal QT interval dispersion and advanced Lown classes. These two arrhythmogenic markers can be considered as interdependent phenomena, cause (abnormal QT interval dispersion) and effect (ventricular ectopy). Furthermore, these two parameters are closely related to the presence of left ventricular hypertrophy. Advanced Lown classes are also weakly correlated with the presence of ventricular late potentials. In contrast, abnormal QT interval dispersion and the presence of ventricular late potentials are not correlated. Differences in the mechanism generating these two electric phenomena may be responsible for this lack of correlation: a fragmentation of final depolarization for late potentials and an inhomogeneity of repolarization for QT interval dispersion.

In the Framingham reports, the presence of echocardiographic left ventricular hypertrophy increased the relative arrhythmic risk up to 8·9 in men and to 4·6 in women; an almost identical risk was identified for electrocardiographic left ventricular hypertrophy. Multiple subsequent clinical studies confirmed the association between left ventricular hypertrophy and ventricular arrhythmias identified on the ECG or at echocardiography in systemic hypertension. A clear connection was established between elevated QT interval dispersion and ventricular fibrillation in patients with acute myocardial infarction or hypertrophic cardiomyopathy. In a recent study no relationship was identified between QT interval dispersion and ventricular arrhythmias in hypertensive left ventricular hypertrophy, even if QT interval dispersion was significantly increased in the presence of hypertrophy or heart failure. However, a subsequent clinical report identified a significant relationship between left ventricular mass index, systolic blood pressure and QT interval dispersion value in hypertensive subjects at greater risk of sudden death, independent of electrocardiographic left ventricular hypertrophy. As a marker of arrhythmic propensity, ventricular late potentials were identified in hypertensive left ventricular hypertrophy, showing a good correlation with ventricular arrhythmias or abnormal Doppler filling patterns. However, only in a small clinical study have they been found associated with syncope, aborted sudden cardiac death, documented ventricular tachycardia, or fibrillation in hypertensive heart disease.

Prognostic value of arrhythmogenic markers in systemic hypertension

Data from the same Framingham study showed that subjects with echocardiographic left ventricular hypertrophy and asymptomatic ventricular arrhythmias, considered as an ominous sign when recorded on Holter ECGs, had a higher mortality. However, in systemic hypertension, the prognostic importance of arrhythmogenic markers has not yet been evaluated. In the present study, we demonstrated that in univariate analysis Lown class IV b and abnormal QT interval dispersion were significantly related to global, cardiac and sudden death in hypertensive patients. In contrast, ventricular late potentials failed to predict mortality. In univariate and multivariate analyses echocardiographic left ventricular hypertrophy is also an independent predictor of cardiac mortality. Finally, in multivariate analysis, only Lown class IV b is an independent predictor of global and cardiac mortality, emphasizing the
prognostic value of high risk ventricular arrhythmias in hypertensive patients. For sudden death not enough events occurred to ensure sufficient statistical power.

**Limits of the study**

One limit of our study is the possible impact of antihypertensive treatment on arrhythmogenic markers and sudden death. Since this was not a randomized study, no conclusion concerning the effects of the drugs was possible. We must therefore underline that the increase in mortality associated with the use of an angiotensin converting enzyme inhibitors is explained by the left ventricular mass index (123 ± 36 vs 112 ± 30 g.m², \( P=0.03 \)) and thus the higher degree of hypertension in patients treated with this drug. The increase in mortality associated with the use of a diuretic could be, in part, due to a pro-arrhythmic effect. In fact, if compared with patients without diuretic, patients treated with diuretics were significantly older (64 ± 12 vs 56 ± 12 years; \( P<0.0001 \)), had a more enhanced left ventricular mass index (123 ± 36 vs 112 ± 30 g.m²; \( P=0.03 \)), greater QT interval dispersion (68 ± 32 vs 51 ± 31 ms; \( P=0.0002 \)) and a higher frequency of Lown classes IV b (231 vs 11-9%; \( P<0.05 \)). Large hypertensive trials provided evidence that the benefit of the antihypertensive effect of potassium-sparing diuretics could be overwhelmed by an excess risk of sudden cardiac death in treated patients[27,28]. In contrast the use of beta-blockers was associated in our study with a decrease in mortality which cannot be explained by the characteristics of patients treated with this drug. Metoprolol was previously found to reduce the incidence of sudden death when compared to thiazide diuretics[29]. Other antihypertensive agents such as calcium blockers are associated with diminished ventricular arrhythmias when compared to diuretics, but their influence on survival is still under discussion[30]. The arrhythmogenic effect of treatment was emphasized by a recent study which showed that in hypertensive subjects with left ventricular hypertrophy only patients under long-term antihypertensive treatment had significantly increased complex ventricular arrhythmias compared to non-treated subjects[31].

Furthermore, we did not study the possible influence on survival of regression of left ventricular hypertrophy by the different antihypertensive treatments. In fact, reduction of cardiac hypertrophy was found to be accompanied by a reduction of ventricular arrhythmias if treated with beta-blockers, calcium entry blockers or angiotensin converting enzyme inhibitors[32]. Experimental studies showed that regression of hypertrophy was associated with normalized ventricular electrophysiology and reduced arrhythmia risk[33]. There is still no evidence that left ventricular regression per se will lead to improved cardiovascular prognosis.

**Conclusion**

The present study demonstrates the prognostic value of two arrhythmogenic markers in systemic hypertension:

- abnormal QT interval dispersion and an advanced Lown class, which are closely related to the presence of left ventricular hypertrophy. In multivariate analysis, only a Lown class IV b is an independent predictor of global and cardiac mortality, increasing the risk of death about three fold. Among hypertensive patients with left ventricular hypertrophy, the presence of non-sustained ventricular tachycardia on ECG Holter individualizes patients at high risk of mortality needing more intensive care.

We gratefully acknowledge the expert technical assistance of Nicole Murcia.

**References**

Arrhythmogenic markers in hypertension


