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ORIGINAL ARTICLE

The spatial QRS-T angle as a marker of ventricular repolarisation in hypertension

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Ventricular repolarisation abnormalities are important in arrhythmia provocation. The dispersion of repolarisation duration is not the only aspect of repolarisation heterogeneity. Spatial vectorcardiographic descriptors constitute a novel approach to quantify ventricular repolarisation. To test the ability of vectorcardiographic descriptors to discriminate between hypertensives with high or low blood pressure (BP), 110 treated hypertensives (mean age 63.6 ± 12.1 years) were classified in the high (systolic BP ⩾160 mm Hg or diastolic BP ⩾95 mm Hg) (n = 67), or the low (systolic BP < 160 mm Hg and diastolic BP <95 mm Hg) (n = 43) BP group. The maximum QT, JT, and T peak–T end intervals and the QT, JT, and T peak–T end dispersion were calculated from a digitally recorded 12-lead electrocardiogram (ECG). X, Y, and Z leads were reconstructed from the 12-lead ECG. The amplitude of the maximum spatial T vector (spatial T amplitude), the angle between the maximum spatial QRS and T vectors (spatial QRS-T angle) and the frontal plane QRS-T angle were calculated. The spatial QRS-T angle was higher in patients with high compared to those with low BP (P = 0.025). All conventional ECG markers of the dispersion of ventricular repolarisation duration failed to demonstrate significant differences between hypertensives with high or low BP. In conclusion, the spatial QRS-T angle was significantly increased in those treated hypertensive patients who showed repeatedly high BP values. Hence, we may suggest that the angle between the directions of ventricular depolarisation and repolarisation is a sensitive marker of the repolarisation alterations in systemic hypertension.
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Introduction

Systemic hypertension affects up to 25% of the adult population and is widely recognised to be a potent cardiovascular risk factor.[1] Left ventricular hypertrophy in hypertensive patients results in inhomogeneity of ventricular repolarisation, favouring the propensity to ventricular tachyarrhythmias.[2] The variability in the QT interval duration among the different leads of a surface 12-lead electrocardiogram (ECG) (QT dispersion) is supposed to reflect local differences in the recovery time of the myocardium.[3] High QT and JT dispersion values have been previously reported in patients with systemic hypertension.[1,2,4,5] Several studies have postulated that QT dispersion values decrease when adequate blood pressure (BP) control is achieved.[5,6] However, well-known difficulties in defining the end of the T wave, the inability to measure the QT interval in all leads, and the possible presence of U waves may contribute to poor reproducibility of QT dispersion and, hence, may reduce its power to assess arrhythmia risk prospectively.[3,7]

The dispersion of repolarisation duration seems to consist only one aspect of the repolarisation inhomogeneity. Several studies have already attempted to quantify the spatial aspects of ventricular repolarisation.[8–11] The spatial aspects of the T wave complexity have been evaluated by the so-called principal component analysis,[8] while T axis has been reported to be a strong risk indicator of cardiac events in the elderly.[9] Furthermore, a recent study concluded that QT dispersion is largely determined by the T-loop morphology, as expressed by T-loop amplitude and width,[10] whereas an older one has reported a widened QRS-T angle in patients with eccentric left ventricular hypertrophy.[11]

The objective of the present study was to evaluate several temporal and spatial descriptors of ventricular repolarisation in a population of treated hypertensive patients. In particular, our study investigated the ability of repolarisation descriptors to discriminate between hypertensive patients with high or low measured BP values.

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Methods

Study population

The study population consisted of 110 consecutively recruited patients (62 women; mean age 63.6 ± 12.1 years) with history of systemic hypertension. All patients received antihypertensive therapy and no one had any other risk factors for coronary artery disease. All patients underwent physical examination, 12-lead ECG, and serial blood tests. Apart from systemic hypertension, no other cardiovascular disease was present in the study population. All patients were in sinus rhythm. Routine medications were not withheld during patients evaluation.

Excluded from the study were patients with left or right bundle branch block, atrioventricular block, ventricular pre-excitation, history of coronary artery disease, atrial fibrillation, sick sinus syndrome, prior pacemaker implantation, clinically overt heart failure (New York Heart Association (NYHA) classes II-IV), or pericarditis. Patients receiving digitalis or any anti-arrhythmic drugs were also excluded.

Repeated BP measurements performed in the outpatient clinic were used to stratify the study patients according to their BP levels. The average from the BP measurements obtained in three different patient visits within the same week was used for this purpose. Patients were categorised in the high BP group when the average systolic BP was $\geqslant$160 mm Hg or diastolic BP $\geqslant$95 mm Hg and in the low BP group when the average systolic BP was $<$160 mm Hg and diastolic BP $<$95 mm Hg. The study was approved by the local ethics committee and informed consent was obtained from all participants.

Twelve-lead surface electrocardiogram

In all subjects, a 12-lead digital ECG was recorded in the supine resting position using a computer-based electrocardiograph (Cardioperfect, version 1.1, CardioControl NV, Rijswijk, The Netherlands). All 12 leads of each ECG were recorded simultaneously for 20 seconds and sampled at a rate of 1200 Hz. From each lead, the average complex was calculated by the MEANS (Modular ECG Analysis) system.[12] These average complexes, sampled at 300 Hz, were used in the analysis. Left ventricular hypertrophy was electrocardiographically defined when at least one of the following three criteria was positive: $S_{V3} + R_{aVL} > 2.4$ mV in men or >2.0 mV in women, a typical strain pattern, or a Romhilt-Estes score $\geqslant$5.[13] The presence of discordant T waves in those patients who fulfilled the ECG criteria for left ventricular hypertrophy was assessed in all the 12 leads of each ECG in both study groups.

QT, JT, and T peak–T end interval measurements

All QT, JT, and T peak–T end interval measurements were performed manually using the digitally stored ECGs displayed on a high-resolution computer screen. Each lead was separately magnified (160 mm/s and 60 mm/mV). On screen calipers were used to define the start and end of all the aforementioned ECG intervals separately. The QT interval was measured from the onset of the QRS complex to the end of the T wave. The point of T-wave offset was defined as the return to the baseline.[3] If a U wave followed the T wave without an isoelectric separation, the end of the T wave was taken as the nadir between the T and U waves. If the end of the T wave could not be reliably determined or when the T wave was of very low amplitude ($<$50 μV), QT measurements were not made and the lead excluded from analysis.[10] No attempt was made to correct for missing leads.[3] The JT interval (from the J point to the end of the T wave) was calculated from the equation JT = QT – QRS, and the T peak–T end interval was measured from the apex of the T wave to its end.[2] All measurements were performed by two independent investigators who were blinded to the clinical data of the patients. The averages of the measurements of the two observers were used for comparisons.

Definition of ECG indices of ventricular repolarisation

The following indices were derived from each measurement of each ECG:

(1) The maximum QT, JT, and T peak–T end intervals in any measurable leads (QT maximum, JT maximum, and maximum T peak–T end, respectively).
(2) QT, JT, and T peak–T end dispersion, defined as the difference between QT maximum and the minimum QT interval (QT dispersion), JT maximum and the minimum JT interval (JT dispersion), maximum T peak–T end and the minimum T peak–T end interval (T peak–T end dispersion) in any measurable leads, respectively.

Twelve-lead vectorcardiogram

To derive vectorcardiographic descriptors of ventricular repolarisation, orthogonal X, Y, and Z leads were reconstructed from the standard 12 ECG leads (Figure 1).[14] Let QRSx, QRSy, and QRSz be the projections of the maximum QRS vector on the X, Y, and Z axes and QRSxy, QRSxz, and QRSyz its projections on the frontal (xy), horizontal (xz), and right saggital (yz) planes, respectively. Similarly, let Tx, Ty, and Tz be the projections of the maximum T vector on the X, Y, and Z axes and Txy, Txz, and Tyz its projections on the frontal (xy), horizontal (xz), and right saggital (yz) planes, respectively. QRSxy, QRSxz, QRSyz, Txy, Txz, and Tyz were automatically calculated by our analysis system. According to previously published equations,[11] and by use of

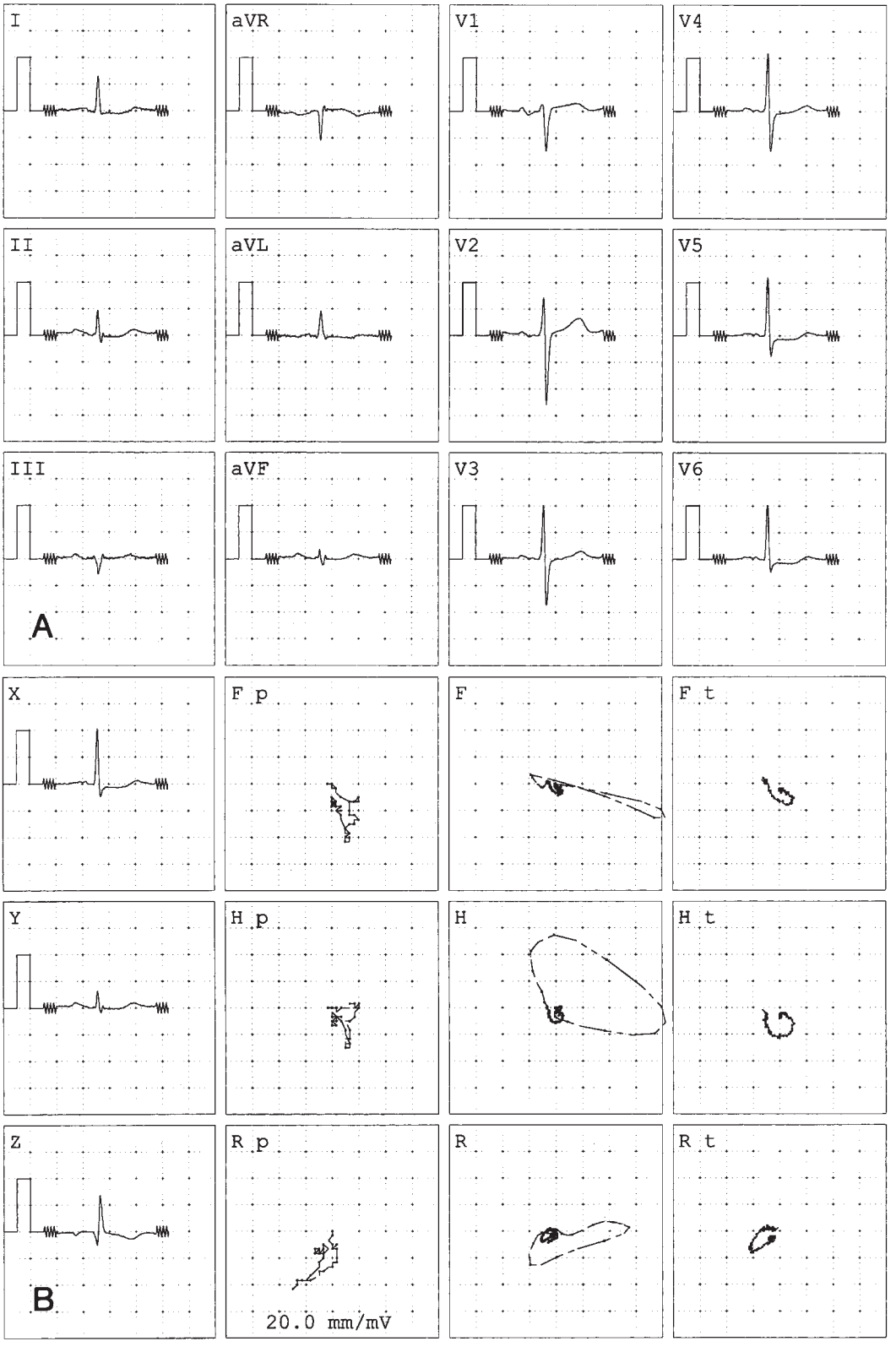

Figure 1 (**A**) Twelve-lead electrocardiogram, (**B**) reconstructed X, Y, and Z leads and P, QRS and T loop projections in the frontal (F), horizontal (H), and right saggital (R) planes from a 73 years old hypertensive patient.

the Pythagorean theorem, the amplitude of the maximum spatial T vector (Spatial T amplitude) was calculated from the formula:

$$\text{Spatial T amplitude} = [(Txy^2 + Txz^2 + Tyz^2)/2]^{1/2}$$

Furthermore, the angle (θ°) between the maximum spatial QRS and T vectors (Spatial QRS-T angle) was calculated from the formula:[11,15]

$$\cos\theta^\circ = (QRSxTx + QRSyTy + QRSzTz)/|QRS|\,|T|$$

The angle between the maximum QRS and T vectors in the frontal plane (Frontal QRS-T angle) was also computed.

Intraobserver and interobserver variability

Intraobserver and interobserver mean percent error (absolute difference between two observations divided by the mean and expressed in percent)[16] for maximum QT, JT, and T peak–T end intervals and for QT, JT, and T peak–T end dispersion measurements were determined in 40 randomly selected study participants. To define intraobserver errors of measurements one of the two investigators measured the QT, JT, and T peak–T end intervals of all 40 ECGs twice.

Statistical analysis

Continuous variables are expressed as mean (± s.d.). For comparisons between the different patient groups, Mann-Whitney test and chi-square test were used where appropriate. Spearman's correlation coefficients were used to assess the association between different variables. To determine the multivariate contribution of other factors to the values of different repolarisation indices, linear regression equations were constructed:

$$X = A_0 + A_1\text{BP control} + A_2\text{LVH} + A_3\text{Age} + A_4\text{Sex} + A_5\text{Heart rate},$$

where X was one of the considered repolarisation indices (QT maximum, QT dispersion, JT maximum, JT dispersion, maximum T peak–T end, T peak–T end dispersion, spatial T amplitude, frontal QRS-T angle, spatial QRS-T angle) and BP control was the presence of high or low BP during patient evaluation. Left ventricular hypertrophy (LVH) and sex were treated as categorical variables in the regression equation. For each repolarisation index, the statistical significance of the regression coefficients A_1, A_2, A_3, A_4, and A_5 was evaluated. P values <0.05 were considered statistically significant.

Results

Of a total enrolment of 110 hypertensive patients, 43 were categorised in the low BP group and 67 in the high BP group. ECG documented LVH was more prevalent in the high BP group. No significant differences were noticed between the two groups concerning age, sex, heart rate, the body mass index, the duration of hypertension, the plasma potassium levels, and the drugs administered (Table 1).

All ECG repolarisation indices were found to have similar values between hypertensive patients with high BP and those with low BP. Only the spatial QRS-T angle was found to be significantly higher in hypertensive patients with high BP values compared to patients with low BP levels. No significant differences were noticed between the two groups concerning the QRS axis in the frontal plane (Table 2). From those patients who fulfilled the ECG criteria for LVH, five (18%) patients with high BP and one (10%) patient with low BP showed discordant T waves to the QRS complexes in both precordial and limb leads ($P = 0.131$), while seven (25%) patients with high BP and two (20%) patients with low BP showed discordant T waves in the precordial leads ($P = 0.456$). In those patients who did not fulfill the ECG criteria for LVH ($n = 72$), the spatial QRS-T angle was the only repolarisation parameter to differ significantly, although only marginally, between hypertensives with high BP (18.1 ± 12.8 degrees) and those with low BP (11.8 ± 6.9 degrees), $P = 0.05$. The differences in the repolarisation parameters between hypertensive patients with ECG documented LVH and those without LVH are shown in Table 3.

Table 1 Clinical characteristics of patients with high or low blood pressure

Characteristic	*Low blood pressure group (n = 43)*	*High blood pressure group (n = 67)*	*P value*
Age (years)	63.7 ± 14	63.5 ± 10.9	0.876
Men	51% (22)	39% (26)	0.206
Body mass index (kg/m^2)	27.5 ± 4	27.9 ± 6.6	0.693
Heart rate (bpm)	68.4 ± 12.4	71.4 ± 9.7	0.190
Systolic blood pressure (mm Hg)	138.4 ± 11.1	169.5 ± 16.4	<0.001
Diastolic blood pressure (mm Hg)	80.7 ± 7.5	100.8 ± 12.8	<0.001
Duration of hypertension (years)	10.3 ± 4.6	10.7 ± 5.9	0.715
Left ventricular hypertrophy	23% (10)	42% (28)	0.018
Serum potassium (mmol/L)	5 ± 0.6	5.1 ± 0.6	0.323
Drug therapy			
Diuretics	23% (10)	31% (21)	0.276
ACE-inhibitors	26% (11)	39% (26)	0.107
Calcium-antagonists	33% (14)	21% (14)	0.102
Beta-blockers	12% (5)	9% (6)	0.513
Other	7% (3)	4% (3)	0.366

Values are mean (± s.d.), or % (no.). ACE, angiotensin converting enzyme.

Table 2 Electrocardiographic data of patients with high or low blood pressure

Parameter	*High blood pressure group (n = 67)*	*Low blood pressure group (n = 43)*	*P value*
QRS axis			
Normal	85% (57)	91% (39)	0.651
Left	13% (9)	7% (3)	0.180
Right	2% (1)	2% (1)	1.0
QT maximum (ms)	396 ± 26.9	402.3 ± 37.1	0.410
QT dispersion (ms)	44.4 ± 17.4	39.8 ± 13.9	0.149
JT maximum (ms)	303.1 ± 27.6	307.1 ± 34.6	0.442
JT dispersion (ms)	40.9 ± 14.4	39.7 ± 12.9	0.734
Maximum T peak–T end (ms)	95.8 ± 17.7	93.7 ± 18.8	0.327
T peak–T end dispersion (ms)	54.8 ± 23.7	52 ± 18.1	0.769
Spatial T amplitude (μV)	342.8 ± 158.1	358.4 ± 121.8	0.516
Frontal QRS-T angle (degrees)	23.4 ± 37	16.8 ± 28.4	0.114
Spatial QRS-T angle (degrees)	19.8 ± 12.6	14 ± 8.1	0.025

Values are mean (± s.d.), or % (no.). See the text for the definition of the individual indices.

Table 3 Electrocardiographic data of hypertensive patients with or without left ventricular hypertrophy

Parameter	*Patients with LVH (n = 38)*	*Patients without LVH (n = 72)*	*P value*
QT maximum (ms)	405.3 ± 37.7	395.6 ± 26.9	0.159
QT dispersion (ms)	47.9 ± 19.4	39.8 ± 13.6	0.024
JT maximum (ms)	309.6 ± 37.5	302.1 ± 25.9	0.235
JT dispersion (ms)	44.1 ± 16.1	38.5 ± 12.1	0.044
Maximum T peak–T end (ms)	102.7 ± 22	90.9 ± 14.3	0.005
T peak–T end dispersion (ms)	62.8 ± 27.8	49 ± 15.8	0.006
Spatial T amplitude (μv)	318 ± 186.8	365.2 ± 114.7	0.023
Frontal QRS-T angle (degrees)	36.2 ± 52.5	12.6 ± 11.4	0.029
Spatial QRS-T angle (degrees)	21.3 ± 12.6	15.2 ± 10.9	0.012

Values are mean (± s.d.), or % (no.). LVH, left ventricular hypertrophy. See the text for the definition of the individual indices.

Associations between repolarisation indices and clinical variables

QT maximum and JT maximum were significantly associated with sex ($P = 0.009$ and $P < 0.001$, respectively) and heart rate ($P < 0.001$). JT dispersion ($P = 0.046$), maximum T peak–T end ($P = 0.006$) and T peak–T end dispersion ($P = 0.006$) were significantly related to the presence of ECG documented LVH. The spatial T amplitude was significantly associated with sex ($P = 0.020$) and the presence of LVH ($P = 0.046$). The frontal QRS-T angle was significantly associated with the presence of LVH ($P = 0.01$), while the spatial QRS-T angle was significantly associated to sex ($P = 0.001$) and the presence of high or low BP in our study population ($P = 0.008$).

The correlation among the different repolarisation indices is shown in Table 4.

Accuracy of the measurements

The intraobserver relative errors for QT maximum, QT dispersion, JT maximum, JT dispersion, maximum T peak–T end, and T peak–T end dispersion were 2.1 ± 1.6%, 24.3 ± 22.2%, 4.3 ± 3.7%, 25.9 ± 21.1%, 7.9 ± 6.2%, and 27 ± 26.1%, respectively. The interobserver relative errors for the same indices were 4.7 ± 3.9%, 32.8 ± 21.9%, 7.8 ± 9.4%, 34 ± 24.4%, 9.8 ± 8.2%, and 35.2 ± 31.3%, respectively.

Discussion

The principal finding of this study is that the spatial QRS-T angle is the only repolarisation marker to be significantly increased in those treated hypertensive patients who show repeat office measurements of high BP. Conventional ECG markers of the dispersion of ventricular repolarisation duration failed to demonstrate significant differences between hypertensives with high or low BP, although they were useful for the discrimination between hypertensives with and those without ECG-documented LVH.

Ventricular repolarisation abnormalities in hypertension

Hypertension affects approximately 25% of the adult population, and a large percentage of these patients develop LVH, which carries a negative prognosis being a recognised harbinger of ventricular arrhythmias and mortality.[1,2] Although the factors predisposing to electrical instability and arrhythmic death are not well established,[17] it has been reported that myocardial hypertrophy alters the ionic channels that are operative during the early repolarisation phase.[18] Furthermore, LVH is characterised by an increase in collagen interstitial matrix that may also lead to alterations in ventricular repolarisation.[19]

Dispersion of repolarisation duration in hypertension

QT dispersion has been suggested to reflect abnormal ventricular repolarisation.[3] High QT dispersion values have been reported in patients with systemic hypertension and LVH,[1,2,4,5] which decrease after the appropriate treatment of high BP.[5,6] However, the rather weak correlation between QT dispersion and BP levels,[5,6] or LVH[1,2,5] and the poor reproduci-

Table 4 Correlation coefficients between electrocardiographic and vectrocardiographic indices

	QT dispersion	*JT maximum*	*JT dispersion*	*Maximum T peak–T end*	*T peak–T end dispersion*	*Spatial T amplitude*	*Frontal QRS-T angle*	*Spatial QRS-T angle*
QT maximum	0.160	0.893‡	0.131	0.266†	0.150	–0.243*	0.265†	0.071
QT dispersion		0.019	0.153	0.249†	0.382‡	–0.036	0.087	0.293†
JT maximum			0.287†	0.179	0.074	–0.281†	0.252†	0.003
JT dispersion				0.082	0.142	–0.155	0.198*	0.174
Maximum T peak–T end					0.741‡	–0.137	0.390‡	0.262†
T peak–T end dispersion						–0.233*	0.321†	0.198*
Spatial T amplitude							–0.205*	–0.059
Frontal QRS-T angle								0.300†

*$P < 0.05$, †$P < 0.01$, ‡$P < 0.001$. See the text for the definition of the individual indices.

bility of QT dispersion measurements,[3,7] may reduce its power to assess arrhythmia risk prospectively.[4]

In this study, to estimate the dispersion of repolarisation duration, apart from QT maximum and QT dispersion, JT maximum and JT dispersion were used as more accurate measures of the ventricular repolarisation time and maximum T peak–T end and T peak–T end dispersion were used as indices of the transmural dispersion of repolarisation.[20] However, no significant differences were noticed between hypertensives with high and those with low BP irrespective of the ECG index used (Table 2). The aforementioned results cannot be attributed either to the extension of hypertension history, nor to heart rate, nor to the plasma potassium levels, and nor to the drugs administered (Table 1). The inability of the dispersion indices to distinguish hypertensives with high BP from those with low BP may be due to the questionable accuracy of the manual measurement of those indices. On the other hand the dispersion indices, and particularly T peak–T end dispersion, showed significant differences between hypertensives with and those without ECG-documented LVH. This is in accordance with previous reports.[1,2,4,5] The associations found between ECG and clinical variables have already been reported.[21,22]

Spatial aspects of ventricular repolarisation in hypertension

The dispersion of repolarisation duration is not the only important aspect of ventricular repolarisation. The complexity of the shape of the T wave is supposed to be a marker of abnormal repolarisation and principal component analysis has been used to quantify T waves with major morphologic abnormalities.[8] However, subtle changes in the shape of a monophasic T wave are not detectable by this method. QT dispersion has recently been reported to be an attribute of T loop morphology,[10] and T axis has been used as an alternative measure of repolarisation abnormality.[9] An abnormal orientation of the T axis has long been known to provide a global measure of repolarisation abnormality,[22] and the spatial angle between the direction of the repolarisation and depolarisation waves (spatial QRS-T angle) has already been used to quantify ventricular repolarisation.[11,23,24]

In this study, the spatial QRS-T angle was the only index found to be significantly higher in hypertensives who showed repeatedly high BP values as compared to those who had low BP levels. Previous studies have already reported a widened spatial QRS-T angle in patients with eccentric LVH,[11] and a decrease in the frontal or the horizontal QRS-T angle in patients following treatment of hypertension.[23] A widened QRS-T angle in patients with LVH was also reported when Grant's vectorial method was used.[25] Similar differences between patients with ECG-documented LVH and those without were also demonstrated in our study concerning the spatial T amplitude, the frontal QRS-T angle and the spatial QRS-T angle. Although most of the ECG and the vectorcardiographic repolarisation parameters showed significant differences between patients with and those without ECG-documented LVH, only the spatial QRS-T angle was able to discriminate hypertensives according to the adequacy of the BP control (Table 3). This is in accordance with a previous report.[23] On the other hand, the spatial T amplitude and the frontal QRS-T angle failed to demonstrate significant differences between hypertensives with high or low BP. Furthermore, scalar ECG characteristics like the QRS axis or the presence of discordant T waves failed to show any differences between the two study groups.

The ability of the spatial QRS-T angle to distinguish hypertensives with low from those with high BP is not probably due to the different prevalence of ECG-documented LVH between hypertensives with low and those with high BP. This is obvious from the fact that the spatial QRS-T angle was the only repolarisation parameter able to distinguish the patients who did not fulfill the ECG criteria for LVH according to their BP levels. The significant

association found between the spatial QRS-T angle and sex has been reported previously.[15]

Hence, the spatial QRS-T angle may serve as a marker of ventricular repolarisation heterogeneity in hypertension, even when ECG documentation of LVH is not obtained. The relationships between the spatial QRS-T angle and the pathophysiology of specific repolarisation abnormalities are not established. The occurrence of electrophysiological changes in response to ventricular pressure or volume overload has been well documented.[26] The ability of the spatial QRS-T angle to detect those stretch-induced electrophysiological changes, still undetectable by the standard surface ECG, cannot be ruled out. However, we do not know if the observed differences in the magnitude of the spatial QRS-T angle are due to movement of the QRS or the T vector. The consistently high BP values obtained in some ($n = 67$) of our patients are less likely to be due to an alerting response (white coat hypertension). The latter is usually attributed to an increased sympathetic activation.[27] This was not probably the case in our study because no sympathetically-induced differences in heart rate were noticed. Therefore, the data of the present study may suggest that the angle between the directions of ventricular depolarisation and repolarisation is a sensitive and early marker of the repolarisation alterations in systemic hypertension. Moreover, it can be measured easily, is not affected by observation biases and is likely to be less susceptible to noise and problems of definition than conventional ECG measures of the dispersion of the repolarisation duration. Future studies should clarify the ability of the spatial T loop morphology analysis and particularly of the spatial QRS-T angle to offer a more precise and reproducible measure of ventricular repolarisation. Furthermore, the ability of those vectorcardiographic descriptors of ventricular repolarisation to identify high-risk patients for ventricular arrhythmias should be prospectively assessed.

Limitations

Only ECG documentation of LVH was obtained in this study. Additional studies using echocardiographically determined LVH may prove to be more informative. Neither coronary angiography, nor other imaging techniques were applied to our study patients to rule out the presence of significant but not clinically apparent coronary artery disease. Finally, our patients were not prospectively evaluated to assess the ability of the vectorcardiographic markers of ventricular repolarisation to predict the occurrence of life-threatening ventricular arrhythmias.

Conclusion

In conclusion, the spatial QRS-T angle was the only repolarisation marker found to be significantly increased in those treated hypertensives who showed repeatedly high BP values. Conventional features of the scalar ECG and markers of the dispersion of ventricular repolarisation duration failed to demonstrate significant differences between treated hypetensives with high or low BP levels.

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