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The value of the spatial QRS–T angle for assessing the severity of heart damage in patients with arterial hypertension

Aim	To study the relationship between clinical, echocardiographic, and laboratory indexes with increased QRS – T spatial angle (sQRS – T) in patients with arterial hypertension (AH).
Material and methods	The study included 160 patients with AH, 61 (38%) men and 99 (62%) women aged 58 [49; 67] years. Patients with ischemic heart disease or His bundle blocks were not included. Echocardiography was used to determine the left ventricular end-diastolic dimension (LV EDD), left ventricular posterior wall thickness (LVPWT), interventricular septal thickness (IVST), relative wall thickness (RWT), left ventricular myocardial mass (LVMM), and LVMM index (LVMMI). Also, the following indexes were analyzed: office systolic and diastolic blood pressure (SBP, DBP), disease duration, body mass index, plasma levels of glucose, cholesterol, and creatinine, and glomerular filtration rate. The QRS-T spatial angle was calculated as an angle between the integral vectors QRS and T using a vectorcardiogram derived from a 12-lead digital electrocardiogram. Data are presented as median (25th percentile; 75th percentile].
Results	The QRS-T spatial angle for the group was 65 [43; 90] °. The QRS – T spatial angle increased with increases in the AH grade (grade 1 AH, 55 [37; 74] °; grade 2 AH, 60 [41; 82] °; grade 3 AH, 88 [62; 107] °; p<0.0001); the AH stage (stage 1, 50 [41; 77] °; stage 2, 68 [44; 93] °; stage 3, 78 [59; 110] °; p=0.0002), and the cardiovascular risk degree (low and moderate risk, 49 [37; 70] °, high risk, 62 [43; 88] ° (p=0.005); very high risk, 88 [61; 117] ° vs. high risk, 62 [43; 88] ° (p=0.0002). The QRS – T spatial angle was greater with diabetes mellitus (78 [60; 117] °) than without it (63 [43; 89] °) (p=0.03). Weak but significant correlations were found between sQRS – T and body mass index (r=0.2; p<0.01), SBP (r=0.4; p<0.0001), DBP (r=0.2; p<0.01), LV EDD (r=0.2; p<0.01), LV PWT (r=0.3; p<0.001), IVST (r=0.3; p<0.001), LVMM (r=0.3; p<0.001), LVMMI (r=0.3; p<0.001), and blood glucose (r=0.2; p<0.01).
Conclusion	In patients with AH, a large QRS-T spatial angle is related with significantly higher values of SBP and DBP, LV dimension, blood glucose, and body mass index.

Keywords	QRS–T spatial angle; arterial hypertension; synthetized vectorcardiogram
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Introduction

A major risk factor for cardiovascular diseases (CVDs), cerebrovascular diseases, and chronic kidney disease (CKD), arterial hypertension (AH) is consequently one of the leading causes of mortality. With a prevalence of AH in the adult Russian population of above 40%, primary health care carries the most significant burden in terms of examination and treatment of these patients.

The current guidelines [1] require a comprehensive review of the overall risks of developing CVDs in all patients with AH to determine treatment strategy. In real-world practice, however, it is not always possible to perform a timely comprehensive examination of all patients with AH. According to the AH register [2], while all patients with AH underwent electrocardiogram (ECG), only 29% of patients received echocardiography in the primary healthcare setting in 2010–2014. It is of note that 94% of patients were classified as being of high and very high cardiovascular risk.

Thus, it seems relevant to increase the diagnostic efficacy of electrocardiographic examination in terms of detecting heart lesions and enabling a more refined risk stratification in patients with AH.

The prognostic value of the voltage electrocardiographic criteria of left ventricular hypertrophy (LVH) with respect to overall mortality and cardiovascular mortality, as well as adverse cardiovascular events both in the general population and in patients with AH, has been confirmed by several meta-analyses [3–5].

However, several large trials have shown that changes in the ST segment and T wave characteristic of LVH are strong independent predictors of all-cause mortality in the general population [6], having the greatest prognostic power and the closest relations with the risk of coronary artery disease (CAD), chronic heart failure (CHF) and stroke compared to other electrocardiographic criteria of LVH [7]. In another large trial in patients with AH, the presence of ST-segment and T-wave changes increased the risk of developing CAD by more than a factor of two, as well as constituting a significant predictor of other CVDs within the 10-year follow-up [8]. At the same time, non-specific minimal changes in the ST segment and T wave in the baseline ECG records in AH patients under the age of 55 years without CVDs and diabetes mellitus (DM) were not associated with increased cardiovascular risks and mortality in the 16-year follow-up period [9].

The so-called spatial QRS-T angle (sQRS-T) can be used to assess the ST segment and T wave changes characteristic of LVH. This indicator has recently captured the great attention of researchers due to its prognostic value for overall and cardiovascular mortality, being linked with sudden cardiac death in the general population and various clinical populations [10]. However, there are still few trials studying this indicator in patients with AH.

Objective

To determine clinical, echocardiographic, and laboratory indicators associated with the increase in sQRS-T in patients with AH.

Material and Methods

The study included 160 patients with AH (61 (38%) male and 99 (62%) female) at the age of 58 [49; 67] years hospitalized at the Myasnikov Research Institute for Cardiology of the Russian National Medical Research Center of Cardiology.

AH was diagnosed according to current guidelines based on a complex clinical examination. The study excluded patients with CAD, valvular heart disease, or conditions with a clinically significant effect on the ECG configuration (bundle branch blocks, ventricular preexcitation, ventricular pacing).

The following case record data were collected from the INTERIN medical information system: diagnosis (including AH grade, AH stage, general cardiovascular risk, concomitant cerebrovascular and DM type 2); duration of AH, systolic blood pressure (SBP), and diastolic blood pressure (DBP) measured on the day of ECG acquisition; drug treatment at the time of admission; age, smoking status, family history of early-onset CVDs, body mass index (BMI), plasma levels of cholesterol, glucose and creatinine. Glomerular filtration rate (GFR) was calculated using the MDRD (Modification of Dietin Renal Disease) formula.

Digital 12-lead ECGs were acquired and processed using the Easy ECG computer-based electrocardiograph. The indicators of interest were calculated automatically based on the mean cardiac complex of a 10-second record following automatic mapping (manually corrected if necessary). The synthesized X, Y, and Z orthogonal leads were calculated using special linear transformations. The dimensions of sQRS-T (in degrees) were calculated as the angle between the QRS and T integral vectors. The following indicators were also analyzed in this paper: the Sokolow-Lyon index (the sum of the S wave amplitudes in the V1 lead and the largest R wave in the V5 or V6 lead), the Cornell index (the sum of the S wave amplitudes in the V3 lead and the R wave in the aVL lead; 8 mm were added in female patients to compensate for the sex-related difference in the threshold values of this indicator) and the Cornell product (the product of the QRS duration and the Cornell index). The electrocardiographic criteria of LVH were Sokolow-Lyon index >35 mm, Cornell index>28 mm (male) and 20 mm (female), Cornell product>2,440 mm·ms.

Transthoracic echocardiography was performed using a Vivid 9 ultrasound device in accordance with the guidelines for the assessment of heart chambers in adults [11]. Left ventricular (LV) mass was calculated based on linear B-controlled M-mode measurements using the formula proposed by the American Echocardiography Society (AES). Relative wall thickness (RWT) index was calculated using the formula:

$$(2 \times \text{LVPWT})/\text{LVEDD},$$

where LVEDD is the end-diastolic LV dimension, and LVPWT is LV posterior wall thickness.

Given a large number of overweight and obese patients in the study group, LV mass was indexed to patient height raised to the power of 2.7 rather than body surface area (BSA). The echocardiographic criteria of LVH were LV mass index (LVMI)/height$^{2.7}$>50 g/m$^{2.7}$ in male patients and >47 g/m$^{2.7}$ in female patients. In patients with normal body weight, LV mass was indexed to BSA, which was calculated using the formula:

$$\text{BSA}=\text{body weight (kg)}^{0.425} \times \text{height (cm)}^{0.725} \times 0.007184.$$

In this case, the LVH criteria were LVMI >115 g/m^2 in male patients and 95 g/m^2 in female patients.

The data were analyzed using MedCalc v.12.7. Continuous variables are presented as the median and the interquartile range (Me [25th percentile; 75th percentile]).

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Qualitative variables are expressed as the absolute and relative values (n (%)). The Student's t-test or Mann – Whitney test were used to estimate the differences between two independent quantitative variables depending on the type of distribution, while Pearson's chi-square was used for the qualitative variables. Pearson's or Spearman's correlation analysis was performed to determine relationships between variables depending on a type of distribution. Characteristic curves (ROC curves) were used, and the sensitivity and specificity of the criteria were determined to describe the diagnostic efficacy of the indicators. The level of statistical significance was $p<0.05$.

Results

The patient characteristics at the time of examination are presented in Table 1. According to the history, 53 (33%) patients had AH grade 1, 50 (31%) patients had AH grade 2, and 57 (36%) patients had AH grade 3. At the time of hospitalization, 40 (25%) patients did not receive antihypertensive therapy, 41 (26%) patients received monotherapy, while 79 (49%) patients were administered combined antihypertensive therapy. At the time of admission, 93 (58%) patients reached the target BP level (<140/90 mmHg). AH stage I, II, and III was established in 46 (29%), 81 (51%), and 33 (20%) patients, respectively. The 10 (6%) patients had low cardiovascular risk, 23 (14%) had moderate cardiovascular risk, 73 (46%) had high risk, and 54 (34%) patients were at very high cardiovascular risk. The low and moderate cardiovascular risk groups were combined for further analysis due to the small number of patients.

sQRS-T was 65 [43.5; 90.5] ° in the general group. There were no significant sex-related differences in sQRS-T: 74 [49; 94] ° in male patients and 61 [43; 89] ° in female patients (p=0.15). There were also no significant differences in sQRS-T based on the presence of family history of early-onset CVDs: 67 [39; 88] ° if present and 63 [45; 94] ° if absent (p=0.82); and based on smoking status: 70 [47; 81] ° in smokers and 62.5 [43; 91] ° in non-smokers (p=0.85).

There were significant differences in sQRS-T depending on the presence of DM type 2: 96 [63; 142] ° in patients with DM type 2 and 62 [43; 88] ° in patients without DM type 2 (p=0.001).

Figure 1 depicts the values of sQRS-T depending on the grades and stages of AH along with the degree of overall cardiovascular risk: sQRS-T grew as AH grade, AH stage, and overall cardiovascular risk increased. This trend was especially clear in the groups of varying overall cardiovascular risk.

The results of ROC analysis with regard to the determination of the diagnostic efficacy of ECG indicators in the groups of patients having low-to-moderate and high-to-very-high cardiovascular risk are presented in Table 2. The groups of AH patients at very high cardiovascular risk and those at lower cardiovascular risk are presented in Table 3.

The correlation coefficients for sQRS-T with various clinical and laboratory indicators are shown in Table 4.

Echocardiographic signs of LVH were detected in 66 (41%) patients when the recommended LVMI/height$^{2.7}$ threshold values were used. Electrocardiographic signs of LVH were detected in 29 (18%) patients when the recommended LVMI/height$^{2.7}$ threshold values were used.

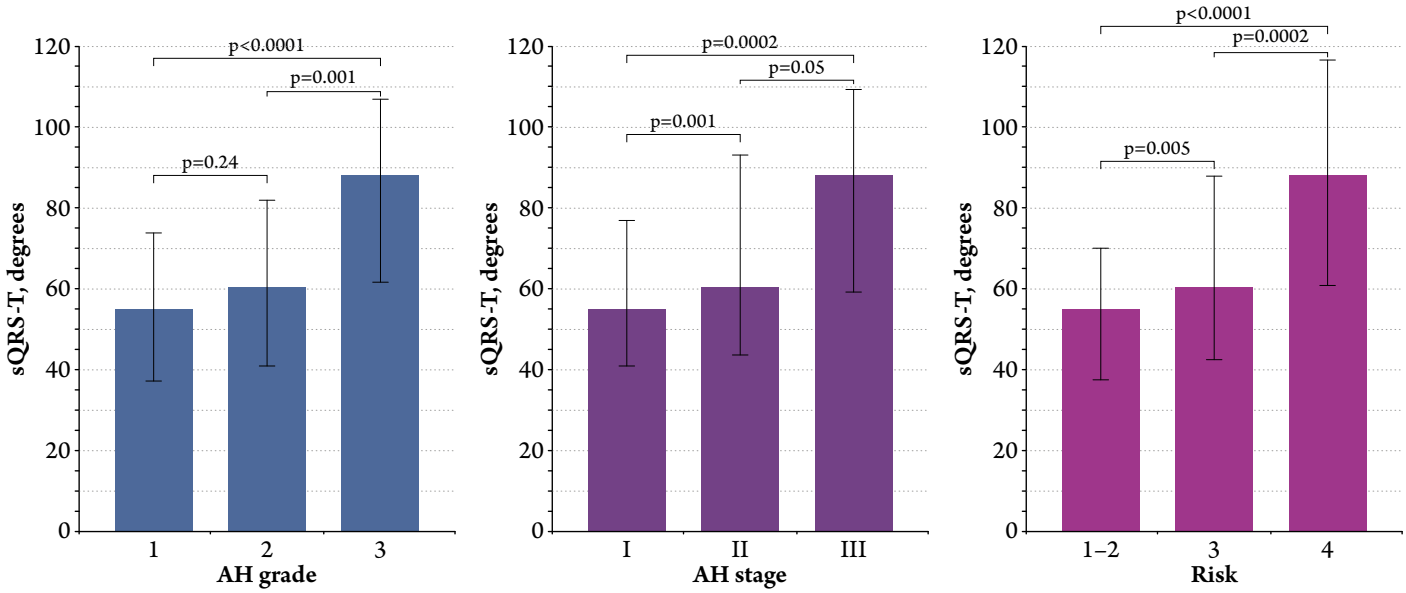
There were no significant differences in the incidence of voltage electrocardiographic signs of LVH in patients with and without obesity (16 (24%) versus 13 (14%), (p=0.19). sQRS – T>90° was significantly more prevalent in obese patients compared to patients without obesity (24 (35%) versus 16 (17%)). At the same time, obese patients had significantly higher LV mass (194 [167; 220] g versus 169 [142; 197] g in patients without obesity; p=0.0006) and LVMI/height$^{2.7}$ (46 [40; 53] g/m$^{2.7}$ versus 42 [36; 47] g/m$^{2.7}$ in patients without obesity; p=0.0003).

Table 1. Clinical characteristics of the examined patients

Parameter	Value
Male	61 (38%)
Age, years	58 [49; 67]
Duration of the disease, years	10 [5; 15]
Burdened family history	47 (29%)
Smoking	30 (19%)
BMI, kg/m^2	29 [26; 33]
Number of overweight patients (BMI 25–29.9 kg/m^2)	64 (40%)
Obesity (BMI > 30 kg/m^2)	68 (42.5%)
Diabetes mellitus type 2	20 (12.5%)
SBP, mm Hg	140 [130; 153]
DBP, mm Hg	84 [80; 90]
Total cholesterol, mmol/L	5.36 [4.45; 6.59]
Total cholesterol > 4.9 mmol/L	103 (64%)
Fasting plasma glucose, mmol/L	5.41 [4.93; 6.0]
Number of patients with fasting plasma glucose >5.6 mmol/L	65 (41%)
Creatinine, μmol/L	71.0 [62.9; 84.0]
GFR, mL/min/1.73m^2	89.9 [72.5; 100.2]
LVEDD, cm	5.0 [4.8; 5.3]
LVPWT, cm	1.0 [0.9; 1.05]
IVST, cm	1.0 [0.9; 1.05]
LV mass, g	176 [153; 213]
LVMI/height$^{2.7}$, g/m$^{2.7}$	43.6 [37.0; 49.5]

BMI – body mass index; SBP – systolic blood pressure;
DBP – diastolic blood pressure; GFR – glomerular filtration rate;
LVEDD – left ventricular end-diastolic dimension;
LVPWT – left ventricular posterior wall thickness;
IVST – interventricular thickness;
LVMI – left ventricular mass index.

Figure 1. QRS–T (Me [25th percentile; 75th percentile]) depending on AH grade, AH stage and overall cardiovascular risk



AH – arterial hypertension.

Table 2. Diagnostic efficacy of electrocardiographic indicators in the groups of patients with low-to-moderate and high-to-very high cardiovascular risk

Parameter	AUC±SE	95% CI	Threshold	Sensitivity, %	Specificity, %
sQRS-T, °	0.71 ± 0.04	0.6–0.77	> 79	43	94
Sokolow-Lyon index, mm	0.66 ± 0.05	0.58–0.73	> 35	4	100
Cornell index, mm	0.77 ± 0.04	0.69–0.83	> 28	11	100
Cornell product, mm x ms	0.77 ± 0.04	0.69–0.83	> 2,440	13	100

QRS-T – spatial angle; AUC – area under the curve; SE – standard error; CI – confidence interval.

Table 3. Diagnostic efficacy of ECG data in the groups of AH patients at very high cardiovascular risk and AH patients with lower cardiovascular risks

Parameter	AUC±SE	95% CI	Threshold	Sensitivity, %	Specificity, %
sQRS-T, °	0.72 ± 0.04	0.64–0.78	> 94	44	89
Sokolow-Lyon index, mm	0.55 ± 0.05	0.47–0.63	> 35	3	94
Cornell index, mm	0.69 ± 0.05	0.61–0.76	> 28	11	99
Cornell product, mm x ms	0.70 ± 0.04	0.62–0.77	> 2,440	22	95

QRS-T – spatial angle; AUC – area under the curve; SE – standard error; CI – confidence interval.

The results of ROC-analysis concerning diagnostic efficacy of electrocardiographic indicators in the groups of AH patients both with and without echocardiographic signs of LVH are given in Table 5.

For patients who had achieved the target BP level at admission, sQRS-T was significantly smaller (59 [40; 79] °) as compared to the patients who had not achieved the target BP level (79 [55; 100] °; p=0.002).

The results of ROC-analysis concerning diagnostic efficacy of electrocardiographic indicators in the groups of AH patients who had achieved the target BP levels and those who had not are given in Table 6.

sQRS-T angles above the 75th percentile (greater than 90°) were not detected in patients at low and moderate cardiovascular risk; however, they were detected in 15 (20%) patients at high risk and 26 (46%) patients at very high cardiovascular risk.

In the high cardiovascular risk group, sQRS-T >90° was detected in 11 (15%) patients without LVH voltage criteria and 4 (5%) patients with LVH voltage criteria, as well as being associated with higher SBP (140 [139; 150] mmHg with sQRS-T>90°; 134 [126;142] mmHg with sQRS-T≤90°; p=0.01); LVEDD (5.30 [4.83; 5.50] cm with sQRS-T>90°; 4.90 [4.80; 5.20] cm with sQRS-T<90°; p=0.04) and LV mass (213 [169; 220] g with sQRS-T>90°; 170 [153] 188 g with sQRS-T≤90°; p=0.04). There was a trend towards more patients having sQRS-T >90° among those who did not take antihypertensive drugs or who

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Table 4. Correlation coefficients for sQRS-T and various clinical and laboratory indicators

Parameter	Correlation coefficient (r)	p
Age	0.00	0.96
AH duration	0.10	0.19
SBP	0.37	< 0.0001
DBP	0.23	0.004
BMI	0.22	0.006
LVEDD	0.21	0.007
LVPWT	0.30	0.0001
IVST	0.30	0.0001
LV mass	0.32	< 0.0001
LVMI/height2.7	0.34	< 0.0001
Fasting plasma glucose	0.17	0.03
Total cholesterol	0.02	0.74
Creatinine	0.04	0.60
GFR	0.04	0.58
Sokolow-Lyon index	0.14	0.08
Cornell index	0.44	< 0.0001
Cornell product	0.51	< 0.0001

QRS-T – spacial angle; SBP – systolic blood pressure; DBP – diastolic blood pressure; BMI – body mass index; LVEDD – left ventricular end-diastolic dimension; LVPWT – left ventricular posterior wall thickness; IVST – interventricular thickness; LVMI – left ventricular mass index; GFR – glomerular filtration rate.

received monotherapy (11 (26%) of 43) compared to those who took combined therapy (4 (13%) of 30; p=0.33).

In the very high cardiovascular risk group, sQRS-T>90° was detected in 7 (13%) patients without LVH voltage criteria, 18 (33%) patients with LVH voltage criteria and were associated with higher LVPWT (1.10 [1.00; 1.10] cm with sQRS-T >90°; 1.00 [0.90; 1.10] cm with sQRS-T≤90°; p=0.03). sQRS-T>90° was significantly more often identified in patients who did not take antihypertensive drugs or who received monotherapy (11 (79%) of 14) as compared to patients who received combination therapy (14 (35%) of 40; p=0.01).

Figure 2 presents the ECG record of a 32-year-old male patient complaining of episodes of headache, shortness of breath, and squeezing in the chest. He had a history of hypertensive heart disease for several years and was not receiving continuous antihypertensive therapy.

He was admitted to the hospital in moderate condition: moderate subcutaneous fat; BMI 26 kg/m^2; muted heart sounds; regular heart rhythm; heart rate (HR) 82 bpm; right-arm BP 160/100 mmHg, left-arm BP 170/100 mmHg.

ECG: regular sinus rhythm, HR 74 bpm; normal position of the heart axis. Sokolow-Lyon index at the upper normal limit (35 mm); Cornell index (20 mm) and Cornell product (1830 mm·ms) within normal limits; sQRS-T significantly increased (120°).

Echocardiogram: cardiac chambers not dilated; walls not thickened. LVEDD 5.5 cm; IVST 1.0 cm; LVPWT 1.0 cm; LV mass 213 g (normal up to 224 g), LVMI/BSA 10^6 g/m^2 (normal up to 115 g/m^2), LVMI/height$^{2.7}$ 44 g/m$^{2.7}$ (normal up to 50 g/m$^{2.7}$).

A secondary origin of AH was not confirmed; total cholesterol increased to 5.13 mmol/L; low-density lipoprotein cholesterol increased to 3.28 mmol/L; hypertensive retinal angiopathy; stenosis up to 20% in the brachiocephalic bifurcation due to a heterogeneous atherosclerotic plaque. Final diagnosis: hypertensive heart

Table 5. Diagnostic efficacy of electrocardiographic indicators in the groups of AH patients with and without echocardiographic signs of LVH

Parameter	AUC±SE	95% CI	Threshold	Sensitivity, %	Specificity, %
sQRS-T, °	0.62 ± 0.05	0.54–0.70	> 55	74	48
			> 117°	15	95
Sokolow-Lyon index, mm	0.60 ± 0.05	0.52–0.67	> 35	2	95
Cornell index, mm	0.69 ± 0.04	0.61–0.76	> 28	3	98
Cornell product, mm x ms	0.69 ± 0.04	0.62–0.76	> 2,440	20	94

QRS-T – spatial angle; LVH – left ventricular hypertrophy; AUC – the area under the curve; SE – standard error; CI – confidence interval.

Table 6. Diagnostic efficacy of electrocardiographic indicators in the groups of AH patients who achieved the target BP levels and did not achieve the target BP levels

Parameter	AUC±SE	95% CI	Threshold	Sensitivity, %	Specificity, %
sQRS-T, °	0.66 ± 0.04	0.58–0.74	> 70	63	68
			> 104	21	93
Sokolow-Lyon index, mm	0.55 ± 0.05	0.47–0.63	> 35	3	97
Cornell index, mm	0.60 ± 0.05	0.52–0.68	> 28	3	95
Cornell product, mm x ms	0.61 ± 0.05	0.53–0.69	> 2,440	18	93

QRS-T – spatial angle; AUC – area under the curve; SE – standard error; CI – confidence interval.

disease stage II, AH grade 2. Risk 3 (high). Brachiocephalic atherosclerosis. Dyspydemia type IIa. Hypertensive retinal angiopathy.

Figure 3 presents the ECG record of a 71-old female patient complaining of shortness of breath at minimal physical exertion, squeezing pain at physical exertion, lower extremity swelling, asthenia. A long history of hypertensive heart disease and DM type 2 (she uses insulin). According to the patient, she receives combined antihypertensive therapy (angiotensin II receptor blocker, calcium channel blocker, beta-blocker, imidazoline receptor agonist, diuretic) in adequate doses.

She was hospitalized in moderate condition; excessive subcutaneous fat; BMI 40 kg/m^2; clear heart sounds; loud S2 over the aorta; regular rhythm; HR 75 bpm; right-arm BP 180/90 mmHg, left-arm BP 170/90 mmHg.

The biochemical blood test showed hyperglycemia (fasting plasma glucose 8.6 mmol/L) at admission.

ECG: sinus rhythm, HR 78 bpm; normal position of the heart axis. LVH voltage criterion (Cornell index 21 mm); Sokolow-Lion index (30 mm) and Cornell product (2,210 mm·ms) are normal; ST-segment decline and negative T waves are recorded in leads I, II, aVL, V4 – V6; sQRS-T significantly increased (156°).

Dilated left atrium, concentric LVH according to echocardiography. LV volume index (LVVI) 51 mL/m^2 (normal up to 34 mL/m^2), LVEDD 4.8 cm; IVST 1.3 cm; LVPWT 1.15 cm; LV mass 226 g (normal up to 162 g), LVMI/BSA 112 g/m^2 (normal up to 95 g/m^2), LVMI/height$^{2.7}$ 70 g/m$^{2.7}$ (normal up to 47 g/m$^{2.7}$). Satisfactory LV wall motion, LVEF 60%. Impaired LV diastolic function shown by tissue Doppler. No signs of pulmonary hypertension.

Stress echocardiography was performed to eliminate latent coronary insufficiency; latent coronary insufficiency test was negative.

Clinical diagnosis: hypertensive heart disease stage III. Uncontrolled AH. Risk 4 (very high). Atherosclerosis of the aorta and extracranial brachiocephalic arteries. Concentric LVH. DM type 2. Obesity grade III. CKD stage II. CHF, NYHA functional class II. Chronic cerebral ischemia. Diabetic polyneuropathy. Diabetic microangiopathy, diabetic macroangiopathy.

Following treatment modification in the hospital (two drugs were replaced within the same classes, spironolactone was added), BP stabilized at 135–139/80 mmHg.

Discussion

Increased sQRS-T is associated with increased risk of all-cause and cardiovascular death in the general population and in patients with suspected CAD and patients with CHF to an even greater extent [12]. Relatively little is known about changes of this indicator in patients with AH.

Figure 2. ECG of a 32-year old male patient diagnosed with hypertensive heart disease stage II, arterial hypertension grade 2, risk 3 (high)

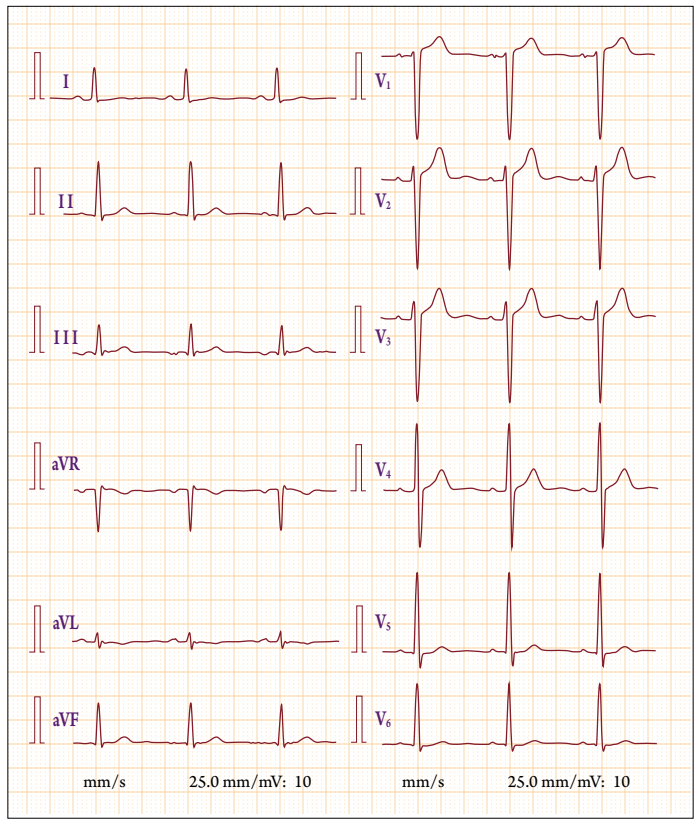
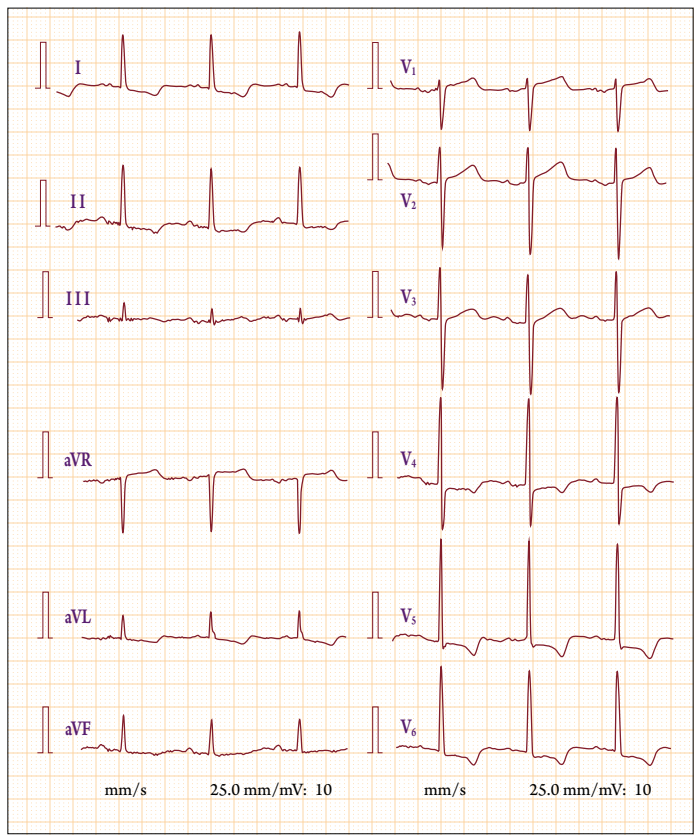


Figure 3. ECG of a 71-year old female patient diagnosed with: hypertensive heart disease stage III, uncontrolled arterial hypertension, risk 4 (very high); diabetes mellitus type 2; obesity grade III; chronic heart failure NYHA functional class II



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This paper demonstrates that sQRS-T grows with AH degree and AH stage – and, especially clearly, overall cardiovascular risk increase. According to the ROC analysis, sQRS-T threshold>79° and >94° suggests that a patient with AH is at high and very high cardiovascular risk, respectively.

The correlation analysis identified highly significant but weak correlations of sQRS-T with SBP and DBP and echocardiographic indicators of LVH. The ROC analysis demonstrated that sQRS-T allows identification of patients with echocardiographic signs of LVH and patients who have not achieved the target BP level in the general AH patient group. However, this analysis is characterized by high specificity, and relatively low sensitivity, which nevertheless is comparable to the sensitivity of the Cornell product and exceeds by several times the sensitivity of the Cornell index and the Sokolow-Lyon index. Lower sQRS-T thresholds lead to a large number of false-positive results. Increased sQRS-T as a manifestation of electrical cardiac remodeling in patients with AH is obviously influenced not only by the BP levels and LV mass but also by some other factors. It should be noted that the correlation of sQRS-T and the Cornell index and Cornell product was significant but relatively low; there was no correlation with the Sokolow – Lyon index. This confirms once again that sQRS-T and voltage electrocardiographic criteria of LVH do not overlap but complement each other.

The correlation of increased sQRS-T with DM type 2 and blood glucose levels in patients with AH should also be considered. Both DM and AH significantly increase the cardiovascular risk and, consequently cardiovascular death. The exact mechanisms of an increase of sQRS-T in DM are not known. There is evidence that this is associated with the level of glycosylated hemoglobin and the presence and severity of autonomous neuropathy [13].

We have not found the association of sQRS-T with blood creatinine and GFR, although sQRS-T increase was associated with CKD in patients with inferior myocardial infarction in our previous work [14]. In this study, a mild decrease in GFR (from 89 to 60 mL/min/1.73 m^2) and a moderate decrease in GFR (from 59 to 40 mL/min/1.73 m^2) was identified in 65 (40%) and 13 (8%) patients, respectively, other patients had normal GFR. Increased sQRS-T may possibly be manifested in more severe kidney damage in patients with AH. Further research is required to address this issue.

Patients with CAD were intentionally excluded since this pathology can independently influence ST segment, T wave, and thus sQRS-T. sQRS-T will be studied in patients with a combination of CAD and AH later.

sQRS-T correlations with only a few simple indicators widely used in routine clinical practice were analyzed in this study. There are published data that the increased frontal QRS – T angle is correlated with the non-dipper status [15]; the ST-segment and T-wave changes are correlated with myocardial deformation in patients with AH [16, 17]. This issues may also be addressed in future studies.

Conclusion

Changes in sQRS-T in patients with hypertension have not yet been very well studied. This paper shows that the spatial QRS – T angle grows as the degree and stage of arterial hypertension increases and especially as the degree of overall cardiovascular risk increases in patients with arterial hypertension.

Correlations of sQRS-T with systolic and diastolic blood pressure and echocardiographic indicators characterizing left ventricular hypertrophy have been identified; correlations of increased sQRS-T with diabetes mellitus type 2 and blood glucose levels have been demonstrated. sQRS-T greater than 90° was identified especially often (in 79% of cases) in patients with arterial hypertension at very high risk who did not receive adequate antihypertensive therapy. Mechanisms of sQRS-T increase in patients with arterial hypertension require further research.

No conflict of interest is reported.

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