KAPITEL 14 / *CHAPTER 14* [14]
INNOVATIVE METHODS FOR ANALYSING HEART RHYTHM TURBULENCE IN PRECISION CARDIOLOGY

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Introduction

Timely identification of patients at high risk of adverse events after acute myocardial infarction (AMI) is a key element in the implementation of effective secondary prevention measures and reduction of cardiovascular mortality. Over the past decades, a number of clinical, electrophysiological, and biochemical risk markers have been proposed, but their prognostic accuracy remains moderate, even when used in combined models.

It is known that post-ischemic cardiac remodeling is accompanied by complex changes in autonomic regulation, which are not always adequately reflected by traditional indicators. In this context, heart rate turbulence (HRTo/HRTs) markers are gaining particular attention as potentially independent predictors of risk, characterizing the dynamic response of the sinus rhythm to ventricular extrasystoles (VES).

With the development of modern analytical technologies, new opportunities are emerging for a more in-depth analysis of these indicators through digital modeling and the application of innovative algorithms within the concept of precision cardiology. This opens up prospects for the creation of new stratification tools capable of improving the accuracy of prognosis and individualizing the approach to patient management.

The study of fluctuations in the duration of sinus rhythm cycles (heart rate turbulence, HRT) after premature heart contractions recorded on an ECG using the Holter method includes the characterization of two numerical descriptors: turbulence onset (TO) and turbulence slope (TS) of the heart rhythm. This approach was proposed in the work of Georg Schmidt et al. [1], where it was noted that after PVCs, patients with low risk experienced early acceleration and subsequent slowing of the sinus

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rhythm, which was not characteristic of patients with high risk.

This approach was continued in subsequent studies [2, 3, 4], as TSR is recognized as a promising noninvasive marker of ventricular tachyarrhythmias and cardiac death, especially in patients with myocardial infarction (MI) or heart failure (HF) with reduced LVEF.

TCP abnormalities demonstrate the strongest prognostic value for composite endpoints (adjusted hazard ratio [aHR] 3.41) or individual events, including cardiac death (aHR 4.08), ventricular tachyarrhythmia (aHR 3.72), and HFH (aHR 4.32) [4].

At the same time, when determining TSR based on acceleration (increase in heart rate after SE, HRTO) and deceleration (decrease in heart rate after previous acceleration, HRTS) [5], there is a discrepancy in the methods used to calculate them. A simple formula is used to calculate HRTO, while HRTS requires the use of specialized software that allows this indicator to be analyzed only with the help of separate Holter systems, since HRTS is defined as the maximum slope of the linear regression of 5 consecutive average R-R intervals after premature contraction (EC), which requires detailed data processing.

14.1. Modern interpretation of quantitative ECG analysis.

A promising area of research is the further optimization of quantitative ECG analysis with the creation and use of an individual formula for calculating HRTO and HRTS in a familiar data analysis software environment, followed by risk assessment and the construction of a predictive model. We examined 32 patients diagnosed with chronic coronary syndrome (CCS), acute coronary syndrome (ACS) of the STEMI type, and hypertension with ventricular arrhythmia.

All patients underwent a comprehensive assessment, including clinical status, blood test results, ECG with evaluation of its quantitative indicators and subsequent digitization and differentiation of ECG, echocardiography, and CAG results. The feasibility of the proposed mathematical approach to determining HRTO and HRTS indicators using an individual formula, which we recommend implementing in a spreadsheet editor, was assessed during Holter monitoring of patients. The study was conducted in accordance with the basic provisions of Good Clinical Practice (GCP) and the World Medical Association's Declaration of Helsinki on ethical principles for conducting scientific medical research involving human subjects, with the informed consent of the study participants.

In the first stage of the study, we analyzed the formulas for determining TSR parameters (HRTO and HRTS) used in some Holter devices for ECG recording. However, in the available literature, we did not find a description of software for their assessment using a simple analysis of two PQRST complexes before/after extrasystole and 15 complexes after with the calculation of specific indicators on the ECG.

Therefore, the formula for calculating Turbulence Onset (TO) is as follows: \[TO=\left(\frac{(RR_avg_post-
\text{RR}{\text{avg\pre}})}{\text{RR}{\text{avg_pre}}}\right) \times 100 \]
where:

• RRavg_postRR_avg_post is the average RR interval for the first two cardiac cycles after SE;

• RRavg_preRR_avg_pre is the average RR interval for the last two cardiac cycles before SE.

This formula allows you to calculate the percentage change in the average RR interval, which shows the heart's response to SE.

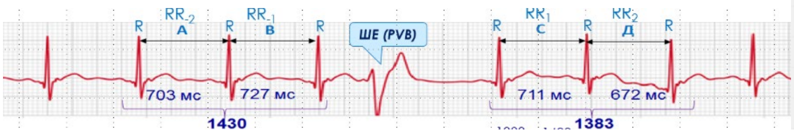
To calculate HRTO in data analysis software tools (Excel):

• The RR intervals before SE should be in cells A3 and B3.

• The RR intervals after SE should be in cells C3 and D3.

Then the HRTO formula will look like this: HRTO=((AVERAGE(D3:E3)-AVERAGE(A3:B3))/AVERAGE(A3:B3))×100

HRTO = ((AVERAGE(D3:E3) - AVERAGE(A3:B3)) / AVERAGE(A3:B3)) \times 100, as shown in Figure 1.



	A	B	C	D	E
1	-2	-1	0	1	2
2	A	B	PVB	C	D
3	703	727	1086	711	672
4	HRTO			1	2
5	-3,287				

Figure 1 - Calculation formula for Turbulence Onset [3].

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However, the situation with the formula for calculating Turbulence Slope (TS) is more complicated, as it is implemented as follows: TS=max (ΔRRΔt)TS=\max \left(\frac{\Delta \text{RR}}{\Delta t} \right)

where:

• ΔRR\Delta \text{RR} - change in RR interval;

• Δt\Delta t - time between changes in RR intervals.

In the context of cardiac turbulence, TS is calculated as the slope of the steepest straight line representing the change in RR intervals after SE during the first 5-15 heartbeats. This can be implemented in a spreadsheet editor such as Excel using the SLOPE or LINEST function. The formula looks like this:

\[TS = \text{SLOPE}(\text{dynamic_RR_values}, \text{timestamps})\]

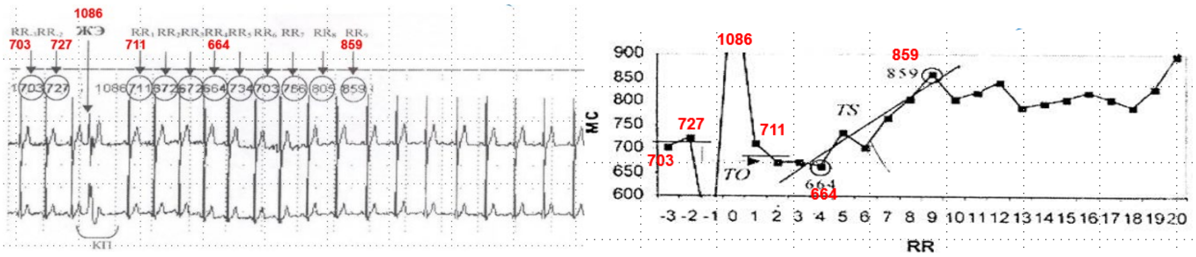
If the RR intervals after SE are located in cells C2:C16 and the time stamps are in cells B2:B16, the formula takes the following form:

TS=SLOPE(C2:C16,B2:B16)TS = \text{SLOPE}(C2:C16, B2:B16)

or

TS=@LINEST(C2:C16,B2:B16)TS = \text{@LINEST}(C2:C16, B2:B16),

as shown in Figure 2.



	A	B	C
1		A1-A15	B1-B15
2	A1	1	711
3	A2	2	672
4	A3	3	672
5	A4	4	664
6	A5	5	734
7	A6	6	703
8	A7	7	766
9	A8	8	805
10	A9	9	859
11	A10	10	800
12	A11	11	820
13	A12	12	825
14	A13	13	760
15	A14	14	770
16	A15	15	780
17	HRTS	linest	9,6429
18	HRTS	slope	9,6429

Figure 2 - Calculation formula for Turbulence Slope [5, 6].

Statistical data processing was performed using spreadsheets, applying parametric and nonparametric comparison methods. Under the condition of normal data distribution, the Student's t-test was used (data presented as mean (M) and standard deviation (m)), while under abnormal distribution, the Mann-Whitney U test was used. Pearson's and Spearman's correlation coefficients were used to analyze correlation relationships. The critical level of statistical significance was set at $p<0.05$, and the trend at $p<0.1$.

14.2. Prognostic value of an innovative approach to HRTO and HRTS assessment.

At the first stage of the study, all examined patients were divided into groups with positive and negative prognoses according to the limits for TSR assessment defined in the literature. The first group (positive prognosis) included patients with HRTO < 0% or HRTS > 2.5 ms/RR. The second group (negative prognosis) included patients with HRTO > 0% and HRTS < 2.5 ms/RR, which may indicate an increased risk of cardiac events.

When evaluating this distribution of TSR indicators in the positive/negative groups depending on the dynamics of HRTO, the average level of the indicator for the group was in the HRTO distribution: -4.84±1.37% versus +4.44±1.32%, $p<0.001$, Δ% 1-2 = 191.67% (patients with positive HRTO distribution, n=17, versus patients with negative HRTO distribution, n=15). In this group, depending on the positive/negative fluctuation of the HRTO indicator, the HRTS distribution was recorded as unreliable and amounted to 1.15±1.02 ms/RR versus 1.42±0.51 ms/RR, $p>0.2$, Δ% 1-2 = 24.00%, respectively.

Conversely, when determining HRTS as the dominant factor in the distribution into groups of positive/negative fluctuations, the discrepancy for HRTS was: 4.32±1.40 ms/RR versus -0.10±0.26 ms/RR, $p<0.005$, Δ%1-2=-102.42% (patients with positive HRTS distribution, n=10, versus patients with negative HRTS distribution, n=22). In this group of positive/negative HRTS fluctuations, the following HRTO distribution occurred with insignificant and unreliable corresponding fluctuations in the opposite direction: 0.17±3.74% vs. 0.79±0.81%, $p>0.5$.

In the group with positive/negative direction depending on HRTO dynamics (HRTO < 0% or HRTO > 0%), a dependence of the prevalence of VAS scores with unfavorable HRTO distribution was found (51.33±3.69% vs. 34.11±2.58%, $p<0.001$, Δ% 1-2 = 50.45%), while at the same time there was an inverse discrepancy in the number of SH (4.00±1.00% vs. 13.50±4.50%, $p<0.05$, Δ% 1-2 = 70.37%).

In the positive/negative direction group, depending on the HRTS dynamics (HRTS > 2.5 ms/RR or HRTS < 2.5 ms/RR), a negative direction of the standard deviation of the intervals between consecutive R-waves (NN intervals) on the ECG was determined, which is an indicator of HRV, SDNN (Standard Deviation of NN intervals) with an unfavorable HRTS distribution (100.43±7.23 ms vs. 129.25±9.25 ms, $p<0.02$, Δ% 1-2 = 22.29%), since a decrease in SDNN may indicate a decrease in HRV, which may be a sign of stress, fatigue, or cardiovascular disease, while high SDNN values indicate good autonomic nervous system function and high HRV, which is a sign of good physical condition and the body's ability to adapt to stress. Analysis of another HRV indicator – RMSSD (Root Mean Square of Successive Differences), which reflects short-term changes between successive heartbeats and is calculated as the root mean square of the differences between successive NN intervals (intervals between normal heartbeats) and indicates low HRV at low values, which may be a sign of stress, as opposed to an increase in the indicator, which indicates good autonomic nervous system function and high HRV, which is a sign of good physical condition and the body's ability to adapt to stress about the positive increase in RMSSD in the group with positive HRTS dynamics (160.65±27.15 ms vs. 22.9±6.29 ms, $p<0.001$, Δ% 1-2 = 95.74%).

Interestingly, the group with positive HRTS dynamics had higher scores on the NAD anxiety scale (8.90±0.40 vs. 7.86±0.40, $p<0.05$, Δ% 1-2 = 11.64%), total cholesterol (5.50±0.39 vs. 4.38±0.18, $p<0.02$, Δ% 1-2 = 20.31%) and alanine aminotransferase (28.42±2.79 vs. 19.43±1.73, $p<0.02$, Δ%1-2=31.64%), which was not observed for the HRTO indicator.

This discrepancy between HRTO and HRTS may explain the Pearson correlation

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coefficient between HRTO and HRTS, which is -0.117 ($p>0.05$), indicating a weak negative relationship between these two parameters. This means that an increase in HRTO is accompanied by a slight decrease in HRTS. The correlation coefficient between HRTO and SDNN is 0.3477 ($p<0.05$), indicating a noticeable positive relationship. This suggests a positive correlation between HRTO and SDNN. The correlation between HRTO and ST depression duration in Pearson's distribution is statistically significant at $p<0.01$, indicating an important relationship between these parameters, which warrants further analysis. The Pearson correlation coefficient between HRTS and the number of SHs is -0.276 ($p<0.05$), indicating a weak but significant negative relationship between these two parameters. The correlation between HRTS and HE couplets is -0.192 ($p<0.05$), which also indicates a weak but significant negative relationship. The correlation between HRTS and the maximum velocity ratio (MVR) of the differentiated ECG is 0.158 ($p<0.05$), indicating a positive weak but significant relationship that may be useful for further research.

The Spearman correlation coefficient between HRTO and HRTS is 0.1661 ($p>0.05$), indicating a weak positive relationship between these two parameters. This means that as HRTO increases, HRTS tends to increase, but this relationship is not statistically significant. The correlation coefficient between HRTO and SDNN is 0.2247 ($p<0.05$), indicating a weak positive correlation, so that as HRTO increases, SDNN tends to increase, indicating a certain degree of dependence between these parameters. The correlation between HRTO and ST depression duration and Spearman's distribution is statistically significant at $p<0.01$, indicating an important relationship between these parameters. The Spearman correlation coefficient between HRTS and the number of HE is 0.0649 ($p>0.05$), indicating a weak relationship between these two parameters. The correlation between HRTS and SE couplets is -0.3091 ($p<0.05$), indicating a weak but significant negative relationship. This means that as HRTS increases, SE couplets tend to decrease. The correlation between HRTS and VMS is -0.0281 ($p>0.05$), indicating a very weak negative relationship that is not statistically significant.

The results of the study show that changes in LVEF, depending on the distribution

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of HRTS (<2.5 ms/RR>), are associated with a significant increase in the HRT index (HRT: 3.11) at a 95% confidence interval (CI: 0.62-15.71) (p<0.05). A significant association was also found between an increase in HRV and HRTO (<0%>) when comparing 1-vessel damage to 2-3-vessel damage to the coronary arteries (HRV: 0.42, CI: 0.08-2.25, p<0.05).

A high level of interaction was found when analyzing the HRV SDNN (<90.18 ms>) and HR (9.92 with CI: 1.6-61.6, p<0.05), with a relative risk of their impact (relative risk (RR) 2.98; CI: 1.38-2.88; p<0.01). An interesting trend of interaction between HRTO (<0%>) and the probability of ventricular extrasystole (<240/24-hour>) during daily Holter monitoring is reflected in the HR index of 3.75 (CI: 0.34-40.81, p>0.1), but not for HRTS (HR: 0.84, CI: 0.13-5.56, p>0.1).

Also, our own study, when analyzing HR for HRTO in comparison with the HRM indicator for ECG differentiation, indicates certain patterns of HR for HRTO. In particular, for the group with a decrease in HRTO (<0%), there is a decrease in the risk of events with a decrease in HR (0.26; CI: 0.05-1.39; p>0.1). Compared to the group with an increase in HRTO (>0%), HR is significantly higher. Regarding the BMH indicator of differentiated ECG, our analysis showed that an increase in this indicator affects HRV. At BMH values >0.6, the HRV indicator (0.53; CI: 0.25-1.13; p>0.1) demonstrates a relatively lower risk compared to BMH<0.6. That is, an increase in the BMSh value is expected to be associated with a decrease in risk.

Our studies also show that high HRTS values (>2.5 ms/RR) are associated with a reduced risk of certain events. The HR for HRTS >2.5 ms/RR is 0.26 (CI: 0.05-1.39, p>0.1), indicating that a high HRTS value is a favorable factor. Conversely, low HRTS values (<2.5 ms/RR) are associated with an increased risk of events. The HR for HRTS <2.5 ms/RR is 3.11 (CI: 0.62-15.71, p<0.05), indicating that low HRTS values are an unfavorable factor.

The current view of the regulation of the cardiac circadian rhythm by the autonomic nervous system (ANS) shows that heart rate variability (HRV), as a change in the R-R interval, is associated with the interaction between the sympathetic and parasympathetic nervous systems. HRV is a recognized non-invasive method for assessing ANS activity; lower HRV correlates with higher cardiovascular events and mortality, while high HRV indicates better heart function. Therefore, current studies also establish a link between HRV and premature ventricular complexes [7]. The use of this approach has sparked interest in determining the effect of sodium-dependent glucose cotransporter 2 (SGLT2) inhibitors, with an assessment of the benefits of dapagliflozin in patients with type 2 diabetes and reduced LVEF, by reducing the burden of ventricular ectopic contractions through its effect on HRV and ventricular repolarization heterogeneity. The results of the EMBODY study on HRV and indicators of sympathetic and parasympathetic nervous activity of the heart show improvement in the empagliflozin group compared to the placebo group [8]. According to the concept of the TSR study, the ratio of time before/after SE in the distribution of early acceleration followed by a slowdown in sinus rhythm, as well as the study of ECG changes in the repolarization phase during the first post-extrasystolic normal contraction with assessment of post-extrasystolic ST segment changes and T wave variation (PEST) are strong independent predictors of all-cause mortality in survivors of acute MI and elderly patients from the general population [9].

Conclusions

The proposed innovative approach to calculating HRTO and HRTS indicators in data analysis software allows for the assessment of patient prognosis and endpoints using the presented calculation model in all patients, regardless of whether the software has Holter monitoring capabilities for these calculations. The assessment of the use of HRTO and HRTS calculation formulas allowed us to find dependencies regarding the distribution of HRTO greater than/less than 0% prevalence of VAS scores with an unfavorable distribution of HRTO ($p<0.001$, Δ% 1-2 = 50.45%) with a reverse discrepancy in the number of SH ($p<0.05$, Δ% 1-2 = 70.37%), and with an unfavorable distribution of HRTS, a negative trend in SDNN was recorded ($p<0.02$, Δ% 1-2 = 22.29%) with a positive increase in RMSSD in the group with positive HRTS dynamics ($p<0.001$, Δ% 1-2 = 95.74%).

Correlations between HRTS and the number of SE, SE couplets, and the VMS index indicate significant relationships according to Pearson's correlation ($p<0.05$), but have different directions of relationships according to Spearman.

Our studies have shown that changes in LVEF are associated with the distribution of HRTS and HRTO indicators. In particular, a decrease in HRTS (<2.5 ms/RR>) is accompanied by a significant increase in HR. In addition, there is a significant association between a decrease in HRTO (<0%) and an increased risk of coronary artery disease (2-3 vessel disease compared to 1-vessel disease). The results of the study prove that low HRTO is associated with a reduced risk, and high BMSh values of differentiated ECG are associated with a lower risk of events, therefore both HRTO and BMSh values of differentiated ECG have a significant impact on HR and can be used for risk assessment. The results of the study show that high HRTS values are associated with a reduced risk, while low HRTS values are associated with an increased risk of events. This indicator can be used to assess risks in cardiovascular diseases.

The interaction between HRV (SDNN) and odds ratio (OR) confirms a high level of risk of cardiovascular events. A tendency toward an increased likelihood of ventricular extrasystoles during 24-hour Holter monitoring was also found in the group with low HRTO values, although this requires further study.

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