Changes in heart rate variability caused by coronary angioplasty depend on the localisation of coronary lesions

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Abstract

Background: Coronary angioplasty (PTCA) is a common treatment method in patients with coronary heart disease, but its effects on heart rate variability (HRV) have not been well established.

Aim: To verify whether the localisation of coronary lesion undergoing PTCA affects HRV parameters.

Methods: Ninety six consecutive individuals underwent elective coronary angiography with subsequent ad hoc successful PTCA. Two five-minute ECG were recorded, one before PTCA and the second 24-hour after PTCA. The HRV indices were determined by means of classical and ‘new’ mathematical models.

Results: The PTCA-induced changes in HRV variables depended on the localisation of dilated lesion. PTCA of the circumflex artery revealed the most significant HRV changes – a decrease in value of domain indices: Yeh DI (0.033±0.031 vs. 0.011±0.006 unitless, p=0.005), Yeh II (0.053±0.039 vs. 0.032±0.013 unit, p=0.017), Organ BAND (9.101±9.245 vs. 4.62±2.205 bpm/beat per minute, p=0.031), Huey STV (208.821±262.248 vs. 76.444±35.281 bpm, p=0.013), Dalton MABB (15.733±16.575 vs. 7.57±4.89 ms, p=0.015), Dalton SD (48.741±37.468 vs. 27.759±10.533 ms, p=0.015), Zugaib STV (0.0129±0.0132 vs. 0.005±0.003 un, p=0.005), SDNN (27.204±18.592 vs. 21.329±18.592 ms, p=0.044) and rMSSD (56.239±19.751 vs. 51.496±43.889 ms, p=0.025) and increased LF/HF (2.384±2.072 vs. 5.632±3.379 un, p=0.044). Angioplasty of the right coronary artery resulted in decreased AR TP (18.273±2.296 vs. 17.085±2.256 ms², p=0.017) and alteration of the sympathovagal balance of the autonomic nervous system towards predominance of sympathetic activity: AR LF (0.264±0.029 vs. 0.284±0.040 un, p=0.007), LF/HF (4.310±4.457 vs. 6.958±7.013 un, p=0.018), HF (0.199±0.165 vs. 0.141±0.157 un, p=0.031), AR HF (0.647±0.043 vs. 0.621±0.054 un, p=0.014). PTCA of the left anterior descending artery caused no change.

Conclusion: Changes in heart rate variability caused by coronary angioplasty depend on the localisation of coronary lesions.

Key words: heart rate variability, percutaneous transluminal coronary angioplasty, stable angina

Introduction

Heart rate variability (HRV) refers to variability of the intervals between successive sinus beats, i.e. R-R intervals. The HRV changes result from the impact of autonomic nervous system tone on sinus node function. Moreover, they reflect the effects of the humoral substances released locally and peripherally. Up to now, HRV parameters have been shown to adequately predict poor prognosis in ischaemic heart disease patients, particularly after myocardial infarction (MI). These parameters are also used to detect autonomic neuropathy. The HRV analysis is not used routinely in clinical practice mainly due to either the fact that it is a time-consuming technique associated with 24-hour ECG (electrocardiographic) signal recordings or a lack of standardisation of diagnostic methods as well as some technical difficulties. An attempt to make analysis easier led to successful replacement of 24-hour signal registration by short-term measures lasting 2 to 15 minutes [1]. New HRV parameters as well as possibilities for their more common application in clinical practice have been sought [2-4].

It has been shown that HRV indices decrease after percutaneous revascularisation [5], in particular parasympathetic-related ones [6,7]. The results of some studies indicate that this drop is accompanied by increased sympathetic tone [6]. Moreover, variable reaction was also...
Changes in heart rate variability caused by coronary angioplasty depend on the localisation of coronary lesions

Methods

Patients

Out of 500 consecutive individuals who underwent elective coronary angiography, 96 subjects (75 men and 21 women at the mean age of 56.8±8.7 years) were enrolled in the study. They presented stable CAD and had successful primary PTCA following angiography. Patients selected for this examination for reasons other than angina were not included. Moreover, patients who did not undergo PTCA due to either a lack of significant lesions in the coronary arteries or culprit lesions not qualified for PTCA were not entered into the further analysis. Patients with suboptimal effect of PTCA and also with an inadequate quality of the ECG recording were not included in the further analysis.

Patients received typical medical treatment for CAD. In the majority of them concomitant cardiovascular risk factors were also present. In order to evaluate the impact of location of the treated coronary artery on HRV, patients were split into three subgroups. The LAD group involved 43 patients who underwent angioplasty of the left anterior descending (LAD) artery. The RCA group comprised 36 individuals with PTCA of the right coronary artery (RCA). Finally, the CX group consisted of 17 patients who underwent PTCA of the left circumflex (CX) artery.

Coronary artery angiography

Coronary angiography and angioplasty were performed according to the well-established standards. The atherosclerotic lesions in the coronary arteries were treated with PTCA when stenosis exceed 70% of the arterial lumen. A procedure was considered successful if the final flow was TIMI 3 (complete, rapid filling of the coronary artery with contrast medium, the presence of residual stenosis not exceeding 20%) and no complications during either the procedure or in-hospital stay.

HRV analysis

Confirmation of the clinical relevance of the HRV analysis derived from 2 to 15-minute ECG recordings [1] allowed us to use 5-minute ECG records. The first ECG was acquired on the admission and the second one within 24 hours after angioplasty. ECG registration was always performed in the same conditions, in the morning, in the horizontal position with maximal limitation of the affecting external stimuli. The electrocardiogram was recorded using the computer software and cardiograph Kardio PC (® MEDEA Gliwice). Acquired ECG analogue signals were transformed by an analogue-digital converter with 500 Hz sampling frequency. The digital signals were then analysed on the basis of the graphic environment of LabVIEW 7.1 Express (National Instruments). The series of the heart rate (HR) derived from the ECG record was analysed using the Berger splin filtration algorithm, where sampling of the flattened HR function occurred with frequency f_s=1.07 Hz [12].

A standard time domain parameters, SDNN (standard deviation of subsequent R-R intervals), rMSSD (root-mean-square of successive interval differences) and frequency domain indices such as TP (total power, including a frequency range 0.003-0.04 Hz), HF (high frequency, frequencies within a range of 0.15-0.4 Hz), LF (low frequency, frequencies within a range of 0.04-0.15 Hz) and LF/HF index were analysed. Spectral analysis was performed using either the Fourier rapid transformation technique or autoregressive method (AR). It allowed us to reach high resolution of frequency, stabilisation of the spectral variation estimators and spectral parameterisation. Except for total spectrum power expressed as ms², the frequency domain parameters were expressed as relative units. They were calculated from the rate of the given index to TP after subtraction of the value of ULF (ultra low frequency, a range of frequencies <0.003 Hz).

Moreover, the analysis was extended by evaluation of the time domain parameters calculated using short- and long-term variability models described by Yeh, Zugaib, Organ, Dalton and Huey [4]. The methods of signal transformation with respect to just either time or frequency are sometimes insufficient for practical use in biomedicine where the signal changes its spectra values as time elapses. Thus, there is a need to employ an analysis combining time and frequency domains with the use of wavelet transform w1-w5 parameters [13, 14]. A description of the time domain parameters and combined time and frequency domains employed in this study is outlined in Table 1.

The PTCA impact on HRV with respect to both the whole studied patient population and subgroups was
evaluated. Baseline and postprocedural HRV profiles were compared. A comparative analysis of the delta parameters following PTCA of the specific coronary arteries was carried out (delta: \( \Delta = \) the value of a given parameter before PTCA – its value following PTCA). Additionally, the impact of the group differentiating factors was analysed.

The study protocol was approved by the Bioethical Committee of the Poznań University of Medical Sciences (No. 887/04 dated 6.05.2004). All patients expressed informed consent to participate in this study.

### Statistical analysis

Data were analysed using the statistical computer software STATISTICA 7.0 (StatSoft Inc.). In the case of a lack of normal distribution, checked with Shapiro-Wilk’s W test, the Mann-Whitney U test for unrelated variables was used. To compare the HRV parameters in the specific groups before and after PTCA procedures Wilcoxon pair rank test was employed. Due to the fact that the parameters are expressed on the interval scale, the results are presented as the arithmetic mean and standard deviation. To assess the correlation on a nominal scale the exact Fisher’s test and \( \chi^2 \) test with Yates’ correction were used. A p value of 0.05 was considered as statistically significant.

### Results

The examined group of patients was not equal regarding coexistence of diabetes mellitus and severity of the atherosclerosis assessed according to the Gensini scale. They also differed with respect to location of MI and previous angioplasty. Complete characteristics of the groups are presented in Table II.

Comparison of HRV indices before and after PTCA in all enrolled patients revealed that myocardial revascularisation
caused a drop in spectrum power within the range of high frequencies (HF) (0.199±0.170 vs. 0.141±0.153 un, p=0.002) and elevation of LF/HF (4.017±4.125 vs. 6.042±5.615 un, p=0.0002) as well as wavelet w3 index (3.176±3.533 vs. 3.503±2.988 ms, p=0.048).

The HRV indices that differentiated the three examined subgroups of patients at the baseline were as follows: rMSSD was found to be significantly higher in the RCA group vs. the LAD group (70.742±53.067 vs. 53.59±31.46 ms, p=0.019), AR TP higher in the RCA group vs. the LAD group (18.273±2.296 vs. 17.096±2.0 ms², p=0.005) or CX group (18.273±2.296 vs. 16.839±2.568 ms², p=0.030) (see Figures 1 and 2).

The PTCA of LAD caused no significant changes in the HRV indices.

Angioplasty of the RCA resulted in a statistically significant decrease of AR TP as well as HF or AR HF accompanied by an increase in AR LF and LF/HF (Table III).

Table II. Characteristics of the examined groups of patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>LAD n=43</th>
<th>CX n=17</th>
<th>RCA n=36</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Women</td>
<td>12 (28)</td>
<td>2 (2)</td>
<td>7 (19)</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>31 (72)</td>
<td>15 (88)</td>
<td>29 (81)</td>
</tr>
<tr>
<td>Age [years]</td>
<td></td>
<td>56.98±9.2</td>
<td>57.4±7.1</td>
<td>56.1±9.1</td>
</tr>
<tr>
<td>BMI [kg/m²]</td>
<td></td>
<td>28.4±4.2</td>
<td>27±3.7</td>
<td>26.7±3.4</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td></td>
<td>33 (77)</td>
<td>11 (65)</td>
<td>19 (53)</td>
</tr>
<tr>
<td>Diabetes mellitus type 2</td>
<td></td>
<td>13 (30)</td>
<td>0 (0)</td>
<td>5 (14)</td>
</tr>
<tr>
<td>Smokers</td>
<td></td>
<td>21 (49)</td>
<td>13 (76)</td>
<td>23 (64)</td>
</tr>
<tr>
<td>Hypcholesterolemia</td>
<td></td>
<td>29 (67)</td>
<td>12 (71)</td>
<td>26 (72)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td></td>
<td>16 (37)</td>
<td>5 (29)</td>
<td>16 (44)</td>
</tr>
<tr>
<td>Inferior wall infarction</td>
<td></td>
<td>8 (18)</td>
<td>3 (18)</td>
<td>19 (53)</td>
</tr>
<tr>
<td>Anterior wall infarction</td>
<td></td>
<td>12 (28)</td>
<td>0 (0)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Lateral wall infarction</td>
<td></td>
<td>1 (2)</td>
<td>4 (24)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Indefinite location of myocardial infarction</td>
<td></td>
<td>3 (7)</td>
<td>4 (24)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Time from myocardial infarction [months]</td>
<td></td>
<td>7.3±2.7</td>
<td>54.7±108.9</td>
<td>10.3±7.6</td>
</tr>
<tr>
<td>Previous CABG</td>
<td></td>
<td>1 (2)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Previous PTCA</td>
<td></td>
<td>7 (16)</td>
<td>4 (24)</td>
<td>6 (17)</td>
</tr>
<tr>
<td>Previous PTCA LAD</td>
<td></td>
<td>3 (7)</td>
<td>0 (0)</td>
<td>2 (6)</td>
</tr>
<tr>
<td>Previous PTCA CX</td>
<td></td>
<td>0 (0)</td>
<td>3 (18)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Previous PTCA RCA</td>
<td></td>
<td>5 (12)</td>
<td>1 (6)</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Time from procedure [months]</td>
<td></td>
<td>13.7±16.8</td>
<td>41.3±33.7</td>
<td>17.5±16.1</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td></td>
<td>26 (60)</td>
<td>9 (53)</td>
<td>18 (50)</td>
</tr>
<tr>
<td>Diuretics</td>
<td></td>
<td>10 (23)</td>
<td>3 (18)</td>
<td>5 (14)</td>
</tr>
<tr>
<td>Ca blockers</td>
<td></td>
<td>6 (14)</td>
<td>1 (6)</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Anti-arrhythmic agents</td>
<td></td>
<td>2 (5)</td>
<td>0 (0)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Statins</td>
<td></td>
<td>34 (79)</td>
<td>12 (71)</td>
<td>32 (89)</td>
</tr>
<tr>
<td>Fibrats</td>
<td></td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td></td>
<td>35 (81)</td>
<td>11 (65)</td>
<td>26 (72)</td>
</tr>
<tr>
<td>Digoxin</td>
<td></td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Vasodilatators</td>
<td></td>
<td>21 (49)</td>
<td>7 (41)</td>
<td>14 (39)</td>
</tr>
<tr>
<td>Genini score</td>
<td></td>
<td>5188±8859</td>
<td>59.38±40.28</td>
<td>35.67±24.93</td>
</tr>
<tr>
<td>One-vessel disease</td>
<td></td>
<td>23 (53)</td>
<td>6 (35)</td>
<td>16 (44)</td>
</tr>
<tr>
<td>Two-vessel disease</td>
<td></td>
<td>14 (32)</td>
<td>8 (47)</td>
<td>13 (36)</td>
</tr>
<tr>
<td>Multi-vessel disease</td>
<td></td>
<td>6 (14)</td>
<td>3 (18)</td>
<td>7 (19)</td>
</tr>
<tr>
<td>Balloon angioplasty</td>
<td></td>
<td>4 (9)</td>
<td>4 (24)</td>
<td>2 (6)</td>
</tr>
<tr>
<td>1 stent implanted</td>
<td></td>
<td>31 (72)</td>
<td>11 (65)</td>
<td>27 (75)</td>
</tr>
<tr>
<td>≥2 stents implanted</td>
<td></td>
<td>8 (19)</td>
<td>2 (12)</td>
<td>7 (19)</td>
</tr>
</tbody>
</table>

Table III. Characteristics of the examined groups of patients

* LAD vs. CX, p <0.001
** LAD vs. RCA, p=0.005
*** LAD vs. CX, p=0.045
****LAD vs. CX, p=0.022; CX vs. RCA, p=0.034
***** LAD vs. CX, p=0.018
****** RCA vs. CX, p=0.015

Figure 1. Comparison of baseline index AR TP

![Figure 1. Comparison of baseline index AR TP](image1)

Figure 2. Comparison of baseline rMSSD values

![Figure 2. Comparison of baseline rMSSD values](image2)
In the CX group, PTCA caused a significant decrease of nine time domain indices and a marked increase of the LF/HF ratio (Table IV). Analysis of PTCA-induced changes in HRV parameters (Δ) revealed several significant differences (Table V).

A trend of the most changes of HRV parameters following PTCA of CX was found to be opposite to that in the case of other coronary artery revascularisation (Figures 3 and 4).

Following angioplasty, the examined subgroups did not differ significantly between each other, although in the CX group the results of most parameters of sinus rhythm variability were the lowest.

No significant differences in the delta analysis including the impact of coexistence of diabetes mellitus, previous MI and infarct location were noted. No differences between patients with single- and multi-vessel disease were seen either. However, a comparative analysis of the HRV parameters calculated after angioplasty in the group of patients with mild (GS ≤32) and significant (GS >32) severity of atherosclerotic changes expressed on the Gensini scale revealed significant changes regarding ΔHF and ΔLF/HF values (Table VI).

### Discussion

Our results indicating a decrease of parasympathetic tone and autonomic balance shift towards sympathetic domination follow PTCA are consistent with the findings of Kanadasi et al. [6] and Ohler et al. [15]. However, they are in opposition to the results reported by Tseng et al. [8], who showed that the profile of the HRV parameters recorded before the procedure did not differ significantly from the profile assessed directly after the procedure.

The observed HRV changes as a result of RCA angioplasty are probably associated with sinus node blood supply most commonly from this artery. However, the results of CX and LAD angioplasty indicate that not only sinus node blood supply plays an important role in HRV profile. So far there is no known mechanism that would cause the observed phenomena. It may be

### Table III. Significant changes of the HRV indices following PTCA of the right coronary artery (RCA)

<table>
<thead>
<tr>
<th>Index</th>
<th>Before PTCA</th>
<th>After PTCA</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>HF [un]</td>
<td>0.199</td>
<td>0.165</td>
<td>0.141</td>
</tr>
<tr>
<td>LF/HF [un]</td>
<td>4.310</td>
<td>4.457</td>
<td>6.958</td>
</tr>
<tr>
<td>AR TP [ms²]</td>
<td>18.273</td>
<td>2.296</td>
<td>17.085</td>
</tr>
<tr>
<td>AR LF [un]</td>
<td>0.264</td>
<td>0.029</td>
<td>0.284</td>
</tr>
<tr>
<td>AR HF [un]</td>
<td>0.647</td>
<td>0.043</td>
<td>0.621</td>
</tr>
</tbody>
</table>

### Table IV. Significant changes of the HRV indices following PTCA of the circumflex branch of the left coronary artery (CX)

<table>
<thead>
<tr>
<th>Index</th>
<th>Before PTCA</th>
<th>After PTCA</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Yeh DI [un]</td>
<td>0.033</td>
<td>0.031</td>
<td>0.011</td>
</tr>
<tr>
<td>Yeh II [un]</td>
<td>0.053</td>
<td>0.039</td>
<td>0.032</td>
</tr>
<tr>
<td>Organ BAND [bpm]</td>
<td>9.101</td>
<td>9.245</td>
<td>4.62</td>
</tr>
<tr>
<td>Huey STV [bpm]</td>
<td>208.821</td>
<td>262.248</td>
<td>76.444</td>
</tr>
<tr>
<td>Dalton MABB [ms]</td>
<td>15.733</td>
<td>16.575</td>
<td>7.57</td>
</tr>
<tr>
<td>Dalton SD [ms]</td>
<td>48.741</td>
<td>37.468</td>
<td>27.759</td>
</tr>
<tr>
<td>Zugaib STV [un]</td>
<td>0.0129</td>
<td>0.0132</td>
<td>0.005</td>
</tr>
<tr>
<td>SDNN [ms]</td>
<td>27.204</td>
<td>18.592</td>
<td>21.329</td>
</tr>
<tr>
<td>rMSSD [ms]</td>
<td>56.239</td>
<td>19.751</td>
<td>51.496</td>
</tr>
<tr>
<td>LF/HF [un]</td>
<td>2.384</td>
<td>2.072</td>
<td>5.632</td>
</tr>
</tbody>
</table>

### Table V. Changes of the means of the HRV parameters (Δ) after PTCA of the individual coronary arteries

<table>
<thead>
<tr>
<th>Index</th>
<th>Δ LAD</th>
<th>Δ CX</th>
<th>Δ RCA</th>
<th>Δ LAD vs. Δ CX</th>
<th>Δ LAD vs. Δ RCA</th>
<th>Δ CX vs. Δ RCA</th>
</tr>
</thead>
</table>
speculated that CX usually supplies the myocardium close to the sinus node compared to LAD. Our observations are not consistent with the findings of Kanadasi et al. [6], who did not find any impact of the location of the artery treated with angioplasty on HRV changes (a relatively small population is a limitation of their study).

One may assume that unequal distribution of individuals with diabetes might affect the findings of the study. It is commonly accepted that diabetic patients and those with diabetes-induced autonomic neuropathy present impaired capacity for vasodilatation and reduced coronary flow. Rich et al. reported an inverse correlation between HRV and diabetes mellitus. However, a study published by Szot et al. [17], like our own analyses, did not reveal a significant impact of diabetes mellitus on the values of HRV parameters in response to PTCA.

Hypothetically, differences in the findings in the particular groups might have resulted from collateral circulation. Its presence may produce only slight improvement in myocardial perfusion following PTCA, contrary to the subjects without collateral circulation presenting a sudden change in myocardial perfusion [18].

Similarly to our study, Rich et al. [16] as well as Tseng et al. did not observe any relationship between Gensini scale and basic HRV profile [8]. In contrast, Hayano et al. noted a significant negative correlation between vagal nerve tone and severity of the lesions in the coronary arteries [19]. Wachowiak-Baszyńska et al. also revealed decreased SDNN and rMSSD in patients with more extensive atherosclerotic coronary lesions [20].

An assessment of the impact of atherosclerotic lesions’ severity on HRV following PTCA in our study was consistent with the findings reported by Osterhues et al. only if the classification proposed by Bruske, Proudfit and Sones was employed [11].

The time period adopted in our study enables us to assume that our findings resulted mainly from the changes in myocardial perfusion. The effects of reperfusion-induced myocardial injury may be also taken into account. It presumes

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**Table VI.** Significant changes of the HRV indices after PTCA in a group of patients with less (GS ≤32) and more advanced atherosclerotic lesions (GS >32), expressed in Gensini scale (GS)

<table>
<thead>
<tr>
<th>Index</th>
<th>GS ≤32</th>
<th></th>
<th>GS &gt;32</th>
<th></th>
<th>Δ GS ≤32</th>
<th></th>
<th>Δ GS &gt;32</th>
<th></th>
<th>p ΔGS ≤32 vs. ΔGS &gt;32</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF [un]</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
</tr>
<tr>
<td>LF/HF [un]</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
<td>Before PTCA</td>
<td>After PTCA</td>
<td>p</td>
</tr>
<tr>
<td>0.222±0.174</td>
<td>0.115±0.132</td>
<td>0.0002</td>
<td>0.181±0.168</td>
<td>0.148±0.145</td>
<td>NS</td>
<td>0.107±0.183</td>
<td>0.033±0.202</td>
<td>0.044</td>
<td></td>
</tr>
<tr>
<td>3.110±3.117</td>
<td>6.877±5.846</td>
<td>0.00001</td>
<td>4.701±4.654</td>
<td>5.547±5.427</td>
<td>NS</td>
<td>−3.767±4.519</td>
<td>−0.846±5.721</td>
<td>0.002</td>
<td></td>
</tr>
</tbody>
</table>

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**Figure 3.** Differences in (∆) Yeh DI index after PTCA

**Figure 4.** Differences in (∆) Dalton SD index after PTCA
that an increase in coronary blood flow may implicate an adverse influence on the myocardium through pressure overload and muscular filaments strain. In consequence, it leads to oedema, excessive constriction or even myocytes’ death [22]. It was shown in experimental settings that many metabolic changes took place after transient coronary artery occlusion such as potassium and adenosine concentration elevation, acidosis, rapid release of free agents and mitochondria overload with calcium and also more pronounced endothelium dysfunction. These phenomena may stimulate functional derervation of the myocardial nervous endings [23]. First, consequences of ischaemia and reperfusion involve the subendocardial layer (the place where the fibre endings of the vagal nerve are located). Then they propagate to subepicardial regions. Thus, the assumption that lowered parasympathetic modulation of the sinus node, in the early period following PTCA, is a consequence of the rapid changes in blood flow. The impact of ischaemia on myocardial performance during angioplasty is seen within a short time period [24]. Meanwhile, a longer period is necessary to restore normal function to the damaged cellular structure or biochemical processes [25]. The findings of the studies published by Kanadasi et al. [6], Osterhues et al. [11] and Wennenbrom et al. [7] showing a gradual increase in parasympathetic tone accompanied by a drop in sympathetic activity in the long-term follow-up suggest a delayed restoration of normal autonomic activity in consequence of successful coronary revascularisation. The results of these reports indicate improvement in vagal nerve function although complete functional restoration was not achieved. This may support the thesis that myocardial ischaemia is not the only mechanism of reduced parasympathetic tone in uncomplicated coronary artery disease.

We are aware of the limitations of our study such as a lack of long-term follow-up and heterogeneous patient population (with concomitant diseases and influenced by medical therapy). However, a lack of many often accepted exclusion criteria was an intentional step to create the most representative group for daily clinical practice.

Conclusions

1. Percutaneous transluminal coronary angioplasty has an impact on the parameters of HRV in the early assessment, i.e. within 24 hours following the percutaneous procedure.
2. Location of the treated coronary artery significantly affects HRV; angioplasty of the left circumflex artery causes a decrease of many time domain parameters but flow restoration in the left anterior descending artery does not manifest in HRV changes. The PTCA of RCA causes changes in the frequency domain that indicate predominance of the sympathetic arm of the autonomic nervous system.
3. The degree of atherosclerotic lesion severity calculated according to the Gensini scale has an impact on the HRV changes following PTCA.

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Wpływ angioplastyki wieńcowej na kształtowanie się zmienności rytmu serca u osób ze stabilną chorobą niedokrwienną serca z uwzględnieniem lokalizacji tętnicy poddanej zabiegowi

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Streszczenie

Wstęp: Kardiologia inwazyjna stworzyła ogromne możliwości terapeutyczne. Nadal jednak brakuje skutecznych, nieinwazyjnych i łatwo dostępnych metod diagnostycznych służących wyselekcjonowaniu osób z podwyższonym ryzykiem rozwoju chorób sercowo-naczyniowych, a co za tym idzie – ukierunkowaniu dalszej diagnostyki i zminimalizowaniu czasu potrzebnego do wdrożenia optymalnego modelu leczenia. Na uwagę zasługuje analiza zmienności rytmu serca (ang. heart rate variability, HRV), jednakże zjawiska leżące u podłoża zmian HRV związanych z niedokrwieniem i poprawą ukrwienia nie zostały dotychczas wyjaśnione.

Cel: Ocena wpływu skutecznej angioplastyki wieńcowej na HRV u osób ze stabilną chorobą niedokrwienną serca (ang. ischaemic heart disease, IHD) z uwzględnieniem lokalizacji naczynia poddawanego zabiegowi.


Wyniki: W wyniku PTCA w całej ocenianej populacji odnotowano spadek mocy widma HF (p=0,002) z jednoczesnym wzrostem wskaźników: LF/HF (p=0,0002) i w3 (p=0,048). Angioplastyka PTW spowodowała spadek mocy całkowitej widma: AR TP (p=0,017), wzrost aktywności współczulnej: AR LF (p=0,007), LF/HF (p=0,018), i spadek przewspółczulnej: HF (p=0,031), AR HF (p=0,014). W wyniku PTCA GPZ nie zaobserwowano istotnych statystycznie zmian wskaźników HRV. Z kolei angioplastyka GO spowodowała obniżenie wielu wskaźników czasowych: Yeh DI (p=0,005), Yeh II (p=0,017), Organ BAND (p=0,031), Huey STV (p=0,013), Dalton MABB (p=0,015), Dalton SD (p=0,015), Zugaib STV (p=0,005), SDNN (p=0,044), RMSSD (p=0,025) wraz z wzrostem współczynnika LF/HF (p=0,044). Analiza porównawcza delt parametrów HRV uzyskanych w wyniku PTCA poszczególnych tętnic wieńcowych (Δ = wartość wskaźnika przed PTCA – wartość po PTCA) potwierdziła zjawiska zaobserwowane w analizach przeprowadzonych dla pojedynczych naczyń.

Wnioski: Zabieg angioplastyki naczyń wieńcowych wpływ na kształtowanie się parametrów zmienności rytmu zatokowego we wcześniejszej ocenie, to jest w okresie 24 godz. od zabiegu. Lokalizacja tętnicy poddanej rewaskularyzacji ma istotny wpływ na HRV; angioplastyka GO lewej tętnicy wieńcowej wywołuje obniżenie wielu wskaźników czasowych, udrożnienie GPZ nie przekłada się na zmiany HRV, PTCA PTW powoduje zmiany w domenie częstotliwościowej świadczące o przewadze składowej współczulnej układu autonomicznego.

Słowa kluczowe: zmienność rytmu serca, angioplastyka wieńcowa, stabilna choroba wieńcowa

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