Coefficient of heart rate reduction - the newest method for predicting the occurrence of life-dangerous arrhythmias, methods of its correction

Vytryhovskiy Andriy Igorovych

Abstract

The aim: Was to develop and implement the concept of prevention and treatment of fatal complications in patients with post-infarction cardiosclerosis based on the evaluation of the heart rate slowdown.

Materials and methods: The article presents an analysis of 100 Holter recordings of patients with post-infarction cardiosclerosis on the background therapy with beta-blockers (bisoprolol) and phosphocreatine (Neoton).

Results: Treatment with bisoprolol leads to a decrease in the rate of acceleration of the cardiac rhythm, the anti-fibrillatory effect of the combination of bisoprolol-phosphocreatine occurs at the expense of more physical protection, namely, an increase in the activity of the parasympathetic system.

Conclusion: Use of phosphocreatine in patients with post-infarction cardiosclerosis and coherent phenomenon of heart rhythm turbulence can optimize the prevention of sudden cardiac death among these individuals.

Keywords: risk factors, ischemic heart disease, arrhythmia, sudden cardiac death, heart rhythm.

Introduction

For many years, ambulatory electrocardiography (ECG) monitoring has been used in risk stratification of post-infarction patients. Assessment of various Holter-based indices gives insight into the autonomic modulation of the cardiovascular system and it has been proven useful in risk stratification in post-infarction and heart failure patients [1-5]. Risk predictors based on heart rate dynamicity such as heart rate variability (HRV) or heart rate turbulence (HRT) have been extensively studied over recent decades [3, 4, 10]. Deceleration capacity (DC) is a new risk stratifier, characterizing heart rate dynamics in the neighborhood of a deceleration.

Decreased DC was proven to be a better risk predictor of mortality in post-infarction patients than left ventricular ejection fraction (LVEF) and standard deviation of normal RR intervals (SDNN) [6]. Although different studies documented that HRV and HRT are influenced by clinical and ECG covariates, and suggested that these associations should be taken into account while using them for risk stratification purposes [6, 7, 8, 11, 12]. The relation of DC to similar variables has not been studied so far. The aim was to develop and implement the concept of prevention and treatment of fatal complications in patients with post-infarction cardiosclerosis based on the evaluation of the heart rate slowdown. A sample of 100 patients who were hospitalized in the Ivano-Frankivsk regional clinical hospital with a diagnosis - ischemic heart disease, postinfarction cardiosclerosis complicated by heart rhyme type ventricular arrhythmia. Patients were divided into two groups receiving bisoprolol and the second to the above preparation was added phosphocreatine. The scheme of treatment was treatment - beta-blockers (bisoprolol) and phosphocreatine (neoton). Daily dose of bisoprolol was 0.07 mg / kg body weight, and phosphocreatine (neoton) 1 g treatment for 10 days. The 24-hour Holter ECG recordings were performed between the third and fifth day after admission to evaluate mean heart rate, ventricular arrhythmia, HRV, HRT, and DC. The RR intervals were exported and used in further analysis of HRV, HRT and DC. The HRV analysis was performed in time and frequency domain according to ESC/NASPE guidelines. Holter recordings were performed using «Cardiosens (Medica - HAI, Kharkiv, Ukraine». We used a signal processing technique of phase rectified signal averaging (PRSA) to process sequences of RR intervals obtained from Holter recordings (PRSA algorithm is accessible for noncommercial use from www.prsa.eu). The technique provides separate characterizations of deceleration related modulations, quantified by DC.
For computation of DC, heartbeat intervals longer than the preceding interval are identified as anchors. Subsequently segments neighboring with anchor points are aligned around anchor points and the signal is averaged. A detailed method of DC calculation has been described [5]. Deceleration capacity was categorized into low (> 4.5 ms), medium (4.5–2.5 ms) and high (≤ 2.5 ms) risk categories according to the original publication [8]. Abnormal DC was defined as ≤ 4.5 ms. The study was approved by the local bioethical committee and all patients gave their informed consent.

Materials and Methods
An analysis of changes in the AC and DC indices was performed on the background of the treatment with one and two schemes is presented in Table 1.

Table 1: Dynamics of the heart rate acceleration (AS) and slowdown (DC) on the background of treatment.

<table>
<thead>
<tr>
<th>Before treatment (bisoprolol)</th>
<th>DC(24 - hours), mc</th>
<th>DC(24 - hours), mc</th>
<th>AC(AC), mc</th>
<th>DC(AC), mc</th>
<th>AC(night), mc</th>
<th>DC(night), mc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before treatment (bisoprolol)</td>
<td>-5.23±1.19</td>
<td>5.95±0.32</td>
<td>-5.21±0.44</td>
<td>5.65±0.33</td>
<td>-6.73±0.62</td>
<td>7.21±0.86</td>
</tr>
<tr>
<td>After treatment (bisoprolol)</td>
<td>-7.17±1.11</td>
<td>7.49±1.12</td>
<td>-6.73±1.67</td>
<td>6.99±1.22</td>
<td>-7.42±1.3</td>
<td>7.99±0.61</td>
</tr>
<tr>
<td>Before treatment (bisoprolol + phosphocreatine)</td>
<td>-6.09±0.21</td>
<td>6.43±0.28</td>
<td>-5.37±0.39</td>
<td>5.99±0.38</td>
<td>-5.99±0.51</td>
<td>6.71±0.41</td>
</tr>
<tr>
<td>After treatment (bisoprolol+phosphocreatine)</td>
<td>-7.2±0.84</td>
<td>7.98±0.39</td>
<td>-6.88±1.26</td>
<td>7.17±0.73</td>
<td>7.23±1.12</td>
<td>8.37±0.33</td>
</tr>
</tbody>
</table>

Notes: *- difference p < 0.01 between before and after treatment.

Table 2: Comparative characteristic of the difference between the absolute values of the deceleration factor (DC) and the heart rate acceleration (AC) on the background of treatment.

<table>
<thead>
<tr>
<th>Before treatment (bisoprolol)</th>
<th>DC i AC (24 - hours), mc</th>
<th>DC i AC (day), mc</th>
<th>DC i AC (night), mc</th>
</tr>
</thead>
<tbody>
<tr>
<td>After treatment (bisoprolol)</td>
<td>0.72</td>
<td>0.41</td>
<td>0.48</td>
</tr>
<tr>
<td>Before treatment (bisoprolol + phosphocreatine)</td>
<td>0.34</td>
<td>0.62</td>
<td>0.72</td>
</tr>
<tr>
<td>After treatment (bisoprolol + phosphocreatine)</td>
<td>0.78</td>
<td>0.29</td>
<td>1.14</td>
</tr>
</tbody>
</table>

Discussion
During the analysis of the influence of different treatment regimens on the heart rate ratios, it was established that both circuits have a fairly prognostic beneficial effect. As exactly bisoprolol itself is, that the combination of bisoprolol with phosphocreatine reduces the rate of acceleration and increases the rate of slowdown in the heart rate, but there is a significant difference in their anti-fibrillation protection. Bisoprolol itself results in a decrease in the rate of acceleration of the cardiac rhythm, the effect of the combination of bisoprolol phosphocreatine occurs at the expense of more physiological protection, namely, an increase in the activity of the parasympathetic system. And this is another important factor in choosing the combination of bisoprolol-phosphocreatine for the treatment of patients with post-infarction cardioclasosclerosis with complicated cardiac rhythm disturbances in the type of ventricular extrasystoles of varying degrees of gradation.

Conclusion
The use of phosphocreatine in patients with postinfarction cardioclasosclerosis and a coherent phenomenon of cardiac rhythm turbulence can optimize the prevention of sudden cardiac death among these individuals. Prospects for further research. An important task is to improve existing and search for new criteria for the primary prevention of sudden cardiac death, screening patients with an increased risk of sudden cardiac death.

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References


