Melatonin — a somniferous option which does not aggravate sleep-disordered breathing in cardiac risk patients: a Holter ECG based study

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Abstract

Background and aim: We hypothesised that melatonin may represent a safe somniferous drug for cardiac patients, and assessed the effects of administering 5 mg of melatonin daily before bedtime for 30 days in patients with coronary artery disease (CAD) regarding changes in the nocturnal breathing pattern.

Methods: Sixty patients with CAD (aged 48–80 years) were randomised to melatonin/placebo treatment in a 2:1 ratio. A Holter ECG-based method (Lifescreen Apnea software) which has been validated as a screening tool for sleep-disordered breathing was used to estimate the apnoea/hypopnoea index (AHI). A 24-h Holter ECG was used to detect nocturnal breathing abnormalities at the beginning and at the end of the observation. The values of estimated AHI (eAHI) £ 15 were classified as optimal (Opt) and those > 15 — as pathological (Pat). A change of the breathing pattern was classified on the basis of the transition between the initial and final eAHI status (Opt→Opt; Opt→Pat; Pat→Pat, Pat→Opt). The mean initial and final value of eAHI and the percent of Opt and Pat values of eAHI in the initial and final assessment were compared between the melatonin and the placebo groups.

Results: The breathing pattern was not affected by melatonin — the mean initial value of the eAHI in the melatonin group was 18.2 ± 9.4, and in the placebo group 19.6 ± 12.3 (p = 0.64), whereas at the end of the observation in the melatonin group it increased by 1.2 ± 11.3, and in the placebo group — by 1.0 ± 9.0 (p = 0.44).

Conclusions: Hypnagogic treatment with melatonin did not worsen the eAHI in patients with CAD.

Key words: melatonin, sleep apnoea syndrome, coronary artery disease

INTRODUCTION

Owing to the increasing availability of diagnostic tools, sleep apnoea syndrome is often diagnosed in patients with coronary artery disease (CAD), arterial hypertension, arrhythmias or heart failure. It is worth remembering that some drugs which improve the quality of sleep may increase sleep apnoea in cardiac patients.

Melatonin is a hormone of the biological clock, produced by the pineal gland. This compound is used as a hypnotic drug which restores the circadian rhythm of sleep. It is used without the control of a doctor because it is available over the counter, without a prescription. Many studies have shown, however, that it has some influence on the parameters of the circulatory system (heart rate, arterial blood pressure) and that it interferes with some medicines used in cardiology i.e. beta-blockers and calcium antagonists [1, 2]. There are data on the abnormal concentration of endogenous melatonin in people with sleep apnoea, but the effects of exoge-
The most precise method for diagnosing sleep apnoea is polysomnography. This examination cannot be used as a screening method on a mass scale, however, because it is extremely expensive as well as labour- and time-consuming. Hence the need for simpler methods, for example those based on 24-h ECG Holter monitoring. One such option is the Lifescreen Apnea software. This method allows the calculation of the estimated apnoea/hypopnoea index — eAHI [4].

The purpose of our study was to assess the effects of exogenous melatonin on the eAHI values in patients with CAD by means of the Lifescreen Apnea software as compared with a placebo.

**METHODS**

This study was part of a programme which assessed the effects of melatonin on the circadian rhythms of the circulatory system in patients with cardiac disorders [5]. In order to assess the probability of the occurrence of apnoea and hypopnoea episodes, 24-h ECG Holter monitoring was used. The study included 60 patients between 48–80 years of age (mean age 58.6 ± 9.3 years); 31% were women. The inclusion criteria were: CAD confirmed by means of coronary angiography, a nondipper profile in ambulatory blood pressure monitoring before the study, and the patient’s informed consent to take part in the study. The exclusion criteria were: sleep apnoea syndrome diagnosed before the start of the study, taking sleeping pills or melatonin within one month before the beginning of the programme, or heart rhythm other than normal sinus rhythm in the initial ECG examination. The demographic parameters and the concomitant diseases are shown in Table 1, and pharmacological treatment in Table 2.

The patients were randomised in the ratio of 2:1 to take either melatonin or placebo. On the day preceding the initiation of melatonin/placebo, all the patients underwent 24-h ECG Holter monitoring (Pathfinder 700; DelMar Reynolds, Hertford, UK). The participants wrote down the time of onset of

Table 1. Comparison of demographic and clinical characteristics of studied patients

<table>
<thead>
<tr>
<th>Risk factor of atherosclerosis</th>
<th>Melatonin (%) n = 40</th>
<th>Placebo (%) n = 20</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years]</td>
<td>61.15 ± 6.75</td>
<td>53.61 ± 13.6</td>
<td>0.004</td>
</tr>
<tr>
<td>Male gender</td>
<td>31 (77)</td>
<td>13 (65)</td>
<td>0.302</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>25 (62)</td>
<td>14 (70)</td>
<td>0.565</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>23 (57)</td>
<td>17 (85)</td>
<td>0.033</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>13 (32)</td>
<td>4 (20)</td>
<td>0.311</td>
</tr>
<tr>
<td>Smoking</td>
<td>8 (20)</td>
<td>6 (30)</td>
<td>0.387</td>
</tr>
<tr>
<td>Obesity</td>
<td>7 (17)</td>
<td>10 (50)</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Table 2. Comparison of pharmacological treatment in the study group and controls

<table>
<thead>
<tr>
<th>Pharmacological group</th>
<th>Melatonin (%) n = 40</th>
<th>Placebo (%) n = 20</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-blockers</td>
<td>40 (100)</td>
<td>20 (100)</td>
<td>1.0</td>
</tr>
<tr>
<td>Anti-platelet compounds (aspirin and/or clopidogrel)</td>
<td>40 (100)</td>
<td>20 (100)</td>
<td>1.0</td>
</tr>
<tr>
<td>Statins</td>
<td>29 (72)</td>
<td>13 (65)</td>
<td>0.319</td>
</tr>
<tr>
<td>Calcium channel antagonists</td>
<td>20 (50)</td>
<td>5 (25)</td>
<td>0.064</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitors</td>
<td>18 (45)</td>
<td>14 (70)</td>
<td>0.067</td>
</tr>
<tr>
<td>Mononitrites</td>
<td>11 (27)</td>
<td>3 (15)</td>
<td>0.218</td>
</tr>
<tr>
<td>Diuretics</td>
<td>10 (25)</td>
<td>8 (40)</td>
<td>0.187</td>
</tr>
<tr>
<td>Angiotensin receptor antagonists</td>
<td>6 (15)</td>
<td>5 (25)</td>
<td>0.345</td>
</tr>
<tr>
<td>Fibrates</td>
<td>4 (10)</td>
<td>4 (20)</td>
<td>0.282</td>
</tr>
<tr>
<td>Trimetazidine</td>
<td>2 (5)</td>
<td>4 (20)</td>
<td>0.067</td>
</tr>
<tr>
<td>Oral anticoagulants</td>
<td>2 (5)</td>
<td>0 (0)</td>
<td>0.309</td>
</tr>
<tr>
<td>Alpha-adrenolytics</td>
<td>1 (2)</td>
<td>1 (5)</td>
<td>0.611</td>
</tr>
<tr>
<td>Sibutramine</td>
<td>0 (0)</td>
<td>1 (5)</td>
<td>0.153</td>
</tr>
<tr>
<td>Oral antidiabetics</td>
<td>10 (25)</td>
<td>3 (15)</td>
<td>0.375</td>
</tr>
<tr>
<td>Insulin</td>
<td>3 (7.5)</td>
<td>1 (5)</td>
<td>0.128</td>
</tr>
</tbody>
</table>
sleep and the time of awakening, to the nearest 30 min. Since the patients were included in this study regardless of the quality of their sleep, no questionnaires assessing this parameter were used. From Day 1 to Day 30, the patients were obliged to take one tablet — 5 mg of melatonin/placebo — every day between 9 pm and 11 pm (more or less one hour before their planned time of going to sleep). On Day 30 (the last day of melatonin/placebo taking) the Holter ECG examination was repeated and the patients were again asked to write down the times of the onset of sleep and of waking up. In order to assess the probability of the occurrence of apnoea and hypopnoea episodes during sleeping, the Holter ECG recordings were analysed by means of the Lifescreen Apnea software option. The method that was used for the evaluation of this risk is based on the detection of episodes of deceleration and acceleration of sinus rhythm on the apnoea-wakening borderline, and on the analysis of field variation under the curve of the QRS complexes as an exponent of chest movements during sleep [4]. Although the entire analysis was performed automatically, a researcher working with the software and inputting the periods of sleep time for every patient was unaware of the treatment applied to the participant of the study. For every patient, we gave the initial result (from Day 0) and the final result (from Day 30) in the form of the eAHI, reporting the number of apnoea and hypopnoea episodes per hour. According to the information given by the producer of the Lifescreen Apnea software, the values of eAHI < 5 were regarded as normal, ≥ 5 and ≤ 15 — as borderline, and > 15 — as abnormal. For the purposes of this study, we decided that the values ≤ 15 would be considered to be optimal (Opt), and the values > 15 to be pathological (Pat). According to this assumption, all the patients, both those taking melatonin and those taking placebo, were divided into the following four categories, on the basis of the initial and the final value of eAHI: Opt→Opt and Pat→Pat, which meant no change in the qualitative eAHI through the duration of the study; or Opt→Pat, meaning a change from an optimal value to a pathological one; or Pat→Opt, meaning a change from a pathological value to an optimal one.

For every patient, we calculated the difference between the initial and the final value of eAHI. The protocol of the study was approved by the Local Commission for Bioethics (Resolution no. RNN/50/04/KE dated February 10, 2004), and all the patients signed an informed consent to take part in this study. The study complied with the Declaration of Helsinki and with the Declaration of Tokyo.

**Statistical analysis**

The results are presented as mean ± SD or numbers and percentages. The t-test for independent samples (unpaired data) was used to compare the melatonin group with the placebo group. The t-test for dependent samples (paired data) was used to compare the initial vs the final results in both groups. The normality of the distribution of the eAHI values was checked by means of the Kolmogorov-Smirnov test. The fractions constituted by the patients from the respective categories according to the initial and final eAHI values between the melatonin and the placebo group were compared by means of the χ² test. A p value < 0.05 was considered significant.

**RESULTS**

All the patients who took part in the study tolerated the melatonin/placebo tablets well, and no one withdrew from the study before its completion.

At baseline, the percentage of patients with optimal eAHI in the melatonin group was 37.5%, and in the placebo group it was 35%; the remaining patients had pathological values. The majority of patients, 50% in the melatonin and 55% in the placebo group, were categorised as having both initial and final eAHI above 15, i.e. pathological. A change in the category according to the eAHI from optimal to pathological occurred in three patients taking melatonin and in one patient receiving placebo. The distribution of the specific categories established on the basis of the baseline and final eAHI was not significantly different between the melatonin and the placebo group (p = 0.72) (Table 3). The mean baseline value of the eAHI in the melatonin group was 18.2 ± 9.6, and in the placebo group 19.6 ± 12.3 (p = 0.64), whereas at the end of the observation in the melatonin group it increased by 1.2 ± 11.3, and in the placebo group it decreased by 1.0 ± 9.0 (p = 0.44) (Table 4). Although patients with an optimal baseline eAHI value had a higher final eAHI value both in the melatonin group and in the placebo group, this difference

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**Table 3.** Comparison of categories according to the value of initial and final estimated apnoea/hypopnoea index (eAHI); p = 0.72

<table>
<thead>
<tr>
<th>Category</th>
<th>Melatonin (%) n = 40</th>
<th>Placebo (%) n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opt→Opt: Initial eAHI &lt; final eAHI</td>
<td>12 (30): 5 (12)</td>
<td>6 (30): 4 (20)</td>
</tr>
<tr>
<td></td>
<td>Initial eAHI &gt; final eAHI</td>
<td>7 (18)</td>
</tr>
<tr>
<td></td>
<td>Initial eAHI &gt; final eAHI</td>
<td>8 (20)</td>
</tr>
<tr>
<td>Opt→Pat</td>
<td>3 (7)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Pat→Opt</td>
<td>5 (13)</td>
<td>2 (10)</td>
</tr>
</tbody>
</table>

Opt→Opt — no change from the initial optimal category; Pat→Pat — no change from the initial pathological category; Opt→Pat — deterioration from the optimal to pathological category; Pat→Opt — improvement from the pathological to optimal category
was not significant. In patients with a pathological initial eAHI, both those taking melatonin and those taking placebo, an opposite tendency was observed — the final eAHI value tended to be lower than the baseline one, but this difference did not reach significance (Table 5).

DISCUSSION
The principal finding of our study is that melatonin did not diminish eAHI values, and its action in this respect was weak, comparable to that of placebo. In both groups, the majority of patients had eAHI > 15, and a change of the category of this index occurred in a comparable percentage of patients.

Although no randomised placebo-controlled studies showed that sleeping pills had a detrimental effect on the apnoea index in healthy people, still, in patients with diseases of the circulatory system, great caution is advised due to the danger of causing hypoxia to such vulnerable organs as the myocardium and the brain.

The reduction of upper airway muscle tone, and impaired arousal responses to airway occlusion or hypoxia are the most important mechanisms of worsening sleep breathing following benzodiazepine use [6]. Also zolpidem, flurazepam or sodium oxybate have been reported to improve sleep but at the expense of a trend towards increased apnoea [7, 8]. Melatonin would seem safe as a sleeping drug because of its natural origin. In many countries, it is available over the counter, without prescription, and can be taken by patients without the control of a doctor.

To date, there have been no studies on the effects of exogenous melatonin on breathing disorders during sleeping. Patients with obstructive sleep apnoea diagnosed on the basis of polysomnography have decreased concentrations of this hormone [3]. On the other hand, using the same methodology, Brzecka et al. [9] found that patients with obstructive apnoea can be divided into those with a deficit or excess of endogenous melatonin, and the latter group had worse parameters of sleeping. Hence, it is vital to determine whether subjects with CAD, concomitant arterial hypertension, obesity or heart failure can safely take melatonin, especially since breathing disorders in this group are more frequent than in the general population [10], which was confirmed also by this study. It has to be emphasised, however, that the method used in our study did not allow to determine precisely the proportion between the obstructive and the central type of apnoea, which may co-exist in cardiac patients.

One of the criteria for inclusion in our study was a non-dipper profile in 24-h arterial blood pressure monitoring; this is a state which often co-exists with sleep apnoea, and we believe that this criterion might have had an influence on the frequency of detecting higher eAHI values among our patients [11, 12]. However, the very presence of CAD and some of its risk factors (arterial hypertension, obesity) predispose people to breathing disorders, and the main purpose of this study was to assess the safety of taking melatonin by patients with a cardiac risk. Our study excluded patients with atrial fibrillation, with numerous ventricular extrasystoles, and after the implantation of electrotherapy devices, because these conditions would make it impossible to analyse the ECG recording for the purpose of evaluating the probability of apnoea.

The method of detecting apnoea by means of the software for analysing ECG Holter recordings was compared to polysomnography, and this comparison showed that the Lifescreen Apnea software effectively detects breathing disorders during sleeping, and the conformity of the results is high, especially at the values of the eAHI > 18 [13]. Also, intra-patient reproducibility of the results during 48-h ECG monitoring was confirmed [14]. It should be emphasised that reproducibility is determined not only by the technical aspects of an

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Melatonin</th>
<th>Placebo</th>
<th>Significance melatonin vs placebo</th>
</tr>
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<tbody>
<tr>
<td>Baseline eAHI</td>
<td>18.2 ± 9.6</td>
<td>19.6 ± 12.3</td>
<td>0.64</td>
</tr>
<tr>
<td>Final eAHI</td>
<td>19.4 ± 12.4</td>
<td>18.5 ± 11.1</td>
<td>0.79</td>
</tr>
<tr>
<td>Change of eAHI</td>
<td>1.2 ± 11.3</td>
<td>1.0 ± 9</td>
<td>0.44</td>
</tr>
<tr>
<td>Significance initial vs final eAHI</td>
<td>0.50</td>
<td>0.61</td>
<td>–</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Baseline eAHI</th>
<th>Final eAHI</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melatonin group with optimal value of initial eAHI (≤ 15)</td>
<td>7.6 ± 3.4</td>
<td>12.8 ± 13.9</td>
<td>0.17</td>
</tr>
<tr>
<td>Melatonin group with pathological value of initial eAHI (&gt; 15)</td>
<td>24.5 ± 5.6</td>
<td>23.4 ± 9.5</td>
<td>0.52</td>
</tr>
<tr>
<td>Placebo group with optimal value of initial eAHI (≤ 15)</td>
<td>6.2 ± 4.2</td>
<td>9.3 ± 8.6</td>
<td>0.14</td>
</tr>
<tr>
<td>Placebo group with pathological value of initial eAHI (&gt; 15)</td>
<td>26.7 ± 8.5</td>
<td>24.5 ± 9.0</td>
<td>0.26</td>
</tr>
</tbody>
</table>
examination based on ECG, but also by the very phenomenon of apnoea. The transient nature of apnoea episodes was signal-
led by Skinner et al. [15], who suggested a repetition of poly-
somnography in patients with an acute coronary syndrome in order to verify the accuracy of the first result.

Limitations of the study
The main limitations of our study are the small sample of patients and the fact that we used an indirect method of assessing breathing abnormalities during sleeping. In order to calculate how many patients were needed to make the tendencies observed in the melatonin-induced changes in the eAHI statistically significant, we extrapolated the study group theoretically. We obtained significant values of p < 0.05 when the study group was multiplied by six, which suggests that an analogous study should be conducted on 360 patients. How-
ever, automatic multiplication of the same study group is not a good method of statistical simulation, because it implies constant variance. After using software for random sampling of an additional 300 people, we obtained random variables; when we analysed these, it turned out that there were still no significant differences between the mean ± SD eAHI values between the melatonin and the placebo group. Another li-
mitation of this study is the imbalance in the distribution of the age of patients as well as the higher prevalence of arterial hypertension and obesity in the placebo group compared to the melatonin group — this disproportion is a result of the relatively small size of our control group.

In the available literature, we have not found studies con-
cerning the effects of exogenous melatonin on the worsening of breathing parameters during sleeping, although the topic of the influence of hypnotic drugs has been vividly discussed re-
cently [16]. Also studies on ramelteon, a synthetic melatonin receptor agonist, did not demonstrate any significant effects of this drug on the mean AHI values in comparison with the pla-

cebo group [17]. Since the abovementioned study was per-
formed by means of polysomnography, it should be emphasised that also assessment of melatonin neutrality in this respect should be conducted and confirmed by this method. Authors of earlier studies on melatonin only warn doctors against giving this supplement to patients who take calcium channel antago-
nists, because combining these two caused an increase in arte-
rional blood pressure and heart rate [2, 5, 18].

CONCLUSIONS
In this placebo-controlled study, we showed that hypnagogic treatment with melatonin was safe and well tolerated. Our data suggests that in patients with CAD melatonin does not worsen eAHI — an indirect indicator of abnormal breathing during sleeping. Interestingly enough, the Holter ECG-based method of apnoea detection (using the Lifescreen Apnea software) revealed sleep-disordered breathing in two thirds of patients with CAD.

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Conflict of interest: none declared

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Melatonina — opcja leku nasennego, który nie nasila bezdechu sennego u pacjentów z ryzykiem kardiologicznym: badanie oparte na holterowskiej ocenie EKG

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Streszczenie
Wstęp: Syntetyczne leki nasenne mogą nasilać zaburzenia oddychania podczas snu u pacjentów z chorobami sercowo-naczyniowego. Melatonina — hormon szyszynki — jest powszechnie używana jako środek pomagający w zasypianiu, ale jej wpływ na zjawisko bezdechu nie był badany.

Cel: Celem pracy była ocena, czy melatonina stanowi bezpieczny środek nasenny dla pacjentów z obciążeniem kardiologicznym. Oceniono efekt przyjmowania 5 mg melatoniny przez pacjentów z chorobą wieńcową (CAD) codziennie przed snem przez 30 dni, ze zwróceniem uwagi na zmiany szacowanego wskaźnika bezdechu/spłyconego oddychania ocenianego na podstawie holterowskiego badania EKG z godzin nocnych.

Metody: Pacjentów (n = 60) z CAD (wiek 48–80 lat, śr. 58,6 ± 9,3 roku; 31% kobiet) losowo przydzielono do grup przyjmujących melatoninę lub placebo w proporcji 2:1. U żadnego pacjenta nie rozpoznano wcześniej zespołu bezdechu sennego. Nadciśnienie tętnicze było obecne u 67%, cukrzyca u 28%, a otyłość u 28% osób. Oprogramowanie LifeScreen Apnea oparte na holterowskim zapisie EKG zostało użyte do oszacowania wskaźnika eAHI definiowanego jako liczba bezdechów i liczba epizodów spłyconego oddychania w ciągu 1 godziny snu. Całodobowe zapisy holterowskie wykonano w dobie przed zażyciem pierwszej dawki melatoniny oraz w ostatnim dniu przyjmowania. Wartości eAHI £ 15 (prawidłowe i graniczne) sklasyfikowano jako optymalne (Opt), a wartości eAHI > 15 — jako patologiczne (Pat). Zmiana profilu oddychania podczas snu została zakwalifikowana do jednej z 4 kategorii na podstawie wyjściowego i końcowego wyniku eAHI (Opt→Opt; Opt→Pat; Pat→Pat; Pat→Opt). Porównano średnie wyjściowych i końcowych wyników eAHI oraz odsetek Opt i Pat wyników eAHI na początku i pod koniec badania w grupie melatoniny i placebo.

Wyniki: Melatonina była dobrze tolerowana i wszyscy pacjenci ukończyli badanie. Podawanie melatoniny przed snem nie wpływało na eAHI. Na początku obserwacji średnia wartość eAHI w grupie melatoniny wynosiła 18,2 ± 9,4, a w grupie placebo 19,6 ± 12,3 (p = 0,64), a pod koniec badania wzrosła o 1,2 ± 11,3 w grupie melatoniny i o 1,0 ± 9,0 (p = 0,44) w grupie placebo.

Wnioski: W niniejszym badaniu podawanie melatoniny jako leku nasennego u osób z CAD było bezpieczne, dobrze tolerowane i, jak sugerują wyniki, nie pogarszało szacowanego wskaźnika bezdechów. Należy podkreślić, że na podstawie 24-godzinnego monitorowania EKG zaburzenia oddychania w czasie snu wykryto u 2/3 pacjentów z CAD.

Słowa kluczowe: melatonina, zespół sennego bezdechu, choroba wieńcowa

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