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A Comparative Study of the Efficacy of Cognitive Behavioral Therapy and Zopiclone in Chronic Insomnia

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Objectives. To assess the efficacy of the cognitive behavioral therapy of insomnia (CBT-I) compared with pharmacotherapy in chronic insomnia in the Russian population. **Materials and methods.** A crossover trial was performed in 42 patients with chronic insomnia (14 men and 28 women) aged 29–80 years old, who received two courses of treatment: using zopiclone and using an educational method with elements of CBT-I. All patients underwent nocturnal polysomnography studies. Treatment efficacy was evaluated using the Insomnia Severity Index, the Pittsburgh Sleep Quality Index questionnaire, the Dysfunctional Beliefs about Sleep Scale, the Sleep Hygiene Index, and the Beck Depression Scale. The efficacy of medication and nonmedication treatment methods in insomnia were found to be comparable. The insomnia severity index after CBT-I decreased by 3.6 (from 17.7 ± 5.3 to 12.8 ± 5.1) points, compared with a decrease by 4.9 (from 16.5 ± 5.8 to 12.9 ± 6.2) points after courses of zopiclone ($p < 0.05$). However, after two weeks, treatment results persisted only after use of CBT (12.9 ± 6.2 points); scores increased to 15.5 ± 4.6 points by the end of zopiclone administration. In addition, CBT-I was followed by significant decreases in values on the Beck Depression Scale (from 11.8 ± 6.9 to 8.5 ± 7.0 points), the Sleep Hygiene Index (from 26.9 ± 7.5 to 23.9 ± 5.7 points), the Dysfunctional Beliefs about Sleep Scale, (from 104.9 ± 29.7 to 84.4 ± 34.2 points) ($p < 0.05$). Patients responding to CBT-I were younger than nonresponders (40.5 ± 12.9 and 57.2 ± 11.7 years, respectively, $p < 0.05$), such that young age can be regarded as a predictor for CBT-I being effective. **Conclusions.** Treatment of chronic insomnia using CBT was as effective as pharmacotherapy, its use was accompanied by additional improvements in emotional status, and its effects lasted longer.

Keywords: chronic insomnia, cognitive behavioral therapy of insomnia, zopiclone.

Chronic insomnia is regarded as a common sleep disorder which significantly degrades the quality of life of treatment-resistant patients and is associated with serious somatic diseases.

The prevalence of chronic insomnia in the general population is at least 6%, which is comparable with that of diabetes mellitus. However, not all doctors have a clear view of who should treat chronic insomnia and how. This leads to underdiagnosis and incorrect treatment based on prescription of benzodiazepines. Economic losses due to insomnia arise from the fact that poorly sleeping people have decreased work capacity. They are often absent from work because of poor wellbeing and they have an overall three times fewer "effective" working days than healthy people [1].

Chronic insomnia with objectively short durations of nocturnal sleep, apart from degrading patients' quality of life, is associated with an increased risk of developing cardiovascular and metabolic diseases [2]. It has been suggested that this may be due to increased sympathetic nervous system tone, impairments to the body's circadian rhythms, and hyperactivity of the renin-angiotensin-aldosterone system [3, 4].

Hypnotic agents (benzodiazepines, Z drugs, histamine receptor antagonists), are not the drugs of choice when the course of insomnia is chronic [5]. Long histories of the disorder, the development of insomnia maintaining factors, and tolerance to medication-based treatment make chronic insomnia refractory to conventional treatment with hypnotics and justify prescription of antidepressants.

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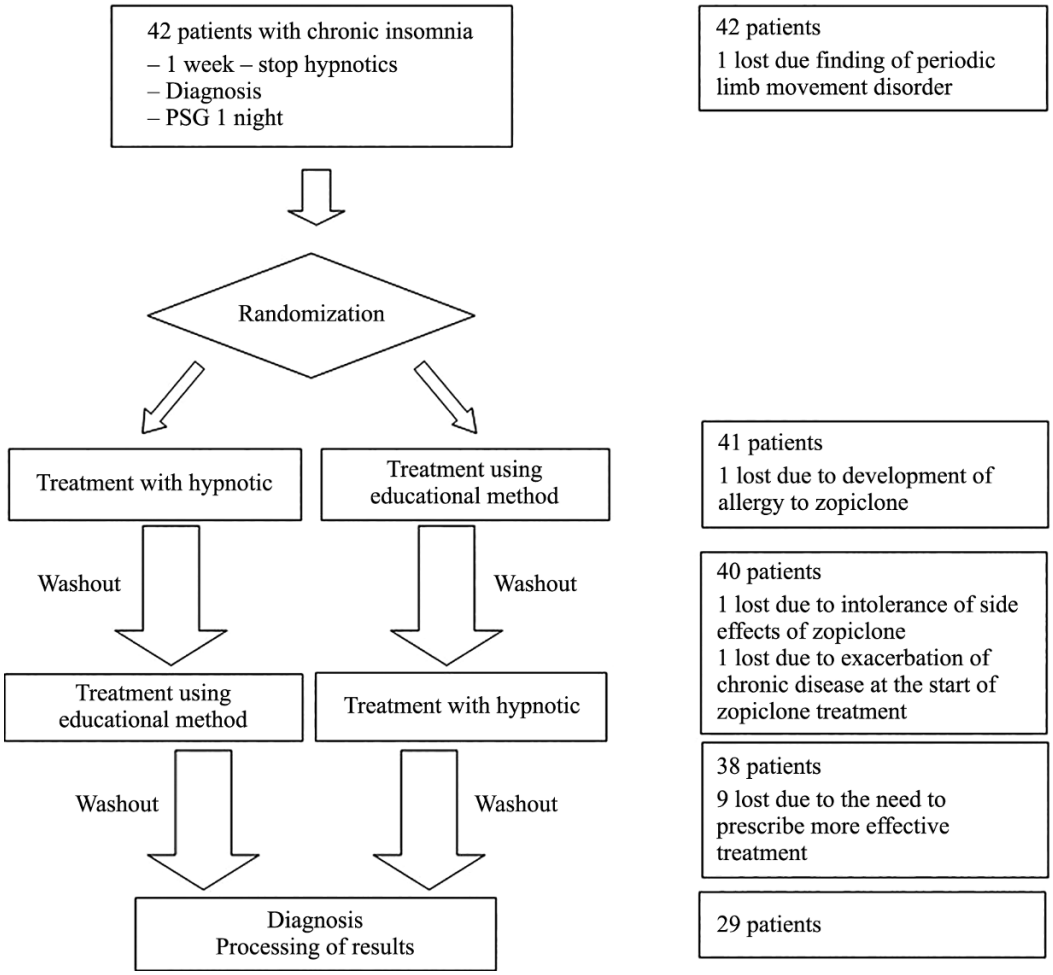

Fig. 1. Scheme of trial and formation of patient pathways.

After courses of medication, there remains a high risk of recurrence, while long-term use of hypnotics increases the risk of habituation and abuse. In addition, drugs for the treatment of sleeplessness have a wide spectrum of side effects [6].

Data from non-Russian guidelines indicate that cognitive behavioral therapy of insomnia (CBT-I) is the method of choice in the treatment of chronic insomnia, and this is confirmed by many clinical trials. The efficacy of this treatment method in the long-term perspective is greater than that of treatment with hypnotic drugs [5]. The use of CBT-I is pathogenetically based, as it corrects dysfunctional beliefs about sleep and associated behavioral factors. The advantages of nondrug treatment are the lack of side effects and the absence of any risk of habituation and abuse. At the same time, complete courses of CBT-I requires large inputs of time from the patient and doctor, so short courses, termed brief behavioral treatment of insomnia (BBT-I), and online courses are increasing in popularity [7].

Cognitive behavioral therapy (CBT) is actively used in many other somatic diseases. Data on the efficacy of its use in pain [8, 9] and obesity [10] have been published. The lack of data on the efficacy of this treatment method in chronic insomnia in the Russian population generates the need to study the comparative efficacies of CBT-I and pharmacotherapy.

Materials and Methods. A total of 42 patients (14 men, 28 women, aged 29–80 (mean 54) years) took part in the trial; all had diagnoses of chronic insomnia in compliance with the criteria of the International Classification of Sleep Disorders, third edition, (ICSD-3) [11]. Participants were selected at the Department of Sleep Medicine, University Clinical Hospital No. 3 (Sechenov First Moscow State Medical University).

The study excluded patients: 1) taking drugs affecting sleep, refusing or being unable to stop taking them for a minimum of one week before the trial started and throughout the trial; 2) with history of abuse of medicines, alcohol, or narcotics; 3) with previously diagnosed endogenous mental disorders; 4) with dementia; 5) pregnant or breastfeeding; 6) working shifts; 7) with other diseases affecting the depth and duration of sleep: moderate-severe obstructive apnea syndrome (apnea/hypopnea index ≥15 episodes/h), restless legs syndrome, periodic limb movement during sleep syndrome

with an index of periodic limb movements of 15 or more episodes/h, or severe pain; and 8) with severe chronic diseases or somatic diseases in exacerbation or decompensation preventing them from taking part in the study. All subjects signed informed consent using a form designed and approved by the Local Ethics Committee at Sechenov University.

The trial was a crossover design in which each subject received two courses of treatment in random order: 1) using zopiclone – 7.5 mg 30 min before going to bed for two weeks; 2) using a standard educational method with elements of CBT-I.

The order of using treatment methods (CBT-I and zopiclone) in each case was determined by randomization using a card method. Patients were placed either in a group in which the first method was use of hypnotic drug and the second was the educational method (group 1) or a group first receiving the educational method and then the hypnotic drug (group 2). The groups had comparable compositions in terms of gender (50% men in group 1 vs. 54% in group 2), age (54.1 ± 15 years in group 1 vs. 47.4 ± 14.1 years in group 2), anthropometric characteristics and polysomnograph (PSG) results, and psychometric and somnological questionnaire results.

After each course of treatment, subjects had two-week rest periods when neither treatment method was used, allowing the stability of therapeutic effects to be evaluated.

Questionnaires were completed before and after each course of treatment, as well as after the two-week washout period. Thus, the total duration of participation in the trial was eight weeks; each participant had six planned visits in which PSG was used, two individual consultations with the doctor, and completed five questionnaires.

Of the 42 patients signing consents to take part in the trial, one patient was lost after PSG due to identification of periodic limb movement disorder, three at the stage of drug treatment due to intolerance of zopiclone (allergic reaction in a patient of group 1, intolerance of side effects of zopiclone (bitter taste in the mouth, group 2), and exacerbation of chronic illness (group 2). The remaining patients tolerated both treatment courses well and reported no deterioration of sleep or overall wellbeing. A total of 38 patients (13 men and 25 women) received whole treatment courses, after which nine were lost to the trial due to the need to prescribe more effective treatment methods for sleep disorders. The remaining 29 patients underwent two-week follow-up periods after the second stage of treatment (Fig. 1).

The structured CBT-I method was the brief behavioral treatment of insomnia method (BBT-I), whose efficacy has been confirmed in the study reported by Buysse et al. [12].

The CBT-I method was delivered as two individual sessions each of 1 h duration once a week. The method included a questionnaire on the patient's sleep problems, a discussion of the mechanisms regulating sleep, the causes of the development and chronicization of insomnia and common strategies for treating it; assessment of sleep diaries kept by the patients; assessment of methods for limiting the time spent in bed and definition of individual regimes; assessment of means of controlling external stimulation; assessment of sleep hygiene rules; issue of reminders including the principles discussed; assessment of relaxation methods. As a relaxation method, patients were given "Relaxation and Rest in Sleeplessness" training guide by author A. A. Tabidze (Psychotherapeutic Pedagogics Science Center, Russian Ministry of Education and Science). Audio recordings were a variant autogenic training method and were of duration 32 min. Patients were told to listen to the audio recording once daily using headphones after going to bed and turning out the light.

Before treatment, all patients underwent PSG during nocturnal sleep in hospital beds (without an adaptation night) to exclude other sleep disorders which might influence its subjective perception such as respiratory disorders and movement during sleep. This was carried out by recording six-channel electroencephalogram traces in monopolar leads Fp_1A_2, Fp_2A_1, C_3A_2, C_4A_1, O_1A_2, O_2A_1, two electrooculogram channels, one electromyogram (EMG) channel from the mentalis muscle, two EMG channels from the tibialis anterior muscles on both sides, the electrocardiogram, and respiratory values during sleep with recording of the oronasal air flow, respiratory movements of the chest and abdominal wall, respiratory sounds, blood oxygen saturations, and the position of the body in bed with parallel video monitoring. Data were interpreted using the 2007 criteria of the American Academy of Sleep Medicine [13] with the 2012 updates [14].

The first visit included structured clinical interview, after which patients completed questions on their own: the Beck Depression Scale [15], the Spielberger Anxiety Scale [16], the Toronto Alexithymia Scale brief version (TAS-20) [17], the five-factor personality questionnaire Big Five Questionnaire (BFQ-2R) [18], the Insomnia Severity Index (ISI) [19], the Pittsburgh Sleep Quality Index questionnaire (PSQI) [20], the Sleep Hygiene Index (SHI) [21], and the Dysfunctional Beliefs about Sleep Scale (DBSS) [22].

At the next four visits, patients completed the Beck Depression Scale, the Spielberger Anxiety Scale, the ISI, PSQI, SHI, and DBSS.

Patients also kept a daily sleep diary throughout the trial, in which they noted the following parameters: time of going to bed, time of getting up, estimated time of going to sleep, number and estimated duration of nocturnal wakings.

Statistical processing. Statistical tables were constructed and data were processed in Statistica 7.0. The per protocol (PP) efficacy of the treatment of insomnia was determined for all patients satisfying the inclusion and exclusion criteria and receiving at least one course of treatment and two sets of diagnostic procedures before and after treatment.

Descriptive data for each patient are presented as means and standard deviations for continuous data. Qualitative and rank data values are presented as percentages.

Significant differences in continuous variables and independent sets, and also for repeat measures, were identi-

TABLE 1. Comparison of PSG Results in Responders and Nonresponders

Parameter	Treatment method					
	CBT-I			zopiclone		
	responders	nonresponders	*p*	responders	nonresponders	*p*
Number of patients, *n*	19	19		8	30	
Index of sleep efficiency, %	65.7 ± 18.8	63.5 ± 17.6	0.78	71.4 ± 10.6	62.8 ± 19.6	0.1
Sleep latency, min	41.1 ± 33.3	44.4 ± 29.4	0.60	48.7 ± 30.4	41.7 ± 31.8	0.97
Waking during sleep, min	105.8 ± 70.4	101.6 ± 76.3	0.75	71.4 ± 40.9	111.7 ± 78.8	0.07
Number of wakings	14.8 ± 6.7	12.5 ± 6.3	0.79	15.9 ± 5.7	12.9 ± 6.8	0.66
SWS stage, %						
1	4.6 ± 4.3	3.4 ± 1.9	0.001	3.4 ± 1.6.	4.1 ± 3.6	0.03
2	60.1 ± 8.1	65.7 ± 8.9	0.43	64.4 ± 6.1	62.2 ± 10.1	0.18
3	19.4 ± 8.2.	15.4 ± 7.3	0.59	13.5 ± 5.0	18.6 ± 8.3	0.17
Fast-wave sleep, %	16.2 ± 6.2	15.0 ± 7.7	0.42	18.6 ± 6.7	14.7 ± 7.0	0.96
Duration of sleep, h	5.6 ± 1.7	5.7 ± 1.6	0.92	5.6 ± 1.1	5.7 ± 1.8	0.22

fied using Student's *t* test after confirming the normality of data distributions using the Kolmogorov–Smirnov method.

The treatment efficacy criteria were: a decrease in ISI by 50% or ≥8 points as compared with pretreatment values and/or a decrease in PSQI by more than 3 points compared with pretreatment scores. The criteria for remission were achievement of an ISI score of ≤7 points and/or PSQI of ≤5 points. These criteria were used in the studies of Buysse et al. and Morin et al. [12, 23]. The absence of treatment response was identified as a decrease in ISI by <50% or 8 points as compared with pretreatment values or a decrease in PSQI by <3 points compared with pretreatment values. Depending on the response to each treatment method, patients were divided into responders and nonresponders; baseline characteristics were compared for quantitative values using Student's *t* test (when the distribution as normal) or the Mann–Whitney U test (non-normal distributions). Analysis of patients' assessments of the efficacies of the treatment methods were carried out using 2 × 2 linkage tables and the Pearson χ^2 test. When the number of observations in one of the cells of the linkage table was <5, significant differences were evaluated using Fisher's exact test. The critical significance level (p) for testing statistical hypotheses was <0.05.

Results. Assessment of the efficacy of CBT-I using sleep quality questionnaires demonstrated a significant reduction in ISI immediately after treatment, from 17.7 ± 5.3 to 12.8 ± 5.1 points ($p < 0.05$), and a significant reduction in PSQI, from 13.3 ± 3.7 to 10.6 ± 4.5 points ($p < 0.05$) (Fig. 2). Low ISI levels persisted during the two-week washout period, at 12.9 ± 6.2 ($p < 0.05$) points. The efficacy of CBT-I in relation to insomnia maintenance factors was apparent as a decrease in the SHI scale from 26.9 ± 7.5 to 23.9 ± 5.7 points ($p < 0.05$) and a significant decrease in DBSS from 104.9 ± 29.7 to 84.4 ± 34.2 points ($p < 0.05$). Analysis of influences on measures of emotional status demonstrated a significant reduction in values on the Beck Depression Scale, from 11.8 ± 7.0 to 8.5 ± 7.0 points ($p < 0.05$); however, the decreases in scores on the Spielberger situational and trait anxiety scales were not significant (from 45.3 ± 8.9 to 43.8 ± 8.7 points and from 48.9 ± 7.0 to 48.2 ± 8.3 points, respectively). Responses to CBT-I identified 19 patients as responders. There were no side effects.

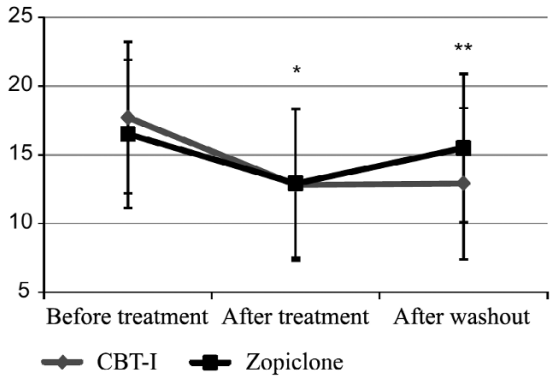

Fig. 2. Dynamics of changes in ISI on the background of CBT-I and zopiclone treatment and two weeks after treatment courses ended ($p < 0.05$). *Significant differences between baseline and end-of-treatment values ($p < 0.05$); **significant differences between baseline and post-washout values ($p < 0.05$).

The effect of zopiclone treatment was apparent as a significant reduction in ISI immediately after treatment, from 16.5 ± 5.8 to 12.9 ± 6.2 points ($p < 0.05$), though

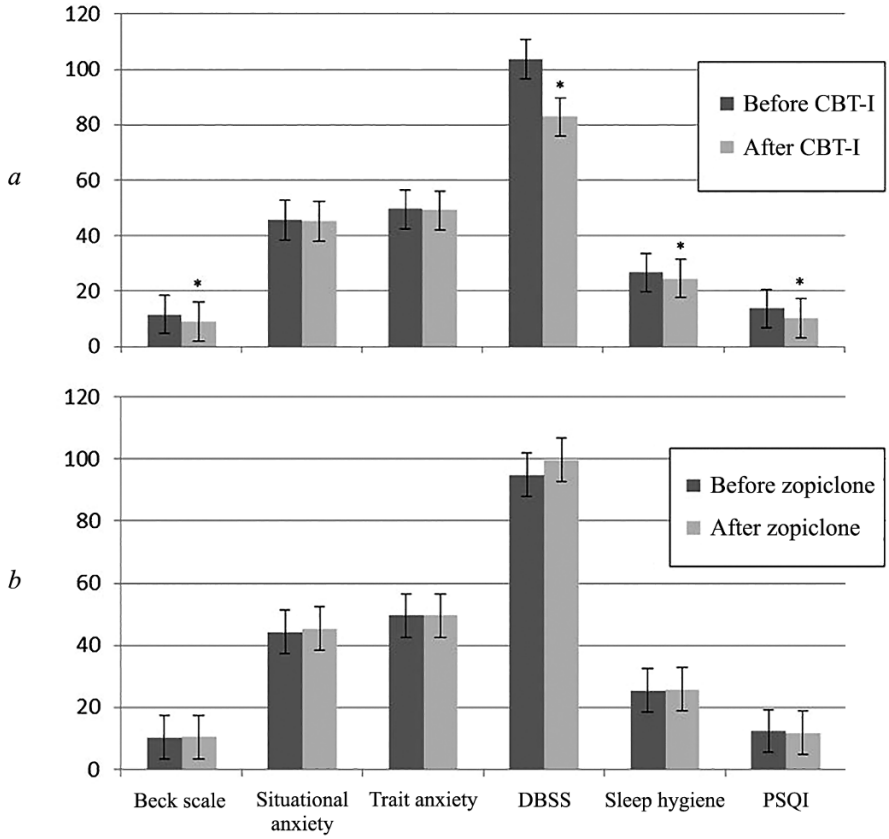

Fig. 3. Dynamics of questionnaire results on the background of treatment with CBT-I and zopiclone. *Significant differences between baseline and end of treatment, $p < 0.05$.

during washout this index increased, to 15.5 ± 4.6 points (the difference between the baseline and post-washout values was not significant). Changes in other scales were also insignificant: PSQI decreased from 12.1 ± 4.1 to 11.7 ± 4.1 points, DBSS increased from 94.0 ± 34.0 to 98.2 ± 35.2 points, SHI from 25.4 ± 5.8 to 25.5 ± 7.0 points, the Spielberger situational and trait and anxiety scales from 43.1 ± 8.8 to 44.5 ± 9.3 points and from 48.3 ± 8.1 to 48.3 ± ± 8.8 points, respectively), the Beck Depression Scale from 9.5 ± 7.3 to 9.6 ± 8.1 points; eight responders were identified. Two cases of intolerance of zopiclone linked with allergic reactions to the drug and severe bitter sensations in the mouth after drug administration were identified.

The efficacies of treatment methods were also evaluated in terms of relative changes to mean values on the sleep quality questionnaire. On the background of CBT-I, the severity of insomnia decreased by 22% on the ISI and 19% on the PSQI. The severity of insomnia on the ISI decreased by 20% on the background of zopiclone, with a 2% decrease on the PSQI (Fig. 3).

Both methods displayed relatively low efficacy, as the value indicated in the criterion (decrease in ISI by eight points and/or PSQI by three points) at the stage of using CBT-I was achieved by only 19 patients, compared with eight at the zopiclone stage, out of a total of 38 patients (50% and 21%, respectively; df = 1; $\chi^2 = 6.95$; $p = 0.084$).

Analysis of the PSG of responders and nonresponders using indicators such as the index of sleep efficiency, sleep latency, nocturnal waking time, the number of nocturnal wakings, the proportions of stages 1–3 slow-wave sleep, the proportion of the fast-wave sleep, and sleep duration showed no significant differences except for the content of stage 1 slow-wave sleep in the two groups (see Table 1).

Analysis of anthropometric characteristics and baseline questionnaire results also showed that there were no significant differences between CBT-I and zopiclone responders and nonresponders, with the exception of mean age in the two groups (40.5 ± 12.9 and 57.2 ± 11.7 years, respectively, $p < 0.05$).

The mean score on the Beck Depression Scale in CBT-I responders was 11.8 ± 5.1 points, compared with 13.0 ± 7.4 points in CBT-I nonresponders; scores in zopiclone responders and nonresponders were 11.7 ± 7.1 and 12.0 ± 6.0 points, respectively.

Mean scores on the Spielberger situational anxiety scale in CBT-I responders and nonresponders were 45.8 ± 9.2 and 44.4 ± 6.3 points and in zopiclone responders and nonresponders 41.7 ± 7.6 and 45.9 ± 8.0 points.

Mean scores on the Spielberger trait anxiety scale in CBT-I responders and nonresponders were 49.7 ± 6.4 and 48.1 ± 9.0 points and in zopiclone responders and nonresponders 47.3 ± 9.2 and 49.7 ± 7.4 points.

Mean scores on the Toronto alexithymia scale in CBT-I responders and nonresponders were 47.4 ± 13.2 and 50.5 ± ± 12.5 points and in zopiclone responders and nonresponders 43.9 ± 6.9 and 50.3 ± 14.1 points.

Mean scores on the DBSS in CBT-I responders and nonresponders were 108.2 ± 24.9 and 103.7 ± 29.8 points and in zopiclone responders and nonresponders 101.4 ± 35.0 and 107.1 ± 26.1 points.

SHI scores in CBT-I responders and nonresponders were 29.6 ± 5.8 and 26.3 ± 6.6 points and in zopiclone responders and nonresponders 25.0 ± 3.5 and 28.8 ± 7.0 points.

PSQI in CBT-I responders and nonresponders were 13.7 ± 3.4 and 14.1 ± 3.1 points and in zopiclone responders and nonresponders 13.1 ± 2.8 and 14.1 ± 3.2 points.

ISI scores in CBT-I responders and nonresponders were 19.3 ± 4.3 and 17.9 ± 5.0 points and in zopiclone responders and nonresponders 15.5 ± 5.3 and 19.3 ± 4.2 points.

For all patients undergoing complete treatment courses, the efficacies of the treatment methods were analyzed in relation to the sequence of presentation. When CBT-I was provided first (group 2), the number of responders was 13/21, compared with 6/17 when it was used second (group 1) (difference not significant). For zopiclone used first (group 1), the number of responders was 4/17, compared with 4/19 when it was used second (group 2) (difference not significant). These results provide evidence that there is no relationship between the efficacy and the order of use of the two treatment methods.

Discussion. The data obtained here confirm the high efficacy of CBT-I in the treatment of chronic insomnia. This is the first Russian clinical trial showing this, while there is a large database of non-Russian trials giving confirmatory results. Thus, studies reported by Morin [23] showed that the use of CBT-I decreased mean ISI scores from 17.3 to nine points, while Sato et al. [24] found a decrease in mean PSQI from 12.7 to 8.9 points. Non-Russian clinical guidelines for the treatment of chronic insomnia gives the CBT-I method the highest levels of evidence rating and clinical efficacy (IA) [5].

The study results confirmed that CBT-I in the longer term is a more effective treatment method for chronic insomnia than courses of zopiclone, which is consistent with data reported by Ma et al. [25].

Mason et al. also [26] noted that an important feature of CBT-I distinguishing it from drug therapy is its influence on the factors maintaining insomnia and concomitant impairments in the emotional domain. The present study also showed that the level of depressive manifestations decreased after courses of CBT-I, in contrast to courses of zopiclone therapy.

Analysis of the anthropometric, polysomnographic and psychometric characteristics of the participants allowed only young age to be identified as a predictor of treatment efficacy. This result devalues one-night PSG as a diagnostic tool for chronic insomnia, while Troxel et al. [27] used PSG data collected over three sequential nights in domestic conditions to show that a longer total duration of sleep was associated with better responses to CBT-I. This negative result is probably due to the fact that one night may be insufficient for evaluation of sleep patterns in chronic insomnia.

The relatively low efficacy of CBT-I can be explained by the fact that the treatment courses were very short and that the method itself was not standardized. The two-week observation periods after completion of treatment courses were also insufficient for assessment of the duration of treatment effects, so longer-term observations are planned with questionnaire studies of participants.

Treatment efficacy in chronic insomnia by CBT-I methods in relation to sleep quality is comparable with that of zopiclone treatment and, in the longer term, greater. Use of CBT-I is accompanied by improvements in measures of patients' emotional status (level of depression), improvements in compliance with sleep hygiene regulations, and changes in dysfunctional beliefs about sleep. Younger age in patients is associated with greater efficacy of CBT-I. The objective characteristics of sleep in patients with insomnia do not provide a predictor of the efficacy of CBT-I or pharmacotherapy.

The treatment of chronic insomnia by CBT is just as effective as pharmacotherapy, and its use is accompanied by additional improvements in emotional state and longer-lasting effects.

The authors have no conflicts of interest.

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