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CLINICAL AND NEUROPHYSIOLOGICAL ANALYSIS OF SLEEP DISORDERS IN ARTERIAL HYPERTENSION AND OPTIMIZATION OF TREATMENT APPROACHES

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Abstract. *Currently, a substantial body of scientific evidence confirms a relationship between sleep disorders, particularly insomnia, and arterial hypertension. However, findings across studies remain inconsistent and sometimes contradictory.*

According to Y. Zhan et al. (2014), the prevalence of arterial hypertension among individuals without insomnia is 37.3%, whereas in patients with insomnia it increases to 48.0%. Moreover, insomnia is observed approximately 1.5 times more frequently in patients with hypertension, suggesting a bidirectional relationship.

A retrospective cohort study conducted in Taiwan (2016) demonstrated that insomnia is associated with a 21% increased risk of developing arterial hypertension. Importantly, objectively measured short sleep duration is strongly associated with elevated blood pressure.

Keywords: *insomnia, arterial hypertension, sleep disorders, polysomnography, neurophysiology*

КЛИНИЧЕСКИЙ И НЕЙРОФИЗИОЛОГИЧЕСКИЙ АНАЛИЗ НАРУШЕНИЙ СНА ПРИ ГИПЕРТОНИЧЕСКОЙ БОЛЕЗНИ С ЦЕЛЬЮ ОПТИМИЗАЦИИ МЕТОДОВ ЛЕЧЕНИЯ

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Аннотация. *В настоящее время накоплено значительное количество научных данных, свидетельствующих о наличии взаимосвязи между нарушениями сна, в частности инсомнией, и артериальной гипертензией. Однако результаты различных исследований остаются неоднозначными и порой противоречивыми.*

Согласно данным Y. Zhan и соавт. (2014), распространённость артериальной гипертензии среди лиц без нарушений сна составляет около 37,3%, тогда как при наличии инсомнии данный показатель увеличивается до 48,0%. Более того, у пациентов с гипертензией нарушения сна выявляются примерно в 1,5 раза чаще, что указывает на вероятное существование двусторонней патогенетической связи.

Результаты ретроспективного когортного исследования, проведённого в Тайване (2016), продемонстрировали, что наличие инсомнии сопровождается увеличением риска развития артериальной гипертензии на 21%. При этом ключевое значение имеет объективно подтверждённое сокращение продолжительности сна, тесно ассоциированное с повышением артериального давления.

Ключевые слова: *инсомния, артериальная гипертензия, нарушения сна, полисомнография, нейрофизиология.*

ARTERIAL GIPERTENZIYADA UYQU BUZILISHLARINING KLINIK VA NEYROFIZIOLOGIK TAHLILI VA DAVOLASH USULLARINI TAKOMILLASHTIRISH

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Annotatsiya. *Bugungi kunga qadar insomniya va arterial gipertenziya o‘rtasidagi bog‘liqlikni tasdiqlovchi katta miqdordagi ma’lumotlar to‘plangan, ammo ayrim tadqiqotlar natijalari ko‘pincha ziddiyatli bo‘lishi mumkin. Ayrim ishlarning natijalari alohida e’tiborga loyiqdir. Masalan, Y. Zhan va hamkasblari (2014) ma’lumotlariga ko‘ra, insomniyasi bo‘lmagan bemorlarda AG chastotasi 37,3% ni tashkil qilsa, insomniyasi bo‘lganlarda — 48,0% ga yetadi. Shu bilan birga, AG bo‘lgan bemorlarda insomniya arterial bosimi normadan yuqori bo‘lmagan shaxslarga nisbatan 1,5 marta ko‘proq uchraydi, bu esa insomniya va AG o‘rtasida ikki tomonlama bog‘liqlik mavjudligini ko‘rsatishi mumkin.*

Tayvan aholisi o‘rtasida o‘tkazilgan retrospektiv kohort tadqiqotiga ko‘ra (2016), insomniyasi bo‘lgan bemorlarda AG rivojlanish xavfi insomniyasi bo‘lmagan bemorlarga nisbatan 21% yuqori ekanligi aniqlangan. Shuningdek, aynan ob’ektiv ravishda o‘lchanadigan qisqa uyqu davomiyligi arterial gipertenziya mavjudligi bilan bog‘liqligi aniqlangan.

Kalit so‘zlar: *Dissomniya, arterial gipertenziya, polisomnografiya, neyroson, nafas mashqlari.*

Introduction.

Sleep disorders, particularly insomnia, are highly prevalent among patients with arterial hypertension. According to epidemiological studies, their prevalence ranges from 19% to 47.9%. At the same time, the prevalence of hypertension among individuals with insomnia ranges from 21.4% to 50.2%, whereas among individuals without sleep disturbances it is significantly lower.

Studies involving ambulatory blood pressure monitoring have shown elevated nighttime blood pressure levels in patients with insomnia. These findings suggest that insomnia may act as a risk factor for hypertension.

The pathophysiological mechanisms linking these conditions include increased activity of the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis, as well as frequent comorbidity with anxiety disorders.

The Pittsburgh Sleep Quality Index (PSQI) is widely used to assess sleep quality. Patients with hypertension typically demonstrate higher PSQI scores, which correlate with systolic and diastolic blood pressure levels as well as with non-dipper circadian patterns.

Both hypertension and insomnia are associated with cognitive impairment. However, the relationship between sleep disturbances and cognitive dysfunction remains controversial due to differences in research methodologies and patient characteristics.

Aim of the Study.

To identify clinical and neurophysiological features of sleep disorders in patients with arterial hypertension at different stages of the disease, and to evaluate their relationship with autonomic regulation, circadian rhythms, and cognitive status, followed by the development of effective approaches to комплекс therapy.

Materials and Methods.

A total of 134 patients diagnosed with arterial hypertension and cognitive impairment were examined in 2024–2025.

Patients were divided into two groups:

Main group (n=70):Mean age — 57.4±1.8 yearsMale-to-female ratio — 1:2.14

Comparison group (n=64):Mean age — 54.5±2.12 yearsMale-to-female ratio — 1:2.16

The main group received standard antihypertensive therapy combined with комплекс correction of sleep disorders. The comparison group received only standard therapy.

All patients underwent:

- clinical examination
- neuropsychological assessment
- EEG
- evaluation of cerebral hemodynamics

Follow-up was performed at:

- baseline (1–3 days)
- 10 days
- 2 months
- 6 months

Table 1.

Parameter	Main Group(n=70)	Comparison Group(n=40)	Parallel Group(n=30)	Control Group(n=32)
Mean Age (years), M±m	54.3 ± 0.89	55.8 ± 1.04	57.1 ± 0.81	56.2 ± 0.92
Females, n (%)	36 (51.4%)	27 (67.5%)	19 (63.3%)	18 (56.3%)
Males, n (%)	34 (48.6%)	13 (32.5%)	11 (36.7%)	14 (43.7%)
Height (cm), M±m	170.4 ± 0.8	168.3 ± 0.7	167.6 ± 0.6	168.6 ± 0.7
Body Weight (kg), M±m	84.1 ± 0.8	86.4 ± 1.2	81.7 ± 1.1	82.7 ± 1.2
Body Mass Index (kg/m^2), M±m	28.7 ± 0.3	29.2 ± 0.3	29.1 ± 0.3	28.5 ± 0.3

The study analyzed the prevalence, clinical features, and complications of sleep disorders in hypertension, as well as the effectiveness of various approaches to their treatment and prevention, with a comparative analysis.

All patients with hypertension were prescribed standard therapy as recommended by a neurologist and cardiologist. Additionally, after assessing the severity and nature of cognitive impairment and sleep disorders, patients in the study group

received a comprehensive treatment program tailored to their clinical and neurophysiological characteristics.

In the comparison group, patients received only standard therapy and generally accepted preventive recommendations without specialized sleep disorder treatment.

In both groups, patients were followed dynamically on days 1-3 and 9-10 of hospitalization, as well as at follow-up periods of 3 and 6 months. The frequency of hypertensive crises, the severity of neurological symptoms, and the incidence of complications were assessed, with subsequent comparative analysis of the data obtained.

All patients included in the study had hypertension associated with sleep disturbances (insomnia, sleep architecture disorganization, non-dipper profile). Therefore, the treatment strategy was based on a two-pronged approach: standard antihypertensive therapy in both groups and pathogenetically based correction of sleep disturbances in the study group.

Standard therapy used in both groups included antihypertensive medications such as angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, calcium channel blockers, and diuretics (as indicated). Individualized chronotherapy was also used, including shifting the administration of certain medications to the evening if necessary. The primary goals of therapy were achieving and maintaining blood pressure below 140/90 mmHg, normalizing the circadian blood pressure profile, and reducing its variability. Along with medication, patients were advised to adhere to sleep hygiene principles, including establishing a stable sleep-wake cycle, limiting stimulant use in the evening, optimizing sleep conditions, and reducing exposure to electronic devices before bed.

Lifestyle modification, including limiting salt intake, weight management, regular moderate-intensity aerobic physical activity, and reducing psychoemotional stress, was an important component of therapy.

This complex of measures was aimed at stabilizing hemodynamic parameters, normalizing circadian rhythms of blood pressure, and reducing the risk of hypertension progression and complications.

Supplemental Complex Therapy in the Study Group

In the study group, pathogenetically justified correction of sleep disorders (Table 2) was added to standard antihypertensive therapy. This included the following:

Table 2.

Component / Treatment Approach	Main / Study Group (n=70)	Standard Antihypertensive Therapy
Standard Antihypertensive Therapy	+	+
Non-pharmacological Correction (Sleep hygiene, CBT-I)	+	-
Pharmacological Correction (e.g., Melatonin, Sedatives)	+	-

• Melatonin at a dose of 2–5 mg at night — to normalize circadian rhythms and reduce sleep latency.

• Reduction of nocturnal sympathetic hyperactivation by restoring circadian mechanisms and improving sleep structure.

• Magnesium supplements at a dose of 300–400 mg/day — to correct neurovegetative imbalance, provide a muscle relaxant effect, and reduce the manifestations of insomnia.

• Electrosleep therapy, lasting 20–40 minutes, in a course of 8–10 sessions — aimed at enhancing cortical inhibition, deepening sleep, and stimulating vagal activity.

• Breathing exercises (diaphragmatic breathing, extended expiration technique) – to increase parasympathetic activity, improve heart rate variability (HRV), and reduce the severity of the non-dipper phenomenon.

Patients were provided with detailed instructions and reminders on the technique, regimen, and duration of five types of breathing exercises. They were trained to independently perform these exercises in an outpatient setting.

Analysis of the dynamics of pathological changes based on EEG data showed that on days 1–2, the parameters of both groups were virtually identical ($p>0.05$), confirming the clinical comparability of the groups at the initial stage of the study.

Based on the results of a two-month observation period, the study group, with the addition of melatonin, magnesium supplements, electrosleep therapy, and breathing exercises to standard therapy, showed a significant reduction in all pathological EEG parameters, demonstrating the high effectiveness of the comprehensive intervention.

Table 3.

EEG Dynamics (%)

Parameter	Main group	Comparison group
Sleep architecture disturbance	20.0%	50.0%
Alpha rhythm desynchronization	14.3%	39.1%
Delta/theta activity increase	11.4%	34.4%
Cortical hyperexcitability	12.9%	37.5%
Paroxysmal activity	1.4%	7.8%
Autonomic imbalance	25.7%	59.4%

Results.

EEG analysis showed no significant differences between groups at baseline ($p>0.05$).

After 2 months, the main group demonstrated significant improvement in all EEG parameters.

• Sleep architecture disturbances: main group — 20.0%, comparison group — 50.0% ($\chi^2 = 13.4$; $p < 0.001$). In the main group, a relative decrease of more than 60% was noted.

• Alpha rhythm desynchronization: main group — 14.3%, comparison group — 39.1% ($\chi^2 = 9.8$; $p = 0.002$), indicating a significant improvement in cortical bioelectrical stability.

• Pathological increase in delta/theta activity: main group — 11.4%, comparison group — 34.4% ($\chi^2 = 9.6$; $p = 0.002$). A decrease in diffuse slow-wave activity is associated with improved cerebral perfusion.

• Cortical hyperexcitability: main group — 12.9%, comparison group — 37.5% ($\chi^2 = 10.1$; $p = 0.001$).

• Paroxysmal activity: main group — 1.4%, comparison group — 7.8% ($\chi^2 = 3.2$; $p = 0.07$). A positive trend was noted, but the difference did not reach statistical significance ($p > 0.05$).

• Signs of autonomic imbalance: main group — 25.7%, comparison group — 59.4% ($\chi^2 = 15.7$; $p < 0.001$). Restoration of autonomic regulation was significantly more pronounced in the main group.

At the initial stage of treatment, statistically significant differences between the groups were not detected ($p > 0.05$). By the 10th day, more pronounced positive dynamics were observed in the main group. By two months, the following were observed in the study group:

• normalization of sleep architecture;

• stabilization of the alpha rhythm;

• reduction in pathological slow-wave activity;

• significant reduction in signs of autonomic imbalance.

In the absence of significant comorbidities, the identified changes are highly likely to be directly associated with long-term arterial hypertension.

Table 4 presents the ECG parameters in the study group (AG, n=70) and the comparison group (TG, n=64) as a percentage with standard error (±).

Table 4.

ECG Indicators

Parameter	Main group	Comparison group
Sinus tachycardia	36.4±1.3	32.1±1.1
Sinus arrhythmia	14.8±1.0	19.0±1.2
P-pulmonale	27.3±1.2	30.9±1.3
P–Q prolongation	18.2±1.1	22.4±1.2
Q–T shortening	12.5±0.9	8.9±0.8
ST depression	20.5±1.2	16.7±1.0
Hypertrophy signs	33.0±1.3	28.2±1.1

ECG analysis revealed that changes reflecting cardiovascular stress manifested differently in patients with hypertension in the study groups.

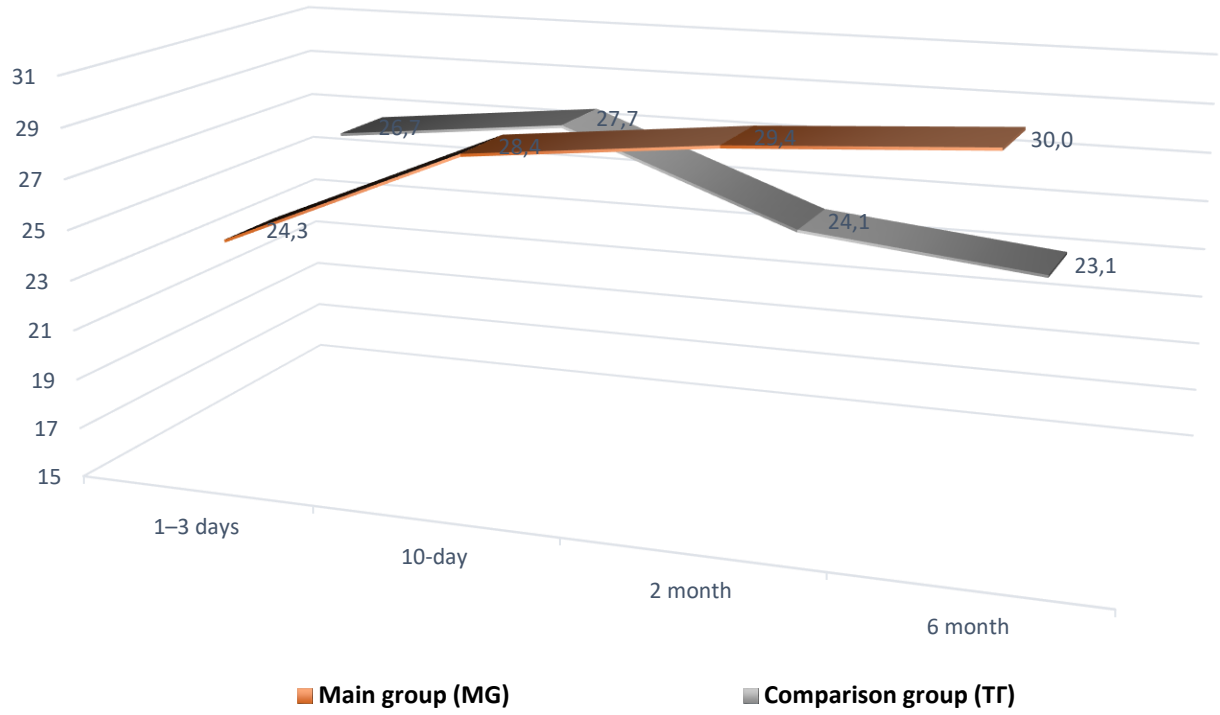
Sinus tachycardia, ST-segment depression, and signs of right heart hypertrophy were more frequently recorded in the study group.

In the comparison group, arrhythmogenic changes (sinus arrhythmia) were more pronounced, as was the P-pulmonale sign, indicating right atrial overload.

Between-group differences ranged from 3% to 7% and were considered statistically significant ($p < 0.05$), indicating different degrees of cardiohemodynamic stress in hypertension depending on the therapeutic approach used.

To evaluate the effectiveness of recommended comprehensive treatment measures over time.

MMSE scores were assessed dynamically on days 1-3, days 7-10, and after two months of follow-up.



Dynamics of cognitive function according to the MMSE scale.

According to the data presented in the diagram (Figure 2), after 10 days, the main group (AG) showed a 17% increase in MMSE scores, while in the comparison group (CG) the improvement was only 3%.

After 2 months, the main group showed a 21% increase in scores, while in the comparison group a 7% decrease was observed.

By the 6th month, the main group showed a 27% increase, while in the comparison group a decrease of 11%.

Analysis of the MMSE scale indicates that the main group showed a steady positive trend in cognitive function against the background of treatment and preventive measures, accompanied by improved sleep quality.

In contrast, the comparison group showed a progressive decline in cognitive scores over time. This fact highlights the importance of a comprehensive pathogenetic approach to the treatment of cognitive impairment associated with hypertension.

Taylor Anxiety Scale - A dynamic analysis of the cognitive and emotional states of patients in the study and comparison groups was also conducted using the Taylor Anxiety Scale.

Anxiety (Taylor Scale)

- **Main group:**
  - decrease by 32.3%
  - sustained improvement
- **Comparison group:**
  - increase up to 41.1%

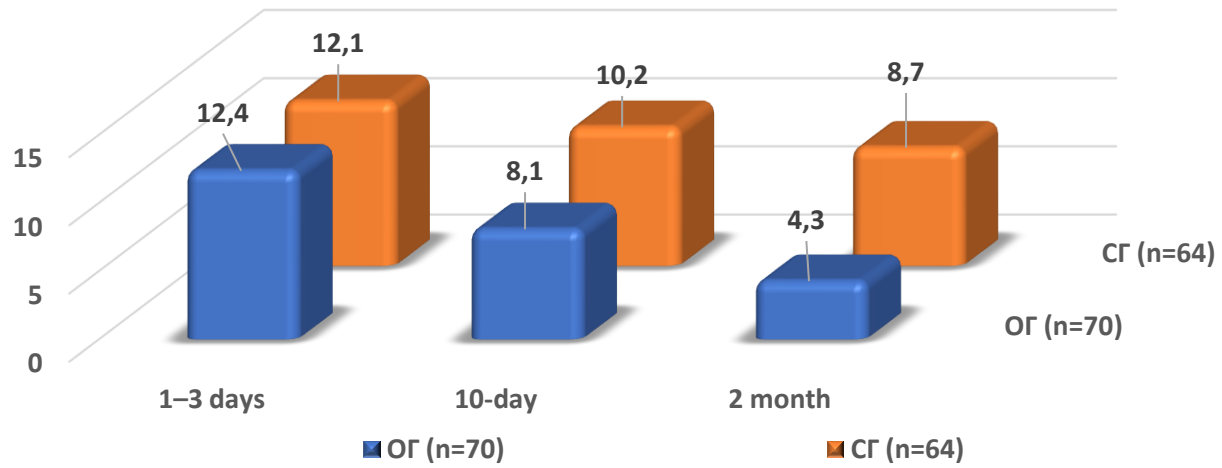
In the main group (AG), the average anxiety level on the Taylor Anxiety Scale was 36.0 points at baseline. After 10 days, this decreased by 32.3%, and after 2 months, to 30.3% of the baseline, indicating significant improvement in psychoemotional state.

In the comparison group (CG), the baseline score was 35.0 points. However, after 10 days, a deterioration (37.5%) was observed, and after 2 months, a further increase in anxiety to 41.1% was observed, reflecting negative dynamics.

These data confirm the effectiveness of the comprehensive treatment and rehabilitation measures used in the main group in the correction of anxiety disorders in hypertension. Sleep Quality Assessment Using the Pittsburgh Sleep Quality Index (PSQI)

Mean PSQI scores (M±m) in patients with hypertension and sleep disorders were analyzed dynamically on days 1–2, day 10, and after two months of follow-up in the study group (AG, n=70) and the comparison group (CG, n=64).

The results demonstrated consistent clinical trends: the study group showed a significant decrease in the total PSQI score, indicating improved sleep quality, while the comparison group showed no significant improvement or only inconsistent changes.



Quality Index (PSQI)

A PSQI score of ≥5 points** was considered a sign of impaired sleep quality.

On days 1–2, no statistically significant differences were found between the groups ($p > 0.05$), confirming their baseline clinical comparability.

By day 10 of observation, the study group (AG) showed a 34.7% decrease in PSQI, while the comparison group (CG) showed a 15.7% decrease. The between-group difference was statistically significant ($t = 2.63$; $p = 0.01$).

By the end of the 2-month observation period, the study group had reduced the PSQI score by 65%, with the final score below 5 points, indicating normalization of sleep quality.

In the comparison group, positive dynamics were also observed, but the score remained at a pathological level (≥5 points). Differences between the groups remained highly significant ($t = 5.48$; $p < 0.001$).

For most parameters, differences between groups reached statistical significance ($p < 0.01$). These data confirm the high neurophysiological efficacy of combination therapy aimed at correcting sleep disorders in arterial hypertension.

Thus, comprehensive correction of sleep disorders in arterial hypertension is a clinically justified, pathogenetically targeted, and practically feasible patient management strategy.

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