ORIGINAL ARTICLE

# PECULIARITIES OF STRUCTURAL CHANGES IN THE BRAIN SUBSTANCE IN PATIENTS WITH ARRHYTHMIAS DEPENDING ON THE SEVERITY OF COGNITIVE DISORDERS

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## ABSTRACT

**The aim:** To evaluate the structural changes of the brain in relation to the formation of cognitive disorders (CD) in patients with arrhythmias

**Materials and methods:** 147 patients with different clinical forms arrhythmias against the background of ischemic heart disease were examine. At the first stage, all patients with arrhythmias assessed cognitive functions. At the second stage, patients were distributed divided into two groups: the main group patients with CD, control – patients without CD. These groups underwent computed tomography examination of the brain.

**Results:** CD were established in 83% patients with arrhythmias. Mild CD were more often diagnosed in patients with persistent form of atrial fibrillation (AF), severe CD – in patients with permanent form of AF and atrioventricular blockade II-III degrees. Neuroimaging changes were found in 73.8% patients with CD and in 36% patients without CD. They were manifested by atrophic changes of the cortex, internal hydrocephalus, a decrease in the density of the brain sub- stance of the periventricular area. In patients with CD, compared to patients without CD, showed lacunar foci with predominant localization in the parietal and frontal lobes of the brain, periventricular and subcortical leukoaraiosis. Multiple correlations were established between CD and structural changes of the brain. **Conclusions:** The increase in the severity of CD in patients with arrhythmias is associated with atrophic changes at the cortical-subcortical level, accompanied by the phenomena of internal hydrocephalus, periventricular and subcortical LA, lacunar foci, with a predominant localization in the frontal-temporal-occipital lobes, in the visual hump and basal ganglia of both cerebral hemispheres.

**KEY WORDS:** computed tomography, arrhythmias, cognitive disorders, leukoaraiosis

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## INTRODUCTION

Violations of higher brain functions are one of the most urgent medical and social problems, as they lead to a decrease in the quality of life, disorders of social and professional activity of a person, and with a long course – to the development of dementia and complete social disadaptation [1-3]. Early diagnosis of cognitive disorders (CD) makes it possible to prescribe timely treatment and postpone the onset of disability. Predemented CD have important clinical significance, as they are more amenable to therapeutic correction [4]. Detection of early, potentially reversible CD against the background of cardiovascular pathology makes it possible to timely identify groups of patients with an increased risk of developing cognitive dysfunction, especially among people of working age.

In most works devoted to the study of CD, their role in the occurrence of arterial hypertension and cerebral atherosclerosis was investigated [5, 6], however, the impact of arrhythmias on the development of cognitive deficits has not been sufficiently studied. In the literature, the question of changes on the part of the nervous system in various forms of arrhythmias has been reflected [7-9], but most of the data on this issue have been obtained in elderly patients, including patients with a history of stroke. The possibilities of the development of CD in young and middle-aged patients with arrhythmias and the absence of obvious morphological cerebrovascular disorders have not been fully explored. The importance of the problem is evidenced by the fact that in March 2018 the joint consensus of the «European Heart Rhythm Association (EHRA), Heart Rhythm Society (HRS), Latin American Heart Rhythm Society (LAHRS), Asia-Pacific Heart Rhythm Society (APHRS) was published on the problem arrhythmias and cognitive functions: what is the best strategy?» [10]. The document deals with approaches to the diagnosis and tactics of treating CD in patients with various arrhythmias.

Possible mechanisms of CD in arrhythmias are: decrease in brain perfusion due to low cardiac output;

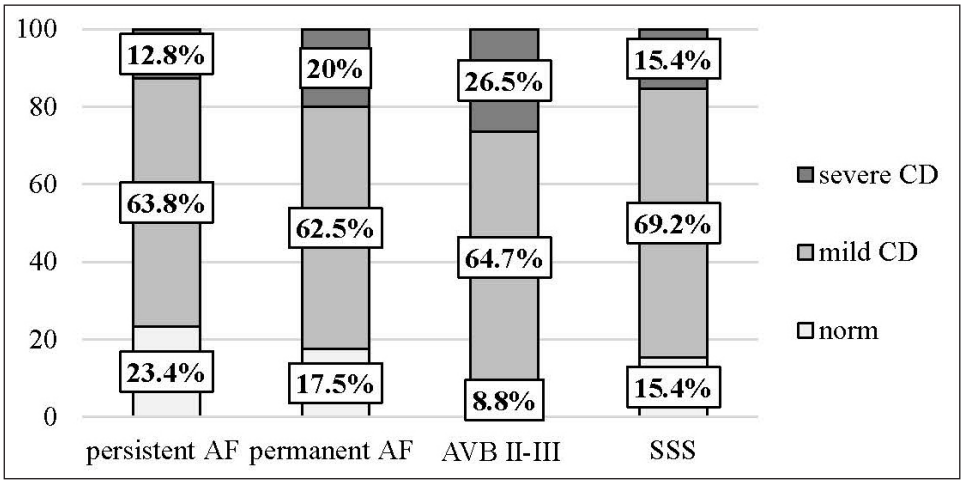


**Fig. 1.** The structure of cognitive disorders in patients of the studied groups

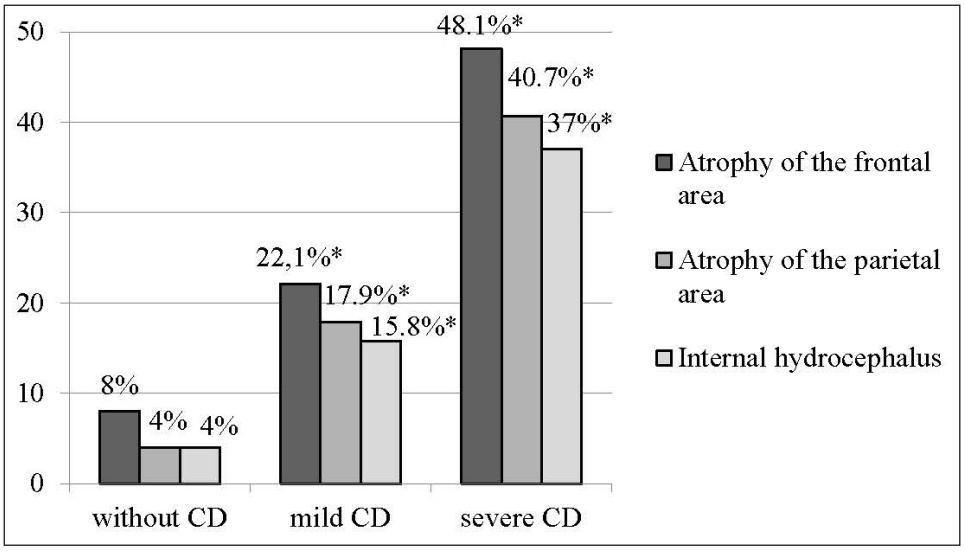


**Fig. 2.** Frequency of neuroimaging changes in patients depending on the severity of CD
* - statistically significant differences when compared with patients without CD ($p<0.05$)

occurrence of leukoaraiosis, «silent» brain infarctions; transient ischemic attacks; hypercoagulation; neurodegenerative cerebral changes [11]. Structural lesions of the brain of a focal or diffuse nature due to ischemia are considered as one of the factors of vascular dementia, and arrhythmias are considered a predictor of CD [12].

An integrated complex approach to the study of the mechanisms of development of CD in patients with arrhythmias, their early diagnosis, the choice of the correct strategy for the treatment of arrhythmias can slow down the progression of cognitive deficits, which will make it possible to improve not only the clinical status of patients, but also their prognosis. The above positions determined the relevance of the chosen direction of research and determined its purpose.

## THE AIM

The aim of our work was to evaluate the structural changes of the brain in relation to the formation of CD in patients with arrhythmias.

## MATERIALS AND METHODS

To solve the set goal of followed 147 patients aged from 30 to 75 years (mean age 62.7 ± 4.6 years) with different clinical forms arrhythmias against the background of ischemic heart disease. Persistent form of atrial fibrillation (AF) diagnostic staged in 47 (32%) patients, permanent form of AF – in 40 (27.2%) patients, atrioventricular blockade (AVB) II-III degrees – in 34 (23.1%) patients, sick sinus syndrome (SSS) – in 26 (17.7%) patients. The group of patients with SSS included patients with persistent sinus bradycardia (42.3%), recurrent sinus auricular block (23.1%), sudden periodic disappearance of the sinus node (sinus node arrest) (15.4%), persistent bradysystolic form of atrial fibrillation (11.5%), the syndrome of «tachycardia-bradycardia» (7.7%).

The criterion for inclusion in the study is arrhythmias was associated with ischemic heart disease. In research not included patients who had acute forms of acute forms of coronary heart disease, expressed female extracardiac pathology, diabetes mellitus, acute history of cerebral circulation. Besides, the study did not include patients with contraindications to proconducting computed tomography (CT) of the brain.

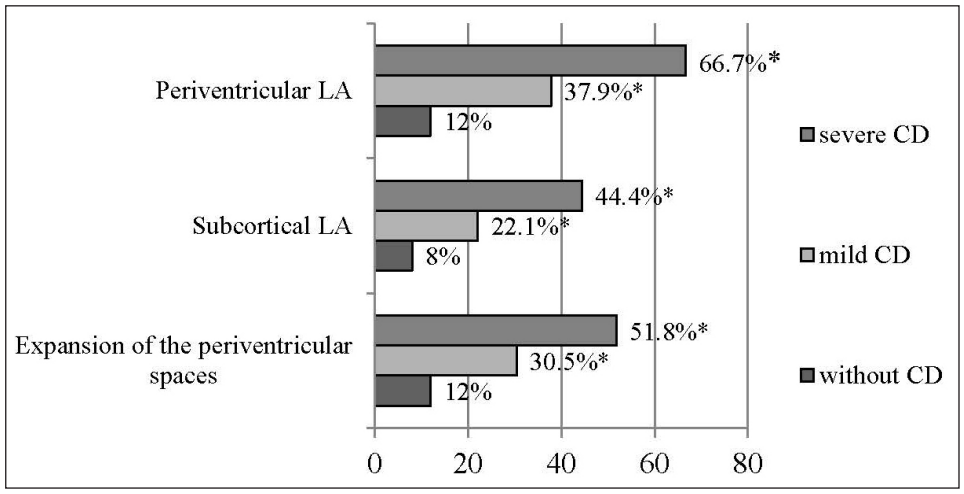


**Fig. 3.** Frequency of diffuse brain changes in patients depending on the severity of CD, * - statistically significant differences when compared with patients without CD (p<0.05)

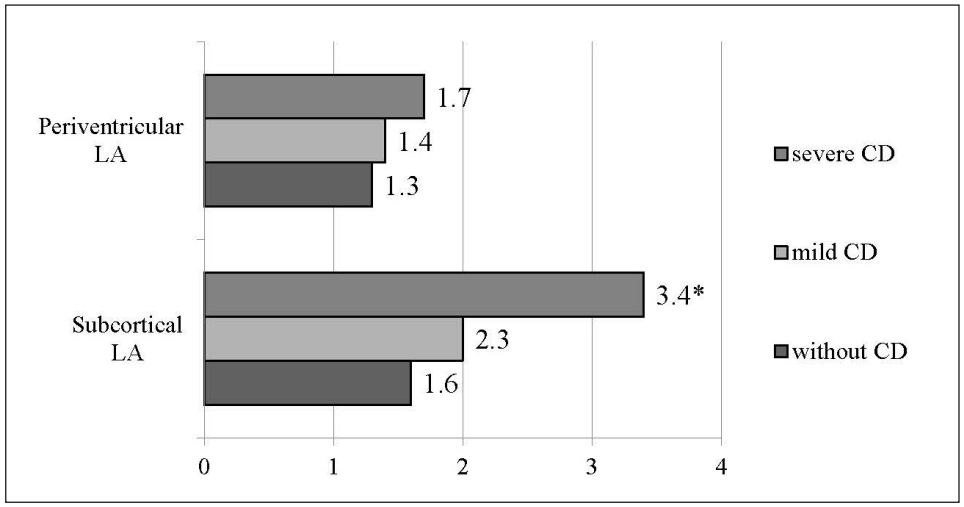


**Fig. 4.** Diffuse changes in the brain (in points) in patients depending on the severity of CD (* p<0.05)

**Table I.** Localization of lacunar foci in patients with arrhythmias depending on the severity of CD

| Localization of lacunar foci | without CD | mild CD | severe CD |
|---|---|---|---|
| Subcortical white matter | 1 (50.0%) | 6 (54.5%) | 8 (44.4%) |
| Basal ganglia and thalamus | 1 (50.0%) | 3 (27.3%) | 5 (27.8%) |
| Pons and cerebellum | 0 | 2 (18.2%) | 5 (27.8%) |
| In total | 2 | 11 | 18 |

**Table II.** Relationship between CD risk and neuroimaging changes in patients

| Indicator | OR | 95% CI | p |
|---|---|---|---|
| CD and external hydrocephalus | 1.61 | 1.14–2.2 | 0.008 |
| CD and internal hydrocephalus | 1.84 | 1.16–2.64 | 0.007 |
| CD and periventricular LA | 3.57 | 1.54–5.82 | 0.006 |
| CD and subcortical LA | 5.68 | 2.21–9.62 | <0.001 |
| CD and lacunar foci | 4.68 | 1.92–7.54 | 0.003 |

The choice of inclusion and exclusion criteria is determined by the following considerations. Thus, ischemic heart disease is considered the most common cause of arrhythmias. Patients with myocarditis, congenital and acquired heart disease and other diseases associated with arrhythmias in many relationships are not comparable with patients suffering from ischemic heart disease, are relatively rare and, obviously, need a separate research. Selection of exclusion criteria is also explained by the need to exclude as much as possible all possible diseases and states capable of being independent cause of brain damage.

The research was carried out in two stages. At the first stage, all patients with arrhythmias assessed cognitive functions using a set of neuropsychological tests. Neuropsychological examination included: Mini-Mental State Examination (MMSE) (Folstein M. et al., 1975), Frontal Assessment Battery (FAB) (Dubois B. et al., 2000), dementia scale of Mattis (DSM) (Mattis S., 1976), test "10 words" (Luria AR, 1969), test "5 words" (Grober E. et al., 1988), verbal association test (Kazdin A., 1982), lines orientation test (Benton A., 1975), test"unpainted objects"(Luria AR, 1969), clock drawing test (Sunderland T. et al., 1989), test of connection of numbers and letters (Trail making test) (Reitan RM, 1958), Boston naming test (Kaplan J. et al., 1978), Spilberger-Khanin anxiety self-assessment scale (Spilberger C.D. et al, 1976), Beck depression scale (Beck A.T. et al, 1975) [13].

At the second stage, patients were distributed divided into two groups: the main group consisted of patients who, in the course of neuropsychological testing, had the presence of cognitive dysfunction; control – patients without CD. These groups underwent computed tomography (CT) examination of the brain on a multisystem spiral computed tomograph «Asteion-4 mod. TSX-021B» (Japan). The severity of internal and external atrophy, periventricular and subcortical leukoaraiosis (LA), lacunar foci (LV) were evaluated.

When analyzing the obtained data, we solved such tasks as describing the studied parameters in groups, evaluating the significance of differences in quantitative and qualitative indicators, checking the empirical distribution of variables for compliance with the law of normal distribution, and identifying the factor structure in the multidimensional space of features. Depending on the type of distribution of the investigated quantitative variables, the results of their statistical processing are represented by the average value and standard deviation M±σ. Frequencies – n (%) were determined for qualitative data.

During the study, the following methods of statistical analysis were used:

- determination of numerical characteristics of variables;
- comparison of parametric data (after checking the number of data for normal distribution using the Kolmogorov-Smirnov and Shapiro-Wilk tests) using the Student's test (for 2 independent elections) and the ANOVA method – for several groups;
- assessment of the significance of differences in quantitative indicators in independent samples: according to the Mann-Whitney U Test – for 2 independent samples and the Kruskel-Wallis test – for several groups. Comparison of paired samples was performed using the Wilcoxon test (for 2 groups);
- to find differences in frequencies, the odds ratio was determined – Odds Ratio (OR), which was calculated as a fraction of the division of the frequency of occurrence of cases in the examined groups. A 95% confidence interval (CI) was calculated for the indicators of OR. An indicator was considered reliable if the CI did not contain an odds ratio value equal to 1;
- assessment of the strength and direction of the relationship between quantitative indicators using the Pearson correlation coefficient (r); the strength of the relationship was assessed by the following values of the correlation coefficient: a value < 0.3 was considered as the absence of a relationship, 0.3–0.7 – a moderate relationship, and > 0.7 – the presence of a strong correlation.

The study used application packages Statistica for Windows v. 8.0 (StatSoft Inc, USA, 2012) in accordance with the recommendations for processing the results of biomedical research.

## RESULTS

Cognitive disorders were established in 36 (76.6%) patients with persistent form of AF, in 33 (82.5%) patients with permanent form of AF, in 31 (91.2%) patients with AVB II-III degrees, in 22 (84.6%) patients with SSS. Mild CD were more often diagnosed in patients with persistent form of AF (OR 1.47, CI 1.13–1.88, p=0.036), severe CD – in patients with permanent form of AF (OR 2.15, CI 1.45–3.32, p<0.001) and patients with AVB II-III degrees (OR 2.62, CI 1.51–4.13, p<0.001) (Fig. 1).

Heart rhythm and conduction disorders are an independent risk factor for cognitive disorders and are characterized by a diffuse deterioration of all cognitive functions. In most cases, these disorders were of moderate and mild degrees.

In patients with a persistent form of AF, a violation of the neurodynamic component of cognitive activity has been established, which do not reach clinically significant CD and do not affect professional and social activity. In patients with a permanent form of AF, violations of regulatory functions come to the fore, forming a polymodal cognitive deficit with a relatively uniform violation of all cognitive functions. In patients with bradyarrhythmias, the leading neuropsychological mechanism of cognitive disorders is the lack of voluntary regulation of activity against the background of a decrease in general mental activity, which is manifested by a significant decrease in cognitive functions and emotional maladaptation, most pronounced in AVB.

Neuroimaging changes were found in 90 (73.8%) patients with CD and in 9 (36%) patients without CD (p=0.037). They were manifested by atrophic changes of the cortex, internal hydrocephalus, a decrease in the

density of the brain substance of the periventricular area, which depended on the duration of the arrhythmia (r=0.42, p=0.026), its severity (r=0.31, p=0.032), the variability of the daily BP rhythm (r=0.36, p=0.035), BP increase episodes (r=0.32, p=0.032). Atrophic changes and internal hydrocephalus occurred more often in patients with severe CD (p=0.04) (Fig. 2).

On the basis of the evaluation of densitometric data, in 45 (36.8%) patients with CD and in 6 (24%) patients without CD, expansion of the ventricular system was established in the absence of changes or with slight expansion of subarachnoid spaces. In 40 (32.8%) patients with CD and in 6 (24%) patients without CD, predominant expansion of the subarachnoid spaces was noted, while expansion of the cerebellar space, frontal and parietal lobes was more common. Combined expansion of the ventricles and subarachnoid spaces was noted in 37 (30.3%) patients with CD and in 5 (20%) patients without CD. At the same time, the frequency of expansion of the ventricular system of the brain among patients with CD significantly exceeded that of patients with severe CD (p=0.033).

Compared to patients without CD, the absolute transverse dimensions of the anterior and posterior horns of the lateral ventricles significantly increased in patients with CD – by 8.7±0,3% and 8.1±0,2%, respectively (p=0.048); indices of the front horns increased by 9.4±1,7% (p=0.036), rear – by 6.5±1,3% (p=0.18). The absolute transverse dimensions of the central parts of the lateral ventricles increased by 8.1±0,4% (p=0.04), the index of the III ventricle – by 25±3,5% (p=0.027), the width of the III ventricle – by 13.1±2,2% (p=0.027), the width IV ventricle – by 8.1±0,7% (p=0.047). The maximum width of the sylvian fissure increased by 23.8±2,9% (p=0.047 and p=0.021), the front sections – by 16.4±2,5% (p=0.047). Expansion of the subarachnoid spaces of the large hemispheres of the brain and cerebellum is more pronounced in patients with severe CD. In patients with CD, a moderate expansion of all sections of the ventricular system and subarachnoid spaces was observed, more pronounced in patients with severe CD (p=0.025).

An increase in the average values of linear indices was found in patients with CD compared to patients without CD. They were uneven: the highest rates were observed in the projection of the parietal lobes (by 1.38 times) and less pronounced – in the projection of the frontal lobes (by 1.2 times). The parameters of the ventriculo-lobular indices, indicating predominantly central atrophy of the brain substance, increased approximately equally in the projection of the parietal and frontal lobes (respectively by 2.3 and 2.4 times) and to a lesser extent in the projection of the temporal lobes (in 1.6 times). The volume of subarachnoid spaces, determined by the value of the subarachnoid-partial indices and which reflects the degree of cortical atrophy of the brain substance, increased with the progression of chronic brain ischemia in the parietal area (by 1.4 times) and to a lesser extent (by 1.3 times ) in the projection of the mediobasal divisions of the temporal lobes. The results in the projection of the parietal lobes were reliable (p=0.034).

Lacunar foci of reduced density with a diameter of up to 2 cm, localized mainly in the deep parts of the white matter of the parietal and frontal lobes of the brain, were found in 29 (23.7%) patients with CD, of them – in 11 (9%) of patients with mild CD (p=0.053) and in 18 (14.8%) patients with severe CD (p=0.027). Lacunar foci occurred in 2 (8%) patients without CD.

In patients with severe CD, multiple hypodense foci were found, localized in almost all departments with a preference in the cerebellar hemispheres, temporal and occipital lobes (Table I). Foci of 1–3 mm indicate the expansion of perivascular spaces as a result of chronic brain ischemia. Their localization is characteristic of damage mainly to the vessels of the vertebro-basilar basin.

Diffuse changes in the white matter of the brain are presented in 57 (46.7%) patients with CD and in 5 (20%) patients without CD by periventricular and/or subcortical leukoaraiosis (LA) and expansion of the periventricular spaces. Subcortical LA was visualized to a greater extent in the occipital and parietal lobes, periventricular LA – in the white matter in the area of the posterior horns of the lateral ventricles (occipital «caps»). In 54 (44.3%) patients with CD, there were signs of periventricular LA, in 33 (27%) patients – subcortical LA, in 43 (35.2%) patients – expansion of the periventricular spaces (Fig. 3).

Intergroup differences related not only to the frequency of detection of LA, but also to the severity of changes in the white matter of the brain. The degree of severity of periventricular LA in 20 (37%) patients with CD reached 1 point, which corresponded to single foci with a diameter of less than 5 mm, limited to the front or back areas. Such changes were considered as a variant of the age norm [14]. Foci with a diameter of 6–10 mm were found in 34 (63%) patients. The expressiveness of periventricular LA in them was estimated at 2 points. In patients without CD, changes in the periventricular substance amounted 1 point in 3 (100%) patients.

In 20 (60.6%) patients with CD, less than 5 small, up to 5 mm in diameter, local subcortical LA foci or less than 2 large, 6–10 mm in diameter, foci were found, which corresponded to 1–3 points. In 10 (30.3%) patients, multiple foci (more than 6, size of 6–10 mm) of subcortical LA were found, which corresponded to 4

points. In 3 (9.1%) patients, a focus with a diameter of more than 10 mm was found, which corresponded to 5 points. In all patients without CD, less than 5 small, up to 5 mm in diameter, local foci or less than 2 large, 6–10 mm in diameter, subcortical LA foci were found, which corresponded to 1–3 points. Differences in the severity of subcortical LA between patients with severe CD and those without CD were significant (p=0.034) (Fig. 4).

We determined the influence of heart rate level (according to the results of daily blood pressure monitoring) on the severity of periventricular LA (r=0.34, p=0.041) and subcortical LA (r=0.37, p=0.038) in the frontal lobes of the brain. The influence of blood pressure on the degree of severity of white matter damage was established in middle-aged and elderly patients: the level of systolic blood pressure was correlated with the severity of subcortical LA in the frontal lobes of the brain (r=0.42, p=0.007), and the level of diastolic blood pressure was correlated with the severity of widespread subcortical LA (r= 0.34, p=0.046) and subcortical LA in the occipital lobes of the brain (r=0.41, p=0.008).

The presence of small vascular foci in the white matter of the large hemispheres of the brain was associated with the daily BP rhythm of the «non-dipper» type (r=0.35, p<0.05). At the same time, the frequency of small foci in «non-dipper» was 2.1 times higher (OR 2.14, CI 1.43–3.00, p=0.012). The presence of small vascular foci was associated with the average night time index of systolic blood pressure (r=0.38, p=0.04). With increased systolic blood pressur load at night, the frequency of small vascular foci in the white matter of the brain was 1.8 times higher (OR 1.81, CI 1.46–2.24, p=0.002). Thus, the systolic blood pressur load at night reliably influenced the risk of structural damage to the brain.

Thus, in patients with CD on the background of arrhythmias, compared to patients without CD, brain spiral computed tomography showed lacunar foci with predominant localization in the parietal and frontal lobes of the brain, periventricular and subcortical LA, signs of an atrophic process.

Correlations were established between the expansion of the lateral ventricles and the results of FAB (r= -0.33, p=0.036), DSM (r= -0.4, p=0.024), tests of «10 words» (r= -0, 37, p=0.028), drawing a clock (r= -0.39, p=0.017), undrawn objects (r= -0.35, p=0.025); between the expansion of the periventricular spaces and the «concentration» subtest of DSM (r= -0.36, p=0.041); between the index of the III ventricle and the total score according to the screening scales (MMSE – r= -0.38, p=0.007; FAB – r= -0.32, p=0.032; DSM – r= -0.32, p=0.027); between the index of the III ventricle and the «10 and 5 words» tests (r= -0.35, p=0.038 and r= -0.33, p=0.026, respectively); between the dimensions of the anterior horn of the lateral ventricle on the left / right and the «orientation» subtest of MMSE (r= -0.36, p=0.008 and r= -0.24, p=0.046, respectively); between the sizes of the anterior horns of the lateral ventricle and the tests of «literal associations» (r= -0.43, p=0.004), «categorical associations» (r= -0.38, p=0.008), «undrawn objects» (r= - 0.4, p=0.01), «line orientations» (r= -0.43, p=0.006); between the total temporal index and MMSE (r= -0.34, p=0.037), FAB (r= -0.32, p=0.041); between the ventriculo-partial temporal index and the tests «10 words» (r= -0.33, p=0.036), «connection of numbers and letters» (r=0.35, p=0.028); between the central linear index and the tests «10 words» (r= -0.31, p=0.034), «line orientations» (r= -0.4, p=0.007); between the posterior linear index and the «underdrawn objects» test (r= -0.33, p=0.035).

A correlation was observed between the presence of the LA phenomen and the degree of cognitive decline (r= -0.41, p=0.006). LA is a sufficiently specific pattern for CD vascular genesis.

We established correlations between the severity of LA and the tests: MMSE (r= -0.41, p=0.032), FAB (r= -0.38, p=0.034), DSM (r= -0.38, p= 0.027), «10 words» (r= -0.36, p=0.035), «5 words» (r= -0.35, p=0.028), literal associations (r= -0.31, p=0.041 ), categorical associations (r= -0.39, p=0.035), undrawn objects (r= -0.35, p=0.04), clock drawing (r= -0.36, p=0.028), both blocks of the number and letter association test (r=0.37, p=0.027 and r=0.38, p=0.03, respectively), the Boston naming test (r=0.39, p=0.017).

The presence of single asymptomatic lacunar foci was correlated with the following tests: MMSE (r= -0.32, p=0.028), FAB (r= -0.35, p=0.024), «10 words» (r= -0.39, p= 0.02), literal associations (r= -0.36, p=0.031), categorical associations (r= -0.40, p=0.007), line orientations (r= -0.38, p=0.026), clock drawing (r= -0.36, p=0.035), both blocks of the number and letter association test (r=0.39, p=0.008 and r=0.38, p=0.026 respectively), the Boston naming test (r=0.34, p=0.04).

In addition to determining the correlations, we calculated the OR of CD development for each of the detected pathological neuroimaging signs. The results of univariate logistic regression indicate that external and internal hydrocephalus, periventricular / subcortical LA and the presence of lacunar foci are additional factors that increase the risk of CD in patients with arrhythmias (Table II).

Thus, the pathological processes that develop in the vascular system in arrhythmias cause damage to the actual substance of the brain with the formation of angioencephalopathy. The morphological substrate of these disorders is small focal and diffuse changes mainly in the deep parts of the brain.

## DISCUSSION

The main morphological substrate of CD in patients with arrhythmias is white matter damage in the frontal lobes of the brain, which causes dysfunction of these parts and manifested by subcortical and periventricular leukoaraiosis. The consequence of such changes is the functional isolation of the frontal lobes due to disruption of their connections with other cortical and subcortical cerebral structures [7].

The changes detected during the neuropsychological examination in patients with arrhythmias, as well as the nature of brain changes according to spiral CT (moderately expressed LA, single lacunae in the absence of pronounced brain atrophy) give grounds to assume the presence subcortical frontal dysfunction as the main mechanism of CR development. The basis of frontal dysfunction probably lies in the damage of various parts of the brain (due to the formation of «silent» lacunae), as well as the disruption of associative connections between the frontal lobe and the visual hump, basal ganglia, and other parts of the brain [15].

In patients with arrhythmias in the presence of CD, compared to patients without CD, «silent» lacunar infarcts in the frontal lobe of the brain, leukoareosis around the bodies of the lateral ventricles, as well as signs of an atrophic process were detected on spiral CT scans. Therefore, taking into account the more pronounced CD in patients with arrhythmias, it is possible to assume that it is the above-mentioned changes in the brain that play a leading role in the development of cognitive deficits.

Neuroimaging changes in patients with arrhythmias were manifested by mild atrophic changes in the cortex, internal hydrocephalus, and a decrease in the density of the brain matter of the periventricular area, which depended on the duration of the arrhythmia, its severity, the variability of the daily rhythm, and episodes of increased blood pressure. Atrophic changes and internal hydrocephalus occurred more often in patients with moderate CD (p=0.04).

In patients with CD, compared to patients without CD, CT scans of the brain showed lacunar foci with predominant localization in the parietal and frontal lobes of the brain, periventricular and subcortical leukoaraiosis, signs of an atrophic process.

We established inverse correlations between the severity of leukoaraiosis / lacunar foci and the results of most neuropsychological tests. The results of univariate logistic regression indicate that external and internal hydrocephalus, periventricular and subcortical leukoaraiosis, and the presence of lacunar foci are additional factors that increase the risk of CD in patients with arrhythmias.

Neuropsychological changes detected in patients with arrhythmias, as well as the nature of changes in the brain according to spiral CT (moderately expressed leukoareosis, isolated lacunae in the absence of pronounced brain atrophy), suggest the presence of subcortical frontal dysfunction as the main mechanism of CD development. The basis of frontal dysfunction probably lies in the damage of various parts of the brain (due to the formation of «silent» lacunae), as well as the disruption of associative connections between the frontal lobe and the visual hump, basal ganglia, and other parts of the brain. The results of our study confirm the connection between CD and structural changes in the brain (leukoareosis, lacunar infarcts, and subcortical brain atrophy), previously shown in a number of works [16].

## CONCLUSIONS

1. The increase in the severity of cognitive deficits in patients with heart rhythm and conduction disorders is associated with atrophic changes at the cortical-subcortical level (OR 1.61, CI 1.14–2.2, p=0.008), accompanied by the phenomena of internal replacement hydrocephalus (OR 1.84, CI 1.16–2.64, p=0.007), leukoaraiosis [periventricular (OR 3.57, CI 1.54–5.82, p= 0.006) and subcortical (OR 5.68, CI 2.21–9.62, p<0.001)] and lacunar foci (OR 4.68, CI 1.92–7.54, p=0.003), with a predominant localization in the frontal-temporal-occipital lobes, in the area of the visual hump and basal ganglia of both cerebral hemispheres.
2. Leukoaraiosis, localized subcortically, leads mainly to a violation of mnestic function, periventricular – to disorders of attention and praxis. With widespread leukoaraiosis, a whole complex of cognitive disorders is revealed, among which there is a violation of speech function, thinking and memory. The combination of leukoaraiosis with the expansion of the ventricular system of the brain leads to a deterioration in such indicators as praxis and attention.

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