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COGNITIVE DISORDERS IN SCHIZOPHRENIA

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ABSTRACT

Currently, there is evidence that schizophrenia is associated with impairment of many cognitive functions (Green M.F. et al., 2004). They are found in healthy relatives of the first degree of kinship, in patients - already in childhood, in the premorbid condition of the disease, most clearly manifested in high-risk states - psychopathological diathesis (Sheinina N.S., Kotsyubinsky A.P., Skorik A.I., Chumachenko A.A., 2008; Sofronov A.G., Spikina A.A., Savelyev A.P., Pashkovsky V.E., 2011). In the prodromal period of schizophrenia, there is an increase in cognitive deficit associated with morphological and functional changes in the brain, which leads to the development of psychosis (Yanushko M.G. et al., 2014; Welham J. et al., 2009), during which cognitive disorders persist (Lesh TA et al., 2011). Patients with endogenous psychosis, already at the first hospitalization, have impaired cognitive functioning, and in patients with schizophrenia they are most pronounced (Reichenberg A. et al., 2009). While in the ICD-10 cognitive impairments are noted only sporadically as diagnostic criteria (for example, speech impoverishment in schizophrenia), the DSM-V emphasizes the clinical importance of cognitive dysfunctions for verifying psychiatric diagnoses (APA, 2013). The possibility of including cognitive impairments in the number of diagnostic signs of schizophrenia was considered, which was rejected due to the lack of sufficient data to differentiate cognitive function in schizophrenia and other disorders. Cognitive deficits, for which effective remedies are currently lacking (Vingerhoets WA et al., 2013), are associated with the problems of social functioning of patients and functional outcome (Sofronov A.G. et al., 2012; Milev P. et al., 2005) ; Prouteau A. et al., 2005; Torrey EF, 2006), prognosis of schizophrenia (Kahn and Keefe, 2013).

KEYWORDS: *Schizophrenia, Cognitive Disorders, Neurocognitive Deficits, Mental Disorders.*

INTRODUCTION

In recent decades, there has been an increased interest in the neurobiological underpinnings of schizophrenia and cognitive functioning in psychiatry. Neurocognitive deficit (NCD) in schizophrenia is considered as the "third group of symptoms" along with positive and negative disorders [12]. It is believed that neurocognitive deficit largely determines the social and therapeutic prognosis of the disease, and also affects the formation of other psychopathological symptoms [8, 27]. The latest advances in molecular biology, genetics, and neuroimaging, together with the accumulated knowledge about the significant similarity of the clinical manifestations of these two endogenous mental disorders, the commonality of diagnostic and therapeutic principles, make us think about changing our ideas about the dichotomous model of endogenous psychoses [6, 19, 24]. One of the factors that initiated the revision of these concepts are data from studies of cognitive impairments in patients with endogenous psychoses. It was the separation of cognitive deficits into a separate cluster of disorders, first in schizophrenia, and then in affective psychosis, that caused a new wave of interest in neuroimaging research and coincided with the latest advances in molecular genetics, which allowed researchers to gain a new look at many previously considered unshakable postulates ... Cognitive dysfunction, as evidenced by the results of numerous studies, is one of the central links in the etiopathogenesis of schizophrenia, and therefore it can be distinguished into a separate pathological cluster, by analogy with positive and negative symptoms [4, 16]. Cognitive decline in patients with schizophrenia precedes the development of mental illness and is a genuinic disruption in the flow of information processes. The need to take cognitive impairments into account in experimental psychological studies of schizophrenia was noted in many works, for example, E. R. Isaeva showed that for the choice of constructive coping with stress, an adequate cognitive assessment of the social situation is important [42]. In cognitive impairment, according to VG Morogin, it is necessary to take into account the motivational side of cognitive processes in schizophrenia [43]. That is why foreign scientists have focused their main efforts on the study of cognitive processes proper. Researchers emphasize the importance of studying, within the framework of neurocognitive deficit, attention disorders (mainly selectivity), limitations of auditory and visual working memory, decreases in the speed of reaction and activity of mental (information) processes and disorders of executive functions - functions of programming, regulation and control of mental activity [26, 27]. Thus, according to foreign authors, the basic cognitive functions include memory, attention, and performance functions. There are several types of cognitive impairments: - I type of impairment is associated with the functions of attention: concentration, stability and selectivity, decreased purposefulness of activity, etc. - II type of impairment is associated with mnestic functions: visual and auditory speech memory, reduced accuracy when copying, etc. - III the type of impairment includes executive functions: hand-eye coordination, speed of learning, planning and change of attitude, control over activity, speed of sensorimotor reactions [44]. Researchers disagree about the stability of ICD in schizophrenia. Some authors consider neurocognitive deficit to be quite stable and not undergoing changes during the course of the disease [28]. Others believe that the dynamics of cognitive impairment is ambiguous: in some cases, there is an improvement in performance compared to the period of the manifest, in others - further deterioration [11]. In addition, at present, researchers are trying to conduct a comparative analysis of basic cognitive functions in patients with schizophrenia, organic brain lesions, depression and neuroses [45].

Cognitive impairments occur even in the prodromal stage of psychosis, remain relatively stable throughout the course of the disease and largely do not depend on its clinical manifestations and antipsychotic therapy [7]. The parameters of cognitive functioning are important indicators of the level of social functioning of patients with schizophrenia, regardless of their clinical status [10]. A wide range of cognitive functions affected by disease include attention, perception, learning ability, and psychomotor skills [31,32]. Cognitive deficits in schizophrenic patients are also manifested in impaired ability to plan, initiate, and maintain targeted strategies. In the implementation of full-fledged cognitive control, many areas of the cerebral cortex are involved, including the dorsolateral prefrontal cortex, mediafrontal cortex, and parietal regions [30]. Studies on the relationship between the prefrontal cortex and working memory led to the assumption that it is the prefrontal cortex that is the main lesion zone in schizophrenia, which leads to impaired working memory, performance skills, abulic symptoms, and behavioral disorganization. Interacting with the sensory, motor, and subcortical regions of the brain, the prefrontal cortex plays a major role in integrating external information and coordinating the subsequent behavioral response [21, 22]. Cognitive control is, in fact, the ability to maintain an algorithm of appropriate behavior in response to a specific situation that requires priority selection. Weakened cognitive control leads to insufficiency of the corresponding clusters of higher psychological functions. Recent studies highlight preventive and reactive types of cognitive control, within the framework of the theory of "double control mechanism" proposed by T.Braver et al. [2].

The preventive type of cognitive control is described as a mechanism of purposeful retention of information that optimally mobilizes attention, perception, and readiness to respond in advance of an event requiring cognitive tension. At the same time, the reactive type reflects the current, momentary cognitive control when performing tasks. Due to its relationship with sensorimotor regions, the dorsolateral prefrontal cortex plays a central role in maintaining a preventative type of cognitive control, which is reflected in increased blood flow when performing appropriate cognitive tests on functional MRI. Reactive control, in turn, is associated not only with an increase in blood flow in the dorsolateral prefrontal cortex, but also in the anterior cingulate gyrus [18]. In healthy volunteers, when performing tasks, the preventive type of cognitive control predominates. Patients with schizophrenia are more likely to include reactive control mechanisms at the initial test performance, including due to insufficient perfusion in the prefrontal cortex. Later, when the tasks are repeated, the prefrontal cortex is activated, leading to a change in the type of cognitive control to the preventive one [2,34,38]. In most cases, the debut of psychosis in schizophrenia occurs in adolescence and young age, while cognitive deficit becomes evident long before the clinical manifestations of the disease, even in childhood or adolescence [4, 23]. It is assumed that cognitive deficit increases during the prodromal period along with morphological and functional brain changes, which ultimately leads to the development of psychosis [20]. Such changes lead to disturbances in various neuropsychological clusters [5]. Thus, cognitive deficits can be a predictor of the subsequent development of psychosis. New data on the role of cognitive deficits in the pathogenesis of schizophrenia have caused an increase in the number of studies aimed at studying the pathophysiological mechanisms of cognitive impairment and on possible ways to correct them [39, 40]. Despite all the advances in molecular genetics and biology, progress in the development of drugs to improve cognitive functioning in schizophrenia has not been significant. To date, there is no consensus on

the effect of second-generation antipsychotic therapy on cognitive functioning in patients with schizophrenia, and on the use of adjuvant drugs to correct cognitive deficits [36, 37]. Clinical studies of the possibility of using various stimulants of nicotinic, GABA-ergic receptors are being conducted, however, none of the investigated drugs has been registered as a stimulant to improve cognitive functioning [3, 9]. However, encouraging results are also emerging. So, N.V. Maslennikov et al. [35] describes the positive dynamics in the state of cognitive functioning in depressed patients with schizophrenia after a course of transcranial magnetic stimulation. Comparison of the profile and severity of neuropsychological deficits in patients with schizophrenia and in patients with other endogenous psychoses can make a significant contribution to understanding the pathogenesis of these disorders and to nosological models of psychotic disorders in general. Data from numerous studies indicate that patients with other endogenous psychoses also exhibit abnormalities in the profile of cognitive functioning [33, 29]. Most studies compare cognitive deficits in schizophrenia and bipolar disorder. There is sufficient evidence that cognitive impairment is common in patients with affective psychosis. For example, one study indicated that cognitive impairment occurs in 84% of patients with schizophrenia, 58.3% of patients with depression with psychotic symptoms, and 57.7% of patients with psychotic symptoms in the context of bipolar disorder [25]. Some studies argue that patients with schizophrenia have more pronounced neurocognitive deficits [25], in other studies, differences in the severity of cognitive impairment between patients with schizophrenia and bipolar disorder with psychotic manifestations have not been identified [14, 15]. Psychotic symptoms in the clinical picture of bipolar disorder are a factor aggravating cognitive deficits. One meta-analysis confirmed the evidence that patients with psychotic depression have more manifestations of cognitive deficits than those with depression without psychotic symptoms [29]. Despite numerous studies of cognitive functioning in bipolar and unipolar affective disorder, there is still no consensus on the neuropsychological profile characterizing affective psychoses. Currently, there are three main hypotheses of cognitive impairment in affective psychosis.The first of these, the so-called "diffuse", provides that patients with depressive disorder suffer from global or diffuse cognitive decline [17]. The second is the hypothesis of specific cognitive decline, which suggests that depressive disorder is associated with a marked decrease in specific cognitive parameters, mainly executive function and memory [1]. According to the third hypothesis, patients with major depression experience cognitive deficits when performing tasks that require cognitive efforts, while they do not show cognitive decline when performing automatic tasks. Automatic cognitive functioning implies a response in response to a stimulus, while tasks requiring cognitive tension include attention functions and cognitive abilities in general in response to presented tasks [13]. Recent studies have demonstrated disagreement in the assessment of cognitive impairment in patients with depression, since none of the cognitive functions characterizes this particular pathology. Moreover, not all patients show the same severity of cognitive deficits. Differences in research results are explained by different methodological approaches, such as the inclusion in the study of patients with different degrees of severity of depression, different clinical subtypes of the studied conditions.

Based on the analysis of literature data and the results of numerous studies of neurocognitive deficits, it can be stated that no convincing differences in neurocognitive deficits in patients with schizophrenia, affective disorders, and organic brain lesions have yet been identified. In general, despite the popularity and high density of scientific research in the field of neurocognitive

deficit, this phenomenon remains poorly understood and, unfortunately, is practically not used in the work of Russian psychologists. In this regard, it should be noted that at present, the methodological approaches to the study of disorders of mental processes in Western clinical psychology and Russian psychology differ significantly. The founders of Russian pathopsychology and neuropsychology A.R. Luria, L.S.Vygotsky, A.N. Leontyev, B.V. Zeigarnik, B.G. quantitative measurements. “For a long time, clinics were dominated by the method of quantitative measurement of mental processes, a method that was based on Wundt's psychology. Investigation of the decay of any function consisted in establishing the degree of quantitative deviation from its “normal standard”, ”wrote BV Zeigarnik [46]. In the Russian school of pathopsychology, the founder of which was B.V. Zeigarnik, violations of the functions of memory, attention, thinking are combined into a system of the leading symptom complex (schizophrenic, organic, etc.), in each of which there is a "core" of violations. Foreign researchers are based on a quantitative measurement of individual basic mental functions.

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