ISSN 0362-1197, Human Physiology, 2015, Vol. 41, No. 4, pp. 372–377. © Pleiades Publishing, Inc., 2015.
Original Russian Text © V.V. Shulgovskiy, M.V. Slavutskaya, I.S. Lebedeva, S.A. Karelin, V.V. Moiseeva, A.P. Kulaichev, V.G. Kaleda, 2015, published in Fiziologiya Cheloveka, 2015, Vol. 41, No. 4, pp. 37–43.

Saccadic Responses to Consecutive Visual Stimuli in Healthy People and Patients with Schizophrenia

V. V. Shulgovskiy[a], M. V. Slavutskaya[a, b], I. S. Lebedeva[b], S. A. Karelin[a, b], V. V. Moiseeva[a, c], A. P. Kulaichev[a], and V. G. Kaleda[b]

[a] *Moscow State University, Moscow, 199992 Russia*
[b] *Mental Health Research Center, Russian Academy of Medical Sciences, Moscow, 115522 Russia*
[c] *Centre for Cognition and Decision Making, National Research University Higher School of Economics, Moscow, 101000 Russia*
e-mail: mvslav@yandex.ru

Received January 27, 2015

Abstract—We investigated cognitive functions of attention and decision-making in 18 healthy subjects and 15 schizophrenia patients using an experimental design with consecutive presentation of two short visual stimuli (double-step). In patients with schizophrenia, an increase in the number of errors and change in the pattern of saccadic responses have been found: an increase of the number of two-saccade responses to each stimulus and a decrease in the number of single-saccade responses to the second stimulus. In schizophrenia patients, the latent period of the first of a pair of saccades has been shorter; and the latent period of single saccade has been increased in comparison with healthy subjects. Opposite lateral differences in latent periods of saccades in healthy subjects and schizophrenia patients have been found. Our results show the deficit of cognitive oculomotor control and a decrease in prognostic processes of saccade programming in schizophrenia patients.

Keywords: attention, programming of saccades, latent period, decision-making, double step, schizophrenia

DOI: 10.1134/S0362119715040143

Saccadic eye movements are used as a natural model in the investigation of the effect of cognitive functions on various behavioral processes. Deficit of saccadic movements is a marker of a number of mental disorders associated with cognitive impairment, including schizophrenia [1–3].

Numerous data provide evidence of a close connection between eye movements, attention and decision-making, and anatomic and functional overlap between structures that control these processes at all levels of the brain [4–7]. In psychophysiology, cognitive functions of attention and decision-making are considered to be individual steps in the programming of a saccade, which takes place during the latent period [8–9].

The connection between attention and decision-making has been shown in many clinical studies. In patients with lesions in the prefrontal cortex, difficulties in the selection of response and behavioral strategy are observed in addition to attention deficits; i.e., the decision-making process is impaired [10, 11]. However, very few studies are focused on the decision-making in schizophrenia patients [12, 13].

One of the approaches in the studies of decision-making is the double-step experimental paradigm [14, 15]. In this paradigm, two short visual stimuli are presented; the subject has to make saccadic response to them. Two types of responses are possible: two consecutive saccades in response to each stimulus and one saccade in response to the second stimulus.

It is hypothesized that a single-saccade response to the second stimulus occurs if the second stimulus is presented before a decision to respond to the first stimulus was made; the first saccade becomes "reprogrammed" as a response to the second stimulus [15]. The double-step test allows evaluating the latent period of a saccade that corresponds to the stage of decision-making.

The aim of this study was to compare saccadic responses in the double-step test in healthy subjects and schizophrenia patients in order to use the results as markers of the decision-making process in the analysis of local EEG potentials in the latent period of a saccade.

METHODS

The study was carried out on 13 healthy volunteers (8 men and 5 women at ages from 19 to 24 years) and 15 schizophrenia patients (men at an age from 19 to 26 years), right-handed and with normal or corrected vision. All subjects gave their informed consent to participate in the study. The protocol of the study was approved by the bioethical committees of Moscow State University and Mental Health Research Center of the Russian Academy of Medical Sciences and met the ethical principles of the World Health Organization (Helsinki Declaration) for human research.

The group of patients included subjects diagnosed with juvenile episodic schizophrenia (F20 in ICD10) with a disease duration no longer than five years from the onset of initial symptoms. All patients received individual medication therapy and were clinically stable. They were studied at the remission stage or during the development of remission.

Horizontal eye movements were registered as a bipolar electrooculogram (EOG). Non-polarizable electrodes 10 mm in diameter were placed at the outer border of the eye sockets. Electroencephalogram (EEG) was registered from 24 cortical areas using the 10–20 system. A joint ear electrode was used as a referent. The sampling rate was 512 Hz; the signal was filtered at 80 Hz; the time constant for EEG recording was 1 s; for EOG, 0.5 s.

The subjects sat in a chair with a head support in a dark chamber. Visual stimuli, white dots 0.2° in diameter, were presented on a black screen 60 cm away from the eyes of the subject. Five visual stimuli were used: the central fixing stimulus (CFS) and four peripheral visual stimuli (PVSs) placed 3° and 7° to the left and right from the central stimulus on a horizontal line.

To focus the attention of the subjects on the experiment, the self-initiation method was used: the subjects initiated presentation of stimuli by a click on a PC mouse button with their right hand (Fig. 1). A fixation stimulus lasting for 800–1000 ms appeared in the center of a monitor 100 ms after the click. The first peripheral stimulus (PVS1) was presented simultaneously with the end of the fixation stimulus; its duration was 150 ms. In 90% of cases, the first stimulus appeared 7° to the left or to the right from the fixation stimulus.

The second peripheral stimulus (PVS2) lasted for 80–120 ms and appeared simultaneously with the end of the first stimulus in the opposite hemifield 3° from the CFS (the pulse-overshoot scheme). 600–700 ms after the end of the second stimulus, it was presented again for 500–700 ms; then, its brightness was two times reduced (muting). The duration of the muting was 600 ms.

The subjects were instructed to press the button of the mouse with their right hand and look at the central stimulus. When the consecutive peripheral stimuli appeared, the subjects should move their gaze to them as fast as possible (to perform saccades). After the muting of the second stimuli, the subject was to release the button of the mouse and look at the center of the screen. If the subject did not release the button during the period of muting or released it before muting, the recordings were excluded from the analysis.

In order to reduce the monotony of the experiment, in 10% of cases, the first stimulus was presented 3° to the left or right of the CFS, and the second stimulus was presented 7° from the CFS in the contralateral hemifield. The responses to these stimuli were not analyzed.

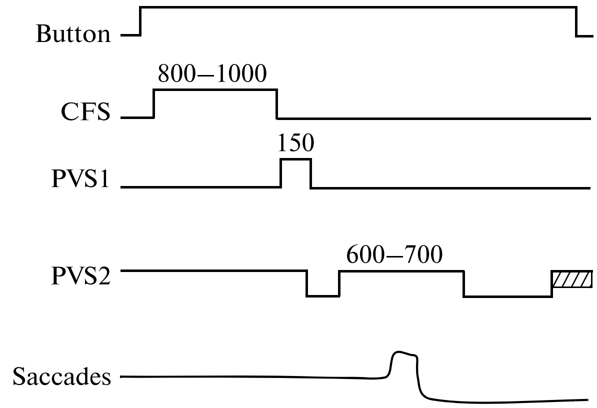

Fig. 1. The experimental design of the double-step visual stimuli presentation test. A hatched rectangle shows muting of the second stimulus after the repeated presentation of the stimulus. CFS is for central fixation stimulus; PVS, peripheral visual stimulus.

Each healthy subject was presented with 350 to 500 stimuli; patients were presented with 300 to 400 visual stimuli during a single experiment. The stimuli appeared in sets of 50 stimuli. Behavioral parameters of the response (the pattern and the number of mistakes) and the latent period of saccades were evaluated.

A CONANm=1.5 integrated system was used to manage the experimental data, collect, store, and analyze the results. The search of saccades and calculating of their latent period were carried out automatically by the original SACCADE SEARCH software. Saccades with latent periods of 85 to 400 ms for the first of the pair of saccades or a single saccade in response to the second stimuli and 85 to 500 ms for the second saccade were included in the analysis.

The statistical analysis of the results was carried out using the STADIA 8.1 software. The relationship between parameters was evaluated using the two-way analysis of variance (ANOVA, saccade type × the number of subjects). The studied factors included the pattern (two saccades in response to both stimuli of a single saccade in response to the second stimulus), laterality (rightward or leftward saccade), and group (healthy subjects or schizophrenia patients). The significance of differences between mean values was calculated using the nonparametric Wilcoxon test (W). Significance of differences between frequencies was evaluated using the Z test.

The analysis of sex-related effects on the latent period in healthy subjects was carried out by comparing the mean values of the latent period of saccades in response to the first visual stimulus in men and women. No statistically significant differences were found (198.1 ± 3.1 and 196.7 ± 3.1 ms respectively, $p >$

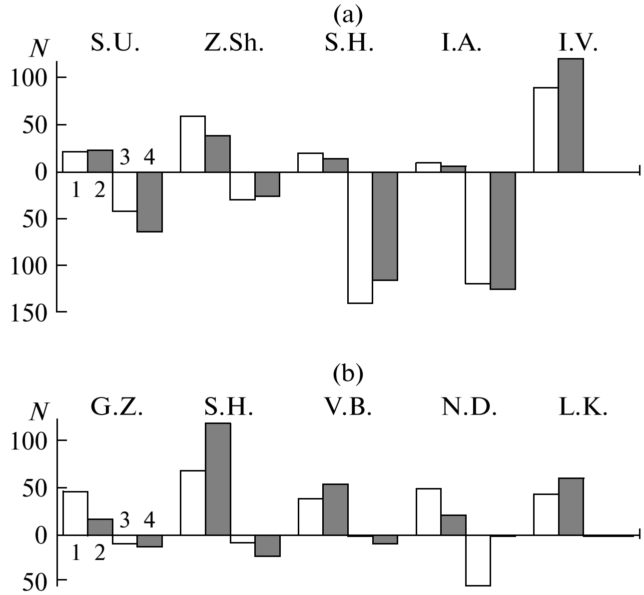

Fig. 2. Individual parameters of saccadic response patterns: (a) in healthy volunteers S.U., Z.Sh., S.H., I.A., and I.V., and (b) in schizophrenia patients G.Z., S.H., V.B., N.D., and L.K. *N* is the number of saccades. White columns show leftward saccades; hatched columns show rightward saccades. Columns above the *X*-axis show the number of two-saccade responses; under the *X*-axis, the number of single-saccade responses to the second stimuli in the contralateral hemifield.

0.05). Therefore, we could compare this group to the group of patients.

RESULTS AND DISCUSSION

The analysis of the results showed a significant difference in the number of errors and the pattern of saccadic responses (two saccades to each stimulus or one saccade to the second stimulus) in healthy subjects and schizophrenia patients.

Among healthy subjects, only three made practically no errors such as failure to respond. A significant difference in number of errors was found between the schizophrenia patients (30 ± 10%) and healthy subjects (17 ± 9%, $W = 139_{(14, 12)}$, $p = 0.005$).

Two-saccade responses to the consecutive stimuli were found in the schizophrenia patients more often (72% and 48%, $Z = 4.25$, $Z = 4.25$, $p = 2.2 \times 10^{-5}$), while healthy subjects responded with single saccades to the second stimulus more often than the patients (52% and 28%, $Z = -3.47$, $p = 0.0005$). In five schizophrenia patients and two healthy subjects, two-saccade responses to the consecutive stimuli prevailed; and single saccades appeared in less than 5–10% of cases. Individual patterns of the saccadic responses are shown in Fig. 2.

ANOVA showed that the factors "pattern × group" and "direction × group" affected the latent period of saccadic responses.

In the case of two-saccade responses in schizophrenia patients, the latent period of the first saccade decreased by 55.2 ± 7 ms for leftward saccades ($F = 251.4$ (871), $p = 2.8 \times 10^{-21}$) and by 72 ± 9 ms for the rightward saccades ($F = 83.87$ (602), $p = 5.3 \times 10^{-18}$) in comparison with healthy subjects (Fig. 3a).

The latent period of single-saccadic responses to the second stimulus increased in schizophrenia patients in comparison with healthy subjects (by 129 ± 6 ms for the leftward saccades, $F = 219$ (347), $p = 8.78 \times 10^{-19}$; and by 103 ± 5 ms for the rightward saccades, $F = 0.53$ (301), $p = 0.606$) (Fig. 3b).

In seven healthy subjects, single saccades in response to the second stimulus had an express latency (from 85 to 130 ms) in 46% of cases; in the subject V.D., all single saccades had an express latency (114.5 ± 0.9 ms). In schizophrenia patients, single saccades with express latency appeared less often (in 8% of cases in six patients, Z = 10.98, $p = 0$). The patients K.Z. and N.D. were an exception, who had express saccades in 67% of cases (latent period 120 ± 1.4 ms). However, in schizophrenia patients, express latency of the first saccade from a pair of saccades appeared more often than in healthy subjects (36% of cases and 9% of cases respectively, $Z = 6.73$, $p = 1.7 \times 10^{-11}$).

The latent period of the response to the second stimulus increased in comparison with the saccade to the first stimulus in the case of two-saccade response in both healthy subjects ($F = 57.23$ (545), $p = 1.26 \times 10^{-16}$ for the leftward saccades; $F = 142.6$ (675), $p = 1.3 \times 10^{-19}$ for the rightward saccades) and schizophrenia patients ($F = 508.9$ (629), $p = 3.19 \times 10^{-21}$ for the leftward saccades; $F = 26.27$ (620), $p = 1.288 \times 10^{-13}$ for the rightward saccades). However, in schizophrenia patients, this increase was greater than in healthy subjects (139 ± 21 ms and 25 ± 7 ms for the leftward saccades, and 156 ± 25 ms and 78 ± 9 ms for the rightward saccades, respectively) (Fig. 3c).

The latent periods of a two-saccade response to the first stimulus and a single-saccadic response to the second stimulus were different in healthy subjects and schizophrenia patients: the latent period of a single saccade in response to the second stimulus was shorter by 49 ± 8ms in healthy subjects, and longer by 151 ± 5.8 ms in schizophrenia patients than the latent period of the first saccade of a two-saccade response ($F = 12.2$ (785), $p = 6.2 \times 10^{-10}$ and $F = 219$ (347), $p = 8.78 \times 10^{-19}$, respectively). However in two patients, single saccades had an express latency in half of cases.

ANOVA showed the significance of the factors "laterality" × "group" in both healthy subjects and schizophrenia patients. However, the lateral differences in the latent period of saccadic responses were opposite in healthy subjects and patients. In most healthy subjects, the latent period of leftward saccades was shorter than of the rightward saccades by 16.4 ± 2.4 ms for the first saccade of the two-saccade response ($F = 15.47$ (1296), $p = 3.6 \times 10^{-14}$) and by 13.8 ± 3.0 ms for a single-saccade response to the second stimulus ($F = 12.2$ (785), $p = 6.2 \times 10^{-10}$) regardless the type of a saccade. In schizophrenia patients, the latent period of the first saccade of a two-saccade response to the right decreased by 15.2 ± 2.4 ms ($F = 219$ (347), $p = 8.78 \times 10^{-19}$); and the latent period of a single saccade to the right decreased by 23.8 ± 9 ms ($F = 7.2$ (145), $p = 5.4 \times 10^{-7}$) in comparison with leftward saccades.

Therefore, this study showed significant differences in behavioral saccadic responses and their latent periods between healthy subjects and schizophrenia patients.

An increase in the number of incorrect saccades in schizophrenia patients in comparison with healthy volunteers was previously shown under various experimental conditions and was considered to be a marker of schizophrenia [16]. The putative cause of this increase in the number of errors was the frontal cortex dysfunction in schizophrenia patients, which is well described in clinical, electrophysiological, and functional magnetic resonance studies [17, 18].

In our study, we found an increase in the number of two-saccade responses to both stimuli and a decrease in the number of single-saccadic responses to the second stimulus in schizophrenia patients; at the same time, the latent period of the first saccade of the two-saccade response decreased in comparison with healthy subjects.

Previously a decrease in the latent period of the saccadic response to visual stimuli was found in schizophrenia patients in comparison with healthy subjects. Several hypotheses of the mechanism of this phenomenon were proposed including the impairment of sensory filtration [19], a decrease in the time of sensory processing [20], intensification of attention disengagement and deterioration of attention engagement [21], and the impairment of voluntary endogenous attention due to a decrease in the top–down control from the dorsolateral frontal cortex and weakening of its hippocampal projections [22].

We suggest that the programming of the first saccade of the two in the double-step paradigm can be more rapid in schizophrenia patients. The decision-making phase ends before the presentation of the second stimulus, which is necessary for the two-saccade response according to the hypothesis of Becker and Jurgens [15].

An increase in the latent period of the first saccade of the two in comparison with the first saccade was found in both schizophrenia patients and healthy subjects. This increase was more prominent in patients than in healthy subjects. We suppose that this phenomenon occurred due to a number of reasons.

First, there is a hypothesis that the programming of the second saccade in the double-step paradigm includes the remapping of its retinal vector depending on the information about the sight position, which is received from the oculomotor structures as an efferent copy of the motor command of the first saccade (corollary discharge) [23]. The impairment of reafferent feedback in the motor system was found in schizophrenia patients [24]. This impairment could also take place in the oculomotor system and slow down the programming of the second saccade.

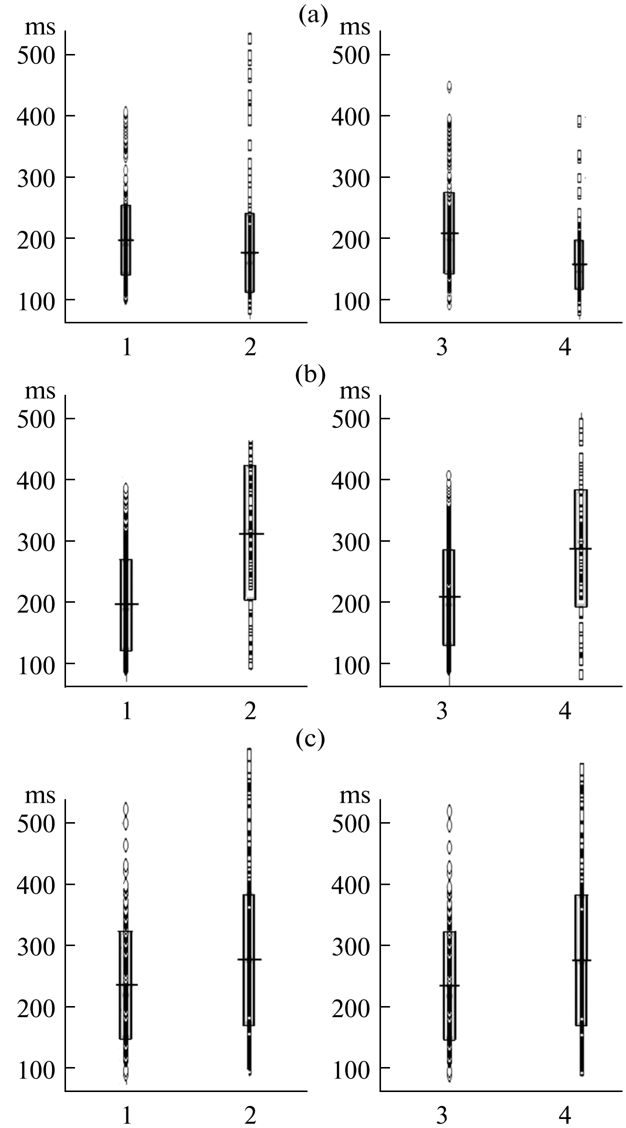

Fig. 3. Diagrams of the latent period (LP) distribution (whisker boxes) of the (a) two-saccade response to the first stimulus, (b) single-saccade response to the second stimulus, and (c) two-saccade response to the second stimulus. (1) Leftward saccades in healthy subjects; (2) leftward saccades in schizophrenia patients; (3) rightward saccades in healthy subjects; (4) rightward saccades in schizophrenia patients. LP values of all subjects were averaged. Vertical rectangles show standard deviation values. Horizontal mark in the center of a rectangle indicates the mean value.

In addition, in the modification of the double-step paradigm used in our study, execution of the second saccade required reorientation of attention from one visual hemifield to the other, which is difficult for schizophrenia patients [25]. The mechanism of this phenomenon is believed to be related to the reduction of the monoaminergic terminals involved in the control of selective attention and activation in schizophrenia [26].

We found a decrease in the number of single saccades in schizophrenia patients and a significant increase in their latent period in comparison with healthy subjects. In healthy subjects, single saccades often had express latency, and their latent period was shorter in comparison with the first saccade. In patients, on the contrary, a significant increase in the latent period was found in comparison with the first saccade in a two-saccade response.

In 1979, Becker and Jurgens found that some healthy subjects respond with single saccades to the second stimulus in the double-step test [15]. They suggested that these subjects used a strategy of minimization of muscle effort by responding only to the second stimulus. The strategy of minimization of muscle effort requires activation of prediction processes related to the motor attention and the preliminary selection of the motor program from the memory during the pre-stimulus period. The interconnected regions of the prefrontal cortex, hippocampus, and basal ganglia are the substrate of these processes. Their dysfunction is found in schizophrenia patients [27]. Our results showed that this strategy appeared less often in schizophrenia patients, which could indicate the impairment of prediction processes in these patients.

Interesting results were obtained after the analysis of lateral differences in the latent period of saccades in healthy subjects and healthy volunteers. In healthy subjects, all types of saccades were characterized by a decrease in the latent period of left saccades in comparison with right saccades. This lateralization was found in other experimental paradigms: anti-saccadic, cost–benefit, and memory-guided saccade paradigms [28]. These paradigms are characterized by an increase in spatial attention regulated by the right hemisphere [29, 30].

Our results provide evidence of an increase in spatial attention in the double-step paradigm in healthy subjects.

The opposite lateralization of the latent period values were found in schizophrenia patients: the latent periods of both the first and the second saccades were shorter for the rightward saccades than for the leftward saccades. This could reflect the deficit of spatial attention in schizophrenia patients. The disengagement of attention at the right visual hemifield necessary for the generation of the leftward saccade is known to be disrupted in schizophrenia patients. This phenomenon is considered to be one of the markers of schizophrenia [25, 31].

Another interesting finding was made: express saccades appeared more frequently in healthy subjects in the case of single-saccade responses to the second stimulus, and in the schizophrenia patients in the case of the first saccade of the two. In psychophysiology, express saccades are considered to be markers of the attention involvement in the programming of a saccade [8]. The mechanism of express saccades is under discussion. According to one of the hypotheses, they have the subcortical origin with the leading role of the superior colliculus and the involvement of the occipital cortex [9]. Another hypothesis suggests that this phenomenon has the cortical and is related to the prediction processes [32] depending on the frontal cortex, which has direct projections to the brainstem saccadic generator bypassing the superior colliculus.

Our results lead to conclusion that express saccades have different origins in schizophrenia patients and healthy subjects in the double-step paradigm. In schizophrenia patients, express latency of the saccadic response to the first stimulus may reflect the activation of automatic attention involving the superior colliculus. It may depend on the impairment of sensory filtration [19].

In healthy subjects, express latency of the single-saccade response to the second stimulus may be related to the effect of the anticipation during the motor preparation in a pre-stimulus period. The brain substrate of this process is the prefrontal cortex [10, 17].

CONCLUSIONS

Experiments with presenting visual stimuli in the double-step test have shown differences in the response pattern, number of errors, and distribution of the latent period values in healthy subjects and schizophrenia patients.

The results of this study provide evidence of the impairment of the relationship between attention and decision-making and disruption of prediction in the saccade programming in schizophrenia patients. Further analysis of EEG parameters and topography related to stimulus presentation or the beginning of saccadic response in the latent period of the saccade and during the pre-stimulus period would provide additional information about neurophysiological mechanisms of cognitive control of the saccade programming in healthy subjects and schizophrenia patients.

ACKNOWLEDGMENTS

This study was supported by the Russian Foundation for Basic Research, project nos. 12-04-00719 and 14-04-01634.

REFERENCES

1. Broerse, A., Crawford, T.J., and Boer, J.A., Parsing cognition in schizophrenia using saccadic eye movements: A selective overview, *Neuropsychologia*, 2001, vol. 39, p. 742.
2. Camchong, J., Dyckman, K.A., Austin, B.P., et al., Common neural circuitry supporting volitional saccades and its disruption in schizophrenia patients and relatives, *Biol. Psychiatry*, 2008, vol. 64, no. 12, p. 1042.
3. Benson, Ph.J., Beedy, S.A., Shphard, E., et al., Simple viewing tests can detect eye movement abnormalities that distinguish schizophrenia cases from controls with exceptional accuracy, *Biol. Psychiatry*, 2012, vol. 72, p. 716.
4. Coull, J.T., Neural correlates of attention and arousal insights from electrophysiology, functional neuroimaging and psychopharmacology, *Progr. Neurobiol.*, 1998, vol. 55, p. 343.
5. De Haan, B., Morgan, P.S., and Rorden, Ch., Covert orienting of attention and overt eye movements activate identical brain regions, *Brain Res.*, 2008, vol. 1204, p. 102.
6. Eimer, M., Van Velzen, J., Cherry, E., and Press, C., Erp Correlates of shared control mechanisms involved in saccade preparation and in covert attention, *Brain Res.*, 2007, vol. 1135, p. 134.
7. Kable, J. and Glimcher, P., The neurobiology of decision: Consensus and controvercy, *Neuron*, 2009, vol. 63, p. 733.
8. Becker, W., Saccadic eye movements as a control system, in *The Neurobiology of Saccadic Eye Movements*, Wurtz, R.H. and Goldberg, M.E., Eds., Amsterdam: Elsevier Sci. Publ. BV (Biomedical Division), 1989, p. 13.
9. Fischer, B. and Breitmeyer, E., Mechanisms of visual attention revealed by saccadic eye movements, *Neuropsychology*, 1987, vol. 25, p. 73.
10. Miller, E.K. and Cohen, J.D., An integrative theory of prefrontal cortex function, *Annu. Rev. Neurosc.*, 2001, vol. 24, p. 167.
11. Kennerly, S.W. and Walton, M.E., Decision making and reward in frontal cortex: Complementary evidence from neurophysiological and neuropsychological studies, *Behaiv. Neurosci.*, 2011, vol. 125, no. 3, p. 297.
12. Brown, J.K., Waltz, J.A., Strauss, G.P., et al., Hypotetical decision making in schizophrenia: The role of expected value computation and "irratational" biases, *Psychiatry Res.*, 2013, vol. 209, no. 2, p. 142.
13. Paulus, M.P., Frank, L., Brown, G.G., and Braff, D.L., Schizophrenia subjects show intact success-related neural activation but impaired uncertainty processing during decision-making, *Neuropsychopharmacology*, 2003, vol. 28, no. 4, p. 795.
14. Lisberger, S., Fuch, A., King, W., and Evinger, L., Effect of mean reaction time on saccadic responses to two step stimuli with horizontal and vertical components, *Visual Res.*, 1975, vol. 15, p. 1021.
15. Becker, W. and Jurgens, R., An analysis of the saccadic system by means of double step stimuli, *Vision Res.*, 1979, vol. 19, no. 9, p. 967.
16. Klein, C., Rockstroh, B., Cohen, R., and Berg, P., Contongent negative variation (CNV) and determinants of the post-imperative negative variation (PINV) in schizophrenic patients and healthy controls, *Schizophrenia Res.*, 1996, vol. 21, p. 97.
17. Goldman-Rakic, P.S., Topography of cognition: Parallel distributed networks in primate association cortex, *Ann. Rev. Neurosci.*, 1988, vol. 11, p. 137.
18. Everling, S. and Fischer, B., The antisaccade: A review of basic research and clinical studies, *Neuropsychology*, 1998, vol. 36, no. 9, p. 885.
19. Lijffijt, M., Lane, S.D., Maiera, S.L., et al., LP50, N100, and P200 sensory gating: Relationships with behavioral inhibition, attention, and working memory, *Psychophysiology*, 2009, vol. 46, no. 5, p. 1059.
20. Strelets, V., Faber, P.L., Golikova, J., et al., Chronic schizophrenics have shortened EEG microstate durations, *Clin. Neurophysiol.*, 2003, vol. 114, no. 11, p. 2043.
21. Nestor, P.G., Faux, S.F., McCarley, R.W., et al., Attention cues in chronic schizophrenia. abnormal disengagement of attention, *J. Abnormal Psychol.*, 1992, vol. 101, p. 682.
22. Spencer, K.M., Nestor, P.G., Valdman, O., et al., Enhanced facilitation of spatial attention in schizophrenia, *Neuropsychology*, 2011, vol. 25, no. 1, p. 76.
23. Sommer, M.A. and Wurts, R.H., What the brain stem tells the frontal cortex: II. Role of the SC-MD-FEF pathway in corollary discharge, *J. Neurophysiol.*, 2004, vol. 91, p. 1403.
24. Bender, S., Schroder, J., Freitag, C., et al., Movement-related potentials point towards an impaired tuning of reafferent sensory feedback by preceding motor activation in schizophrenia, *Psychiatry Res.*, 2012, vol. 202, no. 1, p. 65.
25. Maruff, P. and Hay, D., Asymmetries of the covert orienting of visual spatial attention in schizophrenia, *Neurophysiology*, 1995, vol. 33, no. 10, p. 1205.
26. Liotty, M., Dazzi, S., Fox, P.T., and Laberg, D., Deficits of automatic orienting of attention in schizophrenic patients, *J. Psychiatr. Res.*, 1993, vol. 27, no. 1, p. 119.
27. Selemon, L.D. and Goldman-Rakic, P.S., The reduced neuropil hypothesis: A circuit based model of schizophrenia, *Biol. Psychiatry*, 1999, vol. 45, no. 1, p. 17.
28. Slavutskaya, M.V. and Shulgovskiy, V.V., Presaccadic brain potentials in conditions of covert attention orienting, *Span. J. Physiol.*, 2007, vol. 10, no. 2, p. 277.
29. Bragina, N.N. and Dobrokhotova, T.A., *Funktsional'nye asimmetrii cheloveka* (Functional Asymmetries in Humans), Moscow: Meditsina, 1988.
30. Posner, M., Orienting of attention, *J. Exp. Psychol.*, 1980, vol. 32, p. 3.
31. Moran, M.J. and Thaker, G.K., Covert visual attention in schizophrenia spectrum personality disordered subjects: Visuaspatial cuing and alerting effects, *J. Psychiatric Res.*, 1996, vol. 30, no. 4, p. 261.
32. Pratt, G., Visual fixation offsets effects both the initiated and the kinematics feature of saccades, *Exp. Brain Res.*, 1998, vol. 118, p. 135.

Translated by E. Suleymanova