

Impairment of Gaze Fixation and Holding in Patients with Neurocirculatory Asthenia Syndrome and Associated Vestibular Dysfunction

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The study was performed with a hardware-software complex for the monitoring of human motor functions. Saccadic eye movements and vestibulo-oculomotor interactions were studied in patients with neurocirculatory asthenia and associated vertigo. No changes in saccadic eye movements were found in these patients. Abnormalities of cervico-vestibulo-oculomotor interactions were manifested in the impairment of gaze fixation and holding.

Key Words: *vestibulo-oculomotor interaction; vertigo; neurocirculatory asthenia syndrome; gaze holding*

Vertigo is one of the most common symptoms of various somatic, neurological, and mental diseases. An illusory sense of vertigo is associated with the mismatch between brain signals from vestibular sensory receptors and other sensory organs (eyes, proprioceptors of the neck muscles, *etc.*) [4].

Vertigo is classified into vestibular (systemic) and non-vestibular (non-systemic) vertigo. All types of non-vestibular (inorganic) vertigo (sense of alcoholic intoxication, faintness, ataxia, syncope, pre-collapse, sense of oscillating motions of the objects, abnormal vision, blackout, and movements or stirring in the head) are related to dysregulation and discoordination between the suprasegmental and segmental autonomic centers. This state is followed by functional discoordination in various systems of the body and polymorphous clinical manifestations of the disease [3].

We examined the patients with neurocirculatory asthenia syndrome (NAS). Non-vestibular vertigo is a major symptom of this disease. Vestibulo-oculomotor interactions were evaluated in these patients due to difficulties in the diagnostics of diseases that cause vertigo (as a symptom of the disease).

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MATERIALS AND METHODS

We examined 16 patients with NAS and associated vestibular dysfunction. The control group consisted of healthy volunteers. The age of the subjects was 19-34 years. All patients were also examined by an otolaryngologist, neuro-otologist, and neurologist to exclude organic diseases of the brain (MRT and EEG), diseases of the neck vessels (Doppler ultrasonography of the neck vessels), neurological diseases, endocrine diseases, blood diseases, and craniocerebral injury in the case history. The patients were emotionally labile. Vertigo was mainly observed after stress, mental work, and overstrains. It was accompanied by a sense of anxiety. The degree of vertigo was reported to increase in thick air. Vertigo was not systemic.

Saccadic eye movements and cervico-vestibulo-oculomotor interactions were studied during the performance of directed horizontal movements in response to visual stimulation. This study was performed with a patented hardware-software complex for the evaluation of human motor functions [2]. This complex allows us to monitor and record the path of movement of the eyes and head. Parameters of these movements are analyzed in the follow-up period. Eye movements (saccades) in test 1 were studied with an

electro-oculogram (EOG). Test 2 was conducted to record horizontal head movements in a leftward and rightward direction (with the maximum amplitude). The subject was asked to maintain gaze on a stationary target. Head movements were also recorded in test 3. However, the subject should maintain gaze on the target that moved synchronously with head movement (at the same speed and direction).

The amplitude and frequency of head movements, asymmetry index of head movements, and deviation of the left and right eyes from gaze fixation on the target were recorded in the case of gaze fixation and holding on a stationary target (test 2) or moving target (synchronously with head movements, test 3). The amplitude of head movements was estimated as the difference between the maximum and minimum levels of the curve. The asymmetry index of head movements was calculated (relative value) as the ratio of the time of right-to-left head movement to the time of left-to-right head movement. Deviation of the left and right eyes from gaze fixation on the target was calculated as the difference between the maximum and minimum positions. The difference between the amplitudes of head movements and eye movements, as well as the difference between the zero line and significant changes (bursts) in EOG were estimated over several epochs of observations (30-50 sessions). These epochs were sufficient for the statistical analysis.

The results were analyzed by Statistica 6.0 software.

RESULTS

Studying the path of saccadic eye movements in healthy volunteers (normal) and NAS patients revealed a similarity and synchrony of eye movements in test 1 (only saccades). It was typical of all saccades. No significant differences were found in the latency and

duration of saccades in healthy volunteers and NAS patients ($p > 0.05$). The specific electrophysiological feature of saccades in NAS patients was a greater intraindividual variability (standard deviation) of the latency and, to a lesser degree, of the duration of saccades (as compared to those in healthy volunteers; Table 1).

For steady fixation and holding of the gaze on a stationary target, the head and eyes of the subject should move with the same speed in the opposite directions. The optimal ratio of the speed of eye movements to the speed of head movements (gain) approaches 1. This index in young healthy volunteers is 0.81-1.01 [2].

Two tests for gaze holding were performed. Figure 1 shows the paths of movements in the gaze-holding test with a stationary target. Healthy volunteers were able to perform well the eye movements and head movements (good fixation of the target). In NAS patients, the eyes failed to stay fixed to the target. Deviations of the eyes in various directions were found in patients with NAS (Table 2).

The speed of movement of the left and right eyes was lower than the speed of head movement. Hence, the phases of eye movements did not coincide with those of head movements. Discoordination between eye movements and head movements is accompanied by a poor fixation of the gaze.

The visual target moved simultaneously with head movements in test 3 (Fig. 2). Healthy volunteers were characterized by gaze fixation on the target. EOG appeared as a relatively straight line. In NAS patients, the eyes lost this target and returned to the former position (Table 3).

The shift of the right and left eye from gaze fixation on the target was 3-fold higher in NAS patients as compared to healthy volunteers. The results of both tests indicate that gaze fixation and holding on the

TABLE 1. Saccadic Eye Movements in Test 1 ($M \pm m$)

Parameter		Normal	NAS
Rightward saccades of the left eye	latency	176.7 \pm 9.1	183.6 \pm 25.6
	duration	103.3 \pm 11.6	105.4 \pm 16.4
Rightward saccades of the right eye	latency	181.8 \pm 8.4	185.2 \pm 24.4
	duration	112.1 \pm 10.9	110.3 \pm 14.5
Leftward saccades of the left eye	latency	165.6 \pm 7.2	177.0 \pm 18.7
	duration	106.4 \pm 7.8	104.5 \pm 9.2
Leftward saccades of the right eye	latency	174.9 \pm 8.2	173.2 \pm 15.5
	duration	104.0 \pm 7.5	100.1 \pm 9.4

Note. $p < 0.05$ (paired t -test).

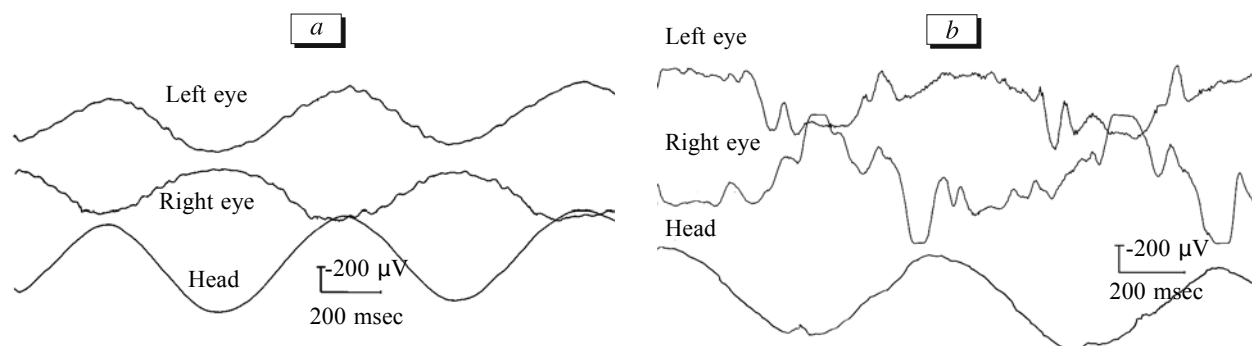


Fig. 1. Gaze holding on a stationary target during head movement in the horizontal plane (from one shoulder to another): healthy volunteer (a) and NAS patients with vertigo (b).

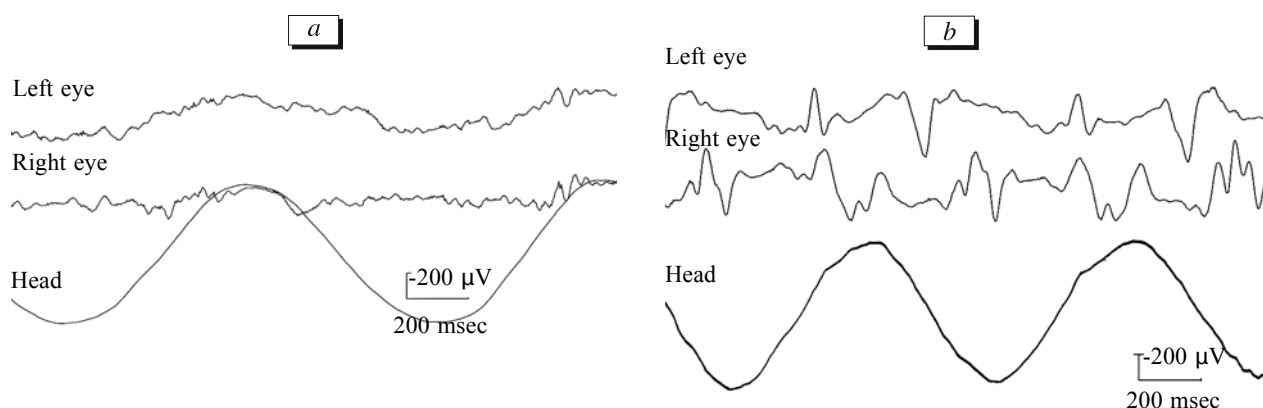


Fig. 2. Gaze holding on the target that moves with the same speed and direction as the head (horizontal movement from one shoulder to another): healthy volunteer (a) and NAS patients with vertigo (b). Calibration amplitude for eye movements in EOG.

TABLE 2. Estimated Parameters for Movements of the Head and Eyes in Studying the Gaze Fixation and Holding on a Stationary Target (Me)

Group	AHM, degrees	FHM, Hz	C_{as}	PELE, degrees	PERE, degrees
Healthy volunteers (normal)	77.7 (75;83.5)	1.4 (1.21;1.5)	0.98 (0.94;1.1)	6.1 (2.5;9.3)	7.4 (3.1;9.9)
NAS patients	78.2 (72;84.2)	1.3 (1.2;1.3)	0.97 (0.93;1.2)	16.9* (11.5;28.5)	19.8* (13.9;31.6)

Note. Here and in Table 3: AHM, amplitude of head movements; FHM, frequency of head movements; C_{as} , asymmetry index of head movements; PELE, phase error of the left eye and head; PERE, phase error of the right eye and head. The lower and upper quartiles are shown in brackets. * $p < 0.05$ compared to normal (Mann-Whitney test).

TABLE 3. Estimated Parameters for Movements of the Head and Eyes in Studying the Gaze Fixation and Holding on the Target that Moves Horizontally and Synchronously with Head Movement (Me)

Group	AHM, degrees	FHM, Hz	C_{as}	DevLE, degrees	DevRE, degrees
Healthy volunteers (normal)	100.9 (88.2;108.5)	1.4 (1.2;1.6)	1.0 (0.95;1.6)	9.0 (7.1;11.3)	7.5 (6.2;9.6)
NAS patients	99.1 (89.2;108.1)	1.5 (1.3;1.7)	1.01 (1.0;1.2)	25.4 (21.5;33.3)	24.1 (19.2;31.4)

Note. DevLE, deviation of the left eye from gaze fixation on the target; DevRE, deviation of the right eye from gaze fixation on the target.

target during simultaneous movements of the head and eyes are impaired in these patients.

The performance of eye movements (relative to head movements) is associated with the vestibulo-oculomotor reflex. Nerve pulses that are transmitted by the vestibular nerve from the semicircular canals serve as the input signal for this reflex. The reflex is realized due to coordinated activity of the vestibular nuclei, oculomotor muscles, and neck muscles.

The increased excitability of nonspecific brain structures and functional imbalance in the segmental apparatus (sympathetic [cervical sympathetic trunk] and parasympathetic autonomic nervous system) and suprasegmental structures (limbic-and-reticular system) in NAS patients contribute to the impaired interaction between these structures and vestibular nuclei during neck movements. This state is accompanied by loss of the ability to coordinate movements of the ocu-

lomotor and cervical muscles. Studying the vestibular and oculomotor system with a new hardware-software complex for the evaluation of human motor functions allows us to identify these changes in test patients. Abnormalities of cervico-vestibulo-oculomotor interactions are manifested in the impairment of gaze fixation and holding in NAS patients with vertigo.

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