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Saccadic and Smooth-Pursuit Eye Movement in Neurootological Diagnostic Procedures

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Abstract: The origin of the generation of saccadic and smooth-pursuit eye movements has not been proved. The goal of the study reported here was to search for the connection of visuooculomotor reactions—saccades and eye-tracking test—with cerebellar damage. Using electronystagmography, we tested 11 patients treated with aminoglycosides because of pneumonia. After a mean of 21 days of pharmacological therapy, a neurological examination revealed cerebellar disability in all cases. On the basis of our previous studies, we suspected cortical Purkinje cell damage. In all tested patients, saccadic movements were disturbed: We observed overshoot, undershoot, and prolonged latencies. In seven patients, eye-tracking test results were not correct: We noted the degradation of the sinusoidal pattern most often. The results pointed to a connection between the generation of these two tested visuooculomotor reactions with cerebellar diseases possibly located in the cortex.

Key Words: cerebellum; electronystagmography; eye-tracking test; saccadic movements

Saccadic and pursuit eye movements have been used as clinical tests for many years. Few controversies surround hyper- and hypometric saccades and eye-tracking disturbances in relation to central nervous system lesions and side preference [1]. Hypometria of the saccadic movement is present in neurodegenerative diseases and intoxication. Hypermetria is observed in cerebellar lesions, where delayed saccades are caused by cerebral cortical disturbances [2].

The smooth-pursuit eye-tracking test is useful in evaluating drug toxicity, central nervous system tumors, posterior or middle cerebral artery thrombosis, encephalitis, whiplash injury, and parkinsonism [3]. Both tests (saccades and eye-tracking) are rarely pathological in peripheral vestibular damage.

Kornhuber [4] stressed that lack of saccadic accuracy is very often cerebellar and that, in such patients, smooth-pursuit eye movement is normal. That effect was observed in Louis-Bar's syndrome in children and in cerebellar cortical atrophy in elder patients. In contrast, disorders of smooth-pursuit eye movements were absent after cerebellar cortical lesions but present in cases of damaged cerebellar nuclei or their connections with the brainstem [5]. The aim of our study was to evaluate saccadic and smooth-pursuit eye movement in patients with cerebellar lesions.

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PATIENTS AND METHODS

Eleven patients participated in our study (eight women, three men; ages 37–46 years; mean age, 41.2 years). All patients were treated for bacterial pneumonia with aminoglycosides (gentamicin), 160 mg/day for 20–25 days (mean, 21 days). Two to three weeks after therapy, vertigo and postural disturbances began in all patients. Audiological and vestibular examination showed the absence of hearing loss; caloric test results were normal as well. Cerebellar function evaluation revealed slight disturbances in the Romberg test, the finger-nose test, diadochokinesis, and the walking test. On the basis of our knowledge of aminoglycoside's negative influence on Purkinje cells, we postulated a cerebellar cortical lesion in all cases.

Saccadic and smooth-pursuit eye-tracking tests were recorded using the electrovideonystagmography system. Results from both eyes were recorded simultaneously. We used a moving light target programmed to turn on

and off sequentially. In the saccadic test, changes of point position (left to right) were in 1-second increments. The tracking test fixation point velocity was 20 degrees/sec. The parameter called *gain* was calculated, comparing the target velocity and eye velocity during smooth pursuit. The ideal value of gain is 1, which means that eye velocity is equal to stimulation velocity.

RESULTS

We did not observe normal saccades in any of our patients. In ten patients, we observed an overshoot and prolonged latencies, whereas in one patient, we observed undershoot and pathological prolonged latency.

In the eye-tracking test, normal smooth pursuit was present in four patients. In the seven patients in whom the eye-tracking test results were abnormal, we saw degradation of the sinusoidal pattern to the saccadic pattern in five and, in two, production of a sinusoidal pattern accompanied by deformation of purity on both sides of the eye movement. The gain in these two patients was from 0.5 to 0.7.

DISCUSSION

In a peripheral vestibular lesion, strong spontaneous nystagmus can produce abnormalities of pendular eye movement [3]. Smooth-pursuit and saccadic disorders are present mainly in central vestibular lesions.

In our patients, the cause of the cerebellar symptoms, in our opinion, was degeneration of Purkinje cells after aminoglycoside use. This type of degeneration was observed in our laboratory during experimental studies with pigeons, and our findings, among others, were presented during the Neurootological and Equilibriometric Society (NES) Congress in Haifa in 1997 [6,7]. Our study confirms the role of cerebellar cortical lesions in producing saccadic abnormality. Conversely, in the majority of cases, smooth-pursuit disturbances also were registered. Possibly, the area of lesion was not limited to the cortical hemisphere in our patients.

The influence of cortical damage on smooth pursuit is equally possible. Dionne et al. [8] observed in Charlevoix-Saguenay spastic ataxia, which highlighted vermis and vestibulocerebellar involvement, disorders seen in eye-tracking test results together with a lack of saccadic accuracy but without changes in saccadic velocity. Oyanagi et al. [9] reported the loss of Purkinje and granular cells in vermis cortex after organic mercury intoxication. The damage of such localization was followed by eye-tracking disturbances. Total ablation of the cerebellum proved to be manifested by disturbances of both saccades and sinusoidal eye movements [10]. Nedzelski [11] reported interesting results: He observed saccadic abnormalities in medium and small cerebellopontine angle tumors. Nedzelski excluded cerebellar surface compression and cortical damage. We suggest that saccadic and smooth-pursuit tests show a central origin of lesion, although detailed topodiagnostic definition is limited.

CONCLUSIONS

Saccadic and smooth-pursuit eye movement tests are useful in diagnosing cerebellar disorders, although saccadic movements were more often disturbed than were eye-tracking test results in cerebellar lesions. Saccadic disorders may be strictly connected with cortical cerebellar damage, which was suspected after aminoglycoside intoxication. However, controversy regarding the origin of abnormalities seen in both tests' results necessitates further investigation.

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