Investigation of Betaserc in Auditory and Vestibular Disturbances

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Abstract: Vestibular vertigo is a primary symptom in neurootological clinical practice and is common among cerebrovascular diseases. The aim of this investigation was to evaluate the effect of betahistine dihydrochloride (Betaserc), 8 and 16 mg, on patients who were transport system workers with vascular auditory and vestibular disturbances. We examined 50 patients, 30 of whom were treated with 16-mg doses and 20 of whom received 8-mg doses of Betaserc. The mean age of the patients was 37 ± 2.3 years. The following evaluative methods were used: questionnaire including detailed neurootological history; ear, nose, and throat and neurological examinations; tonal threshold audiometry; and examination of the vestibular system (spontaneous and provoked reactions). On the basis of the investigations carried out, we recommend 16 mg Betaserc three times daily in the acute phase. The medication is very well tolerated, has no sedative effect and is suitable for long-term treatment.

Key Words: Betaserc; electronystagmography; nystagmus; posturography; vascular auditory and vestibular disturbances

The complicated perception of body position and its separate parts in space is a result of analyzed and synthesized impulses by the vestibular analyzer, by both superficial and deep proprioception, and by the ocular analyzer. Vestibular receptors play a main role in this complex system and determine changes both in linear and angular acceleration and in gravity strength [1-3]. Pathological changes in functioning of the aforementioned systems cause vertigo [4], dizziness, a sensation of floating, noise in the ears, and balance disorders.

Cerebrovascular disease forms the main cause among complaints such as these. Arterial hypertonia or atherosclerosis is accepted to be the most common etiopathogenic factor. Sensory structures, being highly differential, are more vulnerable to ischemia than are other nerve structures [5,6]. The main symptom in neurootological clinical practice—vestibular vertigo—is common among disturbances of cerebral hemodynamics and the blood supply of peripheral and stem parts of the vestibular analyzer. This finding led us to use the medicine Betaserc (betahistine dihydrochloride), which has both peripheral and central effects and brings to normal the imbalance in the vestibular system.

Betaserc is a product of Solvay Pharmaceuticals. Its peripheral effect is vasodilation of the inner ear on the microcircular level [4]. In the central ear, it improves histaminergic neurotransmission by blocking presynaptic H3 receptors, it increases histamine release in synapses, and it normalizes the exciting processes in vestibular nuclei of the medulla oblongata [7].

The aim of our investigation was to evaluate the effect of treatment with 8 mg and 16 mg of Betaserc on patients (workers in the transport system) with vascular auditory and vestibular disturbances.

PATIENTS AND METHODS
We examined a total of 50 men: One group of 30 men received 16 mg of Betaserc and another group of 20 men received 8 mg of Betaserc (mean age, 37 ± 2.3 years). The patients, all transport workers, presented with vascular pathology of the vestibular and auditory analyzer. The otoneurological investigation was conducted before and on the twentieth and the forty-fifth days of treatment in the Department of Neurology and Neurootology of the National Multiprofile Transport
Hospital, King Boris III, Sofia. During the investigation, patients were given either only 16 mg of Betaserc three times daily or 8 mg of Betaserc three times daily, as those with arterial hypertonia underwent simultaneous antihypertensive treatment.

A detailed neurootological history was obtained by means of a questionnaire, and otological and neurological examinations were conducted in each patient. Tonal threshold audiometry was performed using a Siemens (Copenhagen, Denmark) SD-50 audiometer. Examination of the vestibular system was directed at both spontaneous and provoked reactions.

First, with the help of Frenzel’s spectacles, we evaluated spontaneous reactions, checking for spontaneous, positional, and latent nystagmus. Coordinating tests included Bárány’s tests, in which diversity of more than 3 cm/30 sec in one direction is considered pathological, and statokinetic tests, which were performed using static statobilography and the dynamic Unterberger test. Static statobilography using a “TEST-V”-type apparatus (Transport Cybernetics Ltd., Sofia, Bulgaria) was accommodated by the respective software and computer detection of a summary index for stability in a position of closed and open eyes and a coefficient of Romberg (Romberg in square [R/s]). Dynamic Unterberger testing was achieved by way of quantitative computer detection of declinations in the respective direction for 30 seconds, using system U-01/91 (Transport Cybernetics Ltd.).

To assess provoked reactions, a Tönies apparatus was used. Above-threshold reactions were achieved by starting with angle-speed enforcement of 6 degrees/sec²,
steady rotation of 30 seconds, reaching the speed of 90 degrees/sec, and then sudden stop-stimulation.

RESULTS AND DISCUSSION

The examined patients were divided as shown in Figure 1. The total number of patients treated with 16 mg of Betaserc was 30 (18 with central otoneurological syndrome and 12 with peripheral otoneurological syndrome). The total number of patients treated with 8 mg of Betaserc was 20 (12 with central otoneurological syndrome and 8 with peripheral otoneurological syndrome). Vertigo and dizziness were the main symptoms of patients with otoneurological disease, and the symptoms often were accompanied by noise in the ears, hearing decrease, and balance disorders. Very often,

Figure 3. Subdivision of men with spontaneous (SNy), latent (LNy), and positional nystagmus (PNy) (according to Frenzel’s spectacles) during treatment with 8 mg and 16 mg of Betaserc. (Th = therapy.)

Figure 4. Results of investigation of asymmetrical reactions after above-threshold rotatory provocations (by percentage). (SPS = slow-phase speed; Th = therapy.)
patients with dizziness have difficulty in describing their symptoms. Mostly, the complaints are of instability, a sensation of staggering, and giddiness.

Figure 2 shows a decrease in complaints of vertigo (16 patients) and dizziness (14 patients) on the twentieth day in one-half of the 30 patients treated with 16 mg of Betaserc. On day 45, substantial improvement was seen among one-third of the 20 patients treated with 8 mg of Betaserc. Complaints of vertigo remained in 6.7% of the 16 vertigo patients receiving 16 mg of Betaserc, as compared to 20% of the 11 vertigo patients treated with 8 mg of Betaserc. Complaints of dizziness remained in 10% of the 14 dizziness patients treated with 16 mg of Betaserc, as compared to 25% of the 9 dizziness patients treated with 8 mg of Betaserc. The results of the respective treatment of patients with balance disorders, hearing decrease, and noise in the ears are nearly identical.

The frequency of attacks was lower in both treated groups, but the result was considerably better after treatment with 16 mg of Betaserc. The otological and neurological status of all patients was normal before starting treatment. No pathological change was found after tonal audiometry; the results were within normal limits (hearing decrease of 5–10 dB, mainly for the high-frequency zone) for those older than 36 years.

Regarding nystagmus reactions, the general test showed
the presence of different nystagmus forms—horizontal, horizontal rotatory, and rotatory—according to the phase and nosological units. Figure 3 shows the breakdown of patients according to whether they experienced spontaneous, latent, or positional nystagmus (according to Frenzel’s spectacles) and to whether they were being treated with 8 mg or 16 mg of Betaserc.

On the twentieth day of treatment with 16 mg of Betaserc, we noted an improvement in the spontaneous and, especially, the latent nystagmus symptoms. On day 45, these symptoms were barely noticeable (spontaneous nystagmus, 6.7%; latent nystagmus, 3.3%). Positional nystagmus symptoms were less influenced, which could be explained by changes in the cervical spine (i.e., cervical osteochondrosis) proved by roentgenography of the cervical vertebrae. These data correlate with the results of above-threshold provocation, objectified by electronystagmography (Fig. 4). Considering the duration, slow-phase speed, amplitude, and organization of nystagmus, symmetry of reaction is considerably improved after above-threshold provocation and 45 days treatment with 16 mg of Betaserc.

We confirmed the opinion of many scientists that a larger amount of Betaserc leads more rapidly to considerable acceleration of the vestibular function recovery processes [8,9]. Similar results were obtained by using the coordination and statokinetic tests (Fig. 5). As compared to static body changes, better results were registered in kinetic changes on the twentieth day of treatment with 16 mg of Betaserc. These results correlate with the improved subjective complaints about balance disorders.

CONCLUSIONS

Complete investigation revealed decreased complaints of vertigo and dizziness (in frequency and strength of attacks) among one-half of our patients on the twentieth day of treatment with 16 mg of Betaserc. Better results were received after treatment with 16 mg of Betaserc, considering spontaneous and, especially, latent nystagmus symptoms. Also, the symmetry of reaction was considerably improved after above-threshold provocation, mainly in terms of duration, slow-phase speed, and amplitude. Results of coordinating and statokinetic tests were also improved after treatment of patients with 16 mg of Betaserc, as better results were achieved in body kinetic indices.

On the basis of investigations carried out, we recommend 16 mg of Betaserc three times daily in the acute
phase. The medication is very well tolerated, has no
sedative effect, and is suitable for long-term treatment
of transport workers with auditory and vestibular dis­
turbances of vascular etiology.

REFERENCES
1. Blagovestenskaya SN. Otoneurological symptoms and
3. Ivanov I. Vestibular dysfunctions (in Bulgarian). S Med 1
Fick 180, 1982.
4. Aantaa E. Treatment of acute vestibular vertigo. Acta Oto­
5. Balow RW, Harker LA. Central vestibular system disorders.
6. Caplan LR. Vestibular Disease, vol 1. In HJ Barnet, JP
Mohr, MB Stein (eds), Stroke. New York: Churchill Liv­
8. Tighilet B, Leonard J, Lacour M. Betaistine dihydrochloride
treatment facilitates vestibular compensation in the
9. Tighilet B, Lacour M. Distribution of histaminergic axonal
fibres in the vestibular nuclei of the cat. Neuroreport
7:873--878, 1996.