

Neurootological Aspects of Juvenile Vertigo

Beáta Bencsik,¹ Gábor Bencze,² Elemér Nagy,³ Lóránt Heid,⁴
and Claus-Frenz Claussen⁵

¹ Department of Otorhinolaryngology and Head and Neck Surgery, Semmelweis University, ² Central Hospital of Ministry of the Interior, ³ Dr. Nagy & Co. Ltd., Vestibular and Psychological Research Groups, and ⁴ Central Military Hospital, Budapest, Hungary; and ⁵ Neurootological Research Institute of the 4-G-F, Bad Kissingen, Germany

Abstract: Pathologies from childhood to adolescence carry physical, cognitive, motor, linguistic, perceptual, social, emotional, and neurosensory characteristics. The ages between 8 and 14 or 15 especially carry very special traits of a rollover in data processing with respect to balance regulation. Data acquisition of neurootological function provides us with a network of information about the sensory status of our young patients. Major neurootological complaints leading to functional neurootological investigations are vertigo (including giddiness), dizziness, and nausea. These complaints may occur acutely but also are present in some patients at a young age as longer-lasting complaints. Physiological and clinical vertigo syndromes are commonly found as a combination of four principal phenomena: perceptual (vertigo), oculomotor (nystagmus), postural (dystaxia), and vegetative (nausea, vomiting). These four cardinal manifestations of vertigo are related to different levels of the vestibular analyzer and require different methods of investigation. The focus of our study is the phase of restructuring of equilibrium regulation in children between the ages of 8 and 15 years.

Key Words: children; equilibrium; neurootological functional data; nystagmus; vertigo

The differential diagnosis of vertigo in children is extensive. Otitis media and middle-ear effusion could be the most common causes of vertigo in children, but problems exist in detecting the other causes of vertigo. Choung et al. [1] measured 55 children (<16 years old) who had normal eardrums and did not suffer from otitis media or middle-ear effusion, and these authors found that the most common causes of vertigo in children were migraine in 17 (30.9%) and benign paroxysmal vertigo of childhood in 14 (25.5%). Szirmai et al. [2] found that two-thirds of the young patients (<18 years) in their neurootological department had migraine or migraine-equivalent vertigo. The man-

ifestations of the migraine-related vestibular symptoms vary widely, ranging from episodic true vertigo to constant imbalance, movement-associated disequilibrium, and motion sickness.

Uneri and Turkdogan [3] examined 34 children (ages 4–18 years) having paroxysmal dizziness or vertigo attacks or both. Regarding the medical history of the group, chronic headache attacks consistent with migraine were reported in 12 children, and motion sickness was reported in 30 (some patients experiencing both conditions). The authors found spontaneous vestibular nystagmus (41%) and benign paroxysmal positional nystagmus (59%). These authors thought that a considerable number of these vestibular problems might be related to the migraine syndrome (i.e., basically vascular problems).

Examining the prevalence of migraine and migraine equivalents in children of school age (N = 314), Russel and Abu-Arafeh [4] found that benign paroxysmal vertigo is common in children and that, although it is seldom diagnosed, it appears to cause few major problems for the affected children. In common with previous studies, these authors found that benign paroxysmal vertigo appears to be related to migraine. Possible mechanisms that

Reprint requests: Beáta Bencsik, MD, Semmelweis University Faculty of Medicine, Department of Otorhinolaryngology and Head and Neck Surgery, 1083 Szigony u.36, Budapest, Hungary. Fax: (0036)-1-333-3316, E-mail: bencsik@fulo.sote.hu

This research was presented at the Thirty-First Congress of the Neurootological and Equilibriometric Society, Bad Kissingen, Germany, March 26–28, 2004.

can explain the aura in migraine or the paroxysmal vertigo disorder are an ischemia by vasodilatation due to abnormal accumulation of vasoactive substances (prostaglandin, serotonin, tyramine) or a neural dysfunction located in a specific area mediated by neurons containing serotonin [5,6].

Other less frequent causes include trauma [7], Ménière's disease [8,9], cerebellopontine angle tumor [10], seizure, postmeningitic vertigo [11], acute vestibular neuritis, and juvenile rheumatoid arthritis. These findings have been shown to be very different from those related to adult vertigo. The evaluation of vertigo in children requires a questionnaire for extensive and complete history recording, audiograms, and vestibular function tests. In selected cases, electroencephalography, hematological evaluation, and imaging of the brain or temporal bone should be performed.

Metabolic diseases in children can also cause problems for the equilibrium system. Gawron et al. [12] examined children suffering from type 1 (insulin-dependent) diabetes mellitus. They examined 95 children and young adults 6–28 years of age and found that type 1 diabetes causes disturbances in different parts of the vestibular organ but mostly in its central part. The range of vestibular organ impairment seems to depend mainly on the presence and character of hypoglycemic incidents and the duration of the disease (and, to some extent, on the compensation of diabetes).

By stabilography, examined parameters were worse in a diabetic group in comparison to controls, reaching statistical significance in younger children [13], and were better in diabetic patients with a longer history of the disease.

Ménière's disease in children is rare, and its clinical appearance is not as typical as in adults. The triad of vertigo, tinnitus, and deafness are not usually elicited; diagnosis is often made after years of follow-up and batteries of investigations [8,9]. According to some authors, the therapy for Ménière's disease in children is mainly based on drug treatment, but when the disease has progressed, intratympanic gentamicin injections and endolymphatic sac decompression (or, in some cases, nerve section) might be performed subsequently. At the authors' department, the incidence of Ménière's disease in pediatric patients with vertigo was 2.9%.

Acetylsalicylic acid (aspirin) and other nonsteroidal antiinflammatory drugs can also cause tinnitus, deafness, and vertigo [14]. One hypothesis attributes these outcomes to a reactive immunological mechanism, but this hypothesis has been confirmed only in exceptional cases. The theory of the cyclooxygenase pathway, currently the most widely accepted, is based on the ability of nonsteroidal antiinflammatory drugs to inhibit the cyclooxygenase pathway of arachidonic acid metabolism, leading to prostaglandin depletion and an increase in leukotrienes.

In the majority of cases, vestibular dysfunction appears together with cochlear dysfunction. Examining a group of 72 children (ages 4–14 years) with unilateral sensorineural hearing loss of probable viral origin and concomitant vestibular affects, Melagrana et al. [15] found no statistically significant differences between the audiographic findings at the onset of hearing loss and the electronystagmographic findings, but they found a direct correlation between the presence of vertigo or dizziness and electronystagmographic findings.

PATIENTS AND METHODS

We based our evaluation and results on a sample of 35 juvenile patients. All patients complaining of vertigo either with or without hearing loss and tinnitus presented for our neurootological investigation. We performed an inspection for otological causes of the complaints as described. The patients completed an extensive questionnaire of the NODEC III type, and then we performed a series of neurootometric investigations according to our neurootological network system. These investigations consisted of a polygraphic electronystagmography scheme with spontaneous reactions; vestibuloocular reactions to the caloric test; the perrotatory and postrotatory type of optokinetic stimulations; and vestibular cardiac reactions. Simultaneously with electronystagmography, we recorded the cardiac reactions by a three-channel electrocardiogram (ECG) scheme.

The vestibulospinal pathways are controlled by craniocorpography recordings of the standing test [16] and the stepping test [17,18]. We also performed an acoustic analysis: pure-tone audiometry, tympanometry, speech audiometry, and brainstem evoked response audiometry. Sometimes, when children could not cooperate with us or because of their behavior, we had to forgo some investigations. For this reason, the numbers of patients in the investigated group vary.

All the tests are related to standardized test evaluations of the major databanks of NODEC I–IV. They comprise data from approximately 30,000 neurootological patients and normal controls, spanning ten decades of life and both genders. For our study, we transferred all the test results into a special Excel spreadsheet (Microsoft), some of which we used for demonstration here.

RESULTS

The ages of our special young patients ranged from 5 to approximately 15.5 years (mean, 10.5 years; standard deviation [SD], ± 5.27 years). Of these, 29% were male and 71% were female. Their weights constituted a mean of 53.9 kg (SD, ± 16.46 kg), and their heights constituted a mean of 159.19 cm (SD, ± 11.92 cm).

In evaluating the blood pressure of this population, we found an average systolic blood pressure of 90 mm Hg and a diastolic blood pressure of 57.5 ± 3.53 mm Hg. Regarding the history of our patients, we found the parameter frequencies outlined in Table 1. Most of the complaints are related to low blood pressure; however, in a few cases we also found complaints of hypertension. Also, cases with cardiac insufficiency were related to

Table 1. Historical Data of 35 Juvenile Patients

Parameter	Percentage
Background disorders	
Hypotension	71.43
Hypertension	14.29
Cardiac insufficiency	11.43
Diabetes mellitus	2.86
Kidney disorders	2.86
Nephrolithiasis	2.86
Vertigo-triggering factors	
Getting up	28.57
Turning the head	20
Bending	14.29
Gaze mechanisms	8.57
Car	8.57
Train	5.71
Other	11.43
Vegetative or nausea symptoms	
Malaise	34.29
Sweating	25.71
Vomiting	20
Collapse	8.57
Duration of single vertigo attack	
Seconds	34.29
Minutes	28.57
Hours	2.86
Days	2.86
Weeks	2.86
Vestibular symptoms	
Rocking	34.29
Rotating	28.57
Instability	22.86
Blackout	22.86
Falling	8.57
Lifting	2.86
Hearing symptoms	
Hearing loss	57.14
Tinnitus	34.29
Deafness	11.43
Humming	5.71
Whistling	2.86
Ear surgery	2.86
Visual disturbances	
Loss of acuity	34.29
Double vision	17.14
Head and neck trauma	
Traffic accident	8.57
Home accident	8.57
Sports accident	5.71

vertigo and to a low rate of diabetes and kidney disorders. The vegetative and nausea symptoms consisted mainly of malaise. We found higher frequencies of vertigo with rocking movements (as if on board ship) and of rotating sensations (as if riding a carousel) than of blackouts or instability. The most frequent visual complaints were acute loss of visual acuity and double vision. Among the main vertigo-triggering factors were the orthostatic mechanisms of getting up and head turning. Most of the children suffered from vertigo attacks only for seconds' or minutes' duration. Very rare were longer-lasting attacks (hours, days, or weeks).

More than one-half of our sample group complained of subjective hearing loss, which is very frequent for this young age range. We found a relatively high incidence of the symptom of deafness in one or the other ear. Quite a few young patients had head and neck trauma, mostly due to traffic accidents and home accidents and, less frequently, to sports accidents.

The equilibrated, objective, and quantitative investigation results of polygraphic electronystagmography of spontaneous and caloric vestibuloocular nystagmus are shown in Table 2. The central nystagmus frequencies

Table 2. Statistical Evaluation of the Vestibuloocular Caloric Test with Electronystagmography and Electrocardiography (n = 27) and Rotatory Intensity-Damping Test (n = 20)

	Mean	SD
Caloric test (frequencies in Hertz)		
Spontaneous nystagmus, right	0.48	0.41
Spontaneous nystagmus, left	0.35	0.25
44°C, right	1.1	0.49
44°C, left	1.14	0.41
30°C, right	1.22	0.37
30°C, left	1.17	0.52
Electrocardiogram per minute		
Spontaneous electrocardiography	88.24	13.29
44°C, right	86.07	14.98
44°C, left	84.96	13.94
30°C, right	85.03	14.38
30°C, left	85.11	12.49
RIDT nystagmus beat rates per 30 seconds		
Perrotatory nystagmus, right	41.01	16.64
Perrotatory nystagmus, left	41.82	14.94
Postrotatory nystagmus, right	41.68	17.7
Postrotatory nystagmus, left	48.44	18.06
RIDT heartbeat rates per 60 seconds		
Electrocardiography sitting spontaneous, right	80.16	12.45
Electrocardiography sitting spontaneous, left	79.5	12.21
Perrotatory electrocardiography, right	79.45	14.28
Perrotatory electrocardiography, left	80.8	11.89
Postrotatory electrocardiography, right	80.8	12.69
Postrotatory electrocardiography, left	84.5	12.5

RIDT = rotatory intensity-damping test; SD = standard deviation.

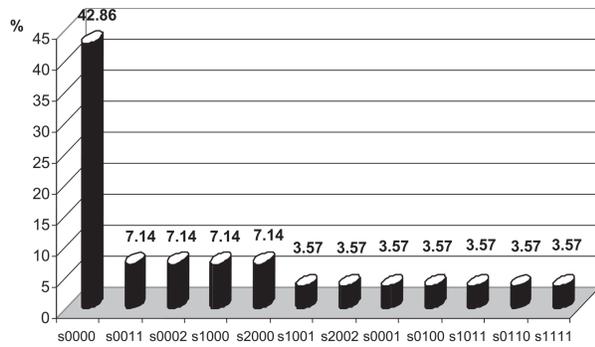


Figure 1. Results of caloric tests. When we synoptically fit together the four results of the caloric tests (right warm, right cold, left warm, left cold), we use trinary coding. If the response was within the normal range, we coded it 0; when it was inhibited, we coded it 1; and when it overshot, we coded it 2. Most frequently (42.86%), the pattern s0000 occurs, which means all four caloric test characteristics were positioned in the normal range. This is followed in frequency by a typical peripheral pattern with a monolateral inhibition of warm left and cold left in 7.14% of subjects. Of equal percentage is the third position in the graph, representing a central disorder with disinhibition of left cold. The fourth bar represents a typical monolateral peripheral receptor lesion s1000 with an inhibition of the right warm and a spontaneous nystagmus to the opposite side. Bar 5 (s2000) stands for a central disorder with a disinhibition of the right warm. In the next stage of s1001, we see a midbrain inhibition of the right-beating nystagmus and, in the pattern s2002 we have a disinhibited nystagmus preponderance pointing at the right temporal lobe, which demonstrates that the dysfunction likely shares peripheral and central sites of origin (n = 27).

follow the established rule: The softest response was for right warm, followed by left warm; the strongest response was for right cold, followed by left cold (Figs. 1 and 2).

During the caloric test, we also studied vestibular cardiac reaction. A spontaneous ECG was defined as an electrocardiographic response during the culmination phase of the nystagmus reaction in the supine position without any stimulation. We saw thereafter that the four caloric responses—ECGs at 44°C right and left and ECGs at 30°C right and left—systematically depicted lowering heart rates, which is systemic and may be due to an action of the vagal system.

The rotatory acceleration test stimulates the right and left inner-ear vestibular canals in opposite directions. We saw few site differences between perrotatory right- and left-beating nystagmus, whereas postrotatory right- and left-beating nystagmus show a major laterality, with higher activities in postrotatory left-beating nystagmus.

During the rotatory intensity-damping test, we recorded the electrocardiographic reactions. Baseline is an electrocardiogram obtained with the subject in a seated position with a spontaneous nystagmus beating to the

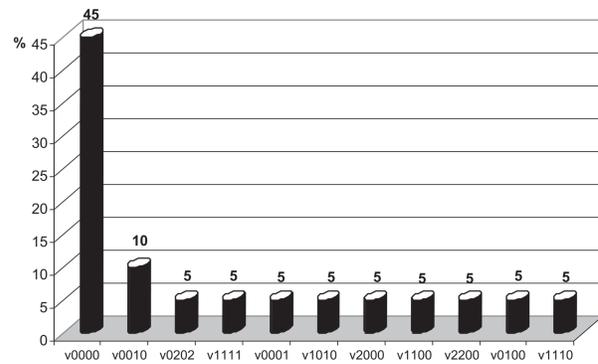


Figure 2. Results of caloric tests. In combining the monolateral caloric warm response as a nystagmus response on a weak stimulus with the perrotatory binaural stimulus as a supraliminal stronger stimulus, we can establish the vestibular response intensity comparison (VSRIC [20]), for which we used trinary coding. We examined 20 children from this group in this way. The three types of VSRIC can again be grouped into three subdivisions each: parallel behavior, recruitment behavior, and decruitment behavior. Most frequently, we found bilateral normal behavior in 45% of our entire sample. This, however, is followed by a monolateral recruiting phenomenon in 10% of our sample (trinary code v0010), demonstrating that peripheral vestibular inhibition can be straightened out and compensated when increasing the power of a stimulus. Other findings were a mixture of disinhibitive recruitments (v0202), parallel inhibition on both sides (v1111), monolateral decruitment phenomenon (v0001), bilateral recruitment (v1010), monolateral overshooting decruitment (v2000), monolateral parallel inhibition (v1100), monolateral parallel disinhibition (v2200), monolateral right decruitment (v0100), and monolateral parallel inhibition with an opposite site recruitment (v1110). We also demonstrate that juvenile vertigo depends not only on peripheral vestibular lesions, which partly are compensated and partly are decompensated, but also on central regulatory deficits.

right or to the left. In the postrotatory reactions, we observed sympathicotonia in postrotatory left-beating nystagmus, which showed a higher release of the ECG rhythm (Figs. 3–5; also see Table 2).

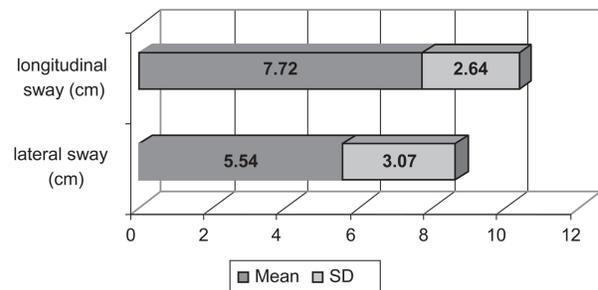


Figure 3. Results of standing craniocorpography (CCG). We recorded the vestibulospinal responses objectively and quantitatively by means of CCG. The results for the standing-test CCGs exhibited the parameters of longitudinal sway and lateral sway. The dimensions are pointing trend-wise to the right (normal) direction (n = 35).

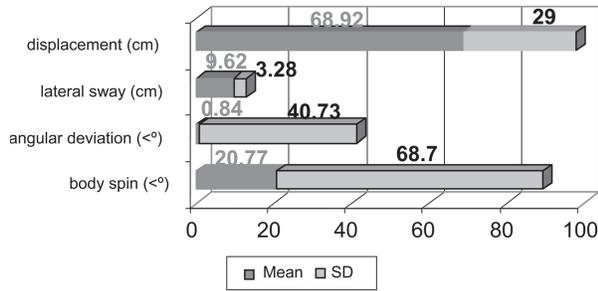


Figure 4. Vestibulospinal test results. The more sensitive test is stepping craniocorpography (CCG), using the stepping test of Unterberger and Fukuda [17,18]. We evaluated the four principle parameters—displacement, lateral sway, angular deviation, and body spin—with mean and standard deviation. All these parameters derive from the arrangement pointing to the right direction; however, the body spin overshoot the normal range (n = 35).

A second field of neurootometry includes audiometry. To compile the best results, we recorded speech audiometry, which gave us an impression of the patients’ social understanding. It is astonishing that the parameters of understanding double-digit numbers and monosyllabic words *and* loss of discrimination are much more pathological in the left ear than in the right ear (Table 3).

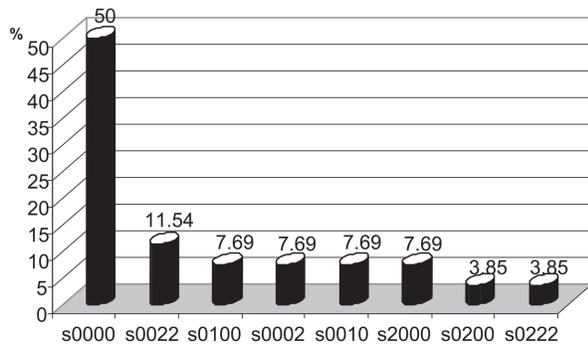


Figure 5. Caloric test responses. We used a trinary coding scheme for the four parameters—displacement, lateral sway, angular deviation, and body spin—depicted in Figure 4. Twenty-six children from our group underwent this type of examination. They are confronted as characteristics with the normal ranges for each of the parameters in our special chart. One-half of our sample demonstrated a normal synoptic pattern (s0000). The second most frequent pattern was s0022, which shows a pathological deviation toward the right, including a pathological body axis spin toward the right. Patterns three and four exhibit minor deficits in the lateral sway or in the lateralization toward the right side, as far as body spin is concerned. The s0010 pattern actually must be included in the group of normal responses. However, overshooting central responses are seen in pattern s2000 and (especially with a centrally enlarged lateral sway) in pattern s0200. Finally, pattern s0222 exhibits a combination of broadened sway with a pathological deviation toward the right side.

Table 3. Statistical Evaluation of Speech Audiometry in 35 Juvenile Patients

Speech Audiometry Parameters	Mean	SD
Right ear		
Numbers	15.76	27.82
Words	61.56	17.58
Loss of discrimination	8	27.6
Left ear		
Numbers	33.79	40.87
Words	71.2	21.56
Loss of discrimination	25.08	40.63

DISCUSSION

Pathology from childhood to adolescence carries physical, cognitive, motor, linguistic, perceptual, social, emotional, and neurosensory characteristics. Associated with ages 8 to 14 or 15 years especially are very special traits of a rollover in data processing with respect to balance regulation.

Physiological and clinical vertigo syndromes commonly are found as a combination of four principal phenomena: perceptual (vertigo), oculomotor (nystagmus), postural (dystaxia), and vegetative (nausea, vomiting). These four cardinal manifestations of vertigo are related to different levels of the vestibular analyzer and require different methods of investigation.

A large number of children suffering from vertigo have additional problems in daily life, such as learning disabilities, reduced attention, decreased endurance and concentration capacity, disturbances of precise motor functions, and hyperactive behavior [19]. Because of the variability in symptoms, no specific therapy exists for children with learning disabilities. Thus, training procedures are of primary importance. These should be individualized to the requirements of an affected child, and the functions that are most markedly affected must be included (e.g., exercises of the static and dynamic equilibrium, training of more complex motions and of memory, tactile and kinesthetic perception). Although the total number of young patients investigated in our study was small (N = 35), we attempted to present a picture of this special group of patients.

REFERENCES

1. Choung YH, Park K, Moon SK, et al. Various causes and clinical characteristics in vertigo in children with normal eardrums. *Int J Pediatr Otorhinolaryngol* 67(8):889–894, 2003.
2. Szirmai A, Ribari O, Repassy G. Migraine related vestibular disorders in childhood and adolescents. *Neurootol Newslett* 6(2):56–59, 2002.

3. Uneri A, Turkdogan D. Evaluation of vestibular functions in children with vertigo attacks. *Arch Dis Child* 88(6): 510–511, 2003.
4. Russell G, Abu-Arafeh I. Paroxysmal vertigo in children—an epidemiological study. *Int J Pediatr Otorhinolaryngol* 49(suppl 1):S105–107, 1999.
5. Skyhoj Olsen T, Friberg L, Lassen NA. Ischemia may be the primary cause of neurological deficits in classic migraine. *Arch Neurol* 44:156–161, 1987.
6. Tusa RJ, Saada AA, Niparko JK. Dizziness in childhood. *Child Neurol* 9:261–274, 1994.
7. Plaza Mayor G, Ferrando Alvarez-Cortinas J, de los Santos Granados G. Pediatric temporal bone fractures. *Ann Otorrinolaringol Ibero Am* 29(3):237–246, 2002.
8. See GB, Mahmud MR, Zurin AA, et al. Vestibular nerve section in a child with intractable Ménière's disease. *Int J Pediatr Otorhinolaryngol* 64(1):61–64, 2002.
9. Akagi H, Yuen K, Maeda Y, et al. Ménière's disease in childhood. *Int J Pediatr Otorhinolaryngol* 61(3):259–264, 2001.
10. Truy E, Furminieux V, Dubreuil C. Acoustic neuroma in children. Report of 5 cases. *Ann Otolaryngol Chir Cervicofac* 116(2):92–97, 1999.
11. Aust G, Lattermann V. Early and Later Lesions in the Auditory and Vestibular System in Children and Juveniles After Meningitis. In *Proceedings of the Twenty-Second Annual Meeting of the NES*. Amsterdam: Excerpta Medica, 1995:301–304.
12. Gawron W, Pospiech L, Orendorz-Fraczkowska K, Noczynska A. Are there any disturbances in vestibular organ of children and young adults with type I diabetes? *Diabetologia* 45(5):728–734, 2002.
13. Gawron W, Pospiech L, Orendorz-Fraczkowska K, Noczynska A. The influence of metabolic disturbances present in diabetes mellitus type I on vestibulo-spinal reflexes in children and young adults. *Otolaryngol Pol* 56(4):451–457, 2002.
14. Porto Arceo JA. Special features of NSAID intolerance in children. *Allergol Immunopathol (Madr)* 31(3):109–125, 2003.
15. Melagrana A, Tarantino V, D'Agostino R, Taborelli G. Electronystagmography findings in child unilateral sensorineural hearing loss of probable viral origin. *Int J Pediatr Otorhinolaryngol* 42(3):239–246, 1998.
16. Romberg M. *Lehrbuch der Nervenkrankheiten*. Berlin: Springer-Verlag, 1848.
17. Unterberger S. Neue registrierbare Vestibularis-Körperdreh-Reaktionen, erhalten durch Treten auf der Stelle. Der Tretversuch. *Arch Ohr Nas Kehlk Heilk* 140:273–282, 1938.
18. Fukuda T. The stepping test: two phases of the labyrinthine reflex. *Acta Otolaryngol (Stockh)* 50:95–108, 1959.
19. Aust G, Schneider D. Vertigo in Children with Disturbances in Central Sensory Perception. In *Transactions of the Twentieth Regular Meeting of the Bárány Society, Würzburg*. Amsterdam: Excerpta Medica, 1998:453–458.
20. Claussen CF. Vestibular compensation. *Acta Otolaryngol Suppl* 513:33–36, 1994.